

40th
Anniversary

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2025 KALAS

40th Anniversary International Symposium

JULY **23** (WED) – **26** (SAT)

📍 ICC JEJU · Jeju Island · KOREA

GIANTS

Global Insights in Advancing Translational Sciences



Organized by



**KOREAN ASSOCIATION
FOR LABORATORY ANIMAL SCIENCE**



MINISTRY OF FOOD AND DRUG SAFETY
**National Institute
of Food and Drug Safety Evaluation**

Sponsored by

KIT 국가독성과학연구소
Korea Institute of Toxicology

KMPC 국가모델동물연구소
Korea Model animal Priority Center

KMEDIHUB
국가중점연구사업
Korea Model animal Priority Center

KBIOHEALTH
오송첨단의료산업진흥재단

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Korea Research Institute of
Bioscience & Biotechnology

KFRI KOREA FOOD
RESEARCH INSTITUTE

KCLAM/KSLAV
한국실험동물수의사회

KRICP
KOREA RADIOISOTOPE CENTER FOR PHARMACEUTICALS

KCFST
Korean Federation of Science &
Technology Societies

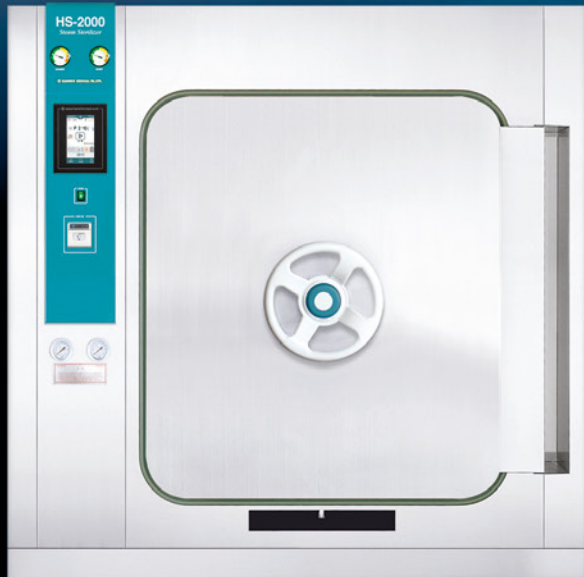
제주관광공사
JEJU TOURISM ORGANIZATION

안전성! 유효성! 내구성!

HANSHIN MEDICAL STERILIZER

50년 멸균기 전문제조기업의 확고한 신념입니다.

한신메디칼은 지난 50년 동안 끊임없는 연구개발과 품질혁신으로
의생명과학(실험동물) 분야의 발전에 적극 헌신하고 있습니다.



Large Steam Sterilizer
HS-2000R(1500~2000L)



Large Steam Sterilizer
HS-1000R(D) (1,073L)



Large Steam Sterilizer
HS-5035PT(350L)



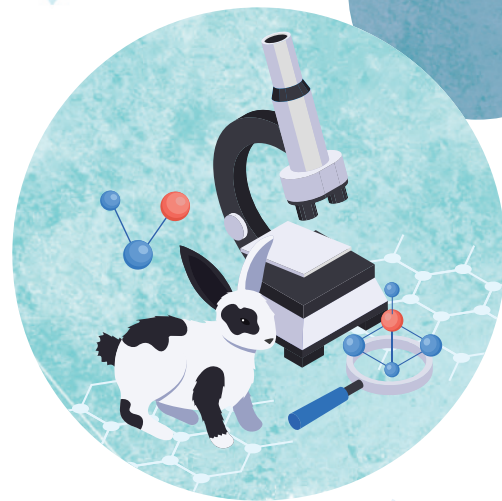
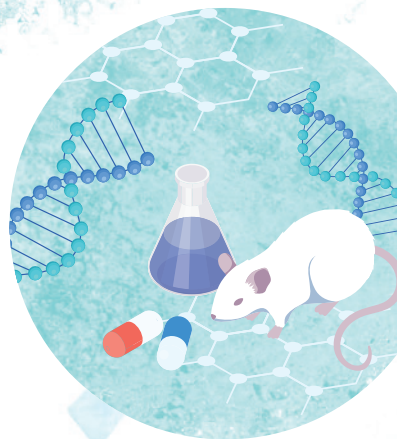
KALAS

www.kalas.or.kr

2026 한국실험동물학회 동계심포지엄

2026. 1. 28.(수) - 1. 30.(금)

강원도 평창 알펜시아 컨벤션센터



주최 |



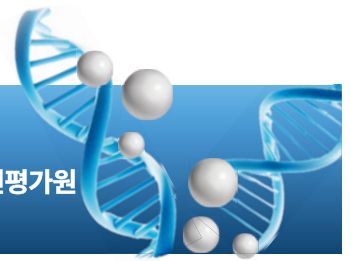
한국실험동물학회



식품의약품안전처

후원 |





실험동물자원은행



주요 업무

- 활용가치가 높은 실험동물자원 기탁·수집
- 실험동물자원 체계적, 안정적 보존 및 정도 관리
- 실험동물자원 이용촉진 및 성과 확산
- 지역별 거점기관 운영

- ▶ 위치 : 대구광역시 동구 침북로 73(대구경북첨단의료복합단지 내)
- ▶ 규모 : 1,134평/지하 1층 ~ 지상 3층
- ▶ 설립 근거 : 『실험동물법』(법률 제18853호, 2003.4.27.)제5조 제1항 3의2

보존 장비



액체질소탱크 초저온냉동고 슬라이드보관장 포르말린보관장 파라핀블록보관장

신청 절차



보유자원 현황 및 주요자원(KNTP 사업)

동결 자원



장기/조직

혈청

세포

고정 자원



포르말린 고정

파라핀블록

슬라이드

디지털 병리 이미지 자원



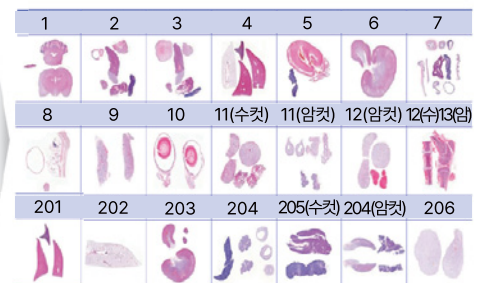
디지털 병리 이미지

주요자원(KNTP 사업)

백수오, 이엽우피소 등 생약재를 투여한

40종의 처치 자원 보유

-파라핀블록
-슬라이드(H&E)
-디지털 병리 이미지(WSI)



보유 실험동물 현황

국산 일반(모체) 쥐 : 4종 Korl ; Korea Laboratory animal

ICR:Korl · 식의약품안전성 유효성 평가에 사용 · 국내 사용량이 많은 자원	BALB/cKorl · 면역질환, 암 연구 등 사용
C57BL/6NKorl · 질환모델동물 제작, 면역학, 독성학 연구 등 사용	DBA/2Korl · 신경질환, 관절 연구 등 사용

국산 질환모델동물

비만 모델 마우스	당뇨 모델 마우스	암 모델 마우스
정상 비만	정상 당뇨	암
신경계 20종	면역계 14종	암 15종
대사계 12종	순환계 8종	피부 3종
호흡기계 2종	기타 11종	

85종



2025년 4월 1일부터 안전성평가연구소가
국가독성과학연구소로 명칭이 변경되었습니다.

국민건강과 안전사회 실현을 위한 글로벌 독성 연구기관

고품질 데이터는 고품질 **실험동물**에서 시작됩니다.



주령/체중
지정 가능



100마리 이상
대량 주문 가능



전국 빠른
배송 가능



중앙실험동물(주)는 아래와 같은 서비스를 제공합니다.

실험동물

- Inbred / Outbred (B6, BALB/c 등)
- 면역부전 (nu, NKG, SCID, BRGSF)
- 질환모델 (노화, 대사질환, 아토피 등)
- 유전자변형
- 수술 모델

사료

- 일반 chow사료
- Research Diet 조제사료 (76A, Western, Atherogenic)
- Dyets 조제사료 (Ethanol, Control)
- 주문제작

시약

- 아토피 유발 진드기 시약
- 알러지 유발 시약 (pollen, Insect)
- 관절염 유발 / 측정 시약
- ELSIA kit
- 동물대체 안점막 / 피부자극

실험용품

- 동물 수술 실습 모형 (개, 돼지, 원숭이, 소동물 피부 등)
- 실험장비 및 도구
- 소도구
- 깔짚 / nestlet



QR코드를 스캔하여
더 많은 마우스 정보를 확인하세요!



중앙실험동물(주)
Central Lab. Animal Inc.

www.LabAnimal.co.kr

Tel: 02-3461-5255 Fax: 02-3461-4274

ONE STOP SOLUTION

1

동물반입을 위한 청정화 및 첨단사육시스템

첨단 동물 사육 시스템

규모 : 5,440 m², IVC Cage
수용 능력 : 35,000 Mice, 3,500 Rats, 50 Guinea pigs, 24 Rabbits, 10 Mini pigs



실험동물 청정화 및 고속증식 서비스



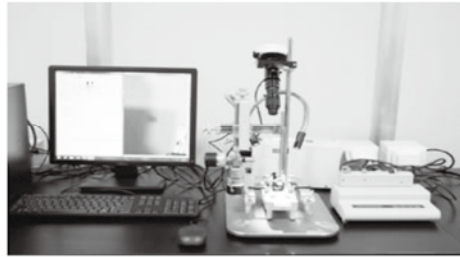
청정화 및 고속증식

자원동결 및 복원

2

소·중동물 모델별 전용 수술/처치 장비 및 시설

Stereotaxic Surgery Station



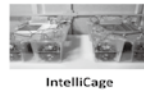
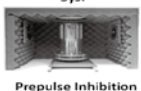
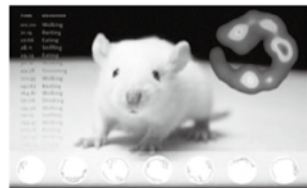
Middle-Animal Surgery System



3

다면적 표현형 분석 장비 및 활용지원 (행동/대사/생체 이미징)

소동물행동분석



Grip Strength

Treadmill

Rotarod

LABORAS

Phenotypic

In vivo 생체 이미징



Micro-CT (PerkinElmer)

IVIS Spectrum (PerkinElmer)

대사사육실험분석



Oxyman-CLAMS (Collins)

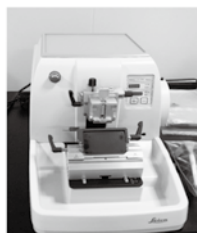
4

생체 유래 조직분석 장비 및 기술지원 (조직배양/부검/조직병리)

조직병리분석



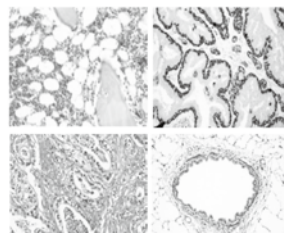
밀폐형 조직처리기(Leica)



회전식 조직절편기(Leica)



조직포매기(Leica)



조직절편 결과물(예시)

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National Medical Research Center

K BIOHEALTH
오송첨단의료산업진흥재단
Osong Advanced Medical Industry Promotion Foundation

KRIIB
Korea Research Institute of
Bioscience & Biotechnology

KFR! KOREA FOOD
RESEARCH INSTITUTE

KCLAM/KSLAV
한국실험동물수의사회
Korean Federation of Laboratory Animal Veterinarians

KRICP
KOREA RADIOISOTOPE CENTER FOR PHARMACEUTICALS
국립방사능연구원

KCFST
Korean Federation of Science &
Technology Societies

제주관광공사
JEJU TOURISM ORGANIZATION

INVITATION

Dear KALAS members,

It is our great pleasure to warmly invite you to the 2025 KALAS (Korean Association for Laboratory Animal Science) 40th Anniversary International Symposium, entitled with 'GIANTS (Global Insights in Advancing Translational Sciences)' at Jeju International Convention Center (Jeju ICC) in Jeju island from July 23 (Wed) to 26 (Sat) in 2025.

The bioscience is rapidly changing and developing, and along with the development of the life science, responsibility in the field of laboratory animals is becoming more important. To celebrate the 40th anniversary, a total of 20 sessions of various lectures have been prepared to reflect the trend of the latest research. These include two keynote lectures, eight sponsored symposiums, a IACUC session, two KLAT educational sessions, a KALAS-CALAS joint symposium, and so on.

The symposium will be supported by several institutions and organizations including KOFST (Korean Federation of Science and Technology Societies), KIT (Korea Institute of Toxicology), KMPC (Korea Model Animal Priority Center), NIFDS (National Institute of Food and Drug Safety Evaluation), KMEDHub, KBIOhealth, KRIBB (Korea Research Institute of Bioscience and Biotechnology), KFRI (Korea Food Research Institute), KCLAM (Korean College of Laboratory Animal Medicine), KIRMS (Korea Institute of Radiological and Medical Sciences), and I would like to thank everyone who participated in this symposium.

I hope all participants will enjoy this KALAS 40th Anniversary International Symposium, where they can broaden their academic knowledge and cherish precious memories in Jeju Island. Once again, I would like to express my sincere gratitude to those who support and contribute to this international symposium.

Yang-Kyu Choi
President, KALAS



KALAS COMMITTEE MEMBERS

	직책		성명	소속		직책		성명	소속
이사회(9인)	이사장	최양규	건국대학교	LAR5.0위원회	위원장	차지영	가천대학교		
	이사	남기택	연세대학교		위원(간사)	김명석	KIST 천연물연구소		
	이사	이정규	중앙실험동물(주)		위원	허진영	서강대학교 생명과학부		
	이사	서준교	한림대학교		위원	임승순	계명대학교 의과대학		
	이사	정자영	KBIO HEALTH		위원	현정은	단국대학교 바이오융합대학 의생명과학부		
	이사	신재호	을지대학교		위원	김주연	한양대학교		
	이사	진희경	경북대학교		학술위원회	동계위원장	이근욱	한림대학교	
	이사	장재진	(주)오리엔트바이오			동계간사	오경진	한국생명공학연구원	
	이사	이태훈	전남대학교			동계간사	서원호	이화여자대학교	
고문위원회	고문/전임회장	이영순	서울대학교	동계위원		이찬희	한림대학교		
	고문/전임회장	한상섭	전북대학교	동계위원		성영훈	서울아산병원		
	고문/전임회장	장자준	서울대학교 (퇴직)	동계위원		오한솔	충북대학교		
	고문/전임회장	조정식	한국실험동물협회	하계위원장		이호	국립암센터		
	고문/전임회장	강종구	충북대학교, (주)바이오독스텍	하계간사		최재훈	한양대학교		
	고문/전임회장	이국현	안동의료원	하계간사		정수명	성균관대학교		
	고문/전임회장	박재학	서울대학교	하계위원	김경미	서울대학교			
	고문/전임회장	현병화	한국과학기술원	하계위원	이현지	고려대학교 의과대학			
	고문/전임회장	김철규	제주대학교	하계위원	최진욱	광주과학기술원			
	고문/전임회장	이범준	충북대학교 (퇴직)	국제위원회	위원장	이순신	건국대학교		
고문/전임회장	이한웅	연세대학교, (주)젠크로	위원(간사)		유대영	서울대학교			
고문/전임회장	김길수	경북대학교	위원(간사)		박현정	서울대학교 수의과대학			
고문/전임회장	성재경	서울대학교	기획/섭외위원회	위원장	윤준원	서울대학교			
자문위원회	자문위원	김대용		서울대학교	위원(간사)	김형식	부산대학교		
	자문위원	김대중		충북대학교	위원	김환	기초과학연구원		
	자문위원	박정규		서울대학교	위원	이병철	숙명여자대학교		
	자문위원	송시환		(주)코아시스템즈	위원	이은우	한국생명공학연구원		
	자문위원	송창선	건국대학교	재무위원회	위원장	이영재	가천대학교		
	자문위원	오구택	이화여자대학교		위원(간사)	김경미	서울대학교		
	자문위원	차신우	안전성평가연구소	기금관리위원회	위원장	남정석	광주과학기술원		
	자문위원	한범석	호서대학교		위원(간사)	신태훈	제주대학교		
	부회장	수석부회장	남기택	연세대학교	홍보위원회	위원장	고혁완	연세대학교	
부회장		서준교	한림대학교	위원(간사)		부해진	제주대학교		
부회장		신재호	을지대학교	위원		김완태	서울시립대학교		
부회장		이태훈	전남대학교	위원		임경태	고려대학교		
부회장	장재진	(주)오리엔트바이오	위원	양한솔		KAIST			
감사	감사	강병철	서울대학교	위원		안은희	한림대학교		
	감사	제정환	서울대학교병원	위원		권혁권	경상국립대학교		
총무위원회	위원장	북진웅	연세대학교	위원		최치은	제주대학교		
	위원(간사)	황성순	연세대학교	위원		이충용	경북대학교		
	위원(간사)	박준원	서울대학교	위원		김다현	성신여자대학교		
	위원(간사)	서진희	한국원자력연구원	위원	장경구	연세대학교			
편집위원회	위원장	성재경	서울대학교	전산위원회	위원장	박종환	전남대학교		
	부위원장	북진웅	연세대학교		위원(간사)	황지연	분당서울대학교병원		
	위원(ME간사)	고혁완	연세대학교	산학협동위원회	위원장	김현일	(주)윙티팜		
	위원(ME간사)	김경미	서울대학교		부위원장(간사)	강경수	신구대학교 자원동물과		
	위원(ME간사)	문창홍	전남대학교		위원	이정규	중앙실험동물(주)		
	위원(ME간사)	박만성	고려대학교		위원	김재길	한신메디칼(주)		
	위원(ME간사)	박종환	전남대학교		위원	박천귀	(주)쓰리사인		
	위원(ME간사)	부해진	제주대학교		위원	유병천	(주)아이티스텐다드		
	위원(ME간사)	서원호	이화여자대학교		위원	이오	(주)오리엔트바이오		
	위원(ME간사)	서준교	한림대학교	인증위원회	위원장	오승현	서울대학교		
	위원(ME간사)	성영훈	서울아산병원		부위원장(총무)	전현정	한국식품연구원		
	위원(ME간사)	윤상선	연세대학교		부위원장(법제)	주영신	가톨릭대학교 의생명산업연구원		
	위원(ME간사)	이근욱	한림대학교		위원(교육 및 교재간사)	오한솔	충북대학교		
	위원(ME간사)	이정수	가톨릭대학교		위원(교육 및 교재간사)	김동재	대구경북과학기술원		
	위원(ME간사)	이주영	가톨릭대학교 약학대학		위원(소통간사)	허승호	서울아산병원		
	위원(ME간사)	이호	국립암센터		위원(인증간사)	양세란	강원대학교		
위원(ME간사)	정주희	덕성여자대학교	위원(인증간사)		이진수	충남대학교			
위원(ME간사)	정호성	연세대학교 의과대학	위원(전산간사)		전희연	가톨릭대학교 의생명산업연구원			
위원(ME간사)	조성대	서울대학교 치과대학	위원(전산간사)		이정민	삼성서울병원			
위원(ME간사)	조종기	서울대학교	위원(총무간사)		김지영	이화여자대학교 마곡병원			
위원(ME간사)	최재훈	한양대학교	위원		강병철	서울대학교			
위원(ME간사)	현영민	연세대학교	위원		강진석	남서울대학교			
위원(ME간사)	홍정주	한국생명공학연구원	위원		권영삼	경북대학교			
위원(ME간사)	황인구	서울대학교	위원		김건아	을지대학교			
위원(RE간사)	황성순	연세대학교	위원		김동환	건양대학교			
위원	구송희	고려대학교	위원		김영욱	경북대학교			
위원	김용환	숙명여자대학교	위원		김배환	계명대학교			
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위원	박성규	서울대학교	위원		김성현	한국폴리텍대학 바이오캠퍼스			
위원	박한수	광주과학기술원	위원		김용범	안전성평가연구소			
위원	윤경아	건국대학교	위원		김종성	아주대학교의료원			
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	위원	남 기택	연세대학교
	위원	남 기 환	한국생명공학연구원
	위원	박 중 환	전남대학교
	위원	박 준 석	대구경북첨단의료산업진흥재단
	위원	백 성 진	(주)크로엔
	위원	서 민 수	경북대학교
	위원	서 준 교	한림대학교
	위원	서 진 희	한국원자력의학원
	위원	석 승 혁	서울대학교 의과대학
	위원	성 영 훈	서울아산병원
	위원	성 하 정	마이더스인터넷서벌코리아
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	위원	신 재 호	울지대학교
	위원	신 태 훈	제주대학교
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	위원	우 계 형	세명대학교
	위원	유 영 춘	건양대학교
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	위원	이 재 훈	연세대학교
	위원	이 준 구	한경국립대학교
	위원	이 총 현	단국대학교
	위원	정 의 숙	대구경북첨단의료산업진흥재단
	위원	정 재 황	충북도립대학교
	위원	정 지 윤	공주대학교
	위원	제 정 환	서울대학교병원
	위원	조 성 대	서울대학교 치과대학
	위원	진 영 배	경상국립대학교
	위원	조 현 무	가톨릭대학교 은평성모병원
	위원	최 양 규	건국대학교
	위원	최 연 식	한국폴리텍대학 바이오캠퍼스
	위원	한 동 운	연암대학교
	위원	한 범 석	호서대학교
	위원	황 대 연	부산대학교
	위원	황 종 익	고려대학교
	위원	황 지 연	분당서울대학교병원
법제위원회	위원장	이 원 우	서울대학교
	위원(간사)	김 기 석	안전성평가연구소
	위원(간사)	임 재 철	서울대학교
연구윤리위원회	위원장	석 승 혁	서울대학교 의과대학
	위원(간사)	이 지 민	서울대학교
	위원(간사)	홍 정 주	한국생명공학연구원
동물복지위원회	위원장	이 상 래	아주대학교 의과대학
	위원(간사)	김 중 성	아주대학교의료원
	위원	전 채 은	동물유위행동
	위원	박 준 석	대구경북첨단의료산업진흥재단
포상위원회 (학술상위원회)	위원장	양 인 속	연세대학교의료원
	위원장	황 대 연	부산대학교
	위원(간사)	임 용	동의대학교
	위원	안 지 윤	한국식품연구원
지회		조 현 무	가톨릭대학교 은평성모병원
	서울지회장	조 성 대	서울대학교 치과대학
	경기지회장	이 희 영	가천대학교 의과대학
	강원지회장	김 태 민	서울대학교
	경상지회장	김 배 환	계명대학교
	전라지회장	권 중 기	전북대학교
	제주지회장	윤 영 민	제주대학교
	충청지회장	정 지 윤	공주대학교

KALAS COUNCIL MEMBERS

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성명	소속
강 경 선	서울대학교
강 병 철	서울대학교
강 종 순	한국생명공학연구원
강 진 석	남서울대학교
고 혁 완	연세대학교 생명시스템대학
구 재 형	대구경북과학기술원(DGIST)
권 동 략	대구가톨릭대학교의료원
권 중 기	전북대학교
김 곤 섭	경상대학교
김 근 형	충북대학교
김 길 수	경북대학교
김 대 중	충북대학교
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김 명 옥	경북대학교
김 배 환	계명대학교
김 욱 진	원광대학교
김 용 범	안전성평가연구소
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김 정 훈	서울대학교
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김 종 춘	전남대학교
김 종 용	(주)오리엔트제니아
김 태 완	경북대학교
김 형 식	부산대학교
김 형 진	(前)한국생명공학연구원
나 이 랑	서울대학교병원
남 기 택	연세대학교
남 기 환	한국생명공학연구원
남 상 윤	충북대학교
남 정 석	광주과학기술원
박 대 훈	동신대학교
박 정 규	서울대학교
박 종 일	양산부산대학교병원
박 종 환	전남대학교
박 준 석	대구경북첨단의료산업진흥재단
박 천 귀	(주)쓰리사인
배 재 성	경북대학교

성명	소속
배 춘 식	전남대학교
복 진 용	연세대학교
서 준 교	한림대학교
석 승 혁	서울대학교
성 제 경	서울대학교
성 하 정	마이더스인터내셔널코리아
송 문 용	한국건설생활환경시험연구원
송 창 선	건국대학교
송 창 우	국가독성과학연구소
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신 재 호	울지대학교
양 세 란	강원대학교
오 경 진	한국생명공학연구원
오 구 택	이화여자대학교
오 승 현	서울대학교
유 경 록	서울대학교
유 영 춘	건양대학교
윤 원 기	한국생명공학연구원
윤 준 원	서울대학교
이 경 선	오송첨단의료산업진흥재단
이 근 욱	한림대학교
이 만 휘	경북대학교
이 민 재	강원대학교
이 범 준	충북대학교
이 병 한	(前)오송첨단의료산업진흥재단
이 병 희	환경부 국립생물자원관
이 상 래	아주대학교
이 순 신	건국대학교
이 영 재	가천대학교
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이 철 호	한국생명공학연구원
이 태 훈	전남대학교 치의학전문대학원
이 학 모	(주)디자인원헬스
이 한 용	연세대학교, (주)젬크로
이 현 용	(주)대중기기산업

성명	소속
이 현 주	(주)샘타코바이오코리아
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장 인 석	경상국립대학교
장 재 진	(주)오리엔트바이오
전 경 희	연세대학교
전 현 정	한국식품연구원
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정 자 영	오송첨단의료산업진흥재단
정 재 황	충북도립대학교
정 지 윤	공주대학교
제 정 환	서울대학교병원
조 규 혁	안전성평가연구소
조 성 대	서울대학교
조 재 진	서울대학교
조 현 무	가톨릭대학교
진 희 경	경북대학교
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차 지 영	가천대학교
천 병 년	(주)우정바이오
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최 병 인	가톨릭대학교
최 양 규	건국대학교
최 연 식	한국폴리텍대학
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허 승 호	서울아산병원
허 용	대구가톨릭대학교
현 병 화	한국과학기술원
황 대 연	부산대학교
황 성 순	연세대학교
황 정 호	안전성평가연구소
황 종 익	고려대학교

발전기금 납입 회원

2025.01.01. 기준

성명	소속	금액(단위:원)
Alan Lee Chedester	NIH	300,000
Toru Takeo	Kumamoto University	USD 1,000
강병철	서울대학교	2,311,200
강종구	(주)바이오톡스텍	1,000,000
강진석	남서울대학교	200,000
권구범	NTCV Vaccine Co.	300,000
권중기	전북대학교	200,000
김길수	경북대학교	200,000
김대용	서울대학교	350,000
김대중	충북대학교	517,000
김덕원	삼양약화학	50,000
김배환	계명대학교	802,280
김윤배	충북대학교	267,680
김종성	삼성생명과학연구원	267,680
김종춘	전남대학교	300,000
김형식	부산대학교	300,000
남정석	광주과학기술원	869,950
박재학	서울대학교	5,955,525
박충권	녹십자 EM	300,000
서경덕	천안연암대학	100,000
서준교	한림대학교	1,769,950
석승혁	서울대학교	2,100,000
성제경	서울대학교	300,000
성하정	크로엔리서치	200,000
손우찬	울산대학교	100,000
송시환	(주)캠온	100,000
송창우	안전성평가연구소	3,650,000
신영수	신구대학교	467,680
신재호	을지대학교	200,000
안병우	충북대학교	200,000
염수청	서울대학교	200,000
오승현	가천대학교	200,000
오양석	한림대학교	1,791,299
원무호	강원대학교	3,000,000
위명복	강원대학교	391,200

성명	소속	금액(단위:원)
유영춘	건양대학교	200,000
이민재	강원대학교	500,000
이범준	충북대학교	200,000
이병한	오송첨단의료산업진흥재단	367,680
이상구	(주)바이오톡스텍	200,000
이상필	(주)한주씨엠아이	4,000,000
이수해	식품의약품안전처	100,000
이영순	서울대학교	10,000,000
이정규	(주)중앙실험동물	1,000,000
이철호	한국생명공학연구원	200,000
이한웅	연세대학교	5,000,000
인증위원회	인증위원회	3,000,000
인증위원회	동물실험길잡이 인세	36,587,000
장동덕	국군의학연구소	200,000
장자준	서울대학교	882,400
정기원	(주)엠제이엘티디	1,000,000
정재황	충북도립대학	100,000
정지윤	공주대학교	200,000
제정환	서울대학교병원	837,630
조기행	서울대학교	300,000
조윤주	서정대학교	200,000
조재진	서울대학교	10,000,000
차신우	안전성평가연구소	200,000
천병년	(주)우정비에스씨	2,000,000
최경철	충북대학교	1,000,000
최양규	건국대학교	2,769,950
최연식	한국폴리텍바이오대학교	200,000
최우성	대구경북첨단의료산업진흥재단	200,000
한남옥	코아텍	30,000,000
한범석	건양대학교	2,200,000
허용	대구가톨릭대학교	200,000
현병화	오송첨단의료산업진흥재단	30,300,000
황대연	부산대학교	200,000
황인구	서울대학교	1,900,000
(재)한국건설생활환경시험연구원		2,000,000

학술상·편집위·실험동물연구장학생 후원금 납입 내역

2025.01.01. 기준

소속	금액(단위:원)	년도
(주)쓰리사인	25,000,000	2005~2009
(주)코아텍	10,000,000	2016
중앙실험동물(주)	235,000,000	2007~2024

2025 한국실험동물학회 평생회비 납입회원

2025.07.01. 기준

성명	소속
강 경 수	신구대학교
강 병 철	서울대학교
강 중 구	충북대학교
강 진 주	경북대학교
고 은 아	제주대학교
곽 동 미	경북대학교
구 재 형	대구경북과학기술원 (DGIST)
권 구 범	우정BSC
권 동 략	대구가톨릭대학교병원
권 명 상	강원대학교
권 은 아	서울대학교병원
권 재 성	연세대학교
권 태 준	대구경북첨단의료산업진흥재단
김 강 현	서울아산병원
김 건 아	울지대학교
김 경 원	서울아산병원
김 근 형	충북대학교
김 길 수	대구경북첨단의료산업진흥재단
김 대 원	강릉원주대학교
김 대 중	충북대학교
김 명 옥	경북대학교
김 무 강	(재)충남동물자원센터
김 배 환	계명대학교
김 보 라	국립암센터
김 상 아	서울대학교 약학대학
김 상 윤	한국생명공학연구원
김 상 현	대구경북첨단의료산업진흥재단
김 선 호	(주)플라스
김 성 곤	대구경북첨단의료산업진흥재단
김 욱 진	원광대학교
김 용 안	충남대학교
김 우 석	한림대학교
김 유 용	서울대학교
김 정 원	한국원자력의학원
김 정 훈	서울대학교병원
김 종 남	서울대학교
김 종 민	바이오이종장기개발사업단
김 종 훈	전남대학교
김 주 완	대구가톨릭대학교
김 중 현	오송첨단의료산업진흥재단
김 진 만	(주)오리엔트바이오
김 철 규	오송첨단의료산업진흥재단
김 총 수	대구경북첨단의료산업진흥재단
김 총 용	대구경북첨단의료산업진흥재단
김 태 완	경북대학교
김 태 환	경북대학교
김 현 일	(주)옵티팜
김 형 식	부산대학교
김 혜 진	한국한의학연구원
김 희 정	대구경북첨단의료산업진흥재단
김 희 진	농림축산검역본부
나 운 성	전남대학교
나 이 랑	서울대학교병원
남 기택	연세대학교
남 병 수	한국한의학연구원
남 상 윤	충북대학교
남 정 석	광주과학기술원
노 재 일	연세대학교
류 동 렬	성균관대학교
류 정 주	대구경북첨단의료산업진흥재단

성명	소속
문 지 영	대전보건대학교
박 대 훈	동신대학교
박 동 주	경상국립대학교
박 민 경	대구경북첨단의료산업진흥재단
박 부 환	인하대학교
박 상 인	(재)스크립스코리아항체연구원
박 인 선	인하대학교
박 재 학	서울대학교
박 종 환	전남대학교
박 준 석	대구경북첨단의료산업진흥재단
박 진 성	서울대학교병원
박 천 귀	(주)쓰리사인
배 재 성	경북대학교
배 춘 식	전남대학교
백 인 정	서울아산병원
변 용 섭	오송첨단의료산업진흥재단
복 진 용	연세대학교
서 민 수	DGMIF-LAC
서 병 부	대구대학교
서 준 고	한림대학교
서 진 희	한국원자력의학원
석 승 혁	서울대학교
성 영 훈	연세대학교
성 제 경	서울대학교
성 하 정	크로엔리서치
손 성 향	아주대학교
손 화 영	충남대학교
송 문 용	한국건설생활환경시험연구원
송 승 우	Harlan Korea Ltd
송 창 우	안전성평가연구소
신 재 호	울지대학교
신 태 훈	제주대학교
신 형 철	한림대학교
안 재 범	가천대학교 이길여암당뇨연구원
안 지 완	서울대학교
오 경 진	한국생명공학연구원
오 구 택	이화여자대학교
오 미 경	한양대구리병원
오 승 현	가천대학교
오 양 석	한림대학교
오 연 수	강원대학교
오 은 석	서울대학교병원
우 계 형	세명대학교
원 무 호	강원대학교
위 갑 인	대구경북첨단의료산업진흥재단
유 대 영	서울대학교
윤 기 영	신구대학교
윤 준 원	서울대학교
이 경 선	오송첨단의료산업진흥재단
이 공 현	인제대학교 부산백병원
이 광 훈	대구경북첨단의료산업진흥재단
이 국 현	서울대학교
이 근 식	강원대학교
이 동 준	부산대학교
이 만 휘	경북대학교
이 민 재	강원대학교
이 범 준	충북대학교
이 병 한	오송첨단의료산업진흥재단
이 상 래	한국생명공학연구원
이 상 필	(주)한주씨엠아이

성명	소속
이 순 신	순천향대학교
이 윤 재	한양대학교 구리병원
이 재 형	바이오지노케어
이 재 훈	연세대학교
이 정 규	(주)중앙실험동물
이 정 범	순천향대학교
이 종 권	식품의약품안전처
이 준 구	경상북도축산기술연구소
이 준 호	(주)바이오피아
이 창 환	울산대학교
이 철 호	한국생명공학연구원
이 중 현	단국대학교
이 학 모	서울대학교병원
이 한 용	연세대학교
이 현 용	(주)대충기기산업
이 현 주	(주)샘타코바이오코리아
임 재 향	이화여자대학교
장 규 태	한국생명공학연구원
장 용 기	(주)베르나바이오테크코리아
장 윤 성	전북대학교병원
장 자 준	서울대학교
장 재 진	(주)오리엔트바이오
전 현 정	한국식품연구원
정 기 원	(주)엔제이엘티디
정 민 재	한림대학교
정 원 일	한국과학기술원(KAIST)
정의숙	대구경북첨단의료산업진흥재단
정 자 영	오송첨단의료산업진흥재단
정 지 운	연암대학교
제 정 환	서울대학교
조 우 리	대구경북첨단의료산업진흥재단
조 익 준	오송첨단의료산업진흥재단
조 재 진	서울대학교
조 정 식	호서대학교
조 준 휘	강원대학교병원
조 현 무	가톨릭대학교 은평성모병원
진 영 배	경상국립대학교
진 희 경	경북대학교
채 갑 용	식품의약품안전처
천 병 년	(주)우정바이오
최 경 철	충북대학교
최 병 태	부산대학교
최 양 규	건국대학교
최 영 석	건국대학교
최 영 현	동의대학교
최 우 성	대구경북첨단의료산업진흥재단
최 은 화	강원대학교
최 정 훈	강원대학교
최 준 영	대구경북첨단의료산업진흥재단
최 혜 윤	대구경북첨단의료산업진흥재단
한 남 옥	코아텍
한 덕 주	건일제약(주)
한 진 수	건국대학교
한 태 원	한국식품연구원
허 승 호	서울아산병원실험동물연구실
홍 성 근	경상대학교
홍 의 주	충남대학교
황 인 구	서울대학교
황 정 호	안전성평가연구소
황 종 익	고려대학교

2025 한국실험동물학회 국제학술대회 협력기관

회사명	대표자	전화번호	홈페이지
AAALAC International		02-3147-9590	www.aaalac.org
GemPharmatech		+1-888-899-5899	www.gempharmatech.com
KB금융	김종선	010-6321-7170	
(재)국가모델동물연구소	성제경	02-885-8392	mousephenotype.kr
농협금융그룹	채장희	010-2824-0790	www.nhfngroup.com
(재)대구경북과학기술원	이건우	053-785-8613	www.dgist.ac.kr
동우메디칼	여한동	053-951-5199	
(주)디비엘	주재훈	032-513-4470	www.dbl.kr
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라이카바이오시스템즈	박영돈, 이정록	02-3416-4500	www.leicabiosystems.com/ko-kr
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식품의약품안전평가원 비임상자원연구과	강석연	043-719-5515	nifds.go.kr
실험동물 고품질관리 기반 구축연구단	강병철	02-2072-1909	
(주)쓰리샤인	박춘자	042-933-3361	threeshine.com
아이빔테크놀로지	김필한	070-4193-5029	www.ivimtech.com
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(주)에코엔테크	기혜란	032-325-6166	www.ecoandtech.co.kr

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(주)엠온솔루션	남수진	070-4115-1110	www.emonsolution.co.kr
(주)영바이오	송영종	031-759-9931	www.youngbio.kr
(주)오리엔트바이오	장재진	031-730-6728	www.orientbio.co.kr
오송첨단의료산업진흥재단	이명수	043-200-9823	www.kbiohealth.kr
(주)오스테오시스	안영복	02-6124-5900	www.osteosys.com
(주)옵티팜	김현일	043-249-7592, 7550	www.optipharma.co.kr
(주)우정바이오	천희정	031-280-5200	www.woojungbio.kr
(주)정도비앤피	정병도	010-3280-0809	jeungdo.co.kr
주식회사 두열바이오텍	최양환	02-575-6598	www.dybiotech.co.kr
주식회사 디케이코리아	유용주	010-7315-4561	www.dkd-korea.kr
주식회사 메디코어스	윤귀영	031-698-4903	www.medikors.com
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중앙실험동물(주)	이정규	010-6287-9375	www.labanimal.co.kr
(주)코리아바이오믹스	장재룡	010-8956-7470	www.koreabiomics.com
(주)코아텍	한남옥	031-611-8221	www.koatech.co.kr
(주)크리스티코리아	김 찬	010-4494-2989	www.cryste.co.kr
한국건설생활환경시험연구원	천영길	010-9180-2714	kcl.re.kr
한국기초과학지원연구원	남연경	042-865-3977	www.kbsi.re.kr
한국생명공학연구원 실험동물자원센터, 모델동물중앙은행	권석윤	043-240-6527	mouse.kribb.re.kr
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한국생명공학연구원 천연물중앙은행	권석윤	043-240-6121	kobis.re.kr/npcb
한신메디칼(주)	김성훈	010-6242-0553	www.hanshinmed.co.kr
(주)한주씨엠아이	이상필	031-366-8600	한주씨엠아이.com

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July 23 (Wed)					
HALL	Halla Hall A	Halla Hall B	Samda Hall	Meeting Room (5F)	Lobby
11:30-13:00				Council Meeting (11:30~13:00) [Ocean view]	
13:00-14:00	Registration				
14:00-15:40	(KOR) Symposium 1 [K-MEDI hub, KBIO HEALTH] Current Trends in Nonclinical Testing With Non-Rodent Animal Models: Focus on Common Marmosets and Pigs	(ENG) Symposium 2 MPS and Organoid Research and Laboratory Animal Science	(KOR) Symposium 3 [IACUC] Strengthening Biosafety Risk Assessment and Management in Laboratory Animal Research		Booth Installation Space Only : 10:00~ Shell Scheme (Space+Frame): 16:00~
15:40-16:00	Refreshment				
16:00-17:40	(KOR) Symposium 4 [KFRI] Animal Models in Nutrition Research and Functional Food Development	(KOR) Symposium 5 [KIT] The Use of Non-Human Primates in Non-Clinical Studies Ranges From Management to Research	(KOR) Symposium 6 [KLAT Education 1] Microbiological Quality Control for Laboratory Rodents		

July 24 (Thu)						
HALL	Halla Hall A	Halla Hall B	Samda Hall	Meeting Room (3F)	Lobby	
09:00-09:30	Registration				Poster Installation Time (09:00~11:00)	
09:30-09:40	Opening Ceremony					
09:40-11:20	(ENG) Symposium 7 Stem Cells and Tissue Regeneration	(KOR) Symposium 8 [KIRAMS] Molecular Imaging Techniques for Evaluation of Drug Development and Disease Animal Models	(KOR) Symposium 9 [KCLAM] Optimizing Post-Approval Monitoring: Navigating the Roles of IACUC, AV, LAT, and Researchers		Board Meeting (10:40-11:30) [Room 301]	Poster & Booth
11:20-11:30	Refreshment & Exhibition visit					
11:30-11:40	40주년 기념 비전 선포					
11:40-12:30	(ENG) Plenary Lecture 1					
12:30-12:40	Refreshment					
12:40-13:00	Luncheon Seminar 1 (IVIM Technology)	Luncheon Seminar 2 (ITstandard)	Luncheon Seminar 3 (Orient Bio Inc.)			
13:00-14:00	Lunch Break				Poster Presentation1 (13:20~14:20)	
14:00-14:40	Academy Award Presentation				KCLAM 회의 (14:00-17:00) [Room 303]	Poster & Booth
14:40-14:50	Refreshment					
14:50-16:30	(ENG) Symposium 10 In Vivo Explorations of Vascular Function and Dysfunction	(KOR) Symposium 11 [KMPC] The Next Waves in Signaling Crosstalk From Cellular to Systemic Metabolism	(ENG) Symposium 12 [KALAS-CALAS] Preclinical Studies of Xenografts and Tissue Transplantation (14:50-16:10)			
16:30-17:00	Refreshment			KALAS-CALAS Joint Round Table Meeting (16:20-17:00) [Room 304]		
17:00-19:00	General Meeting & Welcome Reception [Tamna Hall (5F)]					

TIME TABLE

July 25 (Fri)					
HALL	Halla Hall A	Halla Hall B	Samda Hall	Meeting Room (3F)	Lobby
08:30-09:00	Registration				
09:00-10:40	(ENG) Symposium 13 Metabolic Mechanisms in Health and Diseases	(ENG) Symposium 14 [KRIBB] Translational Research Platform Using Animal Models for Inherited Retinal Disease (IRD)	(ENG) Symposium 15 [NIFDS] Humanize Mouse Models for Preclinical Research		Poster Installation Time (09:00-11:00)
				산학협력 간담회 (10:00-11:00) [Room 301]	
10:40-11:00	Refreshment & Exhibition visit				
11:00-11:50	(ENG) Plenary Lecture 2				Poster & Booth
11:50-12:00	Refreshment				
12:00-12:20	Luncheon Seminar 4 (Bio-Medical Science Co., Ltd.)	Luncheon Seminar 5 (GemPharmatech Co., Ltd.)	Luncheon Seminar 6 (NongHyup Financial Group INC.)		
12:20-14:00	Lunch Break				Poster Presentation 2 (13:00-14:00)
14:00-15:30	(ENG) Symposium 16 Development of 3D Composite Constructs with Immune Modulatory Functions and Tissue Regeneration Technologies	(ENG) Symposium 17 Latest Research Trends in Development of Experimental Animal Models and Biomarkers, and AI-Based Behavioral Analysis Technologies for Substance Use Disorder	(KOR) Symposium 18 [KLAT Education 2] Application and Management of Genetically Modified Animals		Poster & Booth
15:30-15:50			기술원 QUIZ EVENT		
15:50-16:00	Refreshment				
16:00-16:40	Closing Ceremony & Awards				

July 26 (Sat)	
HALL	Halla Hall A
09:30-11:10	Satellite meeting

HALL INFORMATION

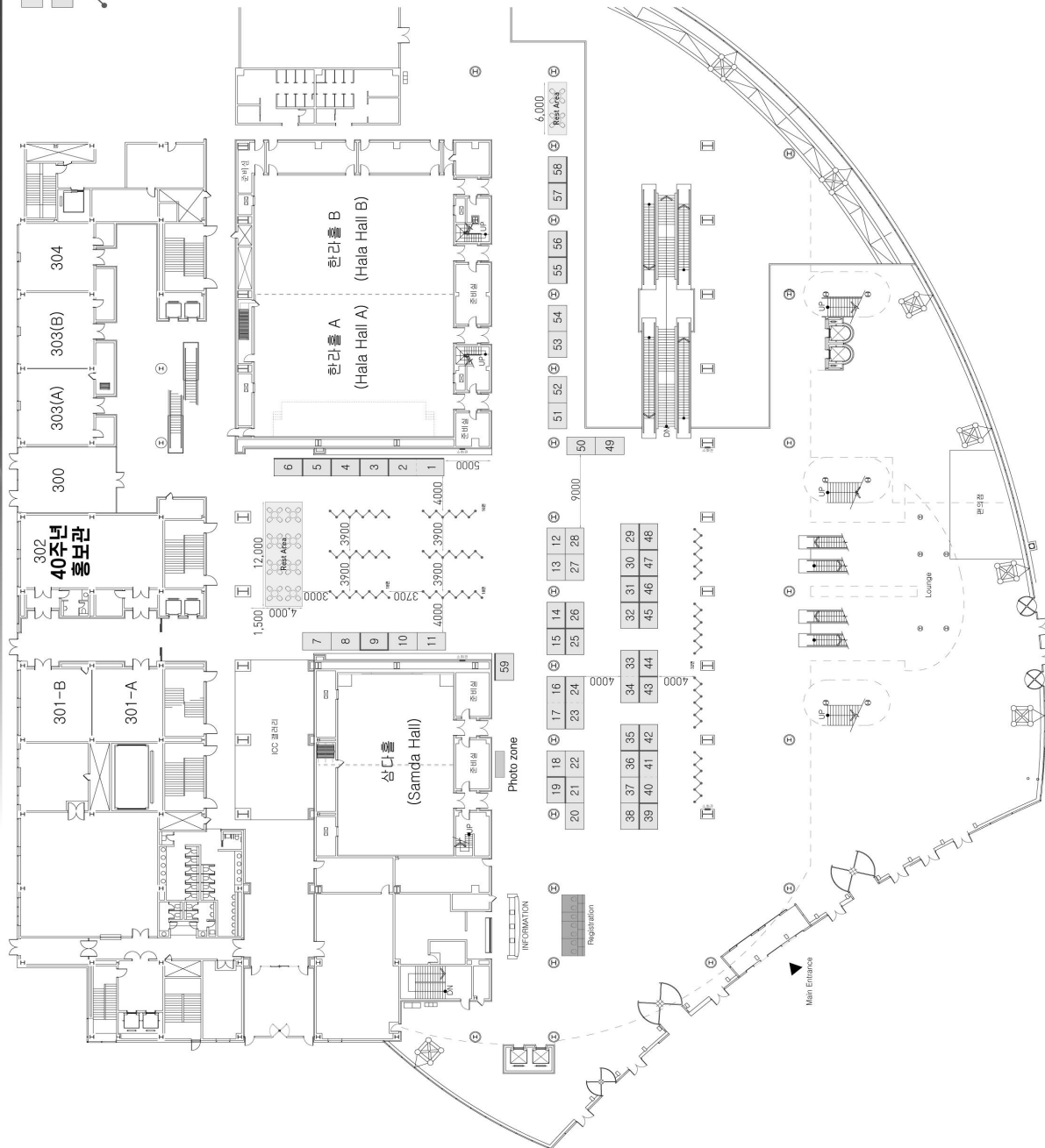
KALAS 2025 KALAS International Symposium

2025. 7. 23. (수) ~ 7. 25. (금)

ICCEJU 제주컨벤션 3F

- 독립전시 Booth(3x2m) : 24ea
- 기본전시 Booth(3x2m) : 35ea
- Poster board : 85ea = 138(side)

- 1.2. 한국기초과학지원연구원
3. 한진해운(주)
4. (주)오스비오시스
5. 라온메타(주)
6. (주)크리아바이옴스
- 7.8. (주)가온셀룰러구스
9. 주식회사 피마노이드
- 10.11. (주)코아텍
- 12.13.27.28. (주)오리엔트바이오
14. 쓰리사인(주)
15. 오송첨단의료산업진흥재단
- 16.17. (주)비이오텍
18. (주)디비엘
19. GemPharmatech
- 20-22. 라온바이오(주)
- 23.24. 학기씨아이엔텍(주)
25. 실험동물 고분자관리 기반 구축연구단
26. (주)셀타판
- 29.30. (주)크리아바이옴스텔즈
31. (주)에코엔테크
32. 아이엠테크놀로지
- 33.34. (주)메트릭코리아
35. (주)정도비엔피
36. 제비타코리아유한회사
- 37.38. 셀타코리아유한회사
39. 식품의약품안전평가원 실험동물자원은행
- 40.41. 한국동물자원관리원 연구원
42. 주식회사 두얼바이오
43. 주식회사 메디코어스
44. 동양메디칼
45. 한국생명과학연구원 실험동물지원센터
46. 한국생명과학연구원 천연물연구센터
47. 한국생명과학연구원 영양특수지원센터
48. AAALAC International
- 49-51. (주)두얼바이오
52. (주)비엠에스
53. (주)셀룰러
54. (주)크리스티코리아
55. (주)영바이오
56. 주식회사 다케이코리아
57. (주)한주이앤아이
58. KB금융
59. 농협생명보험



PROGRAM

July 23 (Wed)		
Symposium 1 [K-MEDI hub, KBIO HEALTH] KOR		Halla Hall A (14:00~15:40)
Current Trends in Nonclinical Testing With Non-Rodent Animal Models: Focus on Common Marmosets and Pigs		
Organizer / Chair : Myung-Hoon Chung (K-MEDI hub), Jayoung Jeong (OSONG Medical Innovation Foundation)		
The Minipig in Medical Device Development	Ryounghoon Jeon	K-MEDI hub
Research on Tissue-Engineered Scaffolds Combined with Stem Cell Therapies Utilizing Animal Models	Sang Jin Lee	Univ. of Hong Kong
Research Methodology Developments for Common Marmoset (Callithrix Jacchus)	Dohyun Lee	OSONG Medical Innovation Foundation
Bridging the Rodent to Human Translational Gap: Marmoset as a Model System for Biomedical Research	Jung Eun Park	Univ. of Pittsburgh
Symposium 2 ENG		Halla Hall B (14:00~15:40)
MPS and Organoid Research and Laboratory Animal Science		
Organizer : Jaejin Cho (Seoul Natl. Univ.) Chair : Hyung-Sik Kim (Pusan Natl. Univ.), Jaejin Cho (Seoul Natl. Univ.)		
Advanced Human Microphysiological Systems for Disease Modeling and Drug Development	Deok-Ho Kim	Johns Hopkins Univ.
MPS and Organoids as New Alternative Methods (NAMs): Current & Upcoming Trend	Nakwon Choi	Korea Univ.
Microfluidics-Embedded 3D Perfusion Cell Culture System	Jeung Sang Go	Pusan Natl. Univ.
Therapeutic Organoid Platform, "Tissue Reforming Technology Platform" and Animal Model	Jaejin Cho	Seoul Natl. Univ.
Symposium 3 [IACUC] KOR		Samda Hall (14:00~15:40)
Strengthening Biosafety Risk Assessment and Management in Laboratory Animal Research		
Organizer : Ji Min Lee (Seoul Natl. Univ.) / Chair : Jung Joo Hong (KRIBB)		
Risk Assessment Strategies for Research Involving Biological Agents	Won-Jong Jang	Konkuk Univ.
Biosafety Management for Animal Experiments in ABL-3 Laboratories	Kyung Min Lee	International Vaccine Institute
Current Status and Future Development of Biosafety Education Programs	Tae Jong Son	Korea Disease Control and Prevention Agency
Predicting Hazards in Laboratory Work by Deep Learning Towards Risk Assessment and Safety Management	Takaaki Harada	Nagoya Univ.

Symposium 4 [KFRI] KOR		Halla Hall A (16:00-17:40)
Animal Models in Nutrition Research and Functional Food Development		
Organizer : Hyunjhun Jhun (KFRI), Ho-Young Park (KFRI) / Chair : Ho-Young Park (KFRI)		
Integrating Chrono-Nutrition and Probiotics in Organoids for Functional Food Development	Miri Park	KFRI
Alterations in Mitochondria-Associated Molecular Patterns and the Role of Dietary Components in Improving Inflammation and Metabolic Disease	Yu Geon Lee	KFRI
Development of New Concept Efficacy Evaluation Analysis Technology Utilizing Mouse Resources for Modeling Human Disease	Hea-Jong Chung	KBSI
Effects of the Complex of Panicum Miliaceum Extract and Triticum Aestivum Extract on Hair Condition	Pan-Young Jeong	Novarex Co., Ltd.

Symposium 5 [KIT] KOR		Halla Hall B (16:00-17:40)
The Use of Non-Human Primates in Non-Clinical Studies Ranges From Management to Research		
Organizer : Han Na Suh (KIT) / Chair : Gee Euhn Choi (Jeju Natl. Univ.)		
Considerations for Research with NHPs	Eun Ju Song	Emory Univ.
Approaches to Microbiological Quality Control in Laboratory Non-Human Primates	Kyoung-Sun Lee	OSONG Medical Innovation Foundation
Application of Non-Human Primates in Advanced Biopharmaceuticals Development	Han Na Suh	KIT

Symposium 6 [KLAT Education 1] KOR		Samda Hall (16:00-17:40)
Microbiological Quality Control for Laboratory Rodents		
Organizer : Dong-Jae Kim (DGIST) / Chair : Seung Hyeok Seok (Seoul Natl. Univ.)		
Major Rodent Diseases in Korean Laboratory Animal Facilities	Joon-Suk Park	K-MEDI hub
Disease Control Strategies for Laboratory Rodents: Quarantine, Disinfection, and Sterilization	Jungmin Lee	Samsung Medical Center
Health Monitoring Techniques for Laboratory Rodent	Hwan Kim	IBS
Cleaning Methods for Infected Rodents and Applicable Cases	Yonghun Sung	Korea Polytechnic

PROGRAM

July 24 (Thu)		
Symposium 7 ENG		Halla Hall A (09:40~11:20)
Stem Cells and Tissue Regeneration		
Organizer : Jinwook Choi (GIST) / Chair : Jinwook Choi (GIST), Hye-Jin Boo (Jeju Natl. Univ.)		
Rare Begets Common: Skin Diseases Reveal Basic Human Biology	Ting Chen	NIBS, Beijing
Aging and Rejuvenation of Skin Stem Cells and Their Niches	Sekyu Choi	POSTECH
Organoid Modelling of Human Lung Development and Cell Fate Alteration in Human Congenital Pulmonary Diseases	Kyungtae Lim	Korea Univ.
The Specification and Diversification of Enteroendocrine Cells from Intestinal Stem Cells	Rongwen Xi	NIBS, Beijing
Symposium 8 [KIRAMS] KOR		Halla Hall B (09:40~11:20)
Molecular Imaging Techniques for Evaluation of Drug Development and Disease Animal Models		
Organizer : Jin-Hee Seo (KIRAMS) / Chair : Yong Jin Lee (KIRAMS)		
Radiopharmaceuticals for Noninvasive Theranostics Imaging	Yun-Sang Lee	Seoul Natl. Univ.
MR Molecular Imaging for Drug Development and Disease Diagnostics Using Animal Models	Yongmin Chang	Kyungpook Natl. Univ.
Preclinical Evaluation and Imaging of Radiopharmaceuticals Using PET	Se Jong Oh	KIRAMS
Pharmacokinetic Study Using C-14 in New Drug Development	Mihye Kwon	KIRAMS
Symposium 9 [KCLAM] KOR		Samda Hall (09:40~11:20)
Optimizing Post-Approval Monitoring: Navigating the Roles of IACUC, AV, LAT, and Researchers		
Organizer : Jun Won Yun (Seoul Natl. Univ.) / Chair : Byeong-Cheol Kang (Seoul Natl. Univ.)		
Enhanced PAM: Experience and Implementation at HKUMED's AAALAC International Accredited Program	Connie Leung	The Univ. of Hong Kong
AV and LAT Roles in Post-Approval Monitoring (PAM)	Ji-Yeon Hwang	Seoul Natl. Univ. Bundang Hospital
Program Review and PAM: Similar Goals, Different Approaches	Young-Shin Joo	The Catholic Univ.
Plenary Lecture 1 ENG		Halla Hall A, B (11:40~12:30)
Organizer / Chair : Ki Taek Nam (Yonsei Univ.)		
Hippo-YAP/TAZ Signaling: A Crucial Pathway in Cell Fate Dynamics	Dae-Sik Lim	KAIST

Luncheon Seminar 1		Halla Hall A (12:40–13:00)
		Chair : Jeong-Hwan Che (Seoul Natl. Univ.)
Intravital Confocal / Two-Photon Microscopy: In Vivo Cellular-Level Imaging of Live Animals Model	Sowon Lee	IVIM Technology

Luncheon Seminar 2		Halla Hall B (12:40–13:00)
		Chair : Keunwook Lee (Hallym Univ.)
Research Ethics and Security in the Age of AI	Byung chun Yoo	ITstandard

Luncheon Seminar 3		Samda Hall (12:40–13:00)
		Chair : Dae-Yong Kim (Seoul Natl. Univ.)
Are Laboratory Animals Still Essential? - The Scientific, Ethical, and Industrial	Jae-Jin Chang	Orient Bio Inc.

Poster Presentation 1		Lobby (13:20–14:20)
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Academy Award Presentation		Halla Hall A (14:00–14:40)
		Chair : Yang-Kyu Choi (Konkuk Univ.)
FXR as a Bridge Between Metabolic Dysfunction and Oncogenesis	Sungsoon Fang	Yonsei Univ.

Symposium 10 ENG		Halla Hall A (14:50–16:30)
In Vivo Explorations of Vascular Function and Dysfunction		
		Organizer / Chair : Jae-Hoon Choi (Hanyang Univ.)
Mechanism and Potential Therapies for Brain Arteriovenous Malformation	Suk P. Oh	Barrow Neurological Institute
Dissecting Cellular Dynamics through Genetic Ablation and Labeling of Vascular Cells	Junyeop Lee	Asan Medical Center
Animal Model and Its Application in Vascular Biology: Endothelial Migration and Differentiation	Heon-Woo Lee	Kyung Hee Univ.
Immune-Vascular Crosstalk within the Tumor Microenvironment	Keehoon Jung	Seoul Natl. Univ.

Symposium 11 [KMPC] KOR		Halla Hall B (14:50–16:30)
The Next Waves in Signaling Crosstalk From Cellular to Systemic Metabolism		
		Organizer : Je Kyung Seong (Seoul Natl. Univ.), Sungsoon Fang (Yonsei Univ.) / Chair : Jang-Seong Kim (KRIBB)
Glutamatergic Metabolic Synapse: Hepatocyte-Kupffer Cell Interactions in Liver Disease	Won-Il Jeong	KAIST
Enhanced Adipose Tissue Reservoir Capacity after Lactation Improves Insulin Sensitivity and Protects against Type 2 Diabetes	Joon Ho Moon	Seoul Natl. Univ. Bundang Hospital
Selenium's Paradoxical Role in Ferroptosis Modulation	Namgyu Lee	Dankook Univ.
Modulating Chronic Inflammation to Lower Cancer Risk: Mechanisms and Therapeutic Approaches	Jongho Park	Keimyung Univ.

PROGRAM

Symposium 12 [KALAS-CALAS] ENG			Samda Hall (14:50-16:10)
Preclinical Studies of Xenografts and Tissue Transplantation			
Organizer : Sun Shin Yi (Konkuk Univ.) / Chair : Ik Jin Yun (Konkuk Univ. Hospital), Dengke Pan (ClonOrgan Biotechnology Co., Ltd.)			
Homozygous Editing of Multiple Genes for Accelerated Generation of Xenotransplantation Pigs	Sen Wu	China Agricultural Univ.	
Major Progress in Xenotransplantation Preclinical and Clinical Research in China	Dengke Pan	ClonOrgan Biotechnology Co., Ltd.	
Current Progress in Corneal Xenotransplantation in South Korea	Mee Kum Kim	Seoul Natl. Univ.	
The Clinical Usefulness of Xenogeneic Skin Substitutes	Jun Hur	Hallym Univ. Hangang Sacred Heart Hospital	

KALAS-CALAS Joint Round Table Meeting	Room 304 (16:20-17:00)
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General Meeting & Welcome Reception	Tamna Hall (5F) (17:00-19:00)
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July 25 (Fri)			
Symposium 13 ENG			Halla Hall A (09:00-10:40)
Metabolic Mechanisms in Health and Diseases			
Organizer : Su Myung Jung (Sungkyunkwan Univ.) / Chair : Kyoung-Jin Oh (KRIBB)			
Phosphatidylethanolamine Content in the Endoplasmic Reticulum Regulates SREBP-1 Activation and Lipogenesis	Shunxing Rong	UT Southwestern	
Urolithin A: A Gut Metabolite as a Novel Therapeutic for Cardiac Dysfunction in Heart Failure	Chang-Myung Oh	GIST	
Exercise-Driven Musculoskeletal Alteration and Health	Hyeonwoo Kim	KAIST	
Dynamic Regulation of Anaplerotic Flux in Metabolic Disease	Haejin Yoon	UNIST	

Symposium 14 [KRIBB] ENG			Halla Hall B (09:00-10:40)
Translational Research Platform Using Animal Models for Inherited Retinal Disease (IRD)			
Organizer : Jeong Hun Kim (Seoul Natl. Univ.) Chair : Jeong Hun Kim (Seoul Natl. Univ.), Ki-Hoan Nam (KRIBB)			
Potential Role of Retinal Astrocytes in Retinopathy of Prematurity	Marcus Fruttiger	UCL	
Autonomous Tissue-Sensing Needle Injection System for Ocular Diseases	WonHyung Ryu	Yonsei Univ.	
Novel Bio-Imaging Technologies for Gene and Cell Therapy	Tae Geol Lee	KRISS	
Translational Research Platform for IRD in SNUH GEC-GCT	Jeong Hun Kim	Seoul Natl. Univ.	

Symposium 15 [NIFDS] ENG		Samda Hall (09:00–10:40)
Humanize Mouse Models for Preclinical Research		
Organizer : Dae-Yeul Yu (GHBio) / Chair : Dae-Yeul Yu (GHBio), Su-Hyung Park (KAIST)		
Generation of a NOG–W41–Based Humanized Mouse Model for In Vivo Human Hematopoiesis	Ryoji Ito	CIEM
4–1BB Co–Stimulation, a Promising Strategy for Treating Cancers	Su-Hyung Park	KAIST
Enhanced Human Leukocyte Engraftment and Reduced GvHD Symptoms in CD47;Rag2;IL2r γ Triple Knockout Mice	Seung-Ho Heo	Asan Medical Center
Target Discovery for Cancer Immunotherapy Using Humanized Mouse Models	Sung-Yup Cho	Seoul Natl. Univ.
Plenary Lecture 2 ENG		Halla Hall A, B (11:00–11:50)
Organizer / Chair : Won-Il Jeong (KAIST)		
Adipocyte Heterogeneity and the Regulation of Energy Metabolism	Christian Wolfrum	NTU Singapore
Luncheon Seminar 4		Halla Hall A (12:00–12:20)
Chair : Jun-Gyo Suh (Hallym Univ.)		
GONAD: Simple Method for Genome–Editing in Mice & Rats	Makoto Matsuyama	Shigei Medical Research Institute
Luncheon Seminar 5		Halla Hall B (12:00–12:20)
Chair : Dae-Youn Hwang (Pusan Natl. Univ.)		
Genetically Engineered Mice Models Accelerate Metabolic Disease Drug Development	Shuxin Xu	GemPharmatech Co., Ltd.
Luncheon Seminar 6		Samda Hall (12:00–12:20)
Chair : Jae-Ho Shin (Eulji Univ.)		
Promoting NH Financial Products	Yeong Gyun Gwon	NongHyup Financial Group INC.
Poster Presentation 2		Lobby (13:00–14:00)

PROGRAM

Symposium 16 ENG		Halla Hall A (14:00~15:50)
Development of 3D Composite Constructs with Immune Modulatory Functions and Tissue Regeneration Technologies		
Organizer / Chair : Kyung-Sun Kang (Seoul Natl. Univ.)		
Bioengineered Liver Crosslinked with Nano-Graphene Oxide Enables Efficient Liver Regeneration via MMP Suppression and Immunomodulation	Da-Hyun Kim	Sungshin Women's Univ.
Humanized Mouse Models in Hematopoietic Research: From Disease Mechanisms to Therapeutics	Kyung-Rok Yu	Seoul Natl. Univ.
Expanding the Frontiers of Spatial Cellular Analysis: Applications of SLACS in Animal Models and Organoid Systems	Chungwon Lee	Meteor Biotech
Advancing Liver Tissue Engineering: Improving Endothelial Cell Attachment in Decellularized Liver Scaffolds	Kyung-Mee Park	Chungbuk Natl. Univ.
Current Status of Human Tissue Substitutes Developed Using Transgenic Animals	Hyunil Kim	Optipharm Inc.

Symposium 17 ENG		Halla Hall B (14:00~15:50)
Latest Research Trends in Development of Experimental Animal Models and Biomarkers, and AI-Based Behavioral Analysis Technologies for Substance Use Disorder		
Organizer / Chair : Hye Jin Cha (Gyeongsang Natl. Univ.)		
TRPA1 as a Therapeutic Target for Cocaine Addictive Disorders	Choon-Gon Jang	Sungkyunkwan Univ.
Impaired Decision-Making in Methamphetamine-Dependent Rats: Neural Mechanism and Novel Treatment	Kiyofumi Yamada	Fujita Health Univ.
Inhaled Synthetic Cannabinoid Self-Administration Model and Its Application in Vaccine Development for Drug Abuse	Jaesuk Yun	Chungbuk Natl. Univ.
Non-Human Primate Models for Addiction Research: Behavior and Imaging	Dohyun Lee	OSONG Medical Innovation Foundation

Symposium 18 [KLAT Education 2] KOR		Samda Hall (14:00~15:50)
Application and Management of Genetically Modified Animals		
Organizer : Hanseul Oh (Chungbuk Natl. Univ.) / Chair : Hyunjhong Jhun (KFRI)		
Health Risks and Optimized Management in Genetically Engineered Lab Animals	Taehoon Shin	Jeju Natl. Univ.
Generation and Use of Genetically-Engineered Mouse Models Using CRISPR-Cas9	Young Hoon Sung	Asan Medical Center
Generation of Mouse Models and Experimental Applications	Kyoungmi Kim	Seoul Natl. Univ.

Closing Ceremony & Awards	Halla Hall A (16:00~16:40)
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July 26 (Sat)	
Satellite meeting	Halla Hall A (09:30~11:10)

CONFERENCE AND EVENT INFORMATION

1. 개회식 (Opening Ceremony)

July 24 (Thu) 09:30 ~ 09:40 / Halla Hall A,B

- 진 행: 복진웅 총무위원장
- 개회사: 이 호 학술위원장
- 인사말: 최양규 이사장

2. 총회 및 환영만찬 (General Meeting & Welcome Reception)

July 24 (Thu) 17:00 ~ 19:00 / Tamna Hall 5F

- 진 행: 복진웅 총무위원장
- 1) 총회 안건
- 2) 공로상 수여

성 명	소 속
故박천귀 대표	(주)쓰리샤인
故천병년 대표	(주)우정바이오

3) 학술상 시상식

① 실험동물학술상(대상)

성 명	소 속
황성순	연세대학교

② LAR 다수논문인용상

성 명	소 속
최영현	동의대학교
김종춘	전남대학교
남기택	연세대학교
고필옥	경상대학교

③ 특별상

성 명	소 속
정원일	KAIST 의과학대학원

④ 실험동물연구장학생

시상 : 이호 학술위원장

구 분	성 명	소 속
그룹 I	리우즈빈(Liu Zhibin) 김채연 최성은 송희진 설아윤 김규래 김솔빈	경북대학교 경북대학교 KAIST 의과학대학원 부산대학교 부산대학교 KAIST 의과학대학원 서울대학교
그룹 II	한승훈	한국생명공학연구원 실험동물자원센터

⑤ 한국실험동물기술원상

구 분	성 명	소 속
1급	공경환 이유진	서울대학교 가톨릭대학교

⑥ 2025년 한국실험동물기술원 우수합격자상

구 분	성 명	소 속
1급	김수연 윤혜빈	대구한의대학교 전북대학교
2급	이유림 노민영 유혜지	이노보테라퓨틱스 경북대학교 충남대학교

[2024년 한국실험동물기술원 우수합격자상]

구 분	성 명	소 속
1급	강소라 장동근	분당서울대학교병원 전임상실험센터 분당서울대학교병원
2급	김가은 신은정	상지대학교 (주)큐라티스

4) 환영만찬

3. 40주년 기념 비전 선포

- 진행: 복진웅 총무위원장
- 비전선포: 최양규 이사장
- 축사: 권석윤 원장 (한국생명공학연구원장)

4. 폐회식 (Closing Ceremony)

- July 25 (Fri) 16:00 ~ 16:40 / Halla Hall A
- 진행: 복진웅 총무위원장
 - 1) 우수포스터상 시상
 - 시상: 이호 학술위원장
 - 2) 경품추첨 및 40주년 기념 포토제닉 수상
 - 3) 폐회사
 - 인사말: 최양규 이사장
 - 폐회사: 이호 학술위원장
 - 4) 기념촬영

5. 관련위원회 회의 및 모임 (Committee & Meeting)

KALAS 평의회 (Council Meeting)

July 23 (Wed) 11:30~13:00 / Ocean view (5F)
참석대상 | 한국실험동물학회 평의원

KALAS 이사회 (Board Meeting)

July 24 (Thu) 10:40~11:30 / Room 301 (3F)
참석대상 | 한국실험동물학회 이사 및 감사

KALAS 우수포스터상 선정 회의

July 25 (Fri) 14:00~15:00 / Room 303 (3F)
참석대상 | 포스터 심사위원

KCLAM 회의

July 24 (Thu) 14:00~17:00 / Room 303 (3F)

KIRAMS-SKBP Joint Meeting

July 24 (Thu) 14:00~14:40 / Samda Hall (3F)

KALAS-CALAS Joint Round Table Meeting

July 24 (Thu) 16:20~17:00 / Room 304 (3F)
참석대상 | KALAS-CALAS 임원 및 관계자

산학협력 간담회

July 25 (Fri) 10:00~11:00 / Room 301 (3F)
참석대상 | 한국실험동물학회 협력기업 관계자

6. 점심식사 안내 (Luncheon Seminar)

런천세미나		기업명	장소	티켓 수령처
July 24 (Thu) 12:40~13:00	LS 1	IVIM Technology	Halla Hall A	09:20부터 기업부스 (선착순 100명)
	LS 2	ITstandard	Halla Hall B	09:20부터 등록대 (선착순 100명)
	LS 3	Orient Bio Inc.	Samda Hall	09:20부터 기업부스 (선착순 100명)
July 25 (Fri) 12:00~12:20	LS 4	Bio-Medical Science Co., Ltd.	Halla Hall A	08:50부터 기업부스 (선착순 100명)
	LS 5	GemPharmatech Co., Ltd.	Halla Hall B	08:50부터 기업부스 (선착순 100명)
	LS 6	NongHyup Life Insurance Co., Ltd.	Samda Hall	08:50부터 기업부스 (선착순 100명)

※ 런천 티켓은 한정 수량으로 조기 품절될 수 있습니다.

※ 런천 세미나 강연을 청취하신 후 퇴실 시 도장을 받고 식당에서 티켓 확인 후 식사가 제공됩니다.

(도장이 없는 경우 식사가 불가능한 점 참고 바랍니다.)

※ 식사장소: 3F 델리자 (Delizia)

LS 1: IVIM Technology

주제: Intravital Confoca / Two-Photon Microscopy: In Vivo Cellular-Level Imaging of Live Animals Model

연자: Sowon Lee

LS 2: ITstandard

주제: Research Ethics and Security in the Age of AI

연자: Byung chun Yoo

LS 3: Orient Bio Inc.

주제: Are Laboratory Animals Still Essential? - The Scientific, Ethical, and Industrial Nexus
Sustaining Progress in Life Sciences

연자: Jae-Jin Chang

LS 4: Bio-Medical Science Co., Ltd.

주제: GONAD: Simple Method for Genome-Editing in Mice & Rats

연자: Makoto Matsuyama

LS 5: GemPharmatech Co., Ltd.

주제: Genetically Engineered Mice Models Accelerate Metabolic Disease Drug Development

연자: Shuxin Xu

LS 6: NongHyup Financial Group INC.

주제: Promoting NH Financial Products

연자: Yeong Gyun Gwon

7. 실험동물연구장학생(그룹) 포스터 발표

발표시간	July 24 (Thu) 13:20~14:20
발표장소	ICC JEJU Lobby 3F (제주 국제컨벤션센터 3층 로비)
포스터 번호	PS-R-01 PS-R-02 PS-R-03 PS-R-04 PS-R-05 PS-R-06 PS-R-07 총 7개
부착 시간	July 24 (Thu) 09:00~11:00
철거 시간	July 24 (Thu) 17:00~17:30

※ 철거 시간 이후의 포스터 분실은 책임지지 않습니다.

※ 부착은 7월 24일 (목) 부착 시간 내에 반드시 진행해야 하며, 미부착시 제재가 주어집니다.

• 실험동물연구장학생 발표

- 포스터 발표는 좌장의 진행에 따라 포스터당 7분 (5분 발표, 2분 질의응답)으로 진행되며, 발표시간에 포스터 앞에 대기하여 주시기 바랍니다.

• 실험동물연구장학생 시상

- July 24 (Thu) 17:00 ~ 19:00 / Tamna Hall 5F (General Meeting & Welcome Reception)
- 실험동물연구장학생 발표자는 총회 및 환영만찬에서 진행되는 시상식에 반드시 참석해야 합니다. (대리수상불가)

8. 포스터 발표 (Poster Session)

발표시간	Poster Presentation 1	Poster Presentation 2
	July 24 (Thu) 13:20~14:20	July 25 (Fri) 13:00~14:00
발표장소	ICC JEJU Lobby 3F (제주 국제컨벤션센터 3층 로비)	
포스터 번호	PS-A-01~26 (해부생리) PS-B-01~44 (독성병리) PS-C-01~10 (미생물) PS-D-01~26 (유전자질환모델) PS-E-01~24 (시설운영 및 기타) 총130개	PS-A-27~49 (해부생리) PS-B-45~88 (독성병리) PS-C-11~20 (미생물) PS-D-27~51 (유전자질환모델) PS-E-25~47 (시설운영 및 기타) 총125개
부착 시간	July 24 (Thu) 09:00~11:00	July 25 (Fri) 09:00~11:00
철거 시간	July 24 (Thu) 17:00~17:30	July 25 (Fri) 17:00~17:30

※ 철거 시간 이후의 포스터 분실은 책임지지 않습니다.

• 포스터 심사

- 포스터 발표는 좌장의 진행에 따라 포스터당 4분 (3분 발표, 1분 질의응답)으로 진행되며, 과학적 성과와 발표자의 발표력 등을 기준으로 우수포스터를 선정하여 시상하오니 반드시 발표시간에 포스터 앞에 대기하여 주시기 바랍니다. (해당 시간에 발표자가 없는 포스터는 우수 포스터상 심사 대상에서 제외)

• 미부착 포스터: 포스터 보드에 2회 이상(개수로 적용) 미부착 시, 교신저자(연구책임자)에게 향후 1년간 포스터 제출 불가의 제재가 주어집니다.

• 우수포스터상 시상

- July 25 (Fri) 16:00 ~ 16:40 / Halla Hall A (Closing Ceremony)
- 우수포스터의 경우 폐회식에서 선정자를 호명합니다. 호명 시 자리에 없으면 다음 우수자에게 상이 수여되오니, 학술대회 종료일까지 학술대회에 꼭 참석해 주시기 바랍니다. (상장과 상금 수여, 대리수상불가)

9. 이벤트 및 경품추첨 안내 (Event & Giveaway)

1) 환영만찬 노래자랑 이벤트

진행일시 | July 24 (Thu) 17:00 (General Meeting & Welcome Reception)

참여방법 | 사전접수 및 현장접수

경 품 | 상품권 30/20/10/1만 원권, 기타 등

2) 40주년 기념 포토제닉 이벤트

진행일시 | July 23 (Wed) 부스 오픈 시 ~ July 25 (Fri) 13:00 / 폐회식(Closing Ceremony) 중 시상

참여방법 | 현장에서 네컷사진 촬영 후 판넬에 부착

경 품 | 상품권 20/10/5만 원권

3) 실험동물기술원 교육강연 퀴즈 이벤트

진행일시 | July 25 (Fri) 15:30 ~ 15:50 [KLAT Education 2] 이후 진행 / Samda Hall

참여방법 | 스마트폰 앱 / Play 스토어에서 퀴즈를 위한 카훗(kahoot)앱 다운로드 후 참여

경 품 | 에어팟 프로(2세대), 상품권 10/5/3/1만 원권

4) 폐회식 경품추첨

진행일시 | July 25 (Fri) 16:00 ~ 16:40 (Closing Ceremony)

참여방법 | ① KALAS2025국제 App 다운로드

② 국제학술대회 설문지 작성

③ App을 열어 40개 부스(40주년 홍보관 필수) 스탬프 투어 달성

※ 부스 투어 이벤트 마감: 7/25(Fri) 14:30 까지

경 품 | 갤럭시탭 S10플러스, 상품권 30/10/5/1만 원권 등



※ KALAS Exhibition Hall 전시장 운영 규칙

- 전시장에서 입장 시에는 항상 명찰이 잘 보이도록 착용하여야 한다.
- 전시장에 참여한 단체의 활동은 배정된 구역 내에서 진행되어야 한다.
- 해당 소속 전시부스 이외의 타사 부스에 허락없이 들어갈 수 없다.
- 사진 촬영은 금지한다.
- 해당 부스의 전시 책임자의 허가 없이 타 참여사의 제품 혹은 장비를 사진, 동영상, 기타 방식으로 기록 및 보관할 수 없다.
- 전시장을 포함하여 컨벤션홀 내부 전체는 모두 금연이다.

Safety & Security

- 모든 참여사의 인원들은 전시장 내부에서 항상 명찰이 잘 보이도록 착용하여야 한다.
- 발급받은 명찰들은 어떠한 방식으로든 변경하거나 삭제할 수 없다.
- 참여 단체의 전시에 참여하는 인원은 해당 전시사의 허가 없이 타사 부스에 들어갈 수 없다.

KALAS 규정

- 전시장에 참여하는 참여사 인원과 학회 참가자는 모두 KALAS의 규정에 따라야 한다.
- 규정에 어긋나거나 학술대회 및 전시장에 심각한 문제를 일으키는 경우, KALAS의 권한으로 전시사 및 참가자는 패널티를 부여받을 수 있다.
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TRANSPORTATION INFORMATION

[제주 국제컨벤션센터(ICC)]

- 주소: 제주특별자치도 서귀포시 중문관광로 224 (중문동)
- 전화: 064-735-1000
- 홈페이지: iccjeju.co.kr

[교통안내]

1. 리무진 버스 (600번 제주공항 ↔ 중문관광단지)

- 탑승장: 공항정문 1층 5번 게이트 왼쪽 리무진 버스 승차장
- 운행표

서귀포칼호텔→파라다이스호텔입구→서복전시관→서귀포항(화인호텔)→샛기정공원→제주월드컵경기장→
서건도→악근천(켄싱턴리조트)→왕대왓→강정새마을금고→월평마을→약천사→대포항→제주국제컨벤션센터→
씨엔에스호텔→리틀프린스뮤지엄→그랜드 조선 제주→중문골프장→파라나스호텔(하얏트)→
스위트호텔(신라호텔입구)→블룸호텔/한국관광공사→켄싱턴리조트 중문점입구(롯데호텔입구)→
여미지식물원입구→중문관광단지입구→예래입구→창천리→동광환승정류장→정존마을→한라병원→
삼무공원사거리/롯데시티호텔→제주 선히호텔→공항

※ 항공기 이착륙 시간 변경, 기상이변, 행사관계 등으로 수송수요에 변동이 생길 경우에는 증회 또는 운행시간이 조정될 수 있음.

※ 공항 출발 22:20, 22:50분 차량은 테디벨리골프장 정류소 정차

※ 공항 출발 22:40분 차량은 수모루 정류소 정차

- 운행시간 | (매 16~40분 간격 운행)

(1) 제주국제공항출발 (06:00~10:40)

(2) 서귀포(칼호텔)출발 (06:37~22:15)

※ 전체 운영시간표 사이트 참고 → <http://bus.jeju.go.kr/schedule/viewNew/600>

- 이용요금: 공항에서 ICC JEJU까지 편도(성인) 4,500원

- 이용문의: 삼영교통 (064) 713-7000

2. 택시 (제주공항 ↔ 중문)

- 택시승차장 장거리, 단거리 확인 - 제주공항 택시승차장에서 이용시, 장거리 승차장에서 출발하여 오십시오.
요금은 미리 정해져 있으므로 승차전에 확인하세요.
- 이용안내: 약 50분 (거리 42km)

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SYMPOSIUM 1 [K-MEDI hub, KBIO HEALTH] KOR

July 23 (Wed) 14:00–15:50 | Halla Hall A

Current Trends in Nonclinical Testing With Non-Rodent Animal Models: Focus on Common Marmosets and Pigs

Organizer / Chair : Myung-Hoon Chung (K-MEDI hub), Jayoung Jeong (OSONG Medical Innovation Foundation)

The Minipig in Medical Device Development

Ryounghoon Jeon (K-MEDI hub)

Research on Tissue-Engineered Scaffolds Combined with Stem Cell Therapies Utilizing Animal Models

Sang Jin Lee (Univ. of Hong Kong)

Research Methodology Developments for Common Marmoset (*Callithrix jacchus*)

Dohyun Lee (OSONG Medical Innovation Foundation)

Bridging the Rodent to Human Translational Gap: Marmoset as a Model System for Biomedical Research

Jung Eun Park (Univ. of Pittsburgh)



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The Minipig in Medical Device Development

Ryounghoon Jeon, Sang-Hyun An, Sang Kyoon Kim, Myung-Hoon Chung

*Preclinical Research Center, Daegu Gyeongbuk Medical Innovation Foundation (K-MEDI hub),
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Minipigs have become increasingly important with the development of biotechnology. In particular, the biological similarities between minipigs and humans make them an irreplaceable species for evaluating medical devices developed for human use.

One of the main characteristics that makes minipigs suitable for selection is their anatomical size, which is optimized for biomedical research. Compared to rodents, they have a body size large enough to accommodate human medical devices, but unlike farm-pigs or other large animals, they have a slow growth rate, allowing for long-term research. The reproductive physiology of minipigs is another advantage that makes them a good choice for research. The short gestation period and rodent-like litter size to are useful for genetic similarity, and the knowledge accumulated from farm pigs over a long period of time enables a stable supply of animals, which not only allows for consistent research, but also reduces the number of animals used, which is advantageous in terms of improving laboratory animal ethics. Despite these advantages, however, there are still difficulties in using minipigs in experiments. Their body size makes them incompatible with rodent-based research facilities, which limits their research infrastructure, reduces accessibility to minipigs, and incurs high costs. In addition, the lack of established disease models compared to rodents is another hurdle for research using minipigs.

Therefore, considering the current research environment, the strategy of expanding minipig research facilities and accumulating public pathophysiological reference data will be the basis for successful development of medical devices using minipigs.

Key words : Anatomy, Infrastructure, Medical device, Minipig, Physiology

Research on Tissue-Engineered Scaffolds Combined with Stem Cell Therapies Utilizing Animal Models

Sang Jin Lee

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Tissue engineering has emerged as a pivotal field in regenerative medicine, particularly through the integration of advanced biomaterials and stem cell therapies. This presentation explores our research on tissue-engineered scaffolds combined with stem cell therapies utilizing various animal models. Bioengineered research work has primarily focused on the development of hydrogels and 3D printing techniques, alongside the application of nano/micro-particles, to reconstruct damaged tissues. It has conducted extensive studies using rodent models (mice and rats) to investigate inflammation responses, immune reactions, and tissue integration. These models have provided critical insights into the biological interactions between engineered scaffolds and the host environment. Additionally, our research on rabbits has included successful trachea reconstruction and calvarial defect repair, demonstrating the potential for complex hard tissue regeneration. Furthermore, It has leveraged pig models for the development of cardiovascular stents, offering valuable data on long-term biocompatibility and functional outcomes. Notably, bioengineered studies involving Beagle dogs and primates have advanced our understanding of tissue engineering applications in larger, more complex biological systems. This research underscores the necessity of animal models in validating the efficacy and safety of tissue engineering strategies before clinical application.

By utilizing diverse species, we can better mimic human physiological responses, paving the way for innovative therapeutic solutions. The ongoing advancements in this field hold significant promise for improving patient outcomes in regenerative medicine, highlighting the critical role of interdisciplinary research in overcoming current challenges and enhancing tissue regeneration techniques. This presentation aims to provide an overview of our findings, the rationale behind using animal models, and the future prospects of tissue-engineered solutions in clinical settings.

Key words : Biomaterials, Implantation, Transplantation, Organ reconstruction, Stem cell therapy

Research Methodology Developments for Common Marmoset (*Callithrix Jacchus*)

Dohyun Lee

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July 23 (Wed)

The common marmoset (*Callithrix jacchus*) offers several advantages in biomedical research, including small body size, low risk of zoonosis, and high fecundity, compared to macaque monkeys. However, marmosets cannot fully replace macaques in all areas of research, and their advantages can also present challenges. For instance, while their small size allows for easier handling and requires less space for housing and experiments, it also makes procedures involving micro-structures or surgery more difficult. Additionally, due to genetic and metabolic differences, certain drugs may exhibit different pharmacological profiles in marmosets compared to macaques and humans. Marmosets also have limitations in therapeutic and vaccine research for infectious diseases because they may respond differently to some human pathogens.

Our center has worked to expand the potential research applications of the common marmoset. For example, we developed a miniaturized laparoscopic ovum pick-up system to overcome limitations related to their small size (Theriogenology Wild, 2025). We also evaluated the pharmacokinetics of human antibody drugs in marmosets, sequenced their genome using next-generation sequencing (NGS), and analyzed the data with artificial intelligence (AI). Furthermore, we developed marmoset infection models for critical human pathogens.

As a result of nearly a decade of work, we have been able to clearly define the areas of research where marmosets can and cannot be effectively used.

Key words : Non-human primates, Assisted reproductive techniques, Animal biosafety level 3, Whole genome sequencing, Pharmacogenomics

Bridging the Rodent to Human Translational Gap: Marmoset as a Model System for Biomedical Research

Jung Eun Park, Gregg E Homanics, Stacey J Sukoff Rizzo,
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Alzheimer's disease (AD), the most common cause of dementia in aging populations, affects over 55 million individuals worldwide, with that number expected to rise as global life expectancy increases. Despite decades of research, a limited understanding of the mechanisms underlying the onset and progression of AD continues to impede the development of therapies capable of halting, preventing, or curing the disease. We propose that the development of a non-human primate model of AD is a critical next step toward addressing the limitations of existing model systems. Such models are essential for investigating primate-specific cellular and molecular mechanisms that underlie the pathogenesis and progression of AD.

We hypothesize that the common marmoset (*Callithrix jacchus*) represents an ideal non-human primate model for AD. To test this hypothesis, we are developing and studying two complementary marmoset cohorts: gene-edited models carrying AD-associated genetic risk variants, and genetically diverse wild-type marmosets that age naturally. These animals are monitored longitudinally from birth throughout their lifespan using non-invasive methods. Our multidisciplinary approach enables detailed characterization of the genetic, molecular, functional, behavioral, cognitive, and pathological changes associated with both normal aging and AD. This comprehensive framework is designed to uncover key primate-specific mechanisms underlying AD pathogenesis and progression.

By establishing and studying these marmoset models, we aim to uncover primate-specific disease mechanisms, overcome the translational limitations of rodent models, and lay a stronger foundation for therapeutic development. Ultimately, this work is poised to accelerate the discovery and implementation of effective treatments for AD.

Key words : Marmoset, Non-human primate, Alzheimer's disease, Genetic engineering

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SYMPOSIUM 2 ENG

July 23 (Wed) 14:00–15:40 | Halla Hall B

MPS and Organoid Research and Laboratory Animal Science

Organizer : **Jaejin Cho** (Seoul Natl. Univ.)

Chair : **Hyung-Sik Kim** (Pusan Natl. Univ.), **Jaejin Cho** (Seoul Natl. Univ.)

Advanced Human Microphysiological Systems for Disease Modeling and Drug Development

Deok-Ho Kim (Johns Hopkins Univ.)

MPS and Organoids as New Alternative Methods (NAMs): Current & Upcoming Trend

Nakwon Choi (Korea Univ.)

Microfluidics–Embedded 3D Perfusion Cell Culture System

Jeung Sang Go (Pusan Natl. Univ.)

Therapeutic Organoid Platform, “Tissue Reforming Technology Platform” and Animal Model

Jaejin Cho (Seoul Natl. Univ.)



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Advanced Human Microphysiological Systems for Disease Modeling and Drug Development

Deok-Ho Kim

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In alignment with the FDA Modernization Act 2.0, there is a growing emphasis on the development and adoption of human-relevant microphysiological systems (MPS), such as organs-on-chips and organoids, to reduce reliance on traditional animal models in preclinical drug development and safety evaluation. These innovative platforms are poised to transform the biomedical research ecosystem by improving translational predictability, accelerating therapeutic discovery, and addressing critical gaps in disease modeling. In this talk, I will present our lab's recent efforts to advance MPS technologies through the integration of multi-scale biofabrication, patient-specific organotypic cultures, and AI-enhanced biosensing platforms. Our work focuses on building scalable, high-throughput organ-on-chip systems embedded with real-time phenotypic biosensors that enable dynamic monitoring of tissue function and drug responses. Specifically, I will highlight three representative case studies: (1) a microphysiological model of Duchenne muscular dystrophy (DMD) for gene therapy; (2) an automated 96-well heart-on-a-chip platform for evaluating drug-induced cardiotoxicity, enabling early-stage compound screening at scale; and (3) a spaceflight-induced muscle aging model developed to study the impact of microgravity on human musculoskeletal health aboard the International Space Station. Together, these studies illustrate how MPS can offer mechanistic insights into complex human pathophysiology under both terrestrial and extraterrestrial conditions. Our approach not only supports safer and more effective drug development but also aligns with regulatory shifts toward next-generation testing paradigms, paving the way for precision medicine and innovation in biomedical science.

Key words : Microphysiological systems (MPS), Organs-on-chip, Organoid, Biomaterials, Biofabrication

MPS and Organoids as New Alternative Methods (NAMs): Current & Upcoming Trend

Nakwon Choi

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This presentation introduces a recently announced National Institutes of Health (NIH) program called Compliment Animal Research in Experimentation (Compliment-ARIE). More specifically, this presentation includes the purpose, objectives, framework, technical components, and how stake holders conceived the Compliment-ARIE. As initial academic background, this presentation covers concept, definition, and applications of complex in vitro systems, representatively microphysiological systems (MPSs) and organoids. This presentation also introduces the history of the NCATS Tissue Chip Project to explain how the Compliment-ARIE has evolved into a next 10-year program. Information from this recent US nation-wide trend would allow for better understanding the expected value of new alternative methods (NAMs) toward recapitulating human physiology and pathology and utilizing new tools for drug discovery.

Key words : Compliment animal research in experimentation, New alternative methods, Complex in vitro systems, Microphysiological systems, Organoids

Microfluidics-Embedded 3D Perfusion Cell Culture System

Donghyuk Park, Jeung Sang Go*

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Recently, 3-dimensional in vitro cell cultures have been gained much attention in biomedical sciences because of their closer relevance between in vitro cell cultures and in vivo environments. This paper presents a microfluidic perfusion 3D cell culture system with consistent control of long-term culture conditions to mimic in vivo microenvironment. It consists of two sudden expansion reservoirs to trap incoming air bubbles, gradient generators to provide a linear concentration, and microchannel mixers. Specifically, the air bubbles disturb a flow in the microfluidic channel resulting in the instability of the perfusion cell culture conditions. For long-term stable operation, the sudden expansion reservoir is designed to trap air bubbles by using buoyancy before they enter the culture system. The performance of the developed microfluidic perfusion 3D cell culture system was examined experimentally and compared with analytical results. Finally, it was applied to test the cytotoxicity of cells infected with Ewing's sarcoma. Cell death was observed for different concentrations of H₂O₂. For future work, the developed microfluidic perfusion 3D cell culture system can be used to examine the behavior of cells treated with various drugs and concentrations for high-throughput drug screening.

Key words : 3D, Cell culture, Microfluidics, Perfusion, Screen

Therapeutic Organoid Platform, “Tissue Reforming Technology Platform” and Animal Model

Eunjeong Seo, Jin Ju Park, HyeRan Gwak,
Jieun Lee, Hyun Ji Kim, Jaejin Cho*

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Therapeutic organoids hold great promise in regenerative medicine, yet their clinical application remains limited due to challenges in structural controllability, reproducibility, scalability, and batch variation. Most current systems rely on artificial scaffolds or matrices and often fail to replicate the complexity of in vivo tissues. To overcome these barriers, we developed the Tissue Reforming Technology Platform (TRTP), a scaffold-free system that enables stem cells to self-organize into millimeter-scale organoid tissue modules (OTMs). TRTP generates highly reproducible and biologically relevant tissue structures without exogenous materials. In this study, we evaluated TRTP-generated OTMs in preclinical animal models, demonstrating successful tissue integration and regenerative efficacy. Histological and functional assessments supported their therapeutic potential across various applications. Our results suggest that TRTP is a next-generation platform that addresses the limitations of conventional organoids and offers a practical pathway toward clinically viable regenerative organoid therapies.

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SYMPOSIUM 3 [IACUC] KOR

July 23 (Wed) 14:00–15:40 | Samda Hall

Strengthening Biosafety Risk Assessment and Management in Laboratory Animal Research

Organizer : Ji Min Lee (Seoul Natl. Univ.) / Chair : Jung Joo Hong (KRIBB)

Risk Assessment Strategies for Research Involving Biological Agents

Won-Jong Jang (Konkuk Univ.)

Biosafety Management for Animal Experiments in ABL-3 Laboratories

Kyung Min Lee (International Vaccine Institute)

Current Status and Future Development of Biosafety Education Programs

Tae Jong Son (Korea Disease Control and Prevention Agency)

Predicting Hazards in Laboratory Work by Deep Learning Towards Risk Assessment and Safety Management

Takaaki Harada (Nagoya Univ.)



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Risk Assessment Strategies for Research Involving Biological Agents

Won-Jong Jang

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Risk assessment is a vital process in laboratories to ensure biosafety, particularly when working with animals. It involves evaluating factors such as research subjects, methods, facilities, equipment, and personnel prior to conducting any experiment. This process helps identify potential hazards and implement strategies to minimize them, ultimately reducing the likelihood of accidents. Over time, this comprehensive and continuous approach has evolved into what is now commonly referred to as "risk management."

Biosafety is a very important consideration in laboratories involving animals. Biosafety refers to guidelines and procedures for preventing and managing risks that may arise during the handling of biological hazards. Experiments involving animals require more stringent safety management because they involve unique risks, such as aerosol generation, animal bites or scratches, and pathogen shedding.

Risk assessment should be conducted for safety protection not only in laboratories that study organisms, but also in conducting animal-related research. Animal biosafety levels (ABSLs) are used to classify safety precautions required to raise experimentally infected animals, animals administered recombinant DNA (rDNA), or laboratory animals that may naturally harbor zoonotic infectious agents. In animal-related research, the risk factors of the animals themselves are very important before the factors related to the research. There are fundamental risk factors such as the sudden behavior of laboratory animals, the presence or absence of zoonotic infectious diseases that animals carry, the method of handling animals, and the conditions of the cages and facilities.

In this way, in animal-related experiments, a risk assessment is conducted, and a research plan that includes safety assurance through risk assessment is approved after deliberation by the Institutional Animal Care and Use Committee (IACUC) and the Institutional Biosafety Committee (IBC), thereby creating a safer research environment.

Key words : Biosafety, Animal experiment, Risk assessment, Risk management

Biosafety Management for Animal Experiments in ABL-3 Laboratories

Kyung Min Lee

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Animal Biosafety Level 3 (ABL-3) laboratories are essential for conducting high-risk pathogen research involving animal models. These facilities require rigorous biosafety practices due to the increased risk of zoonotic transmission and laboratory-acquired infections.

Risk assessment provides a systematic approach to identifying potential hazards associated with specific pathogens, animal species, and experimental procedures. This process enables the implementation of tailored safety measures, including standard operating procedures (SOPs), facility design and personal protective equipment (PPE) requirements. A thorough risk analysis serves as the foundation for minimizing exposure risks and ensuring regulatory compliance.

Scenario-based emergency planning is also a core element of high-containment laboratory management. Developing and analyzing realistic incident scenarios—such as accidental spills, animal escape, or aerosol-generating exposures—allows for the creation of structured response protocols. These include clear chains of communication, containment procedures, and medical follow-up actions. Scenario planning enhances institutional readiness and helps prevent escalation during biosafety incidents.

Combining risk-based planning with scenario-specific emergency response strengthens both the safety culture and operational resilience of ABL-3 laboratories. These approaches support a proactive framework for safeguarding personnel, the environment, and public health, and can serve as a reference for institutions involved in high-containment animal research.

Key words : Biosafety, Animal Biosafety Level 3, Risk assessment, Laboratory-acquired infection

Current Status and Future Development of Biosafety Education Programs

Tae Jong Son

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In ROK, the installation and operation of all biosafety research facilities handling high-risk pathogens, toxins, or LMOs, as well as infectious animals using them, must be reported to or permitted by the government depending on their biosafety level. Institutions operating biosafety level 2 or higher facilities are required to establish an Institutional Biosafety Committee (IBC) to review research plans and manage safety implementation and biosafety training completion.

According to the "Infectious Disease Control and Prevention Act," the "Living Modified Organisms Act," and the "Laboratory Safety Act," biosafety education is mandated by law for personnel working in research facilities handling pathogens or infectious animals using them. To ensure efficiency and expertise in managing biosafety education, the Korea Disease Control and Prevention Agency (KDCA) designates biosafety education institutions and regularly operates biosafety education programs. These programs are categorized according to the roles of the personnel involved such as facility operation managers, biosafety officers, research staff, facility managers, and managers responsible for high-risk pathogens.

KDCA has established a specialized hands-on training facility equipped with specifications and functions equivalent to biosafety level 3 (BSL-3) laboratories. At this facility, biosafety officers and researchers handling high-risk pathogens receive periodic BSL-3 practical training.

KDCA plans to continuously develop and operate biosafety education programs that correspond to the roles and responsibilities of personnel within institutions handling high-risk pathogens, aiming to improve the quality of legally mandated biosafety training.

Key words : Laboratory, Biosafety, High-risk pathogen, LMO

Predicting Hazards in Laboratory Work by Deep Learning Towards Risk Assessment and Safety Management

Takaaki Harada,* Tomita Kengo

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Laboratory work often involves hazardous materials and operations. University researchers and students use fragile glassware for chemical syntheses, sharp scalpels for dissection of mice, and a harmful chloroform/phenol solution for DNA extraction. Such work is necessary for advanced research, but it may result in an accident. A previous study has shown that science-degree students who conduct experiments have the six-times higher risk of an accident than the other students. It is important to assess the risk of a laboratory accident when making a work plan. The first and most critical step is to identify all the hazards associated with laboratory work. However, it is often difficult especially for inexperienced researchers and students. In this presentation, we demonstrate that deep learning models learnt from past laboratory accidents can predict possible hazards associated with a plan of laboratory work. The prediction models were trained on the reports of laboratory accidents that actually occurred in Japanese universities. The training dataset consisted of descriptions of those reports labeled with the most and second most critical hazards to prevent the same accident. The model training was accelerated with a GPU (NVIDIA GeForce RTX 3090). For hazard prediction, we input the description of a laboratory work plan into the trained prediction model, then it predicted the associated hazard as an output. For instance, when a laboratory work plan of "Make a tissue slide" was input into the prediction model, it output "Risk of sharp injury," warning the hazard of sharp items that can cause cut injuries. It was presumed that the model did not simply make a prediction based on keywords such as "glass" and "microtome." Overall, the models showed the sensible performance of hazard prediction. Our deep learning models have the potential application towards risk assessment and safety management of research activities.

Key words : Accident analysis, Laboratory safety, Natural language processing, Multiclass classification, Artificial intelligence

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SYMPOSIUM 4 [KFRI]

KOR

July 23 (Wed) 16:00–17:40 | Halla Hall A

Animal Models in Nutrition Research and Functional Food Development

Organizer : Hyunjhun Jhun (KFRI), Ho-Young Park (KFRI) / Chair : Ho-Young Park (KFRI)

Integrating Chrono-Nutrition and Probiotics in Organoids
for Functional Food Development

Miri Park (KFRI)

Alterations in Mitochondria-Associated Molecular Patterns and the Role of Dietary
Components in Improving Inflammation and Metabolic Disease

Yu Geon Lee (KFRI)

Development of New Concept Efficacy Evaluation Analysis Technology
Utilizing Mouse Resources for Modeling Human Disease

Hea-Jong Chung (KBSI)

Effects of the Complex of Panicum Miliaceum Extract and
Triticum Aestivum Extract on Hair Condition

Pan-Young Jeong (Novarex Co., Ltd.)



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Integrating Chrono-Nutrition and Probiotics in Organoids for Functional Food Development

Miri Park

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Chrono-Nutrition, aligning dietary timing with circadian rhythms, and probiotic supplementation offer innovative approaches to enhance gut and metabolic health. Evidence shows that meal timing influences circadian clocks, shaping gut microbiota and host metabolism. This presentation highlights intestinal organoids as powerful platforms to explore circadian functions in gut epithelial cells, elucidating their roles in nutrient absorption, immune regulation, and barrier integrity.

Probiotics, such as *Lactobacillus* species, support circadian regulation by modulating microbial communities and immune responses, with potential neurocognitive benefits. Synchronizing probiotic administration with circadian cycles, guided by Chrono-Nutrition principles, improves glucose homeostasis and lipid metabolism. Probiotic-derived short-chain fatty acids (SCFAs) further strengthen gut barrier function and maintain microbial balance.

Integrating Chrono-Nutrition with microbiome-based interventions paves the way for next-generation functional foods to prevent and manage metabolic disorders through optimized gut-microbe-host interactions. These organoid-based insights, complemented by preclinical models, underscore the potential for targeted nutritional strategies.

Key words : Chrono-nutrition, Probiotics, Organoids, Circadian rhythms, Gut health

Alterations in Mitochondria-Associated Molecular Patterns and the Role of Dietary Components in Improving Inflammation and Metabolic Disease

Yu Geon Lee

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Mitochondria are subcellular organelles that serve as central hubs for bioenergetic, biosynthetic, and signaling processes. Beyond their classical role in ATP production, mitochondria are essential for cellular adaptation to stress and the regulation of immune and metabolic homeostasis. Dysregulation of mitochondrial function has been implicated in a broad spectrum of pathological conditions, including chronic metabolic disorders, cancer progression, and resistance to chemotherapy. Cells in diseased environments are frequently subjected to metabolic stressors such as nutrient deprivation, hypoxia, and oxidative damage. Prolonged or excessive exposure to these stressors can compromise mitochondrial integrity, leading to impaired metabolism, apoptosis, or oncogenic transformation, thereby aggravating inflammation and metabolic dysfunction. Emerging evidence indicates that certain natural dietary compounds can preserve mitochondrial integrity and modulate associated signaling pathways. These bioactive molecules, often structurally simple and water-soluble, have attracted attention as promising therapeutic candidates for the prevention and management of metabolic diseases and cancer. Our research highlights the anti-inflammatory and anti-proliferative properties of selected dietary components, particularly through their capacity to regulate key mitochondrial functions, including fatty acid oxidation, oxidative phosphorylation, and programmed cell death. These recent findings suggest that targeting mitochondrial health with specific dietary compounds may offer a novel strategy to restore mitochondrial function, reduce chronic inflammation, and suppress tumor development. Collectively, this work supports the potential of mitochondria-targeted nutritional and pharmacological interventions for combating metabolic disorders and cancer.

Key words : Mitochondria, Dietary compounds, Metabolic stress, Chronic inflammation, Mitochondria-targeted therapy

Development of New Concept Efficacy Evaluation Analysis Technology Utilizing Mouse Resources for Modeling Human Disease

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This study aims to establish a preclinical efficacy evaluation platform for microbiome-based therapeutic strategies in response to the increasing demand for translational models that closely reflect human diseases. To this end, we developed disease-specific cell-based and mouse models targeting major conditions such as muscle loss, cognitive impairment, stress-related mood disorders (e.g., depression), and alopecia. Using these models, we screened a wide array of gut microbiota and successfully identified disease-specific microbial candidates with therapeutic relevance. Furthermore, comprehensive efficacy evaluations including molecular, histological, and behavioral analyses, were conducted based on the pathological characteristics of each disease model using the selected microbes. This efficacy evaluation system was established by utilizing the specialized infrastructure of the Animal Facility of Aging Science (AFAS) at the Korea Basic Science Institute (KBSI), which enables the maintenance and long-term monitoring of aged animal models. This in-house capability allows for physiologically meaningful, disease-specific, and reproducible assessments essential for aging-related research. By bridging disease modeling with microbiome-based intervention, this platform provides a robust and scalable framework for developing and validating next-generation microbiome therapeutics. We anticipate that this platform will contribute to advancing of precision microbiome medicine across a wide range of age-related and chronic diseases.

Key words : Microbiome-based strategies, Preclinical disease models, Aging-related conditions, Gut microbiota profiling, Host-microbe interactions

Effects of the Complex of *Panicum Miliaceum* Extract and *Triticum Aestivum* Extract on Hair Condition

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Proso millet (*Panicum miliaceum* L.) and common wheat (*Triticum aestivum* L.) have been used as major crops in multiple regions since ancient times, and they contain various nutrients that can affect human hair health. This study investigated the various biological effects of a complex of millet extract and wheat extract (MWC) on hair health. Human immortalized dermal papilla cells (iDPCs) for an in vitro study and an anagen-synchronized mouse model for an in vivo study were employed. These findings revealed that the application of the MWC in vitro led to an increase in the mRNA levels of antioxidant enzymes (catalase and SOD1), growth factors (IGF-1, VEGF, and FGF7), and factors related to hair growth (wnt10b, β -catenin) while decreasing inflammatory cytokine mRNA levels (IL-6 and TNF α). The mRNA levels of hair follicles (HFs) in the dorsal skin of the mouse model in the early and late telogen phases were also measured. The mRNA levels in the in vivo study showed a similar alteration tendency as in the in vitro study in the early and late telogen phases. In this model, MWC treatment elongated the anagen phase of the hair cycle. These findings indicate that the MWC can suppress oxidative stress and inflammation and may elongate the anagen phase by enhancing the growth factors involved in the wnt10b/ β -catenin signaling pathway. This study suggests that the MWC might have significant potential as a functional food for maintaining hair health.

Key words : Complex of millet extract and wheat extract, Hair health, Anagen elongation, Antioxidant, Growth factor, Functional food ingredient

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SYMPOSIUM 5 [KIT] **KOR**

July 23 (Wed) 16:00–17:40 | Halla Hall B

The Use of Non-Human Primates in Non-Clinical Studies Ranges From Management to Research

Organizer : Han Na Suh (KIT) / Chair : Gee Euhn Choi (Jeju Natl. Univ.)

Considerations for Research with NHPs

Eun Ju Song (Emory Univ.)

Approaches to Microbiological Quality Control in Laboratory Non-Human Primates

Kyoung-Sun Lee (OSONG Medical Innovation Foundation)

Application of Non-Human Primates in Advanced Biopharmaceuticals Development

Han Na Suh (KIT)



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Considerations for Research with NHPs

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Nonhuman primates (NHPs) play a critical role in biomedical research due to their close genetic, physiological, and behavioral similarities to humans. Studies involving NHPs are particularly valuable in neuroscience, immunology, infectious diseases, and pharmacology, where alternative models may lack translational relevance. However, the use of NHPs requires careful ethical, scientific, and logistical considerations. Key factors include rigorous justification of their use, implementation of the 3Rs (Replacement, Reduction, and Refinement), adherence to animal welfare regulations, and ensuring high standards of housing and care. Researchers must also consider the complexity of NHP social and psychological needs, potential impacts on study outcomes, and challenges related to cost, availability, and public perception.

Key words : NHP, Ethics, Refinement, Welfare

Approaches to Microbiological Quality Control in Laboratory Non-Human primates

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Non-human primates (NHPs) are indispensable animal models in laboratory research involving advanced biopharmaceutical development, infectious disease, and aging. However, their close genetic relationship to humans also increases the risk of zoonotic transmission from microbial infections, which can threaten both human health and the Reproducibility of experimental outcomes. Therefore, microbiological quality control (MQC) is a critical component of NHP facility management.

Compared to rodents, which can be maintained in specific pathogen free (SPF) conditions via embryo transfer (ET), NHPs pose unique difficulties due to their longer developmental periods and the necessity of social interactions for normal behavioral development. These factors complicate the MQC and increase the ethical and economic burden associated with euthanizing infected animals.

This presentation outlines practical approaches to implementing effective MQC systems in laboratory NHP facilities. Key considerations include the identification of zoonotic agents, their potential impact on research, and reference to international standards. Based on these factors, institutions should define controlled pathogens, determine appropriate testing frequencies and sampling strategies, and decide whether to conduct testing in-house or through external laboratories. Furthermore, it is essential to establish standardized protocols for reporting test results and managing historical data.

By presenting these approaches, we aim to promote dialogue and alignment among laboratory NHP facilities in Korea, thereby facilitating collaborative research and improving the reproducibility of NHP studies.

Key words : Non-human primates (NHPs), Microbiological quality control, Zoonosis, Research reproducibility

Application of Non-Human Primates in Advanced Biopharmaceuticals Development

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Gene and antibody therapy represent two of the most groundbreaking advancements in drug development. Gene therapy aims to modify or replace defective genes to treat diseases at their root cause, while antibody therapy leverages the immune system to precisely target disease markers.

During the development of these therapies, nonclinical studies using non-human primates (NHPs) are required under FDA guidelines to ensure safety and efficacy before progressing to human trials. NHP toxicology studies help evaluate potential risks, drug metabolism, and immune responses in a biological model that closely mirrors human physiology.

For drugs targeting central nervous system (CNS) diseases, effective delivery to the brain is crucial. Intrathecal (IT) injection, which administers the drug directly into the spinal cord, is the most viable method for bypassing the blood-brain barrier. However, because IT injection is an invasive procedure affecting the nervous system, neurological and physical examinations are essential to verify injection accuracy and assess potential side effects.

In conclusion, NHPs are a precise animal model for assessing safety and efficacy during drug development, particularly for CNS-targeted therapies.

Key words : Non-human primates, Non-clinical study, Intrathecal injection

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SYMPOSIUM 6 [KLAT Education 1]

KOR

July 23 (Wed) 16:00–17:40 | Samda Hall

Microbiological Quality Control for Laboratory Rodents

Organizer : Dong-Jae Kim (DGIST) / Chair : Seung Hyeok Seok (Seoul Natl. Univ.)

Major Rodent Diseases in Korean Laboratory Animal Facilities

Joon-Suk Park (K-MEDI hub)

Disease Control Strategies for Laboratory Rodents:
Quarantine, Disinfection, and Sterilization

Jungmin Lee (Samsung Medical Center)

Health Monitoring Techniques for Laboratory Rodent

Hwan Kim (IBS)

Cleaning Methods for Infected Rodents and Applicable Cases

Yonghun Sung (Korea Polytechnic)



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S6-1

Major Rodent Diseases in Korean Laboratory Animal Facilities

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July 23 (Wed)

Rodent diseases present significant challenges in Korean laboratory animal facilities, impacting both animal welfare and the validity of scientific research. Health screening of rodents used in research is necessary due to the consequences of unwanted infections. This presentation summarizes the major infectious and non-infectious diseases affecting laboratory rodents in Korea, with a focus on prevalent pathogens, transmission routes, clinical signs, and control measures. One determinant of the risk of infection for any given agent is its prevalence. Common diseases include mouse norovirus, mouse hepatitis virus, pinworm infections, and bacterial agents such as *Mycoplasma pulmonis*. These diseases can compromise experimental outcomes and pose biosecurity risks. Regular health monitoring, biosecurity protocols, sanitation, and quarantine procedures are essential for disease prevention and management. This presentation provides crucial data to support effective health management strategies in Korean laboratory animal facilities and emphasizes the need for continued surveillance and research to mitigate disease outbreaks.

Key words : Mice, Rats, Rodent diseases, Prevalence

Disease Control Strategies for Laboratory Rodents: Quarantine, Disinfection, and Sterilization

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To conduct reproducible and reliable animal experiments, researchers must use genetically controlled laboratory animals and ensure that infection control measures are strictly enforced. Infection refers to the state in which pathogenic organisms such as bacteria, rickettsia, viruses, fungi, and protozoa attach to or invade the tissues of a host, such as humans or animals, and undergo growth and proliferation. Asymptomatic infection refers to cases where infection occurs, but no symptoms appear, while infection that manifests as a disease is called an infectious disease. Asymptomatic infections can develop into infectious diseases due to various stresses (such as transportation) and stimuli experienced by laboratory animals. Therefore, it is necessary to quarantine newly introduced animals until their health status and microbiological condition can be assessed. However, for rodents, if the transport is conducted by a facility approved by the relevant authority and the transport process excludes exposure to pathogens, quarantine may not be necessary. Regardless of quarantine status, an acclimatization period is required for imported animals to adapt physiologically, behaviorally, and nutritionally to the new environment. An effective quarantine program minimizes the risk of pathogens being introduced into existing animal populations.

In addition, it includes proper disinfection and sterilization to prevent infection and maintain environmental conditions for animal welfare.

Key words : Disease control, Infection, Quarantine, Disinfection, Sterilization

Health Monitoring Techniques for Laboratory Rodent

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Regular health monitoring of SPF (Specific Pathogen-Free) rodents, such as mice and rats, is essential for guaranteeing the microbiological quality of laboratory animals, which is an important aspect in maintaining the reproducibility and reliability of animal studies. Standardized techniques for health monitoring of laboratory rodents are composed of serological assays, culture-based methods, microscopic examination, and molecular diagnostics. Serological tests, which are primarily conducted on sentinel animals, use ELISA and IFA procedures to detect antibodies against viral or bacterial infections. Culture methods are used to isolate and identify bacteria based on biochemical features. Microscopic examination is to diagnose parasitic infections using morphological characteristic of adult and egg. PCR-based molecular diagnostics, which are more sensitive and specific, have been utilized recently to supplement conventional techniques. These health monitoring techniques are carried out on a regular and systematic basis, in accordance with Ministry of Food and Drug Safety (MFDS) principles and international guidelines such as FELASA, and play an important role in disease prevention and hygiene management in SPF animal facilities.

Key words : SPF rodents, Health monitoring, Serology, Culture, Microscopic test, PCR

Cleaning Methods for Infected Rodents and Applicable Cases

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Research utilizing various experimental animals is actively conducted across all fields related to biotechnology and medical products. In particular, studies involving rodents, such as mice and rats, have become essential for in vivo functional analysis and efficacy evaluation. Recently, to enhance the accuracy of research results using these experimental animals, breeding methods, environmental conditions, and the quality of experimental animals are strictly controlled to maintain and precisely manage breeding facilities. To raise experimental animals in a clean environment, only those that meet specific standards can be introduced and used in experiments following a microbial monitoring process that screens for contaminated microorganisms in vivo. Conducting research in facilities with purified experimental animals requires a thorough process to cleanse contaminated subjects. The purification process for contaminated animals is broadly divided into acclimation, superovulation, embryo collection, culture, and embryo transfer. First, animals undergo an acclimation period to ensure their stability and good condition. Female animals are then administered ovulation-inducing hormones to optimize the timing of ovulation and obtain as many fertilized embryos as possible. The fertilized embryos are collected from mated females, cultured for one day, and observed for their developmental progress. Properly developed embryos are selected and transferred into surrogate mothers raised in clean facilities. After birth, the condition of the offspring is carefully observed, and microbial monitoring is conducted to ensure the experimental animals are free from contamination. By securing clean experimental animals for research, the accuracy of results can be improved, ultimately leading to more reliable experimental outcomes.

Key words : Contaminated animal, Cleaning, Superovulation, Embryo collection, Embryo transfer

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SYMPOSIUM 7 ENG

July 24 (Thu) 09:40–11:20 | Halla Hall A

Stem Cells and Tissue Regeneration

Organizer : Jinwook Choi (GIST) / Chair : Jinwook Choi (GIST), Hye-Jin Boo (Jeju Natl. Univ.)

Rare Begets Common: Skin Diseases Reveal Basic Human Biology

Ting Chen (NIBS, Beijing)

Aging and Rejuvenation of Skin Stem Cells and Their Niches

Sekyu Choi (POSTECH)

Organoid Modelling of Human Lung Development and
Cell Fate Alteration in Human Congenital Pulmonary Diseases

Kyungtae Lim (Korea Univ.)

The Specification and Diversification of Enteroendocrine Cells
from Intestinal Stem Cells

Rongwen Xi (NIBS, Beijing)



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Rare Begets Common: Skin Diseases Reveal Basic Human Biology

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The skin, a multilayered epithelial organ, serves as the body's primary defense against environmental insults, pathogens, and mechanical stress. Despite its critical role, therapeutic advancements for skin disorders remain hindered by gaps in understanding human-relevant biology. Rare genetic skin diseases can reveal conserved pathways that bridge rare and common conditions. By studying these disorders, we uncover fundamental mechanisms—such as stress responses, epithelial regeneration, and immune regulation—that inform therapeutic strategies applicable to both rare and prevalent diseases. This approach transcends limitations of model organisms, enabling insights into human-relevant processes and accelerating translational innovations, from gene therapies to targeted interventions. This integrative approach underscores the value of rare disorders as windows into basic biological principles, driving transformative advancements in dermatology and epithelial biology.

Key words : The skin, Rare genetic skin diseases, Gene therapies, Targeted interventions

Aging and Rejuvenation of Skin Stem Cells and Their Niches

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Throughout adult life, mammalian hair follicles, which are linked to sebaceous glands, go through periodic phases of rest, regeneration, and degeneration. Regulating these hair cycles is a fundamental role of hair follicle stem cells. The activity of melanocyte stem cells, which develop into melanin-secreting melanocytes and contribute to the color of both skin and hair, is another significant aspect. By replacing mature sebocytes, sebaceous gland stem cells also play a key role in sustaining the epidermal barrier. These stem cells are maintained in a specialized microenvironment or niche and are regulated by internal and external signals, determining their dynamic behaviors in homeostasis and hair follicle regeneration. The niches surrounding the hair follicles release a variety of factors that regulate the activity of these skin stem cells. Several environmental factors, including the process of aging and underlying metabolic conditions, can affect skin stem cell activity. Here, I will discuss skin aging and the potential rejuvenation strategy for skin stem cell activity. These viewpoints would provide new insights into the disease mechanisms and treatment of skin and hair disorders.

Key words : Skin aging, Epithelial stem cells, Skin stem cells, Regeneration, Rejuvenation

July 24 (Thu)

Organoid Modelling of Human Lung Development and Cell Fate Alteration in Human Congenital Pulmonary Diseases

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In the developing human lung, alveolar development requires highly systemic, spatiotemporal interactions between subsets of differentiating progenitor cells of epithelial and mesenchymal lineages. In contrast, a congenital pulmonary airway malformation (CPAM) is a rare developmental disorder of fetal lungs during pregnancy around 20 weeks of gestation, which normal alveoli are replaced by atypical airway-like cyst structures. After birth, the CPAM lungs contain one or multiple lesions, which can be either solid or fluid-filled, occasionally with stacked immune cells. However, how the fetal pathogenic niche of CPAM lesions is formed and develops the airway-like epithelial cysts and surrounding mesenchyme are poorly understood. To understand the details of CPAM pathology, we study normal human lung development by generating multiomic cell atlas of human lung development that combines single-cell RNA and ATAC sequencing, high-throughput spatial transcriptomics, and single-cell imaging. Then, we characterised the progression of the pathogenic niche in and around the CPAM lesions by performing single-cell sequencing and spatial imaging, from multiple patients (n=10). Spatial analysis in the CPAM lungs shows that the CPAM niche containing heterogeneous, airway-lineage cells progressively affect cell fate alteration of alveolar cells to airway-like cells that are hardly observed in the normal lung development. Notably, mixed cell states – transitioning alveolar to airway-like cells was frequently observed at the border between normal alveoli and the developing CPAM lesions and at the alveolar regions located among multiple cysts. Our study provides an integrated view of the niche environment as well as cellular heterogeneity within the distal lungs affected by the progression of CPAM.

Key words : Human lung development, Congenital pulmonary disease, Cell fate differentiation, Single cell atlas, Human lung organoid

The Specification and Diversification of Enteroendocrine Cells from Intestinal Stem Cells

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Enteroendocrine cells (EEs) in both vertebrates and invertebrates are derived from intestinal stem cells (ISCs) and are scattered along the length of the digestive tract. EEs represent a heterogeneous population of cells and exert important physiological functions by secreting a diverse array of neuropeptide hormones. Despite the fact that many transcription factors (TFs) required for the specification of EE subtypes have been identified, the process during which the EE cell fate is selected and diversified remain poorly understood. We find that, in the ISC lineage in adult *Drosophila* midgut, in which the Delta-Notch-signaling-guided progenitor cell differentiation into enterocyte is the default mode, the specification of EEs is initiated by a clock-like-mechanism-induced transient activation of an EE-fate inducer in ISCs, which induces asymmetric stem cell divisions that generate EE fate-committed progenitors. Single cell transcriptome analysis of the entire EEs followed by genetic screens have allowed us to classify *Drosophila* EEs into 10 EE subtypes and assign the TF code to each EE subtype. By further analyzing the EE-specific TFs, we find that a combination of a master TF, a secondary-level TF and a tertiary-level TF constitute a “TF code” for generating EE cell diversity. Notably, the differentiated EEs are highly plastic, as changes in the TF code—including as few as one distinct TF—allow efficient switching of subtype identities. Lastly, we show that WNT and BMP intercalating gradients that emanate from the compartment boundaries play an important role in shaping regional ISC identity and consequently regional EE diversity along the length of the midgut. Taken together, our study elucidates the molecular process during which EEs are generated and diversified from ISCs and reveals a hierarchically-organized three-part code that underlies EE diversity and plasticity in *Drosophila*, which can guide investigations of EEs in mammals and inform their application in medicine.

Key words : Enteroendocrine cells, Intestinal stem cells, Transcription factor code, Cell plasticity

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SYMPOSIUM 8 [KIRAMS] KOR

July 24 (Thu) 09:40–11:20 | Halla Hall B

Molecular Imaging Techniques for Evaluation of Drug Development and Disease Animal Models

Organizer : Jin-Hee Seo (KIRAMS) / Chair : Yong Jin Lee (KIRAMS)

Radiopharmaceuticals for Noninvasive Theranostics Imaging

Yun-Sang Lee (Seoul Natl. Univ.)

MR Molecular Imaging for Drug Development and
Disease Diagnostics Using Animal Models

Yongmin Chang (Kyungpook Natl. Univ.)

Preclinical Evaluation and Imaging of Radiopharmaceuticals Using PET

Se Jong Oh (KIRAMS)

Pharmacokinetic Study Using C-14 in New Drug Development

Mihye Kwon (KIRAMS)



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S8-1

Radiopharmaceuticals for Noninvasive Theranostics Imaging

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Radioisotopes have emerged as powerful tools in drug development and evaluation, offering unique advantages in tracing, imaging, and therapeutic applications. This approach enables real-time monitoring of pharmacokinetics, biodistribution, and target engagement with high sensitivity and specificity. In this study, we explore the integration of radiolabeling techniques in the development of novel drug candidates, focusing on both diagnostic and therapeutic isotopes. Preclinical evaluations were conducted using positron emission tomography (PET) and single-photon emission computed tomography (SPECT) imaging to assess in vivo behavior, while therapeutic efficacy was tested with alpha- and beta-emitting isotopes. Our findings demonstrate that radioisotope-based strategies facilitate early decision-making in drug development pipelines by providing quantitative insights into drug performance and biological interactions. This approach holds promise for accelerating translational research and improving precision in personalized medicine.

Key words : Radiopharmaceutical, Theranostics, Positron emission tomography, Radionanotheranostics

July 24 (Thu)

S8-2

MR Molecular Imaging for Drug Development and Disease Diagnostics Using Animal Models

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This lecture aims to explore the applications of MRI molecular imaging techniques in the diagnosis and drug development of various diseases using animal disease models. MR molecular imaging is a technology that implements molecular-level imaging of diseases by leveraging the excellent resolution and soft tissue contrast of MRI compared to other imaging techniques. Particularly in animal models, very high magnetic field MRI imaging is possible, enabling the acquisition of ultra-high-resolution anatomical and molecular images. This lecture will introduce molecular contrast agents and magnetic resonance spectroscopy techniques, which are essential for implementing such molecular imaging. For molecular contrast agents, we will examine application examples of both small molecule and nanoparticle contrast agents.

Key words : MRI, Molecular imaging, Animal model

Preclinical Evaluation and Imaging of Radiopharmaceuticals Using PET

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Preclinical evaluation of novel radiopharmaceuticals is essential for establishing safety, biodistribution, and diagnostic potential prior to clinical application. Positron emission tomography (PET) enables noninvasive, quantitative assessment of tracer pharmacokinetics and organ-specific uptake in vivo, supporting comprehensive early-stage evaluation.

PET-based dosimetry involves calculating time–activity curves from serial imaging and biodistribution data, allowing estimation of absorbed radiation doses in critical organs. This provides a scientific basis for predicting human radiation exposure and assessing safety margins.

For PET tracers targeting Alzheimer’s disease, brain uptake patterns, regional specificity, and potential toxicity can be assessed through quantitative imaging. Correlation with behavioral outcomes in transgenic animal models enables evaluation of diagnostic efficacy and target engagement. Moreover, PET imaging serves as a powerful modality in the investigation of neurodegenerative and psychiatric disorders such as Alzheimer’s disease, Parkinson’s disease, and depression. These studies demonstrate the capacity of PET to visualize disease-specific molecular alterations and monitor pathological progression over time. Integration with behavioral and histological data enhances the interpretation of imaging findings and supports validation of tracer utility.

Such an imaging-based framework allows for simultaneous evaluation of safety and functional performance, offering translational insight into radiopharmaceutical development. In particular, the early detection of disease-related molecular changes and the assessment of diagnostic value in animal models position PET as a key component in preclinical research pipelines.

Key words : Radiopharmaceuticals, PET Imaging, Dosimetry, Neurodegenerative disorders

Pharmacokinetic Study Using C-14 in New Drug Development

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Aim of this study is the application of C-14 radiolabeled compounds in nonclinical pharmacokinetic (PK) evaluation. Pharmacokinetics (PK) is the study of the absorption, distribution, metabolism, and excretion (ADME) of a drug over time using mathematical modeling. Understanding PK is crucial, especially in disease models or co-administration, where alterations in drug-metabolizing enzymes (e.g., CYP, UGT) may affect drug efficacy and toxicity. In this study, radiolabeled compounds with C-14 isotopes were employed to precisely trace and quantify drug behavior in vivo. C-14 labeled compounds (including microtracing) analysis included bioavailability assessment using LC-MS/MS and accelerator mass spectrometry (AMS), tissue distribution via quantitative whole-body autoradiography (QWBA), beta counting (LSC), and metabolite profiling using LC-QTOF and AMS. Representative case studies involved bioavailability evaluation of [14C]-acetaminophen via oral and intravenous routes, tissue distribution and excretion profiling of [14C]-2PE, and the in vivo stability and bio-distribution of a [14C]-labeled mRNA delivery system. These findings highlight that C-14 radiolabeled PK studies provide a powerful approach for evaluating trace-level compounds, large molecules, mRNA vaccines, and other complex analytes.

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SYMPOSIUM 9 [KCLAM] KOR

July 24 (Thu) 09:40–11:20 | Samda Hall

Optimizing Post-Approval Monitoring: Navigating the Roles of
IACUC, AV, LAT, and Researchers

Organizer : Jun Won Yun (Seoul Natl. Univ.) / Chair : Byeong-Cheol Kang (Seoul Natl. Univ.)

Enhanced PAM: Experience and Implementation at HKUMED's
AAALAC International Accredited Program

Connie Leung (The Univ. of Hong Kong)

AV and LAT Roles in Post-Approval Monitoring (PAM)

Ji-Yeon Hwang (Seoul Natl. Univ. Bundang Hospital)

Program Review and PAM: Similar Goals, Different Approaches

Young-Shin Joo (The Catholic Univ.)



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Enhanced PAM: Experience and Implementation at HKUMED's AAALAC International Accredited Program

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This presentation focuses on the enhanced Program of Animal Monitoring (PAM) implemented at Centre for Comparative Medicine Research (CCMR), an AAALAC International Accredited program. Established in 1979, the Centre has long been at the forefront of laboratory animal medicine in the Asia-Pacific region, being the largest and most advanced research animal facility in the Hong Kong. The Centre was also one of the first in the PR China to achieve AAALAC International accreditation in 2005 and is dedicated to providing high-quality research care and use program, state-of-the-art facilities, and professional services and training to support the University of Hong Kong's research and teaching missions.

The presentation will delve into the Post-Approval Monitoring (PAM) process, which involves collaboration between researchers, animal caretakers, technicians, managers, veterinarians, and the Institutional Animal Care and Use Committee (IACUC). Following IACUC approval, a pre-commencement meeting is conducted with researchers and managers to outline monitoring procedures. Utilizing a detailed rack card summary and a series of comprehensive cage cards, animals are monitored daily, with any signs of sickness or specific needs recorded at the cage level for easy reference. Information and animal pictures are also captured on Teams and OneNote software for efficient access and follow-up. The implementation of humane endpoint scoring aims to enhance animal welfare and prevent unnecessary suffering. An electronic form issued by veterinary team allows for immediate culling within 30 minutes if humane endpoints are reached, further safeguarding animal welfare. This collaborative approach between researchers and animal care personnel not only ensures compliance with ethical guidelines but also advances scientific research in a responsible and humane manner. Our work highlights the benefits that comparative medicine research performed to the highest welfare standards brings to human and animal health.

Key words : Post-approval monitoring, Animal welfare

AV and LAT Roles in Post-Approval Monitoring (PAM)

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Post-Approval Monitoring (PAM) is a critical component of animal research oversight, ensuring continued compliance with approved protocols and upholding animal welfare standards throughout the course of a study. While Institutional Animal Care and Use Committees (IACUCs) play a central role in establishing PAM programs, their success relies heavily on the active involvement of the Attending Veterinarian (AV) and Laboratory Animal Technicians (LATs), who serve as the eyes and ears of the program in daily operations.

This presentation highlights the complementary roles of AVs and LATs in implementing effective PAM. The AV provides expert evaluation of animal welfare, reviews procedures for adherence to protocols, and advises on refinement opportunities. As a key liaison between researchers and the IACUC, the AV also facilitates communication and education to support ethical and scientifically sound practices. LATs, on the other hand, are positioned at the frontline of animal care and play a vital role in observing animal conditions, identifying protocol deviations, and reporting concerns in real time. Their close and consistent contact with research animals enables early detection of welfare issues that may otherwise go unnoticed.

The collaboration between AVs and LATs strengthens the PAM process by combining clinical expertise with detailed observational insights. This synergy allows for prompt identification of problems, proactive intervention, and continuous feedback to both researchers and oversight bodies. The presentation will also discuss common challenges faced by AVs and LATs in PAM, such as role ambiguity, communication gaps, and limited resources, and propose practical strategies for enhancing their engagement and effectiveness.

Ultimately, recognizing and formalizing the roles of AVs and LATs within institutional PAM frameworks is essential to ensuring compliance, advancing refinement, and promoting a culture of care in animal research.

Key words : Post-approval monitoring, Attending veterinarian, Laboratory animal technician, Animal welfare, Protocol compliance

Program Review and PAM: Similar Goals, Different Approaches

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The Institutional Animal Care and Use Committee (IACUC) is responsible for overseeing animal research programs to ensure ethical standards and regulatory compliance. Two core continuing oversight activities, Program Review and facility inspection and Post-Approval Monitoring (PAM) share the common objective of promoting animal welfare, yet they differ significantly in scope and methodology.

Program review and facility inspection are mandated by national and international regulations, including the Guide for the Care and Use of Laboratory Animals and the Korean Animal Protection Act and Laboratory Animal Act. Conducted at least twice a year, this process evaluates institutional policies, veterinary care systems, training programs, and physical facilities and so on. Facility inspection involves a direct assessment of animal housing areas, procedure rooms, and support spaces to ensure the environment meets established standards.

On the other hand, PAM is an ongoing oversight mechanism that focuses on the conduct of animal research at the protocol level. PAM involves direct observation, interviews, and review of laboratory practices to confirm that procedures align with approved protocols and standard operating procedures.

While program review emphasizes institutional infrastructure, PAM centers on protocol implementation. Together, they form a complementary system that enhances transparency, consistency, and accountability in animal research.

Understanding the distinctions and interactions between these two processes is especially important for newly established committees or new IACUC members. This presentation highlights key differences and offers practical insights for integrating both approaches to strengthen institutional animal care and use programs.

Key words : IACUC, Continuing IACUC oversight, Program review, Facility inspection, Postapproval monitoring

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PLENARY LECTURE 1

ENG

July 24 (Thu) 11:40–12:30 | Halla Hall A, B

Organizer / Chair : Ki Taek Nam (Yonsei Univ.)

Hippo–YAP/TAZ Signaling: A Crucial Pathway in Cell Fate Dynamics

Dae–Sik Lim (KAIST)



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Hippo-YAP/TAZ Signaling: A Crucial Pathway in Cell Fate Dynamics

Dae-Sik Lim

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The Hippo-YAP/TAZ signaling pathway is a critical developmental program that controls adult stem/progenitor cell proliferation, organ size, and regeneration. The core complex of this pathway comprises the protein kinases MST1 and -2 (MST1/2), WW45 (Sav1), MOB1a/b, and LATS1/2. YAP and TAZ are the downstream effectors of the Hippo signaling pathway, and their activity is determined by phosphorylation status. The upstream kinases LATS1/2 directly phosphorylate YAP/TAZ, resulting in their cytoplasmic localization and degradation. We and others revealed that YAP/TAZ activation leads to hyperproliferation of epithelial precursor cells, differentiation inhibition, surrounding cell proliferation, and cancer development. However, the role of this pathway in regulating cell fate changes or cell plasticity has not been fully characterized *in vivo*. We recently showed that YAP hyperactivation in the liver forces hepatoblasts or hepatocytes to commit to the biliary epithelial cell (BEC) lineage. It also increases BEC and fibroblast proliferation but suppresses hepatoblast to hepatocyte differentiation. In addition, YAP activation in ductal cells of the liver and pancreas led to the rapid development of Cholangiocarcinoma (CC) and Pancreatic Ductal Adenocarcinoma (PDAC). We also found that activation of YAP/TAZ in other organs induces dedifferentiation and/or transdifferentiation, rather than hyperproliferation, of precursor cells. Here, I will discuss how Hippo-YAP/TAZ signaling affects cell fate dynamics of stem/progenitor cells in the liver and lung and how YAP confers "cell plasticity" to certain types of differentiated cells.

Key words : Hippo-YAP/TAZ signaling, Tissue homeostasis, Cell plasticity, Cell fate dynamics, Stem/progenitor cells

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Luncheon Seminar 1~3

July 24 (Thu) 12:40–13:00

Luncheon Seminar 1

Halla Hall A

Chair : Jeong-Hwan Che (Seoul Natl. Univ.)

Intravital Confocal / Two-Photon Microscopy: In Vivo Cellular-Level Imaging of Live Animals Model

Sowon Lee (IVIM Technology)

Luncheon Seminar 2

Halla Hall B

Chair : Keunwook Lee (Hallym Univ.)

Research Ethics and Security in the Age of AI

Byung chun Yoo (ITstandard)

Luncheon Seminar 3

Samda Hall

Chair : Dae-Yong Kim (Seoul Natl. Univ.)

Are Laboratory Animals Still Essential? - The Scientific, Ethical, and Industrial

Jae-Jin Chang (Orient Bio Inc.)



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Intravital Confocal / Two-Photon Microscopy: In Vivo Cellular-Level Imaging of Live Animals Model

Sowon Lee

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Intravital microscopy (IVM) enables high-resolution, real-time imaging of cellular events within living organisms, offering valuable insights into complex biological processes in their native microenvironments. As research demands for dynamic, in vivo analysis continue to rise across fields like neuroscience, oncology, and immunology, advanced imaging platforms are critical.

This presentation introduces the fundamentals of intravital confocal and two-photon microscopy and highlights key strengths of our proprietary IVM system. Our microscope is optimized for deep tissue penetration, long-term imaging stability, and high spatiotemporal resolution, making it suitable for a wide range of preclinical applications.

We will share representative datasets acquired using our system, demonstrating in vivo imaging of immune cell dynamics, drug delivery, and disease-related cellular behavior. Both acute and long-term imaging approaches will be discussed, including strategies for minimizing motion artifacts and maintaining imaging quality over extended periods.

In addition, we will explore the therapeutic applications of intravital imaging, such as visualizing drug distribution, and evaluating treatment efficacy at the cellular level.

In conclusion, we emphasize the versatility and scalability of our IVM platform, which supports imaging of diverse organs and is adaptable for both small and large animal models. These capabilities position our system as a powerful tool for advancing in vivo research and facilitating translational breakthroughs.

Key words : Intravital microscopy, In vivo imaging, Cellular dynamics, Preclinical research

Research Ethics and Security in the Age of AI

Byung chun Yoo

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Many institutions now use online review systems to ensure transparent and prompt evaluations by the Institutional Animal Care and Use Committee (IACUC). Science fiction books and films often depict visions of the future, and with the advancement of AI technology, AI systems such as chatbots have emerged as new security threats. Chatbots equipped with AI systems can manipulate human perception through relational processes. Therefore, it is essential to have strict guidelines in place to recognize and respond appropriately when such systems make unusual or unexpected requests. Generative AI, powered by Large Language Models (LLMs), operates through mechanisms fundamentally different from traditional programs. Instead of producing outputs based on predefined rules written in programming languages, they generate results based on vast datasets they have learned from. Resnik's principles of research ethics also emphasize the importance of respecting intellectual property. This includes respecting patents, copyrights, and other intellectual property rights, and being particularly cautious when using unpublished data, methods, or results without permission. Such caution is especially necessary when working with AI. Rather than using AI recklessly, it is advisable to treat it as a supplementary tool that supports human research, not one that replaces it.

July 24 (Thu)

Are Laboratory Animals Still Essential? – The Scientific, Ethical, and Industrial Nexus Sustaining Progress in Life Sciences

Jae-Jin Chang

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Despite the remarkable advances in biotechnology, laboratory animals continue to play a crucial role as a foundational element in life sciences and the biomedical industry.

This presentation explores the irreplaceable value of laboratory animals at this point in time, focusing on their scientific, ethical, and economic significance.

Laboratory animals are not merely tools for research; they serve as sophisticated platforms that enhance our understanding of biological phenomena and improve the predictability of new drug development.

At the same time, the growing emphasis on animal welfare and responsible ethics directly contributes to scientific reproducibility and data reliability.

This presentation will briefly introduce ORIENT BIO and ORIENT GENIA, two of Korea's key infrastructures in the laboratory animal industry, and explain how a testing system that meets global standards for quality, ethics, and regulatory compliance bridges industry and research.

In conclusion, laboratory animals are not an "optional tool" in life sciences, but rather a "fundamental prerequisite."

An integrated approach encompassing ethics, quality, and industrial infrastructure is essential for the continued advancement of life science.

Key words : Laboratory animals, Animal welfare, Biomedical research, Ethics and responsibility

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ACADEMY AWARD PRESENTATION

July 24 (Thu) 14:00–14:40 | Halla Hall A

Chair : Yang-Kyu Choi (Konkuk Univ.)

FXR as a Bridge Between Metabolic Dysfunction and Oncogenesis

Sungsoon Fang (Yonsei University)



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FXR as a Bridge Between Metabolic Dysfunction and Oncogenesis

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Farnesoid X receptor (FXR), a bile acid-activated nuclear receptor, has emerged as a master regulator of metabolic and immune homeostasis in the liver and intestine. In the context of metabolic dysfunction-associated steatotic liver disease (MASLD), FXR signaling modulates key processes such as hepatic lipogenesis, gluconeogenesis, bile acid synthesis, and inflammatory gene expression. FXR activation suppresses the transcription of lipogenic genes via SHP and regulates bile acid synthesis through feedback inhibition of CYP7A1, collectively reducing hepatic steatosis and improving insulin sensitivity. Dysregulation of FXR signaling—whether through genetic, dietary, or microbial influences—exacerbates MASLD progression, highlighting FXR as a compelling therapeutic target.

Beyond liver disease, FXR also plays a critical role in colorectal cancer (CRC). In the intestinal epithelium, FXR maintains barrier integrity and modulates epithelial turnover and immune responses. Loss of FXR expression is frequently observed in CRC and correlates with increased tumor proliferation, inflammation, and susceptibility to microbial dysbiosis. Mechanistically, impaired FXR signaling alters colonocyte metabolism by promoting glycolysis and mitochondrial dysfunction, creating a tumor-permissive microenvironment. Moreover, FXR deficiency leads to enhanced Wnt/ β -catenin signaling, further driving colorectal tumorigenesis.

Taken together, FXR signaling serves as a central axis connecting metabolic regulation and tumor suppression across the liver–gut axis. Therapeutic activation of FXR not only offers metabolic benefits in MASLD but may also suppress colorectal carcinogenesis by restoring epithelial integrity, reprogramming cellular metabolism, and counteracting oncogenic signaling. These insights position FXR as a promising dual-purpose target in metabolic and cancer therapy.

Key words : Bile acids, Nuclear receptors, Metabolic syndromes, Cancer metabolism

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SYMPOSIUM 10 ENG

July 24 (Thu) 14:50–16:30 | Halla Hall A

In Vivo Explorations of Vascular Function and Dysfunction

Organizer / Chair : Jae-Hoon Choi (Hanyang Univ.)

Mechanism and Potential Therapies for Brain Arteriovenous Malformation

Suk P. Oh (Barrow Neurological Institute)

Dissecting Cellular Dynamics through Genetic Ablation and Labeling of Vascular Cells

Junyeop Lee (Asan Medical Center)

Animal Model and Its Application in Vascular Biology: Endothelial Migration and Differentiation

Heon-Woo Lee (Kyung Hee Univ.)

Immune-Vascular Crosstalk within the Tumor Microenvironment

Keehoon Jung (Seoul Natl. Univ.)



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Mechanism and Potential Therapies for Brain Arteriovenous Malformation

Suk P. Oh

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Brain arteriovenous malformations (bAVMs) are characterized by abnormal blood flow, where blood is shunted directly from feeding arteries to arterialized veins without the presence of capillaries. Spontaneous ruptures of bAVMs account for about 25% of all hemorrhagic strokes in adults under the age of 50, often leading to significant morbidity and mortality. bAVMs are classified into two types: familial and sporadic. Familial bAVMs are primarily linked to hereditary hemorrhagic telangiectasia (HHT), a genetic vascular disorder caused by mutations in BMP signaling genes such as ENG, ALK1, and SMAD4. However, the majority of bAVMs (about 95%) are sporadic, with no clear inheritance pattern. Recent research has identified somatic mosaic mutations in the RAS signaling pathway within endothelial cells of sporadic bAVM lesions, with KRAS mutations present in over 50% of sporadic cases. Current bAVM management involves a multidisciplinary approach, including microsurgical resection, endovascular embolization, and radiation therapy. Unfortunately, there are no pharmacological treatments available to halt progression or induce regression of established bAVMs, making the current invasive treatments risky and potentially hemorrhagic. This underscores the urgent need for alternative pharmacological options. Our laboratory has developed longitudinal mouse models for bAVMs using magnetic resonance angiography and preclinical ultrasound imaging technologies. I will present data from both familial and sporadic bAVM mouse models, highlighting ongoing research into disease mechanisms and exploring novel therapeutic approaches for bAVMs.

Key words : Hereditary hemorrhagic telangiectasia, Arteriovenous malformation, Magnetic resonance angiography, Brain, KRAS

Dissecting Cellular Dynamics through Genetic Ablation and Labeling of Vascular Cells

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All cells in the body have a defined lifespan, and perivascular cells, which envelop endothelial cells and stabilize blood vessels, are no exception. In this study, we investigated the turnover and replacement dynamics of perivascular cells under both physiological and pathological conditions. Using intravital imaging of PDGFR β -creERT2;tdTomato and inducible ROSA26-DTA mice, we tracked the fate of retinal pericytes over time. We found that PDGFR β ⁺ pericytes gradually declined, with a markedly accelerated loss observed in diabetic conditions—approximately 50% were lost within one month following diabetes induction. Interestingly, PDGFR β ⁻/TAGLN⁺ cells persisted and repopulated areas where pericytes were depleted, potentially mitigating the progression of diabetic retinopathy. Tamoxifen-induced expression of DTA effectively ablated PDGFR β ⁺ and Myh11⁺ smooth muscle cells in retinal vasculature. This presentation will highlight approaches to evaluate vascular cell dynamics using genetic labeling and ablation models. In conclusion, perivascular cells are actively replaced even under normal conditions and are rapidly lost in diabetes. However, compensatory recruitment of alternative mural cell populations plays a critical role in preserving vascular integrity. We propose that promoting perivascular recovery may offer a novel therapeutic strategy for preventing or treating vascular diseases.

Key words : Pericyte, Dynamics, Genetic ablation, Intravital imaging

July 24 (Thu)

Animal Model and Its Application in Vascular Biology: Endothelial Migration and Differentiation

Heon-Woo Lee

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Angiogenesis is a dynamic process that involves expansion of a pre-existing vascular network that occurs in a number of physiologic and pathologic settings. Despite its importance, the origin of the new vasculature is poorly defined. In particular, the primary subtype of endothelial cells (capillary, venous, arterial) remains undefined. We identified a novel venous endothelial marker gene (Gm5127) and generated inducible venous endothelial-specific Cre and Dre driver mouse lines. Using a comprehensive set of endothelial subtype-specific inducible reporter mice, including tip-, arterial- and venous- endothelial reporter lines, we showed that venous endothelial cells are the primary endothelial subtype responsible for the expansion of an angiogenic vascular network. During physiologic angiogenesis, venous endothelial cells proliferate, migrating against the blood flow, and differentiating into tip, capillary and arterial endothelial cells of the new vasculature. Using intravital 2-photon imaging, we observed venous endothelial cells migrating against the blood flow to form new blood vessels. Venous endothelial cell migration also plays a key role in pathologic angiogenesis. This was observed both in formation of arterio-venous malformations in mice with inducible endothelial-specific Smad4 deletion mice and in pathologic vessel growth seen in oxygen-induced retinopathy. Our studies establish venous endothelial cells are primary endothelial subtype responsible for the normal expansion of vascular network, formation of arterio-venous malformations and pathologic angiogenesis. These observations highlight the central role of the venous endothelium in normal development and disease pathogenesis.

Key words : Angiogenesis, Endothelial migration, scRNAseq, lineage tracing

S10-4

Immune-Vascular Crosstalk within the Tumor Microenvironment

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Our goal is to better understand the immune microenvironment in cancer using advanced methodologies such as in vivo imaging and single-cell genomics. Specifically, we aim to uncover the vast potential of myeloid populations in cancer, an emerging area of study. Real-time intravital multiphoton microscopy allows us to visualize dynamic cell migration and intercellular communication. Through single-cell RNA sequencing in both preclinical and clinical specimens, we reveal the heterogeneity of myeloid cells. This approach provides new opportunities for cancer immunotherapy by identifying promising targets in tumor immunity. Notably, we identified immunosuppressive monocytes and neutrophils as key contributors to tumor resistance against conventional therapies. Furthermore, we elucidated molecular mechanisms underlying treatment resistance, highlighting potential immunomodulatory strategies to enhance long-term clinical outcomes.

Key words : Cancer immunology, Immune cell, Blood vessel, In vivo imaging, Single-cell omics

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SYMPOSIUM 11 [KMPC] KOR

July 24 (Thu) 14:50–16:30 | Halla Hall B

The Next Waves in Signaling Crosstalk From Cellular to Systemic Metabolism

Organizer : Je Kyung Seong (Seoul Natl. Univ.), Sungsoon Fang (Yonsei Univ.)

Chair : Jang-Seong Kim (KRIBB)

Glutamatergic Metabolic Synapse: Hepatocyte–Kupffer Cell Interactions in Liver Disease

Won-Il Jeong (KAIST)

Enhanced Adipose Tissue Reservoir Capacity after Lactation Improves
Insulin Sensitivity and Protects against Type 2 Diabetes

Joon Ho Moon (Seoul Natl. Univ. Bundang Hospital)

Selenium's Paradoxical Role in Ferroptosis Modulation

Namgyu Lee (Dankook Univ.)

Modulating Chronic Inflammation to Lower Cancer Risk:
Mechanisms and Therapeutic Approaches

Jongho Park (Keimyung Univ.)



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Glutamatergic Metabolic Synapse: Hepatocyte-Kupffer Cell Interaction in Liver Disease

Won-Il Jeong

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We have explored from a distinct perspective how Kupffer cell activity contributes to hepatocyte damage in the progression of alcohol-related steatohepatitis (ASH). In an alcohol-fed mouse model, we discovered that hepatocytes, akin to neurons, form glutamate-containing granules through alterations in the glutamate metabolic pathway. These hepatocytes secrete glutamate via calcium-mediated SNARE complex activation, thereby triggering the activity of the glutamate receptor mGluR5 in neighboring Kupffer cells. Specifically, our findings demonstrated that hepatocytes undergo ballooning, a process that brings them into close proximity to Kupffer cells. At this stage, a metabolic synapse forms between the two cell types, allowing the secreted glutamate to selectively stimulate mGluR5 activity in Kupffer cells. The activation of mGluR5 not only induces reactive oxygen species (ROS) production through the activity of NADPH oxidase 2 in Kupffer cells but also upregulates the expression of Cxcl1, a chemokine that attracts neutrophils and exacerbates hepatocyte damage. However, when hepatocytes and Kupffer cells were spatially distant, these effects were absent, indicating that such interactions occur only when hepatocytes establish close contact with Kupffer cells via the metabolic synapse in the liver. This study thus provides novel insights into the mechanisms underlying alcohol-induced hepatocyte damage and inflammatory responses. Through this research, we have not only introduced a new academic paradigm regarding the presence of an independent metabolic synapse in the liver but also highlighted its potential in advancing drug development for the prevention and treatment of alcohol-related liver disease through targeted regulation.

Key words : Glutamate, mGluR5, Alcohol, Liver disease, Steatohepatitis

July 24 (Thu)

Enhanced Adipose Tissue Reservoir Capacity after Lactation Improves Insulin Sensitivity and Protects Against Type 2 Diabetes

Joon Ho Moon

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Pregnancy imposes a substantial metabolic burden on women. Lactation is known to reduce postpartum type 2 diabetes mellitus and we have reported that lactation improves pancreatic β cell mass and function in postpartum women (Science Translational Medicine, 2020), but little is known on the mechanism how lactation can improve insulin sensitivity. We assessed metabolic phenotypes of lactating and previously lactated mice. Lactating and post-lactation mice at 3 and 6 weeks postpartum respectively demonstrated improved glucose tolerance and enhanced insulin sensitivity, despite exhibiting higher levels of adiposity. We treated prolactin in primary preadipocytes and observed a significant induction of proliferation. RNA & ATAC-sequencing analysis of prolactin-treated 3T3-L1 cells revealed a marked increase in the expression of genes associated with adipocyte dedifferentiation and proliferation which involves JAK/STAT5-Src-Wnt pathway. This study proposes that the increased capacity of adipose tissue reservoirs triggered by prolactin might underlie the sustained enhancement of insulin sensitivity observed during lactation.

Key words : Lactation, Adipose, Gestational diabetes, Type 2 diabetes

Selenium's Paradoxical Role in Ferroptosis Modulation

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Selenium is an essential micronutrient renowned for its antioxidant function, primarily through its incorporation as selenocysteine into 25 selenoproteins, including glutathione peroxidase 4 (GPX4), a key inhibitor of ferroptosis. Beyond this canonical, GPX4-dependent role, our previous work uncovered a GPX4-independent mechanism by which selenium suppresses ferroptosis via CoQ10 reduction mediated by sulfide quinone oxidoreductase (SQOR), following selenide processing.

However, selenium's biology is paradoxical. Despite its protective effects at physiological levels, selenium has a narrow therapeutic window and exhibits pronounced toxicity at higher concentrations—a phenomenon observed across humans, livestock, and wildlife. This cytotoxicity has long attracted attention for anticancer applications, yet the underlying mechanisms have remained elusive.

In this presentation, I will highlight selenium's dual faces in ferroptosis regulation and introduce our recent findings that reveal the molecular basis of selenium-induced cell death. These insights not only resolve key aspects of selenium's paradox but also open new avenues for exploiting selenium metabolism in cancer therapy and for developing strategies to counteract selenium poisoning.

Key words : Metabolism, Selenium, Ferroptosis

July 24 (Thu)

Modulating Chronic Inflammation to Lower Cancer Risk: Mechanisms and Therapeutic Approaches

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Hepatitis B virus (HBV) infection is associated with hepatitis and hepatocellular carcinoma (HCC). Considering that most HBV-infected individuals remain asymptomatic, the mechanism linking HBV to hepatitis and HCC remains uncertain. Herein, we demonstrate that HBV alone does not cause liver inflammation or cancer. Instead, HBV alters the chronic inflammation induced by chemical carcinogens to promote liver carcinogenesis. Long-term HBV genome expression in mouse liver increased liver inflammation and cancer propensity caused by a carcinogen, diethylnitrosamine (DEN). HBV plus DEN-activated interleukin-33 (IL-33)/regulatory T cell axis was required for liver carcinogenesis. Pitavastatin, an IL-33 inhibitor, suppressed HBV plus DEN-induced liver cancer. IL-33 was markedly elevated in HBV+ hepatitis patients, and pitavastatin use significantly correlated with reduced risk of hepatitis and its associated HCC in patients. Collectively, our findings reveal that environmental carcinogens are the link between HBV and HCC risk, creating a window of opportunity for cancer prevention in HBV carriers.

Key words : Chronic inflammation, Cancer, IL-33, Regulatory T cell



SYMPOSIUM 12 [KALAS-CALAS] **ENG**

July 24 (Thu) 14:50–16:10 | Samda Hall

Preclinical Studies of Xenografts and Tissue Transplantation

Organizer : **Sun Shin Yi** (Konkuk Univ.)

Chair : **Ik Jin Yun** (Konkuk Univ. Hospital), **Dengke Pan** (ClonOrgan Biotechnology Co., Ltd.)

Homozygous Editing of Multiple Genes for Accelerated Generation
of Xenotransplantation Pigs

Sen Wu (China Agricultural Univ.)

Major Progress in Xenotransplantation Preclinical and Clinical Research in China

Dengke Pan (ClonOrgan Biotechnology Co., Ltd.)

Current Progress in Corneal Xenotransplantation in South Korea

Mee Kum Kim (Seoul Natl. Univ.)

The Clinical Usefulness of Xenogeneic Skin Substitutes

Jun Hur (Hallym Univ. Hangang Sacred Heart Hospital)



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Homozygous Editing of Multiple Genes for Accelerated Generation of Xenotransplantation Pigs

Duan X, Chen C, Du C, Guo L, Liu J, Hou N, Li P, Qi X,
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Although CRISPR-Cas based genome editing has made significant strides over the past decade, achieving simultaneous homozygous gene editing of multiple targets in primary cells remains a significant challenge. In this study, we optimized a co-selection strategy to enhance homozygous gene editing rates in the genomes of primary porcine fetal fibroblasts (PFFs). The strategy utilizes the expression of a surrogate reporter (eGFP) to select for cells with the highest reporter expression, thereby improving editing efficiency. When applied to simultaneous multi-gene editing, we targeted the most challenging site for selection, while other target sites did not require selection. Using this approach, we successfully obtained single-cell PFF clones (3/10) with seven or more homozygously edited genes, including GGTA1, CMAH, B4GALNT2, CD46, CD47, THBD, and GHR. Importantly, cells edited using this strategy were efficiently used for somatic cell nuclear transfer (SCNT) to generate healthy xenotransplantation pigs in less than five months, a process that previously required years of breeding or multiple rounds of SCNT.

Key words : Pigs, Genome editing, CRISPR-Cas, SCNT, Xenotransplantation

Major Progress in Xenotransplantation Preclinical and Clinical Research in China

Dengke Pan^{1*}, Gang Chen², Yi Wang³, Hidetaka Hara³, Dianyuan Li⁴,
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As of May 2025, there are several groups in China exploring various aspects of xenotransplantation preclinical and clinical studies. Clonorgan Biotech Co., Ltd. has cultivated more than 10 kinds of gene editing donor pig populations, carried out more than 70 cases of liver and kidney transplantation from pigs to monkeys. 9 cases of xenotransplantation from pig liver and kidney to brain-dead recipients and patient. Clonorgan's 6 gene editing donor pigs were provided to most of Chinese xenotransplantation groups.

The main preclinical and clinical collaborators are (i) Professor Gang Chen of Tongji Medical College of Huazhong University of Science and Technology in Wuhan, (ii) Professor Yi Wang and Professor of the Second Affiliated Hospital of Hainan Medical University in Haikou, and (iii) Professor Kefeng Dou and Professor Weijun Qin at Xijing Hospital in Xi'an. Professor Gang Chen's group finished 4 gene-modified pig kidney transplantation into rhesus monkey, Out of six monkeys, almost three survived for more than half a year, with a survival rate exceeding 50%. Professor Yi Wang's group carried out the first two pig kidney transplants in brain-dead decedents in China, with follow-up for 12 days. They observed cellular and antibody-mediated rejection, including antibody-dependent cell-mediated rejection. Kefeng Dou's group performed the first pig heterotopic (auxiliary) and orthotopic liver transplant in a decedent in China and documented near-normal hepatic function (with <10% hepatic necrosis) after 10-days' follow-up. Weijun Qin's team carried out the third pig kidney transplants in brain-dead decedents in China, with follow-up for 22 days, and then the first living patient in Aisa and fifth in the world, now survival time is more than 3 months.

Professor Pan has also provided gene-edited pig skin for clinical experiments in living patients with encouraging results, the pig skin grafts surviving as long or longer than human skin grafts.

Key words : Pig, Gene editing, Xenotransplantation, Preclinical study, Clinical study

Current Progress in Corneal Xenotransplantation in South Korea

Mee Kum Kim

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Purpose: To assess 1) the biocompatibility of corneal endothelial cells in genetically engineered (GE) pigs to identify the most suitable candidate for corneal xenotransplantation and 2) the efficacy of full-thickness corneal xenotransplantation from pigs with targeted disruption of four glycan-related genes and iGb3s (QKO), alongside the transgenic expression of two human immune-regulatory genes, CD55 and CD39.

Methods: QKO pigs were evaluated in combination with human (h) CD55, CD39, CD46, and thrombomodulin (TBM) genes. A total of 27 eyeballs obtained from 14 GE pigs were used to assess corneal endothelial cell density (ECD), doubling time (DT), and immunofluorescence staining of corneal endothelial pump channels and tight junctional proteins. Subsequently, full-thickness corneas from either QKO or QKO.hCD55.hCD39 knock-in (KI) pigs older than 6 months were transplanted into six cynomolgus monkeys. Survival rates, rejection indices, central corneal thickness, and immune profiles were evaluated.

Results: The mean ECD loss per week was significantly higher in GE pigs younger than 6 months in GE pigs older than 6 months. The T(Q)KO/hCD46/hTBM cornea exhibited early severe apoptotic changes in endothelial cells, while the other GE corneas did not show such changes. ZO-1, N-cadherin, ATPase, SLC4A1, and aquaporin 1 were well expressed on endothelial cells, regardless of the modified gene types. Longer survival rates were observed in QKO.hCD55.hCD39 knock-in (KI) pig-grafted monkeys compared to those grafted with QKO pigs. Although the concentrations of IFN- γ -producing CD4⁺/CD8⁺ T cell subsets increased up to 6 months, these levels were not higher than those in rejected QKO pig-grafted group, and immune activity significantly declined thereafter. The concentration of donor-specific IgG antibodies also increased during the first six months, but showed individual variability beyond that period.

Conclusion: These findings suggest that the T(Q)KO/hCD55/hCD39 pig cornea demonstrates significantly improved biocompatibility and prolonged graft survival, as a feasible option for corneal xenotransplantation.

Key words : Cornea, Xenotransplantation, QKO.hCD55.hCD39

S12-4

The Clinical Usefulness of Xenogeneic Skin Substitutes

Jun Hur

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Xenogeneic skin substitutes offer promising solutions for treating damaged tissue using materials from different species. Research focuses on minimizing immune rejection and improving integration, making it a significant area in medical science.

Knockout pigs play a crucial role in skin substitute development, utilizing genetic modifications to enhance biocompatibility and increase graft survival rates. This approach is particularly relevant in xenotransplantation and tissue regeneration.

Ongoing studies explore genetic engineering techniques to suppress immune responses and improve skin substitute performance, with applications in burn treatment and skin repair.

Key words : Xenogeneic skin substitutes, Knockout pigs

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SYMPOSIUM 13 ENG

July 25 (Fri) 09:00–10:40 | Halla Hall A

Metabolic Mechanisms in Health and Diseases

Organizer : Su Myung Jung (Sungkyunkwan Univ.) / Chair : Kyoung-Jin Oh (KRIBB)

Phosphatidylethanolamine Content in the Endoplasmic Reticulum
Regulates SREBP-1 Activation and Lipogenesis

Shunxing Rong (UT Southwestern)

Urolithin A: A Gut Metabolite as a Novel Therapeutic
for Cardiac Dysfunction in Heart Failure

Chang-Myung Oh (GIST)

Exercise-Driven Musculoskeletal Alteration and Health

Hyeonwoo Kim (KAIST)

Dynamic Regulation of Anaplerotic Flux in Metabolic Disease

Haejin Yoon (UNIST)



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Phosphatidylethanolamine Content in the Endoplasmic Reticulum Regulates SREBP-1 Activation and Lipogenesis

Shunxing Rong^{1,2}, Mingfeng Xia¹, Goncalo Vale^{1,2}, Shili Li¹,
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Diacylglycerol acyltransferase (DGAT) -2 catalyzes the final step of triglyceride (TG) synthesis. Deletion of DGAT2 reduces liver TG content in mice and inhibitors of DGAT2 are under investigation for the treatment of non-alcoholic fatty liver disease (NAFLD) in humans. Here, we show that in addition to blocking the final step of TG synthesis, DGAT2 inhibition also suppresses SREBP-1 activation, resulting in reduced hepatic fatty acid synthesis and TG secretion. Additional studies reveal DGAT2 inhibition does not alter the cholesterol/sterol or phospholipid polyunsaturated fatty acid contents of the ER. However, phosphatidylethanolamine (PE) levels were significantly increased in the ER of livers of animals treated with a DGAT2 inhibitor. In vitro, enrichment of PE in ER inhibits SREBP-1 activation, suggesting that PE content in the ER acts as a regulator for SREBP-1 activation. Thus, the inhibition of DGAT2 shunted diacylglycerol into phospholipid synthesis, increasing the PE content of the ER, resulting in reduced SREBP-1 cleavage and less hepatic steatosis. Combined, our studies reveal a novel mechanism by which PE level in the ER regulates SREBP-1 activation and fatty acid synthesis.

Key words : SREBPs, PE, Lipogenesis, Hepatic steatosis

Urolithin A: A Gut Metabolite as a Novel Therapeutic for Cardiac Dysfunction in Heart Failure

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Heart failure with preserved ejection fraction (HFpEF) is a clinical syndrome in which patients have signs and symptoms of heart failure due to high left ventricular (LV) filling pressures while having a normal or near normal LV ejection fraction. Urolithin A (UA) is the well-known small metabolite produced by the gut microbiota of pomegranate fruit. Several studies have reported that UA activates mitophagy signaling and has beneficial effects in metabolic diseases. In this study, we sought to investigate the beneficial effects of UA on HFpEF. To establish the mouse model for HFpEF study, BL6J mice were subjected to metabolic and hypertensive stress by high-fat diet and L-NAME. Then, UA (250 mg/kg/d) was administered with a high-fat diet for 12 weeks. We found that UA treatment improved cardiac function and reduced cardiac hypertrophy, pulmonary edema, and fibrosis in the HFpEF model. Further studies are necessary for the elucidation of the underlying mechanisms of UA in HFpEF, both direct and indirect.

Key words : Heart failure, Mitophagy, Urolithin A

Exercise-Driven Musculoskeletal Alteration and Health

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Exercise induces a dynamic biochemical dialogue between skeletal muscle and bone, mediated by secreted factors such as myokines and osteokines. Osteocytes—the most abundant bone cells—are central to this crosstalk. As key mechanosensors, osteocytes detect fluid shear stress generated during physical activity and convert it into signaling cues that regulate bone remodeling through molecules like sclerostin and RANKL. Despite their established role in bone homeostasis, their contribution to systemic exercise responses has remained largely underexplored.

To investigate the osteocyte secretome in response to mechanical load, we exposed MLO-Y4 osteocyte-like cells to fluid shear stress and simulated microgravity. Proteomic profiling revealed seven proteins significantly upregulated by shear stress and twelve downregulated by microgravity. Several overlapping proteins were identified as candidate osteokines with potential endocrine functions. These findings suggest that osteocytes actively participate in exercise-induced inter-organ communication.

Concurrently, we examined irisin, a myokine secreted by skeletal muscle during exercise. Irisin is a 12-kDa polypeptide cleaved from FNDC5, whose expression increases with endurance activity. Irisin circulates at hormone-like levels and exerts effects on bone, adipose tissue, and brain—many of which mirror those induced by exercise. Using quantitative proteomics, we identified the integrin αV family as the primary irisin receptor in osteocytes. Irisin stimulation activated focal adhesion kinase (FAK) signaling in osteocytes, an effect blocked by integrin inhibition.

Together, our findings reveal a bidirectional communication axis between skeletal muscle and bone, mediated by irisin and osteocyte-derived factors. This muscle–bone dialogue is fundamental to exercise physiology and offers novel therapeutic targets to replicate the benefits of physical activity.

Key words : Osteocyte, Irisin, Exercise, Secretome, Skeletal muscle

Dynamic Regulation of Anaplerotic Flux in Metabolic Disease

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Metabolic dysregulation alters energy production and nutrient utilization, playing both adaptive and pathogenic roles in metabolic disease. Prolyl hydroxylase domain-containing protein (PHD) 3 is essential for maintaining cellular metabolic adaptation in cancer by sensing metabolic status and hydroxylating target substrates. However, knowledge regarding the substrate of PHD3 involved in fuel switching in cancer remains limited. Here, we reveal that PHD3 triggers the repression of anaplerosis into the tricarboxylic acid (TCA) cycle through the hydroxylation of pyruvate carboxylase (PC). PHD3 is dual-localized in both the cytosol and mitochondria, where it interacts with PC. Overexpressing PHD3 limits PC activity, consequently impeding cell growth and metastasis while promoting apoptosis. We find that PHD3 expression is strongly increased in subsets of cancer, including clear cell renal cell carcinoma (ccRCC), and is linked to ccRCC progression. In summary, these data identify an unexpected link between PHD3 and PC, and the role of PHD3 in the metabolic dependencies of ccRCC.

Key words : Anaplerosis, Prolyl hydroxylase domain-containing protein 3, Pyruvate carboxylase, Metabolic adaptation

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SYMPOSIUM 14 [KRIBB] ENG

July 25 (Fri) 09:00–10:40 | Halla Hall B

Translational Research Platform Using Animal Models for Inherited Retinal Disease (IRD)

Organizer : Jeong Hun Kim (Seoul Natl. Univ.)

Chair : Jeong Hun Kim (Seoul Natl. Univ.), Ki-Hoan Nam (KRIBB)

Potential Role of Retinal Astrocytes in Retinopathy of Prematurity

Marcus Fruttiger (UCL)

Autonomous Tissue-Sensing Needle Injection System for Ocular Diseases

WonHyung Ryu (Yonsei Univ.)

Novel Bio-Imaging Technologies for Gene and Cell Therapy

Tae Geol Lee (KRISS)

Translational Research Platform for IRD in SNUH GEC-GCT

Jeong Hun Kim (Seoul Natl. Univ.)



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Potential Role of Retinal Astrocytes in Retinopathy of Prematurity

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Retinopathy of Prematurity (ROP) is a leading cause of childhood blindness, and there is an unmet need for more effective and safer treatments. Current therapies primarily target vascular endothelial growth factor (VEGF) to inhibit abnormal blood vessel growth. While these anti-VEGF treatments have improved patient outcomes, they have limitations such as delayed normal vascular development, potential systemic side effects, and incomplete efficacy in all patients. This presentation focuses on the important yet underappreciated role of retinal astrocytes in the development and progression of ROP. Retinal astrocytes are glial cells that play a key role in the formation and maintenance of the retinal vasculature and there is evidence that dysfunction in these cells can contribute to pathological neovascularization and retinal damage seen in ROP. We will therefore explore potential therapeutic strategies targeting retinal astrocytes, aiming to address the underlying mechanisms of ROP more comprehensively. This approach has the potential to improve therapeutic outcomes, reduce side effects associated with current treatments, and support normal retinal development, preventing vision loss in premature infants.

Key words : Retinopathy of prematurity, Oxygen induced retinopathy, Retinal astrocytes, Pdfra

Autonomous Tissue-Sensing Needle Injection System for Ocular Diseases

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Subretinal injection enables direct delivery of therapeutic agents beneath the retina, thereby enhancing local efficacy while minimizing systemic dosage and side effects. Despite its therapeutic advantages, this procedure remains highly challenging due to the delicate retinal structure, hand tremors during manual operation, and limited depth perception under microscopy. To address these limitations and improve surgical precision, we developed an automated subretinal injection system mounted on a five-axis robotic platform and equipped with fiber Bragg grating (FBG) force sensors. These FBG sensors provide high-sensitivity force feedback, enabling real-time detection of needle–retina contact. Upon detection, the system precisely actuates a linear actuator to advance the needle to the target subretinal space and perform the injection. Real-time monitoring of the injection process is achieved through optical coherence tomography (OCT), which provides micrometer-resolution cross-sectional images of retinal tissue. This imaging enables continuous visualization of tissue deformation, needle positioning, and fluid dispersion dynamics. To further assess the safety and precision of the injection process, histopathological analysis is performed post-procedure to detect potential microstructural damage at the injection site. The integration of real-time imaging and histological evaluation provides a robust feedback loop for system optimization. Based on these multimodal data, we implemented a Bayesian optimization algorithm to identify the optimal injection parameters—including insertion speed, needle depth, and angle—under varying retinal tissue conditions. This data-driven approach significantly reduces the risk of retinal damage while enhancing delivery precision and reproducibility. Our system demonstrates the feasibility of automated, feedback-controlled subretinal injection, offering a promising platform for safe and consistent intraocular drug delivery in both clinical and research settings.

Key words : Subretinal injection, Automated drug delivery, Fiber bragg grating (FBG) sensors, Optical coherence tomography (OCT), Bayesian optimization

Novel Bio-Imaging Technologies for Gene and Cell Therapy

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Label-free bio-imaging measurements are extremely useful in and important to biomedical research and clinical applications because they can be measured in their natural forms. In particular, common concerns include toxicity and inaccurate mode of action, among others, due to labelling of genes and cells for clinical therapy. In this study, we introduce various label-free bio-imaging technologies developed at KRISS for gene and cell therapy, such as high-resolution wide-field spectral-domain optical coherence tomography to achieve marmoset retinal images, dark-field/digital holography hybridized with fluorescent microscopy to measure stem cells and organoids, and mass photometry to analyze gene-encapsulated adeno-associated virus. Our spectral-domain optical coherence tomography allows for detailed analysis of marmoset retinal structures, providing crucial information for the early diagnosis and treatment planning of retinal diseases. In contrast, dark-field/digital holography can measure high-resolution images and monitor dynamic phase changes of organoids in real-time, which are not possible with conventional optical microscopy. Such approaches can be utilized as tools in the field of cell therapy and regenerative medicine, ranging from the cellular level to preclinical experimental models. These label-free bio-imaging technologies developed at KRISS enhance clinical applicability and contribute to understanding biological systems in a non-toxic and safe manner. Ultimately, this is expected to improve patient treatment outcomes and accelerate the development of new drugs and personalized cell therapies.

Key words : Label-free bio-imaging, Gene and cell therapy, Hybrid imaging, Real-time monitoring

Translational Research Platform for IRD in SNUH GEC-GCT

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From the FDA approval of anti-VEGF aptamer to wet-type age-related macular degeneration (AMD) of choroidal neovascularization, anti-VEGF aptamer and antibody have been widely used against all kinds of vaso-proliferative retinopathy. Actually, current therapies directed at controlling vascular abnormalities in vaso-proliferative retinopathy target VEGF and can slow the progression of these diseases. While the general role of VEGF in development has been well described, the specific function of locally synthesized VEGF in the eye is incompletely understood. Recently, RNA-guided genome surgery using CRISPR-Cas9 nucleases has shown promise for the treatment of diverse genetic diseases. Yet, the potential of such nucleases for therapeutic applications in non-genetic diseases including AMD, diabetic retinopathy (DR) as well as retinopathy of prematurity (ROP) is largely unexplored. Those vision-threatening retinopathies such as AMD, DR, and ROP are leading causes of blindness in adults and children, which is associated with retinal over-expression of, rather than mutations in, the VEGFA gene.

Herein I would like to provide some my recent experimental results of therapeutic applications such as small peptide, small molecule as well as nanoparticles beyond anti-VEGF antibody. In addition, therapeutic application strategy targeting mitochondrial regulation on retinopathy would be provided.

Key words : Translational research, Gene therapy, Cell therapy, Inherited retinal disease, Blindness

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SYMPOSIUM 15 [NIFDS] ENG

July 25 (Fri) 09:00–10:40 | Samda Hall

Humanize Mouse Models for Preclinical Research

Organizer : Dae-Yeul Yu (GHBio) / Chair : Dae-Yeul Yu (GHBio), Su-Hyung Park (KAIST)

Generation of a NOG-W41-Based Humanized Mouse Model for In Vivo Human Hematopoiesis

Ryoji Ito (CIEM)

4-1BB Co-Stimulation, a Promising Strategy for Treating Cancers

Su-Hyung Park (KAIST)

Enhanced Human Leukocyte Engraftment and Reduced GvHD Symptoms in CD47;Rag2;IL2 γ Triple Knockout Mice

Seung-Ho Heo (Asan Medical Center)

Target Discovery for Cancer Immunotherapy Using Humanized Mouse Models

Sung-Yup Cho (Seoul Natl. Univ.)



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Generation of a NOG-W41-Based Humanized Mouse Model for In Vivo Human Hematopoiesis

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Humanized mouse models engrafted with human CD34⁺ hematopoietic stem cells (HSCs) provide invaluable platforms for investigating human hematopoiesis, immune cell development, and preclinical drug responses in vivo. However, their broader use has been limited by low engraftment efficiency, poor lineage diversity, and reliance on preconditioning.

To address these issues, we recently developed a next-generation humanized mouse model based on the NOG-W41 strain, which carries a mutation in the c-Kit receptor tyrosine kinase domain. This genetic modification promotes efficient human hematopoietic engraftment without the need for irradiation before transplantation and allows for long-term maintenance of human HSCs in the bone marrow. These features improve the reproducibility and ease of use of the humanized mouse model while preserving a more physiological environment for hematopoiesis.

Using the NOG-W41 strain as a platform, we created several derivative strains incorporating additional genetic elements to support the development of specific blood cell lineages. For example, NOGW-hIL-3/hGM-CSF Tg (NOGW-EXL) and NOGW-EXL hIL-5 Tg mice enable enhanced differentiation of human myeloid cells such as monocytes/macrophages, eosinophils, and mast cells.

Furthermore, we established NOGW-Mpl KO (NOGWM) mice by knocking out the Mpl gene, which encodes the thrombopoietin receptor. These mice supported high levels of human platelet production, over 50% of circulating platelets in peripheral blood, even when a relatively small number of CD34⁺ cells were transplanted.

Overall, NOG-W41-based humanized mice represent a powerful and versatile platform for in vivo studies of human hematopoiesis. Their high engraftment efficiency, ability to support multiple blood lineages, and simplified transplantation procedures make them promising tools for both basic research and translational applications, including disease modeling, immunotherapy development, and drug testing in humanized systems.

Key words : Humanized mice, NOG-W41, Hematopoiesis, Immune system

4-1BB Co-Stimulation, a Promising Strategy for Treating Cancers

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4-1BB expression on CD8⁺ TILs represents a distinct activation state among highly exhausted CD8⁺ T cells in HCC. 4-1BB co-stimulation with agonistic antibodies may be a promising strategy for treating cancers exhibiting prominent T-cell activation. However, dose-dependent hepatotoxicity was observed in clinical trials with monoclonal anti-4-1BB agonistic antibodies due to the activation of 4-1BB signaling in liver resident Kupffer cells. To avoid this on-target liver toxicity, a novel bispecific antibody was specifically designed to activate 4-1BB signaling only in the presence of PD-L1, while also blocking PD-1/PD-L1 signaling. The anti-4-1BB×PD-L1 bispecific antibody augmented T-cell activation in *in vitro* assays, and further enhanced the anti-PD-L1-mediated reinvigoration of tumor-infiltrating CD8⁺ T cells. Furthermore, in humanized PD-L1/4-1BB transgenic mice challenged with huPD-L1-expressing tumor cells, the bispecific antibody induced superior anti-tumor activity and maintained an anti-tumor response against tumor rechallenge. In conclusion, the novel anti-4-1BB×PD-L1 bispecific antibody may exert a strong anti-tumor therapeutic efficacy with a low risk of liver toxicity through the restriction of 4-1BB stimulation in tumors.

Key words : 4-1BB, Bispecific antibody, Cancer immunotherapy, Immune-oncology, Exhausted T cells

Enhanced Human Leukocyte Engraftment and Reduced GvHD Symptoms in CD47;Rag2;IL2 γ Triple Knockout Mice

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Humanized mouse models that recapitulate human biological systems have become increasingly important for preclinical immunotherapy studies. However, these models still have limitations, such as the onset of lethal graft-versus-host disease (GvHD), a variable success rate, and the low accessibility of total body irradiation. To address these limitations, we generated humanized mice using a novel CD47;Rag2;IL2 γ triple knockout NOD (RTKO) mouse strain. The RTKO strain was generated because it combines key advantages: the CD47-signal regulatory protein alpha (SIRP α) signaling system inhibits host cell phagocytosis, the NOD mouse SIRP α gene exhibits strong affinity for human CD47, and the rag mutation confers irradiation tolerance and no T/B cell leakiness. Initially, we generated human CD34⁺ hematopoietic stem cell (HSC)-engrafted humanized mice preconditioned with total body irradiation or busulfan (BSF) injection. Over a long period from 16 to 40 weeks post-transplantation, the percentage of hCD45⁺ cells was sustained the longest and at the highest levels in the RTKO BSF group. Reconstruction of human leukocytes, including hCD3⁺ cells, was also most prominent in the RTKO BSF group. The occurrence of GvHD has been identified as mainly due to human T cells infiltrating tissues and their related cytokines. Considering the improvement in human leukocyte reconstitution and the extended experimental window beyond 48 weeks post-transplantation with alleviated GvHD symptoms, the RTKO BSF group was identified as the most suitable model. Next, we also generated and optimized PBMC-engrafted humanized mice using the RTKO strain. When standard doses of PBMCs ($5-10 \times 10^6$ cells) were used, RTKO mice showed enhanced engraftment of human leukocytes, though GvHD was more severe, resulting in a narrow experimental window. In a subsequent trial using a lower dose of PBMCs, RTKO mice exhibited stable human leukocyte reconstitution with milder GvHD symptoms and no life-threatening lesions. Considering the difficulties in generating hematopoietic stem cell (HSC)-engrafted humanized mice, the extended experimental window provided by this model is particularly noteworthy. In conclusion, RTKO mice are expected to provide a useful platform for various immunological research areas, including the generation of humanized mice and transplantation studies.

Key words : CD47, Graft-versus-host disease, Hematopoietic stem cell, Humanized mice, Peripheral blood mononuclear cells

Target Discovery for Cancer Immunotherapy Using Humanized Mouse Models

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Immune checkpoint therapy (ICT) provides durable responses in select cancer patients, yet resistance remains a significant challenge, prompting the exploration of underlying molecular mechanisms. Tyrosylprotein sulfotransferase-2 (TPST2), known for its role in protein tyrosine O-sulfation, has been suggested to modulate the extracellular protein-protein, but its specific role in cancer immunity remains largely unexplored. To explore tumor cell-intrinsic factors influencing anti-PD1 responsiveness, we conducted a pooled loss-of-function genetic screen in humanized mice engrafted with human immune cells. Through in vivo genome-wide CRISPR screening, TPST2 loss-of-function emerged as a potential enhancer of anti-PD1 treatment efficacy. TPST2 suppressed IFN γ signaling by sulfating IFN γ receptor 1 at Y397 residue, while its downregulation boosted IFN γ -mediated signaling and antigen presentation. Depletion of TPST2 in cancer cells augmented anti-PD1 antibody efficacy in syngeneic mouse tumor models by enhancing tumor-infiltrating lymphocytes. RNA sequencing data revealed TPST2's inverse correlation with antigen presentation, and increased TPST2 expression is associated with poor prognosis and altered cancer immunity across cancer types. We propose TPST2's novel role as a suppressor of cancer immunity and advocate for its consideration as a therapeutic target in ICT-based treatments.

Key words : Immune checkpoint therapy, Humanized mice, CRISPR screening, Tyrosylprotein sulfotransferase-2, Interferon- γ

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PLENARY LECTURE 2 ENG

July 25 (Fri) 11:00–11:50 | Halla Hall A, B

Organizer / Chair : Won-II Jeong (KAIST)

Adipocyte Heterogeneity and the Regulation of Energy Metabolism

Christian Wolfrum (NTU Singapore)



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Adipocyte Heterogeneity and the Regulation of Energy Metabolism

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Excess adipose tissue, as observed in obesity, poses a significant risk for the development of multiple disorders, ranging from cardiovascular complications to certain types of cancers, to name but two. While most studies in the past have focused on the link between adipose tissue mass and metabolic health, work in recent years has clearly demonstrated that adipose tissue quality is similarly an important determinant of metabolic control. So far adipose tissue quality is broadly assessed by depot localization and the average size of the adipocyte. Recently, the content of thermogenic brown and brite/beige adipocytes has been shown to stratify the patient's health status independent of the overall adipose tissue mass.

Only in the last years through the advances in single cell and nucleus sequencing technologies has it become clear that adipose tissue on the level of the mature adipocyte is much more complex than previously envisaged, suggesting that the heterogeneity and plasticity of different adipocyte populations might define adipose tissue quality. Thus, identification of other adipose subtypes might reveal new targets to treat both obesity as well as the associated co-morbidities.

Keywords: Metabolic homeostasis, Adipose tissue quality, Thermogenic adipocyte, Single cell analysis, Adipocyte heterogeneity

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Luncheon Seminar 4~6

July 25 (Fri) 12:00–12:20

Luncheon Seminar 4

Halla Hall A

Chair : Jun-Gyo Suh (Hallym Univ.)

GONAD: Simple Method for Genome-Editing in Mice & Rats

Makoto Matsuyama (Shigei Medical Research Institute)

Luncheon Seminar 5

Halla Hall B

Chair : Dae-Youn Hwang (Pusan Natl. Univ.)

Genetically Engineered Mice Models Accelerate Metabolic Disease Drug Development

Shuxin Xu (GemPharmatech Co., Ltd.)

Luncheon Seminar 6

Samda Hall

Chair : Jae-Ho Shin (Eulji Univ.)

Promoting NH Financial Products

Yeong Gyun Gwon (NongHyup Financial Group INC.)



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GONAD: Simple Method for Genome-Editing in Mice & Rats

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Recent advances in the CRISPR/Cas9 system have demonstrated it to be an efficient gene-editing technology for various organisms. Laboratory mice and rats are widely used as common models of human diseases; however, the current standard method to create genome-engineered animals is laborious and involves three major steps: isolation of zygotes from females, ex vivo micromanipulation of zygotes, and implantation into pseudopregnant females. To circumvent this, we recently developed a novel method named Genome-editing via Oviductal Nucleic Acids Delivery (GONAD). This method does not require the ex vivo handling of embryos; instead, it can execute gene editing with just one step, via the delivery of a genome-editing mixture into embryos in the oviduct, by electroporation. Here, we present a further improvement of GONAD that is easily applicable to both mice and rats. It is a rapid, low-cost, and ethical approach fulfilling the 3R principles of animal experimentation: Reduction, Replacement, and Refinement. This method has been reconstructed and renamed as "improved GONAD (i-GONAD)" for mice, and "rat improved GONAD (rGONAD)" for rats. I will talk about the overview of GONAD method.

Key words : CRISPR/Cas9, Genome-editing, GONAD, Mouse, Rat

LS5

Genetically Engineered Mice Models Accelerate Metabolic Disease Drug Development

Shuxin Xu

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Metabolic diseases pose a severe threat to human health and have emerged as a major global public health challenge. Against this backdrop, the development of therapeutics for metabolic diseases has become a major focus within pharmaceutical research. Drug targets represented by the glucagon-like peptide-1 receptor (GLP-1R) are actively pursued by numerous research pipelines. In the drug discovery process, animal models serve as indispensable and critical tools, playing a decisive role in the success of drug development. This report systematically elaborates on the vital applications of genetically modified animal models in metabolic disease drug R&D. It comprehensively analyzes the value and practical significance of these models throughout the entire drug development workflow, spanning multiple dimensions such as drug target validation, in vitro drug screening, in vivo pharmacokinetic and pharmacodynamic (PK/PD) studies, and pharmacodynamic evaluation. The report aims to provide theoretical references and practical guidance for drug discovery efforts in related fields.

Key words : Metabolic diseases, GLP-1R, Mice model, Preclinical research, In vivo efficacy

July 25 (Fri)

LS6

Promoting NH Financial Products

Yeong Gyun Gwon

NongHyup Financial Group INC.

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SYMPOSIUM 16 ENG

July 25 (Fri) 14:00–15:50 | Halla Hall A

Development of 3D Composite Constructs with Immune Modulatory Functions and Tissue Regeneration Technologies

Organizer / Chair : Kyung-Sun Kang (Seoul Natl. Univ.)

Bioengineered Liver Crosslinked with Nano-Graphene Oxide Enables Efficient Liver Regeneration via MMP Suppression and Immunomodulation

Da-Hyun Kim (Sungshin Women's Univ.)

Humanized Mouse Models in Hematopoietic Research:
From Disease Mechanisms to Therapeutics

Kyung-Rok Yu (Seoul Natl. Univ.)

Expanding the Frontiers of Spatial Cellular Analysis:
Applications of SLACS in Animal Models and Organoid Systems

Chungwon Lee (Meteor Biotech)

Advancing Liver Tissue Engineering: Improving Endothelial Cell Attachment
in Decellularized Liver Scaffolds

Kyung-Mee Park (Chungbuk Natl. Univ.)

Current Status of Human Tissue Substitutes Developed Using Transgenic Animals

Hyunil Kim (Optipharm Inc.)



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Bioengineered Liver Crosslinked with Nano-Graphene Oxide Enables Efficient Liver Regeneration via MMP Suppression and Immunomodulation

Da-Hyun Kim

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Decellularized extracellular matrix scaffold, widely utilized for organ engineering, often undergoes matrix decomposition after transplantation and produces byproducts that cause inflammation, leading to clinical failure. Here we propose a strategy using nano-graphene oxide to modify the biophysical properties of decellularized liver scaffolds. Notably, we demonstrate that scaffolds crosslinked with nano-graphene oxide show high resistance to enzymatic degradation via direct inhibition of matrix metalloproteinase activity and increased mechanical rigidity. We find that M2-like macrophage polarization is promoted within the crosslinked scaffolds, which reduces graft-elicited inflammation. Moreover, we show that low activities of matrix metalloproteinases, attributed to both nano-graphene oxide and tissue inhibitors of metalloproteinases expressed by M2c, can protect the crosslinked scaffolds against in vivo degradation. Lastly, we demonstrate that bioengineered livers fabricated with the crosslinked scaffolds remain functional, thereby effectively regenerating damaged livers after transplantation into liver failure mouse models. Overall, nano-graphene oxide crosslinking prolongs allograft survival and ultimately improves therapeutic effects of bioengineered livers, which offer an alternative for donor organs.

Key words : dECM scaffold, Artificial liver, Nano-graphene oxide, M2 macrophage

Humanized Mouse Models in Hematopoietic Research: From Disease Mechanisms to Therapeutics

Aaron Yu, Hyun Sung Park, Dong-Hoon Chae, Kyung-Rok Yu*

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To explore the pathophysiology of human hematologic diseases, humanized mouse models have been developed using the transplantation of hematopoietic stem and progenitor cells (HSPCs). These immunodeficient mice, generated by engrafting human HSPCs or peripheral blood-derived mononuclear cells (PBMCs), mimic human immune interactions and facilitate the evaluation of immunotherapeutic strategies.

Our studies have illuminated how environmental and viral exposures affect hematopoietic properties. We showed that both cigarette smoke extract (CSE) and SARS-CoV-2 pseudovirus disrupt hematopoietic homeostasis. CSE impairs the niche-supportive function of mesenchymal stem cells (MSCs) via reactive oxygen species and NLRP3 inflammasome activation, reducing HSPC engraftment. Similarly, Omicron pseudovirus infection of HSPCs induced inflammaging signatures, diminished colony-forming capacity, and biased myeloid differentiation in vitro and in humanized mice. These phenotypes were partially reversed by nanographene oxide (NGO), underscoring its therapeutic potential.

I will also discuss our recent findings on NGO's immunomodulatory role in acute graft-versus-host disease (GVHD) using a xenogeneic mouse model. NGO administration improved survival, reduced weight loss and leukocyte infiltration, and alleviated GVHD pathology. In PBMCs from GVHD patients, NGO treatment increased naïve T cells and reduced effector memory subsets, indicating restoration of immune balance. RNA sequencing revealed selective suppression of inflammatory pathways and IFN γ -STAT1 signaling, alongside macrophage polarization toward an anti-inflammatory phenotype.

To enhance safety and translational relevance, we developed NGO-primed macrophages (NGO-Macs), which exhibited an M2-like phenotype, suppressed Th1 responses, promoted regulatory T cell induction, and significantly attenuated GVHD severity in vivo. These findings highlight humanized mouse models as valuable platforms for mechanistic studies and support the therapeutic potential of NGO and NGO-Macs in hematologic and immune disorders.

Key words : Humanized mouse model, Hematopoietic stem cells, SARS-CoV-2 pseudovirus, Graft-versus-host disease, Nanographene oxide

Expanding the Frontiers of Spatial Cellular Analysis: Applications of SLACS in Animal Models and Organoid Systems

Sumin Lee, Amos Lee*

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Advancements in translational research demand increasingly precise tools to dissect cellular heterogeneity within complex tissue environments. We present a novel platform based on Spatially-resolved Laser Activated Cell Sorting (SLACS), which enables high-fidelity isolation of single cells from intact tissue sections in preclinical animal models, while preserving spatial context. This technology overcomes the limitations of conventional flow cytometry and laser capture microdissection by offering non-destructive, subcellular resolution cell separation combined with downstream multi-omics analysis (e.g., transcriptomics, proteomics).

In murine models of cancer and inflammation, SLACS has been applied to spatially isolate immune cell populations and tumor subclones from defined microenvironments. Subsequent single-cell and spatial omics profiling has revealed location-specific gene expression signatures and pathway activation patterns that were not observable via dissociated bulk analysis. Moreover, integration with immunohistochemistry and AI-based pathology has allowed for automated hotspot detection and targeted cell retrieval, enabling mechanism-of-action (MoA) studies, biomarker validation, and drug efficacy evaluation with enhanced spatial resolution.

This integrated approach offers a powerful tool for researchers utilizing animal models to bridge the gap between molecular data and in vivo physiology. It holds significant promise for improving the translational relevance of preclinical studies and optimizing therapeutic development pipelines.

Key words : Spatial cell sorting, Multi-omics, Preclinical model, Microenvironment, Translational research

Advancing Liver Tissue Engineering: Improving Endothelial Cell Attachment in Decellularized Liver Scaffolds

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Liver tissue engineering offers a promising solution to the organ shortage crisis, yet restoring a perfusable and thrombosis-resistant vascular network remains a critical challenge. Without adequate endothelialization, decellularized liver scaffolds often fail due to clot formation and poor cell retention. Previous studies have reported strategies such as antibody-guided re-endothelialization, aptamer-functionalized surfaces, and heparin–gelatin coatings to reduce thrombogenicity and enhance graft patency. However, scalable and economical strategies that also support hepatic function are still needed. In this study, we investigated three biofunctional scaffold modifications—heparin immobilization, sphingosine-1-phosphate (S1P) supplementation, and fibronectin (FN) coating—each targeting key bottlenecks in vascular scaffold performance. Heparin immobilization significantly reduced platelet adhesion and thrombus formation, improving endothelial coverage and in vivo angiogenesis. S1P enhanced endothelial proliferation, retention, and function, with upregulation of vascular genes (VE-cadherin, NOS3, VEGF) and reduced apoptosis. FN coating improved attachment and proliferation of endothelial and hepatic cells, resulting in higher albumin production and lower thrombogenicity, even without anticoagulant agents. These complementary strategies offer an effective platform to enhance vascular stability, hemocompatibility, and hepatic integration in liver scaffold engineering. Their simplicity, biological relevance, and scalability support their potential for future application in clinically viable bioengineered liver grafts.

Key words : Decellularized liver scaffold, Endothelialization, Liver tissue engineering, Recellularization, Thrombosis prevention

Current Status of Human Tissue Substitutes Developed Using Transgenic Animals

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As global aging accelerates, the demand for organ transplantation is steadily increasing. While transplantation between humans remains the most effective approach, the number of organ recipients continues to grow, whereas the number of donors has not kept pace. As a result, the waiting list for organ transplants is expanding.

To address this shortage, research into the use of genetically modified pigs as alternative organ sources is actively underway. Notably, both the United States and China have already begun transplanting organs from genetically engineered pigs into humans. In the U.S., heart and kidney transplants have been conducted, while in China, cornea, liver, and skin transplants have taken place. The U.S. has announced a plan to transplant kidneys from genetically modified pigs into six patients with end-stage renal disease starting in 2025, with potential expansion to 50 patients depending on the outcomes.

Beyond solid organs, genetically modified pigs are also being used to develop therapies for type 1 diabetes (islet cell transplants), corneal replacements for patients with eye injuries, skin grafts for burn victims, and even blood products to address shortages in human blood supplies.

This presentation will review the current global landscape of artificial organ development, with a particular focus on the progress of artificial organs and tissues derived from genetically engineered pigs.

Key words : Miniature pig, Translational research, Transgenic pig, Xenotransplantation

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SYMPOSIUM 17

ENG

July 25 (Fri) 14:00–15:50 | Halla Hall B

Latest Research Trends in Development of Experimental Animal Models and Biomarkers,
and AI-Based Behavioral Analysis Technologies for Substance Use Disorder

Organizer / Chair : Hye Jin Cha (Gyeongsang Natl. Univ.)

TRPA1 as a Therapeutic Target for Cocaine Addictive Disorders

Choon-Gon Jang (Sungkyunkwan Univ.)

Impaired Decision-Making in Methamphetamine-Dependent Rats:
Neural Mechanism and Novel Treatment

Kiyofumi Yamada (Fujita Health Univ.)

Inhaled Synthetic Cannabinoid Self-Administration Model and
Its Application in Vaccine Development for Drug Abuse

Jaesuk Yun (Chungbuk Natl. Univ.)

Non-Human Primate Models for Addiction Research: Behavior and Imaging

Dohyun Lee (OSONG Medical Innovation Foundation)



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TRPA1 as a Therapeutic Target for Cocaine Addictive Disorders

Choon-Gon Jang

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Cocaine is a powerful psychostimulant known to cause severe addiction in users; it enhances drug-seeking behavior to a point where users can lose control of their drug intake. At behavioral and physiological levels, cocaine use leads to compulsory addiction-related behavior and causes long-lasting functional changes in neuronal circuitry. The transient receptor potential ankyrin type 1 receptor (TRPA1) is the ion channel for somatosensory modalities such as pain, cold and itch. However, the role of TRPA1 on cocaine reward and seeking has not been established. The present study was investigated that mice with knockdown of the cation channel TRPA1 were resistant to the cocaine-seeking behavior and reward effects of cocaine compared to their wildtype litter mates. In our study, we demonstrate that TRPA1 inhibition in the NAc reduces cocaine activity and dopamine release, and conversely, that TRPA1 is critical for cocaine-induced synaptic strength in dopamine receptor 1-expressing medium spiny neurons. Taken together, our data support that cocaine-induced reward-related behavior and synaptic release of dopamine in the NAc are controlled by TRPA1 and suggest that TRPA1 has therapeutic potential as target for cocaine addictive disorders.

Key words : Cocaine, TRPA1, Reward, Psychostimulant, Abuse

Impaired Decision-Making in Methamphetamine-Dependent Rats: Neural Mechanism and Novel Treatment

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Patients suffering from neuropsychiatric disorders such as substance-related and addictive disorders exhibit altered decision making patterns, which may be associated with their behavioral abnormalities. However, the neuronal mechanisms underlying such impairments are largely unknown. Using a gambling test for rodents, we have demonstrated that chronic methamphetamine-treated rats prefer high-risk/high-reward actions and assigned higher value to high returns, indicative of altered decision-making. Pharmacological studies as well as DREADD technology revealed that the insular neural system controls decision-making in both healthy and methamphetamine-treated rats. Subsequently, we investigated the effect of nicotine on impaired decision-making strategy in chronic methamphetamine-treated rats since nicotine has been shown to have therapeutic effects on attentional and cognitive abnormalities in psychosis. We found that methamphetamine-induced impairment of decision-making was reversed by daily nicotine treatment (0.3 mg/kg, s.c.). This effect was associated with the reduction of lose-shift behavior after negative reward prediction error. Repeated treatment with nicotine had no effects on arm-choice behavior in naïve rats. Varenicline, an $\alpha 4\beta 2$ -nicotinic acetylcholine receptor partial agonist, also ameliorated the altered decision-making in methamphetamine-treated rats. Our findings suggest that nicotine treatment is useful for ameliorating the altered decision-making caused by methamphetamine treatment, and that the $\alpha 4\beta 2$ -nicotinic acetylcholine receptor is a therapeutic target for poor decision-making.

Key words : Decision-making, Methamphetamine, Drug nicotinic acetylcholine receptor

Inhaled Synthetic Cannabinoid Self-Administration Model and Its Application in Vaccine Development for Drug Abuse

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Synthetic cannabinoid receptor agonists (SCRAs) are novel psychoactive substances with high potency. Their structures change rapidly, and their pharmacological and toxicological profiles remain largely unknown. Because these compounds are often abused by inhalation through electronic cigarettes, timely regulation will require suitable preclinical inhalation models to evaluate their abuse potential.

To address this issue, we developed a nose-poke–restricted vapor self-administration method in mice in which drug vapor is delivered exclusively within the nose-poke port. This design avoids the whole-body aerosol exposure of conventional systems, improves face validity.

With this setup, we demonstrated self-administration behaviors of several SCRAs in C57BL/6N mice, as shown by increased active nose-poke responses. After extinction, both cue- and drug-challenge–induced reinstatement was observed. These results indicate that the nose-poke inhaled self-administration model reproduces SCRA-induced reinforcement and relapse-like behavior in mice.

Because the model directly measures SCRA-taking behavior, we subsequently employed this model to evaluate therapeutic interventions. Effective pharmacological treatments for substance-use disorders remain limited, and vaccines that produce antibodies against the abused-drug itself have emerged as a promising alternative. We immunized mice with SCRA haptens conjugated to carrier proteins and formulated them with adjuvants, then tested them in the same inhalation self-administration paradigm. Vaccinated mice displayed markedly fewer active nose-pokes and reduced vapor deliveries compared with non-immunized controls. These results suggest that our nose-poke vaping self-administration model not only replicates SCRA-induced reinforcement and relapse, but also provides a robust platform for evaluating vaccine candidates targeting inhaled psychoactive substances.

Key words : Synthetic cannabinoid receptor agonist, Inhaled self-administration, Vaporization, Abused drug vaccine

Non-Human Primate Models for Addiction Research: Behavior and Imaging

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To regulate new psychoactive substances (NPS), it is necessary to demonstrate that they possess abuse liability. Drug self-administration (SA) procedures are the most widely used method for assessing the relative abuse liability of drugs across various classes. While both rodents and non-human primates (NHPs) are commonly used in SA testing, NHPs are considered more appropriate for evaluating human abuse liability due to their closer similarity to humans in terms of genetics, anatomy, physiology, and cognitive/behavioral functions. Notably, NHPs exhibit greater similarity to humans than rodents in central nervous system (CNS) receptor systems and drug pharmacokinetics. Among NHPs, the common marmoset (*Callithrix jacchus*) offers specific advantages over macaque monkeys, particularly due to its small body size and favorable reproductive characteristics, which enable large-scale testing.

An oral SA model for cocaine has been established in marmosets, and results have demonstrated their potential for developing drug dependency. Additionally, an intravenous SA model has been developed using a custom-made small infusion pump integrated with a Bluetooth system. This setup allowed for non-restrained testing, thereby improving animal welfare. The effects of abuse liability on the CNS have been investigated using brain imaging techniques such as magnetic resonance spectroscopy (MRS), resting-state functional magnetic resonance imaging (rs-fMRI), diffusion tensor imaging (DTI), and positron emission tomography-computed tomography (PET-CT). The observed brain changes in cocaine-addicted marmosets appear to be consistent with those found in human addiction. Clinical and behavioral assessments were also conducted following the SA procedures. In particular, cocaine-induced behavioral responses were analyzed using deep learning algorithms, focusing on rapid, stereotyped psychomotor behaviors, especially head shaking. The behavioral categorization algorithm achieved 90% accuracy, 94% sensitivity, and a 92% F1-score.

In conclusion, the SA procedure using marmosets offers an efficient and ethically sound model for assessing abuse liability. Brain imaging in marmosets may effectively mimic the dependency and neurotoxicity of NPS in humans, and behavioral analysis using deep learning algorithms can yield reliable and labor-efficient results.

Key words : Artificial intelligence, Neuroscience, Dopamine transporter, Neurotransmitter, 3R

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SYMPOSIUM 18 [KLAT Education 2] KOR

July 25 (Fri) 14:00–15:50 | Samda Hall

Application and Management of Genetically Modified Animals

Organizer : Hanseul Oh (Chungbuk Natl. Univ.) / Chair : Hyunjhong Jhun (KFRI)

Health Risks and Optimized Management in Genetically Engineered Lab Animals

Taehoon Shin (Jeju Natl. Univ.)

Generation and Use of Genetically-Engineered Mouse Models Using CRISPR-Cas9

Young Hoon Sung (Asan Medical Center)

Generation of Mouse Models and Experimental Applications

Kyoungmi Kim (Seoul Natl. Univ.)



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S18-1

Health Risks and Optimized Management in Genetically Engineered Lab Animals

Taehoon Shin

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Developing sophisticated gene editing technologies such as CRISPR/Cas has accelerated the establishment and diversification of genetically engineered or modified animals, particularly genetically engineered mouse models (GEMMs). The widespread use of GEMM has revolutionized biomedical science by allowing more precise modeling of various human diseases. However, these specialized animals harboring the complexity of genetic alterations often represent altered physiology and immune function, resulting in increased susceptibility to infections and disease incidence. This session will provide an overview of health concerns encountered in GEMMs, especially focused on the impact of genetic background or alterations on specific disease susceptibility, and management strategies including breeding optimization and biosecurity practices. By the end of this session, potential or early-career laboratory animal technologists will have a deeper understanding of how to deal with the unique health challenges presented by GEMMs, ensuring reliable research outcomes and animal welfare through proactive care.

Key words : Genetically engineered animals, Disease susceptibility, Health monitoring, Colony management

July 25 (Fri)

Generation and Use of Genetically-Engineered Mouse Models Using CRISPR-Cas9

Young Hoon Sung

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Based on their genetic and physiological similarities to humans, various laboratory animals are used in numerous biomedical studies. Mice are not only easy to raise and breed, but they also have most of the genes found in humans. Furthermore, genetic mutations that cause diseases in humans often cause the same diseases in mice. The mice with the same diseases as humans by a specific mutation are called diseased mouse models. Thus, they are used as essential experimental reagents not only for studying the basic aspects of diseases but also for numerous studies necessary for developing novel strategies for disease prevention and treatment. To produce valuable diseased mouse models, we need to generate genetically-engineered mouse (GEM) models carrying novel mutations and to determine whether they recapitulate the human diseases. In this lecture, I will explain CRISPR-Cas9 and gene editing and how to apply it to create GEM models.

S18-3

Generation of Mouse Models and Experimental Applications

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CRISPR/Cas systems have revolutionized the field of genome engineering, enabling precise and efficient genetic modifications in a wide range of organisms. In our research, we have utilized CRISPR-based tools to generate various genetically engineered mouse models, tailored for investigating gene functions, disease mechanisms, and therapeutic strategies. By optimizing guide RNA design and delivery methods, we have successfully established both knockout and knock-in models with high efficiency and reproducibility. Furthermore, we applied these models to explore gene regulation, developmental biology, and pathophysiological processes in vivo. This presentation will highlight key methodologies for mouse model generation using CRISPR, discuss challenges such as off-target effects and mosaicism, and showcase experimental applications that underscore the value of these models in translational research.

Key words : CRISPR/Cas system, Genome editing, Genetically engineered mouse models, Knockout and knock-in

July 25 (Fri)



실험동물연구장학생 POSTER PRESENTATION

July 24 (Thu) 13:20–14:20 | ICC JEJU 3F Lobby

Chair : Kyoungmi Kim (Seoul Natl. Univ.)	
PS-R-01	Hee Jin Song (Pusan Natl. Univ.)
PS-R-02	Chae Yeon Kim (Kyugnpook Natl. Univ.)
PS-R-03	Zhibin Liu (Kyugnpook Natl. Univ.)
PS-R-04	Ayun Seol (Pusan Natl. Univ.)
PS-R-05	Kyurae Kim (KAIST)
PS-R-06	Sol Pin Kim (Seoul Natl. Univ.)
PS-R-07	Sung Eun Choi (KAIST)



실험동물연구장학생 포스터 발표 안내

1. 포스터 발표 안내

발표시간	July 24 (Thu) 13:20~14:20
발표장소	ICC JEJU Lobby 3F (제주 국제컨벤션센터 3층 로비)
포스터 번호	PS-R-01 PS-R-02 PS-R-03 PS-R-04 PS-R-05 PS-R-06 PS-R-07 총 7개
부착 시간	July 24 (Thu) 09:00~11:00
철거 시간	July 24 (Thu) 17:00~17:30

※ 철거 시간 이후의 포스터 분실은 책임지지 않습니다.

※ 부착은 7월 24일 (목) 부착 시간 내에 반드시 진행해야 하며, 미부착시 제재가 주어집니다.

2. 포스터 발표 및 시상

- 포스터 발표는 좌장의 진행에 따라 포스터당 7분 (5분 발표, 2분 질의응답)으로 진행되며, 발표시간에 포스터 앞에 대기 하여 주시기 바랍니다.
- 장학생은 7월 24일(목) 17:00~19:00, 총회 및 환영만찬에서 진행하는 시상식에 반드시 참석해 주시기 바랍니다. (대리수상불가)
- 포스터 보드에 2회 이상 (개수로 적용) 미부착 시, 교신저자(연구책임자)에게 향후 1년간 포스터 제출 불가의 제재가 주어집니다.

3. 포스터 작성안내

- Poster board의 크기는 **95cm(가로) X 210cm(세로)**이며, 특히 제목이 가로 너비를 초과하지 않도록 준비하여야 합니다.
- 모든 포스터는 지정된 날짜, 기간 동안에 지정된 board에 부착하여야 합니다.
- 포스터 내용은 abstract, purpose, results(figures and tables), conclusions, references의 순서로 작성합니다.
- 전방 2m 위치에서 쉽게 읽을 수 있도록 굵고 명확한 글씨체를 이용하여 제작합니다.
- 모든 포스터의 부착 및 철거는 당일 지정된 시간에 발표자가 수행해주시기 바랍니다.

실험동물연구장학생 Poster

Poster no.	Title	Speaker
PS-R-01	Phenotypic Characterization of Age-Related Macular Degeneration in Complement Component 3 Knockout Mice	Hee Jin Song
PS-R-02	Protective Effects of Imatinib on a DSS-induced Colitis Model through Regulation of Apoptosis and Inflammation	Chae Yeon Kim
PS-R-03	Parishin A Inhibits Oral Squamous Cell Carcinoma via the AKT/mTOR Signaling Pathway	Zhibin Liu
PS-R-04	Effect of Cholesterol-Rich Exosomes Derived from Fatty Liver During Progression of Age-Related Macular Degeneration	Ayun Seol
PS-R-05	Binge Drinking Triggers VGLUT3-Mediated Glutamate Secretion and Subsequent Hepatic Inflammation by Activating mGluR5/NOX2 in Kupffer Cells	Kyurae Kim
PS-R-06	An Innovative Approach Using CRISPR-Ribonucleoprotein packaged in Virus-Like Particles to Generate Genetically Engineered Mouse Models	Sol Pin Kim
PS-R-07	CX3CR1+ Macrophages Interact with Hepatic Stellate Cells to Promote Hepatocellular Carcinoma through CD8+ T Cell Suppression	Sung Eun Choi

PS-R-01

Phenotypic Characterization of Age-Related Macular Degeneration in Complement Component 3 Knockout Mice

Hee Jin Song, Ji Eun Kim, Ayun Seol, Su Jeong Lim, Su Ha Wang, Ye Eun Ryu, Ye Ryeong Kim, Dae Youn Hwang*

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Age-related macular degeneration (AMD) is a leading cause of blindness in the elderly. Recent studies have suggested that complement component 3 (C3) plays a critical role in the pathogenesis of AMD. However, the relationship between C3 and AMD remains controversial due to conflicting findings. This study aimed to clarify the role of C3 deficiency in AMD progression by providing scientific evidence through the characterization of AMD-related phenotypes in C3 knockout (KO) mice at 12 months and 8 weeks of age. In the retina of 12-month-old C3 KO mice, the inner plexiform layer (IPL) was elongated, while the outer segment (OS) was shortened. In addition, ultrastructural analysis revealed significant abnormalities. Transmission electron microscopy (TEM) showed a marked increase in Bruch's membrane (BM) thickness in KO mice compared to wild-type (WT) controls, along with cystic alterations unique to the KO group. Moreover, C3 KO mice exhibited a higher number of damaged mitochondria and an expanded vacuolar area in the retina relative to WT mice. Scanning electron microscopy (SEM) demonstrated irregular IPL structure and OS thinning. In 7-week-old C3 KO mice, the ganglion cell layer (GCL), IPL, and inner nuclear layer (INL) were thinner than those of WT mice. Notably, upon blue light (BL) exposure, KO mice showed a reduced number of GCL cells and clear morphological disturbances. Collectively, these findings suggest that C3 deficiency is closely associated with the development and progression of AMD through structural and ultrastructural retinal degeneration.

*Corresponding author: Dae Youn Hwang

Keywords: Complement component 3, Age-related macular degeneration, Bruch's membrane, C3 knock out mice

PS-R-03

Parishin A Inhibits Oral Squamous Cell Carcinoma via the AKT/mTOR Signaling Pathway

Zhibin Liu, Myoung Ok Kim*

Department of Animal Science and Biotechnology, Research Institute for Innovative Animal Science, Kyungpook National University, Sangju-si, Gyeongsangbuk-do 37224, Republic of Korea

Oral squamous cell carcinoma (OSCC) is an aggressive cancer with limited treatment options. Parishin A, a natural compound derived from *Gastrodia elata*, possesses multiple therapeutic properties. However, its effects on OSCC remain unexplored. Purpose: This study explores the anti-cancer potential of Parishin A on OSCC and its mechanisms. OSCC cell lines YD-10B and Ca9-22 were treated with varying Parishin A concentrations. Cell viability was detected using the CCK-8 assay, and colony formation was evaluated in agarose gel. Migration and invasion ability were assessed through wound healing and Matrigel invasion assays. The protein expression levels involved in the PI3K/AKT/mTOR signaling pathway and epithelial-mesenchymal transition (EMT) markers were examined via Western blotting. Parishin A inhibited OSCC cell viability in both dose- and time-dependent manners, with significant reductions at 20, 40, 60, and 80 μ M, without affecting normal human gingival fibroblasts. Colony formation decreased substantially at ≥ 40 μ M higher Parishin A concentrations in a dose-dependent manner. Also, migration and invasion assays showed significant suppression by Parishin A treatment concentration ≥ 40 μ M in a dose-dependent manner, as evidenced by decreased wound closure and invasion. Western blot analyses revealed increased E-cadherin levels and decreased N-cadherin and vimentin levels, suggesting EMT inhibition. Parishin A also decreased the phosphorylation levels of PI3K, AKT, and mTOR. Collectively, these findings support the potential of Parishin A as an anti-OSCC agent.

*Corresponding author: Myoung Ok Kim

Keywords: EMT, Oral squamous cell carcinoma, Parishin A, PI3K/AKT/mTOR pathway, Proliferation

PS-R-02

Protective Effects of Imatinib on a DSS-Induced Colitis Model through Regulation of Apoptosis and Inflammation

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Background/aim: Inflammatory bowel disease (IBD) is characterized by dysregulated immune responses and a multifactorial etiology. While imatinib has demonstrated efficacy in the treatment of immune-related diseases, its potential effects in IBD treatment remain underexplored.

Materials and methods: This study aimed to investigate the therapeutic effects of imatinib in colitis treatment. A dextran sulfate sodium (DSS)-induced colitis model was used to mimic IBD in mice. Imatinib was administered orally to mice simultaneously with DSS treatment. The effects of imatinib on DSS-induced colitis were evaluated by analyzing colitis-related pathology, including the disease activity index (DAI), histological lesions, inflammatory markers, and tight junction integrity. Additionally, western blot analysis and quantitative real-time polymerase chain reaction were used to assess inflammatory markers, tight-junction proteins, and cell death.

Results: In the DSS-induced colitis model, imatinib treatment exerted protective effects by attenuating weight loss, restoring colon length, reducing spleen weight, and improving the DAI score and histological lesions. Additionally, imatinib reduced the level of proinflammatory cytokines, including TNF- α , IL-6, and IL-1 β . Furthermore, imatinib treatment restored tight-junction integrity and decreased the expression of apoptosis marker proteins.

Conclusion: Overall, imatinib treatment significantly alleviated the symptoms of DSS-induced colitis by influencing the expression of proinflammatory cytokines, tight junction proteins, and apoptotic markers in mice. These findings highlight imatinib as a potential therapeutic candidate for IBD.

*Corresponding author: Myoung Ok Kim

Keywords: Inflammatory bowel disease, Apoptosis, Imatinib, Inflammation, Tight junction

PS-R-04

Effect of Cholesterol-Rich Exosomes Derived from Fatty Liver During Progression of Age-Related Macular Degeneration

Ayun Seol, Jea Sic Jeong, Ji Eun Kim, Hee Jin Song, Su Jeong Lim, Su Ha Wang, Ye Ryeong Kim, Ye Eun Ryu, Beum-soo An, Dae Youn Hwang*

Department of Biomaterials Science (BK21 FOUR Program)/Life and Industry Convergence Research Institute, College of Natural Resources and Life Science, Pusan National University, Miryang, Korea

Age-related macular degeneration (AMD) is a primary cause of vision impairment, initially marked by the formation of drusen. Oxidized cholesterol plays a critical role in AMD development by inducing inflammation, cytotoxicity, and retinal cell death. Exosomes, which function as vital vehicles for intercellular communication, are capable of carrying cholesterol molecules. This study investigated the impact of cholesterol-enriched exosomes derived from fat-accumulated (steatosis) hepatocytes on the progression of AMD using ARPE-19 cells. An in vitro AMD model was created using ARPE-19 cells loaded with A2E and exposed to blue light, to assess changes in oxidative stress, inflammatory responses, apoptosis, and angiogenesis-related pathways. Compared to untreated controls, cells treated with cholesterol-laden exosomes exhibited a marked exacerbation of AMD-like symptoms, including elevated intracellular reactive oxygen species (ROS) and nitric oxide (NO) levels, along with decreased expression of superoxide dismutase (SOD). Additionally, Nrf2 phosphorylation was suppressed, while proteins involved in neovascularization were significantly upregulated. The exosomes further triggered the iNOS-COX-2 inflammatory signaling pathway, activated the NLRP3 inflammasome, and increased levels of pro-inflammatory cytokines and apoptotic markers. These cellular effects observed in ARPE-19 cells were also confirmed in BALB/c mice subjected to blue light-induced retinal injury. Overall, the findings demonstrate that cholesterol-rich exosomes from fatty liver potentiate AMD pathology by intensifying oxidative damage, inflammation, cell death, and abnormal blood vessel formation, suggesting their relevance as potential therapeutic targets in AMD treatment.

*Corresponding author: Dae Youn Hwang

Keywords: Age-related macular degeneration, Cholesterol, Exosome, Retina, Oxidative stress

PS-R-05

Binge Drinking Triggers VGLUT3-Mediated Glutamate Secretion and Subsequent Hepatic Inflammation by Activating mGluR5/NOX2 in Kupffer Cells

Kengmo Yang^{1,2}, Kyurae Kim¹, Tom Ryu¹, Young-Ri Shim¹, Hee-Hoon Kim¹, Sung Eun Choi¹, Kwang Woo Lee¹, Jehwi Jeon^{1,3}, Pilhan Kim¹, Young Seo Kim¹, Taeyun Ku¹, Haengdueng Jeong⁴, Ki Taek Nam⁴, Gyumin Lim⁵, Dong Wook Choi⁶, Seok-Hwan Kim⁶, Hyuk Soo Eun⁷, Won Kim^{8*}, Won-Il Jeong^{1,9*}

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Recently, lines of evidence have emerged that the intercellular crosstalk via glutamate signaling is involved in the development of alcoholic liver disease (ALD). However, the detailed role of hepatic glutamate in the pathogenesis of ALD is still unexplored. Here, we investigated the cumulative process of alcohol-induced glutamate vesicles in hepatocytes. Glutamate accumulates in vesicles around the central veins of hepatocytes via the vesicular glutamate transporter 3 (VGLUT3) protein, which is regulated by the NRF2-AHR signaling pathway in response to chronic ethanol exposure. Additional binge drinking alters intracellular Ca^{2+} levels and triggers glutamate exocytosis from hepatocytes. The released glutamate activates the metabotropic glutamate receptor 5 (mGluR5) on Kupffer cells (KCs), which subsequently induces reactive oxygen species (ROS) production through the NOX2 complex. Protein kinase C (PKC) plays a pivotal role in activating NOX2 after mGluR5 signaling. This interaction is facilitated by pseudosynapse formation between ballooned perivenous hepatocytes and adjacent KCs. Pharmacological inhibition of vesicular glutamate release and genetic ablation of Nox2 or Grm5 specifically in KCs both led to a marked reduction in hepatic inflammation. Analysis of patient samples confirmed a positive correlation between plasma glutamate concentration and hepatic VGLUT3 expression with inflammatory markers in alcoholic patients without cirrhosis. Taken together, our findings demonstrate that hepatic glutamate vesicle formation and release are key drivers of KC activation and ROS-mediated liver injury via spatially confined pseudosynaptic communication in alcoholic steatohepatitis (ASH).

*Corresponding author : Won Kim, Won-Il Jeong

Keywords : Steatosis, Alcohol-associated liver disease, Pseudosynapse, VGLUT3, Reactive oxygen species

PS-R-06

An Innovative Approach Using CRISPR-Ribonucleoprotein Packaged in Virus-Like Particles to Generate Genetically Engineered Mouse Models

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Genetically engineered mouse models (GEMMs) are essential tools for investigating disease mechanisms, validating therapeutic targets, and advancing fundamental biological research. Although CRISPR gene editing has dramatically simplified the creation of these models, the conventional method — microinjection of gene editing tools into zygotes — still requires specialized equipment and expertise, and current techniques continue to face technical challenges and efficiency limitations.

In this study, we introduce a CRISPR-VLP-induced targeted mutagenesis (CRISPR-VIM) strategy. This non-invasive method enables efficient genome editing without the need for microinjection, simply by co-culturing zygotes with virus-like particles (VLPs) delivering gene-editing ribonucleoproteins (RNPs), thereby minimizing physical stress and cellular damage.

We applied this approach to generate knockout and base-edited mouse models targeting *Plin1* and *Tyr*, and confirmed both somatic mutation and germline transmission. Furthermore, by integrating CRISPR-VIM with in vitro fertilization (IVF), we successfully produced GEMMs using a minimal number of donor animals, thereby further improving experimental efficiency. The method proved compatible with both cytosine and adenine base editors and we also demonstrated targeted knock-in capabilities.

Overall, CRISPR-VIM significantly lowers the technical barriers associated with GEMM production and eliminates the need for costly microinjection equipment. This approach provides a simplified and scalable platform that can accelerate the development of disease models and expand access to genome editing technologies, particularly in laboratories lacking advanced microinjection expertise.

*Corresponding author : Kyoungmi Kim, Je Kyung Seong

Keywords : Genetically engineered mouse models, Clustered Regularly Interspaced Short Palindromic Repeats, Virus-Like Particle, In Vitro Fertilization

PS-R-07

CX3CR1+ Macrophages Interact with Hepatic Stellate Cells to Promote Hepatocellular Carcinoma through CD8+ T Cell Suppression

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Background and Aims: Hepatic stellate cells (HSCs) contribute to hepatocellular carcinoma (HCC) progression by regulating multiple factors. However, the entire immunoregulatory functions of HSCs are still obscure. Here, we aim to investigate whether HSCs impose CX3CR1+ macrophages to pro-tumorigenic properties in the peritumoral area.

Approach and Results: In single cell RNA-sequencing analysis of HCC patients, a subpopulation of macrophages specifically expressed *Arg1* and *Cx3cr1* in the peritumoral area, and were highly enriched with retinol metabolism-related genes. Flow cytometry analysis showed significantly increased frequencies of CD14+CD11b+HLA-DR+ macrophages with *CX3CR1* in the HCC adjacent region where activated HSCs (aHSCs) mainly expressed CX3CL1. Accordingly, in tumor-bearing mice, *Cx3cl1* mRNA expression was notably increased in aHSCs within the adjacent HCC, where infiltration of Ly6C+ macrophages was mostly observed with decreased CD8+ T cells. In adoptive transfer and *in vitro* co-culture of myeloid cells, we demonstrated that CX3CR1+Ly6C+ cells migrated and highly expressed arginase-1 by interacting with retinoid-enriched aHSCs in the adjacent HCC. Direct treatment of retinoids or co-culturing with retinol-storing mouse aHSCs or human LX-2 cells significantly increased arginase-1 expression in Ly6C+ myeloid cells and human blood CD14+ cells, leading to the suppression of CD8+ T cell proliferation. Moreover, genetic deficiency of CX3CR1 in myeloid cells or pharmacological inhibition of retinol metabolism remarkably attenuated HCC development.

Conclusion: We showed that CX3CR1+Ly6C+ myeloid cells migrate and interact with aHSCs in the peritumoral region where retinoids induce arginase-1 expression in Ly6C+ macrophages, subsequently depriving CD8+ T cells of arginine and promoting HCC.

***Corresponding author:** Jin-Seok Byun, Hyuk Soo Eun, Won-Il Jeong

Keywords: Tumor-associated macrophage, CX3CL1, Arginase-1, Fibrosis, Retinoic acid

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포스터 초록 POSTER PRESENTATION

Poster Presentation 1

July 24 (Thu) 13:20~14:20
ICC JEJU Lobby 3F (제주 국제컨벤션센터 3층 로비)

PS-A-01~26 (해부생리)

PS-B-01~44 (독성병리)

PS-C-01~10 (미생물)

PS-D-01~26 (유전자질환모델)

PS-E-01~24 (시설운영 및 기타)

Poster Presentation 2

July 25 (Fri) 13:00~14:00
ICC JEJU Lobby 3F (제주 국제컨벤션센터 3층 로비)

PS-A-27~49 (해부생리)

PS-B-45~88 (독성병리)

PS-C-11~20 (미생물)

PS-D-27~51 (유전자질환모델)

PS-E-25~47 (시설운영 및 기타)



KOREAN ASSOCIATION
FOR LABORATORY ANIMAL SCIENCE

포스터 초록 (Poster Presentation) 안내

1. 포스터 발표 안내

발표시간	Poster Presentation 1	Poster Presentation 2
	July 24 (Thu) 13:20~14:20	July 25 (Fri) 13:00~14:00
발표장소	ICC JEJU Lobby 3F (제주 국제컨벤션센터 3층 로비)	
포스터 번호	PS-A-01~26 (해부생리) PS-B-01~44 (독성병리) PS-C-01~10 (미생물) PS-D-01~26 (유전자질환모델) PS-E-01~24 (시설운영 및 기타)	PS-A-27~49 (해부생리) PS-B-45~88 (독성병리) PS-C-11~20 (미생물) PS-D-27~51 (유전자질환모델) PS-E-25~47 (시설운영 및 기타)
	총130개	총125개
부착 시간	July 24 (Thu) 09:00~11:00	July 25 (Fri) 09:00~11:00
철거 시간	July 24 (Thu) 17:00~17:30	July 25 (Fri) 17:00~17:30

※ 철거 시간 이후의 포스터 분실은 책임지지 않습니다.

2. 포스터 심사 및 시상

- 포스터 발표는 좌장의 진행에 따라 포스터당 4분 (3분 발표, 1분 질의응답)으로 진행되며, 과학적 성과와 발표자의 발표력 등을 기준으로 우수포스터를 선정하여 시상하오니 반드시 발표시간에 포스터 앞에 대기하여 주시기 바랍니다.
(해당 시간에 발표자가 없는 포스터는 우수 포스터상 심사 대상에서 제외)
- 포스터 보드에 2회 이상(개수로 적용) 미부착 시, 교신저자(연구책임자)에게 향후 1년간 포스터 제출 불가의 제재가 주어 집니다.
- 우수포스터상 시상: July 25 (Fri) 16:00 ~ 16:40 / Hall A (Closing Ceremony)
- 우수포스터의 경우 폐회식에서 선정자를 호명합니다. 호명 시 자리에 없으면 다음 우수자에게 상이 수여되오니, 학술대회 종료일까지 학술대회에 꼭 참석해 주시기 바랍니다. (상장과 상금 수여, 대리수상불가)

3. 포스터 작성안내

- Poster board의 크기는 95cm(가로) X 210cm(세로)이며, 특히 제목이 가로 너비를 초과하지 않도록 준비하여야 합니다.
- 모든 포스터는 지정된 날짜, 기간 동안에 지정된 board에 부착하여야 합니다.
- 포스터 내용은 abstract, purpose, results(figures and tables), conclusions, references의 순서로 작성합니다.
- 전방 2m 위치에서 쉽게 읽을 수 있도록 굵고 명확한 글씨체를 이용하여 제작합니다.
- 모든 포스터의 부착 및 철거는 당일 지정된 시간에 발표자가 수행해주시기 바랍니다.

Anatomy / Psysiology

Poster no.	Title	Speaker
PS-A-01	Primary ovarian leiomyosarcoma in a dog	Gye-Hyeong Woo
PS-A-02	Xanthogranulomatous metritis in a Korean short-haired cat	Gye-Hyeong Woo
PS-A-03	Establishment of an advanced mouse depression model via sustained release of corticosterone using thermo-responsive hydrogels	Hoon-Seong Choi
PS-A-04	Intravital Microscopy-Based Characterization of a PTZ-Induced Seizure Model Using Thy1-GCaMP Mice	Sowon Lee
PS-A-05	Anti-inflammatory of immature Melon (<i>Cucumis melo</i> L.) extract on paw edema in Carrageenan-induced mice	Jeong Min Kim
PS-A-06	Mouse models reflecting type 2 and non-type 2 inflammation to explore asthma immunological diversity	Yeeun Bak
PS-A-07	Antioxidant properties of Melatonin and related Indoleamines secreted from the Pineal Gland	Yeonje Park
PS-A-08	Protective effects of <i>Curcuma longa</i> L. leaves and pseudostem extract on IgE/BSA-induced mast cell activation and DNCB-induced atopic dermatitis in a mouse model	Arachchige Maheshika Kumari Jayasinghe
PS-A-09	Anti-inflammatory effects of <i>Saccharina japonica</i> fucoidan on particulate matter-stimulated skin cells and TPA-induced ear edema in mice	Kirinde Gedara Isuru Sandanuwan Kirindage
PS-A-10	From Gene Expression to Membrane Protein Trafficking: Cellular Responses in MASLD	GERARDO LUIZ SISON
PS-A-11	Distinct Retinal Pathologies in Model Animal of Neuromyelitis Optica Spectrum Disorder	Wen Li Leow
PS-A-12	Structural and functional characteristics of local immune memory formation in SARS-CoV-2-infected cynomolgus macaques	Dong-Yeon Kim
PS-A-13	Branching morphology of the aortic arch and brachiocephalic trunk in cats	Young-Jin Jang
PS-A-14	<i>Ledum palustre</i> var. <i>diversipilosum</i> ameliorates DNFB-induced allergic contact dermatitis (ACD) by modulating the NF- κ B /NLRP3 pathway	Ji-Eun EOM
PS-A-15	Puriton attenuates the asthma severity in ovalbumin-induced murine model via balancing Th1/Th2 and inhibiting inflammation	Dae-Hun Park
PS-A-16	A custom imaging-guided intranasal system for brain delivery in a cynomolgus monkey (<i>Macaca fascicularis</i>)	Hyeon-Gu Yeo
PS-A-17	Quantitative analysis of region-specific microglia-neuron structural interactions in the ischemic core and penumbra following ischemic brain injury	Dong-Ju Park
PS-A-18	ATP-driven pyroptotic cell death in intestinal mucosal immune system in colitis	Sihyun Jeong
PS-A-19	TXNIP regulated endothelial cells senescence drives liver regeneration	Ga-Yeon Yu
PS-A-20	Cannabinoid receptor type 1 regulates aging-associated intestinal barrier dysfunction: Evidence from a rat model and in vitro studies	Yunna Lee
PS-A-21	Targeted inhibition of TLRs modulates inflammation and enhances efferocytosis in an autoimmune multiple sclerosis model	Yejin Lee
PS-A-22	Korean red ginseng extract powder mitigates fasting and postprandial hyperglycemia in type 2 diabetic Mice	Bum Suk Kim
PS-A-23	A Yeast-Derived Aldehyde-Reducing Compound attenuates oxidative and aldehyde stress across evolutionarily conserved Alzheimer's disease models	SUN SHIN YI
PS-A-24	<i>Agrimonia pilosa</i> extract alleviates CDAHFD-Induced non-alcoholic steatohepatitis and fibrosis in mice	Min-Jeong Jo
PS-A-25	Analysis of fibroblast subtypes specific to postpartum uterine regeneration	Byeongseok Kim

PS-A-26	Rapid Single-Step Aqueous Tissue Clearing for Volumetric Imaging and Fine Neural Detail Visualization in the Mouse Brain	Youngjae Ryu
PS-A-27	High Sucrose Intake Aggravates Airway Hyperresponsiveness and Promotes M1 Macrophage-Mediated Th1/Th17 Cell-Driven Inflammation in Adipose and Intestinal Tissues of OVA-Sensitized C57BL/6 Mice	Manathunge Kumudu Sulakshani Thilakarathna
PS-A-28	Microarray profiling of splenic RNA reveals immune gene modulation following long-term intake of Jeju groundwater	Prabhavi Dayaddra Dassanayake Gameralalage
PS-A-29	Medium-chain inulin modulates immune responses and attenuates experimental colitis	Minhyeok Kang
PS-A-30	TLR4 deficiency alleviates airway inflammation in PM-exacerbated allergic asthma mice by regulating macrophage polarization and Th17 differentiation	Jiwon Yang
PS-A-31	Evaluation of targeted drug delivery and therapeutic efficacy of Glucose-Albumin/Apigenin nanocomplex in Idiopathic pulmonary fibrosis model	Hyeri Chae
PS-A-32	Electroceutical Treatment Reduces Acute Renal Injury by Suppressing Osteopontin-Driven Inflammatory Responses in Rats	Meejung Ahn
PS-A-33	Protocatechuic acid and syringin improve dyslipidemia-induced cardiac damage in mice	Myeongguk Jeong
PS-A-34	Non-invasive detection of hepatic fibrosis using 68Ga-FAPI-46 PET imaging	Yeeun Kim
PS-A-35	Longitudinal Neuroimaging and Neuropathological Evaluation of Autologous Blood-Induced Cortical Hemorrhage in Miniature Pigs	Lee Wha Gwon
PS-A-36	Estrogen-induced expression of Pglyrp1 in the mouse endometrium	Ji Min Lee
PS-A-37	Adenine-induced chronic kidney disease (CKD) model as a platform to reveal the role of trained immunity in sustained systemic inflammation	Seong Ju Lee
PS-A-38	Establishment and Efficacy Evaluation of an Ischemic Stroke Model in Rodents	Na-Hye Park
PS-A-39	Anti-tumor activity of 5-HT2A antagonist 15f on colorectal cancer in vitro and in vivo	Jun Young Choi
PS-A-40	Optimized Flow Cytometry and Functional Assays for Immunophenotyping Common Marmoset Peripheral Blood Cells: Seoul National University Hospital - Marmoset Model Resource Bank (SNUH-MMRB)	Jiyeon Jang
PS-A-41	Comparative Evaluation of Water-Bath Sonication and Pipetting for Dispersing Aggregated Extracellular Vesicles	Hui jae Cho
PS-A-42	Identification of proteins regulated by baicalin in an ischemic stroke animal model	Sihoon Moon
PS-A-43	Enhanced Wound Healing in Rat Anastomotic Leakage and Skin Excision Models Using Gallol-Modified Chitosan Hydrogel Patches	Da Han Hyun
PS-A-44	High fructose Induced Adipose Inflammation Promotes Systemic Cytokine Crosstalk and Exacerbates Allergic Lung Responses in OVA-Sensitized C57BL/6 Mice	Prabhavi Dayaddra Dassanayake Gameralalage
PS-A-45	In Vivo Evaluation of Double-Layer CHI-G/cHA Adhesive Patch for Preventing Anastomotic Leakage and Postoperative Adhesions in a Rat Model	Da Han Hyun
PS-A-46	Nasal epithelial zonation and anatomical features in Infant Marmosets	Gyeong min Yoon
PS-A-47	Behavioral analysis of Low-Intensity focused Ultrasound Therapy in a Traumatic Brain Injury Mouse Model	Seungah Oh
PS-A-48	Philadelphus schrenkii ameliorates NLRP3 inflammasome-induced inflammatory bowel disease in mice	Dahyun Ko
PS-A-49	Yeokwisan extracts inhibits LPS-induced inflammation in RAW 264.7 macrophages and Helicobacter felis infection in mice	Ga-Young Lee

Toxicology / Pathology

Poster no.	Title	Speaker
PS-B-01	The anti-allergic effects of <i>Corchorus olitorius</i> in mice	Kiman Lee
PS-B-02	Anticancer effect of vinblastine is associated with counter-regulation of interleukin-24 expression by YAP in human melanoma cells	Eun-Yi Moon
PS-B-03	Potential of Aloe vera in ameliorating Inflammatory Bowel Disease via tight junction protection	Ji Woong Heo
PS-B-04	Anti-inflammatory and anti-tumor effects of <i>Gastrodia elata</i> Blume in an AOM/DSS-induced colitis-associated cancer model	Da Eun Jung
PS-B-05	<i>Gastrodia elata</i> Blume enhances immune function in a Cyclophosphamide-induced immunosuppressed BALB/c mouse model	Da Eun Jung
PS-B-06	Administration of <i>Gastrodia elata</i> Blume extract attenuated liver fibrosis in a Thioacetamide-Induced Rat Model	Ji won Seo
PS-B-07	<i>Cnidium officinale</i> Hot water Extracts Improve the Intestinal Barrier Dysfunction via Tight Junction Protection in IBD	Ye Jin Yang
PS-B-08	Therapeutic potential of <i>cinnamomum cassia</i> ethanol extracts in preserving intestinal barrier integrity: evidence from in vitro and in vivo models of colitis	Han Nim Choi
PS-B-09	Optimization Study of an Atopic Dermatitis Model Induced by MC903 in C57BL/6 Mice: Comparison of Inflammatory Markers and Application Sites	Sokho Kim
PS-B-10	The therapeutic effects of human neural stem cells overexpressing neuroglobin in APPswe/PS1dE9 mice	Ji Su An
PS-B-11	Optimization Study of MPTP-Induced Parkinson's Disease Mouse Model: Effects of MPTP Dosage and Exposure Duration	Sokho Kim
PS-B-12	Vapor self-administration reveals the efficacy of a vaccine against synthetic cannabinoid reinforcement in mice	Byoung Mo Choi
PS-B-13	Pathological role of cleaved crystallin alpha B in mediating angiogenesis and fibrosis in age-related macular degeneration	Eunhye Yu
PS-B-14	Modeling prodromal Alzheimer's disease: Longitudinal behavioral and activity monitoring in amyloid-induced non-human primates	Yu Gyeong Kim
PS-B-15	The Abuse liability of Cigarette Smoke Condensate in Rodents	Seong Shoon Yoon
PS-B-16	Effects of acetaminophen on cholinergic system and muscle contraction	Jiwon Jeong
PS-B-17	The anti-inflammatory and anti-oxidant effects of <i>Caesalpinia sappan</i> L. heartwood on an LPS-induced mouse model and RAW264.7 cells	Ji-Hye Ha
PS-B-18	<i>Boehmeria nivea</i> mitigated OVA-induced allergic asthma by suppressing Th2-mediated immune responses through the inhibition of Th2 cell differentiation	Ba-Wool Lee
PS-B-19	SARS-CoV-2 pseudovirus dysregulates hematopoiesis and induces inflammaging of hematopoietic stem and progenitor cells	Dong-hoon Chae
PS-B-20	Automated characterization of cocaine-induced behavior in marmosets using deep learning	ChulKyu Lee
PS-B-21	Pretreatment with 2-arachidonoylglycerol attenuates cocaine-taking behavior by modulating dopamine transporter and GPR55 availability in common marmosets	Sun Mi Gu
PS-B-22	E-merging evidence: Activation of $\alpha 4\beta 2$ nicotinic acetylcholine receptors by synthetic nicotine analogs in e-cigarettes	DANIEL CHAVEZ VARIAS
PS-B-23	Fibrotic Liver-Derived Organoids Provide a Physiologically Relevant Model of Liver Fibrosis	Jiyoung Heo
PS-B-24	Hepatic B cells aggravate liver fibrosis through interaction with NKT cells via CD40	Jihyo Byun

PS-B-25	Large-scale profiling of coding and long noncoding transcriptomes in the hippocampus of mice acutely exposed to vaporized CBD or THC	Jihun Kim
PS-B-26	Menthol Enhances Vaporized Nicotine Self-Administration in Mice	Yong-Qing Zhang
PS-B-27	Reprogramming Tumor-Associated Macrophages with Sulfanilamidoindazole (SAI) for Antitumor Immunity	Yeon Ju Ryu
PS-B-28	Potential role of T cells and IL-1 in COVID-19 mRNA vaccine-induced heart injury	Sumin Cho
PS-B-29	Effect of aging on mRNA vaccine-induced hepatotoxicity in mice	Eun-Bin Choi
PS-B-30	Effect of nicotinamide on nicotine dependence liability in mice	Gyebin Lee
PS-B-31	RAGE inhibition attenuates MMP2-mediated alveolar damage and enhances alveolar epithelial cell proliferation in emphysema	Jimin Jang
PS-B-32	The role of Cathepsin K inhibition in respirable crystalline silica-induced silicosis through epithelial-macrophage interactions	Dahee Han
PS-B-33	Abuse liability of CUMYL-5F-P7AICA in a nose-poke operant animal model	Sangmin Lee
PS-B-34	ISO 10993-11-based evaluation of acute systemic toxicity of cultured meat components in a rat model	Jun-Yeong Lee
PS-B-35	Development and in vivo evaluation of medical fluorophore 33 as a theranostic agent for colorectal cancer imaging and therapy in mouse models	Kwang Hee Son
PS-B-36	Key role of acrolein as an odor biomarker during 1,2,3-trichloropropane-induced hepatotoxicity of SD rats	Ye Eun Ryu
PS-B-37	Prolonged antioxidant supplementation enhances locomotor performance but compromises survival in aged <i>Drosophila melanogaster</i>	Xin Yi Yeo
PS-B-38	Effect of Gossypetin from <i>Hibiscus sabdariffa</i> on Oral Cancer	Jiaxi Zhang
PS-B-39	Anticancer Effects of Imatinib in Oral Squamous Cell Carcinoma via PI3K/AKT/mTOR Signaling Pathway	Nangwon Yee
PS-B-40	Gossypetin reduces colitis via regulating COX2 and ROS-JNK axis	Ke Huang
PS-B-41	Effects of PM2.5 associated metal components from pig house exposure on the male reproductive system in mice	Chae Rim Kim
PS-B-42	Silibinin alleviates Vanadium-induced lung Injury via the TLR4/MAPK/NF- κ B pathway in mice	Geun Hye Park
PS-B-43	Rhein-Induced AKT/mTOR Suppression Drives ROS-Mediated Apoptosis in Oral Cancer In Vitro and In Vivo	Yu Rim Cho
PS-B-44	Mixed toxicity evaluation study of Chromium(VI) Oxide and Molybdenum Oxide using Zebrafish Embryo	Ju Young Song, Hwi Won Jo
PS-B-45	Inhibition of IRF1 attenuates inflammation and dysregulated autophagy in acute lung injury mice	Jooyeon Lee
PS-B-46	Inhalation effects of polystyrene microplastics on colonic disease of ICR mice	Su Jeong Lim
PS-B-47	Mitigation of particle overload toxicity induced by ultrafine particles	So Young An
PS-B-48	Platelet promote phenotypic variation of U251 human glioblastoma cells	Seo Won Mun
PS-B-49	Modeling drug-Induced phototoxicity using a vascularized skin assembloid derived from human iPSCs	Wonchae Lee
PS-B-50	Influence of biotransformation on pulmonary clearance of iron oxide nanoparticles following 28-day repeated inhalation exposure	Songyeon Kim
PS-B-51	Real-time in vivo imaging demonstrates hippocampal calcium dynamics induced by bicuculline administration in voluntarily running mice	Hyeokjun Kwon
PS-B-52	Evaluation of the protective effects of <i>Loranthus tanakae</i> Franch. and Sav. on atopic dermatitis in ovalbumin-induced mice and TNF- α /IFN- γ -stimulated HaCaT cells	Ba Reun Jin
PS-B-53	Effects of respiratory exposure to 1,2-Dichloroethylene on female reproductive function	Changsic Youn

POSTER PRESENTATION

PS-B-54	Acute Respiratory Toxicity of Non-Exhaust Brake Wear Particles in Mice	Gyuri Kim
PS-B-55	Immunoregulatory effects of Choerospondias axillaris (Roxb.) B.L.Burt & A.W.Hill fruit extract on mice	Yong Heo
PS-B-56	Functional Disruption of Natural Killer Cells by ZnO, rGO, and CB Nanomaterials under Repeat Exposure	Seo-gyeong Jo
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PS-B-81	Luteolin induces caspase-dependent apoptosis and LC3-II-mediated autophagy in human oral cancer cells	HeeJin Jang
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PS-C-03	Cyclopeptides from gut commensal Phocaeicola vulgatus attenuate muscle atrophy in an an vitro model of pancreatic cancer cachexia	Bo-Young Shin
PS-C-04	Synergistic effect of probiotics and omega-3 fatty acids on blood and liver function biomarkers and gut microbiota diversity in high fat diet-induced obesity	Tae-Jun Kwon
PS-C-05	Bacterial reverse mutation assay(Ames Test) of 2-(Amicomethyl)piperidine	Kyeong Min Shin
PS-C-06	Prospective model for Monkeypox disease in common marmoset (Callithrix jacchus)	Chaewon Kwak
PS-C-07	Establishment of a Nipah Pseudovirus Animal Model for Vaccine Evaluation Under BSL-2	In-Ohk Ouh
PS-C-08	Establishment of a Humanized Mouse Model Expressing MXRA8 Receptor for Chikungunya Virus Vaccine Development	Eun-Young Jang
PS-C-09	Safety assessment of Bojungikgitang: genotoxicity tests of water and ethanol extracts	Na young Jeung
PS-C-10	IRES-based single-stranded RNA adjuvants encoding co-stimulatory molecules enhances T cell stimulatory effects and led to tumor regression	Yun Ji Kim
PS-C-11	Current Status of Mouse Pathogen Standard Cooperation Center in 2025	Na-Won Kim
PS-C-12	Intestinal Organoids Reveal the Anti-Inflammatory and Gut-Protective Potential of Heat-Killed Limosilactobacillus reuteri in Mice and Weaning Piglets	Jae Han Park
PS-C-13	Strain-Specific Exercise Adaptations and Gut Microbiota Interactions in B6 and 129 Mice	Seung Yeon Oh
PS-C-14	Modeling Antibiotic-Induced Gut Dysbiosis in Common Marmosets Using Azithromycin Monotherapy	Ji-Eun Kim
PS-C-15	Design and evaluation of O/CATHAY topotype-specific FMDV vaccine candidates	Yerin Kim

PS-C-16	Management of master seed virus candidates for foot-and-mouth disease vaccine production	Mi-Kyeong Ko
PS-C-17	Foot-and-Mouth Disease Incidence in Asia from 2020 to 2024 (Pool 1 Region)	Ye-Ji Kim
PS-C-18	Immune response and protective efficacy of a bivalent intradermal Foot-and-Mouth Disease Vaccine in pigs	Dong-Wan Kim
PS-C-19	Development of NanoBiT-based virus-like particle assay for rapid screening of neutralizing antibodies against foot-and-mouth disease serotype A	Yeonrea Chae
PS-C-20	Development of an Attenuated FMDV Serotype A Strain via Codon-Pair Deoptimization	Seo-Yong Lee

Genetic Disease Model

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PS-D-02	Recovery of effector memory T cells and balance of regulatory T cell subtypes are considered important for immune tolerance in non-human primate combined kidney-bone marrow transplantation	Bongkum Choi
PS-D-03	Deficiency of peroxisomal bifunctional enzyme HSD17B4 causes dysregulation of primary cilia	Dong-Hyung Cho
PS-D-04	TIM-3 deficiency aggravates liver fibrosis by stimulating pro-inflammatory macrophages	Eunmi Lee
PS-D-05	Galectin-4 potentiates CD8+ T cell immunity by enhancing MHC-I expression on dendritic cells: Therapeutic implications for cancer	Hyeong-Rae Kim
PS-D-06	Glucose-6-Phosphate Dehydrogenase as Metabolic Gatekeeper of Brown Adipocyte Identity and Thermogenesis	Sanghun Lee
PS-D-07	Prostaglandin Signals from Brown Adipose Tissue Accelerate Kidney Cystogenesis in Mettl14 BAT cKO Mice	Hyung Chul Kim
PS-D-08	Contraceptive of the male common marmoset (<i>Callithrix jacchus</i>) using vasectomy	So-Min Lee
PS-D-09	Development of a Human CD3E Transgenic Mouse Model for Preclinical Evaluation of CD3 - targeting bispecific antibodies	Kyeong-Won Yoo
PS-D-10	Establishment and Comparative Analysis of Diabetic Sarcopenia Models in Young and Aged mice	Jihye Choi
PS-D-11	Neuroprotective Effects of Astragalus mongholicus and Scutellaria baicalensis Extracts in Ischemic Stroke: MRI-Based Infarct Reduction and Modulation of Inflammation and Pyroptosis	Ji-Yeon Suh
PS-D-12	Establishment of Osteoporotic Animal Model and Evaluation of Super Active Calcium Supplementation	Yuseong Jang
PS-D-13	Development of obesity mouse model and evaluation of fig vinegar supplementation	Hyeon Gi Paik
PS-D-14	Evaluation of antidiuretic effects and single-dose safety of cephalotocin	Seonmi Jo
PS-D-15	Noncanonical β -adrenergic receptor-G12/G13 pathway regulates lipid utilization and thermogenesis in brown adipocyte	Soyeon Kim
PS-D-16	A host genetic locus on chromosome 4 modulates Akkermansia abundance and microbial squalene metabolism in atherosclerosis	Myungsuk Kim
PS-D-17	Synergistic Neuroprotective Effects of Treadmill Exercise and Vitamin D on Cognition, Inflammation, and Mitochondrial Function in Healthy Mice	Yoonhwan Kim
PS-D-18	Exercise Attenuates Neuropathology and Enhances Cognitive Function in 3xTg-AD Mice via Anti-Inflammatory and Myelin-Repair Mechanisms	Taewan Kim

PS-D-19	Simple gene knockout by single gene-directed multiplex CRISPR-Cpf1	Yeon Ju Jung
PS-D-20	Peptide-based modulation of copper homeostasis: therapeutic insights into Wilson's disease and metal-induced organ damage	Seung-Hyun Jung
PS-D-21	Macrophage-driven immune remodeling in early tumorigenesis initiates resistance to androgen deprivation therapy (ADT) in prostate cancer	Gyu Mi Park
PS-D-22	Engineering porcine fibroblasts with CD39/CD73 knock-in at the B4GALNT2 locus for xenotransplantation	Sang Eun Kim
PS-D-23	KBN2202 Suppresses Adipose Tissue Expansion and Enhances GLP-1 Secretion in High-Fat Diet-Induced Obese Mice	Seok-Hwan Chang
PS-D-24	Phenotypical characterization for Alzheimer's disease in C57BL/6-Tg Kori mice with human mutant Presenilin 2 gene	Ye Ryeong Kim
PS-D-25	Therapeutic potential of targeting deubiquitinase to modulate mitochondrial function in pancreatic cancer	Jae Woong Jeong
PS-D-26	Transcriptomic profiling of genetic adaptation pathways in mice exposed to chronic heat stress	Jei Hyoung Park
PS-D-27	Anti-Angiogenic and Cancer Effects of a Bryopsis plumosa-Derived Peptide in Zebrafish	Jeiha Lee
PS-D-28	Therapeutic Potential of KBN2202 in Alzheimer's Disease Mouse Model through Modulation of Microglial Polarization and Phagocytosis	Jeong-Hyeon Heo
PS-D-29	The Generation of Humanized Liver TK-NOG Mouse for the study of HCV Infections	Jung Hyu Shin
PS-D-30	Effects of a Neuro Protective extract on Alleviating Scopolamine Induced Memory Deficits in C57BL/6 Mice via Enhancing Cholinergic Function	Hyeokjin Kwon
PS-D-31	Dynamic regulation of IL-36 α by estrogen controls uterine immune tolerance during implantation	Joohee Kim
PS-D-32	Immunohistochemical Verification of Genetically Engineered Pigs for Xenotransplantation	JI-JING YAN
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PS-D-34	Hepatocellular carcinoma associated with non-alcoholic fatty liver disease in Leptin KO/Kori mice	Soo-Won Yun
PS-D-35	Irradiation of Tumor Draining Lymph Nodes Undermines PD-1 Blockade by Disrupting Peripheral PD-1 ⁺ CD8 ⁺ T Cell Responses	Ka Yeon Kim
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PS-D-37	Expression of necroptosis effectors is influenced by diet in the mouse ovary	Eunbin Shin
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PS-D-39	Structural and Functional Evaluation of Anti-Cancer Peptides Using Zebrafish Embryo	Heabin Kim
PS-D-40	Regulatory Effects of Pentosan Polysulfate Sodium on Benign Prostatic Hyperplasia through Anti-inflammatory Mechanisms	Han-Jung Chae
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PS-D-42	Establishment and Characterization of a Twindemic Mouse Model Co-Infected with Influenza A and SARS-CoV-2 Virus	Han-Jung Chae
PS-D-43	Development of a House Dust Mite-Induced Lung Dysfunction Model for Evaluating the Protective Effects of Prunus mume Extract	Young Yoon
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PS-D-47	Somatic Nf2 Inactivation Induces Spontaneous Metastasis in a Mouse Model of Gastric Cancer	Na Yun Lee
PS-D-48	Beneficial effects of Lobeglitazone, a new PPAR- γ agonist, on atherosclerosis and valve inflammation	Shin Hee Park
PS-D-49	Kras A146T-Driven Gastric Tumoroid Model Exhibiting Metastasis in Immunocompetent Mice	Yu Jin Lee
PS-D-50	SARS-CoV-2 immunization augments tumor-specific T cell cytotoxicity via TCMO6 antigen mimicry	Jong-Wan Kwon
PS-D-51	KBN2201 ameliorates amyloid pathology, neuroinflammation, neuronal degeneration, and neurogenesis deficits in 9-month-old 5xFAD mice	Sun-Young Lee

Facility / Management / Others

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PS-E-02	White Ginseng ethanol extract inhibits tyrosinase and melanogenesis via MITF downregulation in B16F10 cells	Min Jung Kim
PS-E-03	Microcurrent wave mitigates mouse intracranial arterial dolichoectasia development	Dong Rak Kwon
PS-E-04	Small Extracellular Vesicles derived From Feline Adipose Tissue Mesenchymal Stem Cells Enhance The Effects of Anti-Inflammation Compared to Fibroblasts-derived Small Extracellular Vesicles	Min-Jung Ma, Young-Ju Lim
PS-E-05	The status of postapproval monitoring operation by the Institutional Animal Care and Use Committee in Korea	Jimin Lee
PS-E-06	Eradication of Aspiculuris tetraptera in Various Immunodeficient Mouse Models Using Ivermectin: A Case Report	Ji-Hun Lee
PS-E-07	Nanographene oxide attenuates acute GVHD by modulating macrophage polarization in a xenogeneic mouse model	Aaron Yu
PS-E-08	Extraction and Characterization of Polyphenols from Gynura procumbens (sambungcho) using HPLC-ESI-MS/MS	Sang Joon Lee
PS-E-09	Automated radiosynthesis and efficacy evaluation of tau target diagnostic PET tracer candidate, 2-(6-(2-(2-([¹⁸ F]-fluoro)ethoxy)ethoxy)-1H-pyrrolo [3,2-c]pyridin-1-yl)quinoline	Jae Hun Ahn
PS-E-10	Identification of new odor biomarkers associated with aging in aged ICR mice	Ji Eun Kim
PS-E-11	Enhanced oocyte yield in common marmoset assisted reproductive technology by switching from hMG to rhFSH	Hee Jin Choi
PS-E-12	Amide proton transfer CEST MRI for detecting neuroinflammation in a sepsis-associated encephalopathy animal model	Do-Wan Lee
PS-E-13	Mouse-Derived Cell Powder Technology Enhances Myogenic Differentiation and Alignment on Alginate-Zein Scaffold	Kyung-Mee Park, Jihad Kamel, Jun-Yeong Lee, Sadia Afrin, Usha Yadav, Chandra-Jit Yadav
PS-E-14	Bioengineered liver scaffolds functionalized with heparin improve endothelialization and attenuate liver fibrosis	Chandra Jit Yadav

PS-E-15	Sphingosine-1-Phosphate enhances endothelialization of rat decellularized liver scaffolds by promoting endothelial cell function	Usha Yadav
PS-E-16	Current Status of Animal Experimentation and IACUC Operations in Domestic Animal Experimentation Institutions in 2024 in South Korea	Soohee Leem
PS-E-17	Genipin and VEGF Synergize to Support Recellularization and Angiogenic Remodeling in Bioengineered Rat Liver Scaffolds	Kyung-Mee Park, Sadia Afrin, Jihad Kamel, Jun-Young Lee, Chandra Jit Yadav, Usha Yadav
PS-E-18	Compound X, a Resorcinol derivative, alleviates IgE-mediated allergic inflammation by suppressing mast cell activation	Seungwon Jeong
PS-E-19	Tumor-priming CD8 ⁺ NKT-like cell therapy eliminates tumor and immunosuppressive cells in gastric cancer peritoneal metastasis	Juheon Lee
PS-E-20	Effectiveness Evaluation using Animal Models for International Certification of Dental Medical Devices	Donghyun Lee
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PS-E-22	RNA Sequencing Analysis of Embryos Exposed to Nanoplastics: Impacts on Transcriptomic Alteration	Hyeong-ju You
PS-E-23	Lipolytic effects of edible vitalmelon extract in MDI-stimulated adipocytes through activation of glycerol release and β -oxidation pathways	Su Ha Wang
PS-E-24	A novel F-18 labeled pRIPK1-selective PET radiotracer for noninvasive imaging of necroptosis: utility in preclinical disease models	Sun Mi Park
PS-E-25	Evaluation of tumor-targeting and biodistribution of tecentriq using in-vivo near-infrared fluorescence imaging in xenograft models	Jung Ae Kang, Ji-Hye Lee
PS-E-26	Establishment of a Long-Term Mouse Model of Non-Alcoholic Steatohepatitis Induced by High-Fat Diet and Western diet	Ji-Hye Lee
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PS-E-33	Prediction of Social Hierarchy Based on Individual Aggression Assessment in Cynomolgus Macaques	Sang-Hee Lee
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PS-E-35	National primate infrastructure for biomedical and basic science	Se-Hee Choe, Min-Gyeong Ko
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PS-E-44	The receptor for cacao polyphenol procyanidin C1 and its anti-melanoma effects	Jihye Lee
PS-E-45	Evaluation of the Skin-Whitening Effects of Cotton Bark Extract via α -MSH-Stimulated Melanogenesis in B16F10 Cells	Jihye Lee
PS-E-46	Sample Integrity First: A Roadmap for Error-Free Liver Function Testing in Laboratory Animals	Yong Lim
PS-E-47	Effective Hand-Rearing Protocol for Normal Growth in Triplets Neonate Common Marmosets in Seoul National University Hospital Marmoset Model Resource Bank (SNUH-MMRB)	Do Been Choi

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PS-A-01

Primary ovarian leiomyosarcoma in a dog

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Primary tumors arising from the ovary are rare in animals. In dogs, ovarian tumors occur in about 0.5-1.2% of cases. The most commonly reported ovarian tumors are ovarian carcinomas, granulosa-theca cell tumors, and teratomas. However, ovarian stromal tumors have been reported very rarely. Fibroadenomas and leiomyomas are known as ovarian stromal tumours. Leiomyomas and leiomyosarcoma are tumors derived from smooth muscles that occur in various organs. Leiomyomas have been reported to occur in the ovary, although rarely, and have been reported in pigs, horses, dogs, leopards, and tiger among animals. However, leiomyosarcoma, a malignant tumor derived from the ovary, has not been reported in animals other than humans and guinea pigs. In humans, ovarian leiomyosarcoma is known to occur very rarely, accounting for less than 1% of ovarian malignant tumors. In this case, we report the first case of leiomyosarcoma arising from the ovary in a dog based on histopathologic and immunohistochemical findings. A Bichon Frise, approximately 7 years old, was admitted to a local hospital for sterilization surgery. During the surgery, the right ovary was found to be more than three times larger than the left ovary, so the uterus and ovary were referred for diagnosis. No unusual findings were observed in the left ovary macroscopically, but the right ovary was 2.5x1.8cm in size, had a somewhat bumpy surface, and was mostly occupied by a whitish mass. The uterus was somewhat erythematous, and a very small amount of viscous, colorless, transparent secretion was present in the lumen. The requested tissue was fixed in 10% neutral buffered formalin, and after tissue processing, paraffin-embedded, and the paraffin block was cut into 3-4 micrometer sections. For pathological evaluation, the paraffin sections were stained with hematoxylin and eosin (HE). Additional sections were subjected to immunohistochemical staining. Briefly, vimentin (1:100, monoclonal mouse, clone V9, M0725; Dako, Denmark), desmin (1:100, monoclonal mouse, M0760; Dako, Denmark), and smooth muscle actin (SMA, 1:100, monoclonal mouse, M0851; Dako, Denmark) antibodies were used to identify the cellular components in the ovarian mass of dog. Histopathologically, the ovary was surrounded by a single layer of cuboidal epithelium, mostly replaced by tumor cells, and a tumor mass with some degree of invasiveness was observed. The tumor cells were spindle-shaped, proliferating in a bundle-like pattern, and the cell density was high. The boundaries between the tumor cells were unclear, and the tumor cells had a small to moderate amount of eosinophilic cytoplasm. The nucleus was cigar-shaped and elongated, with blunt ends. There were 1-3 nucleoli in the center. The nuclear/cytoplasmic ratio of tumor cells was slightly increased, anisokaryosis was mild or moderate, and no more than two mitotic figures were observed in a single field of view at 400x magnification. More than eight mitotic figures were observed in 10 fields of view at 400x magnification. No tumor emboli were observed in blood vessels or lymphatic vessels. Multifocal hemorrhages and necrosis were also observed within the tumor mass. Immunostaining results for smooth muscle actin, desmin, and vimentin showed positive reactions in tumor cells. Based on above these findings, we report for the first a leiomyosarcoma arising from the ovary of a dog.

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Keywords : Ovarian leiomyosarcoma, Dog, Immunohistochemistry

PS-A-02

Xanthogranulomatous metritis in a Korean short-haired cat

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Xanthogranulomatous inflammation is a rare, chronic, non-neoplastic lesion in animals. Although uncommon in humans, it is a recognized condition that can affect various organs. This inflammation has been reported in multiple animal species, including dogs, cats, and horses. It has been described in the middle ear and small intestine of dogs, the choroid plexus of horses, and the lungs and uterus of cats. Cholesterol granulomas arising in the feline uterus have been characterized, with emphasis on their histopathological features. Here, we report one such rare case, detailing both the histopathological and immunohistochemical findings. A 5-year-old Korean short-haired cat was referred to a local veterinary hospital due to mild vaginal discharge and loss of appetite. The cat had been living on the streets, and no additional health information was available, except for generally poor looking appearance. Ultrasound examination revealed a uterine nodule, prompting an ovariohysterectomy for diagnostic purposes. Macroscopically, a white to yellowish-white nodule, measuring 1 × 2 cm, was observed in one uterine horn (Fig. 1A). The nodule had a distinct border and extended from the endometrium into the myometrium. The uterine cavity contained a large amount of colorless, viscous fluid (Fig. 1B). Uterine tissue was fixed with 10% neutral-buffered formalin, processed, and embedded in paraffin. Paraffin sections were stained with hematoxylin and eosin, Grocott's methenamine silver, Ziehl-Neelsen, Giemsa, and Gram's stains. Additionally, immunohistochemistry was performed using Iba-1 (1:1,000, polyclonal rabbit, 019-19741; Wako, Osaka, Japan) and major histocompatibility complex (MHC) class II (1:40, monoclonal mouse, M0746; Dako, Glostrup, Denmark) as primary antibodies to identify the cellular components of the uterine nodule. Histopathological examination revealed inflammatory nodules in the endometrium, myometrium, and adjacent perimetrium. The uterine lumen was significantly dilated and contained a large amount of eosinophilic proteinaceous material, macrophages, and cellular debris. The inflammatory nodules primarily consisted of numerous macrophages with foamy cytoplasm, along with a smaller population of multinucleated giant cells and lymphocytes. Fibrosis and hemorrhage also were observed. Immunohistochemically, macrophages with foamy cytoplasm were strongly positive to both Iba-1 and MHC II. No infectious agents were present in the lesion or cytoplasm of the macrophages on appropriate special stains. Based on these findings, a diagnosis of xanthogranulomatous metritis in the feline uterus was diagnosed. Because xanthogranulomatous metritis could be clinically diagnosed as a neoplastic lesion, it is considered to have important obstetric value in the differential diagnosis.

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Keywords : Xanthogranulomatous metritis, Cat, Immunohistochemistry

PS-A-03

Establishment of an advanced mouse depression model via sustained release of corticosterone using thermo-responsive hydrogels

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Depression is the most prevalent psychiatric disorder that affects millions worldwide, significantly impairing life quality of patients. As the significance of depression is increasing, the development of reliable preclinical models is essential for elucidating pathophysiological mechanisms and advancing therapeutic strategies. Chronic stress animal models, which mimic stress-induced depressive episodes in patients, are commonly established via repeated corticosterone administration, either by daily injection or dilution in drinking water for several weeks. However, these traditional methods are limited by excessive stress to animals, inconsistent plasma corticosterone levels, and imprecise dosing. In this study, we present an improved chronic stress-induced depression model using a thermo-responsive, bio-adaptable hydrogel for sustained corticosterone delivery. In vitro, the corticosterone-loaded hydrogel remained in a liquid state at room temperature (24 °C), and underwent liquid-gel transition at body temperature (37 °C), enabling gradual degradation and controlled corticosterone release. In vivo, two injections of corticosterone-loaded hydrogel (on days 1 and 10) resulted in significant body weight reduction and marked depressive-like behaviors. Moreover, glucocorticoid receptor expression in the hippocampal CA3 region and medial prefrontal cortex was significantly decreased at 24 days post-injection. Notably, hydrogel-treated animals exhibited persistently elevated plasma corticosterone levels compared to those receiving daily corticosterone injections. Collectively, this hydrogel-based corticosterone delivery approach more accurately recapitulates the clinical and pathophysiological features of depression than traditional methods, while minimizing animal distress during prolonged treatment. This model provides a robust platform for future mechanistic and therapeutic investigations in depression.

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Keywords : Depression, Animal model, Thermo-sensitive hydrogel, Corticosterone, Chronic stress

PS-A-05

Anti-inflammatory of immature Melon (Cucumis melo L.) extract on paw edema in Carrageenan-induced mice

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This study aimed to evaluate the anti-inflammatory effect of immature melon (Cucumis melo L.) extract (ICME) using a carrageenan-induced acute paw edema model in BALB/c mice. The extract was prepared via hot water reflux extraction (1:10, w/v) for 2.5 hours, concentrated 20-fold, freeze-dried, and administered orally at doses of 500 and 1000 mg/kg. Mice were grouped into Normal, Control (carrageenan only), ICME500, and ICME1000 (n=5 per group), and paw edema was induced by subcutaneous injection of 1% carrageenan. Paw volume was measured hourly for 4 hours and the percent inhibition of edema was calculated. ICME exhibited a dose dependent anti-edematous effect, with significant inhibition rates of 21.7% and 36.2% at 500 and 1000 mg/kg, respectively (p<0.05). ICME also reduced serum levels of pro-inflammatory cytokines (TNF-α and IL-1β) in the ICME-treated groups. In addition, ICME showed moderate antioxidant activity in the DPPH radical scavenging assay, with a scavenging rate of approximately 47% at 1 mg/mL, consistent with previous findings on C. melo extracts. Furthermore, in RAW 264.7 macrophages, ICME significantly suppressed lipopolysaccharide (LPS)-induced nitric oxide (NO) production in a dose dependent manner, suggesting its potential role in modulating inflammatory mediators. These results demonstrate that ICME possesses anti-inflammatory and antioxidant properties and may serve as a natural therapeutic agent for inflammatory conditions.

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Keywords : Cucumis melo L., Anti-inflammatory, Edema, Anti-oxidant, Cytokines

PS-A-04

Intravital Microscopy-Based Characterization of a PTZ-Induced Seizure Model Using Thy1-GCaMP Mice

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Accurate *in vivo* modeling of seizure-related neuronal activity is essential for advancing preclinical epilepsy research. Here, we established and characterized a PTZ-induced seizure model in Thy1-GCaMP mice using intravital microscopy (IVM), which enables real-time, high-resolution imaging of calcium dynamics in live brain tissue. Under baseline conditions, cortical neurons exhibited stable GCaMP fluorescence, confirming the suitability of Thy1-GCaMP mice for calcium imaging. Following intraperitoneal injection of pentylenetetrazole (PTZ), we observed a rapid and pronounced increase in neuronal calcium signaling, visualized as enhanced GCaMP fluorescence. This response reflects seizure-like hyperactivity and validates the model's physiological relevance. Our approach demonstrates that combining PTZ induction with IVM in Thy1-GCaMP mice provides a robust and reproducible platform for studying seizure mechanisms and evaluating therapeutic candidates *in vivo*.

*Corresponding author : Hyunseok Kim

Keywords : Thy1-GCaMP, Intravital microscopy, Preclinical research

PS-A-06

Mouse models reflecting type 2 and non-type 2 inflammation to explore asthma immunological diversity

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Asthma is a prevalent chronic respiratory disease with airway inflammation triggered by external stress and exhibits considerable heterogeneity based on an immunological pathway. Type 2 and non-type 2 asthma differ in immune cells profiles and treatment responsiveness. Type 2 asthma is typically associated with eosinophilic inflammation driven by Th2 responses, and generally effective to corticosteroids, but non-type 2 asthma is predominantly neutrophilic associated with Th1/Th17 responses, and it is often treatment resistant. To develop effective therapeutic strategies suitable for various situations, establishing mouse models that reflect various immune systems is essential. To establish distinct endotype/phenotype asthma models, three different adjuvants, aluminum hydroxide (Alum) for type 2 asthma, complete Freund's adjuvant (CFA) and lipopolysaccharides (LPS) for non-type 2 asthma, were sensitized with allergen, ovalbumin (OVA).

Immunopathological analysis indicated that the OVA/Alum showed classical type 2 inflammatory asthma phenotypes, including increased total/OVA-IgE, IL-5, IL-13, eosinophilic inflammation with mucus hypersecretion. In contrast, the OVA/CFA reflected mild non-type 2 inflammatory asthma phenotypes, by suppressing total/OVA-IgE, IL-5, IL-13, eosinophils and lung inflammation and increasing IFN-γ, neutrophil infiltration compared to OVA/Alum. Interestingly, the OVA/LPS, representing a more pronounced non-type 2 inflammatory asthma phenotypes, exhibited neutrophilic inflammation comparable to that of the OVA/Alum, but showed the least mucus secretion, in contrast to the moderate level observed in OVA/CFA and the most severe in OVA/Alum. Also, each model induced distinct T cell subsets in the lungs: the OVA/Alum upregulated GATA-3+ T cells, OVA/CFA enhanced FoxP3+ T cells, and OVA/LPS promoted RORγt+ T cells. Collectively, these results highlight the immunopathological diversity elicited by three distinct asthma models and underscored their utility as valuable experimental platforms for exploring complex heterogeneity of asthma.

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Keywords : Asthma, Immunopathology, Mouse modeling, Type 2 inflammatory asthma, Non-type 2 inflammatory asthma

PS-A-07

Antioxidant properties of Melatonin and related Indoleamines secreted from the Pineal Gland

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Melatonin, often referred to as the "night hormone", is synthesized by the pineal gland in response to darkness. Alongside melatonin, structurally related indoleamines such as N-acetylserotonin (NAS), serotonin, and N-acetyltryptamine (NAT) are also produced in the pineal gland. In this study, we established a mass spectrometry-based analysis method to quantify the secretion of these compounds from isolated rat pineal glands. Adrenergic stimulation increased the secretion of melatonin and NAS via both α - and β -adrenergic receptors, as confirmed using receptor-selective agonists. This effect was attenuated by the β -adrenergic antagonist propranolol in the presence of β -agonists. To further understand their biochemical roles, we evaluated the antioxidant and potential prooxidant properties of these compounds. Antioxidant activity was assessed using radical scavenging assays (DPPH, ABTS, and nitric oxide), ferric reducing antioxidant power (FRAP), and oxygen radical absorbance capacity (ORAC). Prooxidant potential was examined via intracellular ROS levels (DCFH-DA assay) and cell viability (MTT assay). All four compounds effectively scavenged ABTS radicals and nitric oxide, and significantly inhibited AAPH-induced fluorescein degradation in the ORAC assay. Interestingly, melatonin and NAT showed relatively weak activity in the DPPH and FRAP assays. These findings suggest that melatonin and related indolamines possess distinct antioxidant properties, potentially contributing to their physiological functions in the body.

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Keywords : Pineal gland, Melatonin, Adrenergic receptor, Antioxidant

PS-A-09

Anti-inflammatory effects of Saccharina japonica fucoidan on particulate matter-stimulated skin cells and TPA-induced ear edema in mice

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This study investigated the extraction, characterization, and anti-inflammatory effects of fucoidan derived from *Saccharina japonica* (SJF) in models of particulate matter (PM)-induced skin inflammation. Chemical analysis revealed that SJF contains 15.82 $\pm$ 0.07% sulfate and 29.82 $\pm$ 0.86% fucose. In PM-stimulated HaCaT keratinocytes, SJF treatment significantly enhanced cell viability by reducing reactive oxygen species (ROS) production. It also dose-dependently suppressed the expression of inflammatory cytokines including interleukins (IL-6, IL-8, IL-13, IL-25, IL-33), TNF- α , IFN- γ , and thymic stromal lymphopoietin (TSLP), and chemokines (MDC and TARC) via modulation of the NF- κ B/MAPK signaling pathways. Furthermore, conditioned media from SJF-treated HaCaT cells reduced inflammation and extracellular matrix (ECM) degradation in human dermal fibroblasts (HDFs), as evidenced by decreased expression of TSLP, IL-6, IL-8, IL-13, TNF- α , TARC, MDC, as well as matrix metalloproteinases (MMPs), tissue inhibitors of metalloproteinases (TIMPs), and elastase activity. In an in vivo model using BALB/c mice with 12-O-tetradecanoylphorbol-13-acetate (TPA)-induced ear edema, topical application of SJF significantly reduced ear thickness and downregulated the expression of inflammatory enzymes inducible nitric oxide synthase (iNOS) and cyclooxygenase-2 (COX-2). Overall, these findings demonstrate the potential of SJF to alleviate PM-induced skin inflammation, supported by both *in vitro* and *in vivo* evidence.

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Keywords : Saccharina japonica, Fucoidan, Inflammation, Cytokines

PS-A-08

Protective effects of Curcuma longa L. leaves and pseudostem extract on IgE/BSA-induced mast cell activation and DNCB-induced atopic dermatitis in a mouse model

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The perennial herbaceous plant, *Curcuma longa* L. (turmeric) is primarily grown and harvested for pharmacological purposes in China, Korea, and various tropical regions in South Asia. Importantly, various studies have confirmed the presence of numerous active compounds with health-enhancing biological properties in turmeric leaves and pseudostems. Therefore the present study investigated the effect of turmeric leaves and pseudostem extract (CLE) on type I allergic reactions in IgE/BSA-stimulated bone marrow-derived cultured mast cells (BMCs) and 1-chloro-2,4-dinitrobenzene (DNCB)-induced atopic dermatitis in BALB/c mice. The findings showed that CLE suppressed the degranulation of IgE/BSA-stimulated BMCs. CLE decreased the expression of Fc ϵ RI on the surface of BMCs and its IgE binding. CLE downregulated the production of the cytokines by modulating NF- κ B and Syk-LAT-ERK-Gab2 signaling. CLE effectively attenuated the IgE/BSA-induced PCA reaction in BALB/c mice. Besides, the oral administration of CLE improved AD-like symptoms, including skin severity score, transepidermal water loss, scratching frequency, and ear thickness in DNCB-induced AD mice. Additionally, serum levels of IgE, IgG1, and IgG2a were significantly reduced in CLE-treated mice. CLE decreased inflammatory cell infiltration and mast cell degranulation while downregulating mRNA expression levels of AD-related innate cytokines (thymic stromal lymphopoietin [TSLP], IL-25, IL-33), inflammatory cytokines (IL-4, IL-10, IL-13), and chemokines (thymus and activation-regulated chemokine [TARC], macrophage-derived chemokine [MDC]) in the dorsal skin of AD mice. Furthermore, CLE reduced spleen and lymph node enlargement and downregulated mRNA expression levels of inflammatory cytokines in these tissues in a dose-dependent manner. These results revealed the therapeutic potential of CLE against abnormal immune responses.

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Keywords : Allergy, Mast cells, Turmeric, Atopic dermatitis, Immune responses

PS-A-10

From Gene Expression to Membrane Protein Trafficking: Cellular Responses in MASLD

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Metabolic dysfunction-associated steatotic liver disease (MASLD) is characterized by excessive hepatic lipid accumulation and is the leading cause of chronic liver disease, affecting approximately 30% of the global population. To identify potential therapeutic targets involved in MASLD, we performed RNA-seq analysis on liver tissues from mice (*Mus musculus*) fed with either a normal diet (ND) or a high-fat diet (HFD) for 10, 20, 30, or 40 weeks. Differential gene expression analysis revealed significant alterations in pathways related to inflammation and fatty acid metabolism. Validation by RT-qPCR in 40-week HFD mouse liver samples confirmed the upregulation of several key genes, representing families such as aquaporin (*Aqp*), transient receptor potential vanilloid (*Trpv*), melatonin receptor (*Mtnr*), G protein-coupled receptor (*Gpr*), and the C-C motif chemokine receptor (*Ccr*). To further investigate their functional relevance, we modeled steatosis *in vitro* by treating human hepatoblastoma HepG2 cells with a 2:1 mixture of palmitate and oleate. Fluorescence microscopy revealed distinct trafficking patterns: AQP accumulated around lipid droplets, while TRPV localized more prominently to the plasma membrane. These differential localizations suggest that AQP and TRPV may play specialized roles in lipid accumulation and membrane remodeling during MASLD progression. Together, our findings provide insights into the subcellular dynamics of lipid accumulation and offer a valuable molecular resource for understanding the underlying mechanisms in MASLD progression.

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Keywords : Metabolic dysfunction-associated steatotic liver disease, RNA-seq analysis, Colocalization, Membrane proteins, Lipid accumulation

PS-A-11

Distinct Retinal Pathologies in Model Animal of Neuromyelitis Optica Spectrum Disorder

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Neuromyelitis optica spectrum disorder (NMOSD) is a severe autoimmune disease of the central nervous system (CNS) that primarily affects the optic nerves and spinal cord. It is driven by a humoral immune response against aquaporin-4 (AQP4), a water channel protein abundantly expressed on astrocytic end-feet. The binding of anti-AQP4 IgG (NMO-IgG) to AQP4 activates the classical complement cascade, resulting in complement-dependent cytotoxicity (CDC), astrocyte destruction, secondary demyelination, and neuronal loss. Clinically, NMOSD presents with recurrent optic neuritis, transverse myelitis, and often irreversible visual impairment. While CNS involvement is well characterized, retinal pathology in NMOSD remains underexplored. In this study, we aimed to investigate structural and molecular alterations in the retina using a mouse model of NMOSD. To achieve this, we induced NMOSD by intrathecal administration of patient-derived AQP4-IgG and human complement into mice with an EAE-induced inflammatory background. Retinal tissues were analyzed using hematoxylin and eosin (H&E) staining, immunohistochemistry, and real-time quantitative PCR (RT-qPCR). In NMOSD retinas, astrocyte-specific injury was evident, characterized by reduced glial fibrillary acidic protein (GFAP) and AQP4 expression. H&E staining revealed marked retinal thinning, particularly within the ganglion cell layer (GCL) and inner plexiform layer (IPL). Our immunolabeling experiments showed significant reductions in the IPL and outer plexiform layer (OPL) and loss of retinal ganglion cell (RGC). RT-qPCR revealed upregulation of pro-inflammatory cytokines (IL-1 β , TNF- α , CCL2, CXCL10) and downregulation of anti-inflammatory markers (TGF- β , IL-4, IL-10). These results show that our NMOSD model animals induce severe retinal pathology, suggesting the utility of our NMOSD model for understanding of NMOSD retinal pathogenesis and facilitating therapeutic development.

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Keywords: NMOSD Model Mice, Retinal Pathogenesis, Anti-AQP4 IgG, Demyelination, Inflammation

PS-A-13

Branching morphology of the aortic arch and brachiocephalic trunk in cats

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The aortic arch (AA) branching pattern exhibits species-specific anatomical variation. Although cats are commonly used as experimental animal models, studies focusing on the branching pattern of the AA and its arterial branches remain scarce. In this study, we aimed to anatomically characterize the branching pattern of the AA and the brachiocephalic trunk (BCT) in cats. A total of 35 cats (15 males and 20 females) were examined using silicone vascular casting. In all cases, the AA gave rise to the BCT and the left subclavian artery (LSB). The Pearson's correlation for the branching origins of the BCT and the LSB from the AA was strong ($r = 0.9878$, $p < 0.0001$), indicating a highly consistent anatomical relationship across individuals. The BCT branched into the left and right common carotid artery (LCC and RCC) separately in 88.6%, while a common carotid trunk was observed in 11.4%. The correlation between the origins of the LCC and RCC from the BCT was moderate ($r = 0.5392$, $p = 0.0008$), suggesting greater individual variability. To further classify this variation, we applied the ratio of the distance between the LCC and RCC to the total length of the BCT, termed RDCC. Based on RDCC values, four types of branching patterns were identified: type I (RDCC > 30), type II (15 < RDCC $\leq$ 30), type III (0 < RDCC $\leq$ 15), and type IV (RDCC = 0). Among these, type I was the most common (48.6%), indicating that separate branching of the LCC and RCC was the predominant configuration. Taken together, our findings suggest that the BCT and LSB show consistent branching from the AA, whereas the LCC and RCC display notable individual variation from the BCT. These findings highlight the distinctive branching patterns of the feline AA and its arterial branches, serving a valuable anatomical reference for experimental research.

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Keywords: Aortic arch, Brachiocephalic trunk, Branching pattern, Cats, Common carotid artery

PS-A-12

Structural and functional characteristics of local immune memory formation in SARS-CoV-2-infected cynomolgus macaques

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As SARS-CoV-2 transitions from a pandemic to an endemic phase, reinfections have become increasingly frequent. This study used a cynomolgus macaque model to assess immune memory cell formation following Omicron variant infection. Memory CD4⁺, CD8⁺, and B cells were detected both systemically and in the lungs, with significant activation of memory CD4⁺ T and B cells in the lungs and mediastinal lymph nodes (medLNs). Gene ontology (GO) analysis revealed the activation of germinal center (GC) B cells, as well as tissue-resident memory T (TRM) and B (BRM) cells. Spatial analysis showed inducible bronchus-associated lymphoid tissue (iBALT) formation, characterized by GC-like B cells, BRMs, CD4⁺ and CD8⁺ memory T cells. GO analysis indicated enrichment in pathways related to B and T cell activation, proliferation, and immunoglobulin production. These findings underscore the roles of medLNs and iBALT in localized immune defenses and immune memory formation against SARS-CoV-2.

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Keywords: SARS-CoV-2, Tissue resident memory cell, Local immune memory, iBALT, Mediastinal Lymph node

PS-A-14

Ledum palustre var. diversipilosum ameliorates DNFB-induced allergic contact dermatitis (ACD) by modulating the NF- κ B /NLRP3 pathway

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Allergic contact dermatitis (ACD), a type IV hypersensitivity reaction mediated by hapten-specific T cells, is characterized by erythema, edema, and pruritus. Although *Ledum palustre* var. *diversipilosum* (LPD) has traditionally been used to treat inflammatory conditions, its effects on inflammatory skin diseases such as ACD remain unexplored. In this study, we investigated the anti-inflammatory effects of LPD in a 2,4-dinitrofluorobenzene (DNFB)-induced ACD mouse model by evaluating its ability to alleviate clinical symptoms and suppress pro-inflammatory mediators. Oral administration of LPD markedly reduced ear thickness, clinical symptoms, and histopathological features, including epidermal thickening and immune cell infiltration in DNFB-induced ACD mice. At the molecular level, LPD suppressed the overexpression of pro-inflammatory mediators (IL-6, TNF- α , IL-1 β , MMP12) and chemokines (CCL2, CCL3, CCL4, CCL5, CXCL1, CXCL2) in ACD-induced ear tissues. Mechanistically, LPD inhibited NF- κ B activation by reversing nuclear translocation of phosphorylated p65 and attenuated NLRP3 inflammasome signaling via downregulation of ASC, caspase-1, caspase-8, and pro-IL-1 β , accompanied by a marked reduction in IL-1 β expression. These findings suggest that LPD exerts its anti-inflammatory effects by concurrently suppressing NF- κ B activation and NLRP3 inflammasome signaling, which are two major pathways involved in ACD pathogenesis. Therefore, our results suggest that LPD has potential as a promising natural therapeutic candidate for ACD and other inflammatory skin diseases.

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Keywords: Allergic contact dermatitis, *Ledum palustre* var. *diversipilosum*, Anti-inflammation

PS-A-15

Puriton attenuates the asthma severity in ovalbumin-induced murine model via balancing Th1/Th2 and inhibiting inflammation

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Asthma is a chronic inflammatory condition characterized by hyperresponsiveness in the respiratory system, which is triggered by indoor allergens such as house dust mites, cockroaches, and pet dander, as well as outdoor allergens like pollen, peanut protein, and mold. Symptoms of asthma includes wheeze, shortness of breath, chest tightness, and cough which is caused by airflow limitation and airflow limitation was resulted from the representative morphological change in pulmonary system in asthma patients such as airway remodeling including epithelial cell hyperplasia/apoptosis/fibrosis, mucous hypersecretion, and inflammatory cell infiltration such as neutrophils, M2 macrophages, and eosinophils. In 2019, 262 million asthma patients were estimated, and 455 thousand deaths were caused by asthma. It is an incurable, chronic inflammatory respiratory disease and is more severe in the elderly and the young. Forty-eight BALB/c mice were divided into 5 groups: vehicle group (CON), asthma group (OVA), positive drug group (DEX), and Puriton (700 and 1400 µL/head/day). After the animal experiment, all mice were narcotized for collecting BALF and blood, and then anesthetized for sampling the lungs. WBC and differential count in BALF and IgE in serum were measured. Lung tissues were used for histopathological and immunohistopathological studies. Puriton decreased the populations of WBC and neutrophils and the level of IgE. It prevented OVA-induced morphological changes in the lung and increased the expression levels of Th2 cell-related cytokines such as IL-4, IL-5, and IL-13. It inhibited the inflammatory cytokines such as TNF-α and IL-6 and the inflammatory product PGE2. From the results, we concluded that Puriton is a promising drug candidate for asthma treatment.

*Corresponding author : Dae-Hun Park

Keywords : Asthma, Imbalance of helper T1/helper T2, Inflammation, Puriton

PS-A-17

Quantitative analysis of region-specific microglia-neuron structural interactions in the ischemic core and penumbra following ischemic brain injury

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Ischemic stroke is a leading cause of death and disability worldwide, characterized by irreversible neuronal damage and marked spatial heterogeneity within the brain lesion. Distinct morphological and functional differences are observed between the infarct core, where irreversible damage predominates, and the penumbra, where partial neuronal viability remains. Although several studies have anatomically defined these regions, quantitative assessments of structural interactions between neurons and glial cells within each compartment remain limited.

In the present study, ischemic stroke was induced by transient middle cerebral artery occlusion in male Sprague-Dawley rats. Hematoxylin and eosin staining was used to identify infarcted tissue and distinguish the core and penumbra. Western blot analysis was conducted to examine markers associated with neuronal injury and microglial activation in the cerebral cortex. Immunofluorescence labeling of Iba-1 and NeuN was performed and confocal Z-stack images in the cerebral cortex were obtained. Morphological parameters including microglial ramification, soma circularity, neuronal dendritic integrity, and intersection points between microglia and neurons were quantitatively analyzed.

In the infarct core, microglia exhibited amoeboid morphology with reduced branching complexity, accompanied by severe dendritic loss and decreased expression of synaptic markers in neurons. In contrast, the penumbra showed partially preserved dendritic structures and microglia with ramified or hyper-ramified morphology. The number of microglia-neuron intersections and their spatial proximity varied significantly between regions.

These findings provide a quantitative spatial characterization of microglia-neuron interactions in ischemic lesions and highlight the importance of regional heterogeneity in neuropathological responses. The approach establishes a structural basis for evaluating neuroinflammatory mechanisms and may inform region-specific therapeutic strategies for stroke and related neurological disorders.

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Keywords : Ischemic stroke, Microglia-neuron interaction, Sholl analysis, Morphological quantification

PS-A-16

A custom imaging-guided intranasal system for brain delivery in a cynomolgus monkey (*Macaca fascicularis*)

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Intranasal drug delivery is a promising non-invasive route for accessing the central nervous system (CNS). However, anatomical complexity in non-human primates (NHPs)—such as intricate nasal structures and increased nose-to-brain distance—presents significant challenges compared to rodent models. To address these limitations, we developed an optimized intranasal delivery system for a cynomolgus monkey (*Macaca fascicularis*), incorporating CT imaging for guided administration and post-delivery evaluation. A custom-designed injection frame enabled stable, accurate, and reproducible delivery while minimizing agent loss and preventing off-target diffusion. Real-time CT imaging verified precise injection into the upper region of the ethmoturbinate (EMT), which contains olfactory epithelium and serves as an optimal anatomical target for brain access. Notably, the CT contrast agent reached the olfactory bulb within 1 hour post-administration and remained localized in the EMT region for up to 3 hours. These findings highlight the feasibility of non-invasive nose-to-brain delivery in NHPs. Future studies incorporating nanoparticle-based carriers—such as gold nanoparticles (AuNPs)—are expected to further enhance nose-to-brain delivery efficiency and therapeutic potential.

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Keywords : Intranasal delivery, Brain, CT, Non-human primate

PS-A-18

ATP-driven pyroptotic cell death in intestinal mucosal immune system in colitis

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Background/Aim : Damage-associated molecular patterns (DAMPs) are endogenous molecules released from injured or dying cells, contributing to inflammation and cell death. Among them, extracellular ATP is a well-known DAMP and primarily studied in monocyte for its role in pyroptosis. This study aimed to explore the role of extracellular ATP in mediating pyroptosis within the intestinal mucosa during colitis.

Methods : Experimental colitis was induced in mice using dextran sodium sulfate (DSS). ATP levels and the pyroptosis-associated proteins were evaluated in colonic tissues and serum. *In vitro*, Caco-2 colonic epithelial cells were treated with ATP to assess its effects on cell death, mitochondrial function, and pyroptosis. Co-treatment with flagellin was also performed to assess potential synergistic effects.

Results : DSS-induced colitis led to a significant increase in ATP levels in both colonic tissues and serum, accompanied by upregulation of pyroptosis-associated mediators. *In vitro*, ATP treatment induced cell death and mitochondrial dysfunction in Caco-2 cells, along with enhanced expression of inflammatory and pyroptotic markers, without affecting apoptosis-related proteins. Co-treatment with flagellin and ATP further elevated inflammatory responses; however, ATP did not potentiate flagellin-induced cell death or pyroptosis signaling.

Conclusion : Extracellular ATP is markedly elevated during colitis and functions as a DAMP that promotes pyroptosis in intestinal epithelial cells. Moreover, ATP released during pyroptosis can propagate cell death and cause damage to neighboring cells, further exacerbating mucosal injury. This study broadens our understanding of extracellular ATP-mediated pyroptosis in the intestinal mucosal environment.

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Keywords : ATP, Intestine, Pyroptosis, Inflammation, Colitis

PS-A-19

TXNIP regulated endothelial cells senescence drives liver regeneration

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Liver sinusoidal endothelial cells (LSECs) play a critical role in maintaining hepatic homeostasis and promoting liver regeneration. Senescence in LSECs contributes to inflammation and fibrosis through the senescence-associated secretory phenotype (SASP). However, the specific impact of LSEC senescence on liver regeneration remains unclear. Thioredoxin interacting protein (TXNIP) is a type of α -arrestin family protein, is involved in various cellular functions, including cell proliferation, apoptosis, glycolysis, and redox state homeostasis. Previous research has shown that glucose-induced metabolic stress upregulates TXNIP, thereby promoting cellular senescence. However, the specific role of TXNIP in LSECs senescence remains unclear. In this study, we performed 2/3 partial hepatectomy (PHx) in young and old mice to investigate the relationship between TXNIP and LSECs senescence. Aged mice exhibited impaired liver regeneration and the expression of the cell proliferation marker PCNA was sustained after 24 hours post PHx, whereas it continued to increase in the young controls. In young mice, cell cycle progression was initiated earlier, and the expression levels of Cyclin E1 and CDK2 were higher than those in aged mice, indicating more efficient liver regeneration. In aged mice, the expression of TXNIP, TGF- β , and CXCL1 was up-regulated, while IL-6 and TNF- α expression began to increase from 24 hours after PHx. To investigate the relationship between LSEC senescence and liver regeneration, we performed co-immunofluorescence staining for the senescence marker p16^{INK4a} and the LSEC marker Lyve-1. In aged mice, p16^{INK4a}/Lyve-1 double-positive cells were up-regulated compared to young controls. TXNIP expression in senescent endothelial cells was higher than in normal endothelial cells. However, the senescence marker p21 was not detected by immunohistochemistry. TXNIP regulated senescence of LSECs may be a key regulator of liver regeneration and repair. This may represent an important therapeutic target for liver repair and recovery.

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Keywords : Liver sinusoidal endothelial cells, TXNIP, Senescence, Liver regeneration, Partial hepatectomy

PS-A-21

Targeted inhibition of TLRs modulates inflammation and enhances efferocytosis in an autoimmune multiple sclerosis model

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Toll-like receptors (TLRs) play a crucial role in recognizing molecular patterns of microbes and damages thereby triggering the innate immune responses. In previous work, we demonstrated that selective blockade of TLR signaling mitigated colitis by modulating macrophages. Using a cell-penetrating TLR inhibitor peptide, in this study, we evaluated the potential of TLR inhibition to modulate immune responses in an autoimmune model of multiple sclerosis. The competitive TLR inhibitor peptide, designed to disrupt TLR binding to the adaptor MyD88 and TRIF, was conjugated to a cell-penetrating peptide (cpTLR-i) and shown to suppress the production of pro-inflammatory cytokines such as TNF- α in BV2 microglia cell line. In an experimental autoimmune encephalomyelitis (EAE) mouse, cpTLR-i treatment led to delayed disease onset, reduced demyelination, and decreased inflammation in the central nervous system (CNS). Flow cytometry analyses revealed that cpTLR-i diminished CNS infiltration by monocytes/macrophages and attenuated pro-inflammatory activation of microglia. Notably, we observed a significant reduction of apoptotic cells in the brain, accompanied by an increased in microglia, implying that TLR inhibition might promote the clearance of damaged neurons. Gene expression analysis revealed the significant alteration of genes related to efferocytosis. Furthermore, we validated that cpTLR-i enhanced the uptake of apoptotic neuron cells by phagocytes such as BMDMs, partially through the modulation of TGF- β -induced LXR α signaling pathway. In conclusion, our experimental data suggest that the TLR inhibitor peptide is a potent immune modulator, not only limiting excessive inflammation but also promoting immune resolution processes such as efferocytosis.

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Keywords : Toll-like receptors, Cell-penetrating peptide, Multiple sclerosis, Microglia, Efferocytosis

PS-A-20

Cannabinoid receptor type 1 regulates aging-associated intestinal barrier dysfunction: Evidence from a rat model and in vitro studies

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Introduction : Aging is linked to impaired gastrointestinal (GI) function, notably increased intestinal permeability. While the endocannabinoid system (eCB)-particularly cannabinoid receptor type 1 (CB1)-plays a critical role in maintaining gut homeostasis, its involvement in age-associated intestinal dysfunction remains poorly understood. This study aimed to investigate the role of the eCB system in aging-related changes in intestinal barrier integrity.

Methods : A naturally aged rat model comparing young (6 months) and aged (24 months) animals was used alongside *in vitro* cellular senescence models induced by hydrogen peroxide (H₂O₂) and hydroxyurea (HU). CB1 expression was assessed in both models using qPCR. And senescence cell morphology and beta-galactosidase activity were analyzed by optimal bright field image. Transepithelial electrical resistance (TEER) was measured to evaluate intestinal permeability. miRNA databases (miRDB, TargetScan) were used to identify miRNAs targeting the tight junction protein ZO-1. Functional studies included miRNA/siRNA transfection, qPCR, and western blotting. Results : CB1 expression was significantly reduced in the intestinal epithelium of aged rats and senescent cells. Senescence led to decreased TEER values and downregulation of ZO-1, indicating increased intestinal permeability, which was tightly regulated by the CB1 signaling. *In silico* miRNA analysis identified ZO-1 as a direct target of miR-191-5p. HU treatment increased miR-191-5p expression, which was restored by co-treatment with the CB1 agonist ACEA. Overexpression of miR-191-5p increased intestinal permeability, whereas its inhibition restored barrier function. Notably, NF- κ B p65 activation was implicated in the CB1—miR-191-5p regulatory axis.

Conclusion : Age-related decline in CB1 leads to increased intestinal permeability by promoting miR-191-5p expression and NF- κ B p65 activation, ultimately reducing ZO-1 levels. These findings suggest that CB1 signaling is a potential therapeutic target for mitigating aging- or inflammation-induced intestinal barrier defects.

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Keywords : Aging, CB1, MiR-191-5p, Intestinal permeability, Barrier integrity

PS-A-22

Korean red ginseng extract powder mitigates fasting and postprandial hyperglycemia in type 2 diabetic Mice

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Type 2 diabetes mellitus (T2DM) involves insulin resistance and elevated blood sugar levels, causing complications. Red ginseng extract powder (RGE) from Panax ginseng Meyer shows promise for diabetes treatment. However, its efficacy in managing T2DM remains unclear. Therefore, this study aims to evaluate the effectiveness of RGE in a mouse model of T2DM. The efficacy of RGE in treating T2DM was assessed in db/db mice. Mice were divided into seven groups: control, db/db, metformin, and RGE at 50, 100, 200, and 400 mg/kg. Administered orally for 9 weeks, RGE effects on glucose regulation and insulin sensitivity were assessed through various metabolic parameters. Additionally, mRNA expression levels of genes associated with hepatic gluconeogenesis and insulin sensitivity were examined. Fasting blood sugar showed a significant decrease in all RGE concentration groups, but OGTT and ITT showed a significant decrease at the RGE concentration of 400mg/kg, indicating enhanced glycemic control. Moreover, RGE dose-dependently decreased serum glucose, HbA1c levels, and HOMA-IR values, suggesting its effectiveness in reducing insulin resistance in db/db mice. Furthermore, RGE down-regulated mRNA expression of key components in the gluconeogenesis pathway (G6Pase, FOXO1, GLUT4, and PEPCK), insulin sensitivity (leptin, insulin1, PTP1B, GLP-1, and DPP-4), and mitochondria energy metabolism (PGC1) in either the liver or pancreas, while simultaneously upregulating GLP-1 expression. In conclusion, these findings highlight the potential of RGE as a complementary therapy for T2DM, indicating therapeutic efficacy in managing diabetic complications through improved metabolic parameters.

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Keywords : Fasting blood glucose, Insulin resistance, Postprandial hyperglycemia, Red ginseng extract powder, Type 2 diabetes mellitus

PS-A-23

A Yeast-Derived Aldehyde-Reducing Compound attenuates oxidative and aldehyde stress across evolutionarily conserved Alzheimer's disease models

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Alzheimer's disease (AD) progression is mediated by multiple converging mechanisms, notably amyloid- β (A β) aggregation, oxidative stress through reactive oxygen species (ROS), and lipid-derived reactive aldehydes such as 4-hydroxynonenal (4-HNE). Among central nervous system ROS sources, astrocytic NOX4 plays a pivotal role by initiating a downstream myeloperoxidase (MPO)-osteopontin (OPN) loop, which enhances lipid peroxidation, promotes glial ferroptosis, and perpetuates neuroinflammation. Given the synergistic toxicity of ROS and aldehyde stress, agents capable of attenuating both pathways represent a promising therapeutic strategy.

We evaluated a yeast-derived aldehyde-reducing composition (ARC) in three evolutionarily distinct AD models. In *Drosophila melanogaster* overexpressing human tau (GMR > hTau), ARC treatment improved eye morphology and decreased phospho-Tau/total Tau ratios, suggesting a protective effect on tauopathy. In *Caenorhabditis elegans*, ARC reduced whole-organism ROS, delayed A β -induced paralysis (CL4176), and preserved dopaminergic neurons in the 6-OHDA-challenged BZ555 strain, with improved locomotor behavior, indicating efficacy against both AD- and Parkinsonian-type insults. In mammalian models, 10-month-old 5XFAD mice received oral ARC (20 units/kg/day for 30 days). Hippocampal tissue analysis showed markedly reduced levels of NOX4, MPO, OPN, and 4-HNE, consistent with suppression of oxidative and aldehyde cascades.

Collectively, ARC showed conserved neuroprotective effects across invertebrate and mammalian AD models by mitigating shared upstream stress mechanisms rather than targeting a single downstream hallmark. These findings position ARC as a promising multi-target candidate for further pre-clinical development and mechanistic dissection in age-related neurodegeneration.

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Keywords : Alzheimer's disease, Reactive oxygen species (ROS), NOX4, Lipid peroxidation, Aldehyde-reducing composition (ARC)

PS-A-25

Analysis of fibroblast subtypes specific to postpartum uterine regeneration

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Following parturition, the uterus undergoes a dynamic regeneration and tissue remodeling characterized by injury and subsequent repair. In postpartum uterus, tissue remodeling and regeneration are essential for restoring uterine homeostasis. If this process is disrupted due to postpartum infection or inflammation, reproductive function may be impaired, and the risk of pathological conditions increase. Although implantation and pregnancy have been extensively studied, the biological events that occur in the uterus after delivery remain largely unexplored. To investigate cellular dynamics and injury responses in the postpartum uterus at single-cell resolution, we performed single-cell RNA sequencing (scRNA-seq) using a murine model. scRNA-seq was conducted on uterine tissues collected at postpartum days (PPD) 0, 2, 3, and 5. An injury model was established by applying pelvic irradiation at PPD3, which showed peak regenerative activity based on Ki67 immunostaining, and tissues were collected at PPD5. Temporal analysis of the scRNA-seq data revealed dynamic changes in cellular composition. Immune and endothelial cell populations sharply decreased over time after parturition, while stromal and smooth muscle cell populations progressively increased. Within the fibroblast population, we identified two transcriptionally distinct subtypes, referred to as deep fibroblasts and superficial fibroblasts. Superficial fibroblasts showed a pronounced increase over time during normal regeneration. Additionally, subclusters of deep fibroblasts specific to PPD2 and PPD3 were identified, along with their marker genes and activation of signaling pathways related to wound healing. In irradiated uteri, the proportion of superficial fibroblasts, which are closely associated with epithelial cell survival and differentiation was reduced, whereas deep fibroblasts and endothelial cells were relatively expanded. These results suggest that specific fibroblast subtypes contribute to postpartum uterine regeneration, and that disruption of this process may lead to fibrotic complications and impaired reproductive recovery.

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Keywords : Postpartum, Uterus, Single cell RNA-seq, Regeneration, Fibroblast

PS-A-24

Agrimonia pilosa extract alleviates CDAHFD-Induced non-alcoholic steatohepatitis and fibrosis in mice

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Non-alcoholic steatohepatitis (NASH) is caused by fat buildup, inflammation, and fibrotic changes within the liver. NASH can advance decompensated cirrhosis and carcinoma by inflammation and oxidative stress if untreated. Although *A. pilosa* has traditionally been used for medicinal purposes, its effects on the liver remain incompletely elucidated. In this study, we assessed the therapeutic outcomes of *A. pilosa* extract (APE) on free fatty acid (FFA)-treated HepG2 cellular hepatic steatosis and in a choline-deficient, L-amino acid defined, high-fat diet with 0.1% methionine (CDAHFD)-induced NASH mouse model. In FFA-treated cells, APE inhibited intracellular lipid accumulation and regulated the mRNA and protein expression of modulators of fatty acid (FA) synthesis as well as FA oxidation proteins through downregulation of the AMP-activated protein kinase/sirtuin 1 signaling pathway. In the CDAHFD-induced NASH mouse model, APE significantly enhanced liver histology and serum levels of liver damage. APE also significantly attenuated hepatic expression of mRNA and proteins related to FA synthesis, inflammation, and fibrosis. Furthermore, APE mitigated the expression levels of oxidative stress and endoplasmic reticulum stress. Overall, these findings indicate that *A. pilosa* can treat NASH by regulating hepatic lipid metabolism, alleviating inflammation, and mitigating oxidative stress.

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Keywords : Non-alcoholic steatohepatitis, *Agrimonia pilosa*, Lipid metabolism, Inflammation, Oxidative stress

PS-A-26

Rapid Single-Step Aqueous Tissue Clearing for Volumetric Imaging and Fine Neural Detail Visualization in the Mouse Brain

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Optical tissue clearing is essential for high-resolution imaging of thick biological samples, but many existing methods suffer from high viscosity, insufficient refractive index (RI) matching, and loss of fluorescence signals. Here, we present AICI (Aqueous high refractive Index matching and tissue clearing solution for Imaging), a novel aqueous clearing solution characterized by low viscosity and a high RI of 1.466. AICI enables simultaneous refractive index matching and rapid tissue clearing, achieving full transparency of 1-mm-thick mouse brain slices within 90 minutes. The solution maintains tissue morphology with minimal distortion (<2.2% change after 24 hours) and preserves fluorescence signals over time (GFP retains >50% intensity after 10 days). AICI remains stable in liquid form for over 30 days even under air exposure, supporting flexible experimental timelines. While the AICI clearing method preserves fine neuronal structures such as dendritic spines well, its clearing capacity is restricted to a depth of 3 mm. To extend its applicability, we combined AICI with electrophoretic tissue clearing, enabling whole-brain transparency in mice. We then performed three-dimensional imaging using light sheet fluorescence microscopy (LSFM) and spinning disk confocal microscopy, which are representative techniques for volumetric imaging. LSFM offered advantages over confocal imaging in imaging depth, acquisition speed, and fine-structure preservation, suggesting its usefulness for volumetric imaging of cleared brain tissue.

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Keywords : Tissue clearing, Light sheet fluorescence microscopy, Spinning disk confocal microscopy, 3D imaging

PS-A-27

High Sucrose Intake Aggravates Airway Hyperresponsiveness and Promotes M1 Macrophage-Mediated Th1/Th17 Cell-Driven Inflammation in Adipose and Intestinal Tissues of OVA-Sensitized C57BL/6 Mice

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High sucrose intake drives inflammation, yet its immunopathogenic role in allergic asthma remains elusive. In this study, we delineated the immunometabolism impact of high sucrose intake in ovalbumin sensitized C57BL/6 mice and revealed an important role played by the gut-adipose-lung axis of inflammation. In intestine, sucrose consumption markedly intensified OVA-induced immune responses by enhancing intestinal monosaccharide absorption, demonstrated by elevated SGLT1, GLUT2, and GLUT5 expression. This was accompanied by increased malondialdehyde (MDA) levels, reflecting heightened oxidative stress. Mucosal integrity was also compromised as indicated by reduced MUC2 expression and goblet cell depletion. Activation of TLR4-MYD88 signaling, along with robust polymorphonuclear (PMN) cell infiltration and upregulation of chemokines CXCL8, CCL2, and CCL5, facilitated innate and adaptive immune cell recruitment. These events triggered intestinal NLRP3 inflammasome activation and IL-1 β production, promoting M1 macrophage polarization and increased expression of Th1 (T-bet) and Th17 (ROR γ T) transcription factors. Adipose tissues responded similarly, showing enhanced GLUT4 expression, oxidative stress, adipocyte hypertrophy, mast cell degranulation, and crown-like macrophage aggregation. Inflammatory mediators, including NLRP3, IL-1 β , phosphorylated I κ B, and matrix metalloproteinases (MMP7, MMP12) were elevated, indicating sustained macrophage-driven inflammation and immune cell infiltration. This inflammatory state of intestine and adipose tissues extended to lungs, exhibiting elevated reactive oxygen species (ROS) levels, NLRP3 activation, and a predominance of M1 macrophages in lungs. Collectively, these findings suggest that high sucrose intake can aggravate allergic asthma by initiating intestinal inflammation and facilitating it to propagation through adipose tissues and to interact with lung pathology. This emphasizes the systemic pro-inflammatory potential of excessive dietary sugar and the involvement of the gut-adipose-lung axis.

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Keywords : High Sucrose Intake (HSI), Airway Hyperresponsiveness (AHR), NLRP3 inflammasome, Matrix metalloproteinase (MMP), Th1/Th17 response

PS-A-28

Microarray profiling of splenic RNA reveals immune gene modulation following long-term intake of Jeju groundwater

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Jeju groundwater (JGW), naturally enriched with vanadium and other minerals, has been associated with immune and metabolic health benefits. In this study, we evaluated the immunomodulatory effects of its prolonged intake in a murine model. We fed C57BL/6 mice with either JGW (containing 7.5 μ g/L vanadium) or tap water (1.5 μ g/L vanadium) for 210 days, and examined their splenic tissue transcriptome profiles using microarrays. Gene Set Enrichment Analysis (GSEA) and REACTOME pathway annotation of genes showing differential expression between JGW and tap water mice groups revealed significant enrichment of immune related signaling cascades, including those involving MyD88, MAPK, T cell and B cell receptors, and FOXP3-regulated pathways. Activation of MAPK pathway proteins ERK1/2 and p38 was validated through Western blotting while cytokine (IL-2, IFN- γ) and IgG levels were quantified using ELISA. Compared to controls, JGW treated mice exhibited increased phosphorylation of ERK1/2 ($p < 0.05$) and p38 MAPK ($p < 0.01$) along with elevated levels of cytokines and IgG. These results suggest that chronic consumption of JGW stimulates key immune signaling pathways, particularly those mediated by MyD88, MAPK, BCR, and TCR, thereby enhancing both innate and adaptive immune responses systemically.

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Keywords : Jeju groundwater (JGW), Vanadium, MAPK pathway, Microarray profiling, MyD88 signaling

PS-A-29

Medium-chain inulin modulates immune responses and attenuates experimental colitis

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Inulin, a natural dietary fructan composed of $\beta(2 \rightarrow 1)$ -linked fructose residues terminated by a glucose unit, exhibits prebiotic properties with reported beneficial effects on intestinal inflammation and immune responses. However, these biological activities vary depending on structural characteristics, particularly the degree of polymerization (DP), and the effects of medium-chain inulin (M-inulin, average DP 12) remain unclear. In this study, we investigated the immune responses and intestinal barrier-protective effects of M-inulin isolated from *Helianthus tuberosus*, a sunflower-like plant that grows underground tubers rich in M-inulin. In primary murine splenocytes, treatments with M-inulin alone significantly enhanced immune cell proliferation compared to that of treatments with fructooligosaccharides (FOS) or total inulin (T-inulin). Under an immune stimulation with Concanavalin A (ConA), M-inulin treatments further promoted immune cell proliferation, increasing populations of CD45⁺F4/80⁺ macrophages, CD45⁺Gr-1⁺ granulocytes, CD45⁺Gr-1⁺Ly6G⁺ neutrophils, and CD3e⁺CD8⁺ cytotoxic T-cells. Conversely, under an immune activation using phorbol 12-myristate 13-acetate (PMA), M-inulin treatments significantly suppressed immune cell proliferation, demonstrating M-inulin's context-dependent change of effects on immune cell proliferation. Additionally, in a Caco-2/HT-29 epithelial co-culture model challenged with LPS-primed conditioned medium, M-inulin treatments significantly enhanced barrier integrity, indicated by reduced FITC-dextran permeability and nitric oxide production. Consistent with these in vitro findings, oral administration of M-inulin ameliorated clinical symptoms, reduced histological inflammation, and preserved epithelial architecture in a DSS-induced colitis mouse model. Collectively, our findings indicate that M-inulin effectively modulates immune responses and strengthens epithelial barrier integrity, thereby representing a promising dietary intervention for mitigating intestinal inflammation in conditions like IBD.

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Keywords : Medium-chain inulin, Inflammatory bowel disease, Intestinal barrier, Context-dependent effects

PS-A-30

TLR4 deficiency alleviates airway inflammation in PM-exacerbated allergic asthma mice by regulating macrophage polarization and Th17 differentiation

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Particulate matter (PM) is increasingly recognized as a significant contributor to allergic inflammation in asthma. Recent evidence demonstrates that PM exacerbates airway inflammation in allergic asthma mice characterized by Th2/Th17-mediated immune responses. Furthermore, PM may aggravate allergic inflammation in the murine lung through the TLR2/TLR4/MyD88 signaling pathway, thereby contributing to the exacerbation of allergen-induced lung eosinophilia caused by PM. In this study, we investigated the role of TLR4 in PM-exacerbated allergic asthma mice. PM exposure in OVA-induced WT mice promoted TLR4 expression and Th17 responses, leading to macrophage activation and increased the expression of MMP3 and MMP9, as determined by RNA-seq analysis. PM exposure increased CD80+ M1 macrophages, while OVA exposure elevated CD206+ M2 macrophages. Additionally, PM exposure in OVA-induced WT mice significantly amplified oxidative DNA damage and cytokine secretion in both M1 and M2 macrophages. In contrast, TLR4^{-/-} mice showed reduced secretion of M1 and M2 cytokines, along with lower overall inflammatory cytokine levels compared to WT mice after PM exposure. Additionally, inflammatory cell infiltration, mucus hypersecretion, and mast cell activation in lung tissues were reduced in TLR4^{-/-} mice compared to WT mice upon PM exposure. Notably, neutrophil infiltration which is typically activated in inflammation was attenuated in TLR4^{-/-} mice compared to WT mice upon PM exposure. These findings suggest that TLR4 regulates PM-induced asthma by modulating M1/M2 macrophage polarization and Th17 responses.

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Keywords : Allergic asthma, Particulate matter (PM), Transcriptome sequencing (RNA-Seq), Toll-like receptor 4 (TLR4), M1/M2 macrophage polarization

PS-A-32

Electroceutical Treatment Reduces Acute Renal Injury by Suppressing Osteopontin-Driven Inflammatory Responses in Rats

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Acute kidney injury (AKI) is a common and serious clinical condition characterized by a rapid decline in renal function, often accompanied by inflammation and tubular damage. Despite its prevalence, effective therapeutic interventions remain limited. In this study, we explored the potential renoprotective effects of an electron-producing apparatus (EPA) in a rat model of folic acid (FA)-induced AKI. AKI was induced via intraperitoneal injection of FA at a dose of 250 mg/kg. Subsequently, the animals received EPA treatment at 0.4 mV, administered five times over a 48-hour period. Histological examinations of renal tissues were performed, and inflammation was assessed through immunohistochemical analysis of ionized calcium-binding adapter molecule 1 (Iba-1) and osteopontin (OPN), both of which are markers associated with inflammatory responses in kidney injury. The results demonstrated that EPA significantly reduced renal histopathological damage, decreased inflammatory cell infiltration, and improved overall renal function. Compared to the vehicle-treated group, EPA-treated rats showed markedly lower serum creatinine and blood urea nitrogen (BUN) levels, indicating better kidney performance. Furthermore, EPA effectively suppressed the FA-induced upregulation of OPN and other pro-inflammatory mediators, suggesting that it acts through inhibition of OPN-mediated pathways. These findings highlight the potential of EPA as a novel therapeutic approach for the treatment or prevention of AKI. By attenuating inflammation and preserving kidney structure and function, EPA may offer a promising avenue for clinical intervention in AKI and related renal disorders. (Funding Source: This work was supported by the National Research Foundation of Korea: Grant number NRF-2021M3H9A1097596 and the R&D Support Project for Gangwon Science and Technology Promotion, RS-2025-02173090)

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Keywords : Electroceutical treatment, Folic acid, Inflammation, Osteopontin

PS-A-31

Evaluation of targeted drug delivery and therapeutic efficacy of Glucose-Albumin/Apigenin nanocomplex in Idiopathic pulmonary fibrosis model

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Purpose : Idiopathic pulmonary fibrosis (IPF) is a fatal lung disease characterized by excessive deposition of extracellular matrix (ECM) and irreversible damage to alveolar structures. Current treatments delay disease progression but cannot reverse established fibrosis. Apigenin, a natural flavonoid, has anti-inflammatory and anti-fibrotic effects but is limited by poor solubility and bioavailability. To overcome these limitations, we developed a glucose-albumin/apigenin (Glc-Alb/Api) nanocomplex to improve biocompatibility and enhance lung targeting via glucose moiety.

Materials and Methods : Glc-Alb/Api nanocomplexes were synthesized by conjugating glucose and apigenin to DBCO-modified albumin via copper-free click chemistry, confirmed by UV-Vis spectroscopy and MALDI-TOF mass spectrometry. Pulmonary fibrosis was induced in C57BL/6 mice by intratracheal instillation of bleomycin (3 mg/kg). On day 7, mice were injected intravenously with saline, Alb, Api, Glc-Alb or Glc-Alb/Api. Lung tissues were collected at weeks 2, 3, and 4 for RT-qPCR, Western blot and histology. Radiolabeled nanocomplexes with copper-64 were visualized by PET/MR.

Results : All groups exhibited body weight loss until day 7 post-bleomycin. The Glc-Alb/Api group began recovering weight from day 11 and showed significantly greater recovery over 28 days compared to the saline group (P < 0.0001). Hematoxylin and Eosin (H&E) staining demonstrated reduced alveolar structural damage and decreased ECM deposition in the Glc-Alb/Api group compared to the saline group. Immunohistochemistry (IHC) analysis showed that macrophage infiltration increased across all groups at weeks 2 and 3. By week 4, the Glc-Alb/Api group showed an increase in pro-inflammatory M1 macrophages and a decrease in anti-inflammatory M2 macrophages, whereas the saline group exhibited the opposite pattern. Radiolabeled [⁶⁴Cu]Cu-Glc-Alb nanocomplex accumulated effectively in lung tissue, confirming targeted delivery.

Conclusions : The Glc-Alb/Api nanocomplex enhanced pulmonary targeting and drug delivery, significantly improving apigenin's therapeutic efficacy in a bleomycin-induced pulmonary fibrosis model. These results suggest that Glc-Alb/Api represents a promising novel strategy for IPF treatment.

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Keywords : Idiopathic pulmonary fibrosis, Apigenin, Albumin nanocomplex, Bleomycin, Radiolabeling technique

PS-A-33

Protocatechuic acid and syringin improve dyslipidemia-induced cardiac damage in mice

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Cardiovascular disease (CVD) burdens health and public health systems and is a major cause of morbidity and mortality worldwide. The most well-known risk factor for CVD is dyslipidemia. Dyslipidemia is characterized by low high-density lipoprotein (HDL) cholesterol levels and high triglyceride (TG), low-density lipoprotein (LDL), and total cholesterol (TC) levels. These changes can be major risk factors for CVD. In previous studies, we isolated protocatechuic acid (PCA) and syringin (SY) from the whole plant of *Saussurea neoserrata* Nakai. PCA and SY have been reported to have antioxidant, anti-inflammatory, and myocardial damage protection effects. Therefore, we hypothesized that natural compounds PCA and SY could be used as effective therapeutic strategies to alleviate cardiac damage caused by dyslipidemia. This study demonstrated that oral administration of PCA and SY provided significant cardioprotective effects against dyslipidemia-induced cardiac dysfunction through multiple molecular mechanisms. After feeding C57BL/6 mice a high-fat diet for four weeks, we continued to feed them a high-fat diet and administered treatment for an additional 12 weeks. Administration of PCA and SY improved electrocardiogram parameters, effectively alleviated cardiac fibrosis, and inhibited the TLR4/NF-κB inflammatory signaling pathway. We also found that it normalized dyslipidemia profiles. The combination therapy of PCA and SY showed superior efficacy compared to individual treatments and demonstrated effects similar to those of simvastatin, which is commonly used to treat dyslipidemia, suggesting potential therapeutic possibilities for cardiac damage caused by dyslipidemia.

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Keywords : Protocatechuic acid, Syringin, ECG, Dyslipidemia, Fibrosis

PS-A-34

Non-invasive detection of hepatic fibrosis using 68Ga-FAPI-46 PET imaging

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Hepatic fibrosis is a critical precursor to both hepatocellular carcinoma and hepatic dysfunction, with 80-90% of liver cancers arising in fibrotic or cirrhotic liver tissue. Activated hepatic stellate cells (HSCs) are regarded as the primary mediators of hepatic fibrosis and are known to express fibroblast activation protein (FAP), a key driver of fibrogenesis. In this study, hepatic fibrosis was induced in rats via intraperitoneal injections of thioacetamide (TAA; 150 mg/kg, twice per week) over 8 weeks. Following TAA administration, rats exhibited marked morphological alterations in the liver and an increased liver to body weight ratio. Serum AST and ALT levels were significantly elevated during the fibrotic phase and declined as fibrosis progressed to cirrhosis. Histopathological analysis revealed pronounced inflammatory infiltration, hepatocyte degeneration, and necrosis around the central vein and fibrotic septa in the TAA-treated group. PET imaging demonstrated a significant uptake of 68Ga-FAPI-46 at 8 weeks post-TAA administration, suggesting increased FAP expression. Plasma ELISA indicated elevated levels of TGF-β1, and Western blot analysis confirmed activation of the TGF-β1/Smad signaling pathway, correlating with increased FAP expression. Collectively, these findings suggest that hepatic fibrosis enhances TGF-β1/Smad signaling, promoting FAP expression. PET imaging with 68Ga-FAPI-46 may serve as a valuable tool for the non-invasive diagnosis and quantitative assessment of hepatic fibrosis, offering potential clinical utility in monitoring disease progression.

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Keywords : Hepatic fibrosis, Fibroblast activation protein inhibitor, Positron emission tomography, Thioacetamide, TGF-β1/Smad

PS-A-35

Longitudinal Neuroimaging and Neuropathological Evaluation of Autologous Blood-Induced Cortical Hemorrhage in Miniature Pigs

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Large-animal models of intracerebral hemorrhage (ICH) provide important anatomical and physiological relevance for investigating the progression of brain injury and evaluating therapeutic interventions. This study aimed to establish and characterize a reproducible miniature pig model of cortical ICH induced by stereotaxic injection of autologous blood into the primary motor cortex (M1), incorporating serial neuroimaging and histopathological analysis. Miniature pigs (KRIBB Small Pig, 20–25 kg) underwent MRI-guided stereotaxic injection of autologous arterial blood into the M1 region. Magnetic resonance imaging (MRI) was performed weekly over an 8-week period using T1-weighted, T2-weighted, FLAIR, and susceptibility-weighted imaging (SWI) sequences to monitor hematoma evolution, perilesional edema, and hemosiderin deposition. Histological assessments included hematoxylin and eosin (H&E) and Nissl staining to evaluate neuronal structure and tissue damage. Immunofluorescence staining was used to assess astrocytic and microglial responses, and neuronal degeneration in the perihematomal region. MRI analysis revealed gradual hematoma resolution with persistent signal abnormalities in the perilesional area. SWI consistently detected chronic-phase iron deposition within and surrounding the lesion. Histopathology confirmed neuronal degeneration and astroglial proliferation in perihematomal regions. Immunofluorescence demonstrated marked activation of glial cells and decreased NeuN expression near the hemorrhagic core. This stereotaxically induced cortical hemorrhage model in miniature pigs allows for detailed examination of the spatiotemporal progression of lesion development and associated cellular responses. Longitudinal neuroimaging combined with targeted immunofluorescence provides a methodological foundation for investigating post-hemorrhagic mechanisms and evaluating potential neuroprotective strategies in preclinical research.

Support (Grant)

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Keywords : Cortical hemorrhage, Miniature pig, Neuroimaging, Neuroinflammation, Stereotaxic injection

PS-A-36

Estrogen-induced expression of Pglyrp1 in the mouse endometrium

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Uterine development and function are subject to cyclical regulation primarily driven by steroid hormones such as estrogen and progesterone. Elucidating the molecular mechanisms underlying these periodic changes is essential for ensuring successful embryo implantation and development, as well as proper cellular differentiation and proliferation. During the estrous cycle, the endometrium undergoes coordinated phases of proliferation and apoptosis, tightly regulated by estrogen-dependent signaling pathways, which help maintain a functional uterine environment. Peptidoglycan Recognition Protein 1 (PGLYRP1) is a pattern recognition receptor known for its role in the innate immune response, particularly through binding bacterial peptidoglycan and modulating inflammation. Although well studied in infection related immune responses, recent studies suggest that Pglyrp1 may act as an immune cytokine even in sterile environment. However, the expression and regulatory mechanisms of Pglyrp1 in the uterus remain largely unexplored. In this study, we investigated the expression dynamics and hormonal regulation of Pglyrp1 in the mouse endometrium using *in vivo* and *in vitro* models. We found that Pglyrp1 is periodically expressed, increasing in the proestrus and estrus stages and decreasing in the metestrus and diestrus stages. To evaluate hormonal responsiveness, ovariectomized mice were treated with estradiol or progesterone. We observed that Pglyrp1 expression was significantly induced 24 hours after estradiol treatment, whereas progesterone had no effect. Moreover, the estrogen-induced expression was strongly inhibited by co-treatment with the estrogen receptor α (ER α) antagonist ICI 182, 780, conforming that Pglyrp1 is regulated in an ER α -dependent manner. Our findings suggest that Pglyrp1 may function as a cytokine-like immune regulator in the uterus, controlled by estrogen signaling. This provides new insight into hormone-immune interactions within the endometrium and Pglyrp1 as a potential player in uterine immune modulation.

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Keywords : Uterus, Estrogen, Pglyrp1, Immune response

PS-A-37

Adenine-induced chronic kidney disease (CKD) model as a platform to reveal the role of trained immunity in sustained systemic inflammation

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Chronic kidney disease (CKD), affecting over 10% of the global population, is frequently accompanied by comorbidities such as cardiovascular disease (CVD), often driven by chronic inflammation. Recent evidence suggests that trained immunity—an innate immune memory triggered by uremic toxins—may contribute to persistent inflammatory responses in CKD. However, its physiological relevance has not been validated in suitable animal models. In this study, CKD was induced in male C57BL/6J mice through a 0.2% adenine-containing diet for 2 or 4 weeks. Renal dysfunction and fibrosis were confirmed by elevated serum creatinine (CR) and blood urea nitrogen (BUN) levels, along with histological kidney analysis. To assess trained immunity, mice underwent a resting phase following adenine exposure. Splenic myeloid cells exhibited heightened cytokine production upon *ex vivo* lipopolysaccharide (LPS) stimulation, which was further amplified after the resting phase, indicating peripheral trained immunity. This response was attenuated by treatment with a methyltransferase inhibitor (MTA), suggesting an epigenetic modification. Furthermore, flow cytometry of bone marrow cells revealed an increased population of myeloid-biased multipotent progenitor cells (MPP3), indicating central trained immunity and a long-term myeloid skewing in hematopoiesis. These findings establish a physiologically relevant CKD mouse model and provide *in vivo* evidence that trained immunity contributes to sustained inflammation, potentially linking CKD to its associated comorbidities.

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Keywords : Chronic kidney disease, Trained immunity, Epigenetic modification, Myeloid skewing

PS-A-38

Establishment and Efficacy Evaluation of an Ischemic Stroke Model in Rodents

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Stroke is the most common cerebrovascular disorder, with ischemic stroke being one of the leading causes of mortality worldwide. Despite its high prevalence, effective therapeutic strategies remain limited, highlighting the need for continued research on prevention and recovery. In this study, we aimed to develop a symptom-oriented preclinical efficacy evaluation platform by establishing rodent models of ischemic stroke. Two representative models were utilized: the middle cerebral artery occlusion (MCAO) model and the photothrombosis model, which enables the induction of localized cortical infarction through light activation of a photosensitive dye (e.g., Rose Bengal). These models allow for the selective reproduction of ischemic damage based on lesion location and severity. Comprehensive behavioral assessments and histopathological analyses were conducted to evaluate functional deficits and tissue damage. This integrative approach is expected to provide a foundation for precise and tailored evaluation of therapeutic candidates targeting ischemic stroke.

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Keywords : Stroke, The middle cerebral artery occlusion (MCAO), Photothrombosis

PS-A-40

Optimized Flow Cytometry and Functional Assays for Immunophenotyping Common Marmoset Peripheral Blood Cells: Seoul National University Hospital – Marmoset Model Resource Bank (SNUH-MMRB)

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The common marmoset (*Callithrix jacchus*) has emerged as a valuable nonhuman primate (NHP) model in biomedical research due to its close physiological and immunological similarities to humans. These features make it particularly useful for investigating the pathophysiology of human diseases, including autoimmune and infectious disorders. To enhance the translational relevance of marmoset-based studies, a detailed understanding and comparison of their immune profiles with those of humans is essential.

This study aimed to establish a comprehensive immune profile of healthy marmosets from the Seoul National University Hospital Marmoset Research and Breeding Facility (SNUH-MMRB), the second largest marmoset colony in Korea. Peripheral blood samples were collected from 88 clinically normal healthy marmosets (45 males and 43 females), ranging in age from 12 to 73 months. A standardized protocol was developed to characterize the phenotypic and functional properties of key immune cell populations using commercially available antibodies originally designed for humans and macaques. An 8-color flow cytometry panel was optimized to define major immune cell subsets in peripheral blood of marmosets. For functional analysis, intracellular cytokine staining (ICS) was used to evaluate IFN- γ , IL-2, and IL-17A secretion by T cells, while TNF- α production by monocytes was quantified via enzyme-linked immunosorbent assay (ELISA). Additionally, a microbead-based cell sorting method enabled the isolation of specific immune cell subsets for gene expression analysis using quantitative PCR and ELISA.

The established protocols provide a robust platform for immune monitoring in marmosets and will support a wide range of translational studies requiring detailed immune characterization in this NHP model.

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Keywords : Common marmoset, Nonhuman primate, Immune profiles

PS-A-39

Anti-tumor activity of 5-HT2A antagonist 15f on colorectal cancer in vitro and in vivo

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5-Hydroxytryptamine (5-HT, Serotonin) has been reported to be an important role for modulation of various neurological disease, metabolism disease, and cancer progression. Herein, we reported on the feasibility of 15f (5-HT2A antagonist) as a novel anti-cancer agent for colorectal cancer in vitro and in vivo. Expression level of 5-HT2A receptor (HTR2A) was examined in normal colon cells and colorectal cancer cells using quantitative reverse transcriptase PCR. The effects of 15f on cell proliferation, cell cycle, and apoptosis was examined using CCK8 assay and FACS analysis. Western blotting analysis was conducted to determine the change of acetylated p53 (Ac-p53), p21, cleaved caspase-3, and cleaved PARP level in 15f-treated colorectal cancer cells. Colorectal cancer SW620 and HCT-116 cells were subcutaneously challenged to mice, followed by administration of 10 mg/kg and 50 mg/kg of 15f once a day and by measurement of tumor volume, body weight and bioluminescent imaging. HTR2A mRNA expression was higher in colorectal cancer cells than in normal colon cells. 15f lead to dose-dependent reduction of viability in colorectal cancer cells but not in normal cells. Compound 15f markedly triggered sub-G1 cell cycle arrest and apoptosis in colorectal cancer cells by activating the p53/p21/caspase 3 signaling pathway. Western blotting examination showed higher expression of Ac-p53 and p21 in 15f-treated cells compared with vehicle-treated cells. Cleaved caspase 3 and cleaved PARP level was up-regulated in colorectal cancer cells in response to 15f. Furthermore, in vivo treatment with 15f resulted in a marked inhibition of tumor progression without adverse reduction of body weight in all experimental mice. Consistently, in vivo optical imaging demonstrated that bioluminescent signals in HCT-116/luc tumors were much lower in 50mg/kg 15f-treated mice than in vehicle-treated mice. These findings suggest that 15f as 5-HT2A antagonist is a novel and feasible therapeutic agent for colorectal cancer treatment.

*Corresponding author : Yong Hyun Jeon

Keywords : 5-Hydroxytryptamine, 5-HT2A receptor, Antagonist, Anti-tumor, Colorectal cancer

PS-A-41

Comparative Evaluation of Water-Bath Sonication and Pipetting for Dispersing Aggregated Extracellular Vesicles

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The stability of extracellular vesicles (EVs) during storage remains a critical factor influencing their utility as drug delivery platforms. While freezing at -70°C is a common preservation method, it often leads to vesicle aggregation, ultimately impairing delivery efficiency and reducing measurable particle numbers. However, strategies to reverse such aggregation without compromising EV functionality have not been fully established. In this study, we examined whether water-bath sonication could effectively disperse aggregated EVs derived from respiratory cells. We used nanoparticle tracking analysis (NTA) to measure particle size distribution and concentration, which revealed that sonication improved particle recovery and reduced size heterogeneity compared to pipetting. To evaluate biological relevance, sonicated and non-sonicated EVs were administered intratracheally to mice. Bronchoalveolar lavage fluid (BALF) was collected, and cellular uptake was assessed using microscopic analysis of BALF cytospin preparations. Notably, sonicated EVs showed improved internalization, while non-sonicated EVs remained predominantly in extracellular aggregates. Additionally, molecular dynamics simulations corroborated the physical dispersal effect of sonication on EV membranes. Our findings highlight water-bath sonication as a rapid and reproducible approach to recover the dispersity and functionality of freeze-thawed EVs. This method may improve the reliability of EV-based delivery systems and facilitate their broader application in preclinical and translational research.

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Keywords : Extracellular vesicles (EVs), Water-bath sonication, Vesicle aggregation, Freeze-thaw stability, Drug delivery systems

PS-A-42

Identification of proteins regulated by baicalin in an ischemic stroke animal model

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Importance : Cerebral ischemia arises from insufficient blood flow to the brain, resulting in significant metabolic disturbances. Baicalin, a flavonoid compound derived from various plants, is renowned for its anti-inflammatory, antioxidant, and neuroprotective properties.

Objectives : This study aimed to identify the proteins regulated by baicalin in a stroke model.

Methods : Middle cerebral artery occlusion (MCAO) was performed on adult male rats, with baicalin (100 mg/kg) or a vehicle administered intraperitoneally 1 h prior to surgery. Neurobehavioral assessments were conducted 24 h after MCAO, and cerebral cortex tissues were harvested for analysis. Cortical damage was assessed using TTC and hematoxylin-eosin staining. Protein expression changes between experimental groups were identified using liquid chromatography-tandem mass spectrometry.

Results : MCAO-induced injury resulted in substantial neurological deficits, infarction, and histopathological damage, all of which were significantly mitigated by baicalin treatment. MCAO injury reduced adenylate cyclase type 1 and solute carrier family 25 member 12 levels, baicalin mitigated the decrease in these proteins. Additionally, baicalin alleviated the MCAO-induced elevations of C-reactive protein and apolipoprotein C-II levels. It also attenuated the MCAO-induced increases in alpha-1 macroglobulin, murinoglobulin, and hemoglobin subunit B levels. These proteins are involved in mitochondrial metabolism, lipid metabolism, inflammation, cell signaling, myelination, and cell damage.

Conclusions and Relevance : This study demonstrates that baicalin exerts neuroprotective effects in cerebral ischemia by modulating proteins involved in energy metabolism, myelination, and neuroinflammation. These findings highlight baicalin as a promising therapeutic agent in stroke models.

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Keywords : Baicalin, Ischemic stroke, Neuroprotection

PS-A-44

High fructose Induced Adipose Inflammation Promotes Systemic Cytokine Crosstalk and Exacerbates Allergic Lung Responses in OVA-Sensitized C57BL/6 Mice

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Excessive fructose intake is linked to systemic inflammation and metabolic dysregulation, but its role in allergic airway disease through adipose-lung immune crosstalk remains unclear. In this study, we examined if and how chronic dietary fructose consumption exacerbated pulmonary inflammation using an animal model focusing on coordinated immune responses in adipose and lung tissues. C57BL/6 mice were fed with low (LFr, 30%) or high (HFr, 50%) fructose diets and sensitized with ovalbumin (OVA) to induce allergic airway inflammation. Histological analyses of lung and adipose tissues demonstrated a stepwise increase in immune cell infiltration, mucus hypersecretion, and mast cell activation, most prominently in the OVA+HFr group compared with those in OVA+LFr group. Lung tissues showed elevated expression of proinflammatory cytokines (TNF- α , IL-1 β , IL-6, IL-17A, IL-4, IL-22, IFN- γ), Th2/Th17 transcription factors (GATA3, ROR γ t), and MMPs, indicative of immune amplification and tissue remodeling. Adipose tissues exhibited parallel immune alterations including adipocyte hypertrophy, mast cell activation, and increased populations of CD11c⁺CD80⁺, CD11c⁺CD206⁺, and F4/80⁺CD80⁺ macrophages, along with upregulation of TLR4 and NLRP3 inflammasome signaling. These immune alterations correlated with systemic cytokine elevation and intensified Th2/Th17 responses in the lung. Collectively, these findings suggest that inflammation in adipose tissues caused by high fructose intake promotes immune activation, which in turn worsens allergic airway inflammation through adipose-lung immune crosstalk.

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*Corresponding author : Youngheun Jee

Keywords : Fructose, Adipose-Lung Crosstalk, Allergic airway inflammation, NLRP3, TLR4

PS-A-43

Enhanced Wound Healing in Rat Anastomotic Leakage and Skin Excision Models Using Gallol-Modified Chitosan Hydrogel Patches

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Gallol-conjugated chitosan-based hydrogel (CHC) was developed as a biocompatible, tissue-adhesive material designed for efficient wound sealing and enhanced healing, particularly under moist and internal surgical environments. This study aimed to comprehensively evaluate the in vivo therapeutic efficacy of CHC hydrogel patches using two distinct rat models: a cecal anastomotic leakage model and a full-thickness dorsal skin excision model. In the gastrointestinal model, forty male Sprague-Dawley rats underwent cecal anastomosis and were assigned to control, CHI-G only, and CHC-treated groups. Rats in the CHC group exhibited significantly reduced local inflammation, minimized fibrous adhesion, and improved anastomotic sealing compared to the other groups. The presence of hyaluronic acid and calcium ions in the CHC network enhanced mechanical integrity, anti-adhesive properties, and tissue compatibility, which helped maintain patch function in the abdominal environment.

In the skin wound model, CHC patches were applied to 1.5 cm full-thickness dorsal excisions. Wounds treated with CHC showed faster wound closure and reached more than 90% healing by postoperative day 14. This performance was similar to or better than that of a commercial polyurethane-based wound dressing. Histological analysis revealed increased granulation tissue formation, denser collagen deposition, and more complete re-epithelialization. Immunohistochemical staining also showed decreased IL-1 β expression and a well-regulated presence of CD3⁺ T cells, suggesting that CHC contributes to immune modulation during wound healing. Taken together, these findings suggest that CHC hydrogel patches improve healing in both internal and external wounds. The combined properties of adhesion, sealing ability, anti-adhesion effects, and immune modulation support their potential for clinical use in surgical wound management.

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Keywords : Gallol-modified chitosan, Hydrogel patch, Anastomotic leakage, Wound healing, Rat model

PS-A-45

In Vivo Evaluation of Double-Layer CHI-G/cHA Adhesive Patch for Preventing Anastomotic Leakage and Postoperative Adhesions in a Rat Model

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Anastomotic leakage (AL) and postoperative adhesion are major complications following gastrointestinal surgery. To address both issues simultaneously, we developed a bioinspired double-layer patch comprising gallol-conjugated chitosan (CHI-G) as a tissue-adhesive layer and crosslinked hyaluronic acid (cHA) as an anti-adhesive layer. In this study, we evaluated the in vivo efficacy and safety of the CHI-G/cHA patch using a rat cecal anastomosis model. A total of 40 Sprague-Dawley rats were divided into five groups (n=8): control (suture only), Mediclore™ (anti-adhesive control), Neoveil™ (adhesive reinforcement), CHI-G only, and CHI-G/cHA patch. The patches were applied to the anastomotic sites and evaluated on postoperative days 7 and 21. The CHI-G/cHA group exhibited significantly higher anastomotic bursting pressures (161.1 $\pm$ 21.6 mmHg at day 21) than all other groups. Histological analyses showed reduced fibrosis and adhesion formation compared to CHI-G or Neoveil™. Furthermore, liver and kidney function markers remained within normal ranges across all groups, indicating no systemic toxicity. Gross adhesion scores were lowest in the CHI-G/cHA and Mediclore™ groups, while inflammatory cell infiltration and fibrosis were minimized in the CHI-G/cHA group without inducing abscess formation. These findings demonstrate that the CHI-G/cHA patch provides both mechanical sealing and anti-adhesion functions, making it a promising candidate for safe and effective management of intestinal anastomosis in surgical settings.

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Keywords : Anastomotic leakage, Postoperative adhesion, Double-layer patch, Gallol-chitosan, Hyaluronic acid

PS-A-46

Nasal epithelial zonation and anatomical features in Infant Marmosets

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Background & Aim : The nasal cavity plays key roles in humidification, filtration, and olfaction. Turbinates regulate airflow and define the spatial distribution of epithelial types—olfactory (OE), respiratory (RE), transitional (TE), and squamous (SE). The common marmoset (*Callithrix jacchus*) is increasingly used in translational research, but detailed anatomical and histological data on its early postnatal nasal development are still lacking. Because the nasal anatomy of non-human primates—including turbinate structure and epithelial zonation—closely resembles that of humans, they serve as useful models for studying human nasal development and function. This study aimed to analyze turbinate morphology and epithelial compartmentalization in neonatal and developing marmosets, compare them with *Macaca fascicularis*, and propose a standardized trimming protocol for consistent histopathological evaluation.

Methods & Results : Micro-CT imaging and H&E staining were performed on marmosets across developmental stages, from birth to adulthood. In neonates (PND ≤ 4 weeks), distinct epithelial zonation was already established. However, active cell proliferation in the squamous epithelium (SE) and a low density of goblet cells in the respiratory epithelium (RE) suggested ongoing tissue growth and immature mucous cell development. Turbinate structures remained simple and underdeveloped at this stage, indicating that both epithelial differentiation and structural maturation are underway. By adolescence (~4 months), turbinates became more branched and segmented. Comparative analysis with adult *Cynomolgus* monkeys revealed species-specific differences in turbinate morphology and epithelial distribution. Based on these findings, we proposed optimal trimming levels for coronal sections to include all epithelial types: in *Cynomolgus* monkeys, between the canine and P1, and M1–M2; in marmosets, between I2–C and P2–P3.

Conclusion : We described the nasal epithelial anatomy of infant and adult marmosets and, through comparison with *Cynomolgus* monkeys, highlighted species-specific differences. This supports the value of marmosets as a model for respiratory and developmental studies.

*Corresponding author : Jun won Park, Byeong Choe Kang

Keywords : Marmoset, Nasal Turbinate, Micro-CT, Nasal Cavity

PS-A-48

Philadelphus schrenkii ameliorates NLRP3 inflammasome-induced inflammatory bowel disease in mice

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NLRP3 inflammasome and reactive oxygen species (ROS) are closely related and are promising therapeutic targets for inflammatory bowel disease (IBD). This study investigated the anti-inflammatory and antioxidant effects of *Philadelphus tenuifolius* (DJ-001) in bone marrow derived macrophages (BMDM) and IBD models. We found that DJ-001 inhibited NLRP3 inflammasome activation using western blot in stimulated BMDM cells with LPS and ATP and inhibited the production of IL-1 β and IL-18 in a dose-dependent manner. Moreover, we observed by cell fluorescence staining that DJ-001 inhibited the co-localization of NLRP3, caspase-1 and ASC by a confocal microscopic analysis. Furthermore, in a dextran sulfate sodium salt (DSS)-induced colitis mouse model, treatment with DJ-001 significantly reduced weight loss and disease activity index (DAI) caused by inflammatory bowel disease (IBD), and protected contraction the length of colon. The inhibitory effect of DJ-001 on IBD was also apparently confirmed by histological characteristics of the epithelial tissues of DSS-induced IBD mice. In addition, we found that the activity of NLRP3 inflammasome was inhibited through inhibition of MAPKs and NF- κ B signaling in colon of the DJ-001 oral administration group. DJ-001 also downregulated NOX1, NOX2 and NOX4 expression and suppressed ROS through upregulation of Nrf1, HO-1 and NQO-1. These results strongly suggest that DJ-001 inhibited DSS-induced IBD through regulation of NLRP3 inflammasome and ROS activation, and it is a promising candidate for therapeutics of IBD. This research was supported by the Bio & Medical Technology Development Program of the National Research Foundation (NRF) funded by the Korean government (MSIT, Project No. 2022M3A9E4095340).

*Corresponding author : Yung Choon Yoo

Keywords : *Philadelphus schrenkii*, Inflammation, MAPK, NLRP3 inflammasome, IBD

PS-A-47

Behavioral analysis of Low-Intensity focused Ultrasound Therapy in a Traumatic Brain Injury Mouse Model

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Traumatic brain injury (TBI) is a leading cause of persistent neurological impairments, and effective non-invasive therapeutic interventions remain scarce. Low-intensity focused ultrasound (LIFUS) has recently emerged as a promising neuromodulatory modality capable of altering neuronal excitability and promoting functional recovery. Previous studies have shown that LIFUS can stimulate neurogenesis, attenuate neuroinflammation, and enhance synaptic plasticity in experimental models of brain injury. In this study, we evaluated the therapeutic potential of LIFUS in a mouse model of TBI induced via stereotactically guided needle insertion targeting the primary motor cortex. Six-week-old male C57BL/6N mice were subjected to unilateral focal injury and randomly allocated to either the LIFUS stimulation or sham control group. LIFUS was delivered transcranially under standardized stimulation parameters for multiple consecutive days following injury. Behavioral assessments were conducted at multiple post-injury time points using paradigms designed to evaluate spontaneous locomotion and recognition memory. Although behavioral differences were detected between the LIFUS-treated and sham groups, the specific characteristics and functional implications of these alterations remain to be elucidated. These preliminary findings suggest that LIFUS may exert modulatory effects on post-injury behavioral outcomes, supporting its feasibility as a non-invasive therapeutic modality for focal cortical injury.

*Corresponding author : Daehong Kim

Keywords : TBI, LIFUS

PS-A-49

Yeokwisan extracts inhibits LPS-induced inflammation in RAW 264.7 macrophages and *Helicobacter felis* infection in mice

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We investigated anti-inflammatory and anti-microbial effect of two extracts of natural herbs (YSE and YSW) on LPS-stimulated RAW 264.7 macrophages and *H. felis* infection in mice, respectively. *Helicobacter* species are Gram-negative bacteria that colonization on the stomach or intestinal mucosa of many mammalian hosts and induce inflammation. YSE, 30% ethanol extract of *Yeokwisan* composed of 6 types of herbs and YSW, a water extract of *Yeokwisan*, were used in this study.

Methods : 1) C57BL/6 mice were infected by bacterial suspensions of *H. felis* by oral gavage. 2) We determined bacterial titers and the level of pro-inflammatory cytokines in mouse gastric tissues by qPCR and ELISA, respectively, 5 weeks after bacterial infection. And the effect of YS extracts on dyspepsia caused by *H. felis* infection was investigated. Anti-inflammatory activity of YSE and YSW was evaluated in LPS-stimulated RAW 264.7 cells.

Results : Mice infected by *H. felis* showed 100% infection rate. Treatment with YSE and YSW significantly inhibited the levels of FlaB gene expression and pro-inflammatory cytokines. Furthermore, both of YSE and YSW significantly inhibited inflammatory responses in LPS-stimulated macrophages. These extracts significantly improved the delay of gastric emptying and intestinal transit induced by *H. felis* infection. YSE and YSW are beneficial natural products for prevention of *H. felis* infection, and their anti-microbial effect against *H. felis* infection might be related to their activity to inhibit inflammation. This research was supported by the Bio & Medical Technology Development Program of the National Research Foundation (NRF) funded by the Korean government (MSIT, Project No. 2022M3A9E4095340).

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Keywords : Inflammation, FlaB gene, *Helicobacter* infection, YS extract, Gastritis

PS-B-01

The anti-allergic effects of *Corchorus olitorius* in mice

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The anti-allergic effects of *Corchorus olitorius* (*C. olitorius*) *in vivo* were investigated. Atopic dermatitis (AD) is a chronic inflammatory allergic disease that is partly driven by type 2 inflammation. To examine the anti-AD activity of *C. olitorius*, we measured clinical skin severity and levels of disease-related immunoglobulin E (IgE) and cytokines (IL-4, IFN- γ , and TNF- α) in BALB/c mice with human-like AD induced by 2,4-dinitro-chlorobenzene. Body weights consistently increased in all groups treated with the test article, whereas the dexamethasone-treated positive control group showed a decrease in body weight over the 7-day dosing period, indicating that *C. olitorius* had no harmful effects on the mice. The group dosed with *C. olitorius* at 200 mg/kg/day showed the most significant skin curative effects. Additionally, all test article-treated groups exhibited reduced type 2 inflammation-related IgE and cytokine levels. These findings suggest *C. olitorius* extract may serve as a potential therapeutic agent for AD.

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Keywords : *Corchorus olitorius*, Anti-atopic dermatitis, Body weight changes, Type 2 inflammation related-immuno globulin and cytokines, BALB/c mouse

PS-B-02

Anticancer effect of vinblastine is associated with counter-regulation of interleukin-24 expression by YAP in human melanoma cells

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Yes-associated protein (YAP) is a transcriptional co-activator that regulates tumor cell growth, survival and metastasis via its activity modulation by phosphorylation. Interleukin-24 (IL-24) is a cytokine involved in immune regulation and effects various cellular processes, including apoptosis and tumor suppression. IL-24 expression in primary melanocytes and early stage melanomas reduces in more advanced melanomas. IL-24 is known to selectively induces cell death in multiple human cancers including melanomas. However, it has to be defined the induction of anticancer drug resistance. Here, we investigated whether vinblastine induces anticancer drug resistance through YAP-associated IL-24 expression using A375 human melanoma cells. While an increase in IL-24 expression and YAP phosphorylation was observed, cell viability was decreased under 0.1% serum conditions compared to that in cultured cells with 10% serum. In addition, IL-24 expression and YAP phosphorylation were increased by vinblastine treatment. GA017, an inhibitor to LATS in hippo pathway reduced YAP phosphorylation and IL-24 expression, leading to an increase in melanoma cell death. Association of YAP and TEAD was increased by GA-017 in nuclear fraction and less binding YAP to IL-24 gene promoter was detected by chromatin immunoprecipitation (CHIP) assay. Increased cell death by vinblastine was also observed in IL-24-knockout (KO) cells. In conclusion, YAP counter-regulates transcriptional activity of IL-24 gene and increased IL-24 expression promotes suppresses melanoma cell death. It suggests that IL-24 leads PC formation in A375 melanoma cells, thereby inducing resistance to anticancer drugs.

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Keywords : Interleukin-24, YAP, Vinblastine, Melanoma, Anticancer drug resistance

PS-B-03

Potential of Aloe vera in ameliorating Inflammatory Bowel Disease via tight junction protection

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Inflammatory bowel disease (IBD) is closely associated with structural and functional abnormalities of the intestinal epithelial barrier. Disruption of intestinal epithelial tight junction (TJ), in particular, is a key pathophysiological mechanism that promotes the aberrant permeation of enteric pathogens and toxins, thereby perpetuating chronic inflammatory responses. This study aimed to investigate the regulatory effects of *Aloe vera* water extract (AVWE) on TJ proteins, as well as its antioxidant and anti-inflammatory properties, utilizing an inflammation-induced Caco-2 cell model. Caco-2 cells were treated with AVWE at varying concentrations, and an inflammatory environment was subsequently induced via stimulation with IL-6 (50 ng/mL). The expression of TJ proteins was assessed through Western blotting. AVWE exhibited high radical scavenging capacity and elevated levels of phenolics and flavonoids, supporting its antioxidant potential. AVWE showed no cytotoxicity up to 100 μ g/mL, alone or with IL-6. Analysis of the intestinal epithelial barrier revealed a significant increase in the expression of the TJ proteins ZO-1 and occludin in response to increasing concentrations of AVWE. Conversely, the expression of the inflammatory mediators COX-2 and iNOS exhibited a concentration-dependent decrease. Notably, the AVWE 100 μ g/mL treatment group exhibited the highest expression levels of ZO-1 and occludin, alongside the most significant suppression of COX-2 and iNOS expression. Liquid chromatography with tandem mass spectrometry (LC-MS/MS) analysis identified five key bioactive compounds: catechin, epicatechin, isorhamnetin, aloin, and quercetin. These compounds possess antioxidant and anti-inflammatory properties. Molecular docking analysis revealed that the identified compounds exhibited strong binding affinities with the key TJ protein ZO-1, suggesting their role in barrier regulation. These findings indicate that AVWE may restore intestinal epithelial barrier integrity by enhancing TJ protein expression and modulating inflammatory responses, highlighting its potential as a therapeutic strategy for IBD.

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Keywords : Inflammatory bowel disease, Intestinal epithelial cell, Tight junction, Inflammatory mediator, Aloe vera water extraction

PS-B-04

Anti-inflammatory and anti-tumor effects of *Gastrodia elata* Blume in an AOM/DSS-induced colitis-associated cancer model

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Gastrodia elata Blume (GEB), a traditional medicinal plant, has been reported to possess anti-inflammatory properties. This study aimed to evaluate the therapeutic potential of GEB in a murine azoxymethane (AOM)/dextran sulfate sodium (DSS)-induced CAC model. Thirty BALB/c mice were randomly assigned into three groups: the control group (CON), the AOM/DSS group (AOM), and GEB+AOM/DSS group (GEB). CRC was injected intraperitoneally with AOM (10 mg/kg) and given three 7-d cycles of 2% DSS drinking water with a 14 d inter-cycle interval. GEB (8 g/kg/day) was administered daily via oral gavage for 12 weeks. AOM mice exhibited significant body weight loss during the third DSS cycle, while GEB treatment mitigated this reduction. Disease Activity Index (DAI) scores were significantly lower in the GEB group compared to the AOM group. GEB significantly reduced the number and size of tumors, particularly those >4 mm. Relative weights of the spleen and colon were elevated in the AOM group and suppressed by GEB. Histological analysis revealed marked reductions in inflammatory cell infiltration and tissue damage in GEB-treated mice. In the GEB-treated group, Alcian blue staining revealed an increased number of goblet cells compared to the AOM group, indicating restoration of mucin-producing cell density, whereas Ki-67 immunostaining showed reduced cellular proliferation in both tumor and non-tumor regions. GEB significantly downregulated pro-inflammatory markers (iNOS, IL-1 β , IL-6, TNF- α) and Wnt signaling proteins (β -catenin, cyclin D1, c-Myc) in colon tissue. These molecular changes suggest that GEB modulates both inflammatory responses and oncogenic signaling cascades in the colon. GEB exerts notable anti-inflammatory and anti-tumor effects in the AOM/DSS-induced CAC model, suggesting its potential as a natural therapeutic agent for inflammation-associated colorectal cancer.

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Keywords : *Gastrodia elata* Blume, Azoxymethane, Dextran Sulfate Sodium, Anti-inflammation, Anti-tumor

PS-B-05

Gastrodia elata Blume enhances immune function in a Cyclophosphamide-induced immunosuppressed BALB/c mouse model

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Cyclophosphamide (CP) is a widely used chemotherapeutic agent known to induce immunosuppression. This study aimed to investigate the immune-enhancing effects of *Gastrodia elata* Blume (GEB), a traditional medicinal herb known for its neuroprotective and anti-inflammatory properties, in a CP-induced immunosuppressed mouse model. Thirty-two male BALB/c mice were divided into four groups: the control group (CON), model group (CP) (80 mg/kg/day, i.p. for 3 days), the low-dose GEB (L-GEB, 4 g/kg), the high-dose GEB (H-GEB, 8 g/kg). CP (80 mg/kg/day) was administered intraperitoneally for 3 consecutive days, and GEB was given orally once daily for 10 days. After sacrifice, body weight, relative organ weights (spleen and thymus), splenic lymphocyte proliferation, histopathological changes, and cytokine levels were assessed. CP significantly reduced body weight, spleen and thymus indices, and lymphocyte proliferative responses under both LPS and ConA stimulation. Both L-GEB and H-GEB administration significantly improved these immune parameters in a dose-dependent manner. H&E staining revealed severe structural disorganization of thymic cortex-medulla and splenic white pulp in CP-treated mice, which was partially restored in animals treated with L-GEB and markedly restored in those treated with H-GEB. ELISA results showed that serum IFN- γ levels were suppressed in the CP group but were significantly increased in both L-GEB and H-GEB groups in a dose-dependent manner. Splenic qRT-PCR showed that mRNA levels of IL-2, IFN- γ , and IL-4 were significantly downregulated in the CP group, but were significantly upregulated following both L-GEB and H-GEB treatment, with the high-dose group showing more pronounced effects. GEB effectively mitigates CP-induced immunosuppression by restoring lymphoid organ structure, enhancing lymphocyte proliferation, and upregulating key cytokines. These findings suggest that GEB has potential as a natural immunomodulatory agent for improving immune resilience during or after chemotherapy, and may serve as a complementary approach to conventional cancer treatment.

*Corresponding author : Jae-Ho Shin

Keywords : *Gastrodia elata* Blume, Immunosuppression, Cyclophosphamide, Immunomodulatory

PS-B-07

Cnidium officinale Hot water Extracts Improve the Intestinal Barrier Dysfunction via Tight Junction Protection in IBD

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Inflammatory bowel disease (IBD) in humans is closely associated with the disruption of the intestinal barrier. Epithelial tight junctions (TJs) are key structures that regulate the paracellular trafficking of macromolecules. These multi-protein complexes prevent the penetration of proinflammatory molecules into mucosal tissues and the circulatory system, while also maintaining the boundary between apical and basolateral membrane domains. Disruption of the intestinal TJ barrier often follows perturbations in the mucosal immune system and inflammation, acting as a trigger for the development of both intestinal and systemic diseases. *Cnidium officinale* extracts (CNOE) have been reported to possess anti-tumor and anti-inflammatory properties in various studies. However, their effect on intestinal barrier function and the underlying mechanisms remain unclear. In this study, we investigated whether CNOE could restore intestinal function and maintain TJ integrity in an experimental model of IBD. Our results demonstrated that CNOE significantly increased transepithelial electrical resistance (TEER), reduced FITC-dextran permeability across the intestinal epithelium, and restored the expression of TJ proteins in interleukin-6 (IL-6)-induced Caco-2 cells. Furthermore, CNOE downregulated inflammatory cytokine expression. In a Dextran Sulfate Sodium (DSS)-induced mouse model, CNOE alleviated body weight loss, reduced the disease activity index (DAI), and prevented colon shortening. In summary, CNOE mitigated intestinal barrier dysfunction by preserving TJ integrity in IL-6- or DSS-stimulated models. These findings suggest that CNOE holds potential as a natural therapeutic agent for IBD by maintaining TJ function.

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Keywords : Intestinal barrier function, Tight junction, DSS-colitis mouse, Inflammatory bowel diseases, *Cnidium officinale*

PS-B-06

Administration of *Gastrodia elata* Blume extract attenuated liver fibrosis in a Thioacetamide-Induced Rat Model

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Liver fibrosis is a progressive pathological condition characterized by excessive extracellular matrix (ECM) deposition due to chronic liver injury. This study investigated the therapeutic effects of *Gastrodia elata* Blume (GEB) on liver fibrosis induced by thioacetamide (TAA) in rats. Six-week-old male Sprague-Dawley rats were randomly divided into the following three groups: i) Control (CON) group, ii) Thioacetamide (TAA) group, and iii) GEB-treated (GEB) group. The TAA and GEB groups were intraperitoneally injected with TAA (200 mg/kg BW, 3 times/week) for 8 weeks to induce liver fibrosis. The CON group received intraperitoneal saline injections. The GEB group received GEB extract (700 mg/kg BW, p.o.). The CON and TAA groups received distilled water. All treatments were administered for a total of 8 weeks. At the end of the experiment, liver tissues and sera were collected. The results of serum levels of ALP, ALT and AST were elevated in the TAA group compared to the CON group. However, these levels decreased in the GEB group. Histological analysis using hematoxylin & eosin (H&E) showed that the TAA group exhibited disrupted hepatic architecture and accumulation of ECM, while these pathological changes were alleviated in the GEB group. Using Masson's trichrome staining to reveal the fibrosis area, and quantified using the Ishak scoring system. The TAA group's liver tissue showed an average Ishak score of 5 due to the accumulation of extracellular matrix, but the GEB group showed a decreased fibrosis area with a score of 2.6. The CON group showed a score of 0. In conclusion, GEB extract prevented TAA-induced liver fibrosis by exerting anti-fibrosis and hepatoprotective effects. This study supports the potential of GEB as a functional therapeutic candidate for hepatic fibrosis.

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Keywords : *Gastrodia elata* Blume, Liver fibrosis, Thioacetamide, Hepatoprotective effects, Anti-fibrosis

PS-B-08

Therapeutic potential of *Cinnamomum cassia* ethanol extracts in preserving intestinal barrier integrity: evidence from in vitro and in vivo models of colitis

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Cinnamomum cassia is a well-known medicinal plant that is acknowledged for its reported therapeutic benefits in a range of diseases; however, there is a lack of research on its medicinal properties specifically in inflammatory bowel disease (IBD). The objective of this study was to investigate the therapeutic effectiveness of *Cinnamomum cassia* with Inflammatory Bowel Disease (IBD). Experiments using an ethanol extract of *Cinnamomum cassia* (ECC) will be completed in vitro. In this study, the Caco2 cell line was utilized as a model for colitis, and treated with IL-6. In this study, the Caco2 cell line was utilized as a model for colitis, and treated with IL-6. Significant antioxidant activity was detected even at a low concentration of 10 μ g/mL in the antioxidant activity assay. Cell viability assays conducted to assess the toxicity of cinnamon revealed cytotoxicity at high concentrations, specifically at or above 200 μ g/mL. Therefore, subsequent experiments were conducted using concentrations of 10, 50, and 100 μ g/mL, which exhibited antioxidant effects without demonstrable cytotoxicity. The research focuses on intestinal barrier dysfunction, which are recognized for their significant involvement in the progression and manifestation of Inflammatory Bowel Disease. ECC significantly increased transepithelial electrical resistance (TEER) at 24h accordance with concentration, and restored the dysfunction of intestinal barrier in interleukin-6 (IL-6)-induced Caco-2 cells. High-performance liquid chromatography (HPLC) analysis of ECC showed the presence of coumarin and 2-methoxycinnamaldehyde. The docking analysis revealed a strong binding interaction between the two active components and the ZO-1 protein, a tight junction protein which was well known as the critical role in intestinal integrity. These findings indicate that ECC may have the ability to preserve the integrity of the intestinal barrier, and our goal is to further investigate and confirm this hypothesis in upcoming research studies.

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Keywords : *Cinnamomum cassia*, Therapeutic benefits, Inflammatory bowel disease (IBD), Intestinal barrier dysfunction, Intestinal integrity

PS-B-09

Optimization Study of an Atopic Dermatitis Model Induced by MC903 in C57BL/6 Mice: Comparison of Inflammatory Markers and Application Sites

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This study aimed to optimize an MC903 induced atopic dermatitis (AD) mouse model, commonly used in therapeutic research by evaluating inflammatory markers in relation to induction conditions and application sites. A 28-day experimental period was conducted using MC903, a vitamin D3 analog, instead of conventional house dust mite extract to induce atopic dermatitis. MC903 was applied once daily, with variations in the total number of applications and the dosage per application, in order to determine the optimal induction conditions.

The agent was applied simultaneously to both the ears and the back skin of the mice. On day 28, tissues were collected and RNA was extracted to analyze expression levels of TNF- α , IL-6, TSLP, and IL-13. The results demonstrated that chronic MC903 treatment for 28 days significantly increased inflammatory marker levels compared to a 14-day treatment. Moreover, the ear was found to be more susceptible to inflammation than the back.

Changes in body weight and visible assessment of skin lesions followed similar trends to the expression of inflammatory markers. These findings suggest that if the goal is to establish a moderate-to-severe AD model for therapeutic evaluation, applying MC903 to the ears for a full 28-day period is most effective. Among the inflammatory biomarkers assessed, IL-6, TSLP, and IL-13 are recommended as key markers for evaluation. Based on the findings of this study, additional research will focus on identifying the optimal induction conditions by varying the type of vehicle used to deliver the inducing agent, MC903.

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Keywords : MC903, Atopy, Dermatitis, Inflammatory markers, TSLP

PS-B-11

Optimization Study of MPTP-Induced Parkinson's Disease Mouse Model: Effects of MPTP Dosage and Exposure Duration

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MPTP (1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine) is a neurotoxin widely used to induce Parkinson's disease (PD)-like symptoms in mice and rats, mirroring the pathophysiology seen in human PD. Since its introduction in the early 1980s, MPTP has become a commonly used agent in PD research, yet optimal experimental conditions remain inconsistent across laboratories. While many studies have applied doses up to 30 mg/kg to obtain motor impairment, this study aimed to confirm such conditions and determine the most effective protocol for evaluating motor function deficits over time using tests such as the Rotarod and Grip strength test.

The study first evaluated motor function for up to 49 days following 30 mg/kg MPTP administration. Statistically significant differences between groups were observed as early as day 21, suggesting that prolonged observation may not add further benefit and raises questions about the necessity of chronic induction protocols. Subsequently, the study employed a higher dose of MPTP (40 mg/kg) over a shorter period (22 days), incorporating the Rotarod test, Pole test, and Grip strength test.

The results demonstrated that while 30 mg/kg MPTP led to noticeable motor deficits, administration of 40 mg/kg induced stronger and more sustained impairments, particularly in the Pole and Rotarod tests. However, the Grip strength test showed inconsistent outcomes, likely due to the mice's voluntary effort, making it an unreliable tool for PD assessment.

In conclusion, comparative evaluation between 30 mg/kg MPTP for 49 days and 40 mg/kg for 22 days revealed that the latter yields more discriminative and consistent results. Moreover, the study suggests that the Grip strength test is not suitable for assessing Parkinsonian motor symptoms in mice.

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Keywords : MPTP, Parkinson's disease, Dosage, Motor function deficits

PS-B-10

The therapeutic effects of human neural stem cells overexpressing neuroglobin in APPSwe/PS1dE9 mice

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Background : Neuroglobin (NGB) is a neuron-specific oxygen-binding protein known to protect against hypoxia and oxidative stress. Recent studies have suggested its involvement in Alzheimer's disease (AD) pathology, particularly in the regulation of neuroinflammation, amyloid-beta (A β) accumulation, and neuronal survival. However, the biological significance of NGB in the AD brain remains poorly understood, and it has not been clearly demonstrated whether its overexpression can provide therapeutic benefits. **Methods :** Human neural stem cells overexpressing NGB (F3.NGB) were evaluated for anti-inflammatory and anti-apoptotic effects in vitro using A β -treated N2A cells. The mRNA and protein levels of inflammatory (COX-2, IL-6, iNOS, TNF- α , NF- κ B) and apoptotic (BAX, cleaved caspase-3) markers were measured via qPCR and Western blot. In vivo, F3 and F3.NGB cells were transplanted into APPSwe/PS1dE9 transgenic mice. Behavioral tests and molecular analyses were conducted to assess cognitive function, cholinergic activity, A β burden, neurotrophic factor expression, and neuronal differentiation. **Results :** In vitro, F3.NGB co-culture significantly attenuated A β -induced inflammation and apoptosis while activating PI3K/AKT signaling. In vivo, F3.NGB transplantation improved memory performance to near-normal levels, restored ChAT, VACht, and CHT expression, and normalized acetylcholine levels. Neurotrophic factors (BDNF, NGF, CNTF, GDNF) and PI3K/AKT/mTOR signaling were upregulated, and A β accumulation was reduced via IDE and NEP induction and BACE1 suppression. Immunohistochemistry confirmed reduced plaque burden and neuronal differentiation of transplanted cells.

Conclusion : NGB overexpression enhances the therapeutic effects of human neural stem cells by modulating multiple AD-related pathologies. These findings suggest that F3.NGB cells represent a promising therapeutic strategy for the treatment of Alzheimer's disease.

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Keywords : Alzheimer's Disease, Human Neural Stem Cell, Neuroglobin

PS-B-12

Vapor self-administration reveals the efficacy of a vaccine against synthetic cannabinoid reinforcement in mice

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Synthetic cannabinoid receptor agonists (SCRA) are novel psychoactive substances that act more potently on CB1 and CB2 receptors than Δ 9-THC. These compounds are predominantly abused via inhalation routes using electronic cigarettes, and their higher efficacy and affinity for CB1 receptors compared to Δ 9-THC pose a significant risk of neurological side effects. The growing prevalence of these substances has become a societal burden due to the lack of inhalation-based dependence assessment models and the ability of these compounds to evade legal regulation through structural modifications. To address this issue, we employed a SCRA vaccine and evaluated its efficacy using an inhalation engineered self-administration system designed with a electronic cigarette. Unlike conventional vapor self-administration systems, our setup delivers the drug only within the nose poke port, thereby enhancing voluntary drug-seeking behavior in mice. This feature improves the sensitivity of the experiment and enhances face validity. Male C57BL/6 mice were used for the experiments. Mice that received SCRA delivery exhibited higher active nose-poke responses compared to the vehicle group; however, this increase was not observed in the vaccine-treated group. This suggests a reduction in both reinforcement-like behavior and drug-seeking behavior. This effect is presumed to result from the neutralizing action of the antibodies, which likely decreased the BBB permeability of the drug. Antibody production was confirmed via ELISA, with an average concentration of 1.5 mg/mL. To verify the binding affinity of the generated antibodies, K_d was measured using Octet BLI, indicating neutralization effect. Taken together, our findings provide a refined inhalation-based self-administration model that closely mimics the human route of SCRA intake. By integrating a precisely targeted vapor delivery system with voluntary operant behavior, we demonstrate an approach that enhances face validity, experimental sensitivity. Furthermore, we evaluate the efficacy of SCRA vaccine using this system. These methodological advancements represent important progress in developing preclinical tools for assessing the abuse potential of inhaled psychoactive substances and for testing novel therapeutic strategies.

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Keywords : Addiction, Self-administration, E-cigarette, Synthetic cannabinoid receptor agonist, Vaccine

PS-B-13

Pathological role of cleaved crystallin alpha B in mediating angiogenesis and fibrosis in age-related macular degeneration

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Age-related macular degeneration (AMD) is a leading cause of blindness in the elderly, with macular fibrosis caused by choroidal neovascularization (CNV) being a major contributor to vision loss. In our previous research, we identified an association between elevated levels of crystallin alpha B (Cryab), a member of the small heat shock protein family, and proliferative vitreoretinopathy (PVR), a condition characterized by the growth of fibrotic, contractile membranes. In this study, we further investigated the role of Cryab in fibrosis triggered by CNV. Our findings show that Cryab levels were significantly elevated in the aqueous humor of AMD patients. In a laser-induced CNV mouse model, Cryab knockout (KO) mice exhibited a reduced CNV area and lower expression of angiogenic factors and myofibroblast markers compared to wild-type (WT) mice. Interestingly, we also discovered that Cryab exists in a cleaved form, with the C-terminal removed, in CNV-induced mice. Further investigation revealed Prss1, a serine protease, as a binding partner for Cryab, suggesting that it may facilitate Cryab cleavage and promote its interaction with fibrosis-related molecules. Treatment with a Cryab inhibitor, 3-methylglutamic acid, alleviated the pathological progression of CNV, including the expression of fibrosis markers and VEGF, and also reduced the expression of the cleaved form of Cryab. Collectively, these findings suggest that Cryab plays a key pathogenic role in AMD by regulating both angiogenesis and fibrosis. Further research is needed to fully understand how the cleaved form of Cryab influences these processes.

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Keywords : Age-related macular degeneration, Choroidal neovascularization, Crystallin alpha B, Angiogenesis, Fibrosis

PS-B-14

Modeling prodromal Alzheimer's disease: Longitudinal behavioral and activity monitoring in amyloid-induced non-human primates

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This study investigated activity-related and behavioral alterations associated with the early progression of Alzheimer's disease (AD) using a long-term non-human primate (NHP) model. The model was established by repeated cisterna magna (CM) injections of amyloid beta 1-42 (Aβ1-42) oligomers in NHPs. Behavioral and cognitive functions were longitudinally monitored via a touchscreen-based testing system, while daily activity patterns were continuously tracked using a wearable accelerometer. Following Aβ injections, marked disruptions in rest-activity cycles were observed, including increased daytime and nocturnal activity and a shift in peak activity timing—suggesting circadian rhythm disturbances similar to those in early-stage AD patients. These alterations peaked approximately one month post-injection and gradually attenuated over time. Behavioral assessments revealed psychological changes, such as increased stubbornness and reduced motivation, paralleling affective symptoms of early AD. However, no significant cognitive impairments were detected up to nine months post-injection. This NHP model effectively captures early behavioral and circadian features of AD, offering a valuable platform for future neuroimaging and biomarker-based investigations.

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Keywords : Alzheimer's disease, Non-human primate, Behavioral assessment, Circadian rhythm, Amyloid beta oligomer

PS-B-15

The Abuse liability of Cigarette Smoke Condensate in Rodents

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Tobacco smoke contains nicotine alkaloids that are less potent than nicotine, but are still pharmacologically active. Interestingly, recent studies suggest that these minor nicotine alkaloids may also play a pivotal role in tobacco dependence. Here we examined addiction-related behavior effects of nicotine alone and nicotine dose-equivalent concentration of cigarette smoke condensate (CSC) on self-administration (SA) and conditioned place preference (CPP) in rodents. In the CPP test, male ICR mice were conditioned with nicotine (0.5 mg/kg, s.c.) or CSC (0.125, 0.25, 0.5 mg/kg nicotine content, s.c.) for 6 consecutive days. The CSC at a low dose of 0.125 mg/kg nicotine content showed only a significant place preference compared with vehicle group. Furthermore, the administration of CSC decreased place preference in dose-dependence manner. These results indicated that the high dose of CSC may cause aversive behavioral effects in CPP paradigm. In the SA test, male Sprague-Dawley rats were intravenously self-administered nicotine (0.015 mg/kg/infusion) or CSC (0.015 mg/kg/infusion nicotine content) during a daily session until responding was stable. In this experiment, the CSC was more potent than nicotine alone in drug-maintained responding. Collectively, these findings indicate that the minor nicotine alkaloids of CSC have nicotine's reinforcement-enhancing and aversive effects in animal models.

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Keywords : Cigarette smoke condensate, abuse liability, reinforcing effect, rewarding effect, nicotine

PS-B-16

Effects of acetaminophen on cholinergic system and muscle contraction

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Nonsteroidal anti-inflammatory drugs (NSAIDs) are widely used to manage pain and inflammation in neurodegenerative diseases. However, acetaminophen's effect on cholinergic system and muscle contraction pathways remains unclear. We investigated long-term acetaminophen's impact on these pathways in neural stem cells, myoblasts, and rats. Acetylcholinesterase (AChE) activity was measured. Human neural stem (F3) and myoblast (C2C12) cells were treated with N-arachidonoylphenolamine (AM404) (0, 0.1, 0.3, and 1.0 µg/ml). For the in vivo test, SD rats received oral acetaminophen (0, 10, 100, and 1000 mg/kg) for 4 weeks. Hepatotoxicity and brain and sciatic nerve cholinergic markers were assessed; muscle contraction proteins in the gastrocnemius and soleus muscles were examined. AChE activity was inhibited at 8, 31, 250, 500, and 1000 µg/mL. AM404 treatment significantly increased transient receptor potential vanilloid type 1 (TRPV1), Cannabinoid receptor type 1 (CB1), Choline transporter (CHT), Choline acetyltransferase (ChAT), and Vesicular acetylcholine transporter (VACHT) expression in F3 cells, and Cholinergic receptor nicotinic alpha 1 subunit (CHRNA1), Ryanodine receptor 1 (RyR1), and Sarcoplasmic reticulum calcium ATPase 1 (SERCA1) expression in C2C12 cells. In rats, acetaminophen 1000 mg/kg significantly increased alanine aminotransferase, aspartate aminotransferase, and alkaline phosphatase levels, and H&E staining showed glycogen accumulation. At 100 and 1000 mg/kg, acetaminophen increased brain and sciatic nerve TRPV1, CB1, CHT, ChAT, and VACHT expression and cerebrospinal fluid ACh concentration. CHRNA1, RyR1, and SERCA1 expression increased in the gastrocnemius and soleus muscles. Therefore, long-term low-dose acetaminophen improves cognitive function and muscle activation, while providing antipyretic and analgesic effects in elderly without causing hepatotoxicity.

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Keywords : Acetaminophen, AM404, Cholinergic system, Muscle contraction, Cannabinoid

PS-B-17

The anti-inflammatory and anti-oxidant effects of *Caesalpinia sappan* L. heartwood on an LPS-induced mouse model and RAW264.7 cells

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Caesalpinia sappan L., widely distributed in Southeast Asia, is known for its analgesic, antioxidant, antidiabetic, anti-inflammatory, and anticancer activities. In this study, we explored the anti-inflammatory and antioxidant effects of the methanolic extract of *C. sappan* (CS) using a mouse model of lipopolysaccharide (LPS)-induced acute lung injury (ALI) and LPS-stimulated RAW264.7 cells. Mice were administered CS at doses of 200 and 400 mg/kg/day and dexamethasone (3 mg/kg/day) from days 1 to 5 of the experimental protocol. LPS was intratracheally administered at 20 µg/50 µL/mouse for three days. On day 7, the mice were sacrificed and collected bronchoalveolar lavage fluids (BALF) and lung tissue. In LPS-stimulated RAW264.7 cell, cytokines were measured. Treatment with CS decreased the production of tumor necrosis factor (TNF)-α, interleukin (IL-6), and IL-1β in LPS-induced ALI and LPS-stimulated RAW264.7 cells accompanied by the enhanced expression of mitogen-activated protein kinase (MAPK) and nuclear factor-kappa B (NF-κB). In addition, CS treatment elevated the nuclear translocation of Nrf2-related factor with an increase in antioxidant enzymes. This study demonstrated that CS effectively attenuated anti-inflammation induced by LPS induced through the enhancement of NF-κB and MAPKs suppression and expression of Nrf-2. Therefore, CS may be a potential therapeutic agent for ALI.

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Keywords: *Caesalpinia sappan* L., Airway inflammation, Oxidative stress, MAPKs, Nrf-2

PS-B-18

***Boehmeria nivea* mitigated OVA-induced allergic asthma by suppressing Th2-mediated immune responses through the inhibition of Th2 cell differentiation.**

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Allergic asthma is chronic respiratory disease orchestrated by dendritic cells (DCs) and T cell. There is a need to identify new therapeutic strategies for asthma because current treatments have limitations. Because of these limitation, herbal medicine is considered to be alternative treatment for allergic asthma. *Boehmeria nivea* (BN), a plant of the Urticaceae family, is a traditional medicine that has been used for the treatment of fever, diarrhea, wounds, edema, and colds. This study demonstrated to the potential pharmacological properties of active fractions of BN on ovalbumin (OVA)-induced asthma model and lipopolysaccharide (LPS)-induced bone marrow-derived dendritic cells (BMDCs) with tumor necrosis with factor (TNF)-α-stimulated NCI-H292 cells. Various fractions of BN (100 mg/kg) were orally administered to OVA-induced asthma models to investigate the active fraction by measuring T helper 2 (Th2) cytokine (IL-4, IL-5 and IL-13) levels and counting of macrophages and eosinophils in bronchoalveolar lavage fluid (BALF). This study revealed the significant efficacy of active fraction of BN (EA-BN; ethyl acetate fraction of BN) in the treatment of OVA-induced asthma. EA-BN suppressed extracellular cytokine (IL-10, IL-12, and TNF-α) and surface molecules, including major histocompatibility complex (MHC)-II, cluster of differentiation (CD)80 and CD86 in LPS-induced BMDC and splenic DCs from OVA-induced mice. In addition, EA-BN treatment decreases GATA-3 expression in OVA with or without-stimulated CD3+CD4+ T cell correlated to attenuation of airway hyperresponsiveness and mucus overproduction in OVA-induced mice. EA-BN suppress Th2 cytokines production in TNF-α-stimulated NCI-H292 cells. These findings suggest that EA-BN could serve as a useful therapeutic agent for the treatment of allergic asthma.

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Keywords: Allergic asthma, *Boehmeria nivea*, Bone marrow-derived dendritic cell, Th2 cell differentiation

PS-B-19

SARS-CoV-2 pseudovirus dysregulates hematopoiesis and induces inflamming of hematopoietic stem and progenitor cells

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Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection primarily affects the respiratory system but may induce hematological alterations such as anemia, lymphopenia, and thrombocytopenia. Previous studies have reported that SARS-CoV-2 efficiently infects hematopoietic stem and progenitors (HSPCs); however, subsequent effects on hematopoiesis and immune reconstitution have not yet been described. Here, we evaluated the pathological effects of infection of umbilical cord blood (UCB)-derived HSPCs with a SARS-CoV-2 Omicron variant pseudovirus (PsV). Transcriptomic analysis of Omicron PsV-infected HSPCs showed upregulation of genes involved in inflammation, aging, and the NLRP3 inflammasome, suggesting possible inflamming trigger. Omicron PsV-infected HSPCs exhibited decreased numbers of multipotential progenitors (CFU-GEMM) ex vivo and repopulated primitive HSCs (Ki-67-hCD34+ cells) in an HSPC transplantation NSG mouse model (Omicron mouse). Furthermore, Omicron PsV infection induced myeloid-biased differentiation of HSPCs. Treatment with nanographene oxide (NGO), an anti-viral agent, partially mitigated the myeloid bias and inflamming phenotype both *in vitro* and *in vivo*. These findings provide insights into the abnormal hematopoietic and immune effects of SARS-CoV-2 infection and highlight potential therapeutic interventions.

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Keywords: Hematopoietic stem cell, SARS-CoV-2, Humanized mouse, Pseudovirus

PS-B-20

Automated characterization of cocaine-induced behavior in marmosets using deep learning

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Video-based behavior recognition plays a critical role in neuroscience research by enabling the objective analysis of diverse and complex behavioral patterns. However, current methods for behavior recognition in non-human primates (NHPs), such as marmosets, often require labor-intensive manual analysis, are time-consuming, and lack standardized evaluation protocols. This presents a major barrier, particularly in addition research, where understanding drug-induced behavioral alterations is essential for developing pharmacological interventions. In this study, we utilized deep learning algorithms to examine behavioral responses in common marmosets (*Callithrix jacchus*) following cocaine administration. We focused on detecting rapid and repetitive psychomotor behaviors, especially head shaking, which is commonly observed in response to psychostimulants. To improve both accuracy and scalability in behavioral assessment, we utilized an automatic behavior classification algorithm, an open-source tool capable of analyzing video-based behavioral data without human supervision. The automatic behavior classification algorithm employs both pattern image extraction and animation-based features to capture spatiotemporal characteristics, enabling precise deep neural network-based behavior classification. Our algorithm demonstrated strong performance, achieving 90% accuracy, 94% sensitivity, and a 92% F1 score in correctly identifying cocaine-induced behavior categories. These research results highlight the potential of AI-driven behavior analysis tools for advancing research in behavioral neuroscience, especially in the context of drug-induced behavioral phenotyping, and establish a scalable framework for investigating substance-induced behaviors in neuroscience research.

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Keywords: Marmoset, Deep learning, Cocaine, Stereotyped Behavior, Drug-Induced Behavior

PS-B-21

Pretreatment with 2-arachidonoylglycerol attenuates cocaine-taking behavior by modulating dopamine transporter and GPR55 availability in common marmosets

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Drug addiction is intricately associated with various neurotransmitter and neuromodulatory systems, including the endocannabinoid system. However, the precise impact of endogenous cannabinoids, particularly on cocaine-taking behaviors, remains inadequately understood, especially in non-human primate models. Addressing this knowledge gap, our study established a robust cocaine self-administration model using non-restraint common marmosets (*Callithrix jacchus*). We then used this model to investigate the effects of 2-arachidonoylglycerol (2-AG) on cocaine-taking behavior. In this experimental design, cocaine oral self-administration was performed with cocaine dissolved in dextrose solution at a concentration of 0.4 mg/mL, administered at 0.1 mL per response. The marmosets underwent pretreatment with either a control vehicle or two different doses of the endogenous cannabinoid 2-arachidonoylglycerol (2-AG) at 0.01 or 0.1 mg/kg intraperitoneally to investigate its modulatory effects on drug-seeking behavior. Marmosets showed significant active lever responses in cocaine self-administration, and cocaine altered binding potential of DAT. In ex vivo study, availability of DAT and G protein-coupled receptor 55 (GPR55) was reduced and increased, respectively by cocaine treatment. Furthermore, we observed that an intraperitoneal pretreatment of 2-AG reduced active lever responses for cocaine infusion significantly. While another endocannabinoid, 2-LG, did not. 2-AG concentration of the plasma from cocaine self-administered marmoset has decreased in comparison with these of naïve marmosets. However, 2-AG pretreatment rescued the reduction of plasma 2-AG concentration. Taken together, pharmacological interventions, such as 2-AG treatments, may ameliorate cocaine-taking behaviors.

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Keywords : 2-arachidonoylglycerol, Cocaine, Endocannabinoid system, Marmoset, Self-administration

PS-B-23

Fibrotic Liver-Derived Organoids Provide a Physiologically Relevant Model of Liver Fibrosis

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Conventional liver fibrosis models typically employ healthy liver organoids treated with pro-fibrotic stimuli, such as TGF- β 1 or fatty acids. While these *in vitro* models provide high experimental reproducibility, they fall short of fully capturing the complex pathophysiological features of *in vivo* fibrotic liver tissue, including basal inflammation, extracellular matrix (ECM) remodeling, metabolic dysregulation, and impaired hepatic function. To determine whether organoids derived directly from fibrotic liver tissue better reflect *in vivo* disease states, we compared the transcriptomic and drug response profiles of TGF- β 1-treated healthy mouse liver organoids (*in vitro* fibrosis mLOs) with those of fibrotic liver-derived organoids (*in vivo* fibrosis mLOs). Notably, only the *in vivo* fibrosis mLOs exhibited drug responses consistent with *in vivo* outcomes, as indicated by changes in pro-fibrotic marker expression following anti-fibrotic treatment. Targeted transcriptomic analysis of gene clusters associated with liver metabolism and fibrosis revealed that the expression patterns in *in vivo* fibrosis mLOs closely resembled those observed in fibrotic liver tissue. Volcano plots and heatmaps based on these gene sets highlighted this similarity, whereas *in vitro* fibrosis mLOs showed distinct expression profiles, indicating that *in vivo* fibrosis mLOs retain disease-relevant transcriptional signatures at both the gene and pathway levels. Gene Ontology (GO) analysis of downregulated genes after anti-fibrotic treatment showed significant enrichment of terms related to collagen-containing ECM, supporting the drug's efficacy in attenuating fibrotic activity. Collectively, these results demonstrate that organoids derived from fibrotic liver tissue retain critical transcriptomic and functional features of the diseased state. This model offers a physiologically relevant platform for mechanistic studies and high-fidelity drug screening in liver fibrosis research.

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Keywords : Liver organoid, Original tissue-reflected model, Liver fibrosis, Drug responsiveness

PS-B-22

E-merging evidence: Activation of $\alpha 4 \beta 2$ nicotinic acetylcholine receptors by synthetic nicotine analogs in e-cigarettes

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The increasing popularity of single-use e-cigarettes among global youth has paralleled the rise of synthetic nicotine analogs and non-nicotine additives. These substances are often marketed as alternatives to conventional tobacco smoking, claiming to replicate its sensory experience while avoiding the same regulatory restrictions. However, their safety remains largely unverified, raising concerns about youth exposure to unregulated and potentially harmful compounds. Owing to their structural similarity to nicotine, synthetic analogs found in e-liquids may share the ability to activate nicotinic acetylcholine receptors (nAChRs) critically involved in the development of nicotine dependence. To address this, we conducted receptor-based screening to assess the dependence liability of these compounds using Fura-2, AM calcium imaging in $\alpha 4 \beta 2$ nAChR-expressing HEK293 cells. Based on their receptor activation profiles, compounds were classified into two groups depending on whether they elicited measurable receptor activation. We also investigated how co-application of analogs and flavoring agents modulated receptor activity. This *in vitro* platform provides an essential first step in identifying substances that may mimic or potentiate the addictive effects of nicotine, offering foundational evidence to support regulatory strategies in response to rapidly evolving trends in e-liquid formulations.

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Keywords : Nicotinic acetylcholine receptor, Nicotine analogs, Calcium imaging

PS-B-24

Hepatic B cells aggravate liver fibrosis through interaction with NKT cells via CD40

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Background/Aim : Despite evidence that hepatic B cells contribute to liver fibrosis, the exact mechanisms by which they do so remain undefined. CD40, a co-stimulatory molecule expressed on antigen-presenting cells, including B cells, mediates interactions with T cells and is essential for B cell proliferation, differentiation, and antibody production. So, we explore how B cells influence liver fibrosis through interactions with natural killer T (NKT) cells via CD40.

Methods : To investigate this, carbon tetrachloride (CCl₄) was administered to wild-type (WT) and B cell-specific CD40-deficient (CD40 bKO) mice, followed by blood chemistry, histological staining, and qRT-PCR analyses. Isolated hepatic cells were analyzed using bulk RNA sequencing (bulk RNA-seq), single-cell RNA sequencing (scRNA-seq), and flow cytometry.

Results : Bulk RNA-seq and flow cytometry revealed upregulation of B cell activation genes, including *Cd40*, and increased numbers of infiltrating B cells in the liver of CCl₄-treated WT mice compared to vehicle-treated controls. scRNA-seq confirmed significantly elevated *Cd40* expression in B cells alongside augmented *Cd40lg* levels in NKT cells in fibrotic livers of WT mice. Together with increased CD1d and MHCII protein levels and lipid metabolism in B cells of fibrotic liver, it suggests a potential interaction between B cells and lipid-antigen-sensing NKT cells. However, chemokines facilitating mutual recruitment in both cells lack in the liver. Instead, scRNA-seq and qRT-PCR analyses identified activated hepatic stellate cells (HSCs) as the principal source of chemokines recruiting both cell types. These results suggest that B cells recruited to fibrotic septa by HSCs become activated via CD40-CD40L interactions with NKT cells and exert pro-fibrotic functions. Finally, ALT levels, immune cell infiltration, and collagen deposition were significantly lower in CD40 bKO mice than WT mice.

Conclusion : Collectively, these findings suggest that activated HSCs recruit and mediate interactions of pro-fibrotic B cell and NKT cells through CD40-CD40L, highlighting CD40 as a promising therapeutic target for liver fibrosis.

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Keywords : Liver fibrosis, Hepatic B cell, NKT cell, CD40, Hepatic stellate cell

PS-B-25

Large-scale profiling of coding and long noncoding transcriptomes in the hippocampus of mice acutely exposed to vaporized CBD or THC

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Cannabis vaping, particularly involving cannabidiol (CBD) and delta-9-tetrahydrocannabinol (THC), rapidly delivers highly concentrated cannabinoids to the brain, potentially affecting the hippocampus. This study examined differential expression of long noncoding RNAs (lncRNAs) and mRNAs in the hippocampus after acute exposure to vaporized CBD or THC. Male ICR mice were exposed to vaporized CBD or THC (50 mg, n=5/group), and hippocampal tissues were collected at 1, 3, and 14 days post-exposure. Total RNA sequencing was conducted on day 1 samples, and selected transcripts were validated using qRT-PCR across multiple time points. CBD led to significant up- or downregulation of *L3mbtl1*, *Wnt7a*, and *Camk2b* at day 1. However, *Wnt7a* showed gradual recovery at days 3 and 14. In the THC group, *Grin2a*, *Gria3*, and *Golga2* were significantly upregulated, while *Drd1*, *Drd2*, *Gnal*, and *Adcy5* were significantly downregulated at day 1. Time-course analysis showed that *Drd2* expression returned to baseline by day 14, whereas *Adcy5* remained persistently downregulated through days 3 and 14. In the CBD group, *NONMMUT069014.2* was upregulated, while *NONMMUT033147.2* and *NONMMUT072606.2* were downregulated at day 1; notably, *NONMMUT072606.2* showed a transient increase at day 3 before returning to baseline. In the THC group, *NONMMUT085523.1* and *NONMMUT123548.1* were upregulated, whereas *NONMMUT019734.2*, *NONMMUT057101.2*, and *NONMMUT004928.2* were downregulated, with most showing gradual recovery by day 14. Correlation analysis revealed that THC-responsive lncRNAs—including *NONMMUT004928.2*, *NONMMUT057101.2*, and *NONMMUT019734.2*—were strongly associated with downregulated mRNAs such as *Drd2* and *Adcy5*. These findings highlight cannabinoid-specific hippocampal transcriptomic responses and suggest potential regulatory roles for lncRNA-mRNA interactions in cannabinoid-induced neural changes.

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Keywords : Cannabidiol, Delta-9-tetrahydrocannabinol, Hippocampus, Transcriptome, Vaping

PS-B-26

Menthol Enhances Vaporized Nicotine Self-Administration in Mice

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Self-administration is one of gold standard experimental paradigms used in neuroscience to study animal's drug addiction behaviors with Intraperitoneal or intravenous drug administration. However, these methods are different from the clinical administration route, such as nicotine. We measured reinforcement of nicotine in self-administration for mice following clinical administration route. The vehicle and nicotine group of mice self-administered on differently fixed ratio (FR) schedule for 1 h/day sessions. 3, 6, and 12 mg/mL nicotine were self-administered at FR1 condition. 0, 15, 30 mg/mL menthol flavor were detected at FR2 condition. Compared to the vehicle group, active nose poke responses of 6 mg/mL nicotine group shown increased trend in FR1 and was significantly increased in FR2. Active nose poke responses of 6 mg/mL nicotine group were significantly increased after 30 mg/mL menthol treatment together compared to only 6 mg/mL nicotine group. The vapor delivery number of 6 mg/mL nicotine group were significantly increased compare with vehicle group at FR2 condition. There was no significantly difference in inactive nose poke responses between vehicle and nicotine group. It suggested that nicotine vapor had a higher reinforcing effect in mice. The mouse vapor self-administration can make a significant contribution to the drug reinforcement evaluation.

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Keywords : Self-administration, Nicotine, Menthol, Reinforcement behavior

PS-B-27

Reprogramming Tumor-Associated Macrophages with Sulfanilamidindazole (SAI) for Antitumor Immunity

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Background : Modulating tumor-associated macrophages (TAMs) has emerged as a promising strategy in cancer immunotherapy. TAMs within the tumor microenvironment often exhibit an M2 phenotype that promotes tumor progression and suppresses antitumor immunity. In this study, we investigated the potential of a novel indazole-based compound, sulfanilamidindazole (SAI), to reprogram TAMs and enhance antitumor responses.

Methods : In vitro experiments were conducted using RAW264.7 and THP-1 macrophages to assess the immunomodulatory effects of SAI. M1/M2 macrophage polarization was evaluated through phenotypic markers. For in vivo analysis, Balb/c mice bearing 4T1 breast tumors were orally administered SAI at doses of 10 mg/kg and 50 mg/kg daily. Tumor growth was monitored, and tumor-infiltrating immune cell populations were assessed using flow cytometry.

Results : SAI treatment promoted M1 polarization in both RAW264.7 and THP-1 macrophages in vitro. TAMs isolated from 4T1 tumors also shifted toward the M1 phenotype following SAI treatment. In vivo, oral administration of SAI led to significant, dose-dependent tumor growth inhibition without evident toxicity. Flow cytometry revealed increased infiltration of CD8⁺ T cells, NKT cells, and enhanced expression of interferon-gamma (IFN- γ), while regulatory T cell (Treg) levels remained stable.

Conclusion : SAI demonstrated dual activity by both directly inhibiting tumor growth and modulating the tumor immune microenvironment through TAM reprogramming. These findings support the continued development of SAI as an orally available immunotherapeutic agent for solid tumors.

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Keywords : Sulfanilamidindazole (SAI), Tumor-associated macrophages, M1 polarization, Immunotherapy, Tumor microenvironment

PS-B-28

Potential role of T cells and IL-1 in COVID-19 mRNA vaccine-induced heart injury

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The mRNA vaccine is an advanced vaccine introduced to end the pandemic caused by COVID-19. Although mRNA vaccines have been observed to be highly effective, there have been reports of adverse effects such as anaphylaxis, pericarditis, myocarditis, and thrombosis. Notably, myocarditis is believed to be caused by a combination of T cells and macrophages, although the specific mechanisms and consequences remain unclear.

To investigate the roles of T cells and macrophages in myocarditis induced by mRNA vaccination, we adopted a nude mouse model, which is T cell-deficient. Both models were administered CUK3/LNP128, a novel SARS-CoV-2 mRNA vaccine recently developed, twice at two-week intervals by intramuscular injection.

Following mRNA vaccination, key indicators of myocardial injury, such as serum troponin-I levels and the myocardial Myh7/Myh6 ratio, were significantly elevated in both WT and nude mice. Conversely, histological analysis revealed that mononuclear cell infiltration and inflammatory marker expression were markedly increased only in the heart tissue of WT mice. These findings suggest that although both T cells and macrophages contribute to vaccine-induced cardiac injury, T cells play a more prominent role in mediating the inflammatory response.

To investigate the underlying mechanisms of myocarditis, we performed RNA sequencing and found that T cell-mediated immune responses and IL-1 signaling were significantly altered in WT mice. To further assess the role of these immune components, *in vitro* assays were conducted using cardiomyocytes treated with mRNA vaccines and IL-1 siRNA. The results revealed that IL-1 signaling influenced both cell viability and inflammatory marker expression. Collectively, our results suggest that T cells and IL-1 play key roles in mediating vaccine-associated cardiac injury.

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Keywords : mRNA vaccine, Adverse effects, Myocardial damage, T cells, IL-1

PS-B-30

Effect of nicotinamide on nicotine dependence liability in mice

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The use of liquid e-cigarettes has increased rapidly in recent years. Nicotinamide is employed as a primary ingredient in certain products and as an additive in others in which nicotine is the main component. Despite their growing popularity, the abuse liability of such substances in the vaping products remains poorly understood. Therefore, scientific evidence is needed to support appropriate regulation and safety management. This study was conducted to investigate the effects of nicotinamide co-administration on nicotine dependence-related behaviors using an animal self-administration model of nicotine-containing liquid e-cigarette inhalation. We established a mouse self-administration system that models the inhalation route of human e-cigarette use. In particular, the experimental apparatus was designed to direct vapor toward the nose and mouth, thereby more accurately simulating the human e-cigarette use pathway. Mice were pre-trained to associate vapor reward before testing. During the test phase, the mice first self-administered nicotine under a fixed ratio 1 (FR1) schedule. Upon stable responding, the schedule was shifted to fixed ratio 2 (FR2), and once stabilization was achieved, nicotinamide was added to the nicotine solution at different concentrations. While nicotine alone produced stable self-administration behavior in FR2 schedule, the addition of nicotinamide affected the reinforcing effect of nicotine. Based on these results, nicotinamide appears to influence the degree of nicotine-induced psychological dependence. Therefore, with further refinement through additional studies, these findings may provide a scientific basis for regulating liquid electronic cigarettes containing nicotinamide.

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Keywords : E-cigarette, Nicotine, Nicotinamide, Self-administration, Vapor inhalation

PS-B-29

Effect of aging on mRNA vaccine-induced hepatotoxicity in mice

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As the global elderly population continues to grow due to advances in medical technology and increased life expectancy, it is increasingly important to consider age-related physiological and immunological changes in the development and safety evaluation of vaccines. In this study, we investigated the age-dependent toxicity of CUK3/LNP128, a novel SARS-CoV-2 mRNA vaccine, using both young (9-week) and aged (18-month-old) mice. The vaccine was administered intramuscularly in a two-dose regimen with a two-week interval. Compared to young mice, aged mice exhibited delayed post-vaccination weight recovery and failed to return to baseline body weight even after 14 days. Additionally, aged mice showed significant changes in serum biochemical parameters, increased liver weight, and more severe histopathological abnormalities. Specifically, hematoxylin and eosin (H&E) staining revealed increased cytoplasmic vacuolation and a higher number of inflammatory foci in the livers of aged mice. These results indicate that hepatotoxicity caused by the mRNA vaccine is more severe in aged mice. This increased susceptibility may be associated with age-related declines in mitochondrial function and regenerative capacity. To explore the potential mechanisms behind this increased vulnerability, we conducted transcriptomic profiling of liver tissues. Transcriptomic analysis revealed age-related down regulation of genes associated with mitochondrial function and tissue regeneration, supporting the hypothesis of impaired hepatic recovery in aged mice. In summary, vaccination causes more severe liver toxicity in aged models, potentially linked to mitochondrial dysfunction and impaired regeneration.

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Keywords : mRNA vaccine, Aged model, Hepatotoxicity, Adverse effects

PS-B-31

RAGE inhibition attenuates MMP2-mediated alveolar damage and enhances alveolar epithelial cell proliferation in emphysema

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Chronic obstructive pulmonary disease (COPD) is characterized by progressive alveolar destruction driven by chronic inflammation, oxidative stress, and extracellular matrix (ECM) degradation. The receptor for advanced glycation end products (RAGE) has been implicated in these processes, but its downstream molecular mechanisms remain incompletely understood. In this study, we examined the therapeutic effects of a recombinant RAGE antagonist peptide (RAP) using both *in vivo* and *in vitro* models of emphysema, with a particular focus on matrix metalloproteinase (MMP) activity.

RAP treatment in PPE-induced emphysema mice significantly alleviated alveolar structural damage and reduced inflammation and oxidative stress. AGER knockout (AGER^{-/-}) mice showed resistance to emphysematous changes, confirming the detrimental role of RAGE signaling. Notably, RAP administration led to a marked reduction in MMP2 expression at both the transcript and protein levels in lung tissues and epithelial cells, suggesting that RAP attenuates ECM degradation via MMP suppression. This downregulation of MMP2 was associated with decreased NF-κB and MAPK pathway activation, indicating that RAGE modulates MMP expression through pro-inflammatory signaling cascades.

In addition, RAP suppressed p53 accumulation and modestly increased Ki67 expression in cigarette smoke extract-exposed alveolar epithelial cells, suggesting that RAGE inhibition may also support epithelial repair. Collectively, these findings highlight RAGE as a central regulator of MMP-driven tissue remodeling and inflammation in COPD. Inhibition of RAGE signaling via RAP presents a promising therapeutic strategy to limit alveolar destruction and preserve lung architecture in emphysematous disease.

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Keywords : Alveolar epithelial, COPD, MMP, Oxidative stress, RAGE

PS-B-32

The role of Cathepsin K inhibition in respirable crystalline silica-induced silicosis through epithelial-macrophage interactions

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Background and Aims : Silicosis is a fibrotic lung disease caused by inhalation of respirable crystalline silica (RCS), a fine particulate form of silicon dioxide (SiO₂) with an aerodynamic diameter under 10μm capable of reaching the alveolar region. In this study, RCS was used to model silicosis, aiming to evaluate whether inhibition of Cathepsin K (CTSK), a lysosomal protease involved in tissue remodeling, can reduce RCS-induced pulmonary inflammation and epithelial-mesenchymal transition (EMT) in vitro and in vivo.

Methods : RCS was dispersed in 50μL of deionized water and ethanol (3mg/50μL), then analyzed by particle size analysis (PSA) and transmission electron microscopy (TEM) to determine particle size and morphology. MLE-12 epithelial cells and RAW 264.7 macrophages were treated with RCS (200μg/mL), with or without the CTSK inhibitor CompoundA (300nM). In vivo, mice were received a single intratracheal dose of RCS (50mg/kg), followed by weekly intratracheal administration of CompoundA (3.606 mg/kg).

Results : PSA confirmed that the RCS used had an average particle size of 1μm. RCS exposure increased CTSK expression, inflammatory cytokines, and EMT markers in both cell types. Conditioned media from RCS-treated macrophages induced EMT in epithelial cells, which was attenuated by CTSK inhibition. In mice, RCS-induced lung fibrosis was significantly reduced with CompoundA treatment.

Conclusions : CTSK plays a key role in RCS-induced inflammation and fibrosis by promoting macrophage-driven EMT in alveolar epithelial cells. Targeting CTSK may provide a promising therapeutic approach for silicosis.

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Keywords : Silicosis, Respirable crystalline silica, Cathepsin K, Macrophage

PS-B-34

ISO 10993-11-based evaluation of acute systemic toxicity of cultured meat components in a rat model

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As cultured meat continues to emerge as a promising future food source, ensuring its biosafety is essential for consumer acceptance and regulatory approval. This study focused on evaluating the acute systemic toxicity of cultured meat components using an in vivo rat model, following ISO 10993-11 international standards. The cultured meat construct consisted of film type alginate with bovine myoblasts, which were cultured within a three-dimensional scaffold fabricated using a pattern-film technique. The formulation was calculated based on lyophilization yield. A typical 1cm diameter construct contained approximately 20 mg of 2 % alginate and 0.4156 mg of dried cell powder, resulting in a weight ratio of approximately 50 to 1.

For toxicity assessment, 1 mg of test material per 100 g of body weight was administered orally to rats. The dosage reference was based on previous studies, including a report titled "Characterization of chitosan/alginate/lovastatin nanoparticles and investigation of their toxic effects in vitro and in vivo." A total of 20 rats were used in the study. No mortality, behavioral abnormalities, or clinical signs of systemic toxicity were observed during the test period. Additionally, necropsy and histological examinations revealed no pathological abnormalities related to the test substance.

The results clearly demonstrate that the cultured meat components, composed of alginate and bovine myoblasts, do not induce acute systemic toxicity in vivo under the tested conditions. These findings highlight the importance of including toxicological validation during the early stages of developing food-grade biomaterials. Especially for innovative food technologies intended for human consumption, such validation is crucial. This study supports the safety of alginate-based cultured meat and reinforces the necessity of evaluating the biological safety of engineered food materials.

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Keywords : Acute systemic toxicity, Alginate, Bovine myoblasts, Cultured meat

PS-B-33

Abuse liability of CUMYL-5F-P7AICA in a nose-poke operant animal model

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Synthetic cannabinoids are widely abused new psychoactive substances (NPS) that act as potent agonists at cannabinoid receptor type 1 (CB1), posing significant public health concerns. This study aimed to evaluate the abuse liability of vapor exposure to CUMYL-5F-P7AICA, a novel synthetic cannabinoid. To this end, we established a nose-poke-based inhalation self-administration system using vaporized CUMYL-5F-P7AICA in mice.

Prior to the experimental phase, animals underwent a two-stage training process over 24 days, consisting of preliminary shaping and operant conditioning to acquire active nose-poking behavior for vapor delivery. During the experimental phase, animals were assigned to independent groups, each receiving vaporized CUMYL-5F-P7AICA at one of three concentrations (0.25, 0.5, 1 mg/mL), with an additional group exposed to vehicle vapor as a negative control. Abuse liability was assessed over 14 consecutive days by measuring active hole responses, inactive hole responses, and the total number of vapor deliveries.

The results showed that CUMYL-5F-P7AICA significantly increased active hole responses and numbers of vapor delivery in a dose-dependent manner. In particular, the number of vapor deliveries during sessions 10 to 14 was significantly higher in the 0.5 and 1 mg/mL groups compared to the negative control group. The findings suggest that CUMYL-5F-P7AICA exerts reinforcing effects and may induce substance use disorder. The study provides scientific evidence that may inform regulatory authorities regarding the need to control this NPS.

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Keywords : Vapor, Self-administration, CUMYL-5F-P7AICA, Abuse liability, Cannabinoids

PS-B-35

Development and in vivo evaluation of medical fluorophore 33 as a theranostic agent for colorectal cancer imaging and therapy in mouse models

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Fluorescent dyes are emerging as promising theranostic platforms due to their unique physicochemical and biological properties. In this work, we report the identification and application of Medical Fluorophore 33 (MF33), a newly developed theranostic agent featuring a phenaleno-isoquinolinium salt scaffold. MF33 is synthesized efficiently via a straightforward Rh(III)-catalyzed transformation. The compound exhibits intense fluorescence emission, excellent in vivo microsomal stability, and favorable biocompatibility profiles. Functionally, MF33 triggers marked apoptotic cell death in cancer cells through activation of the p53/p21/caspase-3 axis, thereby conferring selective cytotoxicity toward malignant cells. In vivo fluorescence imaging studies confirmed MF33's capability to visualize sentinel lymph nodes in live murine models. Furthermore, repeated intraperitoneal administration of MF33 demonstrated significant tumor growth inhibition in colorectal cancer-bearing mice. Taken together, these results highlight MF33, based on a phenaleno-isoquinolinium salt core, as a potent theranostic candidate for integrated cancer diagnosis and therapy.

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Keywords : Fluorescent dye, Theranostic, Medical Fluorophore 33, Colorectal cancer, Phenaleno-isoquinolinium salt

PS-B-36

Key role of acrolein as an odor biomarker during 1,2,3-trichloropropane-induced hepatotoxicity of SD rats

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While body odor has been increasingly recognized as a potential tool for diagnosing infectious and chronic illnesses, its application in detecting toxicological responses remains largely underexplored. This study aimed to identify novel urinary odor biomarkers associated with liver injury induced by chemical exposure. Sprague Dawley (SD) rats were orally exposed to 1,2,3-trichloropropane (TCP) at doses of 100 or 200 mg/kg, and changes in urinary volatile compound profiles were examined using gas chromatography-mass spectrometry (GC-MS). The TCP treatment induced significant toxicity, including a decrease in body weight, an increase in serum biochemical factors, and istopathological changes in the liver of SD rats. Six volatile compounds—ethyl alcohol, acrolein (2-propenal), methanesulfonyl chloride, methyl ethyl ketone, cyclotrisiloxane, and 2-heptanone—showed significant changes in urinary concentration after TCP treatment. Among them, acrolein, an acrid and pungent aldehyde, exhibited the most notable increase compared to control animals. This elevation was accompanied by upregulation of spermine oxidase (SMOX), a key enzyme involved in acrolein production, along with increased transcription of interleukin-6 (IL-6), a cytokine that regulates SMOX expression. Correlogram analyses revealed strong associations between acrolein levels and other hepatotoxicity-related indicators. These findings suggest that acrolein may serve as a novel diagnostic odor biomarker for TCP-induced hepatotoxicity, offering new insight into odor-based approaches for chemical toxicity assessment.

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Keywords : Hepatotoxicity, Odor, Acrolein, Spermine oxidase, 1, 2, 3-Trichloropropane

PS-B-37

Prolonged antioxidant supplementation enhances locomotor performance but compromises survival in aged *Drosophila melanogaster*

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Reactive oxygen species (ROS) are natural byproducts typically produced at low to moderate levels during normal cellular processes. At physiological concentrations, ROS serve critical functions in modulating protein activity within signalling pathways that regulate cellular homeostasis and function. However, excessive ROS accumulation—driven by a combination of age-independent environmental and endogenous factors, along with an age-associated decline in redox homeostasis—leads to the generation and accumulation of free radicals. These free radicals can damage vital cellular components, leading to the disruption of cell structure and the loss of genomic integrity which cumulates in premature ageing and age-related conditions. Antioxidants, derived from dietary sources or synthesized as nutraceuticals, are widely believed to mitigate the detrimental effects of oxidative stress and are commonly promoted for improving overall health and preventing age-related conditions. Despite their widespread use, the long-term toxicological risks and safety profiles of these compounds remain poorly understood. *Drosophila melanogaster* serves as an ideal model organism for investigating the long-term effects of such compounds on ageing and longevity, owing to its low maintenance cost, genetic tractability, and short life cycle. In this study, we examined the impact of long-term supplementation with luteolin (LUT) and protocatechuic acid (PA)—two compounds frequently investigated for their antioxidant and health-promoting properties in dietary supplements—on *Drosophila* survival and locomotor function. While high concentrations of LUT and PA attenuated age-related declines in locomotor activity, they also accelerated mortality. Further research is necessary to determine whether these findings are translatable to mammals, and careful consideration of dosage is critical when evaluating the inclusion of such supplements in the diet.

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Keywords : Toxicity, Survival, Movement, Antioxidants, *Drosophila*

PS-B-38

Effect of Gossypetin from *Hibiscus sabdariffa* on Oral Cancer

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Gossypetin, a flavonoid compound derived from *Hibiscus sabdariffa* L, has demonstrated a range of pharmacological properties, including anti-inflammatory and antibacterial activities against various diseases. However, its effects on oral cancer have not yet been elucidated. In this study, we investigated the anticancer activity and underlying mechanisms of Gossypetin in oral cancer (OC). To assess its effects, OC cell proliferation was evaluated using cell viability and soft agar colony formation assays. Cell migration and invasion were examined through wound healing and transwell invasion assays, respectively. Apoptosis and cell cycle progression were analyzed by flow cytometry, while western blotting was employed to explore the molecular mechanisms involved. Gossypetin significantly inhibited OC cell proliferation, migration, and invasion. It also induced apoptosis by increasing the Bax/Bcl-2 ratio and caused cell cycle arrest at the G2/M phase. Furthermore, Gossypetin modulated the activation of extracellular signal-regulated kinase (ERK) and nuclear factor-kappa B (NF-κB) signaling pathways. These findings suggest that Gossypetin exerts potent anticancer effects in OC by inhibiting cell growth and metastatic potential while promoting apoptosis and cell cycle arrest. Thus, Gossypetin holds promise as a potential chemopreventive agent for the treatment of oral cancer.

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Keywords : Apoptosis, Gossypetin, Oral Cancer, Proliferation

PS-B-39

Anticancer Effects of Imatinib in Oral Squamous Cell Carcinoma via PI3K/AKT/mTOR Signaling Pathway

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Oral squamous cell carcinoma (OSCC) is a common malignancy of the oral and maxillofacial region, characterized by aggressive invasion and frequent metastasis to cervical lymph nodes, contributing to its high mortality rate. While Imatinib, a tyrosine kinase inhibitor, has demonstrated promising anticancer activity in various cancer types, its specific therapeutic potential in OSCC remains underexplored. This study aimed to investigate the anticancer effects of imatinib on OSCC cells and elucidate the underlying molecular mechanisms. Cell viability was assessed using the Cell Counting Kit-8 (CCK-8) assay, and morphological analysis was performed to observe proliferation changes. Wound-healing and soft agar assays were employed to evaluate cell migration and anchorage-independent growth, respectively. Apoptotic cell death was analyzed by flow cytometry, and Western blotting was used to determine the expression levels of apoptosis-related and signaling pathway-associated proteins. The results demonstrated that imatinib significantly inhibited OSCC cell proliferation and viability in a time and concentration-dependent manner. Additionally, imatinib suppressed cell migration and colony formation while promoting apoptosis, as evidenced by increased expression of p53, Bax, and cleaved PARP, and decreased expression of Bcl-2. Furthermore, imatinib inhibited the PI3K/AKT/mTOR signaling pathway, contributing to apoptosis induction. These findings suggest that imatinib may serve as a potential therapeutic agent for the treatment of OSCC.

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Keywords : Imatinib, Oral squamous cell carcinoma, Proliferation, Apoptosis, PI3K/AKT/mTOR signaling pathway

PS-B-40

Gossypetin reduces colitis via regulating COX2 and ROS-JNK axis

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Inflammatory Bowel Disease (IBD) is a chronic and recurring inflammatory disease that affects the gastrointestinal tract, with an increasing global prevalence. Surgery and medications are currently used as therapeutic options. However, surgeries are invasive operations, whilst pharmacological therapies frequently result in a variety of negative effects. Gossypetin, a flavonoid found abundantly in plants such as hibiscus, has antioxidant and anti-cancer properties. However, its possible impact on IBD remains unknown. This study aims to evaluate the therapeutic potential of gossypetin for colitis. We used the DSS-induced colitis model to determine gossypetin's therapeutic potential for colitis. The efficacy of gossypetin was evaluated in this animal utilizing the Disease Activity Index (DAI) score and histological examination. In addition, we used qRT-PCR to assess the levels of inflammatory cytokines and Superoxide Dismutase. Immunohistochemistry showed the presence of tight junction indicators, COX-2, and phosphorylated JNK protein, which are often linked with disease development. Western blot analysis was also performed to investigate the SOD levels and anti-apoptotic effects of gossypetin.

In mice with DSS-induced colitis, gossypetin treatment reduced weight loss and colon length. Additionally, gossypetin-treated groups showed lower DAI scores and histological damage. Furthermore, gossypetin therapy boosted tight junction expression, decreased inflammatory responses, reduced ROS levels, inhibited JNK signaling, and reduced apoptosis.

Gossypetin has therapeutic potential for reducing colitis symptoms and development by inhibiting the ROS-JNK signaling pathway implicated in inflammation and tissue damage. This demonstrates the promise of natural chemicals like gossypetin for targeted medicines with fewer side effects and greater efficacy.

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Keywords: Anti-inflammation, DSS-induced colitis, Anti-oxidant, Gossypetin, Inflammatory bowel disease

PS-B-42

Silibinin alleviates Vanadium-induced lung Injury via the TLR4/MAPK/NF- κ B pathway in mice

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This study examined the protective effects and underlying mechanisms of Silibinin against vanadium pentoxide (V₂O₅)-induced pulmonary inflammation in a mouse model. To simulate lung injury, eight-week-old male BALB/c mice were exposed to V₂O₅, and pre-treatment with Silibinin was administered at dosages of 50 mg/kg and 100 mg/kg. Histopathological evaluations were conducted to determine tissue damage and inflammatory cell infiltration, while pro-inflammatory cytokines (TNF- α , IL-6, IL-1 β) and activation of MAPK, NF- κ B, and the NLRP3 inflammasome were analyzed through real-time PCR, western blotting, and immunohistochemistry. In addition, complete blood counts were used to assess systemic inflammation. Results showed that Silibinin significantly reduced inflammatory cell infiltration, improved cellular integrity, and downregulated cytokine expression in V₂O₅-induced lung injury. Moreover, Silibinin markedly inhibited the activation of MAPK and NF- κ B pathways and suppressed NLRP3 inflammasome expression. Blood analysis further demonstrated decreased levels of white blood cells, including neutrophils, lymphocytes, and eosinophils, in Silibinin-treated mice. Collectively, these results support the anti-inflammatory potential of Silibinin in mitigating environmental pollutant-induced lung injury. The findings provide crucial insight into the molecular pathways regulated by Silibinin and its possible therapeutic application in treating pulmonary inflammation caused by toxic metal exposure.

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Keywords: Vanadium pentoxide (V₂O₅), Silibinin, MAPK pathway, NF- κ B pathway, Pulmonary inflammation

PS-B-41

Effects of PM2.5 associated metal components from pig house exposure on the male reproductive system in mice

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This study aimed to investigate the effects of major metal components associated with particulate matter (PM2.5) on the male reproductive system, sperm function, and embryo development. PM2.5 samples were collected from a porcine farm and administered to male mice via intratracheal instillation. The mice were exposed to various concentrations of metal components, including calcium oxide (CaO), iron oxide (Fe₂O₃), aluminum oxide (Al₂O₃), zinc oxide (ZnO), lead oxide (PbO), and a combination of these metals. Fourteen days after exposure, testicular inflammation and abnormal sperm morphology were observed in the treated groups. Histopathological and biochemical analyses revealed elevated expression of inflammatory cytokines and oxidative stress-induced apoptosis in testicular tissue. Additionally, metal exposure adversely affected sperm morphology, capacitation status, serum testosterone levels, and testosterone biosynthesis. Notably, decreased sperm fertilizing capacity resulted in impaired embryo development. These findings demonstrate that PM2.5-associated metal components derived from pig farming environments exert detrimental effects on male reproductive health in mice and may pose reproductive risks in agricultural settings.

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Keywords: Particulate matter, Reproductive system, Sperm, Testis

PS-B-43

Rhein-Induced AKT/mTOR Suppression Drives ROS-Mediated Apoptosis in Oral Cancer In Vitro and In Vivo

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Oral cancer remains a major cause of cancer-related mortality worldwide. Rhein, a naturally occurring anthraquinone derivative isolated from the traditional Chinese medicinal herb Rheum Palmatum (Rhubarb), has demonstrated antitumor properties across multiple cancer types. However, its specific pharmacological effects and underlying molecular mechanisms in oral squamous cell carcinoma (OSCC) remain insufficiently characterized. In this study, we systematically investigated the anticancer potential of Rhein in OSCC, both in vitro and in vivo. The antiproliferative effects of Rhein were evaluated using CCK-8 assays, soft agar colony formation, migration by transwell, and Matrigel invasion assays. Flow cytometry was employed to assess cell cycle distribution and apoptotic induction, while Western blot analysis was conducted to elucidate the involved molecular signaling pathways. In vivo antitumor efficacy was further assessed using an OSCC xenograft mouse model. Our findings revealed that Rhein significantly suppressed OSCC cell viability by inducing apoptosis and S-phase cell cycle arrest. Moreover, Rhein inhibited OSCC cell migration and invasion by modulating epithelial-mesenchymal transition (EMT)-related protein expression. Mechanistically, Rhein treatment led to a significant accumulation of intracellular reactive oxygen species (ROS), which subsequently suppressed activation of the AKT/mTOR signaling pathway. Taken together, these results suggest that Rhein exerts potent antineoplastic effects against OSCC through ROS-mediated inhibition of the AKT/mTOR signaling axis, both in vitro and in vivo. Rhein may serve as a promising therapeutic agent for the treatment of oral cancer.

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Keywords: Oral cancer, Rhein, Apoptosis, Autophagy, MTOR

PS-B-44

Mixed toxicity evaluation study of Chromium(VI) Oxide and Molybdenum Oxide using Zebrafish Embryo

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This study was conducted to investigate the environmental impacts of two heavy metals, Chromium(VI) oxide and Molybdenum oxide, by referring to the Organization for Economic Cooperation and Development Guidelines for the Testing of Chemicals Test No. 236 (Fish Embryo Acute Toxicity [FET] Test) and Chapter 3, Section 16 (Fish Embryo Acute Toxicity Test) of the Regulation on Test Methods of Chemical Substances in the field of Ecological Impact Tests. The experimental results are as follows: 1. The toxicity value (LC50) of Chromium(VI) oxide was calculated to be 54.97 mg/L after 96 hours of observation. 2. The toxicity value (LC50) of Molybdenum oxide was calculated to be 97.29 mg/L after 96 hours of observation. 3. The toxicity value (LC50) of a mixture of Chromium(VI) oxide and Molybdenum oxide was calculated to be 34.35 mg/L after 96 hours of observation. It was observed that the toxicity value calculated for the mixture of the two substances was 1.60 and 2.83 times more toxic than that of the individual substances, respectively. Since various substances exist in the environment in diverse forms, and their interactions can have different effects on organisms depending on the substances involved, further research is necessary.

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*Corresponding author : Dong Hyun Kim

Keywords : Zebrafish, Animal alternative test

PS-B-45

Inhibition of IRF1 attenuates inflammation and dysregulated autophagy in acute lung injury mice

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Acute lung injury (ALI), including its most severe form, acute respiratory distress syndrome (ARDS), is a common cause of acute hypoxemic respiratory failure. Although its clinical characteristics have been well characterized, the relevant mechanism remains unclear. An imbalance in autophagy leads to alveolar remodeling and triggers the pathogenesis of ARDS. In this study, we assessed the therapeutic efficacy of the STAT1 inhibitor fludarabine (Fluda) in ALI. C57BL/6 mice were exposed to lipopolysaccharide (LPS), and their lung tissues were analyzed via next-generation transcriptome sequencing. Interferon regulatory factor 1 (IRF1) was highly expressed and STAT1 was phosphorylated following LPS exposure. Fluda significantly decreased the protein expression of STAT1/IRF1 and inhibited the alveolar infiltration of neutrophils and macrophages. Nitric oxide (NO), inducible nitric oxide synthase, tumor necrosis factor- α (TNF- α), interferon- γ , and interleukin-6 (IL-6) release was decreased in the lungs of mice and RAW264.7 macrophages following Fluda treatment. In LPS-induced GFP-LC3 transgenic mice treated with Fluda, the counts of LC3-expressing neutrophils and macrophages in bronchoalveolar lavage (BAL) fluid were significantly decreased. Furthermore, Fluda decreased LC3 and p62 protein expression, thereby inhibiting the release of NO, IL-6, and TNF- α in BAL. In RAW264.7 cells, the inhibition of STAT1/IRF1 by Fluda decreased LPS-induced ERK and NF- κ B p65 phosphorylation. The inhibition of STAT1/IRF1 by Fluda plays a pivotal role in modulating dysregulated autophagy by suppressing the MAPK and NF- κ B p65 pathways in ALI.

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Keywords : IRF1, Acute lung injury, STAT1, Fludarabine, Autophagy

PS-B-46

Inhalation effects of polystyrene microplastics on colonic disease of ICR mice

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The inhalation effects of airborne microplastics (MP) on the gastrointestinal (GI) tract are rarely investigated, with most studies focusing only on the lungs and other organs. To investigate whether inhaled MPs can cause constipation disease in the colon, alterations in stool parameters, structure and motility of mid colon, enteric nervous system (ENS) function and gastrointestinal (GI) hormone levels were analyzed in ICR mice inhaled by polystyrene (PS)-MPs with 0.5 μ m for 2 weeks. MP inhalation successfully induces the defecation delay accompanied by decrease in stool parameters including weight, number and water contents. Also, MP-caused defecation delay is tightly associated with the dysregulation of GI motility and stagnation of stools in the colon as well as abnormalities in the histopathological structural of the mid colon. Furthermore, MP-caused defecation delay may be associated with the decreasing mucin secretion and transcription of mucin related genes as well as water and ion balance. Moreover, MP-caused defecation delay may be tightly linked to the decrease in the distribution of neuronal cells, alternative regulation of excitatory neurons, and dysfunction of inhibitory neuron in the colon. Therefore, these results suggest that inhaled MPs can cause constipation through dysregulation of mucin secretion and water balance as well as dysfunction of ENS in ICR mice. Therefore, these results provide the first scientific evidence that inhalation of PS-MP can be considered important novel causes of constipation.

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Keywords : Microplastics, Constipation, Colon, Mucin, ENS

PS-B-47

Mitigation of particle overload toxicity induced by ultrafine particles

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Air pollution remains a critical global health issue, comprising various harmful components such as particulate matter (PM), toxic metals, and liquid aerosols. Among these, PM0.1—ultrafine particles with a diameter smaller than 100 nm—is considered particularly hazardous due to its high particle number and surface area, as well as its ability to transport heavy metals and organic pollutants. PM0.1 is found in both indoor and outdoor environments and can penetrate deeply into the alveolar region, potentially triggering respiratory diseases and inflammatory responses. However, the precise mechanisms of PM0.1-induced toxicity remain inadequately understood. One proposed mechanism is particle overload, a phenomenon in which excessive particle accumulation in alveolar macrophages impairs their clearance capacity and motility, resulting in inflammation and tissue damage.

This study employed carbon black and diesel particulate matter (SRM-2975) to comparatively assess ultrafine particle toxicity. We analyzed their physicochemical properties and conducted a single-dose inhalation toxicity study using intratracheal instillation in mice, followed by a 28-day recovery period. Both overload-inducing and non-overload-inducing concentrations were identified for each particle type, and transcriptomic analyses were performed to elucidate their biological impacts and mechanisms of action. Additionally, candidate compounds with potential to facilitate particle clearance were selected and evaluated for therapeutic efficacy.

To examine cellular responses, THP-1-derived macrophages differentiated with PMA were exposed to the particles. While SRM-2975 did not exhibit significant cytotoxicity, carbon black showed mild cytotoxic effects (~7–8%). Co-treatment with selected clearance-enhancing agents reduced carbon black-induced cytotoxicity. Further research is warranted to explore how particulate exposure and subsequent treatment affect macrophage mobility and function. These findings contribute valuable insights into the toxicity mechanisms of ultrafine particles and provide foundational data for the development of potential therapeutic interventions aimed at mitigating their biological effects.

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Keywords : Ultrafine particles, Intratracheal instillation, Lung burden, Particle overload, Pulmonary toxicity

PS-B-48

Platelet promote phenotypic variation of U251 human glioblastoma cells

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Glioblastoma is one of the malignant tumors and has a low patient survival rate. In order to overcome the low survival rate of glioblastoma, in addition to the direct targeting of cancer cells, various approaches such as controlling the interaction with the tumor microenvironment are being made. In addition to hemostasis, platelets have various pathophysiological actions, such as being involved in the metastasis and growth of tumor cells. In this study, the effect of platelets on the properties of U251 human glioblastoma cells was investigated in vitro and in vivo. In the case of platelets, whole blood was collected from volunteers and platelet rich plasma was extracted through centrifugation. Finally, the absence of RBC and WBC was confirmed through a complete blood count machine before the experiment was conducted. In vitro experiments directly treated U251 human glioma cells with platelets, and the properties and state changes of the cancer cells were confirmed through western blotting, real-time PCR, etc., and the results showed that indicators related to the malignancy of glioblastoma cells increased statistically significantly. EMT-related markers were markedly increased in the expression of N-cadherin protein in platelet-treated U251 cells. On the other hand, E-cadherin, a biochemical molecular biological single protein marker of the epithelial type, was confirmed to decrease by platelet treatment. In vivo experiments showed that when platelet-treated U251 glioblastoma cells and control U251 glioblastoma cells were inoculated into experimental animals, the tumorigenic potential of platelet-treated U251 glioblastoma cells was statistically significantly increased. Taken together, these results confirmed that human platelets increase the properties of U251 glioblastoma cells both in vitro and in vivo.

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Keywords: Platelets, U251 glioblastoma, Metastases, Malignancy

PS-B-49

Modeling drug-Induced phototoxicity using a vascularized skin assembloid derived from human iPSCs

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Drugs with phototoxic potential can cause various pathological damage in skin tissue upon exposure to ultraviolet (UV) radiation, including epidermal disruption, dermal degeneration, and vascular dilation, thereby underscoring the importance of phototoxicity assessment during drug development. However, conventional in vitro models, such as 2D cell cultures and artificial skin equivalents, have limited predictive capacity due to the absence of complex tissue architecture and vasculature found in human skin. To overcome these limitations, we developed a vascularized skin assembloid by integrating skin and blood vessel organoid derived from human induced pluripotent stem cells (iPSCs). PCR analysis confirmed the mRNA expression of KRT5 and PECAM1, representing epidermal and vascular markers, respectively. Immunostaining further demonstrated the key skin features, including the stratum corneum, epidermis, dermis, skin appendages, and vascular networks in the assembloids. Using an optimized UV exposure condition not causing epidermal damage, we applied doxycycline and ketoprofen into iPSC-derived skin assembloids as systemic and topical compounds, respectively. Compared to drug-only treatment, co-exposure to UVA and phototoxic drugs led to significant disruption of the epidermis and dermis, accompanied by pronounced vascular dilation in the skin assembloids. In contrast, non-phototoxic agents such as ascorbic acid and octisalate preserved both tissue and vascular integrity. In conclusion, the vascularized skin assembloid provides a physiologically relevant platform that enables reliable phototoxicity testing and mechanistic investigation of skin-vascular interactions.

Acknowledgements: This research was supported by the National Research Foundation of Korea (NRF) grant (No. RS-2021-NR061977) and Korea Basic Science Institute (National research Facilities and Equipment Center) grant funded by the Ministry of Science and ICT (No. RS-2025-00563046).

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Keywords: Skin organoid, Assembloid, Vascularization, Phototoxicity, Drug screening

PS-B-50

Influence of biotransformation on pulmonary clearance of iron oxide nanoparticles following 28-day repeated inhalation exposure

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Understanding the toxicity of nanomaterials is essential for risk assessment and the development of safer materials under the safety-by-design framework. Among key factors, biotransformation after tissue deposition may affect the biokinetics and toxicity of nanomaterials, potentially altering their biological behavior. Recent studies suggest that intravenously administered iron oxide (Fe₃O₄) can transform into new particle types. This study evaluated the lung clearance kinetics and tissue distribution of iron oxide nanoparticles, focusing on their biotransformation following a 28-day repeated inhalation exposure and a 28-day recovery period. A comparative 14-day repeated oral toxicity study with a 28-day recovery phase was also conducted. In the inhalation study, rats were exposed to Fe₃O₄ via nose-only inhalation at concentrations of 0.56, 1.67, and 5.00 mg/m³. The oral study used 250, 500, and 1000 mg/kg bw doses. Organs and blood were collected at 0, 7, and 28 days post-exposure. Tissues were digested with proteinase K for qualitative (biotransformation) and quantitative (iron levels) analysis. Inhalation exposure resulted in gradual lung clearance, with 21–54% clearance at 7 days and 11–40% at 28 days. No significant changes were observed in other organs or oral exposure groups. Transmission electron microscopy revealed that Fe₃O₄ in the lungs was consistently biotransformed into smaller, nanometer-sized particles from day 0 onward, with a progressive increase. Although no significant toxicity was detected, the observed biotransformation highlights the need for further investigation into its potential effects on the lung microenvironment.

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Keywords: Iron oxide, Biotransformation, Distribution, Elimination

PS-B-51

Real-time in vivo imaging demonstrates hippocampal calcium dynamics induced by bicuculline administration in voluntarily running mice

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High-resolution, real-time imaging of deep brain during voluntary behavioral states remains a significant challenge in neuroscience. To address this, we employed fiber-optic confocal (FOC) fluorescence endomicroscopy using the Cell-Vizio system to monitor neuronal calcium dynamics in the hippocampus of voluntarily moving yet head-fixed mice. GCaMP6s transgenic mice, which express a genetically encoded green fluorescent calcium indicator specifically in neurons, were habituated to a voluntary linear treadmill under head-fixed conditions. This setup enabled stable recordings in freely moving mice. Calcium signals in the dentate gyrus (DG) of the hippocampus were continuously recorded before and after administration of saline or bicuculline. To investigate hippocampal hyperexcitability, we administered bicuculline, a GABA_A receptor antagonist known to induce epileptiform discharges by blocking inhibitory synaptic transmission. After saline administration, no significant change in calcium fluorescence was observed. In contrast, bicuculline administration elicited a significant increase in calcium signal intensity, indicating heightened neuronal activity and synchronous neuronal discharges within the hippocampus. These results validate the utility of FOC microscopy combined with voluntary behavioral paradigms for real-time monitoring of deep-brain neural dynamics. Moreover, the head-fixed yet voluntary locomotion condition offers an optimal compromise between neural imaging stability and physiological relevance. This experimental approach can be extended to investigate region-specific neuronal responses to a wide range of pharmacological agents or behavioral stimuli, enabling novel insights into brain function under near-naturalistic conditions.

*Corresponding author: Jaesuk Yun

Keywords: Bicuculline, Cell-Vizio, GCaMP6s, Hippocampus, Voluntary

PS-B-52

Evaluation of the protective effects of *Loranthus tanakae* Franch. and Sav. on atopic dermatitis in ovalbumin-induced mice and TNF- α /IFN- γ -stimulated HaCaT cells

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Loranthus tanakae Franch. and Sav. is a traditional herbal remedy known for its anti-inflammatory and antioxidant activities, and has been traditionally employed in the treatment of joint and respiratory inflammation. In this study, we evaluated the therapeutic potential of ethanol extract of *L. tanakae* (LTE) in atopic dermatitis (AD). An ovalbumin (OVA)-induced AD animal model and a human keratinocyte cell line, HaCaT, were used to assess the efficacy of LTE. An in vitro experiment showed that LTE treatment significantly suppressed the production of regulated upon activation, normal T cell expressed and secreted (RANTES) cytokines and macrophage-derived chemokines (MDC) in tumor necrosis factor- α (TNF- α)/interferon- γ (IFN- γ) (TNF- α /IFN- γ)-stimulated HaCaT cells in a concentration-dependent manner. Additionally, treatment with LTE markedly inhibited the translocation of signal transducer and activator transcription 1 (STAT1) protein to the nucleus and the phosphorylation of Janus kinase 2 (JAK2) in TNF- α /IFN- γ -stimulated HaCaT cells. In the in vivo experiment, administration of LTE significantly decreased the levels of immunoglobulin E (IgE) and interleukin-13 (IL-13) of OVA-induced AD mice, which was supported by histological evidence. LTE treatment markedly reduced inflammatory cell infiltration and tissue edema in the affected skin lesions of the OVA-induced AD model. In addition, applying LTE notably inhibited the phosphorylation of JAK2 and STAT1 in the OVA-induced AD mice, supported by in vitro results. In conclusion, LTE effectively ameliorated OVA-induced skin inflammation in both the OVA-induced mouse model and TNF- α /IFN- γ -stimulated HaCaT cells, likely through inhibition of JAK2 and STAT1 phosphorylation. These findings suggest that LTE has potential as a therapeutic agent for AD management.

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Keywords : *Loranthus tanakae* Franch. and Sav., Atopic Dermatitis (AD), JAK2/STAT1 signaling pathway

PS-B-54

Acute Respiratory Toxicity of Non-Exhaust Brake Wear Particles in Mice

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Particulate matter (PM) is a major contributor to air pollution and is classified into exhaust and non-exhaust sources. While effective regulations have successfully reduced emissions from exhaust PM, non-exhaust PM remains largely unregulated. Among these, brake wear particles have emerged as a significant contributor, as the growing number of electric vehicles with heavy batteries increases vehicle mass and consequently leads to brake wear particle emissions. Although recognized as an important environmental issue, the inhalation toxicity of brake pad dust remains poorly understood. In this study, we investigated the respiratory toxicity of two types of brake wear particles—non-asbestos organic (NAO) and low-metallic (LM)—using a mouse model. The particles were administered into the lungs via pharyngeal aspiration, and bronchoalveolar lavage fluid (BALF) and histopathological analyses were conducted 24 hours after exposure. Our results demonstrated that both NAO and LM induced acute neutrophilic inflammation in a dose-dependent manner, with NAO causing significantly higher inflammatory responses. Furthermore, the generation of reactive oxygen species (ROS) by the particles themselves appeared to contribute to the observed toxicity. These findings indicate that brake pad-derived non-exhaust PM can induce significant acute respiratory toxicity, and that its biological effects vary depending on the chemical composition of the materials. This study suggests the need for regulatory strategies targeting the formulation and manufacturing of brake components to mitigate health risks associated with non-exhaust PM exposure.

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Keywords : Non-exhaust particulate matter, Brake wear particles, Inhalation, Respiratory toxicity, Reactive oxygen species

PS-B-53

Effects of respiratory exposure to 1,2-Dichloroethylene on female reproductive function

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1,2-Dichloroethane (1,2-DCE) is a volatile organic compound (VOC) widely used in the plastics and semiconductor industries. Industrial activities can lead to its release and accumulation in various environmental media, including air, soil, and water. As a VOC, human exposure to 1,2-DCE primarily occurs through the respiratory system, and its detection, particularly in indoor residential environments, raises concerns about potential health risks. Previous studies have shown that 1,2-DCE is toxic to multiple organs such as the liver, kidneys, and nervous system, and causes male reproductive toxicity, specifically testicular damage and reduced sperm quality. However, research on the effects of 1,2-DCE on the female reproductive system is very limited. Therefore, this study aims to comprehensively evaluate the toxic effects of inhaled 1,2-DCE exposure on the female reproductive system using a mouse model. Using a 28-day intratracheal intubation model, we explored how inhaled 1,2-DCE affects the female reproductive system by administering 0.3 mg and 3 mg doses to female mice. Our experimental data indicated that 1,2-DCE exposure was associated with diminished body weight gain and erratic estrous cycles. Crucial female reproductive hormone levels also showed considerable deviations. Microscopic examination of ovarian tissue demonstrated pathological changes and an increased number of atretic follicles. Supporting these observations, Western blot and immunofluorescence analyses confirmed the induction of apoptosis and an elevated fibrotic response within the ovaries. These results strongly imply that the inhalation of 1,2-DCE can induce female reproductive dysfunction. This study highlights the urgent need for further research to fully understand the long-term health effects of environmental 1,2-DCE exposure on women's reproductive health.

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Keywords : 1, 2-DCE, VOC, Ovarian toxicity, Reproduction, Intratracheal intubation

PS-B-55

Immunoregulatory effects of *Choerospondias axillaris* (Roxb.) B.L.Burt & A.W.Hill fruit extract on mice

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Lapsi (*Choerospondias axillaris*), a plant native to Nepal, has been traditionally used in Asian countries to treat cardiovascular conditions. However, its effects on immune regulatory function remain largely unexplored. This study aimed to *in vivo* evaluate the immunoregulatory properties of Lapsi fruit extract on mice humoral and cell-mediated immunities. Five-week-old male Balb/c mice were intragastrically administered various doses of the extract for 21 days. In some mice, immune suppression was induced with cyclophosphamide, and subsequent immune recovery was assessed. To assess the immunomodulatory activity of Lapsi fruit extract, 24 mice were divided into four groups of six mice each, with one group each intragastrically administered 5, 50, or 500 mg/kg-BW/day/100 μ l of extract, and the fourth group administered vehicle. To assess the immune-boosting activity of the extract, mice were divided into five groups, with one group each intragastrically administered extract, as above; one administered vehicle and one cyclophosphamide (CP) model control group. Vehicle control groups were injected intraperitoneally with 100 μ l of saline, whereas the other groups were injected with 100 μ l of 100 mg/kg-BW CP in saline, on days 1, 7, and 14 to induce immune suppression. Administration of extract increased the IgG2a/IgG1 ratio while reducing serum IgE level compared with control mice. Tumor necrosis factor- α and interleukin (IL)-17 levels were lower in splenic culture supernatants of mice administered extract. Lapsi extract also effectively reversed cyclophosphamide-induced immunosuppression by enhancing serum levels of IgA and IgG2a, of interferon- γ and IL-4 secreted by splenic T cells, and of IgG1 and IgG2a secreted by B cells, as well as by increasing immune cell counts. Lapsi extract may act as both an immunostimulatory agent, enhancing type-1 helper T-cell reactivity, and as an anti-inflammatory agent, suggesting that it has potential therapeutic applications [supported by the grant from the DragonImmuno Inc., Republic of Korea].

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Keywords : Lapsi (*Choerospondias axillaris*), Immunomodulation, Immunostimulation, Immunoglobulins, Cytokines

PS-B-56

Functional Disruption of Natural Killer Cells by ZnO, rGO, and CB Nanomaterials under Repeat Exposure

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Nanomaterials (NMs) such as zinc oxide (ZnO), reduced graphene oxide (rGO), and carbon black (CB) possess unique physicochemical properties that make them attractive candidates for diverse biomedical applications, including drug delivery systems (DDSs), imaging, and immunotherapy. While their potential therapeutic benefits are increasingly recognized, concerns remain regarding their interactions with the immune system, particularly in the context of systemic administration. Despite their widespread use, comprehensive evaluations of NM-induced immunotoxicity—especially at the level of innate immune effectors—are still lacking. In this study, we systematically investigated the biodistribution, immunological interactions, and functional consequences of ZnO, rGO, and CB nanoparticles following single and repeated intravenous (i.v.) administration in mice. To enhance translational relevance, we also employed human cord blood mononuclear cell (CBMC)-derived natural killer (NK) cells as an ex vivo model.

Physicochemical characterization revealed surface charge alterations and hydrodynamic size changes in biological media, consistent with protein corona formation. Biodistribution analysis indicated dominant hepatic accumulation of all NMs, with substantial splenic retention observed particularly for rGO and CB after repeated dosing. Although histopathological assessments showed no overt tissue damage, functional assays revealed a significant reduction in splenic NK (sNK) cell cytotoxicity, especially following repeated rGO or CB exposure. In vitro assays confirmed that prolonged NM exposure impaired sNK cell viability, perforin/granzyme B expression, and degranulation (CD107a), with rGO and CB showing the most pronounced effects. Notably, ex vivo exposure of CBMC-derived NK cells to NMs resulted in suppressed cytotoxicity and reduced expression of activation markers, with partially divergent patterns compared to murine cells.

Collectively, our findings demonstrate that nanomaterials can induce functional impairments in NK cells without causing histological pathology, and highlight the necessity of incorporating functional and human-relevant immunological assays in nanotoxicology studies. These insights are critical for guiding the safe design and clinical translation of NM-based therapeutics.

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Keywords : Nanomaterials, NK cell, Immunotoxicity, DDS

PS-B-58

Acute Inhalation Toxicity Study of Manganese dioxide in SD Rats

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Background : This study was conducted to evaluate the inhalation hazards of Manganese dioxide, an industrial chemical commonly used in batteries, as an oxidizing agent in chemical reaction, and in water treatment, glass and ceramics industry. The aim was to provide relevant safety information to workers.

Materials and methods : To assess the toxicity of one-day inhalation exposure to Manganese dioxide, an acute inhalation toxicity study was conducted using a nose-only inhalation chamber and a dust generator. Male and female SD rats were exposed to the substance for 4 hours. Clinical signs were monitored twice daily. Concentrations in the chamber were measured three times a day using a personal air sampler equipped with a 25 mm glass fiber filter.

Results : During the study period, the average analytical concentration of the test group (5 mg/L) was measured as 4.887 mg/L. The mass median aerodynamic diameter (MMAD) was 5.477 μ m with a geometric standard deviation (GSD) of 2.2. No abnormal clinical signs or changes in body weight were observed during the exposure period. Upon necropsy, lung enlargement and pale discoloration were observed in three male and two female animals. Tracheobronchial lymph node enlargement was also observed in the same three male animals. These findings were considered not to be substance-related and were not deemed to affect the overall outcome of the study.

Conclusions : According to the OECD guidelines, the test substance Manganese dioxide is considered to fall outside the GHS classification for acute inhalation toxicity.

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Keywords : Dust, Nose-only inhalation, Manganese dioxide, GHS classification

PS-B-57

Sex-specific differences in glucose metabolism and pancreatic function in streptozotocin-induced diabetic mice: The protective role of estrogen

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Sex differences significantly influence the pathophysiology of diabetes mellitus, with estrogen playing a protective role in glucose metabolism, insulin sensitivity, and beta-cell function. This study investigated sex-specific differences in glucose homeostasis, lipid metabolism, and pancreatic function in a streptozotocin (STZ)-induced diabetic mouse model. Eight-week-old male, female, and ovariectomized female C57BL/6J mice received STZ (40 mg/kg) for five consecutive days. Biochemical assessments, including fasting blood glucose (FBG), hemoglobin A1c (HbA1c), and oral glucose tolerance tests (OGTT), were conducted at 4 and 8 weeks post-STZ treatment. Plasma insulin levels, beta-cell function (%B), and insulin sensitivity (%S) were assessed using the HOMA2 calculator. Pancreatic tissues were analyzed via histology and immunofluorescence, while hepatic gene expression was evaluated using microarray analysis. Male and ovariectomized female mice showed significantly higher FBG and HbA1c levels and impaired glucose tolerance compared to female mice, indicating estrogen's protective role in glucose regulation. Histological analysis revealed severe pancreatic islet atrophy and reduced beta-cell mass in male and ovariectomized females, while intact females maintained better insulin-glucagon balance. Transcriptomic analysis identified sex-specific alterations in key metabolic pathways, including insulin signaling and lipid metabolism, which were more disrupted in males and ovariectomized females. Significant sex-specific metabolic and molecular differences exist in STZ-induced diabetes. Estrogen's protective effects highlight its therapeutic potential for preserving beta-cell function and metabolic stability, especially in postmenopausal women.

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Keywords : Sex difference, Diabetes, Streptozotocin, Estrogen, Glucose metabolism

PS-B-59

Acute Inhalation Toxicity of Barium Carbonate in Sprague-Dawley Rats

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Barium carbonate (CAS No. 513-77-9) is a colorless powder used as a raw material for barium salts, crystal glass and optical glass, as a glaze for ceramics and enamel, and as a carburizing agent in material heat treatment. Additionally, it is used as an insecticide and rodenticide. However, workers and consumers are potentially exposed to this substance through inhalation during use. Data on inhalation toxicity remains insufficient for a comprehensive hazard assessment. Therefore, we conducted an acute inhalation toxicity test using a nose-only inhalation chamber system and a dust generator. Rats (3 males and 3 females per group) were exposed to the test substance for 4 hours at a concentration of 1 and 5 mg/L. The target concentrations of in the chambers were 1 and 5 mg/L, with average concentrations of 0.991 $\pm$ 0.034 and 4.978 $\pm$ 0.085 mg/L, respectively. The mass median aerodynamic diameter was 3.076 and 4.255 μ m with a geometric standard deviation of 1.6 and 2.0. Clinical signs, mean body weight changes, and gross necropsy results were recorded. In two males in the 5 mg/L group, Weakness, abnormal gait or soiled perineal region were observed on the first day of exposure, but all animals had recovered by the next day. No unusual clinical signs, body weight changes, or gross necropsy observations were recorded thereafter.

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Keywords : Barium carbonate, Aerosol, Acute inhalation toxicity study, Sprague-Dawley rat

PS-B-60

Preclinical evaluation of tissue coagulation and safety of the plasma electrosurgical device

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This study aimed to evaluate the tissue coagulation efficacy and in vivo safety of the PlazMagik plasma electrosurgical device, developed by Agnes Medical Co., Ltd., using a porcine skin model. The device generates plasma by ionizing gas with high-frequency current, producing a chemically and electrically active state. Cold plasma, generated at relatively low temperatures (30–60°C), can induce tissue changes through localized thermal effects.

A total of 19 experimental groups were tested, comprising 18 plasma exposure conditions varying by gas type (He, Ar), handpiece type (single or triple), plasma/gas output levels, operation modes (continuous or pulsed), and exposure times (5 or 10 seconds), along with a control group. Tissue temperature changes were measured, and histological changes were assessed using H&E, Masson's trichrome, Victoria blue, and Nitroblue tetrazolium chloride staining. Blood chemistry was also evaluated.

Results showed that low-output groups (e.g., G1, G2, G4) maintained tissue temperatures below 37°C, with no significant histological or hematological changes. In contrast, high-output groups (e.g., G3, G7, G12) reached 41.6–57.1°C, inducing visible tissue responses such as blistering, erythema, coagulation, and epithelial detachment, followed by partial healing. Coagulation-related changes, including collagen and elastic fiber alterations, were observed but not fully restored at day 30.

These findings suggest that the PlazMagik device is capable of inducing targeted tissue coagulation under specific conditions. In particular, continuous exposure at maximum output for ≥5 seconds (single handpiece) or ≥10 seconds (triple handpiece), and ≥10 seconds at minimum output using Ar gas, appears effective for coagulation purposes, with acceptable safety profiles.

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Keywords: Plasma electrosurgery, porcine skin model, Cold plasma

PS-B-62

Evaluation of Immunotoxic Effects of Perinatal Lead Exposure in an Autism-Like Mouse Model

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Autism spectrum disorder (ASD) is a complex neurodevelopmental condition characterized by deficits in social communication and stereotyped behaviors. The etiology of ASD is multifactorial, involving genetic predisposition, environmental toxicants, and immune dysregulation. Lead (Pb), a well-known environmental pollutant, readily crosses the placenta and blood-brain barrier, raising concerns about its potential neurotoxic effects during early life.

This study aimed to evaluate the immunotoxic and behavioral consequences of lead exposure during gestation and lactation using BTBR T+Itpr3tf/J (BTBR) mice, a validated ASD-like model, and C57BL/6J (B6) mice as neurotypical controls. Pregnant dams were divided into three groups: one receiving distilled water, one exposed to lead (100 ppm) during lactation, and one exposed during both gestation and lactation via drinking water. On postnatal day 21, offspring were subjected to the passive avoidance test to assess learning and memory, followed by collection of spleen, blood, and brain tissues.

Cytokines (TNF-α, IFN-γ, IL-4, IL-17) and immunoglobulins (IgG1, IgG2a) were analyzed using sandwich ELISA. Pb-exposed BTBR mice showed significantly reduced latency in the behavioral task, indicating impaired memory retention. TNF-α expression was downregulated, while the IFN-γ/IL-4 and IgG2a/IgG1 ratios were elevated, reflecting a Th1-biased immune profile. IL-17 levels were also suppressed, suggesting diminished Th17 activity.

These findings demonstrate that perinatal lead exposure induces both neurobehavioral and immunological alterations in a genetically susceptible ASD-like model, underscoring the importance of minimizing environmental lead exposure during early development.

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*Corresponding author: Chang Yul Kim

Keywords: Autism spectrum disorder, BTBR mouse, Lead, Behavior test, Immunotoxicity

PS-B-61

Neurodevelopmental and Immuno-inflammatory Effects of Maternal Polyethylene Microplastic Exposure in an Autism-like BTBR Mouse Model

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Autism spectrum disorder (ASD) is a complex neurodevelopmental disorder characterized by social interaction deficits, communication challenges, and repetitive behaviors. While global prevalence increases, underlying mechanisms remain unclear. Recent studies suggest environmental pollutants like microplastics (MPs) may contribute to neurodevelopmental disorders.

This study evaluated neurodevelopmental and immunological effects of maternal polyethylene microplastic (PE-MP) exposure during pregnancy and lactation in autism-like BTBR T+Itpr3tf/J (BTBR) mice, using C57BL/6J (B6) mice as neurotypical controls. Pregnant BTBR and B6 females received PE-MPs (1,000 µg/L) via drinking water during gestation, lactation, or both periods. Control groups received distilled water. Offspring were exposed indirectly through placental transfer and breastfeeding. Behavioral performance was assessed using passive avoidance tests on postnatal day 21 to measure learning and memory. Immune and neurochemical markers included serum immunoglobulin E (IgE), cytokines (TNF-α, IFN-γ, IL-4) from splenocyte cultures, and brain-derived neurotrophic factor (BDNF) levels quantified by ELISA.

Results showed BTBR mice had impaired learning and memory compared to B6 controls, with cognitive deficits exacerbated by PE-MP exposure. BTBR mice displayed Th2-dominant immune profiles with elevated IgE levels significantly influenced by PE-MPs. B6 mice showed increased IgE and pro-inflammatory cytokines after PE-MP treatment, indicating broader immune activation. PE-MP exposure reduced brain BDNF levels, particularly in BTBR mice, suggesting disrupted neurodevelopment.

This study demonstrates that maternal PE-MP exposure during critical developmental periods negatively affects offspring cognitive function, neurotrophic support, and immune regulation, with pronounced effects in genetically autism-like mice. These findings highlight potential neurotoxic risks of environmental microplastics and emphasize reducing maternal exposure during pregnancy and breastfeeding to prevent or mitigate neurodevelopmental disorders like ASD.

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Keywords: Autism spectrum disorder, behavior test, BTBR, cytokines, microplastics, Immunoglobulin

PS-B-63

IRES-modified ssRNA adjuvant enhances humoral and cellular immune responses in neonatal mice with improved safety

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Neonatal immunization is fundamental for protecting neonates from infectious diseases, as infections are the leading cause of death in early life. However, the immaturity of the neonatal immune system and its limited capacity to induce Th1 responses remain major challenges in developing effective vaccine strategies for neonates. In this study, we present a single-stranded RNA (ssRNA) adjuvant, modified from the internal ribosome entry site (IRES) of the encephalomyocarditis virus (EMCV), as a potential vaccine adjuvant for neonates. The modified EMCV-IRES ssRNA adjuvant enhances antigen uptake by antigen-presenting cells and promotes the activation of B cells, macrophages and dendritic cells in infant mice. When co-administered with an inactivated influenza vaccine (IIV), ssRNA adjuvant significantly increases vaccine-specific antibody responses in neonatal mice, particularly vaccine-specific IgG2a, a Th1-biased isotype that plays a critical role in antiviral immunity. Furthermore, the ssRNA adjuvant enhances activation of antigen-specific CD4+ and CD8+ T cells and induces robust secretion of antigen-specific IFN-γ and antigen-specific TNF-α. In terms of safety, the modified EMCV-IRES ssRNA adjuvant demonstrates a superior safety profile compared to alum, a widely used adjuvant, in neonatal mice. Overall, our findings demonstrate that the modified EMCV-IRES ssRNA adjuvant is a promising pediatric vaccine adjuvant, capable of inducing balanced and protective Th1/Th2 immune responses in neonates while maintaining a favorable safety profile and potential for future clinical application.

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Keywords: Single-stranded RNA, Adjuvant, Neonatal Vaccine

PS-B-64

TXNIP ameliorates ductular reaction and liver inflammation and fibrosis

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Ductular reaction is a hallmark of chronic liver diseases (CLDs) and is closely linked to liver disease progression, including hepatic inflammation and fibrosis. However, there are currently no approved therapeutic agents that specifically target the ductular reaction, underscoring the need for a better understanding of its regulatory mechanisms. In this study, we aimed to elucidate the function of thioredoxin-interacting protein (TXNIP), a key mediator of cellular proliferation, apoptosis, and redox signaling, in the pathogenesis of CLDs. Using a cholangiocyte-specific Txnip-knockout mouse model (*Txnip^{ΔSOX9}*), we found that deletion of *Txnip* rendered mice more susceptible to 3,5-diethoxycarbonyl-1,4-dihydrocollidin (DDC)-induced ductular reaction, hepatic inflammation, and collagen accumulation. Loss of *Txnip* in cholangiocytes promoted excessive biliary epithelial cell proliferation while suppressing cell death, thereby disrupting normal epithelial homeostasis. Mechanistically, *Txnip* deficiency enhanced the secretion of pro-inflammatory and pro-fibrotic cytokines, including tumor necrosis factor α (TNF- α) and transforming growth factor β (TGF- β), from cholangiocytes. These cytokines activated liver-resident immune and mesenchymal cells such as Kupffer cells (KCs) and hepatic stellate cells (HSCs), further amplifying the inflammatory response and fibrogenesis. Conversely, overexpression of TXNIP in cholangiocytes significantly attenuated DDC-induced ductular expansion and reduced the extent of liver injury and fibrosis. Clinically, TXNIP expression was markedly reduced in cholangiocytes from liver biopsy samples of patients with various forms of CLDs, and its expression level showed a strong inverse correlation with the severity of ductular reaction. Collectively, our findings identify TXNIP as a crucial regulator of cholangiocyte homeostasis and pathological biliary remodeling, suggesting that therapeutic modulation of TXNIP signaling in cholangiocytes may represent a novel and promising approach for the treatment of CLDs characterized by aberrant ductular reaction.

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Keywords : Ductular reaction, Txnip, Liver, Cholangiocyte

PS-B-66

Estrogen-dependent regulation of FDPS in the mouse uterus and its expression in endometrial cancer

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The uterus is a dynamic organ that undergoes endometrial proliferation, decidualization, and regeneration in response to estrogen and progesterone. Estrogen, in particular, induces endometrial proliferation, which is essential for implantation, maintaining pregnancy, and the pathogenesis of gynecological diseases such as endometrial cancer and endometriosis. Therefore, identifying the genes regulated by estrogen and understanding their functions is crucial for maintaining a healthy uterine environment. FDPS is a key enzyme in the synthesis of FPP, which plays a crucial role in cholesterol biosynthesis and protein prenylation in the mevalonate pathway. Using an *in vivo* mouse model, we found that FDPS expression is regulated by estrogen. We observed that FDPS expression increased during the proestrus stage of the estrous cycle and decreased during the metestrus and diestrus stages. FDPS expression was also elevated at the 24-hour timepoint after estradiol treatment, and its estrogen-dependent response was further supported by treatment with the ER α antagonist ICI 162,780. FDPS overexpression has been reported in various cancers, including prostate cancer and glioma, but its role in endometrial cancer remains poorly understood. Through RNA-seq data and *in vitro* cell experiments, we discovered that FDPS is highly expressed in the human endometrial cancer cell line Ishikawa. This suggests that FDPS may contribute to the growth and survival of cancer cells in endometrial cancer. These results provide important insights into the potential role of FDPS in the uterus and suggest that targeting FDPS could be a viable therapeutic strategy for treating endometrial cancer.

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Keywords : Estrogen, Uterus, FDPS, Endometrial cancer

PS-B-65

Ecklonia cava ethanol extract attenuates ROS-mediated oxidative and inflammatory responses induced by particulate matter in MLE-12 cells

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Ecklonia cava, an edible brown marine alga, ethanol extract (ECE) has been reported to exhibit various pharmacological effects, including anti-inflammatory, anti-tumorigenic, anti-aging, anti-fungal, and radioprotective activities. In this study, we investigated whether ECE can exert antioxidant and anti-inflammatory effects against pathologic symptoms induced by particulate matter (PM) in MLE-12 cells, a lung type II alveolar epithelial cell line. ECE did not exhibit any cytotoxicity up to 250 μ g/mL and significantly attenuated PM-induced reactive oxygen species (ROS) generation and lipid peroxidation in MLE-12 cells in a dose-dependent manner. ECE effectively restored PM-induced morphological damages on the cell surface and chelated transition metals (Mg, Ca, Fe, and Al) in MLE-12 cells under PM exposure conditions. The activation of the NF- κ B pathway, one of the keys signaling pathways regulating the expression of inflammatory genes, upon PM exposure led to a significant increase in p-I κ B- α -positive cells, which was markedly ameliorated through ECE treatment. Furthermore, to assess NF- κ B transcriptional activity, MLE-12 cells were transfected with an NF- κ B luciferase reporter plasmid. ECE treatment effectively suppressed PM-induced NF- κ B transcriptional activation. In addition, PM significantly upregulated the mRNA expression of inflammatory cytokines (IL-1 β , TNF- α , IL-4, IL-6, etc.), whereas ECE treatment significantly attenuated it. These results suggest that ECE exerts protective effects against PM-induced oxidative stress and inflammation in MLE-12 cells, supporting its potential as a therapeutic agent for managing air pollution-related pulmonary diseases.

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Keywords : Ecklonia cava, Particulate matter (PM), MLE-12 cells, Oxidative stress, Metal chelating

PS-B-67

Comprehensive genotoxicity evaluation for a SARS-CoV-2 mRNA vaccine candidate using a four-test battery approach

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The emergence of messenger RNA (mRNA) vaccines has represented a paradigm shift in vaccine development, as exemplified by their critical role in the global response to the COVID-19 pandemic. Despite their widespread use, the safety profiling of mRNA vaccines remains incomplete, and concerns regarding their potential genotoxicity persist. Furthermore, specific regulatory guidelines for genotoxicity assessment of mRNA vaccines have not yet been established. To address this gap, we conducted a comprehensive genotoxicity evaluation of a SARS-CoV-2 mRNA vaccine coding the Omicron variant Spike protein (LNP composition: cholesterol, DMG-PEG, DSPC, and the ionizable lipid SM-102), using a four-test battery: the bacterial reverse mutation test (OECD TG 471), *in vitro* chromosomal aberration test (OECD TG 473), *in vitro* micronucleus test (OECD TG 487), and *in vivo* micronucleus test in ICR mice (OECD TG 474). No significant genotoxic effects of the mRNA vaccine were observed in any of the assays. Specifically, the mRNA vaccine did not induce revertant colonies up to 300 μ g (mRNA)/6.3 mg (LNP)/plate, chromosomal aberrations up to 90 μ g/1.89 mg/ml in CHL cells, or micronucleated cells up to 100 μ g/2.1 mg/mL in CHL cells. Similarly, the *in vivo* micronucleus test showed no significant increase in micronucleated erythrocytes following intramuscular administration of 100 μ g/2.1 mg per mouse. However, a significant (~60%) decrease in the polychromatic erythrocyte (PCE) ratio in bone marrow was observed, suggesting a potential impact on hematopoiesis. These results indicate that while the SARS-CoV-2 mRNA vaccine does not elicit detectable genotoxicity under standard testing conditions, its potential effects on bone marrow function warrant further investigation. Our findings may contribute to the development of regulatory guidelines for the genotoxicity assessment of mRNA-based vaccines.

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Keywords : Genotoxicity, mRNA vaccine, SARS-CoV-2

PS-B-68

Histological assessment of early postnatal-period development of major organs in the Common marmoset (*Callithrix jacchus*)

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Non-human primates, such as the common marmoset (*Callithrix jacchus*), have become valuable models in biomedical research owing to their close resemblance to human biology. In contrast to most animals, humans are born with structurally mature organs, while postnatal histological maturation predominates in other species. The purpose of this study was to characterize the normal histological development of major visceral organs—liver, kidney, spleen, heart, and lung—in the marmoset from birth through key juvenile stages. To establish baseline histological features of postnatal-period development in the common marmoset, tissue samples were collected from marmosets at PND 6, 15, 20. At necropsy, representative tissue samples were fixed in 10% neutral-buffered formalin. Tissues were processed routinely, embedded in paraffin, sectioned, and stained with hematoxylin and eosin (H&E). Microscopic evaluation focused on the assessment of PND-dependent morphological changes in each organ. Progressive postnatal maturation was observed across these organs. In the liver, hepatic cord simplification and ductal plate remodeling advanced, with persistent EMH at PND 20. The kidney showed active nephrogenesis from PND 6, with stage 3 nephrons predominating by PND 20. The lung underwent alveolarization, but alveolar septae remained immature. The heart exhibited increased thickness of all chamber walls and myocardial hypertrophy in the interventricular septum. In the spleen, perivascular lymphoid aggregation began by PND 6 but remained immature at day 20. Notably, the spleen displayed the slowest histological maturation among the organs examined. This study provides the overview of major organ maturation in marmosets based on H&E-stained tissue sections. These results contribute to improved model selection and more accurate assessment of tissue changes in non-human primate research. Longitudinal studies extending beyond PND 20 would also be valuable in defining the full trajectory of organ maturation, especially for organs like the spleen which mature more slowly.

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Keywords : Common marmoset (*Callithrix jacchus*), Histological maturation, Early postnatal-period development

PS-B-69

Luteolin induces apoptosis in human melanoma cancer cells via the MAPK pathway

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Luteolin is a substance found in fruits and vegetables and is known to have biological effects such as anti-inflammation, antioxidant, anti-allergic, and anti-diabetic. This experiment was conducted to confirm whether luteolin has an anticancer effect on human melanoma cells, A375P and A375SM, through in vitro experiments. It was confirmed through MTT that luteolin decreased the viability of A375P and A375SM in a concentration-dependent manner. After that, to confirm whether the decrease in cell viability of A375P and A375SM by luteolin was due to apoptosis, morphological observation was performed using DAPI and quantitative confirmation was performed using FACs. The Western blot results confirmed that apoptosis was induced through the protein expression of cleaved PARP, cleaved caspase-3, -8, and -9 when luteolin was applied to A375P and A375SM. In addition, we confirmed the increase or decrease of extracellular Signal-regulated kinase (ERK), c-Jun N-terminal kinase (JNK), and p38 kinase, which are Mitogen-activated protein kinase (MAPK) pathway proteins that regulate various cellular processes such as proliferation, apoptosis, and stress response among various mechanisms of cancer. To confirm whether apoptosis is induced mediated by ERK, one of the MAPK pathways, pretreatment with ERK inhibitor PD98059 was performed, and the decrease in cleavage of PARP was confirmed through western blot. The results of the previous experiments show that luteolin induces apoptosis through the MAPK/ERK pathway in human melanoma cells A375P and A375SM, suggesting its potential as an anticancer agent.

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Keywords : Luteolin, Human melanoma cancer, Apoptosis, MAPK pathway

PS-B-70

Phloretin induce apoptosis through the MAPK pathway in human melanoma cells

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Melanoma is a type of skin cancer that occurs in melanocytes and is characterized by high metastasis potential and poor prognosis if not treated early. Phloretin, which is known to be contained in the skins of apples and pears, is a natural polyphenol that exhibits pharmacological effects such as anti-inflammatory and antioxidant, and is known to have anticancer effects in various cancer cell species, but studies on the induction and action mechanism of apoptosis in human melanoma cell lines A375P and A375SM are incomplete. Therefore, this study aimed to investigate the anticancer action mechanism of phloretin in human melanoma cell lines A375P and A375SM. MTT assay was performed to determine whether phloretin affects cell viability in A375P and A375SM cells, and as a result, cell proliferation and survival inhibition were confirmed. After that, DAPI staining and Annexin V/PI staining were performed to confirm whether the induced cell death was apoptosis, and it was confirmed that apoptosis increased in a concentration-dependent manner. Western blot was performed to confirm the expression of apoptosis and MAPK pathway-related proteins, and as a result, increased apoptosis-related proteins caspase-8, caspase-9, caspase-3, and PARP fragmentation were confirmed. In addition, increased expression of p-ERK and p-JNK, and decreased expression of p-p38 were confirmed in MAPK pathway-related proteins. After 2 hours of pretreatment with PD98059, an ERK inhibitor, apoptosis protein expression was confirmed by treatment with phloretin. As a result, it was confirmed that the fragmentation of PARP, caspase-3 was reduced, and apoptosis was mediated through the MAPK/ERK pathway. As a result, this study showed the potential of phloretin as a natural anticancer agent for human melanoma.

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Keywords : Phloretin, Apoptosis, Melanoma, MAPK pathway

PS-B-71

Breathing plastic: ROS-mediated lung toxicity of environmental secondary microplastics

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Despite rising awareness of microplastic contamination as a global environmental and public health issue, most toxicological research has concentrated on primary microplastics, leaving the potential health risks of environmentally weathered secondary microplastics less understood. In this study, we examined lung inflammation in mice exposed to 1 µm-sized primary and secondary microplastics, with or without UV-induced oxidation. Microplastics were administered either once or repeatedly via pharyngeal aspiration. On day 1 after a single exposure, microplastics caused acute lung inflammation, characterized by increased levels of lactate dehydrogenase (LDH), interleukin-1β (IL-1β), reactive oxygen species (ROS) in alveolar macrophages, and a higher percentage of neutrophils in bronchoalveolar lavage fluid. These inflammatory markers showed a positive correlation with ROS production by microplastics (LDH: $r=0.83$, $p<0.001$; IL-1β: $r=0.66$, $p<0.05$; cellular ROS: $r=0.65$, $p<0.05$; percentage of neutrophils: $r=0.92$, $p<0.001$). By day 28, however, these parameters returned to levels similar to the control group. Conversely, repeated exposure over 30 days (10 instillations) resulted in persistent lung inflammation, characterized by neutrophil infiltration, elevated LDH levels, and the formation of multinucleated giant cells, indicating granulomatous lung pathology. Overall, these findings suggest that secondary microplastics, especially those altered by environmental aging, may pose a greater risk to respiratory health than previously recognized, potentially leading to chronic pulmonary inflammation. Due to their widespread presence and persistence in the environment, additional research and careful regulation are necessary to better understand and mitigate the potential health impacts of microplastic exposure.

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Keywords : Microplastics, Lung inflammation, Reactive oxygen species, Fragmentation

PS-B-72

Establishment and Histopathological Characterization of a Photothrombotic Internal Capsule Infarction Model in Miniature Pigs

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Despite its recognized contribution to post-stroke disability, white matter infarction remains relatively underexplored in preclinical research. Most existing models focus on cortical gray matter injury, limiting their applicability for modeling subcortical white matter infarction. In this study, we developed a focal infarction model in the posterior limb of the internal capsule of miniature pigs using a photothrombotic approach and characterized histopathological alterations at a subacute time point. Miniature pigs (KRIBB Small Pig, 20–25 kg) received intravenous administration of Rose Bengal (20 mg/kg), followed by stereotactically guided 532 nm laser irradiation targeting the posterior internal capsule. Animals were sacrificed 7 days post-injury, and whole-brain tissues were collected for histological and immunofluorescence analyses. Hematoxylin and eosin (H&E) and Nissl staining were used to localize infarct regions and assess white matter structural changes. Apoptotic cell death was evaluated using TUNEL staining. Glial activation was assessed through immunofluorescence labeling for astrocytic and microglial markers. Photothrombotic induction consistently resulted in clearly delineated infarcts within the targeted internal capsule region. H&E and Nissl staining revealed marked tissue rarefaction and vacuolization in the affected white matter. TUNEL staining revealed apoptotic cell death within both the lesion core and the peri-infarct region. Immunofluorescence analyses demonstrated elevated microglial activation and prominent astrocytic reactivity at 7 days post-injury. This miniature pig model of internal capsule infarction replicates key histopathological characteristics of subcortical white matter injury, including glial responses and apoptotic neurodegeneration. These findings support its utility for investigating the pathophysiology of subcortical stroke and for the preclinical evaluation of therapeutic strategies targeting white matter pathology.

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Keywords : Apoptosis, Glial activation, Miniature pig, Photothrombotic injury, White matter ischemia

PS-B-73

FLASH Irradiation Attenuates Radiation-Induced Intestinal and Testicular Damage in Mice

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FLASH radiotherapy (FLASH-RT), characterized by ultra-high dose rates (>40 Gy/s), delivers radiation doses several times higher than those of conventional radiotherapy (>5 Gy/s). This study aimed to evaluate the differential effects of FLASH and conventional irradiation on acute normal tissue toxicity in mice, focusing on the intestine and testes.

C57BL/6 mice were randomly assigned to three groups: control (0 Gy), FLASH irradiation (3 Gy at 40 Gy/s), and conventional irradiation (3 Gy at 0.067 Gy/s). Total body irradiation (TBI) was performed, and tissue samples were collected at 2, 4, 7, 14, and 28 days post-irradiation.

Morphological analysis revealed a marked reduction in testicular size in both irradiated groups, with significantly less shrinkage observed in the FLASH group at 28 days. Similarly, intestinal villi length was reduced in response to irradiation; however, FLASH-treated mice exhibited relatively preserved villi structure compared to those receiving conventional irradiation.

These results indicate that FLASH irradiation may mitigate radiation-induced toxicity in radiosensitive normal tissues such as the testis and intestine. This suggests the potential for FLASH-RT to enhance therapeutic outcomes by reducing normal tissue damage and treatment duration, and supports its further development as a next-generation radiotherapy modality.

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Keywords : FLASH irradiation, Radiation-induced toxicity, Normal tissue protection

PS-B-74

Integrated Transcriptomic Analysis Reveals Placental Responses to Prenatal Arsenic Exposure

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Arsenic (As) is a metalloid with both metallic and non-metallic properties and is widely distributed in the environment in various chemical forms, representing a major environmental pollutant in soil, water, and air. Most environmental arsenic exists in inorganic forms, entering the human body mainly through ingestion or inhalation. Classified as a human carcinogen, arsenic exposure is associated with numerous adverse health effects, including skin disorders and diseases affecting cardiovascular, respiratory, digestive, and nervous systems. Particularly in Korea, regions with industrial complexes and mining areas exhibit high arsenic contamination levels, posing significant exposure risks.

While cellular responses to arsenic exposure have been extensively studied, the impacts of prenatal arsenic exposure on fetal development through the placenta remain poorly understood. This study re-analyzed publicly available mouse datasets (GSE222092, E-MTAB-14214) integrating bulk RNA-seq and single-cell RNA-seq (scRNA-seq) analyses to elucidate integrated molecular mechanisms underlying placental responses to arsenic exposure.

Bulk RNA-seq analysis of arsenic-exposed mouse placenta identified differentially expressed genes (DEGs), and scRNA-seq data were utilized to evaluate DEG enrichment across various placental cell types. Results revealed notably high DEG enrichment in yolk sac cells and mesenchymal-like cells, indicating their heightened sensitivity to arsenic exposure.

Gene Set Enrichment Analysis (GSEA) of DEGs identified in yolk sac cells and mesenchymal-like cells showed significant alterations in hallmark pathways (FDR q-value < 0.05). Specifically, oxidative phosphorylation (NES = 2.48) and mTORC1 signaling pathways (NES = 2.37) were significantly upregulated in yolk sac cells, whereas epithelial-mesenchymal transition (NES = -2.43), hypoxia (NES = -2.32), TNFA signaling via NFκB (NES = -2.3), and p53 pathway (NES = -2.19) were notably downregulated. Particularly, the suppression of the p53 pathway indicates impairment in DNA damage response and cell cycle regulation, highlighting potential mechanisms for arsenic-induced placental toxicity and increased carcinogenic risk during pregnancy.

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Keywords : Arsenic exposure, Placenta, Single cell RNA sequencing, P53 signaling

PS-B-75

MAPK pathway-driven acute lung injury induced by substance O via oxidative stress in mice

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Substance O, an industrial compound with known pulmonary toxicity, induces acute lung injury (ALI), though its underlying mechanisms remain poorly understood. This study investigated the contribution of oxidative stress and inflammatory signaling in substance O-induced ALI using a murine model. C57BL/6 mice were intratracheally exposed to substance O (1–25 mg/kg) and evaluated 24 hours later. Substance O exposure resulted in dose-dependent body weight loss and decreased survival. The lung weight-to-body weight ratio increased significantly, suggesting pulmonary edema. Histopathological examination (H&E staining) revealed hallmark features of ALI, including alveolar wall thickening, neutrophilic infiltration, and perivascular inflammation. Bronchoalveolar lavage fluid (BALF) analysis showed elevated total immune cell counts, particularly neutrophils, with increased macrophages and lymphocytes. Both BALF and serum levels of IL-6 and TNF- α were significantly elevated, indicating strong local and systemic inflammation. Markers of oxidative stress were significantly altered. Lung tissues showed reduced levels of reduced glutathione (GSH), increased oxidized glutathione (GSSG), decreased total GSH, and a lowered GSH/GSSG ratio—indicative of severe oxidative imbalance. Western blot analysis revealed minimal NF- κ B pathway activation, as evidenced by limited p65 nuclear translocation and weak I κ B α degradation. In contrast, robust activation of the MAPK pathway was observed, with significant phosphorylation of ERK1/2, JNK1/2, and p38, implicating these kinases in mediating the inflammatory and apoptotic responses to substance O. In conclusion, substance O induces acute lung injury in mice through pronounced oxidative stress and strong MAPK pathway activation, while NF- κ B plays a minor role. These findings provide mechanistic insight into heavy metal-induced pulmonary toxicity and suggest MAPK signaling as a potential therapeutic target for mitigating environmental or occupational lung injury. This work was supported by the National Research Foundation of Korea (NRF) grant funded by the Korea government (MSIT) (2020R1A5A8019180, RS-2025-00573202).

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Keywords: Acute lung injury, Oxidative stress, MAPK signaling, Environmental toxicology

PS-B-77

Comparative effects of aerosolized THC and CBD via vaping in mice: a study of exposure responses

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Δ^9 -Tetrahydrocannabinol (THC) and cannabidiol (CBD) are the predominant bioactive phytocannabinoids in Cannabis sativa and have been meticulously investigated for their pharmacological and biological attributes. Nevertheless, the toxicological repercussions of inhalational exposure to these compounds remain insufficiently characterized.

In the present study, we sought to delineate the differential toxicodynamic profiles of THC and CBD administered via vaporization, a methodology recognized for its rigor and translational relevance in comparative biomedical research. A base oil vehicle was employed to enhance the systemic bioavailability of each phytocannabinoid.

Eight-week-old male and female ICR mice (n = 5 per sex per group) received a single 50 mg inhaled dose of either THC or CBD using a vaporizer. Tissue collections were undertaken on post-exposure days 1, 3, and 14. The control group was exposed to vehicle vapor alone and processed identically.

Endpoints encompassed body and organ mass indices, hematological profiling (complete blood count), serum cytokine quantification, and pulmonary histopathology. The investigation furnishes comprehensive evidence of sex-dependent and compound-specific biological effects following exposure to vaporized THC or CBD. Salient findings include significant alterations in body and organ weights, discrete shifts in hematological parameters, and divergent immunological and histopathological manifestations within pulmonary tissue.

Collectively, these data underscore the necessity of compound-specific evaluation to elucidate the inhalational safety profiles of THC and CBD. All experimental protocols conformed to institutional ethical standards (IACUC approval: GNU-241113-M0217).

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Keywords: Δ^9 -tetrahydrocannabinol (THC), Cannabidiol (CBD), Vaping

PS-B-76

Degradation of Extracellular Vesicles by Respiratory Phospholipases Limits Their Drug Delivery Potential

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Extracellular vesicles (EVs) are membrane-bound nanostructures (30–1000 nm in diameter) secreted by various cell types. They have gained attention as promising drug delivery vehicles due to their ability to transport bioactive molecules between cells, as well as their high biocompatibility and low immunogenicity. However, their clinical translation is hindered by limited in vivo stability, particularly in extracellular environments such as the respiratory tract, where enzymatic degradation may compromise vesicle integrity and reduce drug delivery efficiency.

In this study, we investigated whether phospholipase activity in bronchoalveolar lavage fluid (BALF) contributes to the degradation of extracellular vesicles (EVs). EVs were isolated from BALF via differential centrifugation and characterized by nanoparticle tracking analysis (NTA) and scanning electron microscopy (SEM). To evaluate the degradative effect of BALF, isolated EVs were incubated with BALF. Compared to PBS-incubated EVs, BALF-incubated EVs showed a reduced number of larger-sized particles and smaller vesicle size, indicating structural disruption. Comparable degradation was observed following incubation with purified phospholipase. Importantly, treatment of BALF with a phospholipase inhibitor attenuated EV degradation. In addition, liquid chromatography–mass spectrometry (LC-MS) analysis identified specific phospholipid breakdown products after incubation with phospholipase, confirming enzymatic membrane cleavage. Notably, this degradation was effectively prevented by the addition of phospholipase inhibitors, underscoring the enzyme's role in EV destabilization.

These findings suggest that extracellular phospholipase activity in the respiratory environment may impair EV stability and reduce their drug delivery potential. Targeting this degradation pathway could enhance the performance of EV-based therapeutics in respiratory disease applications.

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Keywords: Extracellular vesicles, Phospholipase, Drug delivery system, Lung diseases, Enzyme inhibition

PS-B-78

CA Leaf Extract Attenuates Glutamate-Induced Oxidative Stress in HT-22 Cells and Ameliorates Scopolamine-Induced Cognitive Deficits in Mice

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Cognitive impairment, often associated with aging and neurodegenerative disorders, is characterized by a progressive decline in memory and cognitive function. A significant pathogenic mechanism linked to cognitive impairment involves oxidative stress-induced neuronal apoptosis and cholinergic hypofunction. CA has been reported to exhibit various biological activities, including antioxidant and anti-inflammatory effects. However, the neuroprotective potential of CA leaves, a by-product of the plant, has yet to be investigated. In this study, we aimed to investigate the neuroprotective effects of CA leaf extract against glutamate-induced oxidative stress in HT-22 hippocampal neuronal cells, as well as its cognitive-enhancing effects in a scopolamine-induced cognitive impairment mouse model. The CA leaf extract significantly reduced glutamate-induced oxidative stress and enhanced cell viability in HT-22 cells in vitro. In vivo, the extract ameliorated scopolamine-induced cognitive dysfunction, as demonstrated by behavioral assessments, including the Y-maze, novel object recognition (NOR), and passive avoidance tests. It also restored acetylcholine and acetylcholinesterase levels in the hippocampus, suggesting a recovery of cholinergic function. Furthermore, the extract promoted the nuclear translocation of the antioxidant transcription factor Nrf2 and upregulated downstream antioxidant proteins such as HO-1 and NQO1. Additionally, it activated the TrkB/CREB signaling pathway and enhanced the expression of brain-derived neurotrophic factor (BDNF). These findings suggest that CA leaf extract exhibits neuroprotective properties by attenuating oxidative stress and cholinergic dysfunction, potentially through the activation of the Nrf2 and TrkB/CREB pathways. Thus, CA leaves may serve as a promising natural agent for the prevention or improvement of cognitive impairment.

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Keywords: Cognitive impairment, Neuroprotection, Oxidative stress, Cholinergic dysfunction

PS-B-79

Fermented *Lonicera japonica* Flos extracts Alleviate IL-6-induced Ulcerative colitis by Restoring Intestinal Barrier Function and Modulating Tight Junction

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Inflammatory bowel diseases (IBD) is a special chronic intestinal inflammatory disease, which is mainly divided into Crohn's disease (CD) and ulcerative colitis (UC). Tight junctions (TJs) play an important role in intestinal barrier function. TJs in intestinal epithelial cells are composed of different junctional molecules, such as claudin and Occludin (OCLN), and regulate the paracellular permeability of water, ions, and macromolecules in adjacent cells. One of the most crucial roles of the TJ structure is to provide a physical barrier to luminal inflammatory molecules. Impaired integrity and structure of the TJ barrier result in a forcible activation of immune cells and chronic inflammation in tissues. This study aim was to investigate the protective effects of fermented *Lonicera japonica* Flos hot water extracts (FLE) on the intestinal barrier function and elucidate focusing on tight junction activation. As a result, FLE has not cytotoxicity in Caco-2 cell and has an excellent anti-oxidants effect. Also, FLE increased TEER value and decreased permeability compared with IL-6 group. Especially, it was significantly increased in FLE 200µg/ml+IL-6 group. Additionally, the result of immunofluorescence, ZO-1 and OCLN expression were shown clearer without interruption in a concentration-dependent manner with FLE. In conclusion, FLE strengthened the intestinal TJ barrier by recovering tight junction levels in Caco-2 cells.

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Keywords : *Lonicera japonica* Flos, Intestinal barrier function, Tight junction, Ulcerative colitis, Inflammatory bowel disease

PS-B-81

Luteolin induces caspase-dependent apoptosis and LC3-II-mediated autophagy in human oral cancer cells

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Conventional treatment modalities for oral cancer, encompassing surgical interventions, chemotherapy, and radiotherapy, are associated with significant adverse effects and demonstrate limited effectiveness against recurrent or metastatic cancers. Consequently, naturally occurring substances with fewer adverse effects have garnered interest as alternative treatments. Luteolin, a flavonoid found in various fruits and vegetables, has been reported to have anticancer, anti-inflammatory, and antioxidant effects. In this study, the anticancer effects of luteolin were evaluated using human oral cancer cell lines (MC-3, HSC-4) and a mouse xenograft model.

In vitro study, 3-(4,5-dimethylthiazol-2-yl)-2,5-diphenyltetrazolium bromide(MTT) and wound healing assays demonstrated that luteolin inhibited cell viability and proliferation in a concentration-dependent manner. The induction of apoptosis was confirmed by 4',6-diamidino-2-phenylindole(DAPI) staining, Annexin V/PI flow cytometry, and increased expression of cleaved- caspase 3, 8, 9, and cleaved poly (ADP-ribose) polymerase (cleaved-PARP). Furthermore, an augmented level of phosphorylation of c-jun N terminal kinase (JNK) in the Mitogen-Activated Protein Kinase (MAPK) pathway was identified. The induction of autophagy was confirmed by an increase in Microtubule-associated protein 1A/1B-light chain 3 (LC3)-II, a decrease in p62, and acridine orange staining. *In vivo* study, intraperitoneal injection of luteolin (20 mg/kg) into immunodeficient mice implanted with MC-3 cells led to a reduction in tumor volume and weight, although this reduction was not statistically significant. No evidence of toxicity was observed in the liver and kidney tissues, and the alterations in apoptosis and autophagy-related proteins in the tumor tissue were consistent with the results of *in vitro* experiments.

These results suggest that luteolin inhibits oral cancer progression by inducing apoptosis and autophagy through the JNK signaling pathway. These findings suggest that luteolin may be a good alternative for oral cancer treatment if further studies are conducted on the relationship between apoptosis and autophagy.

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Keywords : Luteolin, Apoptosis, MAPK pathway, Autophagy, Oral cancer

PS-B-80

Polystyrene nanoplastics exacerbates lipid accumulation and mitochondrial dysfunction through JNK signaling in hepatocytes under free fatty acids-induced metabolic stress

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Nanoplastics (NPs) are originated from microplastics and may act as environmental toxins and pose potential health risks. Polystyrene nanoplastics (PS-NPs) exposure has been shown to accumulate in hepatocytes and induce liver damage. However, the hepatic lipid-associated effect and underlying mechanism of PS-NP exposure in normal hepatocytes remains unclear. In this study, we investigated the effects of PS-NPs exposure on lipid metabolism in human Chang liver cells under metabolic stress induced by free fatty acids. Human hepatocytes were exposed to increasing concentrations of PS-NPs and intracellular accumulation with minor cytotoxicity and lipid accumulation was observed using flow cytometry. When PS-NPs were exposed under free fatty acids, significant lipid accumulation was induced with increased lipogenesis-related marker proteins, PPAR γ , FAS, and CD36. Under these conditions, reduction in live mitochondria, fatty acid oxidation, ATP production accompanied by NAD⁺ to NADPH ratio, indicating disruption in mitochondrial function and redox balance. During this process, PS-NPs accumulated in mitochondrial fraction and translocated TFAM, a key marker of mitochondria transcription from mitochondria to nucleus, which led to its inactive state. SP60012, a JNK inhibitor, ameliorated PS-NPs-induced mitochondrial dysfunction and lipid accumulation under free fatty acids exposure as observed by recovered live mitochondria and lipid-associated marker proteins comparable to normal conditions. Therefore, our findings suggest that PS-NPs sensitizes and exacerbates lipid accumulation under free fatty acids through JNK-mediated mitochondrial dysfunction in normal hepatocytes.

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Keywords : Nanoplastics, JNK signaling, Hepatocytes, Lipid accumulation, Mitochondrial dysfunction

PS-B-82

Evaluation of immunotoxic effects of polyethylene microplastics and lead exposure during pregnancy and lactation on neonates in an autism-like mouse model.

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Autism spectrum disorder (ASD) is a chronic neurodevelopmental disorder characterized by persistent deficits in social interaction, communication, and repetitive or stimulatory behaviors. The global prevalence of ASD continues to rise. With the acceleration of industrialization and urbanization, human exposure to environmental pollutants such as polyethylene microplastics and lead has increased. These substances are increasingly suspected of contributing to the pathophysiology of ASD through immunological and neurobiological mechanisms, potentially interacting with genetic susceptibility.

This study investigated the effects of developmental co-exposure to PE-MPs and lead during pregnancy and lactation using BTBR T+Itpr3tf/J mice, a widely accepted ASD-like animal model, with C57BL/6J mice serving as neurotypical controls. Mice were given a mixture of polyethylene microplastics at a concentration of 1,000 µg/ml and lead at a concentration of 0.1 mM dissolved in sterile Exposure was carried out from conception to delivery and continued through the lactation period until postnatal day 21.

Results included body weight, behavioral tests, cytokines (TNF- α , IL-17, IFN- γ , IL-4) from spleen cells, serum immunoglobulins (IgE, IgG1, IgG2a), and brain neurotrophic factor (BDNF). Body weight of mice decreased when exposed to PE-MP and lead. In the passive avoidance test, combined exposure decreased the time spent in the light box, resulting in impaired behavioral abilities. Simultaneous exposure to polyethylene microplastics and lead decreased immune cell counts and BDNF. Analysis of spleen cytokines in BTBR mice showed increased TNF- α and IFN- γ , decreased IL-4 and IL-17, and increased serum IgE, IgG1, and IgG2a levels. These results suggest Th1 immune overactivation and suppression of Th2 and Th17 responses. Reduced BDNF levels have negative effects on neurodevelopment.

This study suggests that developmental co-exposure to PE-MPs and lead exacerbates immune dysregulation and neurodevelopmental impairments in genetically susceptible offspring, highlighting the potential role of environmental pollutants in the pathogenesis of ASD.

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Keywords : Autism spectrum disorder(ASD), Polyethylene microplastics, Lead, BTBR mouse

PS-B-83

Polyhexamethylene guanidine hydrochloride-phosphate induces restrictive ventilation defect and alters lung resistance and compliance in mice

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Background : Polyhexamethylene guanidine phosphate (PHMG-p), a major ingredient of humidifier disinfectants, is known to induce inflammation, interstitial pneumonitis, and fibrosis in the lungs. Many studies have evaluated the histopathologic pulmonary toxicities of PHMG-p in rodent models. However, there is limited research specifically addressing changes in pulmonary function test (PFT) following exposure to PHMG-p. **Objectives :** This study aimed to investigate the acute and chronic effects, as well as the dose- and time-response, of PHMG-p on the lungs in mice using PFT and histopathologic examinations.

Materials and Methods : This study utilized single and five-time intratracheal instillation models. In the single instillation model, mice received either saline or PHMG-p (0.3, 1, or 3 mg/kg) and were sacrificed at 2, 4, and 8 weeks after instillation. In the five-time instillation model, saline or PHMG-p (0.1, 0.3, or 1 mg/kg) was administered five times at one-week intervals, and mice were sacrificed 10 weeks after the first instillation. Using a PFT system, parameters related to lung volume, resistance, and compliance were measured and analyzed. Inflammation and fibrosis were scored through histopathologic examination.

Results : In both the single and five-time instillation models, decreases in lung volume and alterations in resistance and compliance parameters indicated a restrictive ventilation defect, decreased lung compliance, and increased lung resistance due to PHMG-p instillation. Histopathologic examination showed increases in lung inflammation and fibrosis scores. Changes in several lung volume and compliance parameters, as well as histopathology, were dose-dependent. Lung resistance and compliance parameters had significant correlations with lung inflammation and fibrosis scores.

Conclusion : PHMG-p exposure in mice resulted in a restrictive ventilation defect with altered lung resistance and compliance, along with histopathologic lung inflammation and fibrosis.

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Keywords : Polyhexamethylene guanidine phosphate, Pulmonary function test, Fibrosis, Mouse

PS-B-84

Regulation of Apoptosis and Fibrosis via the CHI3L1-p53 Pathway in IPF

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Idiopathic Pulmonary Fibrosis (IPF) is a chronic and progressive interstitial lung disease marked by excessive extracellular matrix (ECM) accumulation, epithelial damage, and fibroblast activation. CHI3L1 (Chitinase-3-like protein 1) is highly expressed in IPF lungs and plays a key role in promoting fibrotic macrophage activation, myofibroblast differentiation, and resistance to apoptosis by inhibiting p53 signaling.

To explore the therapeutic potential of CHI3L1 inhibition, we administered an anti-CHI3L1 antibody intravenously at 3 mg/kg and 10 mg/kg on days 6 and 10 following bleomycin exposure in mice. Histological analysis (H&E and Masson's trichrome staining) showed that CHI3L1 antibody treatment significantly reduced fibrotic remodeling and collagen deposition, improving Ashcroft scores. Treated mice also exhibited enhanced body weight recovery and reduced lung weight-to-body weight ratios.

BALF cytology demonstrated decreased macrophage and neutrophil infiltration in antibody-treated groups. Immunofluorescence staining revealed downregulation of α -SMA and CHI3L1 expression, and restoration of epithelial marker RAGE. qRT-PCR and western blot analysis confirmed molecular changes consistent with restored apoptosis: decreased expression of pro-apoptotic genes (Caspase-3, NOX4, Bax) and increased levels of Bcl2 and p53.

These findings indicate that CHI3L1 promotes lung fibrosis by suppressing the p53-mediated apoptotic pathway, and that antibody-based CHI3L1 inhibition reactivates this pathway to facilitate fibrosis resolution. Our study supports the potential of anti-CHI3L1 therapy as a promising treatment strategy for IPF.

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Keywords : Idiopathic Pulmonary Fibrosis (IPF), Chitinase 3-Like-1 (CHI3L1), P53 signaling, Apoptosis

PS-B-85

Toxicological Evaluation of Silk-Derived Protein through Genetic and General Toxicology Studies

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This study aimed to evaluate the toxicological safety of silk-derived protein. With the growing demand for functional ingredients, the application of silk-derived protein as a promising material for functional foods and biomedical fields is becoming increasingly crucial. However, the absence of comprehensive toxicological data prohibits regulatory approval and broader industrial use. To address this issue, we conducted genetic and general toxicology studies, adhering OECD test guidelines and the principles of Good Laboratory Practice (GLP). The test substance, a silk-derived protein powder provided by 'cefo', was dissolved in sterile distilled water and used in the experiments. To evaluate genetic toxicology, we performed the Bacterial Reverse Mutation Test (TG 471), In Vitro Chromosomal Aberration Test (TG 473), and In Vitro Micronucleus Test (TG 474). In parallel, general toxicology was evaluated through a Single-Dose Oral Toxicity Study (TG 423) and a Repeated Dose 90-day Oral Toxicity Study (TG 408). No toxicological effects were observed in genetic testing. Although minor changes were noted in certain organs in the 90-day study, these were not dose-dependent and biologically significant. Our findings suggest that silk-derived protein is safe for use, offering a favorable toxicological profile for potential applications in the biomedical and food industries. Future studies will focus on conducting clinical trials in humans, and this study will provide essential safety data to support subsequent research.

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Keywords : Silk-derived proteins, Genotoxicity assays, Single-Dose Oral Toxicity Study, Repeated Dose 90-day Oral Toxicity Study

PS-B-86

Evaluation of SARS-CoV-2 mRNA Vaccine Candidate-induced Toxicological Responses in hACE2 Transgenic and C57BL/6J Mice

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The rapid development of SARS-CoV mRNA vaccines, unlike traditional drug development, limited comprehensive toxicological assessment of potential adverse effects. This study aimed to evaluate whether the binding between Spike protein expressed in vivo following mRNA vaccination and the ACE2 receptor elicits distinct toxicological phenotypes. The mRNA vaccine candidate (CUK3-1/LNP128) were injected intramuscularly in the left thigh muscle of 6-week-old female C57BL/6J and hACE2 transgenic mice at a dosage of 100 µg/100 µL/head for three times at 2-week intervals. We observed clinical signs, body weight changes, estrous cycle monitoring via vaginal smear. Necropsy was performed 2 days post-secondary injection (dpi), and complete blood count, blood chemistry, and organ weights were analyzed. Both groups showed similar responses following mRNA vaccination, including a transient decrease in body weight that subsequently recovered. Hematopoietic suppression was observed by reductions in RBC, hemoglobin, platelets, reticulocytes. Hepatic changes included increased AST and ALT, and decreased albumin, total protein (TP). Renal effects were indicated by reduced blood urea nitrogen. Cardiac stress was suggested by increased NT-proBNP. Inflammation responses was shown by increased neutrophils, lymphocytes and basophils. Coagulation changes included increased in D-dimer and reductions in fibrinogen, prothrombin time, and activated partial thromboplastin time. In terms of organ weights, increases were noted in the spleen, lymph nodes, whereas the thymus weight was decreased. Although both genotypes showed elevated AST and triglyceride levels, the increases were relatively higher in hACE2 mice than in B6 mice, along with more irregular estrous cycles. Other observed genotype-specific changes were not statistically significant or toxicologically relevant. These results indicate that mRNA vaccination induced hematopoietic suppression, inflammatory and physiological stress responses, changes in lipid and metabolic profiles, hyperactivation of the coagulation system, and increased cardiac stress markers in both genotypes. This study suggests that genotype-specific toxic effects attributable to ACE2-spike protein interaction should be considered

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Keywords : Messenger RNA vaccine, SARS-CoV2, Toxicological phenotype, ACE2 receptor

PS-B-88

Digital Pathology Image Resources at LAREB: Representative Whole Slide Images and Research Applications

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National Institute of Food and Drug Safety Evaluation (NIFDS) established and has operated the Laboratory Animal Resource Bank (LAREB) since 2018 to promote the sharing of laboratory animal-derived resources such as frozen tissues, cells, serum samples, formalin-fixed tissues, paraffin-embedded blocks, and histopathological slides. The LAREB collects resources used in experiments for food and drug safety efficacy evaluation, as well as biological research, and distributes them for use in new research purposes. Additionally, LAREB support the 3Rs (Replacement, Reduction, and Refinement) in research, which promote the substitution of animal models with alternatives, the minimization of animal use and the improvement of animal welfare through refined experiential techniques. In particular, histopathological slides have been digitalized into high-resolution whole slide images (WSIs), which are utilized for toxicological analysis, education, and AI-based applications. To expand access and enhance research utility, the LAREB has digitalized, preserved, and distributed a diverse set of WSIs obtained from various studies. We will introduce five representative WSI categories preserved by LAREB: (1) Korea National Toxicology Program (KNTD) herbal and chemical compound studies, (2) untreated aged rodent studies, (3) inhalation toxicology studies, (4) non-human primate studies, and (5) carcinogenicity studies. These key characteristics and potential research applications are outlined, along with guidance for researchers on how to access and utilize these digital resources.

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Keywords : Laboratory Animal-derived Resources, Whole Slide Images, 3Rs, Laboratory Animal Resource Bank

PS-B-87

A Comparative Toxicity Study of Intravenously Administered Human Adipose-Derived Mesenchymal Stem Cells: Pulmonary Embolism Study in Mice, Rat and Common Marmoset

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Human adipose-derived mesenchymal stem cells (hADMSC) represent a promising therapeutic option, as they are more easily obtained from abundant adipose tissues compared to bone marrow or umbilical cord MSCs. In preclinical safety evaluations of hADMSC using NOG mice, their small blood vessels often lead to fatal pulmonary embolism. To address these challenges, SCID rats and common marmosets were considered as alternative animal models for evaluating the safety of stem cells. In this study, we intravenously infused 1.25 (low), 2.5 (medium), and 5.0 (high) × 10⁸ cells/kg of hADMSC into NOG mice, SCID rats, and common marmosets (control and high-dose groups only) and compared their survival rates. In mice, dose-dependent mortality was observed, with 4 out of 6 (66.7%) animals in high-dose group and 1 out of 6 (16.7%) in medium-dose group dying due to pulmonary vascular obstruction immediately after infusion. *Ex-vivo* imaging of organs from the deceased mice revealed that the majority of the infused cells were trapped in the lungs, and histopathological analysis confirmed that the deaths were caused by pulmonary embolism. Conversely, despite a similar pattern of initial pulmonary cell distribution, both SCID rats and common marmosets tolerated the high dose (5.0 × 10⁸ cells/kg) with no mortality, adverse clinical signs, changes in body weights, or significant changes in hematological and serum chemistry parameters. Therefore, the No-Observed-Adverse-Effect-Level (NOAEL) was determined to be at least 5.0 × 10⁸ cells/kg in these species. These findings indicate that rats and common marmosets, which have larger vascular diameters, are more suitable animal models for injected stem cell toxicity studies. This study underscores the importance of selecting appropriate animal models for preclinical safety evaluations and demonstrates the translational value of using rats and marmosets to develop safe and effective cell-based therapeutics.

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Keywords : hADMSC, Pulmonary embolism, NOG mouse, SCID rat, Common marmoset

PS-C-01

Identification of cast EIJ mouse and NOG mouse lethal to MPOX virus clade IIb

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Monkeypox virus (MPXV) is a member of the Orthopoxvirus genus, which includes variola virus (VARV), the causative agent of smallpox. Beginning in May 2022, mpox outbreak has resulted in more than 91,000 infections around the world. MPXV-ROK-P1-2022 was isolated from the first patient in Republic of Korea and belonged to the B.1.1 lineage of Clade IIb. To confirm whether inbred mice are suitable animal models for MPXV infection, we experimentally infected CAST/EiJ mice and NOG mice with MPXV-ROK-P1-2022. Low passage stock of MPXV-ROK-P1-2022 was obtained from the Korea Disease Control and Prevention Agency. MPXV-ROK-P1-2022 was propagated and titrated on Vero E6 cells prior to use. CAST/EiJ mice and NOG mice were challenged IP with 1x10⁶ plaque-forming units (PFUs) of MPXV-ROK-P1-2022. Blood was collected by cardiac puncture from all animals under isoflurane anesthesia and sacrificed at either day 5 or 7 post-infection. Following euthanasia, lungs, liver, spleen, testes, kidneys and brain were collected, weighed and stored at -80°C until used. After thawing and homogenization, titers of the infectious virus in tissue were determined via plaque assay on Vero E6 cells. To test whether mice exhibit a systemic and circulating infection, we tested blood for the presence of viral genomic material using qPCR. Analysis of organ titers after IP inoculation showed that the virus was present in the lungs, livers, spleens, brains, kidneys, and testes. At 5d after IP infection, the mean titers of MPXV-ROK-P1-2022 were >10⁶ PFU in the lungs and testes. Circulating viral genomes were detected on days 5 and 7 after IP infection of MPXV. In addition, confirmation of circulating infectious virus was obtained by plaque titration of serum from the infected mice. In this study, we experimentally infected CAST/EiJ mice and NOG mice with MPXV-ROK-P1-2022. MPXV intraperitoneal challenge led to systemic infection in CAST/EiJ mice and NOG mice. These results suggest that CAST/EiJ mice and NOG mice are helpful animal models for evaluating vaccine candidates and therapeutics against VARV and MPXV.

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Keywords : Monkeypox virus, Mouse model, Cast/EiJ

PS-C-03

Cyclopeptides from gut commensal *Phocaeicola vulgatus* attenuate muscle atrophy in an an vitro model of pancreatic cancer cachexia

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Cancer cachexia, a multifactorial metabolic syndrome involving severe skeletal muscle wasting, is highly prevalent in pancreatic cancer mouse models and patients. The gut microbiota is a promising source of novel therapeutics, yet the direct effects of its metabolites on cachectic muscle are largely unknown. The gut commensal *Phocaeicola vulgatus* produces bioactive cyclopeptides, like diketopiperazines, known for their stability and anti-inflammatory properties. This study aimed to evaluate if specific cyclopeptides isolated from *P. vulgatus* could directly protect against muscle wasting in a cellular model relevant to pancreatic cancer.

To model the cachectic environment found in laboratory animal models, murine C2C12 myotubes were treated with conditioned media (CM) collected from the Panc02 murine pancreatic cancer cell line. Nine distinct cyclopeptide compounds containing the 2,5-diketopiperazine scaffold were isolated from *P. vulgatus* cultures. The efficacy of these compounds in mitigating skeletal muscle atrophy was evaluated by treating the CM-exposed myotubes with cyclopeptides at concentrations of 10, 20, and 40 µM. Our findings demonstrate that specific *P. vulgatus*-derived cyclopeptides significantly attenuated myotube atrophy. Notably, one compound significantly downregulated the expression of key muscle degradation genes, *Fbxo32* and *Mstn*, which are central to the ubiquitin-proteasome and myostatin pathways, respectively. Another compound partially restored myotube formation and structural integrity, as assessed by myosin heavy chain (MHC) immunofluorescence.

These results suggest that specific cyclopeptides from the gut commensal *P. vulgatus* can directly counteract cachexia-induced muscle atrophy by modulating key catabolic signaling pathways. This study provides a strong mechanistic rationale and identifies promising lead compounds for testing as a novel therapeutic intervention in future *in vivo* animal models of pancreatic cancer cachexia.

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Keywords : Cancer cachexia, *Phocaeicola vulgatus*, Cyclopeptides, C2C12, Panc02

PS-C-02

Phocaeicola vulgatus mediates the anti-cachectic effects of micheliolide in colon cancer by modulating gut microbiota and SCFAs

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Cancer cachexia, a devastating wasting syndrome, severely impacts patient outcomes. The gut microbiota is increasingly recognized as a modulator of cachexia. This study investigated how micheliolide (MIC), a sesquiterpene lactone, and a specific gut commensal, *Phocaeicola vulgatus* (PV), affect colon cancer-induced cachexia in a CT26 mouse model. We assessed MIC's impact on muscle wasting, systemic inflammation (splenic CD4⁺ T cell phenotypes), gut microbiota composition, cecal short-chain fatty acid (SCFA) production, and predicted microbial metabolic functions. Subsequently, the direct effects of oral PV administration were evaluated. MIC treatment robustly improved muscle mass and function and reduced systemic inflammation (decreased activated/exhausted splenic CD4⁺ T cells) in CT26 tumor-bearing mice. MIC also restored gut microbial diversity, increased cecal propionate and butyrate, and notably, selectively increased PV abundance while reducing *Enterococcus faecalis* (EF). Predicted microbial biotin biosynthesis was enhanced by MIC. Crucially, increased PV abundance and SCFA levels correlated with reduced cachexia severity. Validating PV's direct role, oral administration of PV to cachectic mice significantly attenuated muscle wasting, increased cecal butyrate, and beneficially altered the gut microbiota, including a reduction in EF. These findings highlight PV as a key beneficial microbe capable of mitigating cachexia in a laboratory animal model, potentially through SCFA production and modulation of the gut ecosystem. Both MIC-induced PV enrichment and direct PV supplementation represent promising microbiota-targeted strategies for managing cancer cachexia.

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Keywords : Cancer cachexia, Micheliolide, *Phocaeicola vulgatus*, Gut-immune axis, SCFAs

PS-C-04

Synergistic effect of probiotics and omega-3 fatty acids on blood and liver function biomarkers and gut microbiota diversity in high fat diet-induced obesity

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This study investigates the synergistic effects of probiotics and omega-3 fatty acids on blood and liver function biomarkers, as well as gut microbiota diversity, in a high-fat diet (HFD)-induced obesity model in mice. Cardiovascular diseases (CVD), often linked to obesity, are major global health issues driven by abnormal cholesterol and triglyceride levels in the blood. Omega-3 fatty acids, known to lower triglycerides and prevent CVD, and probiotics, which enhance gut microbiota balance and nutrient absorption, were evaluated for their combined effects. The experiment involved five groups of C57BL/6J mice: a normal diet group, an HFD group, an HFD + probiotics group, an HFD + omega-3 group, and an HFD + combined probiotics and omega-3 group. Over six weeks, the combined treatment group showed significant reductions in body weight gain, improved liver function biomarkers, and enhanced anticoagulation markers, such as prothrombin time and activated partial thromboplastin time, compared to the HFD group. Gut microbiota analysis via 16S rRNA sequencing also revealed significant increases in microbial diversity in the combined treatment group. These findings suggest that co-administration of probiotics and omega-3 offers potential therapeutic benefits in reducing obesity-related metabolic dysfunctions by improving lipid metabolism, liver health, and blood circulation.

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Keywords : Probiotics, Obesity, Omega-3, Microbiome, Synergistic effect

PS-C-05

Bacterial reverse mutation assay(Ames Test) of 2-(Aminomethyl)piperidine

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To evaluate the mutagenic potential of 2-(aminomethyl)piperidine, a bacterial reverse mutation assay(Ames test) was conducted using histidine-requiring strains of *Salmonella typhimurium*(TA98, TA100, TA1535, TA1537) and a tryptophan-requiring strain of *Escherichia coli*(WP2 uvrA pKM101). The test substance was applied at concentrations of 0.13, 0.25, 0.5, 1, and 2 µL/plate. As a result, 2-(aminomethyl)piperidine produced positive responses in *S. typhimurium* TA98 and TA1535, as well as in *E. coli* WP2 uvrA pKM101, indicating its potential to induce base pair substitution mutations. Specifically, in the WP2 uvrA pKM101 strain, positive responses were observed at 0.25–1 µL/plate without metabolic activation and at 1–2 µL/plate with metabolic activation. In TA100, positive responses were detected at 0.13–0.5 µL/plate without metabolic activation and at 1 µL/plate with metabolic activation. TA1535 also showed a positive response at 0.5 µL/plate in the presence of metabolic activation. In contrast, no increase in revertant colonies indicative of frameshift mutations was observed in either the presence or absence of metabolic activation in the TA98 and TA1537 strains. Based on these results, 2-(aminomethyl)piperidine is considered capable of inducing base pair substitution mutations under the conditions of this assay.

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Keywords : Ames Test, 2-(Aminomethyl)piperidine

PS-C-06

Prospective model for Monkeypox disease in common marmoset (*Callithrix jacchus*)

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The recent global emergence of the zoonotic monkeypox virus (MPXV), a member of the *Poxviridae* family with a double-stranded DNA genome, has raised significant concerns regarding its pandemic potential since 2022. A total of 173 MPXV-infected patients were reported in Korea from June 2022 to March 2025. MPXV may also have potential as a weaponizable pathogen in addition to its harmful effects on humans. Therefore, an animal model that mimics human infection should be developed to support effective therapeutic and preventive strategies such as therapeutics and vaccines. According to previous research, the common marmoset is suggested as a more suitable model for MPXV infection than macaque monkeys, since macaques require a high dose for inoculation and have a shortened incubation period, which differs from the infection characteristics observed in humans.

In this study, we evaluated the susceptibility of common marmosets (*Callithrix jacchus*) to MPXV infection. Marmosets were intravenously inoculated with varying concentrations of MPXV using an up-and-down method to determine the viral dose that produces a disease course similar to that in humans. Onset of clinical signs (rash, vesicles, hemorrhage, and lymphadenopathy), hematological changes (marked leukocytosis and significant thrombocytopenia), and viremia were observed in a dose-dependent manner. Notably, the virus incubation period was approximately 10 days, closely matching that of humans. Even low-dose inoculation elicited pronounced clinical symptoms, indicating high sensitivity of this model.

These findings suggest that the common marmoset is a highly susceptible and physiologically relevant animal model for MPXV infection, offering a valuable platform for the preclinical evaluation of candidate vaccines and antiviral therapeutics for human use.

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Keywords : Zoonosis, Pathogenesis, Virulence, Non-human primate, Animal biosafety level 3

PS-C-07

Establishment of a Nipah Pseudovirus Animal Model for Vaccine Evaluation Under BSL-2

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Nipah virus (NiV), a highly pathogenic member of the henipavirus genus in the Paramyxoviridae family, is a bat-borne zoonotic pathogen first identified during a severe outbreak in Malaysia in the mid-to-late 1990s. NiV continues to cause recurrent outbreaks in South Asia, particularly in Bangladesh and India, and poses a significant transboundary threat to global health. Due to its high mortality and epidemic potential, NiV has been classified as a high priority pathogen by the Centers for Disease Control and Prevention (CDC), World Health Organization (WHO), and Coalition for Epidemic Preparedness Innovations (CEPI), underscoring the urgent need for effective medical countermeasures, particularly vaccines. However, research on NiV is limited due to the requirement for biosafety level 4 (BSL-4). In this study, we demonstrated a pseudovirus-based animal model using a recombinant vesicular stomatitis virus (rVSV) vector system, which allows for safe experimentation under biosafety level 2 (BSL-2) conditions. Luciferase-tagged Nipah pseudoviruses were inoculated into Balb/c mice, Syrian golden hamsters, and C57BL/6-based human ephrin B2-B3 transgenic (TG) mice. Pseudovirus infection was monitored by measuring luciferase luminescence at 4-, 6-, and 24-hours post-inoculation. The highest luminescence signals were observed at 6 hours in Balb/c mice, 4 hours in Syrian golden hamsters, and 6 hours in human ephrin B2-B3 TG mice. Although further optimization of the time points and infection conditions is required, this pseudovirus-based animal model provides a valuable tool for the evaluation of vaccine efficacy and antiviral strategies against NiV under lower biosafety conditions, which may accelerate NiV vaccine development.

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Keywords : Nipah virus, Biosafety level 4, Human ephrin b2/b3 transgenic mice

PS-C-08

Establishment of a Humanized Mouse Model Expressing MXRA8 Receptor for Chikungunya Virus Vaccine Development

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Chikungunya virus (CHIKV) is a positive-sense single-stranded RNA virus transmitted by mosquitoes and belongs to the Alphavirus genus in the *Togaviridae* family. It causes acute febrile illness accompanied by joint pain in humans. CHIKV was designated as a priority pathogen in the WHO R&D Blueprint in 2017 and has been recognized by CEPI as a key target for vaccine development. Although various vaccine platforms—including virus-like particle (VLP), mRNA, and vector-based approaches—are currently under development, there remains a critical need for appropriate animal models to study viral pathogenesis and evaluate vaccine efficacy. In this study, we established a humanized mouse model expressing the human matrix-remodelling associated 8 (MXRA8) receptor, which is known to mediate CHIKV entry into host cells. MXRA8 is a membrane protein with two Ig-like domains and plays a key role in viral attachment and cell entry. We plan to utilize this model to assess the infectivity and pathogenicity of CHIKV in vivo. The model is expected to serve as a valuable preclinical platform for evaluating the efficacy of CHIKV vaccine candidates and for building a vaccine library in the future.

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Keywords : Chikungunya virus, MXRA8, Vaccine, Humanized mouse model

PS-C-09

Safety assessment of Bojungkigatang: genotoxicity tests of water and ethanol extracts

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Bojungkigatang, a herbal medicine, is widely used to relieve symptoms related to chronic fatigue, gastrointestinal disorders, and immune dysfunction. Despite its long-standing clinical use, comprehensive safety evaluations based on international regulatory standards are limited. To address this gap, we investigated the genotoxic potential of Bojungkigatang's water and ethanol extracts based on a standard battery of genotoxicity tests compliant with OECD test guidelines and conducted under Good Laboratory Practice (GLP) conditions.

The *in vitro* genotoxicity assessment included the bacterial reverse mutation assay (OECD TG 471) and the chromosomal aberration test in CHL cells (OECD TG 473). Both extracts exhibited no mutagenic activity in bacterial strains regardless of metabolic activation and no induction of structural chromosomal aberrations in mammalian cells. No dose-dependent increase in chromosomal aberrations was observed across all tested concentrations. In the *in vivo* micronucleus test using rat bone marrow (OECD TG 474), neither extract induced micronuclei formation, indicating the absence of clastogenic or aneugenic effects under the tested conditions. Bone marrow cytotoxicity, as evaluated by the PCE/(PCE+NCE) ratio, also remained within the historical data range.

These data across three independent genotoxicity assays provide strong evidence that Bojungkigatang water and ethanol extracts do not have a genotoxic risk. This study delivers critical toxicological data that may support the safe use of Bojungkigatang and contribute to its potential consideration in modern evidence-based therapeutic practices and regulatory frameworks.

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Keywords : Bojungkigatang, Herbal medicine, OECD test guidelines, In vitro genotoxicity assessment, In vivo micronucleus test

PS-C-11

Current Status of Mouse Pathogen Standard Cooperation Center in 2025

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Health monitoring of laboratory animals is critical for the prevention and control of infectious diseases, as well as for ensuring the reliability and reproducibility of experimental outcomes in biomedical research. Pathogenic infections in laboratory mice can significantly affect research results, underscoring the need for accurate and timely pathogen surveillance. To establish a robust and standardized diagnostic framework for mouse pathogen monitoring, the Mouse Pathogen Standard Cooperation Center (MPSC) was officially established in September 2021, with its operations based in Korea.

Prior to the establishment of the center, a total of 11 mouse pathogens had been collected, including 7 viruses, 2 bacterial species, and 2 fungal species. Between 2022 and 2024, the center expanded its repository by acquiring an additional 2 viruses and 36 bacterial strains, significantly enhancing the diversity and comprehensiveness of the pathogen collection. In addition, the MPSC has secured tissue samples from mice experimentally infected with several clinically relevant bacterial species, including *Proteus mirabilis*, *Klebsiella oxytoca*, and *Staphylococcus aureus*. Genetic analyses have been performed on bacterial isolates collected from domestic sources. Moreover, microbial resources have been actively distributed to support research and diagnostics. Between 2022 and the first half of 2025, microbial materials were distributed a total of 15 times. These included bacterial strains (6 vials), DNA samples (250 vials, including 135 positive and 115 negative), RNA-positive samples (3 vials), virus samples (2 vials), and serum samples (67 vials, including 47 positive and 20 negative).

Through the systematic collection and standardization of diverse pathogenic microorganisms derived from laboratory animals, the MPSC provides essential foundational resources to support effective mouse health monitoring. These resources are preserved and distributed through national and international pathogen management systems and are made widely available to researchers for pathogen surveillance, diagnostic development, and microbiological research, thereby contributing to the quality and reproducibility of animal-based studies.

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Keywords : Helth monitoring, Mouse pathogen, Resource, MPSC

PS-C-10

IRES-based single-stranded RNA adjuvants encoding co-stimulatory molecules enhances T cell stimulatory effects and led to tumor regression

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Enhancing T-cell responses through co-stimulatory signaling is critical for improving anti-cancer immunity. In this study, we developed single-stranded RNAs (ssRNAs) incorporating the encephalomyocarditis virus (EMCV) internal ribosome entry site (IRES) to drive the expression of co-stimulatory molecules, including OX40L, 4-1BBL, and ICOSL. These ssRNAs were effectively expressed in B16 melanoma and TC-1 tumor cells. *In vitro*, co-culture of mouse splenocytes with tumor cells transfected with these ssRNAs enhanced CD4⁺ and CD8⁺ T-cell proliferation and cytokine secretion, while reducing PD-1 and Foxp3 expression, thereby shifting the T-cell subset composition. *In vivo*, intramuscular injection of ssRNA along with antigen led to the expansion of antigen-specific CD8⁺ T cells and enhanced production of cytotoxic cytokines. Furthermore, intratumoral administration of ssRNAs together with tumor antigens not only suppressed tumor growth but also led to clinical tumor regression in some cases, while promoting immune cell infiltration into the tumor microenvironment, characterized by increased cytotoxic CD8⁺ T cells and reduced regulatory T cells (Tregs) in tumor and secondary lymphoid organs in melanoma-bearing mice. Taken together, the treatment effectively suppressed tumor growth, modulated T-cell subsets, and enhanced immune responses in both *in vitro* and *in vivo* settings. These findings suggest that IRES-based ssRNAs capable of expressing co-stimulatory molecules represent a promising platform for enhancing immunogenicity and advancing the development of effective cancer immunotherapies.

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Keywords : IRES, OX40L, ICOSL, 4-1BBL, T cell responses

PS-C-12

Intestinal Organoids Reveal the Anti-Inflammatory and Gut-Protective Potential of Heat-Killed *Limosilactobacillus reuteri* in Mice and Weaning Piglets

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Organoids, which recapitulate the cellular complexity of native organs, provide a physiologically relevant *in vitro* platform for investigating host responses to microbial stimuli. In this study, we established a screening system using intestinal organoids derived from mice and pigs to evaluate the anti-inflammatory potential of heat-killed (HK) lactic acid bacterial strains. Through comparative screening, *Limosilactobacillus reuteri* was identified as the most effective strain in suppressing inflammatory responses and enhancing epithelial barrier function in both murine and porcine organoid models. The therapeutic efficacy of HK *L. reuteri* was subsequently validated in two *in vivo* systems. In a dextran sulfate sodium (DSS)-induced colitis mouse model, oral administration of HK *L. reuteri* significantly reduced pro-inflammatory cytokine levels, improved survival, and alleviated histopathological damage in the colon. In weaning piglets, dietary supplementation with HK *L. reuteri* led to a marked reduction in diarrhea incidence and a significant improvement in growth performance. These physiological improvements were accompanied by reduced systemic levels of IL-1 β , IL-6, and TNF- α , along with the upregulation of intestinal health markers such as *LGR5*, *MUC2*, and *LYZ1* in organoids derived from treated piglets, indicating enhanced epithelial regeneration and mucosal protection. Furthermore, colonic contents from treated piglets showed elevated concentrations of short-chain fatty acids, particularly butyric and valeric acids, and an increased efficiency in intestinal organoid formation, supporting the strain's role in promoting intestinal homeostasis. In addition, microbiota profiling revealed beneficial modulation of the gut microbial community, characterized by an increase in alpha diversity, suggesting a favorable impact of HK *L. reuteri* on intestinal microbiota balance. Collectively, this study demonstrates that intestinal organoid-based screening is a powerful tool for identifying beneficial bacterial strains and highlights heat-killed *L. reuteri* as a promising paraprobiotic candidate for improving gut health and mitigating inflammation-associated disorders.

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Keywords : Intestinal organoid, Paraprobiotics, Anti-inflammatory activity, Epithelial barrier function

PS-C-13

Strain-Specific Exercise Adaptations and Gut Microbiota Interactions in B6 and 129 Mice

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Genetic background significantly influences how organisms respond to physiological conditions such as exercise, obesity, and interactions with the gut microbiome. These elements are closely connected, and their combined effects play a key role in shaping metabolic outcomes. To investigate genotype-dependent adaptations to exercise, we compared the physiological and metabolic responses of two inbred mouse strains, C57BL/6N (B6) and 129/SvEv (129), following four weeks of voluntary wheel running. Despite exercising only one-third as much as B6 mice, 129 mice exhibited a significantly lower rate of weight gain compared to B6 mice, resulting in no apparent change in body weight with exercise. This strain-dependent difference in running distance observed in SPF mice was not present under germ-free conditions. These observations suggest that differences in the microbiome may reflect compensatory mechanisms that enable 129 mice to maintain energy balance despite limited exercise and impaired glucose metabolism. To investigate this, we systematically analyzed exercise performance, tissue remodeling, and metabolic gene expression profiles in both strains. We further investigated whether alternative energy pathways and gut microbiota composition might contribute to metabolic compensation. Our findings reveal strain-specific adaptations to exercise and underscore the potential role of host-microbiome interactions in regulating metabolic flexibility. To further determine whether the metabolic benefits of exercise can be mediated via the gut microbiota, we plan to perform fecal microbiota transplantation (FMT) into germ-free mice.

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Keywords : Genetic background, Exercise, Microbiome, Energy metabolism

PS-C-15

Design and evaluation of O/CATHAY topotype-specific FMDV vaccine candidates

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Foot-and-mouth disease (FMD) is a highly contagious viral disease affecting cloven-hoofed animals. Among the seven FMD endemic pools worldwide, the Republic of Korea (ROK) belongs to Pool 1 region, where serotype O, such as O/ME-SA/Ind2001, O/SEA/Mya-98, O/CATHAY, and O/ME-SA/PanAsia1 are reported. According to the recent WRLFMD and WOA/FAO reports, the O/CATHAY topotype has emerged in pigs in neighboring countries including Vietnam, Hong Kong, and China. Vaccine matching tests indicate that currently used vaccine strains show poor cross-reactivity with these newly circulating O/CATHAY viruses, highlighting necessity of the topotype-specific vaccine. In this study, we aimed to develop a vaccine strain targeting the O/CATHAY topotype, which has not been reported in the ROK.

Phylogenetic analysis of the VP1 gene sequences from international field isolates showed that viruses originating from Vietnam, Hong Kong, and China formed separate genetic clusters. Two representative strains were selected as vaccine candidates. Using reverse genetics and transfection, recombinant viruses were successfully produced, and antigen particles (146S) were confirmed by transmission electron microscopy (TEM). Guinea pigs were immunized with the inactivated vaccine candidates, and serum samples were collected for immunogenicity analysis. Neutralization testing showed that the vaccine candidate conferred a log(1.2) or higher antibody response against the recently circulating Vietnamese O/CATHAY topotype.

These results suggest that the developed recombinant vaccine candidates are antigenically matched with recent O/CATHAY topotype originated outbreaks and could serve as effective seed strains for emergency vaccination. Further studies, including pig trials and safety assessments, are warranted to validate their field applicability.

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Keywords : Foot-and-mouth disease, CATHAY topotype, Vaccine

PS-C-14

Modeling Antibiotic-Induced Gut Dysbiosis in Common Marmosets Using Azithromycin Monotherapy

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Gut dysbiosis is an imbalance in the intestinal microbiota that involves reduced microbial diversity along with overgrowth of harmful bacteria and decreased beneficial bacteria. Although dysbiosis models using a combination of multiple antibiotics have been extensively studied, there are a few studies demonstrating dysbiosis induced by treatment of single antibiotic in common marmosets (*Callithrix jacchus*). To investigate this, we administered azithromycin (AZT) at 7500 ppm in drinking water for 4 weeks to three male adult common marmosets. Fecal samples were collected before and after treatment to assess microbiome composition using 16S rRNA sequencing. Results showed that alpha diversity significantly decreased after antibiotic administration. This was confirmed by reduced amplicon sequence variant counts and Shannon index values that represent within-community richness and evenness. In beta diversity result, which examines between-individual diversity, also decreased when using weighted UniFrac distances. At the taxonomic level, we examined genera with mean relative abundance $\geq 1\%$ in three marmosets before and after AZT. Analysis revealed that five genera showed decreased relative abundance, whereas ten genera exhibited increased abundance. Although there were inter-individual differences at the individual taxonomic analysis, all three marmosets showed decreased *Bifidobacterium*, and *Clostridium* levels while *Paraprevotella* and *Intestinibacillus* increased. Our results demonstrate that azithromycin monotherapy is sufficient to induce gut dysbiosis in common marmosets. These findings highlight the need for cautious use of AZT and further suggest that single-agent antibiotic treatment can serve as a useful and simplified approach for modeling antibiotic-induced dysbiosis in nonhuman primates.

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Keywords : Antibiotics, Common marmosets (*Callithrix jacchus*), Dysbiosis, Microbiome, 16S rRNA sequencing

PS-C-16

Management of master seed virus candidates for foot-and-mouth disease vaccine production

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Foot-and-mouth disease (FMD) is a highly contagious viral disease that affects cloven-hoofed animals and causes significant economic losses to the livestock industries worldwide. Vaccination remains as a critical strategy for controlling and preventing FMD outbreaks. The selection and management of master seed virus (MSV) candidates play a critical role in ensuring the quality, safety, and efficacy of the FMD vaccines.

In this study, contamination checks and titer tests were routinely performed on MSV candidates developed for the commercialization of FMD vaccines. The MSV candidate strains were stored in ultra-low temperature freezers and liquid nitrogen, respectively, and their titers were periodically evaluated based on the storage duration. Additionally, the viruses were tested for potential contaminants using methods such as bacterial culture and polymerase chain reaction (PCR) to detect the presence of unplanned agents, including bacteria, fungi, and other viruses.

Upon evaluating the MSV candidates stored under cryogenic conditions and in liquid nitrogen, no significant difference in virus titer was observed. However, a gradual decrease in virus titer was noted in some strains. Additionally, contamination checks showed no signs of bacteria, fungi, or other viruses. The most significant observation from this study is the decrease in virus titer in certain strains after prolonged storage. While cryopreservation and storage in liquid nitrogen are widely accepted as effective methods for preserving viral samples, the observed loss of virus potency suggests that these preservation methods may not be universally applicable to all strains over extended periods.

In conclusion, maintaining the efficacy of FMD vaccines requires the regular monitoring of virus potency, and the selection of a more suitable preservative for the FMDV is necessary.

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Keywords : Master seed virus (MSV), FMDV, Management, Vaccine

PS-C-17

Foot-and-Mouth Disease Incidence in Asia from 2020 to 2024 (Pool 1 Region)

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Foot-and-mouth disease (FMD) is a highly contagious viral disease affecting domestic and wild ruminants, characterized by vesicular lesions and causes substantial economic losses. FMD viruses are classified into seven distinct serotypes (O, A, Asia1, C, SAT1, SAT2, and SAT3), each of which circulates independently within seven endemic pools worldwide. Because immunity conferred by FMD vaccines is serotype-specific, ongoing surveillance of FMD outbreaks in neighboring countries (Pool 1 region) is essential for making vaccine policy decisions. In this study, we analyzed FMD outbreak data from the World Organisation for Animal Health (WOAH) and the World Reference Laboratory for Foot-and-Mouth Disease (WRLFMD) from 2020 to 2024. A total of 370 outbreaks were reported in the Pool 1 region during this period. Among these, serotype O was the most frequently identified (59.73%), followed by SAT2 (24.05%) and serotype A (8.65%). In countries within the Pool 1 region, including the Republic of Korea (ROK), serotype O was detected in 95.7% of outbreaks, reflecting a pattern consistent with global FMD prevalence. The predominant strain among serotype O viruses was O/ME-SA/Ind-2001, accounting for 54 cases, followed by A/ASIA/Sea-97 with 3 cases. Most of the O/ME-SA/Ind-2001 viruses were classified into the Ind-2001e sublineage (95%). Notably, the FMD viruses isolated in the ROK in both 2020 and 2024 also in this year, belonged to this lineage (O/ME-SA/Ind-2001e). In the PR China, both O/ME-SA/Ind-2001e and O/CATHAY strains were reported between 2021 and 2023. Overall, diverse serotypes and topotypes were identified in the Pool 1 region. The incursion of novel serotypes into the ROK presents a significant threat to the current FMD vaccination strategy. These findings highlight the importance of continuous surveillance and will contribute to the development of effective domestic isolates-originated vaccines and preventive strategies.

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Keywords: FMDV, Outbreak, WOAH, WRLFMD, ROK

PS-C-18

Immune response and protective efficacy of a bivalent intradermal Foot-and-Mouth Disease Vaccine in pigs

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Foot-and-mouth disease (FMD) is a highly transmissible viral disease of cloven-hoofed animals, associated with significant economic losses. In response to repeated outbreaks in the Republic of Korea, including a serotype O epidemic from 2010 to 2011, a national immunization program was implemented using a bivalent (O + A) oil-adjuvanted vaccine. However, intramuscular (IM) administration in pigs has been linked to local reactogenicity, necessitating evaluation of alternative delivery routes.

This study assessed the immunogenicity and protective efficacy of a bivalent intradermal (ID) FMD vaccine against a field strain isolated in 2019. The challenge virus was the O Anseong strain that occurred in 2019 (O/AS/SKR/2019). FMDV were inoculated with 1×10^6 tissue culture infectious dose (TCID)₅₀/mL (volume, 0.1 mL) into the hoof of donor swine. The donor swine was allowed to coexist with other groups to induce contact infection. Donors were sacrificed 3 d post-challenged (dpc). Humoral responses were quantified via structural protein enzyme-linked immunosorbent assay (SP-ELISA) and virus neutralization test (VNT).

The ID vaccine test group showed positive antibody titer from 6 dpc. The test vaccine group showed VN titer titers of 1:100 from 0 dpc. At the end of the experiment, showed the VN titer was approximately 1:512. The ID-vaccinated group showed complete protection, with no clinical manifestation in any of the 5 animals.

As a result, the ID vaccine showed protection against the FMD virus that recently occurred in South Korea (2019). These results demonstrate that the bivalent ID vaccine elicited effective immune responses and conferred full clinical protection against homologous FMDV challenge. Given its favorable safety profile and protective efficacy, intradermal vaccination represents a viable alternative to conventional IM delivery in FMD control strategies in pigs.

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Keywords: FMDV, Intradermal, Vaccine

PS-C-19

Development of NanoBiT-based virus-like particle assay for rapid screening of neutralizing antibodies against foot-and-mouth disease serotype A

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Foot-and-mouth disease (FMD) is a highly contagious viral disease that affects cloven-hoofed animals, including cattle, pigs, and goats. Among the seven FMDV serotypes, type A is particularly challenging due to its high genetic variability and antigenic diversity. Traditional virus neutralization tests (VNTs), which require live virus and biosafety level 3 (BSL-3) facilities, are labor-intensive, costly, and pose biosafety risks. To overcome these limitations, we developed a NanoBiT-based virus-like particle (VLP) assay for the safe and rapid screening of neutralizing antibodies against FMDV serotype A.

In this study, structural proteins of FMDV serotype A were used to generate VLPs tagged with HiBiT, a small luciferase peptide. These VLPs were introduced into cells expressing LgBiT, the complementary luciferase fragment, and luminescence signals were measured to assess VLP entry.

When mixed with anti-serum prior to cell inoculation, the luminescence signal was significantly reduced, indicating the effective neutralization. The assay showed a strong correlation with the conventional VNT ($R^2 = 0.83$).

Our results demonstrate that the NanoBiT-based VLP assay is a sensitive and reliable method for detecting neutralizing antibodies, which can be performed in BSL-2 laboratories without handling live virus. This platform offers a safer, faster, and more scalable alternative to the conventional methods and holds the potential for broader application in FMDV serological studies and vaccine evaluation.

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Keywords: Foot-and-mouth disease virus, Serotype A, NanoBiT assay, Virus-like particles, Neutralizing antibodies

PS-C-20

Development of an Attenuated FMDV Serotype A Strain via Codon-Pair Deoptimization

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Foot-and-mouth disease virus (FMDV) is a highly contagious pathogen of cloven-hoofed animals, requiring BSL-3 containment due to its high pathogenicity and potential for rapid spread, which significantly limits research accessibility and vaccine development. To enable safer and more accessible FMDV research under BSL-2 conditions, we aimed to develop an attenuated FMDV serotype A strain using a genetic deoptimization strategy, specifically codon-pair deoptimization (CPD).

A recombinant FMDV was generated by replacing the structural protein region (P1) of the O1 Manisa vaccine strain with that of the field isolate serotype A. For attenuation, some of the non-structural protein region was deoptimized using CPD. To differentiate the attenuated strain from wild-type strains, the 3A region was deleted as a DIVA (Differentiating Infected from Vaccinated Animals) marker.

All four recombinant virus constructs were successfully rescued in cell culture. Pathogenicity testing in ICR suckling mice demonstrated complete survival, unequivocally confirming effective attenuation. These findings not only support the feasibility of CPD-based viral engineering for the development of BSL-2 compatible FMDV strains but also provide a crucial platform for safer and more accessible vaccine research and development.

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Keywords: Foot-and-mouth disease virus, Attenuated, Codon-Pair Deoptimization, Vaccine

PS-D-01

Robust human immune cell reconstitution in the humanized N2G Mouse: A NOD-based strain with complete Prkdc and Il2rg gene deletions

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Introduction : Humanized mouse models are indispensable tools for studying human immune responses and evaluating immunotherapeutics. While NSG mice are widely used, their human hematopoietic reconstitution remains suboptimal. In this study, we employed a novel immunodeficient mouse strain (N2G) to evaluate whether busulfan-based preconditioning enhances human immune cell engraftment and function.

Methods : Human CD34⁺ hematopoietic stem cells were isolated from umbilical cord blood and transplanted into N2G or NSG mice preconditioned with busulfan (20, 25, or 30 mg/kg). At 24 weeks post-transplantation, peripheral blood, spleen, and bone marrow were harvested. Human leukocyte reconstitution was assessed by flow cytometry. Spleen tissues were analyzed by H&E staining and immunohistochemistry. T and NK cell functionality was evaluated using CFSE-based proliferation assays and intracellular cytokine staining following stimulation with IL-12, IL-15, and IL-18.

Results : N2G mice preconditioned with 30 mg/kg busulfan (hu-N2G) exhibited significantly enhanced reconstitution of hCD45⁺ leukocytes and hCD3⁺ T cells in peripheral blood and spleen compared to NSG controls. Notably, hu-N2G mice demonstrated robust expansion of functional hCD8⁺ cytotoxic T cells and hCD56⁺ NK cells, characterized by increased proliferation and elevated IFN- γ and TNF- α production following cytokine stimulation. Histological analysis of spleen tissues revealed abundant hCD3⁺ T cells, hCD20⁺ B cells, and hCD68⁺ macrophages.

Conclusions : Preconditioning with 30 mg/kg Busulfan significantly enhances human immune cell engraftment and functionality in N2G humanized mice. This model provides a robust platform for *in vivo* investigations of human immunity and the preclinical evaluation of immunotherapeutic strategies.

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Keywords : Humanized mouse, N2G mouse, Immune cell reconstitution, Immunotherapy evaluation

PS-D-03

Deficiency of peroxisomal bifunctional enzyme HSD17B4 causes dysregulation of primary cilia

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Primary cilia are dynamic sensory organelles orchestrating key signaling pathways, and disruption of primary ciliogenesis is implicated in a spectrum of genetic disorders. The peroxisomal bifunctional enzyme HSD17B4 is pivotal for peroxisomal β -oxidation and acetyl-CoA synthesis, and its deficiency profoundly impairs peroxisomal metabolism. While patients with HSD17B4 deficiency exhibit ciliopathy-like symptoms due to dysfunctional primary cilia, the molecular connection between HSD17B4 and ciliopathy remains poorly understood. Here, we demonstrate that HSD17B4 deficiency impairs primary ciliogenesis and alters cilia-mediated signaling, suggesting a potential link between peroxisomal metabolism and ciliary function. Notably, elevation of acetyl-CoA rescues ciliary defects via HDAC6-mediated ciliogenesis in HSD17B4-deficient cells. Strikingly, acetate administration restores motor function, enhances primary cilia formation, and preserves the Purkinje layer in Hsd17b4-knockout mice. These findings provide insights into the functional link between HSD17B4 and primary cilia, highlighting acetyl-CoA as a potential therapeutic target for HSD17B4 deficiency and ciliopathy.

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Keywords : Peroxisomal bifunctional enzyme, HSD17B4, Primary cilia

PS-D-02

Recovery of effector memory T cells and balance of regulatory T cell subtypes are considered important for immune tolerance in non-human primate combined kidney-bone marrow transplantation

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Introduction : Combined kidney and bone marrow transplantation (CKBMT) can induce immune tolerance through transient mixed chimerism. We investigated the immunological mechanisms of tolerance using a non-human primate (NHP) model.

Methods : CKBMT was performed in five Bw6⁻ NHPs using Bw6⁺ donors. The pre-conditioning regimen included rituximab, cyclophosphamide, fludarabine, rabbit anti-thymocyte globulin (rATG), and thymic irradiation. Sirolimus was given post-transplant as maintenance therapy and then tapered gradually. Based on graft outcomes observed during immunosuppression tapering, recipients were categorized as tolerant (n=3) or non-tolerant (n=2). Peripheral blood was collected pre-transplant, and at 3 and 6 months post-transplant. Effector memory T cell subsets and regulatory T cell (Treg) phenotypes were assessed via flow cytometry.

Results : All CKBMT recipients developed transient chimerism. Tolerant groups maintained stable graft function after complete withdrawal of immunosuppression, accompanied by robust reconstitution of CD4⁺ and CD8⁺ effector memory T cells and sustained suppressive Tregs. In contrast, non-tolerant groups experienced early rejection, impaired memory T cell recovery, and an increased proportion of non-suppressive Tregs (FoxP3lowCD45RA⁻).

Conclusion : Successful tolerance after CKBMT correlates with efficient memory T cell reconstitution and sustained presence of suppressive Tregs. Conversely, impaired memory T cell recovery and expansion of non-suppressive Tregs may contribute to graft rejection. These findings underscore the importance of Treg phenotype and memory T cell homeostasis in maintaining transplant tolerance in the NHP CKBMT model.

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Keywords : Combined Kidney and Bone Marrow Transplantation (CKBMT), Non-human primate (NHP), Effector memory T cells, Non-suppressive Treg cells

PS-D-04

TIM-3 deficiency aggravates liver fibrosis by stimulating pro-inflammatory macrophages

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Liver fibrosis is a globally prevalent disease, yet effective treatments remain elusive. Recent studies have highlighted the importance of immune checkpoint molecules in regulating both innate and adaptive immune responses. Among them, T-cell immunoglobulin and mucin-domain containing-3 (TIM-3) is known to modulate the function of macrophages and has been implicated in the progression of chronic liver diseases. In this study, we examined the spatiotemporal dynamics of TIM-3 expression in macrophages during liver fibrosis. Single-cell RNA sequencing (scRNA-seq) analysis revealed that Galectin-9 (Gal-9) was broadly expressed across multiple cell types, while TIM-3 expression was predominantly confined to Ly6Clow macrophages. Flow cytometry analysis in healthy mice further confirmed that TIM-3 is widely expressed in macrophages from blood and bone marrow. Notably, in liver tissue, TIM-3 expression was significantly upregulated in Ly6Clow macrophages but downregulated in Ly6Chigh subsets. This pattern was preserved under fibrotic conditions. TIM-3 expression remained enriched in Ly6Clow macrophages, which were frequently located in proximity to activated hepatic stellate cells. Functionally, liver fibrosis was more severe in TIM-3 knockout (KO) mice compared to wild-type (WT) controls, despite similar levels of carbon tetrachloride (CCl₄)-induced liver injury. Additionally, fibrogenic genes such as *Col1a1* and *Tnf* were markedly upregulated in TIM-3 KO livers. *In vitro* experiments supported these findings. Gal-9 treatment suppressed lipopolysaccharide-induced activation in WT bone marrow-derived macrophages (BMDMs), but not in TIM-3 KO BMDMs. Collectively, our data demonstrate that TIM-3 is specifically expressed in Ly6Clow macrophages, where its activation via the Gal-9/TIM-3 axis plays an inhibitory role in liver fibrosis. These results suggest a novel therapeutic target for liver fibrosis by modulating Ly6Clow macrophages.

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Keywords : Liver fibrosis, Macrophage, TIM-3, Galectin-9

PS-D-05

Galectin-4 potentiates CD8⁺ T cell immunity by enhancing MHC-I expression on dendritic cells: Therapeutic implications for cancer

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Galectin-4 (Gal-4), a member of the β -galactoside-binding galectin family, plays a role in various physiological processes, including tumor progression and intestinal disorders. Gal-4 exerts their role in various physiologic processes especially in the gastrointestinal tract. However, its role in immune cell function remains poorly understood. In this study, we found Gal-4 plays a critical role in regulation of effective CD8⁺ T cell responses against tumors. In syngeneic mouse cancer models, Gal-4-deficient mice exhibited significantly increased tumor growth and reduced survival rates. This phenotype was not due to intrinsic defects in CD8⁺ T cells but instead linked to decreased surface expression of antigen-MHC-I complexes on dendritic cells. The reduction in antigen presentation caused by Gal-4 deficiency critically impaired the activation and function of CD8⁺ T cells. Conversely, exogenous administration of Gal-4 upregulated MHC class I expression on dendritic cells, thereby enhancing CD8⁺ T cell responses. Combined with cancer vaccines or PD-1 blockade therapy, Gal-4 treatment effectively suppressed tumor growth and improved survival rates. Collectively, our findings highlight a central role for Gal-4 in regulating MHC-I-mediated antigen presentation by dendritic cells and underscore its potential as a therapeutic target for the development of next-generation cancer immunotherapies.

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Keywords: Galectin-4, Dendritic cells, CD8⁺ T cells, Antitumor immunity

PS-D-06

Glucose-6-Phosphate Dehydrogenase as Metabolic Gatekeeper of Brown Adipocyte Identity and Thermogenesis

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Brown adipose tissue (BAT) exhibits remarkable glucose-metabolic capacity in response to ambient temperature, notably increasing glucose utilizations in cold conditions. However, whether glucose utilizations directly contribute to control of brown adipogenesis during adaptive thermogenesis has remained elusive. Here, we show that Glucose-6-phosphate dehydrogenase (G6PD), the rate limiting enzyme of the pentose phosphate pathway, serves as a key regulator of brown adipogenic commitment. Mice with BAT-specific deletion of G6PD were not able to maintain the body temperature under acute-cold stress. While the expression levels of thermogenic and brown adipogenic markers remain unchanged, ectopic expression of myogenic genes is observed in the BAT. This is associated with the emergence of 'zones of myogenics' within BAT of depots, characterized by robustly elevated expression of myogenic genes. The phenotype is recapitulated in vitro upon G6PD loss, implying an altered commitment to the brown adipocyte lineage. Stable isotope tracing reveals that G6PD loss significantly alters glucose flux into nucleotide and hexose metabolism pathways, suggesting potential mechanisms underlying brown adipocyte lineage plasticity. Thus, the formation of 'zones of myogenics', a spatially restricted disruption in lineage specification is, possibly driven by local alterations in metabolic cues. These findings underscore that glucose metabolism is not solely involved in thermogenic bioenergetics, but also plays an active role in shaping cell fate decisions during brown adipogenesis.

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Keywords: Brown Adipose Tissue, Cell Differentiation, Metabolic Physiology, Glucose-6-Phosphate Dehydrogenase, Brown Adipocyte Progenitor Cells

PS-D-07

Prostaglandin Signals from Brown Adipose Tissue Accelerate Kidney Cystogenesis in Mettl14 BAT cKO Mice

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N6-methyladenosine (m6A) RNA modification, catalyzed by METTL14, regulates mRNA stability and translation across tissues. To explore its role in systemic signaling, we generated a brown adipose tissue (BAT)-specific Mettl14 conditional knockout (cKO) mouse model. Surprisingly, these mice developed spontaneous renal cysts, despite Mettl14 deletion being restricted to BAT. Transcriptomic and KEGG pathway analysis of BAT revealed upregulation of prostaglandin biosynthesis and arachidonic acid metabolism, including increased expression of Ptgs and PGF synthases, suggesting elevated production of PGF2 α and related lipid mediators. Although Ptgs2 mRNA was modestly reduced, prior work demonstrates that PTGES2 protein levels increase in Mettl14-deficient BAT due to reduced m6A-mediated mRNA decay (Xiao et al., 2024), supporting a functional rise in PGE2 synthesis.

Importantly, PGE2 is known to promote cystogenesis in the kidney by activating EP2 and EP4 receptors on renal epithelial cells. EP2 mediates cAMP signaling and protects cystic cells from apoptosis (Elberg et al., 2007), while EP4 promotes both proliferation and chloride secretion, contributing to cyst expansion (Rohatgi et al., 2012). These findings suggest that prostaglandins released from BAT in Mettl14 cKO mice may circulate and stimulate renal EP2/EP4 signaling, driving cystogenesis. This work reveals a novel epitranscriptomically regulated BAT-to-kidney communication axis in the pathogenesis of renal cystic disease.

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Keywords: Mettl14, Poly cystic kidney disease, BAT-Kidney axis

PS-D-08

Contraceptive of the male common marmoset (*Callithrix jacchus*) using vasectomy

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The recipient female of embryo transfer (ET) should be housed with a vasectomized male, as vasectomy may serve as an effective contraceptive for male marmosets due to reduced recovery times, lower complication rates, and minimal impact on the animals behavior. Confirmation of azoospermia via post-vasectomy semen analysis (PVSA) is required, and computer-assisted semen analysis (CASA) is an appropriate method for PVSA.

Two adult male marmosets underwent vasectomy in preparation for co-housing with ET recipient females. The procedure durations were 42 and 27 minutes, and neither animal experienced complications or exhibited abnormal behavior postoperatively. PVSA was performed using CASA, confirming azoospermia from 5 weeks post-surgery. Both vasectomized males were placed with recipient females at 6 weeks after vasectomy.

Vasectomy followed by PVSA appears to be a safe and reliable contraceptive method for male marmosets in ET research, and may also have applications in captive breeding management within zoological settings.

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Keywords: Assisted reproductive technology, Non-human primate, Ductus deferens, Vasal disruption, Monkey

PS-D-09

Development of a Human CD3E Transgenic Mouse Model for Preclinical Evaluation of CD3 – targeting bispecific antibodies

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Species specificity of therapeutic antibodies remains a significant barrier in the preclinical evaluation of T cell-engaging immunotherapies. To overcome this challenge, we generated a human CD3E (hCD3E) transgenic mouse model that expresses the human CD3 epsilon chain, thereby enabling functional assessment of anti-human CD3 antibodies in an immunocompetent environment.

In hCD3E tg mice, human CD3 expression was confirmed in the absence of mouse CD3 expression. Flow cytometric analysis showed normal T cell development, with CD4⁺ and CD8⁺ T cell populations similar to those observed in wild-type (WT) mice. Upon stimulation with the anti-human CD3 monoclonal antibody OKT3, hCD3E splenocytes exhibited robust T-cell activation and proliferation, whereas WT splenocytes showed no such response.

Binding assays demonstrated that CD3-targeting bispecific antibodies bound effectively to both PBMCs and splenocytes from hCD3E mice, with binding profiles comparable to those of conventional anti-CD3 monoclonal antibodies.

The therapeutic efficacy of anti-human CD3 antibody was demonstrated in vivo, where treatment suppressed tumor growth in a CT26 model. These results validate the hCD3E tg mouse as a translationally relevant platform for the preclinical evaluation of efficacy of CD3-targeting immunotherapies

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Keywords : CD3, Genetic Engineering, Humanized mouse, Immunotherapies, Therapeutic antibodies

PS-D-10

Establishment and Comparative Analysis of Diabetic Sarcopenia Models in Young and Aged mice

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The increasing elderly population has emphasized the importance of studying age-related diseases, among which diabetic sarcopenia—a combination of diabetes and muscle loss—is emerging as a significant health concern. However, research on the pathophysiology of diabetic sarcopenia remains scarce, and existing animal models focus solely on either diabetes or sarcopenia individually. This study aimed to establish and compare diabetic sarcopenia models in young and aged mice to facilitate future research and therapeutic evaluations. Eight-week-old (young) and over twelve-month-old (aged) male C57/BL6 mice were assigned to four groups (n = 6 / group): control, diabetes, sarcopenia, and diabetic sarcopenia. Diabetes was induced by feeding a high-fat diet (HFD) for eight weeks, and muscle loss was induced by dexamethasone injection for seven consecutive days. In the aged diabetic sarcopenia (ODS) group, body weight gain was limited compared to the diabetes-only group but higher than the sarcopenia-only group, indicating concurrent metabolic disruption and muscle loss. Grip strength and limb endurance significantly declined in both sarcopenia and diabetic sarcopenia groups, with the ODS group exhibiting the most severe impairment. Muscle loss analysis confirmed the greatest reduction in the ODS group. Organ index analysis showed increased liver and fat mass under diabetic conditions, highlighting systemic metabolic alterations. Blood glucose tests revealed exacerbated hyperglycemia and impaired glucose tolerance in diabetic sarcopenia models, demonstrating that sarcopenia worsens metabolic dysfunction. These findings suggest that aging, diabetes, and sarcopenia collectively accelerate muscle atrophy and metabolic dysfunction, establishing a reliable model for understanding disease mechanisms and evaluating potential therapies.

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Keywords : Diabetic Sarcopenia, Insulin resistance, Muscle loss, Animal Model

PS-D-11

Neuroprotective Effects of Astragalus mongholicus and Scutellaria baicalensis Extracts in Ischemic Stroke: MRI-Based Infarct Reduction and Modulation of Inflammation and Pyroptosis

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Ischemic stroke is a leading cause of mortality and disability, largely due to irreversible neuronal damage triggered by impaired cerebral blood flow, followed by inflammation and pyroptosis. However, the current therapeutic option—tissue plasminogen activator (tPA)—is limited by a narrow time window and safety concerns. In this study, we evaluated the therapeutic potential of a combined herbal extract of *Astragalus mongholicus* (AM) and *Scutellaria baicalensis* (SB), two medicinal plants commonly used in East Asian traditional medicine. We administered AM-SB (200 mg/kg, orally) to mice subjected to transient middle cerebral artery occlusion (tMCAO), and assessed its neuroprotective effects using MRI and MRS analysis on post-stroke day 3. Structural imaging showed a significant reduction in infarct volume in the AM-SB-treated group, with a ~50% decrease compared to controls. In vivo magnetic resonance spectroscopy revealed improved metabolic profiles, including increased levels of inositol, taurine, and glutamate. To investigate the underlying mechanisms, we conducted Western blot analyses of inflammation- and pyroptosis-related markers. AM-SB treatment significantly reduced the expression of TNF- α , IL-1 β , NLRP3, GSDMD, ASC, and cleaved caspase-1 in the ischemic penumbra. Furthermore, behavioral assessments demonstrated functional recovery, including improved motor coordination (Rotarod test) and memory retention (Passive Avoidance test). These results suggest that AM-SB exerts multi-modal neuroprotective effects against ischemic stroke by reducing infarct volume, restoring brain metabolism, and suppressing inflammation and pyroptosis. The findings support the potential of AM-SB as a promising complementary therapeutic strategy for ischemic stroke management.

*Corresponding author : Hyun-Man Baek, Keun-A Chang

Keywords : Ischemic Stroke, Neuroprotection, Magnetic Resonance Spectroscopy, Pyroptosis Inhibition, Herbal Extract Treatment

PS-D-12

Establishment of Osteoporotic Animal Model and Evaluation of Super Active Calcium Supplementation

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Osteoporosis is a significant public health concern, particularly among postmenopausal women, due to the sharp decline in estrogen levels that leads to a loss in bone density and an increased risk of fractures. Since current pharmacological treatments may involve adverse side effects, there is an urgent need to explore alternative, safe, and effective preventive strategies. In this study, we investigated the effects of super active calcium (SAC) supplementation on osteoporosis using an established ovariectomized (OVX) rat model that mimics postmenopausal conditions. Six-week-old female rats underwent bilateral ovariectomy to induce osteoporosis and were allowed a one-week recovery period. Following this, the animals were divided into groups and given low, medium, or high doses of ionized calcium orally for eight weeks. The OVX group showed significant physiological changes, including increased body weight, reduced uterus weight, elevated bone resorption markers (CTX1, NTX1), and deterioration in bone microarchitecture, as indicated by reduced BMD, BV/TV, Tb.Th, and other structural indicators. SAC supplementation effectively reversed these changes in a dose-dependent manner. Bone formation marker Gla-OC levels increased, while bone resorption markers decreased. Additionally, improvements were seen in lipid metabolism markers (TC, TG, HDL, LDL), liver enzymes (ALT, AST), and normalization of serum calcium levels. These findings demonstrate that ionized calcium supplementation can significantly ameliorate bone loss and metabolic disturbances associated with estrogen deficiency, supporting its potential use as a safe preventive intervention for postmenopausal osteoporosis.

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Keywords : Postmenopausal Osteoporosis, Ionized Calcium Supplementation, Ovariectomized Rat Model, Bone Microarchitecture, Bone Turnover Markers

PS-D-13

Development of obesity mouse model and evaluation of fig vinegar supplementation

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Obesity, a primary factor in numerous lifestyle-related diseases, demands safe and effective preventive approaches suitable for everyday life. Vinegar is widely recognized for its preventive properties, and combining it with figs led to the development of fig vinegar. This study aimed to investigate the anti-obesity effects and lipid accumulation inhibitory potential of fig vinegar in an animal model. Six-week-old male C57BL/6 mice were fed a high-fat diet (HFD) for 8 weeks to induce obesity. Fig vinegar was administered at doses of 0.5 ml/kg, 1.0 ml/kg, and 2.0 ml/kg throughout the experimental period. Metformin served as a positive control. Fasting blood glucose was measured at weeks 0, 2, 4, 6, and 8. At week 8, glucose tolerance was assessed using OGTT and IPGTT. Body weight in fig vinegar groups tended to decrease compared to the obese control. High-dose fig vinegar significantly reduced fasting glucose at weeks 6 and 8, comparable to metformin effects in glucose tolerance tests. Liver, kidney, and epididymal fat weights decreased significantly in fig vinegar-treated groups. Histological analyses (H&E staining) revealed reduced hepatic fat droplet formation and renal glomerular hypertrophy. Epididymal fat tissue exhibited increased lipid droplet counts and reduced droplet size in the high-dose group. Serum cholesterol (TC) and triglycerides (TG) were also notably reduced. Additionally, fig vinegar significantly decreased lipid accumulation and droplet size in 3T3-L1 cells in vitro. These results strongly suggest that fig vinegar effectively inhibits lipid accumulation and exhibits potent anti-obesity effects, positioning it as a promising dietary intervention for obesity prevention.

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Keywords : Fig vinegar, Anti-obesity, Lipid accumulation, High-fat diet (HFD), Antioxidant

PS-D-15

Noncanonical β -adrenergic receptor-G12/G13 pathway regulates lipid utilization and thermogenesis in brown adipocyte

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The molecular cascade underlying thermogenic activation in brown adipocyte is predominantly described through Gs-dependent canonical β -adrenergic receptor (b-AR) signaling, a GPCR pathway stimulated by norepinephrine; however, this model does not fully capture the physiological complexities of thermogenesis. GPCRs are capable of engaging multiple G proteins, hinting the existence of additional b-AR pathways that modulate brown adipocyte function. Here, we identify a distinct b-AR-G12/G13 pathway playing a critical role in regulating brown adipocyte's fuel utilization and thermogenesis. Transcriptomic analysis following pharmacological PKA inhibition reveals gene clusters regulated independently of Gs signaling. A GPCR transducer screen demonstrates that both human and murine b-ARs additionally couple to non-Gs proteins including G12/G13. CRISPR/Cas9-mediated deletion of G12/G13 (double knockout; DKO) in brown adipocytes results in markedly enlarged and unevenly distributed lipid droplets, accompanied by elevated expression of genes associated with lipid metabolism and thermogenesis. Time-course lipidomic profiling and metabolic phenotyping assays indicate enhanced oxidative lipid utilization flux in G12/G13 DKO brown adipocytes. These findings reveal a previously unrecognized role for G12/G13 as a downstream of b-adrenergic receptor signaling, offering new insights for b-adrenergic control of thermogenesis and potential targets for metabolic intervention.

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Keywords : Brown Adipose Tissue, Thermogenesis

PS-D-14

Evaluation of antidiuretic effects and single-dose safety of cephalotocin

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Cephalotocin is a neuropeptide belonging to the oxytocin/vasopressin superfamily, naturally found in marine molluscs of the family Octopoda. Our previous study showed that cephalotocin selectively activates human vasopressin receptor subtypes V1b and V2, and exerts significant antidiuretic effects when administered intravenously in rats. In the present study, we evaluated the detailed efficacy and safety of cephalotocin to assess its potential as a functional food ingredient or pharmaceutical material. Cephalotocin was administered intraperitoneally, subcutaneously, or orally to normal C57BL/6 mice, and urine osmolality was measured. All routes of administration showed significant antidiuretic effects. In a polyuria mouse model, cephalotocin produced a markedly enhanced urine-concentrating effect. In addition, the duration of efficacy following subcutaneous administration was evaluated according to dosage, revealing that the antidiuretic effect could be sustained for more than 6 hours in a dose-dependent manner. Next, we assessed the safety of cephalotocin through an acute oral toxicity study and a single-dose subcutaneous injection study. No mortality or serious adverse effects were observed at the maximum dose of 2,000 mg/kg, indicating a high safety profile. These findings suggest that cephalotocin has strong potential as a functional food or therapeutic candidate for the management of urinary disorders such as enuresis, nocturnal enuresis, and diabetes insipidus.

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Keywords : Cephalotocin, Peptide, Antidiuretic, Nocturia, Vasopressin

PS-D-16

A host genetic locus on chromosome 4 modulates Akkermansia abundance and microbial squalene metabolism in atherosclerosis

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Atherosclerosis, a leading cause of cardiovascular disease, is influenced by complex interactions between host genetics and the gut microbiome. However, the specific genes and microbial pathways that mediate this interplay remain largely unknown. Here, we leverage a large-scale systems genetics approach using a hyperlipidemic Diversity Outbred mouse population to comprehensively map the interplay between host genetics, the gut microbiome, and atherosclerosis susceptibility. Integrating high-resolution phenotyping of cardiometabolic traits, 16S rRNA sequencing of the gut microbiome, and quantitative trait locus (QTL) mapping, we identify novel genetic loci and microbial taxa that influence atherosclerosis development. We found that host genetics has a profound, sex-specific influence on gut microbiota composition. We identified a significant QTL hotspot on chromosome 4 that regulates the abundance of *Akkermansia*, a genus often associated with metabolic health. This same locus was associated with the predicted abundance of microbial metabolic pathways, including those involved in squalene biosynthesis. While *Akkermansia* abundance was not correlated with disease in the full cohort due to its low prevalence (absent in ~75% of mice), a stratified analysis of mice where it was present ($n = 118$) revealed a significant negative correlation with both atherosclerotic lesion area and plasma cholesterol. Furthermore, the predicted abundance of microbial enzymes involved in squalene metabolism that co-localized with the *Akkermansia* QTL also showed a negative correlation with these traits. Our findings demonstrate the power of systems genetics to dissect complex gene-microbiome interactions and provide a mechanistic framework for understanding the pathogenesis of atherosclerosis. This work illuminates a novel link between a host genetic locus, *Akkermansia*, and microbial squalene metabolism in atherosclerosis, highlighting this axis as a potential therapeutic target to mitigate cardiovascular disease risk.

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Keywords : Atherosclerosis, Gut microbiome, Systems genetics, Akkermansia, Squalene metabolism

PS-D-17

Synergistic Neuroprotective Effects of Treadmill Exercise and Vitamin D on Cognition, Inflammation, and Mitochondrial Function in Healthy Mice

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Purpose : This study aimed to investigate whether the combined application of treadmill exercise and vitamin D injections exerts synergistic neuroprotective effects in non-pathological mice by enhancing cognitive function, regulating immune responses, maintaining blood-brain barrier (BBB) integrity, and improving mitochondrial efficiency.

Methods : Forty male C57BL/6 mice were randomly assigned to control (Con), exercise (EX), vitamin D (VD), or combined treatment groups (EXVD). After treatments for 20 weeks, behavioral, molecular, and cellular analyses were conducted to assess cognitive function, immune responses, BBB integrity, oxidative stress, and mitochondrial efficiency.

Results : The EXVD group showed superior spatial learning and memory compared to other groups. Vitamin D significantly increased plasma IL-10 and tight junction proteins, while exercise alone increased hippocampal TNF- α . Combined treatment reduced astrocyte activation, maintained BBB integrity, and improved mitochondrial respiratory efficiency without increasing oxidative stress. Antioxidant enzyme expression was reduced in VD and EXVD groups, suggesting improved redox balance. **Conclusions :** Treadmill exercise and vitamin D injections exert synergistic neuroprotective effects in non-pathologic mice by enhancing cognition, reducing neuroinflammation, and improving BBB and mitochondrial function. These findings highlight the potential of non-pharmacological strategies to support brain health even in the absence of disease.

본 연구성과물은 2024년도 정부(교육부)의 재원으로 한국연구재단의 지원을 받아 수행된 기초연구사업임(No.RS-2024-00463501)

*Corresponding author : Jeonghyeon Kim

Keywords : Exercise, Vitamin D, Neuroinflammation, Oxidative Stress, Mitochondria

PS-D-18

Exercise Attenuates Neuropathology and Enhances Cognitive Function in 3xTg-AD Mice via Anti-Inflammatory and Myelin-Repair Mechanisms

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Background : Alzheimer's disease (AD), a leading cause of dementia, is characterized by amyloid-beta (A β) plaques, neurofibrillary tangles (NFTs), and neuroinflammation, which collectively drive cognitive decline and neuronal damage. Emerging evidence highlights the potential of exercise to counteract AD pathology by reducing inflammation, enhancing neuroprotection, and facilitating myelin repair.

Purpose : This study investigated the impact of exercise on inflammation, glial cell phenotypes, oligodendrocyte maturation, and cognitive function in 3xTg-AD mice, hypothesizing that exercise would attenuate these pathological features.

Methods : Forty mice, comprising 20 wild-type (WT) and 20 3xTg-AD transgenic mice, were randomly divided into wild-type control (WT), wild-type exercise (WTEX), AD control (AD), and AD exercise (ADEX) groups. At 4 months of age, the mice underwent 20 weeks of treadmill exercise training. Cognitive function was assessed using the Morris water maze (MMW), followed by biochemical analyses of plasma and brain tissues to examine cellular and molecular changes.

Results : We observed that 20 weeks of treadmill running attenuated AD pathology and enhanced cognitive function in 3xTg-AD mice. Specifically, treadmill running balanced systemic and hippocampal cytokines and promoted a shift in glial cell phenotypes toward anti-inflammatory and neuroprotective states. Enhanced mitochondrial antioxidative capacity and quality control mechanisms reduced oxidative stress. Treadmill running further facilitated intercellular mitochondrial transfer, a process critical for neuronal repair and survival under pathological conditions. In addition, treadmill running promoted oligodendrocyte maturation and facilitated remyelination of damaged neurons, an essential process for restoring neural connectivity and function.

Conclusion : Collectively, this study demonstrates that exercise induces profound adaptations at the cellular and molecular levels, including inflammation reduction, glial cell modulation, mitochondrial migration, and myelin repair, highlighting exercise as a potent non-pharmacological therapeutic strategy for mitigating AD-related cognitive decline and neuropathology.

본 연구성과물은 2024년도 정부(교육부)의 재원으로 한국연구재단의 지원을 받아 수행된 기초연구사업임(No.RS-2024-00463501)

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Keywords : Alzheimer's disease, Exercise, Neuroinflammation, Glial Cell, Mitochondrial Transfer

PS-D-19

Simple gene knockout by single gene-directed multiplex CRISPR-Cpf1

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CRISPR-Cpf1 (Cas12a) is a unique genome editing system with a specialized RNase activity, enabling autonomous processing of precursor crRNAs into mature forms. This property allows multiplex gene editing from a single transcript without the need for an independent precursor crRNA-processing enzymes and has been successfully applied for targeting multiple genes simultaneously. In this study, we designed a Cas12a-based crRNA array targeting four distinct sites within a single gene. After selecting four highly active crRNAs, we constructed a tandem array—termed 4CR—in which each downstream crRNA was designed to target potential in-frame mutations Without disturbing the upstream premature stop codons. This sequential targeting strategy increases the likelihood of generating frameshift mutations, thereby ensuring complete gene disruption. Lentiviral vectors carrying the 4CR array and Cas12a were used to infect both cancer and normal cell lines, followed by antibiotic selection to establish stable cell lines. Gene editing efficiency was assessed using the T7 endonuclease I (T7E1) assay and deep sequencing of genomic DNA. In addition, gene knockout was validated at the protein level by Western blotting and immunostaining. Collectively, our data demonstrate that the CRISPR-Cas12a-4CR system enables highly efficient gene knockout without the need for clonal selection. This method is effective in both immortalized cancer cells and primary cells with limited proliferative capacity and represents a versatile tool for functional genomics and high-throughput genetic screening.

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Keywords : Gene Knockout, CRISPR, Cpf1, Multiple crRNAs, Polyclonal isogenic cell

PS-D-20

Peptide-based modulation of copper homeostasis: therapeutic insights into Wilson's disease and metal-induced organ damage

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Copper (Cu) is an essential trace element involved in numerous physiological processes, but excessive accumulation can cause cytotoxicity and organ failure. One of the most well-known genetic disorders related to copper dysregulation is Wilson's disease, characterized by mutations in the ATP7B gene that impair hepatic copper excretion, leading to copper buildup in the liver, brain, and other tissues. Current treatments involve chelating agents, which are limited by toxicity and lack of tissue specificity. In this study, we identified a novel copper-binding peptide from the genome of Octopus minor, a marine mollusk with highly specialized copper-based metabolism. Structural predictions using AlphaFold and in vitro binding assays confirmed the peptide's strong affinity for copper ions. To assess its biological activity, we used Danio rerio (zebrafish) as a vertebrate model. Zebrafish exposed to toxic copper levels, with or without peptide treatment, were monitored for survival, growth, and tissue-specific effects. Peptide-treated groups showed enhanced survival, reduced tissue damage, and significant protective effects across multiple organ systems, including neuroprotective effects and dermal, cardiac, and hepatic recovery responses. RNA-seq analysis revealed the peptide modulates pathways involved in oxidative stress response, metal ion transport, and cellular repair. Furthermore, stability and toxicity profiling supported the peptide's potential for therapeutic use. These findings underscore the promise of marine-derived peptides as safer, more targeted alternatives to conventional chelation therapy for Wilson's disease and other heavy metal toxicity-related conditions. This research was supported by a grant (RS-2025-02313461) from the Ministry of Oceans and Fisheries.

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Keywords : Wilson's disease, Copper-binding peptide, Metal detoxification, Marine biopharmaceuticals, Zebrafish

PS-D-21

Macrophage-driven immune remodeling in early tumorigenesis initiates resistance to androgen deprivation therapy (ADT) in prostate cancer

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Androgen deprivation therapy (ADT), a hormone therapy for prostate cancer, is initially effective but gradually loses its effectiveness over time. Combination treatment strategies with ADT for castration-resistant prostate cancer (CRPC) patients have been extensively studied. However, why ADT resistance develops in prostate cancer, and how ADT resistance can be prevented before it emerges, remain unknown. Here we report that tumor-associated macrophages (TAMs) begin shaping an ADT-resistant tumor microenvironment (TME) during the early tumor stage. We used the TRAMP (Transgenic Adenocarcinoma of the Mouse Prostate) model, in which prostate cancer arises spontaneously, that closely resembles the early-stage human prostate cancer TME. Mice were treated with Enzalutamide (an androgen receptor antagonist) and PLX-3397 (a CSF1R inhibitor that depletes TAMs). Macrophage depletion and vehicle treatment were performed before tumor formation, and the TME was analyzed at the early neoplastic stage (12 weeks). This analysis revealed that TAM accumulation and M2-like polarization contribute to the establishment of an immunosuppressive niche with limited T cell infiltration during early prostate tumorigenesis. We further show that combination therapy with ADT and macrophage depletion suppresses tumor growth and increases the cytotoxic T cell ratio, at the progressive tumor stage (16 weeks). Our study reveals that macrophages promote ADT resistance by shaping an immunosuppressive TME, and suggests that initiating macrophage-targeted immunotherapy in parallel with ADT can prevent progression to CRPC. These findings provide insight into how early immune changes affect prostate cancer and offer direction for overcoming ADT resistance.

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Keywords : Tumor-associated macrophages, Tumor microenvironment, Prostate cancer, Androgen deprivation therapy, Immunotherapy

PS-D-23

KBN2202 Suppresses Adipose Tissue Expansion and Enhances GLP-1 Secretion in High-Fat Diet-Induced Obese Mice

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Obesity is a chronic and multifactorial metabolic disorder characterized by excessive fat accumulation, hormonal imbalance, and low-grade chronic inflammation. These pathological features contribute to increased risk of comorbidities such as type 2 diabetes, cardiovascular disease, and non-alcoholic fatty liver disease. Although GLP-1 receptor agonists are widely used as effective anti-obesity agents, their therapeutic potential is often limited by gastrointestinal side effects, loss of muscle mass, high treatment costs, and reduced efficacy in specific populations, including patients with type 2 diabetes. The present study aimed to evaluate the anti-obesity potential of KBN2202, a novel salicylic acid derivative, using a high-fat diet (HFD)-induced obesity mouse model. C57BL/6J mice were fed a high-fat diet to induce obesity and then orally administered KBN2202 for 8 weeks. Physiological parameters, food intake, body weight, adipose tissue mass, muscle mass, serum metabolic hormones, inflammatory cytokines, thermogenic gene expression, and anxiety-like behavior were measured using ELISA, immunohistochemistry, and behavioral tests including the open field test. KBN2202 administration significantly reduced the weight and size of epididymal white adipose tissue (eWAT) without affecting overall body weight or skeletal muscle mass, despite increased food intake. Circulating GLP-1 levels were elevated, and expression of UCP1 in eWAT was partially restored, indicating enhanced thermogenic and hormonal signaling. Serum TNF- α levels were significantly reduced, suggesting modest systemic anti-inflammatory effects. In addition, KBN2202-treated mice exhibited increased center-zone activity in the open field test, indicative of reduced anxiety-like behavior. KBN2202 exerts its anti-obesity effects primarily through adipose tissue remodeling and metabolic hormone modulation, without suppressing appetite or inducing muscle loss. These multi-target properties position KBN2202 as a promising candidate for future development of safer and more effective therapies for obesity and its associated metabolic disorders.

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Keywords : Efficacy Test Center for Mental & Behavioral Disorders, High-fat diet (HFD), Obesity

PS-D-22

Engineering porcine fibroblasts with CD39/CD73 knock-in at the B4GALNT2 locus for xenotransplantation

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To overcome immunological barriers inherent to xenotransplantation, we developed porcine fibroblasts engineered to suppress xenoreactive expression and promote immunomodulation. Specifically, the B4GALNT2 gene, responsible for the synthesis of xenogeneic antigens, was targeted for knock-in of the human CD39 and CD73 genes. To generate donor animals suitable for xenotransplantation, we adopted a transgenic strategy that excluded the use of antibiotic selection markers.

A CRISPR/Cas9 system was employed, consisting of a gRNA targeting the B4GALNT2 locus and a donor vector expressing CD39 and CD73 under the control of the EF1 α promoter. The gene editing components—ribonucleoprotein complexes and donor DNA—were introduced into dermal fibroblasts derived from GTKO/CMHA(HO1/CD47) pigs. CD39/CD73-positive cells were isolated by fluorescence-activated cell sorting (FACS), and 37 single-cell-derived clones were established. PCR screening identified site-specific knock-in events at the B4GALNT2 locus in two clones (#23 and #27), which were further validated by DNA sequencing. The surface expression of CD39 and CD73 was confirmed by flow cytometry analysis.

Clones #23 and #27 will be used as nuclear donors for somatic cell nuclear transfer (SCNT) following the assessment of immunomodulatory function and karyotype stability. This attempt aims to produce genetically engineered pigs that are immunologically compatible for xenotransplantation and suggests a novel genotype for next-generation donor animal development.

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Keywords : Xenotransplantation, B4GALNT2, CD39, CD73, CRISPR/Cas9

PS-D-24

Phenotypic characterization for Alzheimer's disease in C57BL/6-Tg Krl mice with human mutant Presenilin 2 gene

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The C57BL/6-Tg (NSE-hPS2(*N141I)) Krl (NSE-hPS2m Tg) transgenic mice developed by the Korea FDA exhibit Alzheimer's disease (AD)-like characteristics. However, the precise age at which these pathological features become evident has not yet been clearly identified. To determine the onset timing of AD-related phenotypes, we examined both behavioral and anatomical changes in NSE-hPS2m Tg mice at 10, 12, and 14 months of age. Across all ages (10, 12, and 14 months), NSE-hPS2m Tg mice demonstrated significantly longer escape latency compared to Non-Tg controls in the Morris water maze, while also showing reduced resting time and fewer target crossings during probe trials. In the passive avoidance test, 12- and 14-month-old Tg mice were observed to have significantly shorter transfer latency than age-matched Non-Tg mice. MRI analysis revealed a noticeable reduction in hippocampal area beginning at 12 months in Tg mice, whereas cortical area remained stable across all ages examined. Biochemical assays further showed elevated cortical amyloid beta₄₂ (A β ₄₂) levels in Tg animals older than 12 months, yet hippocampal A β ₄₂ concentrations remained constant across ages, underscoring region-specific plaque accumulation. These combined findings indicate that the NSE-hPS2m Tg mice begin to display distinct and robust AD-like phenotypes starting from 12 months of age onward. This model may therefore be a highly valuable tool for preclinical evaluation of therapeutic candidates targeting Alzheimer's disease.

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Keywords : Alzheimer's disease, Transgenic mice, Presenilin 2, A β accumulation, MRI

PS-D-25

Therapeutic potential of targeting deubiquitinase to modulate mitochondrial function in pancreatic cancer

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Pancreatic ductal adenocarcinoma (PDAC), the major subtype of pancreatic cancer, is lethal disease showing a five-year survival rate of about 10%. The rapid proliferation and metabolic characteristic of PDAC require an increased protein turnover rate, leading proteotoxic stress. These conditions underscore the need for enhanced proteasome function in PDAC cells, making it a promising therapeutic target. Two proteasomal deubiquitinases, ubiquitin-specific peptidase 14 (USP14) and ubiquitin carboxyl-terminal hydrolase isozyme L5 (UCHL5), act as ubiquitin trimmers on substrates. Analysis of published PDAC data revealed that aggressive ductal cells exhibited high expression levels of USP14 and UCHL5, correlating with poor patient survival rates. This observation was further validated through immunohistochemistry assays, suggesting that targeting these deubiquitinases could be a promising therapeutic strategy.

To address USP14 and UCHL5 as potential therapeutic targets, this study investigated what phenotypic changes of PDAC cell lines exhibited after treatment with b-AP15, a specific dual inhibitor of USP14 and UCHL5. As expected, b-AP15 treatment showed significant proliferation inhibition in PDAC cell lines and even xenograft models, suggesting potential therapeutic efficacy. To elucidate mechanisms of cellular phenotypic changes, transcriptomics and proteomics were employed. These comprehensive analyses revealed that a critical impact on mitochondrial function in PDAC cells. These findings suggest the potential of b-AP15 as a therapeutic strategy targeting PDAC.

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Keywords : PDAC, Proteasome

PS-D-26

Transcriptomic profiling of genetic adaptation pathways in mice exposed to chronic heat stress

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As global temperatures continue to rise, understanding the genetic mechanisms that underlie mammalian adaptation to prolonged heat stress has become increasingly important. In this study, we investigated the transcriptomic and physiological responses of C57BL/6J mice exposed to chronic heat stress (35°C, 35–50% relative humidity) for 11 weeks. This thermal condition resulted in approximately 50% lethality, with no significant differences between sexes, indicating substantial thermal stress. To evaluate physiological adaptations, we monitored body weight, rectal temperature, and food and water intake. Heat-exposed mice exhibited reduced weight gain, elevated core body temperature, and decreased consumption of food and water, suggesting systemic disruption of growth and homeostatic regulation. In addition, behavioral tests revealed a significant reduction in swimming endurance and voluntary wheel-running activity, prompting further investigation of skeletal muscle tissue using RNA sequencing. Transcriptomic profiling identified 1,007 differentially expressed genes in skeletal muscle following long-term heat exposure. Gene ontology analysis showed enrichment of pathways involved in neural development, Wnt signaling, and mitochondrial protein synthesis, suggesting potential roles in heat-induced muscle remodeling. Gene set enrichment analysis further revealed upregulation of extracellular matrix organization and PI3K-Akt signaling pathways, whereas pathways related to thermogenesis and oxidative phosphorylation were notably downregulated. These findings indicate a shift in energy metabolism and tissue structure in response to sustained thermal stress. Taken together, these results demonstrate that prolonged heat exposure induces both physiological and gene regulatory adaptations in mice, particularly in skeletal muscle. This study provides an important first step toward elucidating the genetic pathways and molecular mechanisms underlying thermo-adaptation, offering valuable insights into how mammals may respond to climate-related environmental stress.

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Keywords : Climate change, Chronic heat stress, Thermal adaptation, Skeletal muscle, Transcriptomic profiling

PS-D-27

Anti-Angiogenic and Cancer Effects of a Bryopsis plumosa-Derived Peptide in Zebrafish

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The development of novel angiogenesis inhibitors with minimal toxicity is crucial for advancing cancer treatments. Peptides represent a promising class of therapeutic agents due to their efficient cellular penetration, target-specific activity, and low cytotoxicity. In this study, we synthesized novel peptides derived from the marine alga *Bryopsis plumosa* and evaluated their anticancer and anti-angiogenic activities. Impact on cell viability, migration, and self-renewal capacity was investigated using the human non-small cell lung cancer cell line H1299. The anti-angiogenic properties of the peptides were further assessed through in vitro tube formation assays using human umbilical vein endothelial cells (HUVECs) and corroborated with zebrafish embryo angiogenesis models. Using zebrafish (*Danio rerio*), an established vertebrate model for assessing disease mechanisms and toxicity, we examined the anti-angiogenesis effects of peptides. Our findings indicate that these peptides hold significant potential as dual-function therapeutic agents, exhibiting both potent anticancer and anti-angiogenic activities, thereby offering promising opportunities for innovative cancer therapeutics.

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Keywords : Angiogenesis, Zebrafish, Cancer, Peptide, Epithelial-mesenchymal transition

PS-D-28

Therapeutic Potential of KBN2202 in Alzheimer's Disease Mouse Model through Modulation of Microglial Polarization and Phagocytosis

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Alzheimer's disease (AD) is marked by progressive amyloid- β (A β) accumulation and chronic neuroinflammation. Microglia are critical for A β clearance and neuroimmune regulation. Here, we evaluated the therapeutic potential of KBN2202, a novel salicylic acid derivative, in the 5xFAD transgenic mouse model. Starting at 9 months of age, 5xFAD mice received daily oral gavage of KBN2202 for 3 months. At study end, mice were perfused with PBS and brains were extracted for histological analysis. A β plaques were detected by anti-A β immunostaining and Thioflavin-S (ThioS) staining. Microglial activation was assessed via TREM2, Iba1 and ThioS triple immunofluorescence, while lysosomal stress and neurodegeneration were evaluated by Lamp1 immunostaining. Brain cytokine profiles were quantified using a multiplex immunoassay. KBN2202 treatment significantly reduced interferon- γ (IFN- γ) levels in brain homogenates without affecting other measured cytokines, suggesting a shift away from pro-inflammatory M1 microglial polarization. Immunohistochemistry demonstrated a significant decrease in hippocampal A β burden and Lamp1 expression, indicating reduced plaque load and lysosomal stress. Thioflavin-S analysis confirmed a lower dense-core plaque density, and the A β : ThioS ratio decrease implied enhanced microglial compaction and clearance of diffuse A β aggregates. KBN2202 promotes microglial A β clearance, attenuates neuroinflammation, and reduces neurodegenerative markers in 5xFAD mice. These results support its potential as a candidate therapeutic for Alzheimer's disease.

This research was supported by the Bio&Medical Technology Development Program of the National Research Foundation (NRF) funded by the Korean government (MSIT) (RS-2023-00223559) and grants from the Korea Bio-health Technology R&D Project (RS-2023-00267453) and the Korea Health Technology R&D Project (RS-2021-KH113822) through the Korea Health Industry Development Institute (KHIDI), funded by the Ministry of Health & Welfare, Republic of Korea.

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Keywords : Alzheimer's Disease, 5xFAD, Microglia, Phagocytosis

PS-D-29

The Generation of Humanized Liver TK-NOG Mouse for the study of HCV Infections

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Hepatitis C virus (HCV) remains a major global health concern, with drug resistance necessitating the development of new therapeutics. However, progress is hampered by the limited species tropism by HCV, with susceptibility confined to humans and chimpanzees. In this study, human hepatocytes were transplanted into immunodeficient TK-NOG mice that specifically express the thymidine kinase (TK) gene in the liver to establish a highly efficient humanized liver mouse model, which was subsequently used as a hepatitis C virus (HCV) infection model. Ganciclovir (30 mg/kg) was administered to selectively eliminate endogenous hepatocytes, thereby creating a permissive hepatic niche for the engraftment, proliferation, and function maturation of transplanted human hepatocytes, which were subsequently introduced via intrasplenic injection. The transplanted cells maintained high engraftment efficiency and functional stability. The degree of liver humanization was evaluated by human albumin levels in the blood and the replacement index (%) through immunohistochemical analysis using cytokeratin 8/18 and mitochondrial DNA (mt-DNA) markers, confirming a repopulation rate exceeding 60%. Additionally, the chimeric hepatocytes were isolated from the humanized liver tissue by enzymatic digestion, and the proportion of human hepatocytes was quantitatively evaluated by flow cytometry, thereby demonstrating the engraftment and repopulation of human hepatocytes within the liver. After HCV infection, HCV RNA was detected in liver tissue, and histopathological analysis revealed expression of the HCV core protein in human hepatocytes. This study demonstrates that the TK-NOG-based humanized liver mouse model serves as a valuable platform for studying the pathogenesis of viral hepatitis and for the preclinical evaluation of antiviral agents, with significant utility in infectious liver disease research.

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Keywords : Human Hepatocyte, Hepatitis C Virus, TK-NOG, Humanized Liver Mouse

PS-D-31

Dynamic regulation of IL-36 α by estrogen controls uterine immune tolerance during implantation

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The uterus undergoes cyclical remodeling regulated by ovarian steroid hormones, primarily estrogen and progesterone. In mice, the estrous cycle comprises four phases: proestrus, estrus, metestrus, and diestrus. These hormonal changes not only affect the structural dynamics of the endometrium but also orchestrate immune responses. Around gestation day (GD) 3.5, the implantation window, immune balance is especially critical to allow embryo invasion while preventing excessive inflammation. Dysregulation of this immune-endocrine interface contributes to reproductive diseases such as endometriosis and endometrial cancer.

We identified IL-36 α , a pro-inflammatory cytokine from the IL-1 family, as an estrogen-responsive gene expressed specifically in mouse uterine epithelial cells. RNA-seq analysis and 17 β -estradiol treatment of ovariectomized mice confirmed its estrogen receptor alpha (ER α)-dependent regulation. Immunofluorescence revealed epithelial-restricted localization, and primary culture further validated its epithelial specificity. Notably, IL-36 α expression was significantly reduced in endometrial cancer.

During the normal estrous cycle, IL-36 α expression peaked at estrus, while in early pregnancy, its expression declined markedly at GD3.5. This downregulation may serve to suppress neutrophil migration and create an immune-tolerant uterine environment required for successful implantation. Single-cell RNA-seq supported this hypothesis by showing reduced expression of neutrophil-related genes at GD3.5.

To further explore its function, we developed an IL-36 α overexpression vector and plan to administer it in vivo between GD2.5 and GD3.5 in pregnant mice. This will allow us to evaluate whether enforced IL-36 α expression disrupts immune balance and impairs implantation.

In summary, IL-36 α is a dynamically regulated cytokine linking estrogen signaling to local immune modulation in the uterus. Understanding its role could provide insight into implantation biology and offer potential therapeutic strategies for immune-related uterine disorders.

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Keywords : Uterus, IL36 α , Implantation, Estrogen, Gestation day

PS-D-30

Effects of a Neuro Protective extract on Alleviating Scopolamine Induced Memory Deficits in C57BL/6 Mice via Enhancing Cholinergic Function

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Alzheimer's disease (AD) is a disease of progressive neurodegeneration characterized by cognitive decline, including acetylcholine depletion, cholinergic dysfunction, memory loss, disabilities, and behavioral changes. This study investigated the efficacy of plant-derived neuroprotective extract (NPX) in a scopolamine-induced memory loss mouse model. The NPX product is a natural ingredient consisting of broccoli, kohlrabi, radish powder, Gastrodia elata, Tissue-cultured wild ginseng, and concentrated broccoli extract. A group of male C57BL/6 mice were divided into four groups (n=7): control, scopolamine-treated, tacrine-treated (standard AD drug), and NPX-treated. Both NPX (1000 mg/kg) and tacrine (10 mg/kg) were administered orally for 4 weeks. For the memory decline assessment, performance of the behavior was assessed weekly using the Y-maze test, and brain samples were collected to assess biochemical and histological changes. The results of the study showed that NPX administration significantly improved spontaneous transition behavior, and immunohistochemical analysis showed a decrease in amyloid-beta (A β) and caspase-3 levels and an increase in choline acetyltransferase (ChAT) expression. This suggests the alleviation of neurodegeneration and the restoration of cholinergic signaling. This effect was observed with tacrine treatment. Collectively, the NPX showed neuroprotective and cognitive support effects, suggesting its potential as a functional herbal medicine for memory disorders associated with Alzheimer's disease.

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Keywords : Alzheimer's disease, Amyloid-beta, Scopolamine, Memory loss

PS-D-32

Immunohistochemical Verification of Genetically Engineered Pigs for Xenotransplantation

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Genetically engineered (GE) pigs lacking xenoantigens and expressing human immune-regulatory proteins are essential for successful clinical xenotransplantation. To overcome immune rejection, three glycosyltransferase genes GGTA1, B4GALNT2, and CMAH, responsible for the synthesis of α -Gal, Sd(a), and Neu5Gc were knocked out. In addition, pigs were engineered to express human transgenes: CD39, CD55, CD46, and thrombomodulin (hTBM) to enhance immunological compatibility. This study aimed to verify the expression of glycan antigens and human transgenes in multiple GE pig kidneys using immunohistochemistry (IHC).

Paraffin-embedded kidney sections from GE pigs were stained with GSL I-B α lectin for α -Gal, DBA lectin for Sd(a), and an anti-Neu5Gc IgY antibody. Human transgenes (hCD46, hCD55, hCD39, hTBM) were detected using specific monoclonal antibodies. Wild-type (WT) pig kidneys used as positive controls for glycan antigens and negative controls for human proteins, while human kidneys served as the reverse. IHC analysis demonstrated strong expression of α -Gal, Sd(a), and Neu5Gc in wild-type pig kidneys. α -Gal was mainly localized to tubular epithelial cells, while Sd(a) and Neu5Gc were prominent in the glomeruli, vessels, and capillaries. Neu5Gc was not detected in tubules. In contrast, TKO/QKI pig kidneys showed complete loss of all three xenoantigens, confirming successful gene deletion.

The expression of human transgenes was successfully detected in a tissue-specific manner, and their distribution patterns varied between human and TKO/QKI pig kidneys. In human kidney tissue, the hCD39 was primarily expressed in vessels, whereas it was strongly detected in tubules, glomeruli, and to a lesser extent in vessels and capillaries in pigs. hCD55 showed minimal tubular expression in human kidneys but was clearly expressed in pig tubules, glomeruli, and vessels, with no signal in capillaries. Both hCD46 and hTBM were expressed in pig tubules and glomeruli, but not in vessels, contrasting with their vascular expression in human tissue. Notably, hCD46 showed stronger expression than hTBM across all regions of the TKO/QKI pig kidney.

In conclusion, this study demonstrates that immunohistochemical validation is essential to confirm both the elimination of glycan xenoantigens and the functional expression of human immune-regulatory genes in genetically modified pigs.

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Keywords : Xenotransplantation, Genetically Engineered Pigs, Immunohistochemistry

PS-D-33

Prophylactic administration of *Lactobacillus sakei* CVL-001 enhances bacterial clearance and attenuates intestinal inflammation in mice infected with *Citrobacter rodentium*

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The rising incidence of bacterial infections, compounded by climate change and increasing antibiotic resistance, highlights the urgent need for alternative therapeutic strategies that are both effective and sustainable. *Lactobacillus* spp. are known to exert host defense and immune regulatory properties against microbial infections through modulation of mucosal immunity and enhancement of epithelial barrier function. A cytosolic receptor Nod2 triggers host innate immune responses by recognizing muramyl dipeptide, a conserved motif of bacterial peptidoglycan found in both Gram-positive and Gram-negative bacteria. This study was aimed to clarify whether and how *Lactobacillus sakei* CVL-001 when administered prophylactically, suppresses *Citrobacter rodentium* infection in mice and contributes to bacterial clearance and intestinal immune homeostasis. Through this investigation, we seek to explore the immunomodulatory effects of CVL-001 and provide insights into the potential Nod2-dependent pathways involved in probiotic-mediated protection against enteric bacterial pathogens.

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Keywords : *Citrobacter rodentium*, NOD2, Innate Immunity, Mucosal Immunology, Probiotics

PS-D-35

Irradiation of Tumor Draining Lymph Nodes Undermines PD-1 Blockade by Disrupting Peripheral PD-1⁺CD8⁺ T Cell Responses

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Elective nodal irradiation (ENI) is a widely practiced radiotherapy technique aimed at suppressing regional lymphatic dissemination in various malignancies. Nevertheless, emerging evidence has raised concerns regarding its potential to hinder effective antitumor immunity. In this study, we explored how ENI influences immune responses in a CT-26 murine colon carcinoma model. Mice received radiation either confined to the primary tumor or extended to include the tumor-draining lymph nodes (TDLNs). When combined with anti-PD-1 immune checkpoint therapy, concurrent irradiation of both the tumor and TDLNs was associated with continued tumor progression. In contrast, irradiating the tumor alone led to superior tumor suppression. Immunophenotyping via flow cytometry revealed that tumor-only irradiation promoted an increase in PD-1⁺, CD8⁺ T cells within distal lymphoid organs, thereby enhancing the efficacy of PD-1 blockade. These findings indicate that ENI may impair systemic antitumor immunity by disrupting T cell priming in TDLNs. Consequently, excluding ENI in radiotherapy regimens may strengthen systemic immune responses and improve therapeutic outcomes when combined with PD-1-targeted immunotherapy.

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Keywords : Immune Checkpoint Blockade (PD-1), Tumor-Draining Lymph Nodes (TDLNs), Elective Nodal Irradiation (ENI), Antitumor immunity, CT-26 murine colon carcinoma model

PS-D-34

Hepatocellular carcinoma associated with non-alcoholic fatty liver disease in Leptin KO/Korl mice

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Hepatic steatosis, characterized by excessive lipid accumulation in hepatocytes, can trigger inflammation, which may subsequently lead to liver fibrosis, cirrhosis, and even hepatocellular carcinoma (HCC). This study aimed to investigate the effect of a choline-deficient, L-amino acid-defined, high-fat diet (CDAHFD) on liver cancer progression and its associated pathological factors.

Six-week-old male Leptin KO/Korl mice were fed either a chow diet (Lep KO group) or a CDAHFD (CDAHFD group) for 38 weeks. After the feeding period, blood and liver tissues were collected for analysis. Leptin KO/Korl mice showed a gradual increase in body weight, which stabilized or slightly declined, with no significant differences in liver weight between the two groups. The CDAHFD group exhibited a significant increase in both the number and maximum size of hepatic nodules compared to the Lep KO group, suggesting that CDAHFD promotes liver tumor progression in leptin-deficient mice.

Serum levels of alanine aminotransferase (ALT), tumor necrosis factor- α (TNF- α), and hepatic hydroxyproline were significantly elevated in the CDAHFD group, indicating increased liver injury, inflammation, and fibrosis. Sirius red staining also revealed marked collagen deposition. Furthermore, the expression levels of α -fetoprotein (AFP), lipocalin-2 (LCN2), and β -catenin were significantly upregulated in the CDAHFD group, indicating tumor-associated molecular alterations. Histological analysis (H&E staining) confirmed the presence of both hepatocellular adenoma (HCA) and HCC in the CDAHFD group.

These findings suggest that CDAHFD promotes liver tumorigenesis in leptin-deficient mice by exacerbating liver injury, inflammation, and fibrosis. Further studies are needed to clarify the molecular mechanisms by which leptin deficiency and CDAHFD contribute to liver tumorigenesis, and follow-up experiments are currently underway to investigate their respective roles in this process.

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Keywords : Leptin KO/Korl mice, CDAHFD, NAFLD, HCC

PS-D-36

Age-related changes in Rnf130 expression in the mouse ovary

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Reproductive aging affects all women regardless of race, environment, or ethnicity and is marked by an early decline in ovarian hormones. Cholesterol is a universal precursor for steroid hormones, and its uptake is mediated by the low-density lipoprotein receptor (LDLR). A recent study has shown that the display of LDLR on the plasma membrane surface is modulated by Ring finger protein 130 (RNF130), an E3 ubiquitin ligase. RNF130 ubiquitinates LDLR and sequesters it away from the plasma membrane, thereby reducing LDLR availability. Whether RNF130 is expressed in the ovary, the major site of steroid hormone production, and whether aging influences its expression is not known. To investigate the association between age and RNF130 expression *in vivo*, female mice at distinct reproductive stages [8-week-old, 24-week-old, 48-week-old, 51-week-old] were used for ovary sampling. Mice received 5 IU PMSG to synchronize follicular development. Ovaries were collected 48 hours post-PMSG. Eight-week-old mice were also sacrificed at each time point for ovary collection to ensure the integrity of the samples. Total RNA and protein were extracted from ovarian samples and analyzed by quantitative PCR (qPCR) and western blotting. qPCR and western blot analysis results show that Rnf130 is expressed at significantly higher levels in ovaries of 24-week-old and 48-week-old mice compared to those of 8-week-old mice. Among human ovarian cell lines, SKOV3, a human ovarian adenocarcinoma cell line, expresses both *RNF130* and *LDLR*. Immunofluorescence staining confirmed that *RNF130* is localized in the cytoplasm of SKOV3 cells. Our data demonstrate that aging may influence *Rnf130* expression in the mouse ovary. Given the role of RNF130 in LDLR regulation, this suggests that ovarian cholesterol utilization may be altered with age. Further studies are warranted to determine the functional impact of elevated RNF130 on LDLR availability, cholesterol uptake, and steroidogenesis in the ovary.

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Keywords : Ovary, Reproduction, Aging, RNF130, LDLR

PS-D-37

Expression of necroptosis effectors is influenced by diet in the mouse ovary

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Necroptosis, a programmed cell death pathway triggered by inflammatory cytokines, is mediated by actions of Receptor-Interacting Serine/Threonine-Protein Kinase 1 (RIPK1), RIPK3, and Mixed Lineage Kinase domain-Like pseudokinase (MLKL). Inhibiting *Ripk1* expression in diet-induced obesity mice has been shown to alleviate obesity phenotypes, and *Ripk3* is also implicated in diverse metabolic diseases with conflicting results. While the importance of necroptosis in obesity is emerging, its implications in ovarian functions are not known. We aimed to investigate the expression of necroptosis effectors in the mouse ovary after a short-term high-fat diet. We also examined their status in *Ripk3*^{-/-} mice and in human ovarian cell lines. C57BL/6J mice (6-week-old) were fed either a normal diet (ND, 10% fat) or a high-fat diet (HFD, 60% fat) for 4 weeks (short-term group). *Ripk3*^{-/-} mice (6-week-old) were fed the same diets for 4 months (long-term group). All mice received 10 IU PMSG, and ovaries were collected 48 hours later. We conducted quantitative PCR (qPCR), western blotting, and immunofluorescence staining (IF) to assess the status of necroptosis effectors. In HFD-fed mouse ovaries, qPCR revealed no significant differences in mRNA levels between ND and HFD groups; however, western blotting showed a significant increase in RIPK3 levels. In ovarian sections, pMLKL was localized in subsets of theca and granulosa cells. pMLKL was also detected in *Ripk3*^{-/-} ovaries after long-term HFD, suggesting that the necroptosis pathway may still be operative in the absence of *Ripk3*. pMLKL IF in TSZ-treated KGN (a human ovarian adenocarcinoma cell line) cells showed abundant puncta-like pMLKL signals, suggesting that granulosa cells respond to TSZ and induce necroptosis. The results collectively indicate that ovarian cells respond to a necroptosis-inducing signal and that diet may influence the pathway. Whether RIPK3 plays any necroptosis-independent role in the ovary requires further investigation.

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Keywords: Necroptosis, RIPK3, Obesity, Reproduction, Ovary

PS-D-38

An integrated nonclinical platform for enhanced immune checkpoint inhibitor evaluation: combining calcein-AM cytotoxicity assay with MHC-deficient NOG mice

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Accurate *in vitro* and *in vivo* assessment of immune checkpoint inhibitor (ICI) efficacy is critical for identifying promising therapeutic candidates prior to clinical evaluation. Conventional cytotoxicity assays, such as chromium-51 and lactate dehydrogenase release assays, are limited by low specificity, high background noise and complication associated with radioactive materials. To address these issues, we developed a non-radioactive, fluorescence-based cytotoxicity assay using Calcein-acetoxymethyl ester (Calcein-AM)-labeled cancer cells and human peripheral blood mononuclear cell (PBMC)-derived T cells. This method directly quantifies fluorescence released exclusively from lysed cancer cells, resulting in enhanced sensitivity and specificity compared to conventional assays. Among the tested cancer cell lines, only MDA-MB-231 cells, which express high levels of programmed death-ligand 1 (PD-L1), exhibited significantly increased T cell-mediated cytotoxicity in response to treatment with the clinically approved ICI Tecentriq, while PD-L1-low cell lines showed minimal response. These findings underscore the assay's specificity to detect PD-L1-dependent responses and support its utility as a screening platform for ICI candidates. To extend evaluation into physiologically relevant *in vivo* settings, we addressed a key limitation of conventional NOG (NOD.Cg-Prkdcscidll2rgtm1Sug/ShiJic) mice—early-onset graft-versus-host disease (GVHD) following human PBMC engraftment by employing major histocompatibility complex class I/II double knockout (dKO) NOG mice. This model maintained comparable levels of human immune cell engraftment, while significantly extending survival, enabling repeated ICI dosing and more consistent monitoring of antitumor responses. Together, our integrated approach—combining Calcein-AM-based *in vitro* screening with GVHD-delayed dKO-NOG *in vivo* modeling—provides a robust and translationally relevant platform for nonclinical ICI evaluation. This system enables more efficient candidate selection and improves the predictive accuracy of clinical efficacy in immuno-oncology development.

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Keywords: Immune checkpoint inhibitor, Calcein-AM release assay, MHC dKO-NOG humanized mouse model, GVHD, PD-L1

PS-D-39

Structural and Functional Evaluation of Anti-Cancer Peptides Using Zebrafish Embryo

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The structural conformation and functional activity of anticancer peptides (ACPs) are crucial for their effective interactions with target molecules in cancer cells. Specific amino acid sequences primarily mediate these biological functions; however, conformational changes induced during peptide modification or formulation may significantly impair their therapeutic potential. Furthermore, certain ACPs exhibit sensitivity to lipid-related environments, markedly influencing their efficacy.

Most ACPs share key characteristics, including membranolytic or intracellular-damaging activity, amphipathicity, and net positive charges. These attributes enable their roles in disrupting cell membranes and enhancing innate or adaptive cytotoxic mechanisms. Amino acid modifications can alter these functions and influence their suitability as drug delivery systems.

In recent research, zebrafish embryos have become an essential vertebrate model for evaluating drug efficacy and toxicity due to their optical transparency, rapid development, genetic manipulability, and compatibility with high-throughput screening methodologies. This study assessed the anticancer and anti-angiogenic effects of peptides derived from genetic resources of 30 marine organisms. Cytotoxic activities were initially determined in cancer cell lines, and subsequent developmental toxicity evaluations were conducted using zebrafish embryos.

Interestingly, some peptides exhibited minimal toxicity toward cancer cells but induced severe developmental toxicity in zebrafish embryos, resulting in embryonic lethality even at low concentrations. Conversely, conventional anticancer agents showed negligible embryonic toxicity at substantially higher concentrations. These findings highlight the necessity of utilizing zebrafish embryo assays extensively for comprehensive evaluation of biocompatibility and therapeutic efficacy of novel biomaterials and peptide-based drug delivery systems.

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Keywords: Zebrafish, Anti-cancer peptide

PS-D-40

Regulatory Effects of Pentosan Polysulfate Sodium on Benign Prostatic Hyperplasia through Anti-inflammatory Mechanisms

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Pentosan polysulfate sodium (PPS), marketed as Penpol, is an oral medication primarily used for the treatment of interstitial cystitis and bladder pain syndrome (BPS). In addition to its established therapeutic applications, PPS has been reported to exhibit anti-inflammatory properties and promote extracellular matrix degradation. However, its potential role in benign prostatic hyperplasia (BPH) has not been investigated. In this study, we evaluated the efficacy of Penpol in a rat model of BPH. BPH was induced by orchiectomy followed by administration of testosterone propionate and β -estradiol for eight weeks to simulate hormonal imbalances associated with the condition. Penpol was administered during the final four weeks of the induction period. The effects of PPS on prostate weight, histopathological changes, inflammatory cytokine expression, and extracellular matrix remodeling were assessed. The results showed that while prostate weight was significantly increased in the BPH group, Penpol administration did not lead to a significant reduction in prostate weight. However, histopathological analysis revealed a significant improvement in epithelial cell thickness and inflammation scores in the Penpol-treated group, suggesting a potential reduction in urethral compression. Additionally, Penpol administration reduced key BPH-related markers, including prostate-specific antigen (PSA), dihydrotestosterone (DHT), 5- α reductase type 2 (5A-R2), and androgen receptor (AR) levels in prostate tissue. These findings suggest that Penpol may have therapeutic potential in alleviating BPH-related pathology, particularly through its anti-inflammatory effects.

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Keywords: Benign prostatic hyperplasia, Castration, Testosterone, Penpol

PS-D-41

T cell ratio in the lung inflammation predicts prognosis in a COVID-19 mouse model

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Background & Aim : The hACE2 transgenic mouse model has been widely used to evaluate the preclinical effects of SARS-CoV-2 infection. Although body weight loss is a widely used standard indicator of disease severity, we aimed to identify more diverse and refined histopathological parameters to more accurately predict disease outcomes. **Results :** We analyzed approximately 100 lung samples collected from multiple studies, comprising 3 experimental cohorts that included no infection, infection only, and vaccine-treated groups. We focused our analysis on a single lung lobe, as is commonly done in many studies due to practical constraints in sample availability. However, inflammation varied markedly between lung lobes, making single-lobe analysis potentially unreliable. In some vaccinated cohorts, inflammation persisted despite body weight recovery, revealing a discrepancy between histological severity and clinical indicators. Given this limitation, we additionally evaluated the composition pattern of infiltrating immune cells. A convolutional neural network (CNN) was trained using Neuro-T software, with body weight loss scores (10%, 15%, or ≥20%) used as classification labels to teach the model histological patterns associated with clinical outcomes. Low-magnification images were used to evaluate the overall degree of inflammation across the tissue, while high-magnification images were used to capture detailed immune cell composition within inflamed regions. Interestingly, immune cell composition showed a stronger correlation with body weight changes than overall inflammation severity. Pathologist review confirmed T cell-dominant infiltration in low-score images identified by the CNN. This observation was further validated by CD3 immunohistochemistry and deep learning-based quantification using QuPath. Despite regional differences in inflammation, T cell ratios within infiltrates remained consistent across lobes.

Conclusion : Our study demonstrates that the T cell ratio within lung infiltrates can serve as a supportive histopathological biomarker for prognosis in COVID-19 mouse models, complementing conventional indicators such as body weight loss.

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Keywords : T cell, COVID-19, Lung inflammation, Deep learning, CNN

PS-D-43

Development of a House Dust Mite-Induced Lung Dysfunction Model for Evaluating the Protective Effects of Prunus mume Extract

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Prunus mume, commonly known as Japanese apricot, has traditionally been used for its anti-inflammatory, antioxidant, and fatigue-reducing properties. During its processing, large amounts of by-products such as peel, pulp, and seeds are generated; however, their functional utilization remains limited due to an insufficient understanding of their value. In this study, we evaluated the protective effects of Prunus mume extract against house dust mite (HDM)-induced lung injury using a BALB/c mouse model. The extract was administered orally at doses of 50, 100, and 200 mg/kg/day for 6 days. It significantly attenuated HDM-induced pulmonary tissue damage and inflammatory cell infiltration. Furthermore, the extract markedly reduced the phosphorylation of key inflammatory signaling proteins, including AKT, ERK, JNK, and NF-κB in vivo. In addition, it exhibited strong antioxidant activity by dose-dependently suppressing the production of reactive oxygen species (ROS) and nitric oxide (NO). These findings suggest that Prunus mume extract has potential as a functional natural material for alleviating HDM-induced pulmonary inflammation through its anti-inflammatory and antioxidant actions.

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*Corresponding author : Young Yoon

Keywords : Prunus mume, Animal experiment, HDM, Inflammation, Antioxidant

PS-D-42

Establishment and Characterization of a Twindemic Mouse Model Co-Infected with Influenza A and SARS-CoV-2 Virus

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The concurrent circulation of influenza A virus and SARS-CoV-2 variants has raised concerns over the potential for synergistic respiratory infections, commonly referred to as a "twindemic." While each virus independently causes significant respiratory illness, the order and dynamics of co-infection may critically influence disease severity. In this study, we established and evaluated a twindemic mouse model using human angiotensin-converting enzyme 2 transgenic (hACE2-Tg) mice co-infected with the H1N1pdm strain of influenza A virus and the SARS-CoV-2 XBB.1.5 variant. To assess the impact of infection sequence, two co-infection groups were designed: one receiving influenza A virus followed by SARS-CoV-2 (IAV-CoV), and the other receiving SARS-CoV-2 followed by influenza A virus (CoV-IAV).

Clinical parameters including body weight, survival rate, and lung-to-body weight ratios were measured. Lung tissues were collected to quantify viral titers by real-time PCR and to assess histopathological changes through hematoxylin and eosin (H&E) staining. A semi-quantitative inflammatory scoring system was used to evaluate the severity of lung inflammation. Both co-infection groups exhibited more severe disease progression compared to single-virus controls, with significant weight loss, increased lung weights, and reduced survival. However, the IAV→CoV group showed more pronounced pathology overall. Viral titer analysis confirmed active replication of both viruses in the lungs of co-infected mice, with higher viral loads observed in sequential infections. Histological analysis revealed extensive alveolar destruction, epithelial degeneration, and immune cell infiltration, with significantly elevated inflammatory scores in both twindemic groups.

These findings suggest that sequential co-infection with H1N1pdm influenza A virus and SARS-CoV-2 XBB.1.5 exacerbates pulmonary pathology in a manner that is influenced by the order of infection. The established twindemic model provides a valuable platform for investigating the mechanisms of viral synergy and for evaluating therapeutic interventions targeting co-infections of respiratory viruses.

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Keywords : Twindemic, Influenza A, SARS-CoV-2, Lung inflammation

PS-D-44

Standardization of an Ex Vivo Chicken Ileum Assay for Pre-Screening Gut-Active Compounds in Functional Food Research

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To reduce reliance on live animal experimentation in functional food screening, we established an ex vivo chicken ileum contraction assay as a preclinical platform to evaluate intestinal activity of candidate compounds. Ileal segments were harvested post-mortem at 1, 3, and 6 hours and mounted in organ baths. After stabilization, contractility was induced using acetylcholine (100 μM), and the area under the curve (AUC) of isometric contractions was recorded for 10 minutes. A time-dependent decline in contractile response was observed, with values decreasing to 100%, 53%, and 28% at 1, 3, and 6 hours, respectively. Histological examination revealed progressive autolysis consistent with the functional deterioration. These findings identified 1 hour post-mortem as the optimal tissue viability window for reliable data acquisition.

To validate the model's responsiveness to pathological and therapeutic stimuli, tissues were challenged with lipopolysaccharide (LPS, 100 μg/mL), which reduced contractility to 38% of control, and then co-treated with fructooligosaccharide (FOS, 1 mg/mL), which restored contractile function, suggesting protective efficacy. However, as FOS is a non-digestible compound that bypasses enzymatic degradation, its compatibility with the ex vivo setting may not be generalizable to metabolized compounds.

This study highlights the utility of ex vivo ileum assays as a reductionist tool for rapid screening of gut-active agents. Nevertheless, the model is limited by its inability to reflect systemic metabolism, absorption, and host-microbe interactions present in vivo. Therefore, further validation through comparative in vivo efficacy studies and reproducibility assessments is critical to confirm translational relevance and support standardization of the model for broader application in functional food research.

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Keywords : Alternative methods, Animal experiment, Ex vivo, Health functional food, In vitro

PS-D-45

Disrupted PDIA3-PEMT Signaling Under Chronic ER Stress Fuels NASH via ER Membrane Destabilization

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The endoplasmic reticulum (ER) stress response, particularly the unfolded protein response (UPR), is a well-established contributor to the pathogenesis of metabolic dysfunction-associated steatohepatitis (MASH). However, under prolonged and severe ER stress, the adaptive UPR threshold can be breached, resulting in UPR failure and irreversible hepatocellular damage. This study investigates a novel mechanism by which ER stress-induced decay of the chaperone PDIA3 disrupts PEMT maturation, ultimately altering ER membrane integrity and accelerating MASH progression. Liver samples from patients with MASH and a CDA-HFD-induced MASH mouse model were analyzed alongside palmitic acid-treated LO2 cells in vitro. Histological, biochemical, immunoprecipitation, and immunohistochemical analyses were conducted to evaluate the PDIA3-PEMT axis. PEMT overexpression was used to test its protective role in hepatocytes under ER stress conditions. Severe and chronic ER stress led to IRE1 α -dependent RIDD-mediated degradation of PDIA3, a key folding chaperone. Loss of PDIA3 impaired the proper folding and stabilization of PEMT, its client protein, resulting in a significant decrease in PEMT activity. This disruption was accompanied by a compositional shift in ER phospholipids, marked by a reduced PC/PE ratio and increased ER membrane fluidity and permeability. Morphological and functional alterations in the ER membrane were associated with heightened inflammasome activation (NLRP3), increased expression of TLR4 and IL-6, and histologic progression of MASH. Restoration of PEMT expression mitigated these pathological changes. Our findings identify PDIA3 as a critical RIDD target during severe ER stress and reveal that its loss destabilizes PEMT, disrupting phospholipid homeostasis and ER membrane integrity. This mechanistic cascade contributes to a morpho-functional transformation of the ER, serving as a tipping point toward irreversible liver injury and MASH progression. Enhancing PEMT stability may offer a novel therapeutic approach to halt the transition from adaptive ER stress to pathological outcomes in MASH.

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Keywords : Endoplasmic reticulum (ER) stress, MASH, PDIA3, Phospholipids, PEMT

PS-D-47

Somatic Nf2 Inactivation Induces Spontaneous Metastasis in a Mouse Model of Gastric Cancer

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Background & Aim : While genetically engineered mouse (GEM) models have significantly advanced our understanding of gastric cancer (GC) initiation and progression, few faithfully recapitulate spontaneous metastatic behavior observed in patients. The lack of in vivo models limits the investigation of metastatic mechanisms and therapeutic strategies. To address this gap, we aimed to establish a spontaneous gastric cancer GEM model with distant metastasis.

Results : We performed genome-wide CRISPR screening in S1M mouse GC cells (*Cdh1*^{+/+}, *Trp53*^{F/F}, *Smad4*^{F/F}) using a peritoneal dissemination model and identified *Nf2* as an enriched candidate through amplicon sequencing. To investigate the role of *Nf2*, we induced somatic mutations of both *Cdh1* and *Nf2* in *Anxa10a*-Cre-ERT2; ROSA26-LSL-Cas9-GFP; *Trp53*^{F/F} mice via AAV-sgRNA delivery and monitored until they became moribund. Compared to the *Cdh1*-single-deficient model (*Trp53*^{F/F}; *Cdh1*^Δ AAV, CP), the *Cdh1*/*Nf2*-double-deficient model (*Trp53*^{F/F}; *Cdh1*/*Nf2* AAV, CPN) showed significantly poorer survival and a tendency toward increased lung metastases. RNA sequencing of *Nf2* deficient mouse GC cells revealed elevated EMT-related gene expression, and Gene Set Enrichment Analysis (GSEA) confirmed enrichment of the EMT gene signature. Immunofluorescence analysis revealed increased Vimentin expression in *Nf2* deficient mouse GC cells. Western blot analysis of *Nf2* deficient mouse and human GC cells showed increased YAP and decreased p-YAP, the inactive form of YAP, indicating enhanced YAP activity. Consistently, IHC exhibited nuclear YAP accumulation in CPN gastric tumors compared to CP tumors. The expression levels of YAP transcriptional targets were upregulated in *Nf2* deficient S1M mouse gastric cancer cells, as determined by RT-qPCR (*Cyrt1*, *Ctgf*) and Western blot (Survivin).

Conclusion : In this study, we generated a GEM gastric cancer model that spontaneously develops metastases by introducing somatic *Nf2* deletion into a *Cdh1* and *Trp53*-deficient background. This somatic model exhibited a tendency for spontaneous lung metastasis and EMT, with YAP pathway activation, providing a platform for investigating the metastatic biology of gastric cancer.

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Keywords : Stomach Neoplasms, CRISPR-Cas Systems, Epithelial-Mesenchymal Transition (EMT)

PS-D-46

SERPINE1 promotes epithelial-mesenchymal transition in Smad4-deficient gastric cancer

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Background & Aims : *SMAD4* mutations are observed in approximately 14% of human gastric cancers. *SMAD4* is a central molecule of tumor-suppressive TGF- β /BMP signaling, and its deficiency is thought to contribute to tumor progression. However, how *SMAD4* deficiency interacts with tumor microenvironment (TME)-derived signals to promote malignancy remains unclear. This study aimed to investigate how *SMAD4* deficiency cooperates with microenvironmental signals to drive tumor progression in gastric cancer, using a genetically engineered mouse model.

Results : Clinically, E-cadherin-negative gastric tumors with low *SMAD4* expression display enhanced epithelial-mesenchymal transition (EMT) signatures and are significantly associated with poor survival. In our mouse model, *Cdh1* heterozygous and *Trp53* knockout (ChetPE) mice failed to initiate tumor formation, whereas concurrent *SMAD4* deletion (ChetPS) drove tumorigenesis accompanied by loss of E-cadherin expression. Comparative analysis between *Cdh1*/*Trp53* double-knockout (CP) and *Cdh1*/*Trp53*/*SMAD4* triple-knockout (CPS) tumors demonstrated that *SMAD4* deficiency accelerated local invasion. Gene set enrichment analysis (GSEA) of microarray data revealed significant enrichment of EMT programs in *SMAD4*-deficient mouse gastric tumors relative to *SMAD4* wild-type tumors. In vivo, *SMAD4*-deficient mouse gastric tumors showed E-cadherin loss, which was less pronounced when cells of the same genotype were cultured in vitro without a tumor microenvironment. This highlights the critical role of the tumor microenvironment in promoting E-cadherin loss. Immunohistochemistry of mouse gastric tumors revealed more abundant cancer-associated fibroblasts (CAFs) expressing α SMA and PDGFR α in *SMAD4*-deficient tumors. SERPINE1, primarily derived from CAFs but also expressed in cancer cells, was associated with the induction of EMT-related gene expression and PI3K-AKT signaling, particularly in *SMAD4*-deficient cells, promoting E-cadherin loss and migration. Pharmacological inhibition of SERPINE1 suppressed EMT features and reduced PI3K signaling in *SMAD4*-deficient cells, but led to compensatory activation of the MAPK-ERK pathway. Co-treatment with a SERPINE1 inhibitor and the MEK inhibitor trametinib resulted in a greater reduction in cell viability and migration in *SMAD4*-deficient mouse gastric cancer cells compared to wild-type cells.

Conclusion : *SMAD4* loss is associated with increased sensitivity of gastric cancer cells to SERPINE1-induced EMT via PI3K-AKT signaling. Combined inhibition of SERPINE1 and MEK reduces viability, suggesting a therapeutic strategy for *SMAD4*-deficient gastric cancer.

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Keywords : EMT, Tumor microenvironment, Cancer associated fibroblast, Serpine1

PS-D-48

Beneficial effects of Lobeglitazone, a new PPAR- γ agonist, on atherosclerosis and valve inflammation

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Objectives : Lobeglitazone, one of the thiazolidinediones (TZDs), is an activator of PPAR- γ and is used as an oral medication for Type 2 diabetes. Although activated PPAR- γ , known for its anti-inflammatory properties, suggests that lobeglitazone could be effective in various diseases, its therapeutic effects on advanced atherosclerosis have not been confirmed. Therefore, this study investigated the effects of lobeglitazone on established atherosclerosis and valve inflammation in mice induced with hyperlipidemia. **Methods** : The ApoE (-/-) mouse model of atherosclerosis was subjected to a high-fat, high-cholesterol Western diet from 7 weeks of age to induce atherosclerosis for a duration of 16 weeks after reaching adulthood. Following this, while maintaining the Western diet, the mice were divided into two groups: 1) control group receiving saline and 2) group receiving Lobeglitazone (10mg/kg) administered orally once every other day for 8 weeks.

Results : Histological analysis of the aortic root in hypercholesterolemic mice following Lobeglitazone administration revealed a reduction in lipid content and macrophage infiltration in the lesions and plaques. Additionally, an increase in collagen content in the lesions due to Lobeglitazone treatment was observed. These findings suggest that Lobeglitazone administration contributes to the stabilization of lesions, indicating its potential for mitigating atherosclerotic progression.

Conclusions : This study revealed through histological examinations that Lobeglitazone administration reduces lipid accumulation and inflammatory cell infiltration within the lesions, while also showing an increase in the proportion of collagen, which plays a crucial role in lesion stability. While previous research has demonstrated that activation of PPAR- γ leads to increased expression of LXR α , ABCA1, ABCG1, and CD36, thereby enhancing oxLDL uptake or cholesterol efflux associated with decreased lipid deposition, further studies are needed to understand the mechanisms underlying the increase in collagen content.

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Keywords : Atherosclerosis, PPAR γ Agonist, Lobeglitazone

PS-D-49

Kras A146T-Driven Gastric Tumoroid Model Exhibiting Metastasis in Immunocompetent Mice

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Background & Aims : Gastric cancer (GC) is one of the most lethal malignancies, with metastasis being the primary cause of poor prognosis. However, spontaneous metastatic models that reflect human disease are rare in immunocompetent mice. We aimed to establish a metastatic gastric cancer model by exposing mouse GC models to N-methyl-N-nitrosourea (MNU) to induce random mutations and identify key genetic alterations involved in metastatic progression.

Results : Anxa10a-Cre; Cas9-GFP; Trp53 F/F; Cdh1 F/F (CP) mice were treated with tamoxifen and MNU (CP;MNU). Among 40 mice, 2 were found moribund at 5 months, exhibiting multiple metastatic lesions in the lungs, liver, and malignant ascites. CP;MNU mice exhibited multiple distant metastases, whereas CP mice showed no evidence of distant metastases. Tumoroids derived from CP;MNU tumors exhibited a compact and solid morphology with densely packed cells, lacking cystic structures, and displayed features consistent with epithelial-mesenchymal transition (EMT). Upon allograft transplantation into immunocompetent mice, CP;MNU tumoroids gave rise to rapidly growing tumors with widespread metastases to the lungs, liver, and peritoneum. In contrast, CP tumoroids remained slow-growing and non-metastatic in immunocompetent mice. Whole-exome sequencing of CP;MNU tumoroids revealed a Kras A146T mutation, previously reported in human gastric cancer. To explore its potential role in tumor progression, Kras A146T was overexpressed in Trp53-deficient organoids, which normally fail to form tumors. When transplanted into immunocompetent mice, P organoids overexpressing Kras A146T formed solid tumors with distant metastases.

Conclusion : We developed a spontaneous metastatic gastric cancer tumoroid harboring a Kras A146T mutation, associated with enhanced tumor growth and metastasis, in immunocompetent mice. This model could facilitate investigations into the metastatic progression of gastric cancer and potential Kras-targeted therapeutic approaches.

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Keywords : Metastasis, Gastric cancer, Kras A146T, Tumoroid model

PS-D-51

KBN2201 ameliorates amyloid pathology, neuroinflammation, neuronal degeneration, and neurogenesis deficits in 9-month-old 5xFAD mice

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Alzheimer's disease (AD) is a progressive and currently incurable neurodegenerative disorder that affects millions of people worldwide. In this study, we evaluated the therapeutic potential of the compound KBN2201 [2-hydroxy-4-(trifluoromethyl)benzoic acid] in 5xFAD mice, a transgenic model of AD. Chronic oral administration of KBN2201 from 9 to 12 months of age significantly ameliorated AD-related pathologies. Treatment with KBN2201 reduced the expression of amyloid precursor protein (APP), APP C-terminal fragments (APP CTFs), and BACE1, resulting in a marked reduction in amyloid- β (A β) plaque number and size in both the cortex and hippocampus. Structural integrity in the hippocampal CA1 region was maintained, and dendritic complexity was preserved in cortical and hippocampal neurons. Furthermore, neurogenesis was enhanced in the subventricular zone (SVZ) and hippocampus, as indicated by increased DCX and Ki67 expression. Behavioral analysis using the Y-maze test demonstrated improved memory performance following treatment. Collectively, these findings suggest that KBN2201 exerts multifaceted therapeutic effects through anti-amyloidogenic, anti-inflammatory, neuroprotective, and neuroregenerative mechanisms, highlighting its potential as a promising candidate for AD treatment.

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Keywords : Alzheimer's disease (AD), 5xFAD mice, KBN2201, Multitarget drug

PS-D-50

SARS-CoV-2 immunization augments tumor-specific T cell cytotoxicity via TCMO6 antigen mimicry

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Background and aims : Emerging clinical observations suggest delayed cancer progression or regression following SARS-CoV-2 infection or vaccination; however, mechanistic insights remain limited for therapeutic translation. This study aimed to elucidate how viral immunization modulates tumor immunity and to uncover actionable mechanisms contributing to metastatic suppression and enhanced responsiveness to immunotherapy. **Results :** In murine melanoma and gastric cancer models, SARS-CoV-2 immunization suppressed metastatic colonization and improved responsiveness to anti-PD-1 therapy. Single-cell RNA sequencing of metastatic lung tissues revealed increased expression of cytotoxicity-related genes in CD8⁺ T cell lineages, alongside expanded TCR diversity and enrichment of tumor-associated T cell clones—indicative of enhanced tumor-specific cytotoxic T lymphocyte (CTL) responses. To identify genes involved in antigen mimicry, we conducted a BLASTP-based homology search using SARS-CoV-2 T cell epitopes, prioritizing genes with >60% sequence similarity, predicted MHC-I binding affinity, and tumor-associated expression. Functional relevance was assessed through an in vivo CRISPR knockout screen under immune pressure induced by viral immunization. Among the candidates, the tumor antigen TCMO6 exhibited over 65% sequence similarity with the SARS-CoV-2 nucleocapsid 219–228 peptide (LALLLDRL), supporting potential antigenic cross-reactivity. Tcmo6-overexpressing cancer cells significantly increased T cell activity, and in vivo, such tumors displayed greater reductions in metastatic burden following SARS-CoV-2 immunization. Therapeutically, administration of the nucleocapsid 219–228 peptide suppressed metastasis and was accompanied by increased CTL infiltration.

Conclusion : These findings demonstrate that SARS-CoV-2 immunization enhances tumor-specific CTL activity and promotes immune-mediated tumor suppression. Leveraging viral peptide-based immunization—via antigen mimicry exemplified by TCMO6—offers a promising strategy to improve cancer immunotherapy.

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Keywords : COVID-19, Cytotoxic T cells, Metastasis, Molecular mimicry

PS-E-01

Oscillatory and network-level changes following MPTP-induced Parkinsonism in a non-human primate

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Abnormal neural oscillations, particularly within the beta frequency band (13–30 Hz), have been widely implicated in the motor symptoms of Parkinson's disease (PD), such as bradykinesia and rigidity. However, the longitudinal evolution of these oscillatory dynamics within large-scale cortical networks remains insufficiently understood, especially in translationally relevant non-human primate (NHP) models. In this study, we employed chronic electrocorticographic (ECoG) recordings in a freely moving rhesus macaque to investigate cortical network changes before and after induction of parkinsonism using unilateral intracarotid administration of MPTP, a neurotoxin known to replicate PD-like pathology.

A subdurally implanted ECoG array over the primary motor cortex (M1) enabled high-resolution monitoring of cortical activity throughout behavioral and pharmacological states. Following MPTP treatment, the subject exhibited clear parkinsonian symptoms, including decreased spontaneous movement and motor rigidity. Spectral analysis revealed a marked increase in beta-band power in the lesioned hemisphere. Pairwise coherence analysis showed elevated beta coherence during the parkinsonian state (0.77 vs. 0.55 baseline), suggesting increased synchronization across the motor cortex. Similar trends were observed in alpha and gamma bands, potentially indicating reduced network flexibility.

Importantly, administration of L-DOPA resulted in partial behavioral recovery and normalization of beta coherence (0.59), reinforcing the potential of coherence metrics as modifiable biomarkers. These results underscore the utility of ECoG-based network analysis in capturing dynamic changes in PD and support its potential for informing neuromodulatory interventions.

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Keywords: Electrocorticography, Parkinson's Disease, Non-Human Primate Model, Beta Oscillations

PS-E-03

Microcurrent wave mitigates mouse intracranial arterial dolichoectasia development

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Objective: To investigate the therapeutic effect of microcurrent electrical stimulation in a mouse IADE model.

Methods: The study involved dividing twenty 8-week-old C57BL/6J male mice into five groups to investigate the effects of microcurrent on cerebral arteries after intracranial arterial dolichoectasia (IADE). Group 1 served as the control, Group 2 modeled IADE, Group 3 received preventative microcurrents, Group 4 received microcurrents starting after surgery for IADE treatment, and Group 5 evaluated the toxicity of microcurrents. After four weeks of treatment, the mice were euthanized for the analysis of arterial diameter, thickness, and composition using ImageJ, focusing on the proportions of smooth muscle cells, elastin, and collagen within specific arterial regions.

Results: The cerebral arterial diameter was significantly higher in group 2-D (117.79±17.05) compared to group 1-C (76.64±12.03), and group 3-M+D (77.29±24.47). Additionally, the cerebral arterial thickness in group 2-D (9.31±2.26) was significantly lower than in group 1-C (16.16±1.6), and group 3-M+D (15.67±2.86) (Figure 2, Table 1). The diameter of group 4-D+M (100.28±25.99) was lower than that of group 2 and the thickness of group 4-D+M (12.82±5.17) was higher than that of group 2-D although no significant difference was observed (Figure 2, Table 1). The proportion of SMC and elastin in the cerebral arterial wall was significantly lower in group 2-D (SMC: 38.05±10.32, elastin: 53.13±9.08) compared to group 1-C (SMC: 70.93±7.18, elastin: 53.13±9.08), group 3-M+D (SMC: 67.03±6.17, elastin: 47.22±8.73) and group 4-D+M (SMC: 70.45±9.35, elastin: 51.2±6.82), respectively. Additionally, the proportion of collagen in the cerebral arterial wall was significantly lower in group 2-D (42.46±14.12) compared to group 1-C (6.94±2.76), group 3-M+D (13.31±4.67), and group 4-D+M (13.3±3.84), respectively (Figure 2, Table 1) with no toxicity observed in the toxicity evaluation group (Group 5-M).

Conclusion: The study revealed that microcurrent is effective in preventing IADE development, although these beneficial effects warrant further investigation.

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Keywords: Intracranial Arterial Dolichoectasia (IADE), Microcurrent Therapy, Hypertension and Inflammation, Cerebral Arterial Wall Remodeling, Mouse Model

PS-E-02

White Ginseng ethanol extract inhibits tyrosinase and melanogenesis via MITF downregulation in B16F10 cells

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This study investigated the potential of white ginseng ethanol extract (WGEE), derived from the Geonsam variety of *Panax ginseng*, as a functional ingredient for skin whitening. UPLC analysis confirmed the presence of ginsenosides Rg1 (12.03 ppm) and Rb1 (37.59 ppm), while the total polyphenol and flavonoid contents were 33.11 ± 0.43 mg GAE/g and 33.33 ± 0.41 mg QE/g, respectively. WGEE showed strong antioxidant activity, with 56.95% DPPH and 43.97% hydrogen peroxide scavenging at 1,000 µg/mL, and noticeable effects starting at 40 µg/mL. In B16F10 melanoma cells, melanin production increased by 27.96% following α-MSH (100 nM) treatment, but this was reduced by 33.65% with WGEE (40 µg/mL). Western blot analysis revealed that WGEE downregulated α-MSH-induced expression of melanogenesis-related proteins such as MITF, tyrosinase, TRP-1, and TRP-2, exhibiting effects similar to arbutin. Molecular docking studies showed binding affinities of -8.1 kcal/mol for Rg1 and -6.2 kcal/mol for Rb1 with tyrosinase (PDB ID: 3NM8), suggesting direct inhibitory potential.

These results indicate that WGEE and its active compounds possess significant antioxidant and anti-melanogenic properties, supporting their use as safe and effective ingredients in functional cosmetic formulations for skin whitening.

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Keywords: Antioxidant, B16F10 Cell, MITF, Tyrosinase, White Ginseng

PS-E-04

Small Extracellular Vesicles derived From Feline Adipose Tissue Mesenchymal Stem Cells Enhance The Effects of Anti-Inflammation Compared to Fibroblasts-derived Small Extracellular Vesicles

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Adipose tissue-derived mesenchymal stem cells (AD-MSCs) release extracellular vesicles such as small extracellular vesicles (sEVs), apoptotic bodies, and microparticles. In particular, sEVs are formed inside cells via multivesicular bodies (MVBs), thus their protein, DNA, and RNA content are similar to those of the parent cells. sEVs research is rapidly expanding, with an increase in the number of related publications observed in recent years; therefore, the function and application of MSC-derived sEVs could emerge as cell-free therapeutics. sEVs were isolated from culture medium collected during culture of feline AD-MSCs using ultracentrifugation. Feline sEVs have been characterized by Fluorescence-Activated Cell Sorting (FACS), nanoparticle tracking analysis, and transmission electron microscopy imaging. Moreover, cytokine levels were detected by sandwich enzyme-linked immunosorbent assay in sEVs and LPS-induced THP-1 macrophages. The size of the isolated sEVs was that of a typical exosome, i.e., approximately 150 nm, and they expressed tetraspanins CD9 and CD81. The anti-inflammatory factor IL-10 was increased in feline AD-MSC-derived sEVs. However, pro-inflammatory factors such as IL-1β, IL-8, IL-2, RANTES, and IFN-γ were significantly decreased in feline AD-MSC-derived sEVs. This was the first demonstration that feline AD-MSC-derived sEVs enhance the inflammatory suppressive effects and have potential for the treatment of immune diseases or as an inflammation-inhibition therapy.

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Keywords: Small Extracellular Vesicles, Feline Adipose Tissue Mesenchymal Stem Cells, Fibroblasts, Anti-Inflammation

PS-E-05

The status of postapproval monitoring operation by the Institutional Animal Care and Use Committee in Korea

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Postapproval monitoring (PAM) is a critical component of Institutional Animal Care and Use Committee (IACUC) oversight, ensuring compliance with approved protocols and ethical animal research practices. While the PAM is not explicitly mandated under U.S. federal regulations, it has been widely recognized as an essential mechanism for verifying adherence to animal welfare standards. In contrast, Korea has formally integrated the PAM into its legal framework, making it a mandatory function of IACUCs. This study examines the implementation and perception of the PAM in Korean institutions through a survey of relevant professionals. These findings indicate that while awareness of the PAM is high, challenges such as limited manpower and institutional support hinder its effective execution. Additionally, the pandemic highlighted the potential for online remote monitoring as a supplemental method, although concerns remain regarding its effectiveness in assessing real-time animal welfare conditions. The study suggests that a hybrid PAM model, that combines onsite and remote monitoring, could improve oversight efficiency while addressing resource constraints. Strengthening administrative support, increasing professional staffing, and enhancing researcher training are crucial steps for optimizing PAM operations in Korea. These insights contribute to the broader discourse on the evolution of animal research oversight and the need for adaptable monitoring strategies in diverse regulatory environments.

Jimin Lee and Na Ahn contributed equally to this work.

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Keywords: Animal ethics, Animal welfare, IACUC, Postapproval monitoring

PS-E-06

Eradication of *Aspiculuris tetraptera* in Various Immunodeficient Mouse Models Using Ivermectin: A Case Report

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Despite advancements in laboratory animal facility management, pinworm infections remain a persistent issue in immunodeficient mouse colonies. Rapid diagnosis and treatment are crucial to mitigating potential scientific and economic consequences. Effective control requires both the administration of anthelmintic agents and rigorous environmental decontamination. However, the safety and efficacy of these treatments in genetically modified mouse models remain under-evaluated. In this report, *Aspiculuris tetraptera* infestation was identified in multiple lines of immunodeficient mice housed in a laboratory animal facility. Diagnosis was confirmed via fecal flotation for egg detection and necropsy for adult worm identification in the large intestines. To treat the infestation, mice received three subcutaneous ivermectin injections (1 mg/kg) at two-week intervals, coupled with weekly ivermectin spray application for four consecutive weeks. The facility was additionally disinfected using commercial bleach. Following the treatment regimen, fecal and postmortem examinations confirmed the complete elimination of *A. tetraptera* eggs and adult worms from all mouse colonies, with no recurrence observed for 50 weeks. No treatment-related mortality or adverse effects were observed. This combined approach—subcutaneous ivermectin injections and ivermectin spray—proved effective and safe for eradicating *A. tetraptera* in immunodeficient mouse colonies. It offers a practical strategy for managing pinworm infections in genetically modified models.

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Keywords: *Aspiculuris tetraptera*, Ivermectin, Immunodeficient mouse colonies, Facility management, Pinworm

PS-E-07

Nanographene oxide attenuates acute GVHD by modulating macrophage polarization in a xenogeneic mouse model

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Nanographene oxides (NGOs) exhibit potent immunomodulatory properties, making them promising therapeutic candidates for graft-versus-host disease (GVHD). Using a xenogeneic GVHD (xGVHD) mouse model, we found that NGO administration improved survival, mitigated weight loss, reduced human leukocyte engraftment, and lowered GVHD pathological scores. In GVHD patient-derived PBMCs, NGO treatment increased the proportion of naïve T cells while reducing effector memory T cells, suggesting a shift toward immune homeostasis. RNA sequencing of GVHD patient PBMCs revealed that NGOs selectively downregulated antigen presentation and inflammatory pathways while having no significant effects in nonGVHD PBMCs. Further transcriptomic analysis demonstrated that NGOs inhibited the IFN γ -STAT1 signaling axis, suppressing M1 macrophage polarization and enhancing an anti-inflammatory phenotype. To address safety and translational concerns, we developed NGO-primed macrophages (NGO-Macs) as a cell-based therapy. NGO-Macs exhibited an M2-like phenotype, suppressed T cell activation, reduced Th1 differentiation, and enhanced Treg induction. *In vivo*, NGO-Mac therapy reduced M1 macrophage infiltration, increased Treg frequencies, and significantly alleviated GVHD pathology. These findings highlight NGOs and NGO-Macs as promising GVHD immunotherapies by selectively regulating innate and adaptive immune responses. While NGOs demonstrate strong immunomodulatory potential, further studies on long-term safety, biodistribution, and clinical feasibility will be essential for therapeutic translation.

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Keywords: Nanographene oxide, Macrophage, Humanized mouse, Graft versus host disease, Nanomedicine

PS-E-08

Extraction and Characterization of Polyphenols from *Gynura procumbens* (sambungcho) using HPLC-ESI-MS/MS

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Cancer is one of the major public health concerns in both developing and developed countries. Currently, the available treatments for cancer are inadequate and there is an urgent requirement to identify novel therapeutic agents that can decrease the mortality of cancer patients with fewer side effects. Due to the side effects of chemical drugs, the world is interested in developing safe and non-toxic drugs from natural plants. In the current study, we used *Gynura procumbens* commonly known as sambungnyawa (eternal life) which has shown to exhibit anti-diabetic effect in Korean traditional medicine. The polyphenolic mixture was isolated from leaf and stem of *Gynura procumbens* by methanol extraction method and each polyphenolic compound was analyzed using high-performance liquid chromatography coupled with tandem mass spectrometry (HPLC-ESI-MS/MS), and compared with the reported data. The results revealed the total mass value and the divided mass value of each compound. HPLC data shown difference in the set of polyphenols, when compared to leaf and stem extracts. A total of 16 compounds were identified in the leaves, and main compounds were traced after 40 minutes of retention time (RT). But, in case of stem, 15 compounds were identified with main compounds traced before 40 minutes of RT. The obtained list of polyphenols may be considered to have physiological activities such as antioxidant, anti-inflammatory, anti-cancer effects by the previously reported studies. Further *In-vitro* and *In-vivo* investigation needed to confirm these physiological activities of obtained polyphenols from *Gynura procumbens*.

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Keywords: Natural plant, HPLC, Polyphenols, Mass spectrum

PS-E-09

Automated radiosynthesis and efficacy evaluation of tau target diagnostic PET tracer candidate, 2-(6-(2-(2-([¹⁸F]-fluoro)ethoxy)ethoxy)-1H-pyrrolo[3,2-c]pyridin-1-yl)quinoline

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Alzheimer's disease (AD) is neurodegenerative disorder characterized by abnormal tau protein aggregation. Several tracers, such as [¹⁸F]florbetapir, [¹⁸F]flutemetamol, and [¹⁸F]florbetaben, have received FDA approval for amyloid-beta imaging. In contrast, [¹⁸F]T807 (also known as flortaucipir or AV-1451) remains the only first-generation tracer approved by the FDA for tau imaging. To advance diagnostic development, multiple second-generation tracers have been developed. In this study, we developed a novel compound, 2-(6-(2-(2-([¹⁸F]fluoro)ethoxy)ethoxy)-1H-pyrrolo[3,2-c]pyridin-1-yl)quinoline ([¹⁸F]II-25), attempted its automated radiolabeling synthesis, and evaluated its efficacy.

The II-25 precursor was stirred with [¹⁸F]fluoride and purified. For automated labeling, the established by manual radiolabeling protocol was applied using the sCUBE module. Tau-specific binding was evaluated in brain samples and further assessed by in silico molecular docking. In vivo PET/CT imaging was performed in Tg(Pmp-MAPT*P301L) JNPL3HImc transgenic mice.

The radiochemical yield was 12.6% for manual and 17.6% for automated labeling. In vitro studies showed that II-25 inhibited prototype compound uptake by 88.68% to 99.97%. In silico analysis predicted a binding energy of -9.062 kcal/mol for II-25. PET/CT imaging showed higher mean brain uptake in the transgenic group than in controls, with maximum standardized uptake value at T_{max} of 11.32 ± 2.19 in transgenic mice versus 8.87 ± 2.11 in controls.

Automated synthesis of II-25 demonstrated radiochemical results comparable to manual labeling, supporting its suitability for large-scale production with reduced radiation exposure. Tau-specific binding was confirmed in brain tissue and further validated by molecular docking. In vivo PET/CT imaging showed higher [¹⁸F]II-25 uptake in tau transgenic mice than in controls. Taken together, these results suggest that [¹⁸F]II-25 is a promising radiotracer for tau pathology imaging in AD.

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Keywords : Alzheimer's disease, Tau, PET tracer, Automated radiosynthesis

PS-E-10

Identification of new odor biomarkers associated with aging in aged ICR mice

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Most studies on the identification and characterization of age-related odors have been conducted on humans. Even though experimental animals play a crucial role in the study of biological systems in scientific research, studies on age-related odors in animal models are rare. To characterize the odor markers associated with aging in mice, alterations in the concentration of odorants in the urine, expression of metabolic enzymes, the histopathological structure of the sweat glands, and the expression of regulatory factors in sweat secretion were analyzed in ICR mice of four different ages (2, 6, 8 and 10-months-old). The aging of the ICR mice was confirmed by the shortening of telomere length and increase in ammonia concentrations. The concentrations of 15 odorants including ethylenimine and trimethylamine (TMA), and total volatile organic compounds (VOCs) were significantly higher in the urine of 8- and 10-month-old ICR mice when observed in gas chromatography-mass spectrometry (GC-MS) analyses. Among them, TMA was selected as an important odor biomarker associated with aging in the current study based on the results of aging-related TMA upregulation and a higher fish and ammonia-like odor. Also, the increase in TMA concentrations was reflected in the transcription levels of the gene encoding TMA monooxygenase in the livers and the feet of mice. Furthermore, tissues obtained from mouse feet showed an age-dependent increase in the lumen area and secretory coiled portion of the sweat glands, while the transcriptional level of the genes encoding sweat secretion regulators was partially increased in an age-dependent manner. These results provide novel scientific evidence that the age-dependent changes in the volatiles in the urine are indicative of aging-related odors in ICR mice. Specifically, the study shows that TMA has potential as a novel diagnostic odor biomarker in aging ICR mice.

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Keywords : Aging, Odor, VOCs, Trimethylamine, Sweat gland

PS-E-11

Enhanced oocyte yield in common marmoset assisted reproductive technology by switching from hMG to rFSH

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The common marmoset is a nonhuman primate with early sexual maturation and high fertility, making it a valuable model for generating genetically modified (GM) marmoset. However, no GM marmoset has been developed in Korea yet. At SNUH-MMRB, we have been optimizing assisted reproductive technology (ART) conditions to produce TG marmosets exhibiting human-like disease phenotypes. In human ART, recombinant human FSH (follicle-stimulating hormone) is typically used in patients with normal ovarian response, while human menopausal gonadotropin (hMG) is used to those with poor ovarian response. Initially, we used hMG following the ovarian stimulation protocol of the research group that first succeeded in generating TG marmosets in 2009. However, meta-analyses in human clinical studies reported that pure FSH alone leads to higher oocyte retrieval in general populations, excluding specific cases such as severe LH deficiency or hypo-responders. Based on this evidence, we switched from hMG to follicle-stimulating hormone and evaluated outcome changes. We compared the number of available oocytes (AO) per cycle for in vitro maturation (IVM) between groups using different gonadotropins. In the second cycle, the group switched to follicle-stimulating hormone showed a higher number of retrieved oocytes (15.3±8.0 vs. 11.2±5.9). Furthermore, when comparing the mean difference in oocyte yield between the first and second ovum pick-up (OPU) cycles, the group that continued with hMG showed a statistically significant decrease in the second cycle (p = 0.01). These findings suggest that switching from hMG to follicle-stimulating hormone may enhance oocyte retrieval for TG marmoset production. This study provides foundational evidence to improve the efficiency of TG marmoset development and supports further refinement of ART protocols for nonhuman primate models.

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Keywords : Assisted reproductive technology (ART), Ovarian stimulation protocol, Human menopausal gonadotropin (hMG), Recombinant human FSH (Follicle-stimulating hormone), Common marmoset (*Callithrix jacchus*)

PS-E-12

Amide proton transfer CEST MRI for detecting neuroinflammation in a sepsis-associated encephalopathy animal model

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Sepsis-associated encephalopathy (SAE) is characterized by neuroinflammatory changes and cognitive dysfunction in the absence of direct CNS infection. This study aimed to investigate molecular and physiological changes in the hippocampus using amide proton transfer-weighted (APT_w) chemical exchange saturation transfer (CEST) imaging and multi-parametric MRI (mpMRI) in a rat model of lipopolysaccharide (LPS)-induced SAE. A total of 21 rats were assigned to three groups: control (CTRL), SEPS05 (5 mg/kg LPS), and SEPS10 (10 mg/kg LPS). All animals underwent 7T MRI 24 hours after LPS or vehicle injection. APT_w imaging demonstrated significantly increased signal intensities in both SEPS05 and SEPS10 groups compared to controls, suggesting elevated mobile amide proton levels associated with neuroinflammatory processes. Consistently, mpMRI revealed significantly increased apparent diffusion coefficient (ADC) and T1 relaxation values in the hippocampus, indicative of vasogenic edema and blood-brain barrier disruption. CBF and T2 values did not show significant changes. These results highlight the value of APT_w and mpMRI techniques in detecting SAE-related neuroinflammatory alterations. Although APT_w signals can be influenced by T1 and water content, the concurrent increases in APT_w, ADC, and T1 support the interpretation that the observed contrast changes reflect true molecular and structural injury. The integration of APT_w CEST imaging with mpMRI provides a sensitive and non-invasive approach for visualizing early brain injury in SAE, offering potential for preclinical evaluation of therapeutic interventions.

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Keywords : Amide Proton Transfer CEST, Neuroinflammation, Sepsis-Associated Encephalopathy, MRI, Molecular Imaging

PS-E-14

Bioengineered liver scaffolds functionalized with heparin improve endothelialization and attenuate liver fibrosis

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Liver fibrosis, characterized by excessive extracellular matrix (ECM) deposition, progressively disrupts hepatic architecture and function, leading to end-stage liver disease (ESLD). While liver transplantation remains definitive treatment, donor scarcity necessitates alternative therapeutic strategies. Bioengineered livers based on decellularized liver scaffolds (DLS) show promise; however, the lack of a functional endothelium often results in thrombosis and poor graft integration. This study aimed to develop heparinized decellularized liver scaffolds (HEP-DLS), to enhance endothelialization and angiogenesis, thereby enhancing therapeutic outcomes in fibrotic liver models. Rat livers were decellularized by perfusing Triton X-100/ammonium hydroxide. Heparin was covalently immobilized onto the scaffolds to promote endothelialization, angiogenesis, and cellular regeneration. These heparinized scaffolds were recellularized with human umbilical vein endothelial cells (HUVECs) and culture in bioreactor for 7 days. To assess angiogenic potential, acellular HEP-DLS were implanted into the omentum of mice for 21 days. Therapeutic efficacy was assessed by implanting recellularized HEP-DLS into the interlobular space of a thioacetamide-induced liver fibrosis mouse model. Heparin immobilization was confirmed by FTIR and Toluidine blue staining. Histological analysis of the heparinized recellularized group revealed the formation of a uniform endothelial cell lining within the vessels and vascular coverage (87 ± 10.71%) compared to controls (38.43 ± 10.51%). In vivo implantation confirmed the superior angiogenic potential of HEP-DLS, showing enhanced neovascularization indicating strong biocompatibility. In the liver fibrosis mouse model, implantation of HEP-DLS recellularized tissue resulted in reduced serum ALT/AST levels and histological evidence of liver regeneration and ECM remodeling.

Heparin-functionalized liver scaffolds promote endothelialization, angiogenesis and significantly improve tissue regeneration in fibrotic livers. These findings support the therapeutic potential of bioengineered liver constructs for addressing ESLD. This research was supported by Korea Institute of Planning and Evaluation for Technology in Food, Agriculture and Forestry (IPET); Ministry of Agriculture, Food and Rural Affairs (321027-5) and Korean Fund for Regenerative Medicine (KFRM) grant No. 22A0101L1-11.

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Keywords : Liver bioengineering, Heparin, Re-endothelialization, Angiogenesis, Fibrosis

PS-E-13

Mouse-Derived Cell Powder Technology Enhances Myogenic Differentiation and Alignment on Alginate-Zein Scaffold

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Effective muscle regeneration requires not only the proliferation and differentiation of myogenic cells but also their alignment within a supportive 3D microenvironment. In this study, we present a novel approach of bioactive cell powder derived from murine cells to enhance myogenesis and tissue organization in 2D and 3D cultures. Myotube Powder (MP) was prepared from differentiated C2C12 myotubes, while Adipocyte Powder (AP) was from adipogenically induced mouse preadipocytes. These powders were incorporated into culture media and alginate-zein (AZ) scaffolds to investigate their effects on C2C12 viability, differentiation, and alignment. In 2D culture, MP at 0.1 µg/µL significantly upregulated myogenic genes (Pax7, Myod1, Cdk1, and Myoz1) and enhanced desmin myotube alignment. The combination of AP and MP at 1:1 ratio promoted structural organization without cytotoxicity. In 3D, AZ scaffolds supplemented with AP:MP powder (AZ(AP:MP)) demonstrated superior bioactivity, with scanning electron microscopy revealing improved fiber alignment. FTIR analysis confirmed molecular integration of bioactive components into the scaffold. Immunofluorescence staining of AZ(AP:MP) showed organized expression of Myosin, indicating advanced differentiation and cytoskeletal organization. Myotubes aligned concentrically around the scaffold's inner structure. Furthermore, metabolomic profiling via heatmap revealed that AZ(AP:MP) displayed biochemical signatures more closely resembling native beef than plain scaffolds, suggesting enhanced tissue mimicry. These findings highlight the potential of murine-derived cell powders as low-cost, bioactive supplements to improve muscle tissue engineering. This platform holds translational promises for applications in regenerative medicine, artificial meat production, and the development of animal-model-based biomaterials. This work was supported by the Korea Institute of Planning and Evaluation for Technology in Food, Agriculture and Forestry (IPET) through the High Value-added Food Technology Development Program, funded by the Ministry of Agriculture, Food and Rural Affairs (MAFRA) (321027-5) and the Korean Fund for Regenerative Medicine (KFRM) grant No. 22A0101L1-11.

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Keywords : Alginate, Adipocyte, C2C12 myoblast, Cell powder, Zein

PS-E-15

Sphingosine-1-Phosphate enhances endothelialization of rat decellularized liver scaffolds by promoting endothelial cell function

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Endothelialization is crucial for tissue bioengineering, particularly in developing functional blood vessel linings to ensure proper vascularization. Effective re-endothelialization of the vasculature in bioengineered organs is challenging, often leading to blood coagulation and hindering successful engraftment. Endothelial cell proliferation, migration, and angiogenesis are essential processes for constructing functional, vascularized bioengineered organs. Sphingosine-1-phosphate (S1P), a low molecular weight phospholipid mediator, regulates various biological activities in endothelial cells.

In this study, we present a novel approach to enhance the re-endothelialization of decellularized rat liver scaffolds (DLS) by seeding Human Umbilical Vein Endothelial Cells (HUVECs) in the presence of S1P, aiming to bioengineer a fully endothelialized liver. HUVECs were seeded in the presence of S1P into DLS and maintained in perfusion bioreactor for 7 days. Histological and molecular analyses were performed to evaluate endothelial coverage and integrity of recellularized scaffolds.

S1P treatment enhanced HUVEC re-endothelialization of liver scaffolds, achieving 80.44 ± 7.38% vascular coverage compared to 33.07 ± 5.4% in controls. Immunofluorescence showed increased CD31 and ZO-1 expression, indicating improved endothelial integrity and tight junction formation. Resazurin reduction assays revealed higher metabolic activity in S1P-treated scaffolds. qPCR analysis showed significant upregulation of endothelial genes NOS3, VE-cadherin and VEGF in S1P-treated constructs. Additionally, thrombogenicity assays demonstrated reduced thrombus formation with fewer platelet aggregates in S1P scaffolds.

Our results indicate that S1P is a key regulator of endothelialization processes, promoting HUVEC proliferation, survival, and facilitating the formation of a functional endothelial layer on the vascular structure of re-endothelialized liver scaffold. These findings highlight the pivotal role of S1P as a promising approach for generating vascularized bioengineered livers suitable for transplantation. This research was supported by the Korea Institute of Planning and Evaluation for Technology in Food, Agriculture and Forestry (IPET); Ministry of Agriculture, Food and Rural Affairs (321027-5); and Korean Fund for Regenerative Medicine (KFRM) grant No. 22A0101L1-11.

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Keywords : Bioengineered Liver, S1P, HUVEC, Re-endothelialization, Proliferation

PS-E-16

Current Status of Animal Experimentation and IACUC Operations in Domestic Animal Experimentation Institutions in 2024 in South Korea

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This study investigates the structure and functioning of Institutional Animal Care and Use Committees (IACUCs) established in accordance with Article 51 of the Animal Protection Act. Utilizing annual performance reports submitted to the Animal and Plant Quarantine Agency, the analysis aims to provide foundational data for improving laboratory animal welfare in Korea.

In 2024, a total of 556 institutions—including industrial facilities (308), universities (129), public organizations (81), and medical institutions (38)—reported having IACUCs in operation. These committees consisted of 3,222 members, averaging approximately 5.79 members per committee. Over the year, the IACUCs reviewed 43,090 animal research proposals, with 5.8% either returned for revision or not approved. The most common reasons for rejection included unjustified animal usage numbers, improper experimental design, and inadequate consideration of animal pain and distress.

The total number of animals used in research was 4,592,958. Of these, 78.6% were used in legally regulated testing (40.5%) and basic scientific research (38.1%), indicating the predominance of these two categories in domestic animal experimentation.

The outcomes of this study are intended to support future policy development in the field of laboratory animal welfare. Moreover, the findings will contribute to designing targeted education and training programs for IACUC members, ensuring the committees are better equipped to evaluate ethical and scientific standards in animal research. This will help advance a more humane and accountable research environment across institutions in Korea.

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Keywords: Animal Experiment, IACUC, Animal Protection Act

PS-E-17

Genipin and VEGF Synergize to Support Recellularization and Angiogenic Remodeling in Bioengineered Rat Liver Scaffolds

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A major turning point in liver tissue engineering is the creation of physiologically realistic bioengineered liver scaffolds able to simultaneously support the adhesion, survival, and functional integration of hepatic parenchymal cells and vascular endothelial cells. Replicating the intricate structure and perfusion-dependent function of natural liver tissue requires this dual-lineage support, which enhances the translational potential of synthetic grafts for use in research and therapy. In this work, we used HepG2 and EA.hy926 cells to examine the synergistic effects of genipin crosslinking and VEGF stimulation on the recellularization of decellularized rat liver scaffolds. Both cell lines were planted in a 2D environment to assess cellular reactions. To enhance scaffold integrity, rat livers were decellularized and subsequently crosslinked with 0.1% genipin, followed by VEGF stimulation throughout the scaffold. Cell viability assays confirmed that genipin did not impair survival. VEGF treatment (0.01%) significantly enhanced EA.hy926 proliferation, indicating a positive angiogenic response. Hematoxylin and eosin (H&E) staining of seeded scaffolds showed visible cell attachment in vascular and parenchymal regions. Gene expression analysis (RT-qPCR) revealed upregulation of albumin (ALB) and VEGFA in VEGF-treated conditions, indicating enhanced hepatic function and angiogenic signaling. These findings suggest that genipin-crosslinked liver scaffolds provide a supportive environment for dual-lineage recellularization, and that VEGF enhances endothelial integration and remodeling. This platform may contribute to the development of vascularized bioengineered liver constructs, with further validation underway in 3D and perfusion-based systems.

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Keywords: Genipin, VEGF, Liver, Scaffolds, Cells

PS-E-18

Compound X, a Resorcinol derivative, alleviates IgE-mediated allergic inflammation by suppressing mast cell activation

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Mast cells, as rapid responding early effector cells, play a central role in the initiation and progression of allergic reactions and various inflammatory diseases. Among potential therapeutic agents, resorcinol derivatives have shown dermatological, antiseptic, and anti-inflammatory activities in previous studies. In this study, we investigated the effects of various synthesized resorcinol derivatives using cell-based screening systems designed to target mast cells. After screening, Compound X was evaluated *in vivo* using mast cell-mediated allergic diseases models, such as ovalbumin (OVA)-induced allergic asthma and passive cutaneous anaphylaxis (PCA). This study demonstrated that Compound X attenuated asthmatic symptoms in the OVA-induced allergic asthma model. These symptoms included immune cell infiltration, increased levels of total and OVA-specific immunoglobulin (IgE) in serum and bronchoconstriction. Furthermore, Compound X demonstrated anti-inflammatory effects by reducing levels of type 2 cytokines (IL-4, IL-5, IL-6 and IL-13) and inflammatory mediators, such as eosinophil peroxidase (EPO) and myeloperoxidase (MPO), in bronchoalveolar lavage fluid and lung tissues. Mast cell activation was reduced in the lungs of asthmatic mice. *In vitro* assays, the efficacy of Compound X was confirmed by evaluating IgE-mediated allergic responses in mast cells. Compound X inhibited degranulation, as evidenced by decreased release of histamine and β -hexosaminidase, and suppressed the production of inflammatory cytokines (IL-4, IL-6, IL-13, and TNF- α) in activated mast cells. These results were associated with the suppression of PI3K/Akt signal transduction and regulation of transcription factors, including NF- κ B and NFAT. Building upon these *in vitro* findings in mast cells, our subsequent *in vivo* analysis using PCA model further confirmed that Compound X significantly alleviated IgE-mediated allergic responses. In conclusion, our results suggest that Compound X is a promising candidate for the treatment of allergic diseases by suppressing mast cell activation

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Keywords: Allergic asthma, Mast cell, Resorcinol, Anaphylaxis

PS-E-19

Tumor-priming CD8⁺ NKT-like cell therapy eliminates tumor and immunosuppressive cells in gastric cancer peritoneal metastasis

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Tumor-priming CD8⁺ NKT-like cell (TPNC) demonstrated potent antitumor activity against hematologic malignancies in our previous study. To explore its potential in solid tumors, we generated TPNC using ascitic fluid from patients with advanced gastric cancer (GC), a microenvironment enriched with stromal complexity and immunosuppressive cell populations. TPNC maintained stable expansion and exhibited upregulated gene expression related to cytolytic function, cell cycle progression, viability, and movement. Functional assays showed enhanced migration compared to cytokine-induced killer (CIK) cells, along with superior cytotoxicity and resistance to exhaustion upon repeated tumor exposure. Exhaustion markers remained low after rechallenge, indicating sustained function. *In vivo*, TPNC suppressed tumor progression and showed increased persistence in a subcutaneous xenograft model, confirming systemic antitumor activity. In a peritoneal carcinomatosis model, TPNC reduced disseminated tumor nodules without ascites accumulation. TPNC also selectively eliminated tumor-associated macrophages (TAMs) and myeloid-derived suppressor cells (MDSCs) in ascites, while sparing normal cells. T cell receptor (TCR) sequencing showed clonal enrichment and biased TRBV usage, and HLA-blocking assays confirmed MHC-restricted killing of immunosuppressive targets. These findings suggest that TPNC can overcome immunosuppressive barriers and mediate robust antitumor responses in solid tumors, supporting its translational potential for gastric cancer.

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Keywords: Gastric cancer peritoneal metastasis, Cellular therapy

PS-E-20

Effectiveness Evaluation using Animal Models for International Certification of Dental Medical Devices

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As public interest in and awareness of oral health improves, regulatory barriers to approval of related medical devices have also increased. We have designed specialized animal models to support the domestic and international approval of dental medical devices. In this presentation, we would like to introduce specialized animal models and studies to support the domestic and international approval of dental medical devices. For the evaluation of dental medical devices in the mandible of beagles, premolars were extracted and then beagles were spontaneously healed for about 12 weeks. Depending on the kinds of devices, other types of tooth extraction and application surgeries were performed. The effectiveness of the dental medical devices was confirmed through qualitative and quantitative evaluation of biocompatibility, reconstruction function of defective tissue, osteogenesis/osteointegration/osseointegration ability, and foreign body reaction using computed tomography (CT) scan, micro-CT analysis, and histopathological analysis. Our established preclinical evaluation methods for dental medical devices are expected to contribute to dental medical device research and development, as well as to shorten the product development time and overseas expansion of related companies.

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Keywords : Dental medical device, Preclinical research, Dental implant, Bone graft

PS-E-22

RNA Sequencing Analysis of Embryos Exposed to Nanoplastics: Impacts on Transcriptomic Alteration

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Nanoplastics (NPs), as emerging environmental contaminants, are drawing increasing concern due to their potential risks to ecosystems and human health. To investigate their impact on preimplantation embryo development, mouse 1-cell zygotes were treated with NPs, resulting in 2-cell or 4-cell stage arrest and delayed blastocyst formation. To further elucidate the underlying mechanisms, fertilized zygotes were exposed to 0, 0.1, and 1 mg/mL concentrations of NPs, followed by RNA-sequencing analysis at the 4-cell stage. Differential gene expression and pathway analyses were conducted to interpret the transcriptomic data. Results revealed 0.1 mg/mL exposure activated DNA damage repair processes, nuclear structural components, and transcriptional regulators, suggesting a stress response aimed at maintaining embryonic viability. In contrast, 1 mg/mL NPs suppressed pathways related to embryonic development, intracellular transport, and protein-DNA interactions, implying impaired metabolism and transcription. KEGG pathway enrichment showed activation of DNA repair and autophagy-related pathways at 0.1 mg/mL, while 1 mg/mL exposure inhibited developmental signaling (e.g., Notch) and enhanced ATR-mediated stress response. PPI and MCODE analysis revealed that low-dose NPs induced networks involved in cell survival and gene regulation, whereas high-dose exposure shifted the network toward apoptosis-related protein clusters. These results suggest dose-dependent transcriptomic disruptions, with high NP levels leading to impaired development and cell death. These findings suggest that NP exposure disrupts early embryonic development by inducing specific transcriptional alterations. This study provides new insight into the potential developmental toxicity of nanoplastics and advances our understanding of their biological effects in mammalian embryos.

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Keywords : Pre-implantation embryo development, Nanoplastics, RNA Sequencing

PS-E-21

Recombinant human IL-15 enhances expansion of NK cells from Non-human primates

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Non-human primates (NHPs), which share approximately 98% genetic homology with humans, are increasingly recognized as essential experimental models in biomedical research. Their close physiological, behavioral, and immunological similarities to humans make them especially valuable for modeling human diseases. Unlike murine models, NHPs possess an immune system that more closely resembles that of humans, allowing the development of disease models for conditions such as human acquired immune deficiency syndrome (AIDS) and hepatitis. As such, NHPs play a critical role in advancing our understanding of disease pathophysiology and in supporting the development of effective vaccines and therapeutics against infectious diseases. Among immune cells, natural killer (NK) cell is a key component of the innate immune system. They are responsible for identifying and eliminating abnormal or virus-infected cells through cytolytic mechanisms involving perforin and granzymes. Additionally, NK cells have been shown to participate in adaptive immune responses through the development of immunological memory. Despite their importance, the ex vivo expansion of NHP-derived NK cells has traditionally relied on recombinant human interleukin-2 (rhIL-2). However, NK cells from NHPs often show limited activation and proliferation under low concentrations of rhIL-2, posing a challenge for experimental applications. In this study, we evaluated recombinant human interleukin-15 (rhIL-15) as an alternative cytokine for the expansion of NHP NK cells. IL-15 shares receptor components with IL-2, including the common gamma chain (γ c) and IL-2/IL-15 receptor beta (CD122), and both activate the JAK-STAT signaling pathway. Given the well-established role of IL-15 in promoting human NK cell expansion, we speculated that it might exert similar effects in NHPs. Our results demonstrate that rhIL-15 promotes the in vitro expansion of NHP NK cells, offering a robust alternative to rhIL-2 and providing a foundation for future NK cell-based immunological studies using NHP models.

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Keywords : Non-human primate (NHP), Peripheral blood mononuclear cell (PBMC), Immune cell, NK cell

PS-E-23

Lipolytic effects of edible vitalmelon extract in MDI-stimulated adipocytes through activation of glycerol release and β -oxidation pathways

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Background and Objective : Vitalmelon, a hybrid fruit developed in Korea, has shown anti-obesity potential primarily through suppression of adipogenesis and lipogenesis in adipocytes and animal models. However, its effects on lipolysis—the breakdown of stored lipids—have not been well characterized. This study aims to evaluate the lipolytic effects of edible vitalmelon fruit water extract (VW) and investigate its underlying molecular mechanisms in differentiated adipocytes.

Methods : To evaluate the lipolytic activity of VW, we administered the extract to 3T3-L1 adipocytes differentiated with MDI (MiSt-3T3 cells) and assessed free glycerol release, intracellular cAMP concentrations, and the phosphorylation status of hormone-sensitive lipase (HSL). Additionally, we investigated the expression of β -oxidation-related genes such as ACADs, ACO1, and CTP, as well as the regulatory activity of PPAR α .

Results : Successful adipogenic differentiation was validated by increased Oil Red O staining and PPAR γ mRNA levels. Upon VW treatment, there was a dose-dependent increase in glycerol release and intracellular cAMP levels, accompanied by enhanced HSL phosphorylation. VW also upregulated both mRNA and protein levels of β -oxidation regulators (ACADs, ACO1, and CTP), while reducing ATPCL phosphorylation. These molecular changes were closely linked to enhanced expression and phosphorylation of PPAR α , suggesting a regulatory role in fatty acid oxidation. These effects of VW observed in adipocytes are now being verified in animal models with obesity.

Conclusion : Our findings indicate that vitalmelon water extract enhances lipolytic activity in MiSt-3T3 adipocytes by activating the cAMP-HSL signaling axis and promoting β -oxidation through PPAR α -mediated pathways. This suggests VW may have potential as a functional food component targeting obesity via the stimulation of lipid catabolism.

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Keywords : Vitamelon, Lipolysis, Adipocytes, B-oxidation

PS-E-24

A novel F-18 labeled pRIPK1-selective PET radiotracer for noninvasive imaging of necroptosis: utility in preclinical disease models

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Necroptosis is a form of programmed cell death distinct from necrosis and is mediated by death receptor signaling pathways involving receptor-interacting protein kinases 1 and 3 (RIPK1 and RIPK3). Among these, phosphorylated RIPK1 (pRIPK1) serves as a critical regulator of necroptotic signaling and has been shown to be effectively inhibited by agents such as necrostatin-1. Despite its central role, molecular imaging studies targeting pRIPK1 remain extremely limited, and imaging efficacy also remains a significant challenge. In this study, we developed a novel positron emission tomography (PET) radiotracer, [¹⁸F]RIPA56-3-F, designed to selectively bind pRIPK1, and evaluated its potential as a biomarker for necroptosis in preclinical models of diseases where necroptosis is implicated, including acute drug-induced liver injury (DILI), dry age-related macular degeneration (AMD), and osteoarthritis (OA). The radiotracer was derived from RIPA-56, a RIPK1 inhibitor with an EC₅₀ (effective concentration) of approximately 13 nM, through structure-activity relationship (SAR) study, and subsequently labeled with fluorine-18. [¹⁸F]RIPA56-3-F exhibited a binding affinity of 82 nM and demonstrated high radiochemical stability in human serum for over 4 hours. Biodistribution and pharmacokinetic profiles were assessed in normal ICR mice via dynamic PET imaging over 90 minutes. The tracer showed rapid and high uptake in most tissues, excluding the stomach and muscle, followed by clearance predominantly through the intestinal tract. In an acetaminophen-induced DILI model, hepatic uptake of [¹⁸F]RIPA56-3-F was approximately 1.8-fold higher compared to controls (3.48 ± 0.92 vs. 1.98 ± 0.51 %ID/g). In the dry AMD model induced by intraperitoneal sodium iodate, ocular uptake increased 2.1-fold (3.40 ± 0.46 vs. 1.60 ± 0.25 %ID/g), while in the OA model induced by monosodium iodoacetate (MIA), joint uptake was 1.6-fold higher (2.10 ± 0.07 vs. 1.34 ± 0.11 %ID/g) than in the respective controls. In addition, the tissue of interest was extracted after PET imaging and the pRIPK1 expression level was evaluated. Tissue analysis confirmed elevated pRIPK1 expression in affected organs, which correlated with PET signal intensity, supporting the radiotracer specificity. These findings demonstrate that [¹⁸F]RIPA56-3-F enables in vivo visualization and quantification of necroptosis by targeting pRIPK1. This novel radiotracer shows strong potential as a noninvasive imaging tool for the diagnosis of necroptosis-related diseases and may serve as a valuable platform for future translational research in necroptosis.

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Keywords : Necroptosis, Positron emission tomography, Drug-induced liver injury, Dry macular degeneration, Osteoarthritis

PS-E-26

Establishment of a Long-Term Mouse Model of Non-Alcoholic Steatohepatitis Induced by High-Fat Diet and Western diet

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Non-alcoholic steatohepatitis (NASH) is a progressive liver disease that can lead to cirrhosis and hepatocellular carcinoma. With the increasing global prevalence of NASH due to rising rates of obesity and sedentary lifestyles, establishing reliable animal models that mimic human disease progression is crucial for preclinical research and drug development.

In this study, we developed and characterized a long-term mouse model of NASH using C57BL/6N mice fed with either a Western diet (WD) or High fat diet (HFD) for 10 to 50 weeks. Mice receiving a normal diet served as controls. Body weight, liver weight, serum biochemical parameters (ALT, AST, lipid profiles, glucose), MRI imaging (PDFF and T2*), and liver histopathology were monitored at multiple time points.

Both WD and HFD-fed mice showed progressive body and liver weight gain from week 4. PDFF values increased significantly after week 10, indicating hepatic fat accumulation, while T2* values decreased, reflecting tissue injury. Histopathological analysis showed steatosis and inflammation by week 20, followed by stage 1A fibrosis. WD-fed mice exhibited more severe liver pathology compared to HFD-fed mice.

These findings suggest that the WD/HFD-induced NASH mouse model recapitulates key pathological features of human NASH and provides a valuable platform for evaluating candidate therapeutics and determining optimal intervention time points using combined histological, biochemical, and imaging markers.

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Keywords : NASH mouse model, Western diet / High-fat diet, Liver histopathology, MRI imaging

PS-E-25

Evaluation of tumor-targeting and biodistribution of tectentriq using in-vivo near-infrared fluorescence imaging in xenograft models

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Background : In-vivo near-infrared fluorescence (NIRF) imaging enables noninvasive, real-time monitoring of molecular interactions, offering a powerful approach for assessing targeted drug delivery. This study investigates the tumor-targeting and biodistribution properties of Cy5.5-labeled Tectentriq in xenograft models.

Method : BALB/c nude mice were subcutaneously implanted with RKO and PD-L1-overexpressing RKO tumor cells. Tectentriq was labeled with Cy5.5 and intravenously administered. In-vivo imaging was performed using IVIS before and at 1, 2, and 3 days post-injection. Ex-vivo fluorescence imaging was conducted on harvested tumors and organs (lungs, heart, liver, kidneys, spleen). PD-L1 expression was assessed via immunohistochemical staining of tumor tissues.

Results : Peak tumor accumulation of Tectentriq-Cy5.5 was observed at day 1 post-injection. Mice with PD-L1-overexpressing RKO tumors exhibited higher fluorescence intensity than those with wild-type RKO tumors. Tumor fluorescence in Tectentriq-Cy5.5-treated mice remained higher than that in the free-Cy5.5 control group. PD-L1 expression was confirmed in both tumor types, and probe accumulation corresponded with PD-L1 presence, indicating target specificity.

Conclusions : Cy5.5-labeled Tectentriq demonstrates strong tumor-targeting capability in PD-L1-expressing xenografts, suggesting its potential as a noninvasive imaging tool and a predictive biomarker for immunotherapy efficacy.

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Keywords : Near-infrared fluorescence (NIRF), Tectentriq, Tumor-targeting

PS-E-27

Toward the Harmonization of Record Management for Non-Human Primates in South Korea: A Survey and Review

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Non-human primates (NHPs) are essential animal models in aging, neuroscience, and biopharmaceutical research. As demand continues to grow, many research institutions are planning to establish new NHP facilities. Due to their long lifespans and biological complexity, NHPs require high standards of welfare and ethical management. In this study, a web-based survey was conducted across nine NHP facilities in Korea to assess current practices in individual record management and to identify opportunities for harmonization. The survey focused on three key areas: (1) methods of individual record management, (2) items and frequency of records, and (3) the need for harmonization of record management systems.

The results have shown that only four facilities have adopted integrated digital management systems, while the other five have relied on manual or semi-digital methods, such as handwritten notes or spreadsheets. All facilities have recorded basic information, including import history, daily observations, and body weight. However, records related to reproductive history, microbiological testing, and veterinary care have varied across facilities. Notably, only two facilities have recorded enrichment data. Respondents consistently highlighted challenges arising from inconsistencies in data categories, formats, and collection procedures, emphasizing the need for harmonized record management systems across NHP facilities.

This is the first survey conducted on individual record management of non-human primates in Korea and may serve as a reference for improving the quality of NHP production, management, and welfare. To ensure reproducible results in non-human primate research, it is time to initiate discussions on the harmonization of individual record management across NHP institutions in Korea. This research was supported by the Bio & Medical Technology Development Program of the National Research Foundation (NRF), funded by the Korean government (MSIT) (No. RS-2023-00260025).

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Keywords : Individual record management, Non-human primates (NHPs), Animal welfare

PS-E-28

Development of a Mouse-Based Experimental Platform for CDX Models and Antibody Therapeutics Evaluation.

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Animal studies are crucial for elucidating disease mechanisms and developing novel therapeutic strategies. Many medical advancements—including vaccines, anticancer drugs, and surgical techniques—have relied on preclinical testing using animal models. As the fields of precision medicine and biologics rapidly expand, there is an increasing need for advanced animal-based platforms that enable comprehensive evaluation of drug mechanisms and therapeutic efficacy.

In cancer research, the cell line-derived xenograft (CDX) model is widely used *in vivo* systems for research on tumor growth and drug responsiveness. This model involves transplanting cultured tumor cells into immunodeficient mice, providing a reproducible and biologically relevant platform for preclinical evaluation of anticancer agents.

In addition, antibody-based drugs require rigorous *in vitro* pharmacological assessments during early-stage development to elucidate mechanisms of action and determine candidate suitability. In response to these needs, we established an integrated murine experimental platform offering two independent evaluation pipelines: a CDX-based efficacy assessment line and an antibody pharmacology line.

CDX pipeline supports systematic analysis of therapeutic response through tumor growth curves, body weight monitoring, blood biochemical markers, and survival data. And, the antibody evaluation platform includes serum collection post-administration, followed by ELISA-based pharmacokinetic (PK) analysis and target-binding specificity assays. This allows for linkage between *in vitro* results and *in vivo* performance.

This platform is adaptively designed to support either CDX or antibody evaluation depending on research goals, with comprehensive support from model generation to data analysis. The system provides a robust foundation for proof-of-concept validation and mechanism studies in pre-clinical drug development, offering reproducible and translationally relevant insights.

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Keywords : Cell line-derived xenograft(CDX) model, Antibody pharmacology, Preclinical drug evaluation

PS-E-30

Evaluation of the effect of positive reinforcement training on blood cortisol levels in cynomolgus monkeys (*Macaca fascicularis*)

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Cortisol, a primary stress hormone secreted via the hypothalamic-pituitary-adrenal (HPA) axis, is widely used as a physiological biomarker for assessing stress in nonhuman primates. Laboratory procedures such as capture, blood collection, and routine handling can induce acute stress responses, elevating cortisol levels and affecting clinical parameters such as ALT, AST, and CK. These changes may compromise the reliability and reproducibility of experimental outcomes.

This study aimed to evaluate the impact of positive reinforcement training (PRT) on stress responses in cynomolgus monkeys (*Macaca fascicularis*) by measuring changes in serum cortisol levels. Twenty monkeys underwent a 30-day statutory quarantine followed by a 30-day acclimatization period, after which a 5-week phased PRT program was implemented. Blood samples were collected before and after training, during both morning and afternoon sessions. Serum cortisol concentrations were quantified using a competitive enzyme-linked immunosorbent assay (ELISA).

Following PRT, cortisol levels showed significant reductions in both morning and afternoon samples, with average decreases of 34.0% ($p = 0.0003$) and 20.6% ($p = 0.0013$), respectively. These reductions reflect the effects of training, and the greater decrease observed in the morning samples is likely due to the circadian rhythm of cortisol secretion. Cortisol typically peaks in the early morning and naturally declines throughout the day, which may explain the baseline differences between time points. These findings suggest that PRT may help alleviate physiological stress in laboratory-housed primates. However, considering the limitations of using a single physiological marker, further studies incorporating control groups, repeated trials, and additional behavioral or immunological indicators are warranted to more comprehensively validate the effects of PRT.

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Keywords : Positive Reinforcement Training, Cortisol, Stress Biomarker, Nonhuman Primate, Animal Welfare

PS-E-29

Targeting Endometrial Stem Cell Senescence with Traditional Herbal Medicines for Reproductive Aging

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Age-related infertility is a growing concern in reproductive medicine, closely linked to the aging of endometrial stem cells (EnSCs), which are essential for endometrial regeneration and implantation success. Despite the widespread use of assisted reproductive technologies (ART), current hormonal treatments often yield limited efficacy and have been shown to exacerbate EnSC senescence. In this study, we aimed to identify traditional herbal prescriptions with anti-senescent effects on EnSCs and to investigate their molecular mechanisms.

To investigate the molecular basis of age-associated senescence in EnSCs, we isolated EnSCs from mice of varying ages and assessed their lineage-specific differentiation capacity, stemness marker expression, and senescence-associated phenotypes. EnSCs from older mice exhibited significantly reduced adipogenic and osteogenic differentiation potential, accompanied by decreased expression of stemness markers and increased expression of senescence markers, supporting a progressive decline in stem cell functionality with age. In parallel, bulk RNA sequencing was performed to profile transcriptomic alterations associated with aging in EnSCs. This dataset is currently being analyzed to identify age-specific biomarkers and phenotypic signatures that characterize aged EnSCs.

Based on these findings, we conducted a targeted screen of 36 traditional herbal medicines historically used for treating reproductive disorders and postpartum recovery. Several candidates demonstrated the ability to ameliorate cellular senescence under *in vitro* aging conditions by enhancing proliferative capacity and reducing senescence-associated gene expression in EnSCs. These candidate prescriptions are now undergoing further validation in aged murine models through histological examination and functional evaluation of the uterus and EnSC compartments.

Together, these results support the therapeutic potential of specific herbal medicines in reversing age-associated EnSC dysfunction and enhancing endometrial receptivity, offering a viable strategy for fertility preservation in women of advanced maternal age.

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Keywords : Endometrial stem cell, Aging, Herbal Medicine, Improved endometrial receptivity

PS-E-31

Active Hexose Correlated Compound Attenuates Gut Dysbiosis and Improves Intestinal Barrier Function in a High-Fat Diet-Induced NAFLD Rat Model

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Nonalcoholic fatty liver disease (NAFLD) is a rapidly increasing global health concern, closely linked to obesity and metabolic syndrome. Recent evidence highlights the pivotal role of gut microbiota dysbiosis and compromised intestinal barrier function in the pathogenesis of NAFLD. Active hexose correlated compound (AHCC), a standardized extract derived from *Lentinula edodes* mycelia, demonstrates promising immunomodulatory properties; however, its influence on gut-liver axis homeostasis in NAFLD remains to be elucidated.

In this study, thirty-two male Sprague-Dawley rats were randomized into three groups: control diet (CD), high-fat (HF) diet, and HF diet supplemented with 2% AHCC for six weeks. We employed comprehensive microbiome analysis, intestinal barrier function assessment, and inflammatory marker evaluation to determine AHCC's therapeutic potential.

AHCC supplementation significantly enhanced gut microbiota alpha diversity and altered microbial clustering patterns compared to HF controls. Notably, beneficial bacteria *Blautia hansenii* and *Blautia producta*, recognized for anti-inflammatory and anti-obesity properties, showed marked abundance increases. These microbiome modifications correlated with improved intestinal barrier integrity, evidenced by significant mucin 2 upregulation and enhanced tight junction protein expression trends. AHCC treatment effectively reduced both local inflammation in colon and liver tissues and attenuated systemic inflammatory responses.

These findings highlight the capacity of AHCC to beneficially modulate gut microbiota composition, thereby alleviating oxidative stress, attenuating inflammatory cascades, and enhancing intestinal barrier integrity in diet-induced NAFLD. Collectively, the results support the therapeutic promise of AHCC as a dietary intervention targeting the gut-liver axis in the management of metabolic liver disorders.

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Keywords : Active hexose correlated compound, Gut microbiota, Inflammation, Intestinal barrier integrity, Non-alcoholic fatty liver disease

PS-E-32

Plant stem cell-derived extracellular vesicles enhance skin wound healing by alleviating LPS-induced inflammation.

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Plant stem cells are undifferentiated cells that possess the dual capacity for self-renewal and differentiation into various specialized cell types. These cells play a vital role in plant development, growth, and tissue regeneration. They are predominantly located in meristems specialized regions at the tips of shoots and roots, as well as in vascular tissues which serve as centers for continuous cell division and organ formation. Stem cells demonstrate totipotency or pluripotency, making them key players in plant tissue development and repair. Wound healing in mammals is a dynamic and coordinated biological process involving four overlapping stages: hemostasis, inflammation, proliferation, and tissue remodeling. While plant stem cells are traditionally studied for their role in plant physiology, recent interest has shifted toward their biomedical applications. Contemporary research highlights their anti-inflammatory properties, their impact on skin conditions such as atopic dermatitis, and their utility. In our study, we examined the regenerative and anti-inflammatory potential of Plant stem cell-derived extracellular vesicles (PDEVs). We observed that these cells significantly enhanced cellular proliferation and promoted wound healing in vitro. To validate these findings, an in vivo experiment was conducted using a murine wound induced by lipopolysaccharide (LPS) derived from *Klebsiella pneumoniae*. PDEVs were topically applied to wounds on the dorsal skin of mice, and wound healing was assessed at intervals on days using dermascope imaging and histological analysis. Our results demonstrated that PDEVs effectively mitigated LPS-induced inflammation and accelerated wound closure. These findings suggest a promising therapeutic potential of PDEVs in treating inflammatory wound healing. Further studies will be demonstrating the molecular mechanisms underlying these effects.

*Corresponding author : Keunsun Ahn, Sanghyun An

Keywords : Plant stem cell-derived extracellular vesicles, Lipopolysaccharide, Skin wound healing, Anti-inflammation

PS-E-34

Assessment of Bone Healing Enhancement Using Experimental Agents in an Osteoporotic Rat Model

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This study aimed to investigate the potential of novel experimental agents in enhancing bone repair following surgically induced bone defects in an osteoporotic rat model. Osteoporosis was induced in 8-week-old female Sprague-Dawley (SD) rats by performing bilateral ovariectomy, and the onset of osteoporotic conditions was confirmed through bone mineral density (BMD) analysis using Micro-Computed Tomography (MicroCT).

A standardized 4-mm defect was surgically introduced in the right radius of each animal. Experimental agents were applied directly to the defect site. To monitor bone regeneration over time, Dual-Energy X-ray Absorptiometry (DXA) imaging was conducted at 2, 4, and 8 weeks post-defect to evaluate bone density changes at the injury site. At 12 weeks, animals were sacrificed for final analysis, and MicroCT imaging was utilized to assess both morphological changes and BMD.

Compared to the sham-operated controls, the ovariectomized group exhibited a significant increase in body mass and a marked reduction in BMD. Imaging analyses, including DXA and MicroCT, were supplemented by quantitative measurements of the defect area using ImageJ software. The group treated with experimental substances showed a notable decrease in defect size, indicating enhanced bone regeneration.

These findings support the effectiveness of the tested agents in stimulating bone formation under osteoporotic conditions. Furthermore, this study establishes a validated protocol for modeling osteoporosis and evaluating the regenerative capacity of therapeutic interventions in bone defect models.

*Corresponding author : Yoon Beom Lee

Keywords : Osteoporosis, Ovariectomy, Bone regeneration

PS-E-33

Prediction of Social Hierarchy Based on Individual Aggression Assessment in Cynomolgus Macaques

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The cynomolgus macaque (*Macaca fascicularis*) is a widely used nonhuman primate model for investigating the effects of social hierarchy on behavior and physiological responses. However, in laboratory settings, individual housing is common, which limits natural social interactions and complicates group integration, potentially increasing stress and reducing data consistency.

This study aimed to determine whether individual aggression scores could reliably predict social hierarchy and to assess sex-specific differences in dominance formation. The research was conducted in 2 phases.

In Phase 1, 40 male macaques were assessed for aggressive behaviors, including open-mouth stares, lunges, and bites. Social ranks were determined through dyadic pairings based on observed dominant and submissive behaviors. Among 20 pairings, 80% exhibited clear hierarchical structures, and in 75% of these, the more aggressive individual attained the dominant rank. A binomial exact test confirmed that this pattern was statistically significant (12 of 16 dyads, $p = 0.038$), supporting the predictive value of individual aggression scores.

In Phase 2, 8 males and 8 females underwent a structured 4-stage mingling protocol involving 32 pairings per sex. Among males, 91% of pairings showed that the more aggressive individual assumed the dominant position, and aggression scores were strongly correlated with social rank (Spearman's $\rho = 0.843$, $p = 0.0085$). In contrast, among females, only 31% of pairings reflected this alignment, and the correlation between aggression and social rank was weak and not statistically significant ($\rho = 0.527$, $p = 0.179$), suggesting that affiliative and submissive behaviors may play a greater role in female social structuring. No stereotypic behaviors were observed, and body weight remained stable or increased, indicating that the assessment protocol did not induce physiological or behavioral stress.

These findings demonstrate that individual aggression assessment is a reliable and quantifiable method for predicting social dominance in male macaques, supporting its application in pre-integration strategies to improve animal welfare and experimental reproducibility. For females, a broader behavioral framework may be required for accurate hierarchy assessment.

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Keywords : Nonhuman primate, Hierarchy, Aggression, Laboratory Animal

PS-E-35

National primate infrastructure for biomedical and basic science

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Primates are valuable resources for different research fields including genetics, evolutionary biology, biomedical research, neuroscience, regenerative research, microbiology, vaccine development and pharmacology. Because primates have more biological and behavioral similarities and closer genetic relationship to humans than other animal models. However, primate resources are limited to access for individual researchers. In 2005, Republic of Korea government established National Primate Research Center (NPRC). First purpose of NPRC is production and supply of specific pathogen free (SPF) primate in Republic of Korea. Second purpose is supporting the regenerative medicine (bio organ transplantation, stem cell and gene therapy). The last one is supporting the basic biomedical research and basic science. Recently, NPRC established primate resource bank with various primate samples (Tissue deoxyribose nucleic acid (DNA), Blood DNA, ribose nucleic acid (RNA), cDNA, paraffin blocks (brain), etc) from crab-eating monkey, marmoset monkey, rhesus monkey, African green monkey, and squirrel monkey. And also, we established cutting edge medical imaging technique using 3 Tesla magnetic resonance imaging (3T-MRI), positron emission tomographic-computed tomographic (PET-CT), micro PET-CT, and angiography imaging system. Therefore, researchers who want to access the primate resources and use the imaging analysis with primate for research purpose could get various national primate infra service, easily (<http://portal.kribb.re.kr/primate>).

*Corresponding author : Young-Hyun Kim

Keywords : Basic science, Biomedical, Infrastructure, Primate, Monkey

PS-E-36

Nanobody-based CAR-NK cells for Immunotherapeutic targeting of Mesothelin+ tumors

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Chimeric antigen receptor (CAR)-based cellular therapies have demonstrated remarkable success in treating hematologic malignancies. However, their efficacy against solid tumors remains limited due to the immunosuppressive tumor microenvironment and poor antigen accessibility. To address these challenges, we aimed to develop CAR-NK cells targeting solid tumors, with a particular focus on pancreatic cancer, a highly treatment-resistant malignancy among solid tumors. For this purpose, we designed nanobody-based CAR-NK cells that specifically recognize mesothelin, a tumor-associated antigen highly overexpressed in pancreatic cancer.

In this study, we constructed a novel CAR using an anti-MSLN nanobody to enhance tumor specificity and transduced it into NK cells to generate anti-MSLN nanobody CAR-NK cells. The engineered cells were successfully produced and characterized, exhibiting stable surface expression of the CAR. Functional assays demonstrated that these CAR-NK cells displayed enhanced cytotoxicity against PDAC cell lines in a mesothelin-dependent manner, as evidenced by increased tumor cell lysis and upregulated secretion of cytotoxic granules such as granzyme B and perforin.

In vivo validation further supported the mesothelin-dependent antitumor efficacy of the anti-MSLN nanobody CAR-NK cells. Treatment with these cells resulted in significant suppression of tumor growth and revealed clear infiltration of CAR-NK cells within tumor tissues, indicating effective tumor targeting and functional activity in the tumor microenvironment. This study demonstrated the potential to overcome the therapeutic limitations of CAR-NK cell therapy in solid tumors, including the treatment resistance of pancreatic cancer, and to improve clinical outcomes for patients

*Corresponding author : Seok Ho Kim

Keywords : CAR-NK cell, Nanobody, Mesothelin, Pancreatic cancer

PS-E-37

Extracellular vesicle-derived microRNAs as biomarkers and regulators of porcine muscle cell differentiation

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The differentiation status of porcine muscle stem cells (pMuSCs) is a key determinant of cultured meat quality. Conventional analytical methods, including Western blotting, immunocytochemistry (ICC), and quantitative PCR (qPCR), are commonly employed to assess myogenic differentiation. However, these techniques are invasive and limited in scalability and compatibility with food-grade systems. To address these limitations, we investigated extracellular vesicles (EVs) as a non-invasive source of microRNAs (miRNAs) that both reflect and regulate the differentiation status of pMuSCs. EVs were isolated from undifferentiated pMuSCs and differentiated porcine muscle fibers (pMFs), and their miRNA contents were profiled. miRNA sequencing of cells and corresponding EVs identified nine differentially expressed miRNAs (DEmiRNAs). Among these, two miRNAs significantly upregulated in pMFs were selected for further investigation. Quantitative PCR confirmed consistent expression patterns in both cells and EVs, correlating with the progression of myogenesis. Functional assays revealed that overexpression of these miRNAs in pMuSCs enhanced expression of the myogenic marker MYH7, indicating a promotive role in differentiation. Integrated mRNA sequencing and *in silico* target prediction identified five potential target genes. Transfection of the miRNAs led to the downregulation of these targets, including CREB5, supporting their function as post-transcriptional regulators of myogenesis. In conclusion, EV-derived miRNAs serve as both non-invasive biomarkers and functional modulators of porcine muscle differentiation. These findings establish a foundation for scalable, food-compatible monitoring strategies in cultured meat production.

*Corresponding author : Chang-Kyu Lee, Kyung-Rok Yu

Keywords : Extracellular vesicle, Muscle differentiation, Biomarker

PS-E-38

Mapping the Evolution of Stem Cell-Scaffold Research in Bone Regeneration: A Bibliometric and Trend Analysis

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Bone defects arising from aging, trauma, or surgical interventions often exceed the body's natural regenerative capacity. While mesenchymal stem cells (MSCs) have been extensively investigated for their osteogenic and immunomodulatory properties, recent approaches increasingly integrate MSCs with scaffolds or biomaterials to enhance therapeutic efficacy through improved delivery, mechanical support, and biological signaling.

A bibliometric and trend analysis was conducted on global literature from January 2015 to June 2025 using the Web of Science Core Collection. The dataset included articles and reviews, and was analyzed using Bibliometrix (R package) and VOSviewer. The search strategy combined keywords related to bone tissue engineering, MSCs, scaffolds, and preclinical studies.

A total of 3,211 publications were analyzed. China led in research output (1,706 publications), followed by the USA and South Korea. The most productive institutions were Shanghai Jiao Tong University and Sichuan University. Co-occurrence analysis identified six major thematic clusters: stem cell biology, scaffold properties, immune regulation, *in vivo* evaluation, signaling molecules (e.g., exosomes, miRNAs), and biomaterial fabrication. Over time, the research focus shifted from traditional structural considerations (e.g., biocompatibility, porosity) to advanced biological strategies involving immune modulation and extracellular vesicle-mediated signaling.

The field of stem cell-scaffold research in bone regeneration is evolving from passive structural support paradigms toward active, biofunctional, and immune-responsive therapeutic strategies. This transformation reflects the broader movement toward precision regenerative medicine, emphasizing microenvironmental control and cell-free signaling with enhanced translational potential.

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*Corresponding author : Kyenghee Kwon

Keywords : Stem Cell, Scaffold, Tissue engineering, Bone regeneration, Bibliometric analysis

PS-E-39

Regulatory Considerations for Stem Cell-Scaffold Therapies for Osteoarthritis (OA) : Case study of Product

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Osteoarthritis (OA) is a typical degenerative joint disease, which is on the rise in aging societies. Although various surgical and pharmacological management options exist, most aim to alleviate suffering rather than target the causative factors or cure the condition. Regenerative treatment with autologous chondrocyte implantation (ACI) and stem cell-based therapies have shown promising results, but the clinical use is currently limited by issues like low cell viability, insufficient long-term stability. To date, Cartistem in Korea remains the only approved stem cell therapy for OA, reflecting the challenges in commercializing such products.

Recently, combining stem cells with biodegradable scaffolds has emerged as a promising strategy to enhance their therapeutic efficacy by improving cell survival, promoting tissue regeneration, and enabling targeted delivery. However, the fabrication of these products faces complex scientific issues of biocompatibility, immunogenicity, biodegradability, and mechanical stability. Moreover, the double identity of biologics and medical device makes the process for their authorization more difficult due to the need for a multidisciplinary approach.

This study aims to provide foundational insights for the development and regulatory strategy of stem cell-scaffold combination products for OA treatment. Although no such products have been approved to date, relevant insights can be drawn by analyzing existing cases of stem cell-based therapies and scaffold-based treatments for OA. To this end, nonclinical study design requirements and approval procedures were examined, and approval documents and regulatory guidelines were reviewed to derive practical implications that may support the efficient development and global commercialization of future stem cell-scaffold therapies. In addition, current clinical trends of mesenchymal stem cells (MSCs)-scaffold combination therapies were also investigated.

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*Corresponding author : Kyeng Hee Kwon

Keywords : Osteoarthritis (OA), Stem Cell, Scaffold, Regulatory Science, Pre-clinical

PS-E-40

Evaluating the Glucose-Lowering Efficacy of Substance A in ob/ob Diabetic Mice

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This study investigates the glucose-lowering effects of Substance A, a potential candidate for development as a health functional food, using ob/ob mice, a genetically engineered model of type 2 diabetes. Substance A was administered orally once daily for four weeks. Throughout the treatment period, fasting blood glucose levels were measured weekly to evaluate changes in glycemic control. During the final week, an oral glucose tolerance test (OGTT) was conducted to further assess the impact of Substance A on glucose metabolism and clearance.

Substance A was administered orally once daily for a total duration of four weeks. During this treatment period, fasting blood glucose levels were monitored weekly to assess improvements in glycemic control over time. In the final week of the experiment, all animals underwent an oral glucose tolerance test (OGTT), which is a standard method for evaluating the ability of the body to metabolize and clear exogenous glucose.

The results indicated a trend toward decreased fasting blood glucose levels in mice treated with Substance A compared to untreated diabetic controls. No adverse effects or signs of toxicity were observed in either the treated diabetic group or the healthy control group, demonstrating the compound's safety under the experimental conditions.

These findings suggest that Substance A has a beneficial glucose-lowering effect and potential as a functional food ingredient for managing hyperglycemia associated with type 2 diabetes. Given its demonstrated efficacy in a well-established diabetic animal model, Substance A may be a promising candidate for further development in the functional food industry. Future studies are warranted to explore its mechanism of action and clinical applicability.

*Corresponding author : Na-Hye Park

Keywords : Health functional food, Blood-glucose lowering effect, Diabetes

PS-E-41

Cell-scaffold study design in bone regeneration for Key Factor: in vitro & in vivo perspectives in tissue engineering

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Tissue engineering (TE) is an interdisciplinary field increasingly regarded as a promising alternative to traditional grafting procedures, such as autografts, allografts, and xenografts. Due to the poor self-healing capacity of cartilage, diseases such as osteoarthritis and articular cartilage defects remain difficult to treat. In this perspective, cartilage tissue engineering offers a compelling therapeutic strategy. Conventional TE strategies consist of the materials, cells, bioactive molecules themselves, and their combinations. Among these, bioactive combined systems including all three components have been considered as the most promising and powerful for repairing tissues.

Scaffolds provide a three-dimensional (3D) structure that mimics the native extracellular matrix (ECM), recreating a cellular microenvironment conducive to adhesion, spreading, proliferation, and differentiation. Scaffolds need to have excellent biocompatibility, biodegradability, non-toxicity and other structural characters, including suitable porosity, pore size and interconnection to promote an optimal cartilage regenerating performance. A great variety of scaffolds made of natural or synthetic polymers were designed to satisfy these requirements.

In addition to scaffolds, the selection of cell type is critical. MSCs are widely used due to their safety, ethical, and regenerative capacity. Bioactive molecules also optimize the regenerative potential by controlling the properties of the scaffold and cells, promoting a better quality tissue formation.

In this review, we summarize key design parameters for scaffold-cell-bioactive molecule systems and propose standardized in vitro and in vivo evaluation methods for osteoarthritis, osteogenesis, and bone regeneration. This work aims to support the development of nonclinical testing standards and facilitate the clinical translation of tissue-engineered therapies

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*Corresponding author : Kyenghee Kwon

Keywords : Tissue engineering, Scaffold, Stem cell, Bioactive molecule, Bone regeneration

PS-E-42

Application of Three-Dimensional Motion Analysis to Assess Joint Dysfunction and Natural Locomotion in Beagle Dogs and Non-Human Primates

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This study aimed to develop an advanced evaluation platform for toxicity and efficacy of novel pharmaceutical candidates by employing three-dimensional (3D) motion capture technology in beagle dogs and non-human primates (NHPs). Existing neurotoxicity assessment protocols predominantly utilize rodent models and in vitro assays; however, rodent-based evaluations are hindered by interspecies metabolic discrepancies and slower physiological processes, which compromise clinical translational validity and reduce the precision of behavioral outcome measures. Conversely, non-rodent models, especially NHPs, present superior translational value due to their anatomical, physiological, and neurobehavioral homology to humans, encompassing comparable central nervous system structures, locomotor dynamics, and metabolic pathways.

An experimental arthritis model was established in beagle dogs via surgical excision of the meniscus and anterior cruciate ligament. Reflective markers were affixed to both control and arthritic animals, and locomotor behaviors were quantitatively recorded using a 3D motion capture system. Comparative analysis revealed a non-significant trend toward reduced gait velocity in arthritic subjects, while a statistically significant decrease in knee joint flexion angle was observed, likely reflecting joint inflammation and cartilage deterioration consequent to arthritis induction.

In parallel, healthy NHPs donned custom-fitted motion capture suits with strategically positioned markers on the wrist, elbow, shoulder, ankle, knee, and hip joints. Their spontaneous locomotion—including ascent, descent, and ambulation—was captured and analyzed to establish normative biomechanical parameters, such as joint range of motion and gait characteristics.

In summary, this research successfully established a behavior-based quantification method capable of discerning functional impairments induced by arthritis in beagle dogs and constructed normative locomotor profiles for NHPs. The results support the applicability of 3D motion capture technology as a robust, non-invasive tool for preclinical toxicity and efficacy evaluation in higher-order animal models

*Corresponding author : Ji Woon Kim

Keywords : Non-Human Primate, Beagle, 3D motion, Arthritis Model, Normative locomotor

PS-E-43

Current trends and knowledge mapping of ionizing radiation in preclinical animal research

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Ionizing radiation is one of major intervention in biomedical research involving animal models, particularly in oncology, radiobiology, and toxicology. To map the global research landscape and identify thematic developments in animal research with ionizing radiation, we conducted a scientometric analysis of 3,189 original research articles published between 2000 and 2024, retrieved from the Web of Science Core Collection. The search strategy targeted topic terms including "animal" or "in-vivo" in combination with "ionizing radiation." Data were analyzed using VOSviewer 1.6.20 and KnowledgeMatrix Plus 0.8.

Annual number of publication output has shown a consistent upward trend over the past two decades, reaching a peak of 163 articles published in 2024. The most represented Web of Science Categories were Oncology (22.5%), Radiology, Nuclear Medicine & Medical Imaging (20.7%), and Biochemistry & Molecular Biology (14.0%). Keyword co-occurrence analysis revealed key research clusters centered on radiation-induced toxicity, DNA damage, oxidative stress, immune response, and radioprotective strategies.

The United States accounted for the highest proportion of publications (42.1%), followed by China (15.0%) and Germany (7.5%). South Korea, while ranking 8th globally (4.1%), has shown notable growth in recent years, with leading contributions from institutions such as the Korea Institute of Radiological and Medical Sciences (KIRAMS) and Korea University. Major global institutions ranked by number of scientific articles included the University of California System (131 articles), National Institute of Health USA (101 articles), University of Texas System (101 articles), and Harvard University (80 articles). On the one hand, KIRAMS ranked 30th globally (36 articles).

This scientometric analysis underscores the global expansion of radiation-based animal research and highlights the emerging role of Korean institutions. These insights contribute to shaping future directions in preclinical radiobiology, institutional strategy, and the development of ethically grounded research within Specific Pathogen Free (SPF) laboratory animal facilities.

*Corresponding author : Kyung Mi Park

Keywords : Ionizing radiation, Preclinical research, Knowledge mapping, Scientometrics, Web of Science

PS-E-44

The receptor for cacao polyphenol procyanidin C1 and its anti-melanoma effects

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Scope: Grape seed extract is rich in polyphenols, including catechin and epicatechin oligomers, also known as procyanidins. These procyanidins are key bioactive elements in grapes, with numerous studies highlighting their anti-cancer properties in vivo. Specifically, procyanidins have been shown to inhibit the growth of melanoma tumor cells. Procyanidin C1 (PC1), an epicatechin trimer, is one of the most prevalent procyanidins in grape seeds. Despite its known health benefits, the exact molecular mechanisms behind PC1's anti-cancer effects remain largely unknown. This study aims to uncover the molecular functioning of PC1.

Methods and Results: The research identifies the 67-kDa laminin receptor (67LR) as an essential receptor for PC1 on cell surfaces, with a dissociation constant (Kd) of 2.8 μ M. PC1 suppresses cell growth by causing the dephosphorylation of CPI17 and MRLC proteins, leading to the reorganization of the actin cytoskeleton in melanoma cells. This is facilitated by the activation of protein kinase A (PKA) and protein phosphatase 2A (PP2A) when PC1 binds to 67LR.

Conclusion: The human equivalent dose (HED) for PC1 is calculated to be 2.43 mg/kg, translating to a 145.8 mg dose for a 60 kg individual, based on the formula for dose translation by surface area. This dose corresponds to the procyanidin content found in approximately 5 grams of grape seeds. The data suggest that consuming 5 grams of grape seeds may help inhibit melanoma tumor growth. The research demonstrates that PC1 inhibits the proliferation of melanoma cells by activating the 67LR/PKA/PP2A/CPI17/MRLC signaling pathway.

*Corresponding author : Jaehoon Bae

Keywords : Procyanidin C1, Epicatechin, Melanoma, Sensing molecules, Wine polyphenols

PS-E-45

Evaluation of the Skin-Whitening Effects of Cotton Bark Extract via α -MSH-Stimulated Melanogenesis in B16F10 Cells

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Skin pigmentation disorders, such as hyperpigmentation, are caused by various internal and external factors including ultraviolet (UV) exposure, hormonal imbalances, and inflammation. These issues are not only associated with cosmetic concerns but are also closely related to overall skin health. As interest in skin-brightening agents continues to rise, the demand for safe and effective natural ingredients with low toxicity and high biocompatibility has become increasingly important in cosmetic science.

In this study, we evaluated the skin-whitening effects of a 70% ethanol extract derived from the bark of Gossypium spp. in α -MSH-induced B16F10 melanoma cells. The extract was shown to reduce melanin production in a concentration-dependent manner, as measured by melanin content assays. Furthermore, Western blot analysis demonstrated a decrease in the expression of several pigmentation-related proteins, including tyrosinase, tyrosinase-related protein 1 (TRP1), tyrosinase-related protein 2 (TRP2), and microphthalmia-associated transcription factor (MITF), indicating that the extract influences the regulation of key factors involved in pigmentation.

These results indicate that cotton bark extract effectively reduces melanin synthesis and expression of related regulatory proteins in pigment-producing cells. Therefore, it shows strong potential as a natural and functional ingredient for cosmetic applications aimed at improving skin brightness and tone. This study offers useful insight for the development of innovative skin-whitening products using botanical extracts.

*Corresponding author : Jihye Lee, Ji Wook Jung

Keywords : Cotton bark extract, Melanin, Skin whitening, Gossypium indicum

PS-E-46

Sample Integrity First: A Roadmap for Error-Free Liver Function Testing in Laboratory Animals

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Reliable liver function data are pivotal for evaluating systemic toxicity and metabolic health in animal studies. Yet up to 70% of laboratory mistakes arise in the pre-analytical phase, where specimen identification, collection, and handling routinely introduce bias. Such variability compounds the life-science reproducibility crisis, with >50% of published animal experiments reportedly irreproducible. We reviewed common liver biomarkers alanine aminotransferase (ALT), aspartate aminotransferase (AST), alkaline phosphatase (ALP), γ -glutamyl transferase (GGT), total bilirubin, albumin, and total protein and identified key error vectors for each. Hemolysis artificially elevates ALT/AST, while delayed centrifugation skews ALP and GGT; bilirubin degrades under light, and improper anticoagulant ratios alter protein fractions. Evidence-based solutions include point-of-care barcoded labeling, time-stamped transport within 30 minutes, use of light-protected tubes for bilirubin, and immediate serum separation at 2,000 g for 10 minutes. Implementing systematic heterogenization splitting experiments into mini-batches over time further buffers unknown day-to-day variance, boosting external validity without inflating animal numbers. Collectively, meticulous pre-analytical control coupled with smart experimental design offers a pragmatic route to reproducible, ethically responsible liver studies.

*Corresponding author : Yon Lim

Keywords : Pre-analytical error, Reproducibility crisis, Serum separation, Liver function biomarkers, Quality control

PS-E-47

Effective Hand-Rearing Protocol for Normal Growth in Triplets Neonate Common Marmosets in Seoul National University Hospital Marmoset Model Resource Bank (SNUH-MMRB)

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Common marmosets (*Callithrix jacchus*) frequently give birth to litters of three or more, which exceeds the natural nursing capacity of the dam. To prevent undernutrition and ensure survival, Seoul National University Hospital Marmoset Model Resource Bank (SNUH-MMRB) applies a standardized hand-rearing protocol which involves supplemental feeding with formula to triplet neonates. In this study, we compared the body weight changes of naturally-reared singletons (n=4) and twins (n=14) versus hand-reared triplets (n=105) for eight weeks after the birth. The body weight changes were analyzed weekly to assess both absolute growth and relative weight gain (fold-change from birth). While triplets consistently exhibited lower absolute body weights compared to singletons and twins, the relative growth rates were not significantly different, indicating normal developmental progress. These findings demonstrate that our hand-rearing protocol effectively compensates for the biological limitations of maternal nursing in triplet marmoset litters. This highlights the reliability and utility of our protocol as a practical strategy for managing early development in marmoset colonies.

*Corresponding author : Byeong-Cheol Kang

Keywords : Common marmoset, Hand-rearing protocol, Colony management

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홍보 동영상 1



홍보 동영상 2

(부가세 별도)

서비스 유형	세부 서비스 유형	서비스 내용	신청 절차	추가 제공 정보	분양 수수료 (추출물/원시료)	정보 이용료 (효능정보)	담당자
천연물 분양서비스	국내식물 전락소재	개별시료 : 70% 발효주정 추출물 10mg 대량추출 : 70% 발효주정 추출물 1g 원시료 : 추출물 1g 상당의 원시료	천연물중앙은행 홈페이지 (kobis.re.kr/npcb)에서 분양신청	HPLC/UPLC Chromatogram (MS/LV/ELSD) 및 구성성분 정보	10,000원 500,000원 150,000원	10,000원 / 바이오마커	김정희 043-240-6121 plantext1@kribb.re.kr
	해외식물 전락소재	개별시료 : 70% 발효주정 추출물 10mg 대량추출 : 70% 발효주정 추출물 1g 원시료 : 추출물 1g 상당의 원시료	1. 소재분양신청 회원가입 ▶ 분양신청 ▶ 접수 ▶ 분양수수료 청구 ▶ 발송 ▶ 연구결과 Feedback	UPLC-MS Chromatogram	10,000원 500,000원 150,000원	효능 I 산업화 후보소재 대상으로만 '24.~'26.에 구축예정	
	국내식물 (舊)한국식물 추출물은행	개별시료 : 99.9% MeOH 추출물 20mg 대량추출 : 99.9% MeOH 추출물 1g 원시료 : 추출물 1g 상당의 원시료	2. 분석정보 검색 회원가입 ▶ 소재/성분정보검색 ▶ 분석정보열람 신청 ▶ 분석정보 열람 ▶ 분석정보 신청		6,000원 300,000원		
	해외식물 (現)해외생물 소재센터	개별시료 : MeOH, EtOH, 추출물 20mg 분쇄시료 : 담당자에게 문의 대량시료 : 담당자에게 문의	3. 효능정보 검색 회원가입 ▶ 효능정보 신청 (효능정보 활용협약서 작성필수) ▶ 접수 ▶ 분양수수료 청구 ▶ 발송 ▶ 연구결과 Feedback		100,000원 3,000원 10,000원	효능 II	
	용매 분석서비스	식물추출물을 Hexane, Chloroform, Ethyl acetate, Butyl alcohol, Distilled water로 단순 용매분획하여 제공하는 서비스	천연물중앙은행 홈페이지(kobis.re.kr/npcb)에서 분석 신청		1,000,000원 / 추출물 1-10g 기준		
	천연물 분리 분석서비스	Column을 이용한 분리 분석서비스			5,500,000원 / 추출물 1-5g 기준		
소재기탁 서비스	천연추출물 및 원시료 기탁	국가연구개발사업을 통해 확보된 식물소재를 천연물중앙은행에 기탁하여 다른 연구자들이 기탁된 소재를 공유하여 연구할 수 있도록 하는 기탁서비스	회원가입 및 기탁접수(연구성과를 웹사이트, https://biopr.kribb.re.kr/) ▶ 서류검사(기탁가능 여부조사) ▶ 식물자원 접수 ▶ 자원확인 ▶ 기탁번호부여 ▶ 연구성과를 기탁발출 발행		무료 (기탁자 동의 시)		김정희 043-240-6121 plantext1@kribb.re.kr
	Column을 이용한 분리 분석서비스	UPLC-QTOF-MS(Waters社)를 이용하여 식물추출물 또는 분획물의 주요 화합물의 Mass data 분석을 통해 잠정적으로 확인(Tentative Identification) 해주는 서비스 ※ 정량분석 가능(표준품 제공필수)	회원가입 및 분석신청(http://www.zeus.go.kr/main) ※ 장비명 : 이온 모빌리티 Q-TOF Mass Spectrometer (시설장비등록번호 : NFEC-2020-10-265378 장비선택) ※ 정량분석 가능(표준품 제공필수)		1단계 : 400,000원 / 점 2단계 : 400,000원 / peak 3단계 : 400,000원 / peak		안종민 043-240-6143 jmahn@kribb.re.kr

(추가제공정보 참고사항)

· 분석정보 1) 분석데이터의 저작권은 '천연물중앙은행'에 있으며, 분석데이터는 회원에게 무료공개로 원칙으로 하나, 무단캡처, 배포 및 2차 가공을 통한 재배치를 금지 2) 논문, 특허작성용으로 사용될 분석데이터를 원하시면 'Mass분석서비스'를 이용

· 효능정보 효능 I, MAO A, MAO B, 뇌신경세포생존율, ACE, IL-1, sICAM-1, DPP-4, IL-6, DPPH, ABTS, TAPC, β-hexosaminidase, MUC5AC, IL-5, NO, Cell viability, IL-1β, ALP, 지방세포분화, Cell viability, ROS, IL-8, MMP-1, Procollagen

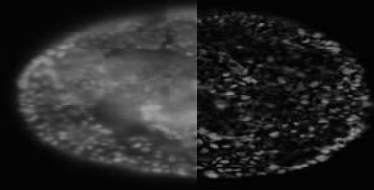
1) 효능정보의 저작권은 '천연물중앙은행'에 있으며, 효능정보는 지식재산권의 보호를 위해 홈페이지에 미공개

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효능 II, 항염, 세포생존율, 살충, 항산화, 항암, 미백, 항당뇨, 항알

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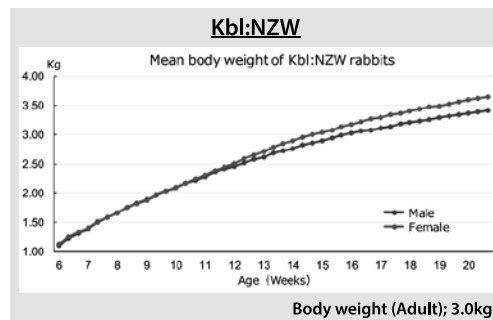
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(New Zealand White)



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	Myxoma virus PCR
	Rabbitpox PCR
	Group A Rotavirus PCR
	Pinworm Screen PCR
Parasite	Cryptosporidium PCR
	Rabbit Eimeria PCR
	Leporacarus gibbus PCR
	E. cuniculi PCR (Encephalitozoon cuniculi)
	B. bronchiseptica PCR (Bordetella bronchiseptica)
Germ	C. piliforme PCR (Clostridium piliforme)
	P. multocida PCR (Pasteurella multocida)
	Salmonella PCR
	M. pulmonis PCR (Mycoplasma pulmonis)

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Research Models

Guinea Pig

Hartley Guinea Pig



Nomenclature _
ElmSim-11A

Origin _
ELM HILL (U.S.A)

Color _
Albino

Research Use _

- 면역학 (Immunology)
- 청각 연구 (Auditory Studies)
- 백신 (Vaccine)
- 농약 (Pesticides)



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Research Models

Rabbit

Newzealand White Rabbit (NZW Rabbit)



Nomenclature _
Sam-NZW

Origin _
Karinfarm (EUROPE)

Color _
White (Albino)

Research Use _

- 생리학 연구 (Biomedical Research)
- 피부과학 (Dermatology)
- 항체 생산 (Antibody Production)
- 신장 연구 (Renal Studies)
- 안과학 (Ophthalmology)
- 독성학 (Toxicology)
- 죽상동맥경화증 (Atherosclerosis) 연구
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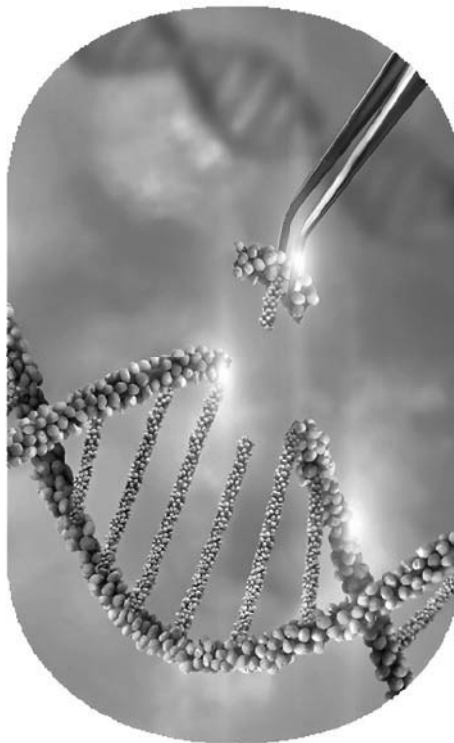
동물진단
(Animal Diagnostics)



동물약품
(Animal Drug)



메디피그
(Experimental Medicine)



이종장기
(Xenotransplantation)

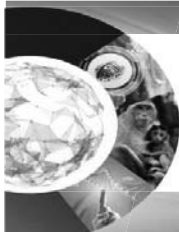


VLP 백신
(VLP Vaccine)



박테리오파지
(Bacteriophage)

OPTIPHARM



Primate resources center (PRC) supports non-human primate infrastructure for biomedical and bioscience research

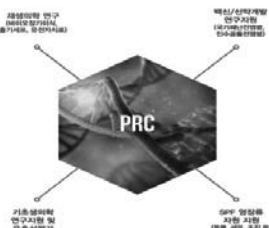
Primate Resources Center (PRC), Korea Research Institute of Bioscience and Biotechnology (KRIIBB), Jeongup 56216, Republic of Korea



<http://portal.kribb.re.kr/prc>

영장류자원지원센터 소개

영장류자원지원센터 주요미션



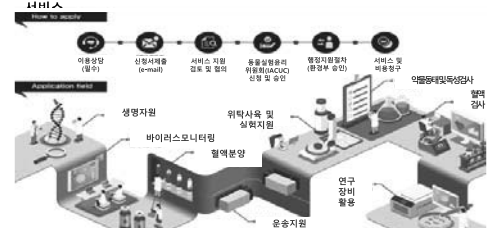
영장류자원지원센터 주요연혁



영장류자원지원센터 주요업무



영장류자원지원센터 자원/소재



영장류자원지원센터 인프라현황

영장류자원지원센터 시설 인프라



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그동안 복잡한 동물실험계획서 심의업무 번거롭고 불편하셨죠? 이제부터 편리하고 사용하기 쉬운 온라인 동물실험관리 시스템을 경험하세요!

1



온라인 심의

- 온라인 심의 시스템 (자동 이메일, SMS 발송)
- 복잡한 심의 과정 간소화
- 심의 단계별 현황 파악 용이
- 심의 기간 단축 및 비용 절감 효과

2



온라인 신청

- 동물구매, 반입, 반출, 실험실예약
- 실험연장, 공동기기 사용신청, 시료 신청, 소모품 신청 등
- 각종온라인 신청 시스템

3



온라인 교육

- 동물보호법 및 실험동물에 관한 법률관련, 이용자교육 등의 각종 온라인 교육 시스템
- 동영상 교육자료 열람 후 문제은행 랜덤 출제방식 등 구현

4



온라인 정산

- 사육비, 구매비, 실험실사용료
- 기술지원, 사체처리비 등
- 각종 온라인 정산 시스템

5



온라인 사육 및 개체관리

- 설치류 현황 등록 관리
- 중대형동물 개체 관리
- 사육시설 등록 관리
- Rack cage 관리

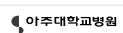
6



LMO 관리

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- 시설현황 점검표 작성
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- 책임자에게 제출, 책임자 확인
- 총괄 관리자 현황 모니터링

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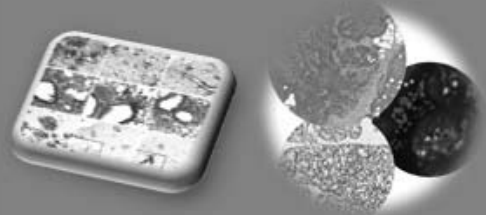
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information



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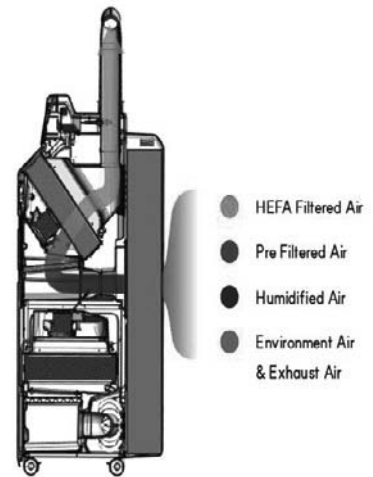
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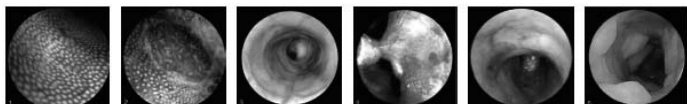
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Derived parameters

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pCO₂(T)
cHCO₃-(P)
cBase(B)
cBase(B,ox)
cBase(Ecf)
cBase(Ecf,ox)
cHCO₃-(P,st)
cH⁺
cH⁺(T)
ctCO₂(P)
ctCO₂(B)
pH(st)
pO₂(T)
pO₂(A)
pO₂(A,T)
p50
p50(T)
p50(st)
pO₂(A-a)
pO₂(A-a,T)
pO₂(a/A)
O₂(a/A,T)
pO₂(a)/FO₂(I)
O₂(a,T)/FO₂(I)
cCa²⁺(pH=7.40)
Anion Gap(K⁺)
Anion Gap
DO₂
Hct
pO₂(x)
pO₂(x,T)



ctO₂(B)
ctO₂(a-v-)
BO₂
ctO₂(x)
FShunt
FShunt(T)
RI, RI(T)
VO₂
mOsm
Qx
Qt
V(B)
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Applications



In Vitro

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Cells,
Adherent Cells,
Organoids



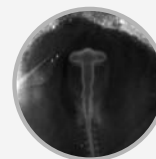
In Utero

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Rat



In Vivo

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조 직 검 사 술 선

간소화된 일차 조직염색 및 커버슬리핑의 새로운 시대를 여십시오.

HistoCore CHROMAX WS는 고품질 조직염색 솔루션에서 수십 년의 경험을 통해 생산성 향상, 최적화된 시약 소비 및 디지털 문서화를 제공합니다.

손끝에서 데이터 관리

업무량을 한 눈에 파악할 수 있습니다.
HistoCore CHROMAX ST를 사용하면
검사실에서 워크플로우 추적 및 문서화를 위해
신뢰할 수 있는 동반자를 확보할 수 있습니다.

상세한 로그에 쉽게 액세스하여 표준 준수를
보장하고 검사실 전체의 원활한 커뮤니케이션을
촉진합니다.

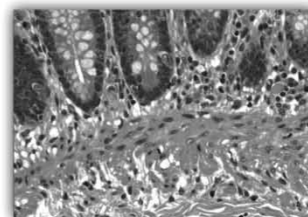


마지막 한 방울까지 활용하세요.

HistoCore CHROMAX ST는 슬라이드 카운팅의 자동화를 통해 시약 소비를 최적화하여 폐기물을 줄이고
비용 효율성을 높입니다. 염색된 표본의 품질을 저하시키지 않으면서 최적화된 시약 사용을 도입해 보세요.

커버슬리퍼 1대의 다기능성

HistoCore CHROMAX CV의 다기능성은
검사실의 고유한 요구를 충족하도록
맞춤화되어 있습니다. 자일렌이 없는
염색을 포함하여 일반 브랜드의
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커버글래스 크기를 지원하며, 시장을
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HistoCore CHROMAX CV에서 제공하는 유리
커버슬립의 뛰어난 광학적 선명도에 몰입하여 정밀한
디지털 스캐닝 및 영상 보관을 위한 토대를 마련해
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- 기타 연구분야 (Others)

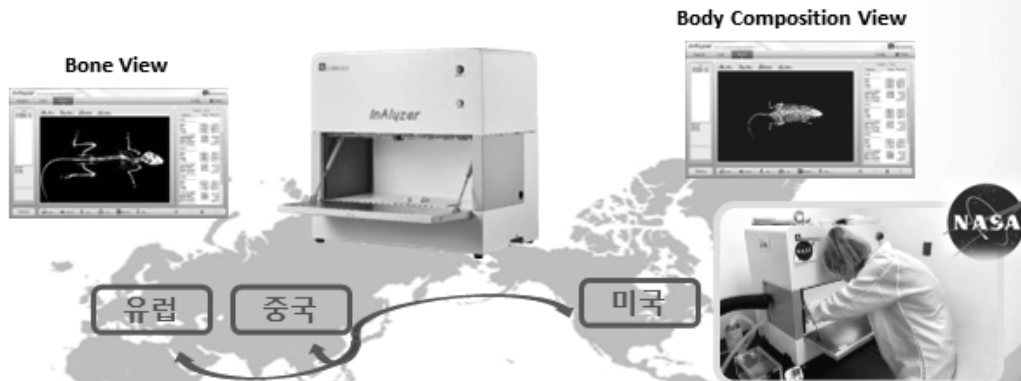


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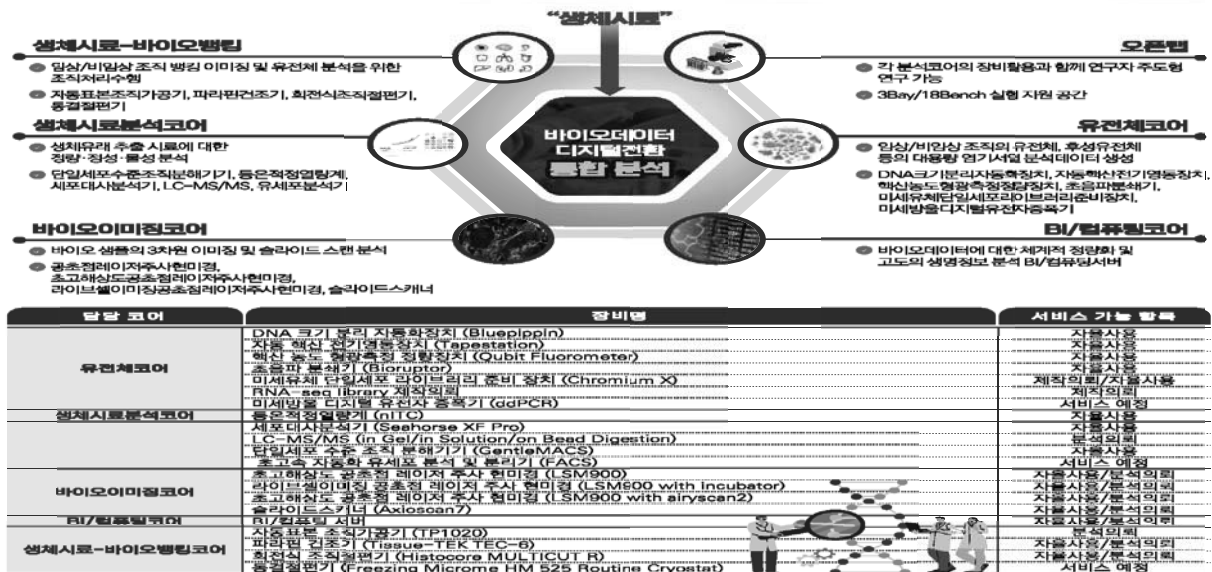
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- ① 소재 개발 고도화 지원
- ② 장비활용 지원
- ③ 시험분석 서비스 제공
- ④ 전주기 시험평가 지원 (연계인프라활용-안전성평가)
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✓ 약물전달기반 바이오소재의 시험평가 및 기술자문 서비스를 통한 연구개발 애로사항 해결

① 소재 개발 고도화 지원

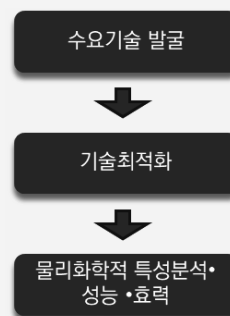
- 구축/연계장비를 활용한 개별기업에서 접근하기 어려운 분석 서비스 제공 및 기술 컨설팅 제공

② 범용장비 공동활용

- 신규구축 장비를 활용한 개별기업의 접근하기 어려운 장비의 사용지원
- 기구축 장비를 활용한 단백질체 및 대사체 전문 분석지원

③ 시험분석 서비스 제공

- 물리화학적 특성분석, 방출, 평가, 효력에 이르는 일련의 연구개발 단계를 지원하는 신뢰성 있는 시험평가



글로벌 기준 평가 체계 구축

- ① GLP·KOLAS 국제 공인 시험기관 체계 구축 (Global Standard Compliance)
- ② 글로벌 시장 진출을 위한 신뢰성 검증 시험·평가 제공 (Validated Testing for Global Market)
- ③ 국제 공인 기술, 고객 중심 맞춤 서비스 (International Compliance, Personalized Services)

규제국제신뢰성 확보, 해외진출 지원

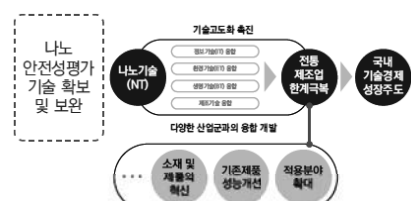
기술서비스

- ① GLP 독성시험
 - 흡입독성
 - 생태독성
 - 대체독성
- ② 물리·화학적 특성평가
- ③ 산업체 R&D 및 현장애로기술 지원
- ④ 장비 활용 지원



화학물질 및 농약에 대한 시험평가 지원

구축 산업·기업연계



국내·외 인프라와 연계한 협력체계 구축

✧ 글로벌 규제 대응을 위한 나노소재·제품 안전성 평가, 기업의 든든한 파트너

✓ 흡입독성시험(Inhalation Toxicity Study)

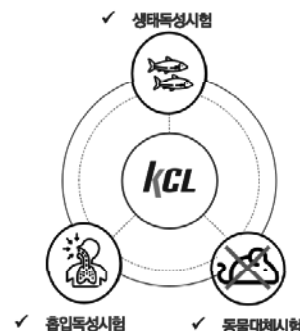
- OECD 등 국제적으로 공통 적용되는 기준 하에 호흡성물질(nanoparticle, gas, vapor, dust, mist, fume, smoke 등)을 전신 및 비부노출 방법을 통해 동물의 호흡기에 노출시키고 흡입된 물질들의 유해성 평가.

✓ 생태독성시험(Ecotoxicity study)

- 화학물질이 생태계(또는 환경)에 노출 되었을 때 생물에 미치는 독성 영향을 먹이사슬(생산자-중간소비자- 최종소비자)을 구성하는 대표 생물군(담수·육상조류, 물벼룩, 어류)을 통해 확인하기 위한 시험.

✓ 동물대체시험(In vitro study)

- 물질이 피부 또는 눈에 접촉했을 때 발생할 수 있는 대한 자극 가능성을 OECD 가이드라인 및 GLP 시험 규정 등에 따라, 동물대체시험인 세포 기반의 In-vitro 시험법으로 평가하기 위한 시험.



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차세대 실험동물실

저비용 고효율과 유지관리비 절감으로 미래경쟁력을 갖추세요!

(주)쓰리샤인은 기획, 설계, 예산, 컨설팅을 무료제공하며 상세설계, 시공, 사육장비, 관리장비, 실험장비, 수술장비, 소독장비 등 50여종 200여 품목을 직접생산 유지관리 하는 전무후무한 토탈서비스 기업입니다. 많은 이용바랍니다