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Aims and Scope

The Korean Journal of Physiology & Pharmacology (Korean J. Physiol. Pharmacol., KJPP) is the official journal of both the Korean Physiological Society (KPS) and the Korean Society of Pharmacology (KSP). The journal launched in 1997 and is published bi-monthly in English. KJPP publishes original, peer-reviewed, scientific research-based articles that report successful advances in physiology and pharmacology. KJPP welcomes the submission of all original research articles in the field of physiology and pharmacology, especially the new and innovative findings. The scope of researches includes the action mechanism, pharmacological effect, utilization, and interaction of chemicals with biological system as well as the development of new drug targets. Theoretical articles that use computational models for further understanding of the physiological or pharmacological processes are also welcomed. Investigative translational research articles on human disease with an emphasis on physiology or pharmacology are also invited. KJPP does not publish work on the actions of crude biological extracts of either unknown chemical composition (e.g. unpurified and unvalidated) or unknown concentration. Reviews are normally commissioned, but consideration will be given to unsolicited contributions. All papers accepted for publication in KJPP will appear simultaneously in the printed Journal and online.

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Editorial Board

2022 대한생리학회 임원명단

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김종환 김중수 나흥식 남숙현 남택상 류판동 민병일 박경표 박병림
박양생 박재식 박춘식 박형진 배선호 문창현 서덕준 서창국 신형철
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학술이사 배재성

학술위원 강다원 곽효범 김선광 김성준
박규상 박재형 배영민 손종우
우선희 이용석 이정범 임승순
홍재우 홍장원 황선욱

이 사 강다원 강대길 강동묵 강봉균 강 엽 공인덕 곽지연 구용숙 권성춘
권혁일 김경년 김나리 김동욱 김명준 김민선 김병주 김보경 김상정
김선희 김성주 김성준 김세훈 김양인 김영미 김용운 김원재 김의용
김재호 김정훈 김종연 김진혁 김창주 김형찬 나창수 박규상 박명규
박사훈 박소라 박소영 박우현 박원선 박종성 박중진 박지호 박진봉
방효원 배영민 배재성 배재훈 배혜란 백은주 서상원 서석효 서인석
송대규 신동민 심은보 안덕선 안도환 안동국 안승철 양훈모 염재범
오석배 우선희 우재석 우현구 윤신희 윤영욱 이 광 이덕주 이무열
이문영 이배환 이상진 이석호 이성중 이수환 이영호 이은희 이장헌
이정범 이지희 임인자 임채헌 장성호 장연진 장은화 전병화 전양숙
전제열 정성우 정승수 정승준 정진섭 정창섭 정한성 조영욱 조하나
진영호 천상우 최세영 최윤희 최장규 최한석 한 진 한상준 한승호
한인옥 한재희 한호재 한희철 호원경 홍성근

감 사 박중진 임승순

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Contents

Welcome Message

대한생리학회 회원여러분,

안녕하십니까?

지난 2년 동안 코로나 사태로 인해 저희 사회의 많은 면이 빠른 속도로 달라지면서 저희 일상 또한 큰 변화를 맞이하게 되어 비대면 활동이 대부분인 상황이 되었습니다. 저희 학회도 어쩔 수 없이 지난 2년 동안 hybrid 학회를 개최할 수 밖에 없었지만 금년에 드디어 10월 27일-28일 양일간 이화여대에서 회원여러분을 직접 뵐 수 있는 장을 만들 수 있게 되어 무척 기쁘게 생각합니다.

이제 저희 학회는 내년 FAOPS개최와 더불어 지속적이고 확장되고자 하는 염원을 담아 이번 학회 슬로건을 “Continued and Expanded”로 정하였습니다. 모든 학문분야가 그렇지만 융합이라는 측면에서 생리학회 또한 그 학문적 역할을 해야하는 상황에 처해 있으며 생리에 기반하고 연관된 융합과학이라는 측면으로 그 저변을 확대하는 것이 향후 학회가 지향해야할 방향일 수 밖에 없습니다.

이번 학회에서 plenary lecture, 인문학특강, 그리고 15개의 심포지움을 준비하여 과거를 돌아보고 미래를 지향하고자 하는 마음을 담았습니다. 특히 인문학분야는 최신 연구를 선도하기 위해 정신없이 달리는 회원여러분께 잠시나마 쉬는 마음으로 생리학을 돌이켜보는 시간을 가질 수 있을 것으로 생각합니다. 이번 가을 학회는 회원여러분의 우수한 학술활동과 임원분들의 헌신적 노력으로 이러한 프로그램을 담을 수 있어 무척 기쁘게 생각합니다.

아무쪼록 많이 참석하셔서 그동안 뵙지 못해 서운했던 마음을 달래며 이번에 열심히 준비한 프로그램으로 생리학의 향연을 마음껏 즐길 수 있기를 희망합니다.

그럼 가을 학회에서 뵙고 인사드리겠습니다.

대한생리학회 회 장 **임 채 헌**

Welcome Message

제74회 대학생리학회 학술대회 개최를 학회 회원 여러분들과 함께 진심으로 축하하며, 특히 코로나 팬데믹으로 인해 비대면으로 개최되다가 3년만에 대면으로 이화여자대학교 의과대학에서 학술대회를 개최하게 된 것을 기쁘게 생각합니다.

1886년 근대여성교육의 장을 연 이화여자대학교는 1887년 이화여자대학교 의과대학의 전신인 보구녀관(保救女館)을 시작으로, 1945년 행림원 의학부의 본격적인 정규 의학교육을 통해 의학 교육의 불모지였던 우리나라에 현대의학교육의 기초를 뿌리내리고 의학 발전을 선도하기 위하여 부단히 노력해 왔습니다. 그리고 2019년 2월 새로운 의대 부속병원인 이대서울병원 개원과 함께, 의과대학은 서울 강서구 마곡지구에 새 캠퍼스로 이전하였습니다. 신축 의학관은 학생중심의 최신 교육 환경과 첨단 연구 환경을 갖추어 새로운 도약의 발판을 마련하고 있습니다.

마곡으로 새롭게 이전한 이화여자대학교 의과대학에서 개최되는 이번 학술대회를 통해, 학회 슬로건 “Continued and Expanded”의 의미처럼 생명의 본질을 연구하는 생리학에 기반하여 생명과학, 더 나아가 인문학 영역까지 학문적 성과가 융합되고 확장되는 현장을 여러 회원분들과 함께 할 수 있기를 기대합니다.

학술대회의 성공적인 개최를 위해 수고해주신 대한생리학회 회장 그리고 학술이사를 포함한 임원진 및 학회 관계자 여러분께 깊은 감사의 말씀을 드립니다. 이화여자대학교 의과대학 생리학교실의 교수진 또한 학술대회의 성공적인 개최를 위해 최선을 다하겠습니다.

이화여자대학교 의과대학 생리학교실 주임교수 **박 성 희**

Schedule (일정표)

▶ 10월 27일 목요일

Time	Contents		
	Room A	Room B	Room C
09:20–09:30	개회사		
09:30–12:00	**S01.** Neurodevelopmental and Neurodegenerative diseases : 신경발달 및 신경퇴행성 질환	**S02.** Pathophysiology of tissue fibrosis : 조직섬유화 병태생리	**S03.** Smooth muscle cells and their regulator cells in physiology and pathophysiology : 평활근과 그 친구들
12:00–13:30		점심식사 및 포스터세션	이사회
13:30–14:10	**인문학 특강 I**		**좌장: 연세의대 안덕선**
	'The Kiss', a Beautiful Atlas of Early Human Development: Embryology embedded in art		유임주 (고려의대)
14:10–16:10	**S04.** Application of cutting-edge microscopy imaging technology to physiology research : 생리학 연구에 있어 첨단 현미경 이미징 기술의 활용	**S05.** Structure and function of the nephron : 신장 세관의 구조와 기능	**S06.** Pathophysiology of cardiovascular diseases : 심장질환의 병태생리
16:10–16:30	휴식		
16:30–18:30	**S07.** Cellular and Molecular regulation of synaptic functionality : 시냅스 기능	**S08.** Exercise physiology : 운동생리학	**S09.** Young Scientists session I : 신진과학자 세션1
18:30–	저녁만찬		

▶ 10월 28일 금요일

Time	Contents		
	Room A	Room B	Room C
09:00–11:30	**S10.** Organoids as in vitro models of human physiology : 인체 생리학 연구를 위한 3차원 오가노이드	**S11.** Physiology of Immune System : 면역생리	**S12.** Environmental Physiology 기후환경의 적응 : 건강한 삶을 위한 환경적응 전략
11:30–13:00		점심식사 및 포스터세션	
12:00–12:45			연구자와 함께하는 NRF 기초연구사업 간담회 **좌장: 배재성** 김성준 (한국연구재단 의약학 단장)
13:00–14:00	**Plenary Lecture**		**좌장: 조선의대 전제열**
	Propulsive colonic contractions are mediated by inhibition-driven post-stimulus responses that originate in interstitial cells of Cajal		고상돈 (Univ. of Nevada)
14:00–14:10	휴식		
14:10–14:50	**인문학특강 II**		**좌장: 울산의대 임채헌**
	혁명과 낭만의 과학, 그리고 과학사 속의 의과학자들		민태기 (에스앤에이치기술연구소)
14:50–15:00	휴식		
15:00–17:00	**S13.** Pathophysiology of airway disease – airway response to various stimuli : 기도질환의 병태생리	**S14.** Inflammation, aging, and cancer : 염증, 노화와 암	**S15.** Young Scientists session II : 신진과학자 세션2
17:00–17:20	유당학술상 강연		
17:20–17:30	신진생리학자상 강연		
17:30–18:20	시상 및 총회		
18:20–18:30	폐회사		

Venue Guide (학술대회장 안내)

층별 안내(Floor Plan) / 의과대학

3F	등록데스크 및 프리뷰 룸	Room A (계림홀)	Room B (Rm.301)	후원사 전시 부스 및 Poster Session
2F	병원 연결 통로 (의과대학 출입구)			
1F	Room C (Rm.105-106)			

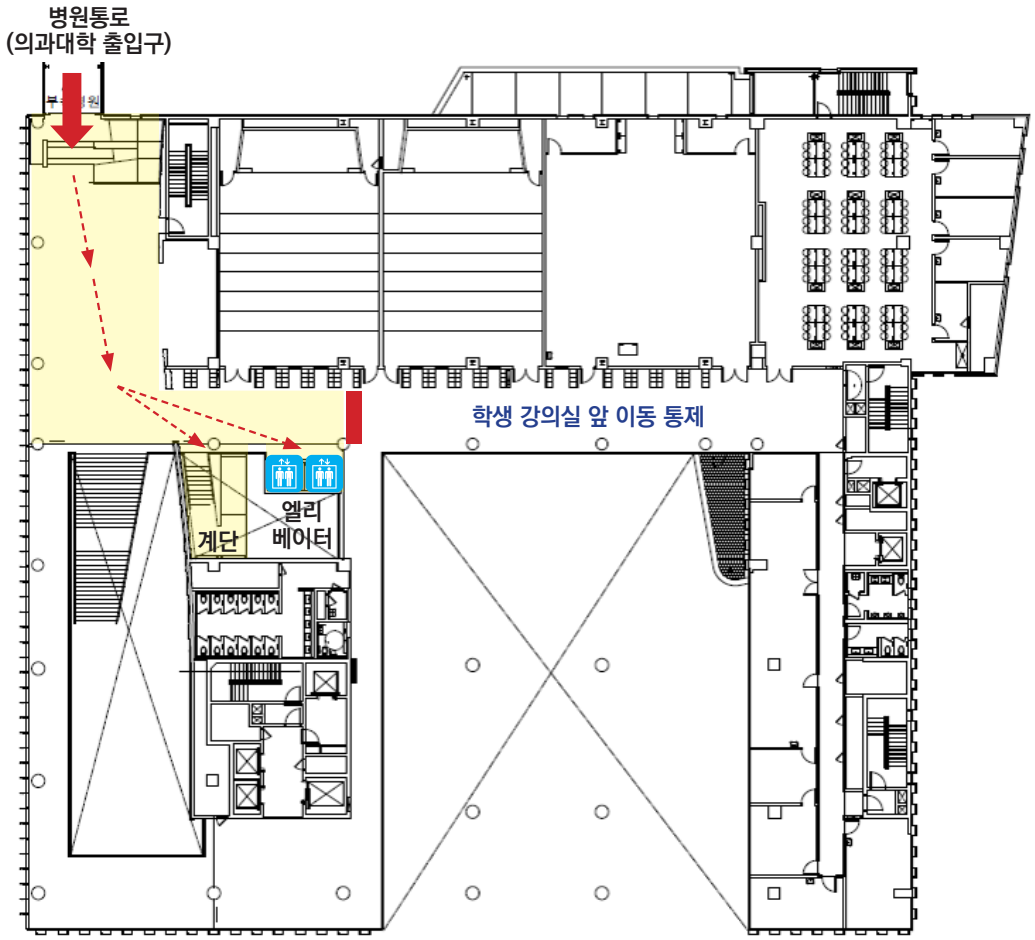
행사 장소 안내

3F

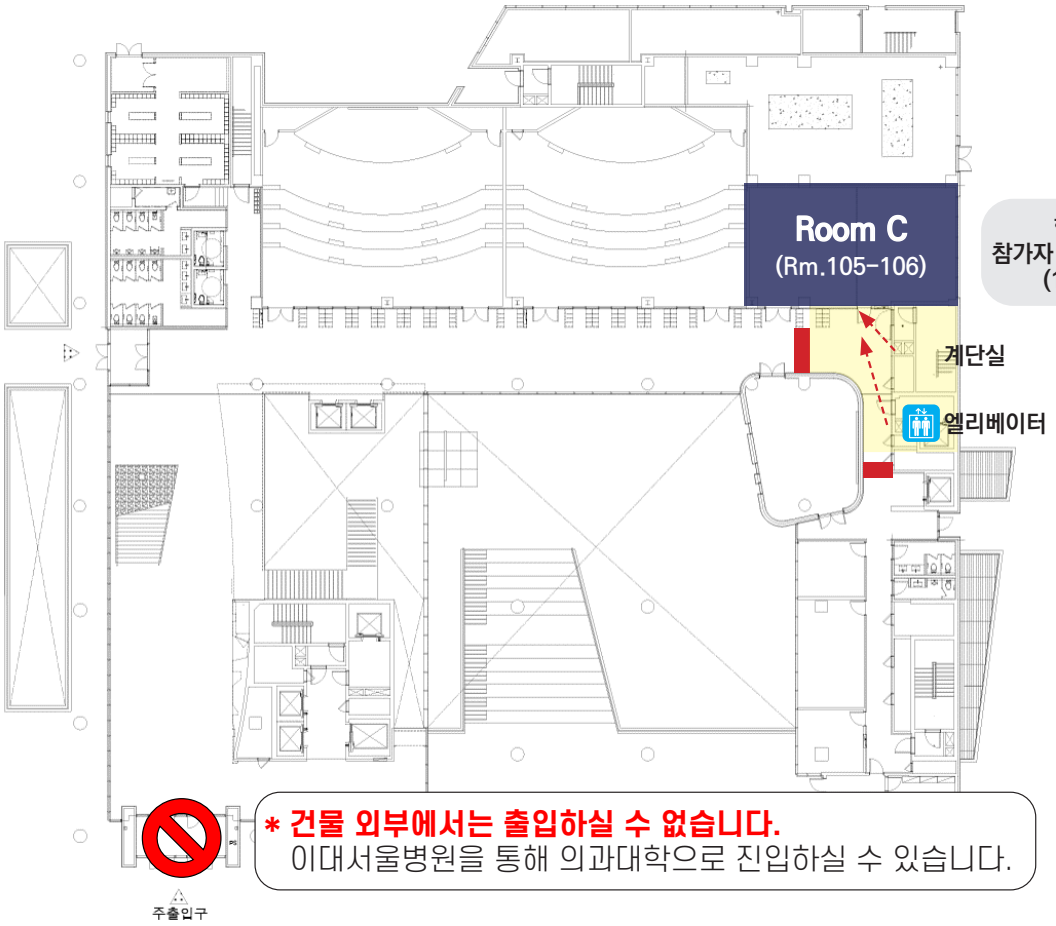
부스번호	후원사명
1	㈜ 싸이텍코리아
2	고마바이오텍 ㈜
3	라이노바이오 주식회사
4	㈜ 필코리아테크놀로지
5	주식회사 코넥스트

부스번호	후원사명
6	네스트사이언티픽코리아 유한회사
7	범문에듀케이션
8	㈜ 웅비메디텍
9	㈜ 바이오엔진

2F


병원통로
(의과대학 출입구)
학생 강의실 앞 이동 통제
엘리
베이터
계단

1F


Room C
(Rm.105-106)
학술대회
참가자 이용 가능 공간
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이대서울병원을 통해 의과대학으로 진입하실 수 있습니다.
주출입구

▶ Plenary Lecture (10월 28일 금요일)

Contents	
Plenary Lecture (13:00–14:00)	**Organizer: *전제열 (조선의대)***
Propulsive colonic contractions are mediated by inhibition-driven post-stimulus responses that originate in interstitial cells of Cajal	고상돈 *(Univ. of Nevada)*

▶ 인문학 특강 I (10월 27일 목요일)

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인문학 특강 I (13:30–14:10)	**Organizer: *안덕선 (연세의대)***
'The Kiss', a Beautiful Atlas of Early Human Development; Embryology embedded in art	유임주 *(고려의대)*

▶ 인문학 특강 II (10월 28일 금요일)

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인문학 특강 II (14:10–14:50)	**Organizer: *임채헌 (울산의대)***
혁명과 낭만의 과학, 그리고 과학사 속의 의과학자들	민태기 *(에스엔에이치기술연구소)*

▶ 연구자와 함께하는 NRF 기초연구사업 간담회 (10월 28일 금요일)

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연구자와 함께하는 NRF 기초연구사업 간담회 (12:00–12:45)	**Organizer: *배재성 (경북의대)***
연구자와 함께하는 NRF 기초연구사업 간담회	김성준 *(한국연구재단 의약학 단장)*

▶ Symposium (10월 27일 목요일)

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S01. Neurodevelopmental and Neurodegenerative diseases : 신경발달 및 신경퇴행성 질환 (09:30–12:00)	**Organizer: *장용우 (한양대), 홍규상 (한국과학기술연구원)***
1. Non-canonical codes for behavioral sequences in neurodevelopmental diseases	김정진 *(KIST 뇌과학연구소)*
2. Potent prostaglandin A1 for orphan nuclear receptor Nurr1 as a therapeutic target for Parkinson's Disease	장용우 *(한양의대)*
3. Conditional coexpression of AIMP2 and α-synuclein models Lewy body dementia	이연종 *(성균관의대)*
4. PET imaging reveals reactive astrocyte-mediated neuronal hypometabolism in Alzheimer's disease patients	남민호 *(KIST 뇌과학연구소)*
5. Hypothalamic neural stem cells in aging	김민수 *(KIST 뇌과학연구소)*

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S02. Pathophysiology of tissue fibrosis : 조직섬유화 병태생리 (09:30–12:00)	**Organizer: *임승순 (계명의대)***
1. Role of PRMT1 in NAFLD-associated hepatic fibrosis	구승회 *(고려대)*
2. Loss of SREBP-1c ameliorates iron-induced liver fibrosis via decrease of lipocalin-2	임승순 *(계명의대)*
3. Identification of novel targets for pulmonary fibrosis	이윤실 *(이대약대)*
4. Kidney fibrosis: is it reversible?	이은영 *(순천향의대)*
5. Targeting autotaxin improves pathophysiologic features of fibrocalcific aortic valve disease	장은주 *(울산의대)*

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S03. Smooth muscle cells and their regulator cells in physiology and pathophysiology : 평활근과 그 친구들 (09:30–12:00)	**Organizer: *김성준 (서울의대)***
1. Endothelium–mediated control of vascular contractility in physiological and pathophysiological conditions	서석효 *(이화의대)*
2. Vasodilatory effects and the underlying mechanisms of the medicinal plants extracts in rat mesenteric resistance arteries	최수경 *(연세의대)*
3. GI motility – organ level investigation	유승범 *(서울대병원 외과)*
4. The role of K_{ATP} channel activation in lymphatic contractile dysfunction associated with metabolic disease	김혜진 *(University of Missouri)*
5. Increased diphosphorylation of MLC2 is responsible for the impaired relaxation state of pulmonary arteries in the monocrotaline–induced pulmonary arterial hypertension	김성준 *(서울의대)*
6. Spontaneous vasomotion in human arteries and their ion channel–based mechanism in the smooth muscle	김영철 *(충북의대)*

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S04. Application of cutting–edge microscopy imaging technology to physiology research : 생리학 연구에 있어 첨단 현미경 이미징 기술의 활용 (14:10–16:10)	**Organizer: *김선광 (경희대)***
1. Role of lysophosphatidylcholine in neutrophil–gated immune response during sepsis	현영민 *(연세의대)*
2. In vivo two–photon microscopy imaging of glia–mediated synapse remodeling during chronic pain	김선광 *(경희대)*
3. Intelligence at the nanoscale: super–resolution imaging of brain structure and function	Valentin Nägerl *(University of Bordeaux)*
4. Brain micro–anatomy revealed by 2–photon shadow imaging *in vivo*	Yulia Dembitskaya *(University of Bordeaux)*

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S05. Structure and function of the nephron : 신장 세관의 구조와 기능 (14:10–16:10)	**Organizer: *김근호 (한양의대), 한기환 (이화의대)***
1. Structure of epithelial cells in nephron segments	한기환 *(이화의대)*
2. Single cell transcriptome reveals cell diversity in the kidney	박지환 *(광주과학기술원)*
3. Regulation of renal aquaporin–2 in kidney collecting duct	권태환 *(경북의대)*
4. Renal Na^+ transporters and salt–sensitive hypertension	김근호 *(한양의대)*

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S06. Pathophysiology of cardiovascular diseases : 심장질환의 병태생리 (14:10–16:10)	**Organizer: *홍장원 (경북의대)***
1. PSME4 degrades acetylated YAP1 in the nucleus of mesenchymal stem cells to induce cardiac commitment	엄광현 *(전남의대)*
2. Targeting smooth muscle cell phenotypic switching in vascular disease	허경선 *(충남대)*
3. Study of non–coding RNAs in diverse disease models	김영국 *(전남의대)*
4. Translational and clinical research of diabetic cardiomyopathy	조성우 *(인제대 일산 백병원)*

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S07. Cellular and Molecular regulation of synaptic functionality : 시냅스 기능 (16:30–18:30)	**Organizer: *김성현 (경희의대)***
1. Myristoylation–dependent palmitoylation of cyclin Y modulates synaptic protein trafficking, LTP, and spatial learning	박미경 *(KIST)*
2. Synaptic cell adhesion–like molecule Sy regulates excitatory synaptic density and activity–dependent gene expression	서영호 *(서울의대)*
3. Investigating physiological and pathophysiological features of neuronal mitochondria using advanced imaging and analysis tools	권석규 *(KIST)*
4. Modulating and monitoring the functionality of corticostriatal circuits using an electrostimulable microfluidic device	김성현 *(경희의대)*

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S08. Exercise physiology : 운동생리학 (16:30–18:30)	**Organizer: *한진 (인제의대), 곽효범 (인하대)***
1. Mechanoregulation of Endothelial Mitochondrial Phenotype	박준영 *(Baylor University)*
2. Exercise–induced muscle injury, muscle stem cell senescence, and novel therapeutic options	류동렬 *(성균관의대)*
3. Exercise type and exercise intensity on circulating myokines	이세원 *(인천대)*
4. Apelin–AMPK axis in mediating maternal exercise effects on offspring non–shivering thermogenesis	손준석 *(University of Maryland)*

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S09. Young Scientists session I : 신진과학자 세션1 (16:30–18:30)	**Organizer: *박규상 (연세대 원주의대)***
1. Update on Alzheimer's disease therapeutics	노지훈 *(고려의대)*
2. The activation of lysosomes decreases the tumor growth of colon cancer cells in vivo	홍재우 *(대구가톨릭의대)*
3. A growth–factor–activated lysosomal K^+ channel regulates Parkinson's pathology	위진홍 *(건국대학교)*
4. N–terminally truncated hERG channels generated by KCNH2 frameshift mutation (c.453delC) induces LQT phenotype in patient–derived iPSC–CMs	최성우 *(동국의대)*
5. A study for red blood cell as physiological marker	손민국 *(동아의대)*

▶ Symposium (10월 28일 금요일)

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S10. Organoids as in vitro models of human physiology : 인체 생리학 연구를 위한 3차원 오가노이드 (09:00–11:30)	**Organizer: *조인호 (범부처재생의료기술개발사업단)***
1. Modeling G2019S–LRRK2 Sporadic Parkinson's Disease in 3D Midbrain Organoids	김종필 *(동국대)*
2. Organoid model–based safety test	김시윤 *(건국대)*
3. Generation of human tonsil epithelial organoids as an ex vivo model for SARS–CoV–2 infection	유종만 *(차의대)*
4. Human pluripotent stem cell–derived intestinal organoids and their applications	손미영 *(한국생명공학연구원)*
5. 3D Bioprinting and its Applications	이준희 *(한국기계연구원)*

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S11. Physiology of Immune System : 면역생리 (09:00–11:30)	**Organizer: *홍재우 (대구가톨릭의대), 홍장원 (경북의대)***
1. Escherichia coli mimetic gold nanorod–mediated photo– and immunotherapy for treating cancer and its metastasis	진준오 *(울산의대)*
2. Oral microbiota–epithelium crosstalk regulates local and distal carcinogenesis	송나영 *(연세치대)*
3. The Emerging role of autophagy–related pathway in immune–driven malignant evolution of tumor cells	송권호 *(대구가톨릭의대)*
4. T cell's self–recognition: shaping diversity beyond specificity	조재호 *(전남의대)*
5. DDS using Salmonella for treatment of cancer	최현일 *(전남의대)*

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S12. Environmental Physiology 기후환경의 적응 : 건강한 삶을 위한 환경적응 전략 (09:00–11:30)	**Organizer: *이정범 (순천향의대)***
1. Physiological and psychological assessments for the Establishment of evidence–based forest healing programs	박수진 *(국립산림과학원)*
2. Splitting up exercise training in morning and afternoon for 14 days in a hot environment: consideration of total body fat and physical fitness	이주영 *(서울대)*
3. The role of occupational and environmental medicine in the subsea space creation and utilization technology development project	민영선 *(순천향대학 천안병원)*
4. Health monitoring through health assessment and bio–signals of habitat in subsea space	이화영 *(순천향대학 천안병원)*
5. The effect of the program to improve adaptation with the change of living environment	오세현 *(키위 심리발달 클리닉)*

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S13. Pathophysiology of airway disease – airway response to various stimuli : 기도질환의 병태생리 (15:00–17:00)	**Organizer: *박해심 (아주대병원 알레르기 내과)***
1. Emerging roles of Innate lymphoid cells in airway inflammations	김혜영 *(서울의대)*
2. Chronic cough and cough hypersensitivity	송우정 *(울산의대 내과)*
3. The clinical impact of air pollutants on COPD and its underlying pathophysiology	이세원 *(울산의대 내과)*
4. Gut–lung axis in adult asthma	박한기 *(경북의대 내과)*

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14. Inflammation, aging, and cancer : 염증, 노화와 암 (15:00–17:00)	**Organizer: *최윤희 (이화의대)***
1. Targeting the stress support pathways in senescence for healthy aging	강찬희 *(서울대)*
2. Exploring the molecular mechanisms to connect metabolism, DNA damage response, and Aging	이인혜 *(이화여대)*
3. The role of senescent tumor cells in cancer progression	박태준 *(아주의대)*
4. The role of caveolin–2 in age–related neuroinflammation	최윤희 *(이화의대)*

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S15. Young Scientists session II : 신진과학자 세션2 (15:00–17:00)	**Organizer: *강동묵 (성균관의대)***
1. CXCR4 Regulates Temporal Differentiation of Embryonic Salivary Glands via PRC1 Complex	이상우 *(서울치대)*
2. Anticancer effect of verteporfin on non–small cell lung cancer via downregulation of ANO1	우주한 *(동국의대)*
3. Intestinal microphysiological systems for investigating the interactions of the human gut with the gut microbes	김래현 *(홍익대)*
4. Systems analysis to dissect network mechanisms of drug resistance in cancer	박상민 *(충남대)*

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Yudang Academic Award (17:00–17:20)	
SREBP–1 regulates autophagy and macrophage polarization in metabolic diseases	임승순 *(계명대)*

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Young Physiologist Award (17:20–17:30)	
Mitochondrial energetic metabolism in Blood brain barrier maintenance	허준영 *(충남의대)*

Plenary Lecture

S29 PL-1 Propulsive colonic contractions are mediated by inhibition-driven post-stimulus responses that originate in interstitial cells of Cajal
Sang Don Koh
Department of Physiology and Cell Biology, University of Nevada, Reno School of Medicine, Reno, NV, USA

인문학 특강 I

S 29 'The Kiss', a Beautiful Atlas of Early Human Development; Embryology embedded in art
유임주
고려의대

인문학 특강 II

S 29 혁명과 낭만의 과학, 그리고 과학사 속의 의과학자들
민태기
에스엔에이치기술연구소

연구자와 함께하는 NRF 기초연구사업 간담회

S 29 연구자와 함께하는 NRF 기초연구사업 간담회
김성준
한국연구재단 의약학 단장

Symposium

S01. Neurodevelopmental and Neurodegenerative diseases : 신경발달 및 신경퇴행성 질환

S 30 S-1-1 Non-canonical codes for behavioral sequences in neurodevelopmental diseases
Jeongjin Kim[1,2]
[1]Brain Science Institute, Korea institute of science and technology (KIST), Seoul, South Korea, [2]Division of Bio-Medical Science & Technology, University of science and technology (UST), Daejeon, South Korea

S 30 S-1-2 Potent prostaglandin A1 for orphan nuclear receptor Nurr1 as a therapeutic target for Parkinson's Disease
Yongwoo Jang
Departments of Pharmacology, College of Medicine, Hanyang University, Seoul, Korea

S 30 S-1-3 Conditional coexpression of AIMP2 and α-synuclein models Lewy body dementia
Yunjong Lee
Department of Pharmacology, Sungkyunkwan University School of Medicine, Suwon, Korea

S 30 S-1-4 PET imaging reveals reactive astrocyte-mediated neuronal hypometabolism in Alzheimer's disease patients
Min-Ho Nam
Brain Science Institute, Korea Institute of Science and Technology (KIST), Seoul, Republic of Korea

S 31 S-1-5 Hypothalamic neural stem cells in aging
Min Soo Kim
Brain Science Institute, Korea Institute of Science and Technology (KIST), Seoul, Korea,
Division of Bio-Medical Science & Technology, KIST school University of Science and Technology (UST), Seoul, Korea

S02. Pathophysiology of tissue fibrosis : 조직섬유화 병태생리

S 31 S-2-1 Role of PRMT1 in NAFLD-associated hepatic fibrosis
Dahee Choi, Seung-Hoi Koo
Department of Life Sciences, Korea University, Seoul, Korea

S 31 S-2-2 Loss of SREBP-1c ameliorates iron-induced liver fibrosis via decrease of lipocalin-2
Seung-Soon Im
Departments of Physiology, Keimyung University School of Medicine, Deagu, Korea

S 31 S-2-3 Identification of novel targets for pulmonary fibrosis
Yun-Sil Lee
College of Pharmacy, Ewha Womans University, Seoul, Korea

S 32 S-2-4 Kidney fibrosis: is it reversible?
Eun Young Lee
Division of Nephrology, Soonchunhyang University Cheonan Hospital, Cheonan, Korea and BK21 Four project, Soonchunhyang University College of Medicine, Cheonan, Korea

S 32 S-2-5 Targeting autotaxin improves pathophysiologic features of fibrocalcific aortic valve disease
Eun-Ju Chang
Department of Biomedical Sciences, Asan Medical Center, University of Ulsan College of Medicine, Seoul, Korea

S03. Smooth muscle cells and their regulator cells in physiology and pathophysiology : 평활근과 그 친구들

S 32 S-3-1 Endothelium-mediated control of vascular contractility in physiological and pathophysiological conditions
Suk Hyo Suh
Department of Physiology, Medical School, Ewha Womans University, Seoul, Korea

S 33 S-3-2 Vasodilatory effects and the underlying mechanisms of the medicinal plants extracts in rat mesenteric resistance arteries
Soo-Kyoung Choi
Departments of Physiology, Yonsei University College of Medicine, Seoul, Korea

S 33 S-3-3 GI motility – organ level investigation
Seung-Bum Ryoo
Division of Colorectal Surgery, Department of Surgery, Seoul National University College of Medicine, Seoul, Korea

S 33 S-3-4 The role of K_{ATP} channel activation in lymphatic contractile dysfunction associated with metabolic disease
Hae Jin Kim
Department of Medical Physiology and Pharmacology, School of Medicine, University of Missouri, Columbia, MO, USA

S 34 S-3-5 Increased diphosphorylation of MLC2 is responsible for the impaired relaxation state of pulmonary arteries in the monocrotaline-induced pulmonary arterial hypertension
Sung Joon Kim
Department of Physiology, Ischemic/Hypoxic Disease Institute, Seoul National University College of Medicine, Seoul, Republic of Korea

S 34 S-3-6 Spontaneous vasomotion in human arteries and their ion channel-based mechanism in the smooth muscle
Young Chul Kim[1], Dae Hoon Kim[2], Jin Young Choi[3], Su Mi Kim[3],
Seung Myeung Son[4], Ra Young You[1], Chan Hyung Kim[5], Woong Choi[5], Hun Sik Kim[5], Wen-Xie Xu[6], Seung Hwa Hong[3], Sang Jin Lee[1],
Hyo-Yung Yun[2]
[1]Dept. of Physiology, College of Medicine, CBNU, Cheongju, Korea, [2]Department of Surgery, CBNU, [3]Department of OBGY, CBNU, [4]Department of Pathology, CBNU,
[5]Dept. of Pharmacology, CBNU, [6]Dept. of Physiology, College of Medcine, Shanghai Jiaotong University, Shanghai, China

S04. Application of cutting-edge microscopy imaging technology to physiology research : 생리학 연구에 있어 첨단 현미경 이미징 기술의 활용

S 34 S-4-1 Role of lysophosphatidylcholine in neutrophil-gated immune response during sepsis
Young-Min Hyun
Department of Anatomy and BK21 PLUS Project for Medical Science, Yonsei University College of Medicine, Seoul Korea

S 35 S-4-2 In vivo two-photon microscopy imaging of glia-mediated synapse remodeling during chronic pain
Sun Kwang Kim
Departments of Physiology, Kyung Hee University College of Korean Medicine, Seoul, Korea

S 35 S-4-3 Intelligence at the nanoscale: super-resolution imaging of brain structure and function
U. Valentin Nägerl
Interdisciplinary Institute for Neuroscience, University of Bordeaux, Bordeaux, France

S 35 S-4-4 Brain micro-anatomy revealed by 2-photon shadow imaging *in vivo*
Yulia Dembitskaya[1], Guillaume Le Bourdelles[1], Stéphane Bancelin[1], Jordan Girard[1], Marie Sato-Fitoussi[1], Sun Kwang Kim[1,2],
U. Valentin Nägerl[1]
[1]Interdisciplinary Institute for Neuroscience, University of Bordeaux/CNRS, France, [2]Department of Physiology, Kyung Hee University College of Korean Medicine, Seoul, Korea

S05. Structure and function of the nephron : 신장 세관의 구조와 기능

S 35 S-5-1 Structure of epithelial cells in nephron segments
Ki-Hwan Han
Department of Anatomy, Ewha Womans University, Seoul, Korea

S 36 S-5-2 Single cell transcriptome reveals cell diversity in the kidney
Jihwan Park
School of Life Sciences, Gwangju Institute of Science and Technology, Republic of Korea

S 36 S-5-3 Regulation of renal aquaporin-2 in kidney collecting duct
Tae-Hwan Kwon
Department of Biochemistry and Cell Biology, School of Medicine, Kyungpook National University, Korea

S 36 S-5-4 Renal Na^+ transporters and salt-sensitive hypertension
Gheun-Ho Kim
Departments of Internal Medicine, Hanyang University College of Korean Medicine, Seoul, Korea

S06. Pathophysiology of cardiovascular diseases : 심장질환의 병태생리

S 37 S-6-1 PSME4 degrades acetylated YAP1 in the nucleus of mesenchymal stem cells to induce cardiac commitment
Gwang Hyeon Eom
Departments of Pharmacology, Chonnam National University Medical School, Gwangju, Korea

S 37 S-6-2 Targeting smooth muscle cell phenotypic switching in vascular disease
Kyung-Sun Heo
Departments of Pharmacology, Chungnam National University College of Medicine, Daejeon, Korea

S 37 S-6-3 Study of non-coding RNAs in diverse disease models
Young-Kook Kim
Departments of Biochemistry, Chonnam National University Medical School, Jeollanam-do, Korea

S 37 S-6-4 Translational and clinical research of diabetic cardiomyopathy
Sung Woo Cho[1,2], Hyoung Kyu Kim[2], Jin Han[2], Chang-Myung Oh[3]
[1]Division of Cardiology, Department of Internal Medicine, Inje University College of Medicine, Ilsan Paik Hospital, Cardiac & Vascular Center, Goyang, Korea,
[2]Cardiovascular and Metabolic Disease Center, Smart Marine Therapeutics Center, Inje University College of Medicine, Busan, Korea,
[3]Department of Biomedical Science and Engineering, Gwangju Institute of Science and Technology, Gwangju

S07. Cellular and Molecular regulation of synaptic functionality : 시냅스 기능

S 38 S-7-1 Myristoylation-dependent palmitoylation of cyclin Y modulates synaptic protein trafficking, LTP, and spatial learning
Mikyoung Park
Brain Science Institute, Korea Institute of Science and Technology, Seoul, Korea

S 38 S-7-2 Synaptic cell adhesion-like molecule Sy regulates excitatory synaptic density and activity-dependent gene expression
Young Ho Suh
Department of Biomedical Sciences, Seoul National University College of Medicine, Korea

S 38 S-7-3 Investigating physiological and pathophysiological features of neuronal mitochondria using advanced imaging and analysis tools
Seok-Kyu Kwon
Brain Science Institute, KIST, Seoul, Korea

S 38 S-7-4 Modulating and monitoring the functionality of corticostriatal circuits using an electrostimulable microfluidic device
Sung Hyun Kim
Departments of Physiology, Kyung Hee University College of Medicine, Seoul, Korea

S08. Exercise Physiology : 운동생리학

S 39 S-8-1 Mechanoregulation of Endothelial Mitochondrial Phenotype
Joon Young Park
Departments of Health, Human Performance, and Recreation, Robbins College of Health and Human Sciences, Baylor University, Waco, Texas, U.S.A.

S 39 S-8-2 Exercise-induced muscle injury, muscle stem cell senescence, and novel therapeutic options
Dongryeol Ryu
Departments of Molecular Cell Biology, Sungkyunkwan University (SKKU) School of Medicine, Suwon Korea

S 39 S-8-3 Exercise type and exercise intensity on circulating myokines
Sewon Lee
Division of Sport Science, College of Arts & Physical Education, Incheon National University, Incheon, Korea

S 39 S-8-4 Apelin-AMPK axis in mediating maternal exercise effects on offspring non-shivering thermogenesis
Jun Seok Son
Department of Physiology, Department of Obstetrics, Gynecology, and Reproductive Sciences, University of Maryland School of Medicine, Baltimore, Maryland, United States

S09. Young Scientists session I : 신진과학자 세션1

S 40 S-9-1 Update on Alzheimer's disease therapeutics
Jee Hoon Roh
Departments of Physiology, Korea University College of Medicine, Seoul, Korea,
Departments of Biomedical Sciences, BK21 4Plus, Korea University Graduate School of Medicine, Seoul, Korea,
Departments of Neurology, Korea University Anam Hospital, Seoul, Korea

S 40 S-9-2 The activation of lysosomes decreases the tumor growth of colon cancer cells in vivo
Jaewoo Hong
Departments of Physiology, Daegu Catholic University School of Medicine, Daegu, Korea

S 40 S-9-3 A growth-factor-activated lysosomal K^+ channel regulates Parkinson's pathology
Jinhong Wie
Department of Physiology, Konkuk University School of Medicine, Chungju, Korea

S 40 S-9-4 N-terminally truncated hERG channels generated by KCNH2 frameshift mutation (c.453delC) induces LQT phenotype in patient-derived iPSC-CMs
Na Kyeong Park[1], Sung Joon Kim[1], Sung Woo Choi[2]
[1]Department of Physiology, Department of Biomedical Sciences, Seoul National University College of Medicine, Korea, [2]Departments of Physiology, Dongguk University College of Medicine, Gyengju, Korea

S 41 S-9-5 A study for red blood cell as physiological marker
Minkook Son
Department of Physiology, College of Medicine, Dong-A University, Busan, Korea

S10. Organoids as in vitro models of human physiology : 인체 생리학 연구를 위한 3차원 오가노이드

S 41 S-10-1 Modeling G2019S-LRRK2 Sporadic Parkinson's Disease in 3D Midbrain Organoids
Jongpil Kim
Departments of Chemistry & Biomedical Engineering, Dongguk, Seoul, Korea

S 41 S-10-2 Organoid model-based safety test
C-Yoon Kim
College of Veterinary Medicine, Konkuk University, Seoul, Republic of Korea

S 41 S-10-3 Generation of human tonsil epithelial organoids as an ***ex vivo*** model for SARS-CoV-2 infection
Jongman Yoo
CHA University, Seongnam, Republic of Korea,
ORGANOIDSCIENCES, Ltd., Seongnam, Republic of Korea

S 42 S-10-4 Human pluripotent stem cell-derived intestinal organoids and their applications
Mi-Young Son
Korea Research Institute of Bioscience and Biotechnology (KRIBB), Daejeon, Republic of Korea, KRIBB School of Bioscience, Korea University of Science and Technology (UST), Daejeon, Republic of Korea

S 42 S-10-5 3D Bioprinting and its Applications
Junhee Lee*, Seunghun Son, SuA Park
Departments of Nature-Inspired System and Application, Korea Institute of Machinery & Materials, Daejeon, Korea

S11. Physiology of Immune System : 면역생리

S 42 S-11-1 Escherichia coli mimetic gold nanorod-mediated photo- and immunotherapy for treating cancer and its metastasis
Jun-O Jin
Department of Microbiology, University of Ulsan College of Medicine, ASAN medical center, Seoul, Korea

S 43 S-11-2 Oral microbiota-epithelium crosstalk regulates local and distal carcinogenesis
Na-Young Song
Department of Oral Biology, Yonsei University College of Dentistry, Republic of Korea

S 43 S-11-3 The Emerging role of autophagy-related pathway in immune-driven malignant evolution of tumor cells
Kwon-Ho Song
Daegu Catholic University School of Medicine

S 43 S-11-4 T cell's self-recognition: shaping diversity beyond specificity
Jae Ho Cho
Medical Research Center, Department of Microbiology & Immunology, Chonnam National University Medical School, Hwasun Hospital, Hwasun-up, Jeonnam, Korea

S 43 S-11-5 DDS using Salmonella for treatment of cancer
Hyonil Choy
Departments of Microbiology, Chonnam University Medical School, Kwangju, Korea

S12. Environmental Physiology : 환경생리학

S 44 S-12-1 Physiological and psychological assessments for the Establishment of evidence-based forest healing programs
Sujin Park, Yeji Choi, Geonwoo Kim, Eunsoo Kim, Soojin Kim
Forest Human Service Division, Future Forest Strategy Department, National Institute of Forest Science, Seoul, Korea

S 44 S-12-2 Splitting up exercise training in morning and afternoon for 14 days in a hot environment: consideration of total body fat and physical fitness
Joo Young Lee
College of Human Ecology, Seoul National University, Seoul, Korea

S 44 S-12-3 The role of occupational and environmental medicine in the subsea space creation and utilization technology development project
Young-Sun Min, In Ho Lee
Department of Occupational and Environmental Medicine, Soonchunhyang University Cheonan Hospital, Cheonan, Korea

S 45 S-12-4 Health monitoring through health assessment and bio-signals of habitat in subsea space
Hwa-Young Lee
Department of Psychiatry, Soonchunhyang University Cheonan Hospital, Cheonan, Korea

S 45 S-12-5 The effect of the program to improve adaptation with the change of living environment
Se-Hyun Oh
Kiwi Development Clinic, Suwon, Republic of Korea

S13. Pathophysiology of airway disease - airway response to various stimuli : 기도질환의 병태생리

S 45 S-13-1 Emerging roles of Innate lymphoid cells in airway inflammations
Hye Young Kim
Laboratory of Mucosal Immunology, Department of Biomedical Sciences, Seoul National University College of Medicine, Seoul, Korea

S 45 S-13-2 Chronic cough and cough hypersensitivity
Woo-Jung Song
Departments of Allergy and Clinical Immunology, University of Ulsan College of Medicine, Asan Medical Center, Seoul, Korea

S 46 S-13-3 The clinical impact of air pollutants on COPD and its underlying pathophysiology
Sei Won Lee
Departments of Pulmonology and Critical Care Medicine, University of Ulsan, Asan Medical Center, Seoul, Korea

S 46 S-13-4 Gut-lung axis in adult asthma
Han-Ki Park
Department of Allergy and Clinical Immunology, School of Medicine, Kyungpook National University, Kyungpook National University Chilgok Hospital, Daegu, Korea

S14. Inflammation, aging, and cancer : 염증, 노화와 암

S 46 S-14-1 Targeting the stress support pathways in senescence for healthy aging
Chanhee Kang
School of Biological Sciences, Seoul National University, Seoul, Korea,
Center for Systems Geroscience, Seoul National University, Seoul, Korea

S 46 S-14-2 Exploring the molecular mechanisms to connect metabolism, DNA damage response, and Aging
In Hye Lee
Department of Life Science, EWHA Womans University, Seoul, South

S 47 S-14-3 The role of senescent tumor cells in cancer progression
Sun Sang Park[1], Young Hwa Kim[1], Yong Won Choi[2], Jang-Hee Kim[3], Tae Jun Park[1,4]
[1]Department of Biochemistry and Molecular Biology, Ajou University, School of Medicine, [2]Department of Hematology-Oncology, Ajou University School of Medicine, [3]Department of Pathology, Ajou University School of Medicine, [4]Inflammaging translational research center, Ajou University Medical Center, Suwon

S 47 S-14-4 The role of caveolin-2 in age-related neuroinflammation
Youn-Hee Choi
Department of Physiology, Inflammation-Cancer Microenvironment Research Center, Ewha Womans University College of Medicine, Seoul, Korea

S15. Young Scientists session II : 신진과학자 세션2

S 47 S-15-1 CXCR4 Regulates Temporal Differentiation of Embryonic Salivary Glands via PRC1 Complex
Sang-Woo Lee, Junchul Kim, Kyungpyo Park*
Department of Oral Physiology, School of Dentistry, Seoul National University

S 48 S-15-2 Anticancer effect of verteporfin on non-small cell lung cancer via downregulation of ANO1
JooHan Woo
Department of Physiology, Dongguk University College of Medicine, Gyeongju, the Republic of Korea

S 48 S-15-3 Intestinal microphysiological systems for investigating the interactions of the human gut with the gut microbes
Raehyun Kim, Nancy L. Allbritton
Departments of Biological and Chemical Engineering, Hongik University, Sejong, Korea

S 48 S-15-4 Systems analysis to dissect network mechanisms of drug resistance in cancer
Sang-Min Park
College of Pharmacy, Chungnam University, Daejeon, Korea

Yudang Academic Award

S 49 SREBP-1 regulates autophagy and macrophage polarization in metabolic diseases
Seung-Soon Im
Department of Physiology, Keimyung University School of Medicine

Young Scientist Session

S 49 Mitochondrial energetic metabolism in Blood brain barrier maintenance
Jun Young Heo
[1]Department of Medical Science, [2]Department of Biochemistry, [3]Infection Control Convergence Research Center, Chungnam National University School of Medicine, Daejeon, South Korea

Poster Presentation

P01: Basic neurophysiology and Pain

S 50 P-01-001 Improvement of cognitive impairments in post-menopausal depression via restoration of hippocampal silent synapses with (-)-gallocatechin gallate-enriched green tea
Sohyun Kim[1], Sukjin Ko[2], Ji-woong Ahn[2], Young-hwan Kim[2], Ji-hyun Jeong[2], Seungsoo Chung[1,2]
[1]Brain Korea 21 Plus Project for Medical Science, Department of Physiology Yonsei University College of Medicine, Seoul, Republic of Korea, [2]BnH Research Co., LTD. Goyang, Republic of Korea

S 50 P-01-002 Inhibitors of angiotensin converting enzyme (ACE) activates the expression of substance P or bradykinin in cultured astrocyte of mice
Jae-Gyun Choi[1], Dong-Wook Kang[1], Hyun Jin Shin[1], Miae Lee[1], Sheu-Ran Choi[2], Jung-Mo Hwang[3], Cuk-Seong Kim[1], Sang Do Lee[1], Byeong Hwa Jeon[1], Hyun-Woo Kim[1]
[1]Department of Physiology and Medical Science College of Medicine and Brain Research Institute, Chungnam National University, [2]Department of Pharmacology Catholic Kwandong University College of Medicine, [3]Department of Orthopaedic Surgery Chungnam National University School of Medicine

S 50 P-01-003 Generation of an optimized autaptic culture system for studying synaptic functions in autonomic ganglia
Seong Jun Kang[1], Choong-Ku Lee[2], Huu Son Nguyen[1], Jeong Seop Rhee[2], Seong-Woo Jeong[1]
[1]Department of Physiology Yonsein University Wonju College of Medicine, Wonju, Republic of Korea, [2]Department of Molecular Neurobiology Max plank Institute for Multidisciplinary Sciences, Göttingen, Germany

S 51 P-01-004 Multiplexed representation of itch and pain and their interaction in the primary somatosensory cortex
Seunghui Woo[1], Yoo Rim Kim[2,3], Myeong Seong Bak[1], Geehoon Chung[1,4], Sang Jeong Kim[2,3,5], Sun Kwang Kim[1,4]
[1]Department of Science in Korean Medicine, Graduate School Kyung Hee University, [2]Department of Physiology Seoul National University College of Medicine, [3]Neuroscience Research Institute Seoul National University College of Medicine, [4]Department of Physiology, College of Korean Medicine Kyung Hee University, [5]Department of Biomedical Sciences Seoul National University College of Medicine

S 51 P-01-005 Inwardly rectifying potassium channel, Kir4.1 mediates Ca^{2+} entry in the satellite glial cells of sympathetic ganglia under a hypokalemic condition
Huu Son Nguyen, Seong Jun Kang, Kyu-Sang Park, Seong-Woo Jeong
Department of Physiology Yonsei University Wonju College of Medicine, Wonju, Republic of Korea

S 51 P-01-006 Analgesic effect of intermittent fasting-related orexin A pathway on the formalin-induced acute pain.
Hyun Jin Shin, Jae-Gyun Choi, Dong-Wook Kang, Miae Lee, Cuk-Seong Kim, Sang Do Lee, Byeong Hwa Jeon, Hyun-Woo Kim
Physiology and Medical Sciences, College of Medicine and Brain Research Institute, Chungnam National University Daejeon, Korea

S 51 P-01-007 Porphyromonas gingivalis directly interacts with nociceptive sensory neurons to produce analgesic effects in chronic inflammatory pain condition
Sena Chung[1], Doyun Kim[2], Yeon Kyeong Ko[3], Hayun Kim[4], Hyun Young Kim[3], Bong-Kyu Choi[3], Youngnim Choi[3], Seog Bae Oh[2,4]
[1]Department of Brain and Cognitive Sciences College of Natural Sciences, Seoul National University, [2]Department of Neurobiology and Physiology School of Dentistry and Dental Research Institute, Seoul National University, [3]Department of Immunology and Molecular Microbiology School of Dentistry and Dental Research Institute, Seoul National University, [4]Interdisciplinary Program in Neuroscience College of Natural Sciences, Seoul National University

S 52 P-01-008
Effect of exercise on the reserpine-induced pain and depression-like responses via the modulation of brain-derived neurotrophic factor expression in mice
Dong-Wook Kang, Jae-Gyun Choi, Hyun Jin Shin, Miae Lee, Cuk-Seong Kim, Sang Do Lee, Byeong Hwa Jeon, Hyun-Woo Kim
Department of Physiology and Medical Science College of Medicine and Brain Research Institute, Chungnam National University

S 52 P-01-009
Electrical stimulation of the insular cortex attenuates neuropathic pain via modulation of synaptic plasticity
Kyeongmin Kim[1], Myeounghoon Cha[1], Guanghai Nan[1,2], Leejeong Kim[1,2], Heejin Jeong[1], Bae Hwan Lee[1,2]
[1]Department of Physiology Yonsei University College of Medicine, Seoul, Republic of Korea, [2]Brain Korea 21 PLUS Project for Medical Science Yonsei University College of Medicine, Seoul, Republic of Korea

S 53 P-01-010
Inhibition of Nav1.7 channels in the trigeminal ganglion alleviates pulpitis-induced pain in rats
Guanghai Nan[1,2], Kyeongmin Kim[1], Leejeong Kim[1,2], Heejin Jeong[1], Myeounghoon Cha[1], Bae Hwan Lee[1,2]
[1]Department of Physiology Yonsei University College of Medicine, Seoul, Republic of Korea, [2]Brain Korea 21 PLUS Project for Medical Science Yonsei University College of Medicine, Seoul, Republic of Korea

S 53 P-01-011
Long-axon adjacent local lymphadenopathy is responsible for vincristine-induced pain via mediating infiltration of the CXCL13+ CX3CR1+ macrophage into the sciatic nerve
Wheedong Kim[1], Da Hee Roh[2], Tae Hyun Lee[2], Doyun Kim[2], Seo Yeon Yoon[3], Seog Bae Oh[2]
[1]Department of Brain and Cognitive Sciences College of Natural Sciences, Seoul National University, Seoul, South Korea, [2]Department of Neurobiology and Physiology School of Dentistry and Dental Research Institute, Seoul National University, Seoul, South Korea, [3]Department of pet total care Daejeon Health Institute of Technology, Daejeon, South Korea

S 53 P-01-012
Modulation of neuropathic pain through regulation of glial cells in the insular cortex
Leejeong Kim[1,2], Kyeongmin Kim[1], Guanghai Nan[1,2], Heejin Jeong[1], Myeounghoon Cha[1], Bae Hwan Lee[1,2]
[1]Department of Physiology Yonsei University College of Medicine, Seoul, Republic of Korea, [2]Brain Korea 21 PLUS Project for Medical Science Yonsei University College of Medicine, Seoul, Republic of Korea

S 54 P-01-013
Clemastine attenuates paclitaxel-induced neuropathic pain by improving myelin repair in the sciatic nerve
Heejin Jeong[1], Kyeongmin Kim[1], Guanghai Nan[1,2], Leejeong Kim[1,2], Myeounghoon Cha[1], Bae Hwan Lee[1,2]
[1]Department of Physiology Yonsei University College of Medicine, Seoul, Republic of Korea, [2]Brain Korea 21 PLUS Project for Medical Science Yonsei University College of Medicine, Seoul, Republic of Korea

S 54 P-01-014
A semi-automated cell counting method for TH-positive dopaminergic neurons in a mouse model of Parkinson's disease using convolutional neural networks
Myeong Seong Bak[1], Doyun Kim[2], Haney Park[1], In Seon Baek[3], Sora Ahn[4], Hi-Joon Park[4], Sun Kwang Kim[1,2,3]
[1]Neurogrin Inc. Seoul, Korea, [2]Department of Physiology College of Korean Medicine, Kyung Hee University, Seoul, Korea, [3]Department of Science in Korean Medicine Graduate School, Kyung Hee University, Seoul, Korea, [4]Acupuncture & Meridian Science Research Center Kyung Hee University, Seoul, Korea

S 54 P-01-015
mGluR5-mediated deactivation of mPFC in the neuropathic pain mice
Mirae Jang[1,2], Sang Jeong Kim[1,2]
[1]Department of Physiology Seoul National University College of Medicine, Seoul, Korea, [2]Department of Biomedical Sciences Seoul National University College of Medicine, Seoul, Korea

S 55 P-01-016
Real-time decoding of spontaneous pain from two-photon microscopy images of brain cellular calcium using deep learning
Heera Yoon[1], Myeong Seong Bak[1], Seung Ha Kim[3], Haney Park[1], Geehoon Chung[1,2], Sang Jeong Kim[3], Sun Kwang Kim[1,2]
[1]Neurogrin Inc. Seoul, Korea, [2]Physiology College of Korean Medicine, Kyung Hee University, Seoul, Korea, [3]Physiology Seoul National University School of Medicine, Seoul, Korea

S 55 P-01-017
Investigation of C. elegans learning and memory regulation mechanism by Mitochondrial Calcium Uniporter (MCU-1)
Hee Kyung Lee, Saebom Kwon, Jessica Antonio, Kyoung-hye Yoon
Department of Physiology Mitohormesis Research Center, Yonsei University Wonju College of Medicine

S 55 P-01-018
Nuclear hormone receptor NHR-49 in the body cavity neurons mediate pathogen avoidance in C. elegans
Saebom Kwon, Hee Kyung Lee, Jessica Antonio, Kyoung-hye Yoon
Department of Physiology Mitohormesis Research Center, Yonsei University Wonju College of Medicine, Wonju, South Korea

S 55 P-01-019
Rapamycin, an mTOR inhibitor suppresses orofacial neuropathic pain and p-mkk4/p-p38 MAPK-mediated microglial activation in TNC in trigeminal nerve injured mice
Ji-Hee Yeo, Dae-Hyun Roh
Department of Oral Physiology School of Dentistry

S 56 P-01-020
Involvement of reactive oxygen species in cocaine addiction-like behavior in rats
Hee Young Kim[1], Mi Jin Yeo[3], Hyung Kyu Kim[2]
[1]Department of Physiology, College of Medicine Yonsei University, [2]Department of Oral Physiology, School of Dentistry Kyungpook National University, [3]Department of Physiology, College of Oriental Medicine Daegu Haany University

S 56 P-01-021
Homeostatic plasticity of Purkinje cell excitability balances fear-related memory
Jaegeon Lee[1,2], Seung Ha Kim[1,2], Dong Cheol Jang[1,3], Mirae Jang[1,2], Myeong Seong Bak[1,2], Hyun Geun Shim[1,2], Yong-Seok Lee[1,2], Sang Jeong Kim[1,2]
[1]Department of Physiology Seoul National University College of Medicine, [2]Department of Biomedical Science Seoul National University College of Medicine, [3]Department of Brain and Cognitive Science Seoul National University College of Medicine

S 56 P-01-022 Effect of monosodium urate on the terminal of substantia gelatinosa neurons of the trigeminal subnucleus caudalis in juvenile mice
Seon-Ah Park, Soo-Joung Park, Seong-Kyu Han
Department of Oral Physiology School of Dentistry and Institute of Oral Bioscience, Jeonbuk National University, Jeonju, Republic of Korea

S 57 P-01-023 Effect of alpha-lipoic acid on substantia gelatinosa neurons of the trigeminal subnucleus caudalis in mice
Seon-Hui Jang[1], Seong Kyu Han[1], Won Jung[2]
[1]Department of Oral Physiology School of Dentistry and Institute of Oral Bioscience, Jeonbuk National University, Jeonju, Republic of Korea, [2]Department of Oral Medicine School of Dentistry and Institute of Oral Bioscience, Jeonbuk National University, Jeonju, Republic of Korea

S 57 P-01-024 The inhibition of neuronal peroxisome proliferator-activated receptor-γ attenuates motor function improvement after spinal cord injury in rats
Youngkyung Kim[1,2], Kyu-won Park[2], Eunji Lee[2], Young Wook Yoon[2]
[1]Institute of Neuroscience, Department of Physiology, Korea University College of Medicine, Seoul, Korea, [2]Medical Science Research Center, Korea University College of Medicine, Seoul, Korea

P02: Neuronal pathophysiology

S 57 P-02-001 N-AS-triggered SPMs are direct regulators of microglia in a mouse of Alzheimer's disease
Kang Ho Park[1], Md Riad Chowdhury[1], Hee Kyung Jin[2], Jae-sung Bae[1]
[1]Department of Physiology School of Medicine, Kyungpook National University, [2]Department of Laboratory Animal Medicine College of Veterinary Medicine, Kyungpook National University

S 57 P-02-002 Discovery of a novel dual-action small molecule that improves multiple Alzheimer's disease pathologies
Kang Ho Park[1], Md Riad Chowdhury[1], Hee Kyung Jin[2], Jae-sung Bae[1]
[1]Department of Physiology School of Medicine, Kyungpook National University, [2]Department of Laboratory Animal Medicine College of Veterinary Medicine, Kyungpook National University

S 58 P-02-003 Chronic obstructive sleep apnea induces miRNA expression profiles associated with Alzheimer's disease in male rat
Hyeyun Kim[1], Ju Yeon Pyo[2], Jiyeon Moon[3], Seungeun Lee[3], Minchae Kim[3], Yein Choi[3], Dong-Ick Shin[4], Byong-Gon Park[3]
[1]Department of Neurology Sleep Medicine Research Center, International St. Mary's Hospital, Catholic Kwandong University, Incheon, Republic of Korea, [2]Department of Pathology International St. Mary's Hospital, Catholic Kwandong University, Incheon, Republic of Korea, [3]Department of Physiology College of Medicine, Catholic Kwandong University, Gangneung, Republic of Korea, [4]Department of Neurology Chungbuk National University Hospital, Cheongju, Republic of Korea

S 58 P-02-004 Short-term administration of Poria cocos extracts enhances sleep quality in rodent models with sleep disturbance
Hyeyun Kim[1], Kyunyong Park[2], Seohyun Park[2], Jiyeon Moon[2], Seungeun Lee[2], Minchae Kim[2], Yein Choi[2], Byong-Gon Park[2]
[1]Department of Neurology The Convergence Institute of Healthcare and Medical Science, International St. Mary's Hospital, Catholic Kwandong University, Incheon, Republic of Korea, [2]Department of Physiology College of Medicine, Catholic Kwandong University, Gangneung, Republic of Korea

S 58 P-02-005 Green tea epigallocatechin-3-gallate (EGCG) improves hippocampal neurogenesis and memory performance impaired by X-irradiation in mice
Kyung-Joo Seong, Hyo-Seon Park, Yeon-Jin Jeong, Sam-Young Park, Song-Yeon Park, Ji-Yeon Jung, Won-Jae Kim
Dental Science Research Institute, Stem cell Secretome Research Center, Hard-tissue Biointerface Research Center, Department of Oral Physiology, School of Dentistry Chonnam National University

S 59 P-02-006 Reactive microglia and mitochondrial unfolded protein response following ventriculomegaly and behavior defects in kaolin-induced hydrocephalus
Jiebo Zhu[1,2,3], Min Joung Lee[1,2,3], Jonghun An[1,2,3], Woosuk Chung[1,5,6], Eungseok Oh[1,4], Jun Young Heo[1,2,3]
[1]Department of Medical Science Chungnam National University School of Medicine, Daejeon, South Korea, [2]Department of Biochemistry Chungnam National University School of Medicine, Daejeon, South Korea, [3]Infection Control Convergence Research Center Chungnam National University School of Medicine, Daejeon, South Korea, [4]Department of Neurology Chungnam National University Hospital, Daejeon, South Korea, [5]Department of Anesthesiology and Pain Medicine Chungnam National University School of Medicine, Daejeon, South Korea, [6]Department of Anesthesiology and Pain Medicine Chungnam National University Hospital, Daejeon, South Korea

S 59 P-02-007 Transcutaneous Auricular Vagus Nerve Stimulation Enhances Cerebrospinal Fluid Circulation and Restores Cognitive Function in the Rodent Model of Vascular Cognitive Impairment
Seunghwan Choi[1], Dong Cheol Jang[2], Geehoon Chung[2], Sun Kwang Kim[1,2]
[1]Department of East-West Medicine Graduate School, Kyung Hee University, [2]Department of Physiology College of Korean Medicine, Kyung Hee University

S 59 P-02-008 Bicarbonate permeability of synaptic GABAAR mediates neuronal excitation
Dong Hoon Shin[1], Ki jung Kim[2], Jea Kwon[2], Jaekwang Lee[3], Ikhyun Jun[1], C. Justin Lee[2], Min Goo Lee[1]
[1]Department of Pharmacology Yonsei University College of Medicine, [2]Center for Cognition and Sociality Institute for Basic Science (IBS), [3]WCI Center for Functional Connectomics and Center for Neuroscience Institute of Science and Technology (KIST)

S 60 P-02-009 Transcriptional alterations of TRPC1/C5 channel in Huntingtin knock-in striatal cells accelerate Ca^{2+}-dependent cytotoxicity by Diamide-induced oxidative stress
Hana Lee[1], Insuk So[2], Chansik Hong[1]
[1]Department of Physiology Chosun University College of Medicine, Gwangju, South Korea, [2]Department of Physiology Seoul National University College of Medicine, Seoul, South Korea

S 60 P-02-010

Hyperactive ERK signaling in astrocytes impairs hippocampal learning and memory
Minkyung Kang[1,2], Jeongho Han[3], Jihye Choi[4], Hyun-Hee Ryu[1], Sunyong Kim[1,2], Kyoung-Doo Hwang[1,2], Jaegeon Lee[1,2], Pojeong Park[5], Ja Eun Choi[5], DaeHee Han[5], Sang Jeong Kim[1,2,7], Bong-Kiun Kaang[5], Benjamin G. Neel[6], Chul Hoon Kim[4], Hyungju Park[3], Yong-Seok Lee[1,2,7]
[1]Department of Physiology Seoul National University College of Medicine, Seoul, Korea, [2]Department of Biomedical Sciences Seoul National University College of Medicine, [3]Department of Structure & Function of Neural Network Korea Brain Research Institute (KBRI), Daegu, Korea, [4]Department of Pharmacology Yonsei University College of Medicine, Seoul, Korea, [5]School of Biological Sciences Seoul National University, Seoul, South Korea, [6]Laura and Isaac Perlmutter Cancer Center New York University Langone Medical Center, New York, USA, [7]Neuroscience Research Institute Seoul National University College of Medicine, Seoul, Korea

S 60 P-02-011

ASD-like phenotypes in a mouse model of Noonan syndrome
Soobin Kim[1,2], Sohyeon Park[3], Gaeun Park[1,2], Minkyung Kang[1,2], Jae Jin Shin[1,2], Sang Jeong Kim[1,2], Moo Kyun Park[4], Yong-Seok Lee[1,2]
[1]Department of Physiology Seoul National University College of Medicine, Seoul, Korea, [2]Department of Biomedical Sciences Seoul National University College of Medicine, Seoul, Korea, [3]Interdisciplinary Program in Neuroscience Seoul National University College of Natural Sciences, Seoul, Korea, [4]Department of Otorhinolaryngology-Head and Neck Surgery Seoul National University College of Medicine, Seoul, Korea

S 61 P-02-012

ASD-like phenotypes in a mouse model of Noonan syndrome
Soobin Kim[1,2], Sohyeon Park[3], Gaeun Park[1,2], Minkyung Kang[1,2], Jae Jin Shin[1,2], Sang Jeong Kim[1,2], Moo Kyun Park[4], Yong-Seok Lee[1,2]
[1]Department of Physiology Seoul National University College of Medicine, Seoul, Korea, [2]Department of Biomedical Sciences Seoul National University College of Medicine, Seoul, Korea, [3]Interdisciplinary Program in Neuroscience Seoul National University College of Natural Sciences, Seoul, Korea, [4]Department of Otorhinolaryngology-Head and Neck Surgery Seoul National University College of Medicine, Seoul, Korea

S 61 P-02-013

Neuroinflammation and microglial NOD2/RIPK2 signaling in Parkinson's disease
Bo Am Seo[1,2], Seung-Hwan Kwon[1], Han Seok Ko[1]
[1]Department of Neurology The Johns Hopkins University School of Medicine, Baltimore, Maryland, United States of America, [2]Department of Convergence Medicine Yonsei University Wonju College of Medicine, Wonju, Republic of Korea

S 61 P-02-014

Downregulation of TREK channels alleviates cognitive impairment in a mouse model of $A\beta_{1-42}$-induced Alzheimer's disease
Marie Merci Nyiramana[1,2], Eun-Jin Kim[1], Min Seok Woo[1], Dang Long Cao[1,2], Dong Kun Lee[1,2], Seong-Geun Hong[1], Jaehee Han[1], Dawon Kang[1,2]
[1]Department of Physiology and Institute of Health Sciences, College of Medicine Gyeongsang National University, [2]Department of Convergence Medical Science Gyeongsang National University

S 62 P-02-015

Peripheral Substance P induces hippocampal memory deficits
Sun Yong Kim[1,2], Kyeong-No Yoon[2,5], Dong Hun Lee[2,4,5], Yong Seok Lee[1,2,4]
[1]Department of Physiology Seoul National University College of Medicine, Seoul, Korea, [2]Department of Biomedical Sciences Seoul National University College of Medicine, Seoul, Korea, [3]Department of Biomedical Sciences, Neuroscience Research Institute Seoul National University College of Medicine, Seoul, Korea, [4]Medical Research Center, Institute of Human-Environment Interface Biology Seoul National University, Seoul, Korea, [5]Department of Dermatology Seoul National University College of Medicine, Seoul, Korea

P03: Electrophysiology and Ca2+ signaling

S 62 P-03-001

Blockade of voltage-dependent K^+ channels by olanzapine, atypical antipsychotic, in rabbit coronary arterial smooth muscle cells
Minji Kang, Ryeon Heo, Seo-Yeong Mun, Wenwen Zhuang, Won Sun Park
Department of Physiology Kangwon National University School of Medicine

S 62 P-03-002

Inhibitory effects of the atypical antipsychotic, clozapine, on voltage-dependent K^+ channels in rabbit coronary arterial smooth muscle cells
Minji Kang, Seo-Yeong Mun, Ryeon Heo, Wenwen Zhuang, Won Sun Park
Department of Physiology Kangwon National University School of Medicine

S 62 P-03-003

Plakophilin-2 deficiency augments Cx43 hemichannel-mediated ATP release and subsequent autocrine non-selective cation currents in HL-1 atrial myocytes under shear stress
Phuong Kim Luong, Anh TV Vu, Qui A. Le, Sun-Hee Woo
Department of Physiology Chungnam National University College of Pharmacy

S 63 P-03-004

Asenapine, an atypical antipsychotic, blocks voltage-gated potassium channels in rabbit coronary artery smooth muscle cells
Seo-Yeong Mun, Ryeon Heo, Minji Kang, Wenwen Zhuang, Won Sun Park
Department of Physiology Kangwon National University School of Medicine

S 63 P-03-005

Calcium homeostasis modulator 2 (calhm2) is responsible for the slowly activating outwardly rectifying current in mouse B cells
Si Won Choi[1], Kyoung Sun Park[2], Sung Joon Kim[1]
[1]Department of Physiology Seoul National University College of Medicine, Seoul, Republic of Korea, [2]Wide River Institute of Immunology Seoul National University College of Medicine, Hongcheon, Republic of Korea

S 63 P-03-006

Blockade of voltage-dependent K^+ channels by the class Ic antiarrhythmic agent lorcainide in coronary arterial smooth muscle cells
Wenwen Zhuang, Minji Kang, Seo-Yeong Mun, Ryeon Heo, Won Sun Park
Department of Physiology Kangwon National University School of Medicine

S 63 P-03-007

Mass spectrometry-based identification of phosphorylation sites in Cav3.1 calcium channel and characterization of their roles by site-directed mutagenesis

Sua Jeong[1], Ji Seon Shim[2], Seok Kyo Sin[2], Kang-Sik Park[2], Jung-Ha Lee[1]
[1]Department of Life Science Sogang University, [2]Department of Physiology College of Medicine, Kyung-Hee University

S 64 P-03-008

Bi-directional sensitivity of CALHM1 channel to protons from both sides of plasma membrane

Jae Won Kwon[1,2], Young Keul Jeon[1,2], Sung Joon Kim[1,2,3]
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S 64 P-03-009

Fucoxanthin suppresses NMDA and AMPA receptor-mediated excitation on substantia gelatinosa neurons of the trigeminal subnucleus caudalis in immature mice

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S 64 P-03-010

G protein beta2 subunit regulates the activity and current kinetics of Cav3.3 T-type channel via its association with the Cav3.3 C-terminus

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S 65 P-03-011

Identification of a novel tricyclic antidepressant binding site within opioid receptor using molecular dynamics and functional assays for TRPC4

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S 65 P-03-012

Shear stress increases junctional Ca^{2+} sparks in atrial myocytes via NADPH oxidase 4-dependent mitochondrial ROS generation

Long Nguyen Hoang Do[1], Tran N. Trinh[1], Yun Soo Bae[2], Phuong Kim Luong[1], Sun-Hee Woo[1]
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S 65 P-03-013

Inhibitory effect of benztropine, a muscarinic acetylcholine receptor inhibitor, on voltage-dependent K^+ channels in coronary arterial smooth muscle cells

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S 66 P-03-014

Hydrogen peroxide affects the post-synaptic $GABA_A$ receptor-mediated neurotransmission on gonadotropin-releasing hormone neurons

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S 66 P-03-015

Trp434 and Trp435 residues are crucial for sensing Calcium ions and PI(4,5)P2 molecules in TRPC5 channel

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S 66 P-03-016

Electrophysiological properties of TRPC1/4 heteromer determined by its pore residues

Christine Haewon Park, Insuk So
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S 67 P-03-017

Activation of TRPV3 is required for keratinocyte differentiation and epidermal barrier formation

Elina Da Sol Chung[1], Hyun Jong Kim[2], Yu Ran Nam[2], Joo Hyun Nam[2], Sung Joon Kim[1]
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S 67 P-03-018

Molecular basis for PI(4,5)P2 modulation of proton-activated chloride (PAC) channels

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S 67 P-03-019

Developmental up-regulation of voltage-gated Na^+ channel and its electrophysiological function in rat hippocampal neurons

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S 67 P-03-020

Phosphate-mediated calcium regulation in podocyte integrity

Bao T.N. Dang[1,2,3,4,5], Ji-Hee Kim[1,2,3,4,5], Phan Anh Nguyen[1,2,3,4,5], Kyu-Sang Park[1,2,3,4,5], Seung-Kuy Cha[1,2,3,4,5]
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S 68 P-03-021 Protective effect of tomatidine against cardiac hypertrophy induced by isoproterenol in cellular system and electrophysiology
Seung Hak Choi[1], Ye Seul Kim[2], Jae Ho Kim[2], Jae Boum Youm[1]
[1]Department of Physiology college of medicine, Inje university, [2]Department of Medical Science School of Medicine Pusan national university

S 68 P-03-022 αKlotho ameliorates podocyte injury and proteinuria in diabetic nephropathy via stabilizing podocyte Ca^{2+} channels
Ji-Hee Kim[1,4], Bao T.N. Dang[1,2,4], Kyu-Hee Hwang[1,4], Nghia Thi Pham[1,2,4], Minseob Eom[3], Kyu-Sang Park[1,2,4], Seung-Kuy Cha[1,2,4]
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S 68 P-03-023 NR2A-containing NMDARs detect increased ambient glutamate concentration in supraoptic nucleus of DOCA-salt hypertensive model rats
Ramesh Sharma[1,2,3], Hyun Jin Shin[1,2], Chiranjivi Neupane[1,2,3], Thuy Linh Pham[1,2], Hyun-Woo Kim[1,2], Jin Bong Park[3]
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S 69 P-03-024 Roles of Zn^{2+} in Mg^{2+}-free-induced epileptiform activity in the CA3 region of rat hippocampal slices
Ji Seon Yang[1], Hyun-Jong Jang[1], Duck-Joo Rhie[1], Ki-Wug Sung[2], Shin Hee Yoon[1]
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S 69 P-03-025 Simultaneous identification of dose-response curves for ion channels using deep-learning
Jaekyung Song[1,2], Chae Hun Leem[1,2], Yu Jin Kim[1]
[1]Department of Physiology Asan Medical Center, Seoul, Republic of Korea, [2]Department of Physiology University of Ulsan College of Medicine, Seoul, Republic of Korea

S 69 P-03-026 The role of STING-IRF3 signaling in GATs expression and its implications in cognitive functions
Chiranjivi Neupane[1], Ramesh Sharma[1,2,3], Thuy Linh Pham[2,3], Jin Bong Park[1], Sanghoon Lee[1]
[1]Laboratory of Veterinary Pharmacology College of Veterinary Medicine and Research Institute for Veterinary Science, Seoul National University, Seoul, Korea, [2]Department of Medical Science School of Medicine, Chungnam National University, Daejeon, Korea, [3]Department of Physiology School of Medicine, Chungnam National University, Daejeon, Korea

S 69 P-03-027 CFTR Channel Regulation of Bicarbonate Permeability by WNK1
Min Jae Kim, Yon Jung Kim, Min Goo Lee
Dept. of Pharmacology Yonsei University, College of Medicine, Seoul, Republic of Korea

S 70 P-03-028 In vitro electrophysiological assessment for proarrhythmia risk prediction under CiPA initiative
Jin Ryeol An, In Kyo Jung, Kwan Soo Kim, Chan Hyeok Kwon, Sun Ok Choi
Pharmacological Research Division, Toxicological Evaluation and Research Department National Institute of Food and Drug Safety Evaluation, Ministry of Food and Drug Safety

S 70 P-03-029 Inhibition of voltage-dependent K^+ channels by antimuscarinic drug fesoterodine in coronary arterial smooth muscle cells
Seo-Yeong Mun, Minji Kang, Ryeon Heo, Wenwen Zhuang, Won Sun Park
Department of Physiology Kangwon National University School of Medicine

S 70 P-03-030 Blockade of Kv3.1 by MK801, a PCP-derivative NMDA receptor inhibitor
Tae Jun Park[1], Sang Woong Park[2], Young Min Bae[1], Mi Seon Seo[1]
[1]Department of Physiology Konkuk University School of Medicine, [2]Department of Emergency Medical Services Eulji University

P04: Muscle physiology

S 71 P-04-001 Vasodilation by trelagliptin, a DPP-4 anti-diabetic drug, via activation of Kv channels and SERCA pumps in rabbit aorta
Ryeon Heo, Minji Kang, Wenwen Zhuang, Seo-Yeong Mun, Won Sun Park
Department of Physiology Kangwon National University School of Medicine

S 71 P-04-002 Vasodilatory effect of antidiabetic omarigliptin by activating Kv Channels and SERCA pump in rabbit aorta
Ryeon Heo, Minji Kang, Seo-Yeong Mun, Wenwen Zhuang, Won Sun Park
Department of Physiology Kangwon National University School of Medicine

S 71 P-04-003 A novel regulator of skeletal muscle functions
Jun Hee Choi[1,2], Seung Yeon Jeong[1,2], Jooho Kim[1,2], Jin Seok Woo[3], Eun Hui Lee[1,2]
[1]Department of Physiology College of Medicine, The Catholic University of Korea, [2]Department of Biomedicine & Health Sciences Graduate School, The Catholic University of Korea, [3]Department of Physiology David Geffen School of Medicine, UCLA

S 71 P-04-004 Calsequestrin 1 Is an Active Partner of Stromal Interaction Molecule 2 in Skeletal Muscle
Seung Yeon Jeong[1,2], Mi Ri Oh[1,2], Jun Hee Choi[1,2], Jin Seok Woo[3], Eun Hui Lee[1,2]
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S 72 P-04-005 Ultra-weak light emission improves mitochondrial respiration in heart and skeletal muscle of mice
Dae Yun Seo[1], Jun Hyun Bae[2], Mi Jung Park[3], Hae Lim Jang[3], Jeong Su Yang[3], Hyo Bum Kwak[2], Jin Han[1]
[1]Department of Physiology, College of Medicine Cardiovascular and Metabolic Disease Center, Smart Marine Therapeutic Center, Inje University, Busan, Korea, [2]Department of Biomedical Science and Engineering Inha University, Incheon, Republic of Korea, [3]Biolight Corporation Gyeonggi-do, Republic of Korea

S 72 P-04-006 Fetuin-B ameliorates dexamethasone-induced atrophy in C2C12 mouse skeletal muscle cells
Hengzhe Jin[1], Seung Hyo Jung[1], Hwan Myung Lee[2], Bokyung Kim Kim[1], Kyung Jong Won[1]
[1]Department of Physiology and Medical Science, School of Medicine Konkuk University, [2]Division of Cosmetic and Biotechnology, College of Life and Health Sciences Hoseo University

P05: Organ physiology

S 72 P-05-001 miR204 potentially promotes non-alcoholic fatty liver disease by inhibition of cpt1a in mouse hepatocytes
Seonhee Kim, Shuyu Piao, Minsoo Kim, GiangHuong Vu, Byeong Hwa Jeon, Cuk-Seong Kim
Department of Medical Science & Physiology, School of Medicine, Chungnam National University

S 72 P-05-002 DPP-4 inhibitor prevents cardiomyopathy via improvement of mitochondrial function and reduction of cardiac fibrosis in type 2 diabetic mice
Hyoung Kyu Kim[1,2], Pham Trong Kha[1,2], Gwang Sil Kim[1], Jong Chul Won[1]
[1]Cardiovascular and Metabolic Disease Center Inje University, [2]Smart Marine Therapeutic Center Inje University

S 73 P-05-003 Long-term exposure of ethylenethiourea induces nephrotoxicity in male C57BL/6 mice
Hyeyun Kim[1], Jiyeon Moon[2], Seungeun Lee[2], Minchae Kim[2], Yein Choi[2], Byong-Gon Park[2]
[1]Department of Neurology The Convergence Institute of Healthcare and Medical Science, International St. Mary's Hospital, Catholic Kwandong University, Incheon, Republic of Korea, [2]Department of Physiology College of Medicine, Catholic Kwandong University, Gangneung, Republic of Korea

S 73 P-05-004 Functional analysis of novel SCN5A mutations related to Brugada syndrome
Hyun Namgoong[1], Na Kyeong Park[1], Sung Joon Kim[1], Seong Woo Choi[2]
[1]Department of Physiology, Department of Biomedical Sciences Seoul National University College of Medicine, Korea, [2]Department of Physiology College of Medicine, Dongguk University, Korea

S 73 P-05-005 Role of Rho-associated kinase (ROCK) in the different speed of relaxation between pulmonary arteries and mesentery arteries of rats
Seung Beom Oh[1], Suhan cho[1], Sung Joon Kim[1,2]
[1]Department of Biomedical Sciences Seoul National University College of Medicine, [2]Ischemic/Hypoxic Disease Institute Seoul National University College of Medicine

S 74 P-05-006 BH4 activates CaMKK2 and rescues the cardiomyopathic phenotype in rodent models of diabetes
Van Nam Bui
Physiology Department Inje University, College of medicine

S 74 P-05-007 Integrin αvβ3 alteration by fluid shear stress in podocyte
Nghia T. Pham[1,2,4], Ji-Hee Kim[1,2,4], Bao T.N. Dang[1,2,4], Minseob Eom[3], Jae Seok Kim[5], Seung-Kuy Cha[1,2,4]
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S 74 P-05-008 Echinochrome A reverses kidney abnormality and reduces blood pressure in a rat model of preeclampsia
Cui Huixing[1], Liu Junxian[2], Zhang Yinhua[3]
[1]Seoul National University College of Medicine Seoul National University College of Medicine, [2]Seoul National University College of Medicine Seoul National University College of Medicine, [3]Seoul National University College of Medicine Seoul National University College of Medicine

S 74 P-05-009 TRPC6 deficiency causes adipocyte dysfunction and obese-like phenotypes
Phan Anh Nguyen[1,2,3,4,5], Kyu-Hee Hwang[1,2,3,4,5], Duyen Tran Thi Thuy[1,2,3,4,5], Tung Hoang Kim[1,2,3,4,5], Kyu-Sang Park[1,2,3,4,5], Seung-Kuy Cha[1,2,3,4,5]
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S 75 P-05-010 TRPC6 deficiency causes hepatosteatosis through deregulation of adipocyte lipid handling
Phan Anh Nguyen[1,2,3,4,5], Duyen Tran Thi Thuy[1,2,3,4,5], Kyu-Hee Hwang[1,2,3,4,5], Tung Hoang Kim[1,2,3,4,5], Kyu-Sang Park[1,2,3,4,5], Seung-Kuy Cha[1,2,3,4,5]
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S 75 P-05-011 Maintaining integrity of hair follicles by 3-dimensional co-culture of hair follicles and dermal fibroblast spheroids in collagen hydrogels
Ji Woo Im, Hae-Rahn Bae
Department of Physiology College of Medicine, Dong-A University, Busan, Korea

S 75 P-05-012

ROS-mediated feedforward upregulation of TRPC6 initiates hepatic stellate cell activation and fibrosis
Kyu-Hee Hwang[1,2,4,5,6], Ji-Hee Kim[2,4,5,6], Phan Anh Nguyen[2,3,4,5,6], Soo-Jin Kim[2,3,4,5,6], Kyu-Sang Park[2,3,4,5,6], Seung-Kuy Cha[2,3,4,5,6]
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S 76 P-05-013

Hidden re-initiation of transcription in a KCNH2 frameshift mutation (c.453delC) produces impaired hERG K^+ channels and the heterozygote patient-derived iPSC-CMs show LQT phenotype
Na Kyeong Park[1], Sung Joon Kim[1], Seong Woo Choi[2]
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S 76 P-05-014

Capsanthin Prevents Atherosclerosis and Vascular Inflammation in $ApoE^{-/-}$ mice
Sungmin Kim[1,2,3], Yu-Ran Lee[1,2], Eun-Ok Lee[1,2], Hao Jin[1,2,3], Yeon-Hee Choi[1,2], Hee-Kyoung Joo[1,2,3], Byeong-Hwa Jeon[1,2,3]
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P06: Endocrine and Energy Metabolism

S 76 P-06-001

Mitochondrial modulation protects blood-brain barrier integrity by increasing junctional protein expression in cerebrovascular cell
Min Joung Lee[1], Jiebo Zhu[1,2,3], Jong Hun An[1,2,3], Jun Young Heo[1,2,3]
[1]Department of Biochemistry Chungnam National University School of Medicine, [2]Department of Medical Science Chungnam National University School of Medicine, [3]Infection Control Convergence Research Center Chungnam National University School of Medicine

S 77 P-06-002

Reduced branched-chain aminotransferase activity alleviates metabolic vulnerability caused by dim light exposure at night in Drosophila
Gwang-Ic Son[1], Mari Kim[2], Yun-Ho Cho[1], Gye-Hyeong Kim[2], Eunil Lee[1], Joong-Jean Park[1]
[1]Physiology Korea University College of Medicine, [2]Preventive Medicine Korea University College of Medicine

S 77 P-06-003

Empagliflozin prevents diabetic cardiomyopathy by attenuating cardiac lipotoxicity in type 2 diabetic db/db mice
Trong Kha Pham, Hoai To Thi Nguyen, Sun Woo Kim, Hyoung Kyu Kim, Jin Han
Department of Physiology, Inje University

S 77 P-06-004

Activation of Sarco/Endoplasmic Reticulum Ca^{2+} ATPase Increases Mitochondrial Biogenesis and Protects Pancreatic β-cells from Lipotoxicity
Ha Thu Nguyen[1,2], Carlos Noriega Polo[1,2], Andreas Wiederkeh[3], Claes B. Wollheim[4,5], Kyu-Sang Park[1,2]
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S 77 P-06-005

Peri-lysosomal Calcium Overload by Palmitate in Pancreatic β-cells
Ha Thu Nguyen[1,2], Kyu-Sang Park[1,2]
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S 78 P-06-006

Loss of SCAP induces obesity through shifting macrophage polarization in adipose tissue
Soo-Young Park, Jae-Ho Lee, Eun-Ho Lee, Hee-Kyung Han, Min-Hee Seo, Seung-Soon Im
Department of Physiology Keimyung University School of Medicine, Daegu, Korea

P07: Epithelium and Exocrine Physiology

S 78 P-07-001

Humanin and formylated Humanin promote skin wound healing through the STAT3 signaling pathway
Airr Yeuanmany[1,3,4,5], Kyu-Hee Hwang[2,3,4], Kyu-Sang Park[1,3,4], Seung-Kuy Cha[1,3,4]
[1]Department of Physiology Yonsei University Wonju College of Medicine, Wonju-Si, Gangwon-do, Republic of Korea, [2]Department of Convergence Medicine Yonsei University Wonju College of Medicine, Wonju-Si, Gangwon-do, Republic of Korea, [3]Department of Global Medical Science Yonsei University Wonju College of Medicine, Wonju-Si, Gangwon-do, Republic of Korea, [4]Mitohormesis Research Center Yonsei University Wonju College of Medicine, Wonju-Si, Gangwon-do, Republic of Korea, [5]Department of Basic Health Science, Faculty of Medicine Faculty of Medicine, University of Health Science, Vientaine, Laos

S 78 P-07-002

The Role of JAK3 in Skin Wound Healing
Won-Tae Jo, A-Young Kim, Eun-Joo Baik
Department of Physiology Ajou University School of Medicine, Suwon, Korea

S 79 P-07-003

Hyperbaric oxygen therapy promotes diabetic wound healing via AKT and ERK signaling pathway
Kyu-Hee Hwang[1,3], Subo Lee[1,2,3], Taeui Hong[1,3], Kyu-Sang Park[1,2,3], Seung-Kuy Cha[1,2,3]
[1]Department of Physiology Yonsei University Wonju College of Medicine, [2]Global Medical Science Yonsei University Wonju College of Medicine, [3]Mitohormesis Research Center Yonsei University Wonju College of Medicine

S 79 P-07-004 Bitter taste, a possible new function.
Kyung-Nyun Kim[1,2], In-Sun Choi[1], Ki-Myung Chung[1,2], Young-Kyung Cho[1,2]
[1]Department of Physiology & Neuroscience College of Dentistry, Gangneung-Wonju National University, [2]Research Institute of Oral Sciences Gangneung-Wonju National University

P08: Inflammation and Immune Physiology

S 79 P-08-001 Effects of trehalose, an autophagy enhancer on implant surface and inflamed and infected bone.
Song-Yeon Park[1], Min-Young Park[1], Sam-Young Park[1], Kyung-Joo Seong[1], Yeon-Jin Jeong[1], Ji-Hye Jeon[1], Hyo-Seon Park[1], Suk-Gyun Park[2], Ji-Yeon Jung[1], Won-Jae Kim[1]
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S 79 P-08-002 In vivo administration of Gas6 inhibits epithelial-mesenchymal transition and enhances PGE2 and PGD2 in alveolar type II epithelial cells following bleomycin treatment
Ye-Ji Lee, Jihee Lee
Physiology Inflammation-Cancer Microenvironment Research Center, College of Medicine, Ewha Womans University, Seoul, Korea

S 80 P-08-003 CRIF1 upregulated homocysteine production by suppressing DHFR expression in vascular endothelial cells
Minsoo Kim[1], Shuyu Piao[1], Seonhee Kim[1], GiangHuong Vu[1], Ikjun Lee[1,2], Cuk-seong Kim[1,2]
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S 80 P-08-004 Optimization and characterization of exosomes from mouse brain: evaluation of it for pathogenic role in delayed onset brain injury
Jong Hun An[1,2,3], Jiebo Zhu[1,2,3], Min Joung Lee[1,2,3], Jun Young Heo[1,2,3]
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S 80 P-08-005 Ulinastastin Attenuates Vascular Damage in IDH2-Deficient Endothelial Cells via TGF-β/MMP7/SDS2 signaling pathway
Gianghuong Vu[1,2], Sujeong Choi[1], Shuyu Piao[1], Seonhee Kim[1], Minsoo Kim[1,2], Byeonghwa Jeon[1,2], Cukseong Kim[1,2]
[1]Department of physiology Department of Physiology & Medical Science, College of Medicine, Chungnam National University, [2]Department of physiology Brain Korea 21 FOUR project for medical science, Chungnam national University

S 80 P-08-006 Protective effect of myricetin in RINm5F β-cells under exposure to interleukin-1β
Seo-Yoon Chang, Yongjun Ko, Myung-Jun Kim
Department of Physiology College of Medicine, The Catholic University of Korea, Seoul, Republic of Korea

S 81 P-08-007 Alleviation of inflammatory parameters by fermented and aged mountain cultivated ginseng sprouts and its main component, compound K, in acute lung injury and asthma models
Dang Long Cao[1,2], Min-Seok Woo[1], Eun-Jin Kim[1], Ji Hyeon Ryu[3], Kye Man Cho[4], Sang Soo Kang[2,5], Dawon Kang[1,2]
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S 81 P-08-008 Elevated Plasma Apurinic/Apyrimidinic Endonuclease 1/Redox Effector Factor-1 Levels in Refractory Kawasaki Disease
HAO JIN[1,2,3], Yu Ran Lee[3], Hee Kyoung Joo[3], Eun Ok Lee[3], Sungmin Kim[1,2,3], Yeon Hee Choi[1,2,3], Byeong Hwa Jeon[1,2,3]
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P09: Cellular Physiology and Cancer

S 81 P-09-001 In vivo injection of apoptotic cancer cells inhibits CAF activation and lung metastasis via Notch1-WISP-1 signaling
Kyungwon Yang, Jihee Lee
Department of Physiology, Inflammation-Cancer Microenvironment Research Center College of Medicine, Ewha Womans University

S 82 P-09-002 SUV39H1-driven NFATc1 methylation is essential for the c-Cbl-mediated degradation of NFATc1 in an osteoclast lineage
Do-Won Jeong[1,2], Hye-Jin Kim[1,2], Jong-Wan Park[2], Seulbee Lee[1,2], Hyeryeon Jung[3], Eugene C. Yi[3], Nacksung Kim[4], Yang-Sook Chun[1,2]
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S 82 P-09-003 Tumor-Treating Fields (TTFields), an Anti-Cancer Therapeutic Modality, Induces Cell Death in Liver Cancer Cell
Seung Hoon Lee[1], Chul Huh[2], Soo Jun Park[2], Hyung Ju Park[2], Min-Kyung Yeo[1], Hyon-Seung Yi[1], Seok-Hwan Kim[1], Sun-Hyeong Kang[1], Sang-Il Lee[1], Hyun-Jin Jo[1], Jong-Il Park[1]
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S 82 P-09-004

Apoptotic cancer cells stimulate WISP-1 secretion from cancer-associated fibroblasts (CAFs) to inhibit migration and invasion of lung cancer cells and CAFs

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S 82 P-09-005

Enhancement of the Notch ligand Dll1 expression in UV-irradiated apoptotic cancer cells activates Notch1 signaling in cancer-associated fibroblasts (CAFs)

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S 83 P-09-006

Neddylation blockade accelerates cancer cell migration under the condition of insulin resistance

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S 83 P-09-007

Atractylodes macrocephala Koidz induces apoptosis in human gastric cancer cells through Activation of the ROS and MAPK Signaling Pathway

Na Ri Choi, Woo-gyun Choi, Byung Joo Kim
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S 83 P-09-008

High diagnostic and therapeutic performance of exosomal miR-34 family for brain metastasis in lung cancer

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S 83 P-09-009

Effects of N-acetyl cysteine and buthionine sulfoximine in propyl gallate-treated lung cancer cells: cell death, reactive oxygen species, and glutathione

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S 84 P-09-010

Tempol inhibits the growth of lung cancer and normal cells through apoptosis accompanied by increased $O2^{\cdot-}$ levels and glutathione depletion

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S 84 P-09-011

Identification of the role of SIRT6 as a tumor suppressor in liver cancer

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Department of Physiology, Institute for Medical Sciences Jeonbuk National University Medical School, Jeonju, Republic of Korea

S 84 P-09-012

A role of Hematopoietic- and neurologic-expressed sequence 1 in ER-stress and autophagy in Heptocellular carcinoma cells

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S 85 P-09-013

Enhanced efficacy of 5-fluorouracil combined with histone deacetylase inhibitor panobinostat against Gastric Cancer

Yanling Wu[1], Soo Mi Kim[2]
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S 85 P-09-014

Effects of rapamycin and hydroxychloroquine in auranofin-treated lung cancer cells: cell death, reactive oxygen species, and glutathione

Xia Ying Cui, Woo Hyun Park
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S 85 P-09-015

The molecular mechanism of TMEM16E-mediated plasma membrane repair (PMR) system

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S 85 P-09-016

Neddylation blockade modulates the positive effect of FIH on breast cancer cells.

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S 86 P-09-017

Cancer cells promote lipolysis of adipocyte derived stem cells to obtain free fatty acids for migration by using a cytokine

Jeong-Eun Yun[1,3], Jieun Seo[4,5], Yang-Sook Chun[1,2,3]
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S 86 P-09-018 CRIF1 siRNA-encapsulated PLGA nanoparticles suppress tumor growth in MCF-7 human breast cancer cells
Shuyu Piao, Seonhee Kim, GiangHuong Vu, Minsu Kim, Miae Lee, Byeong Hwa Jeon, Cuk-Seong Kim
Department of Physiology & Medical Science, College of Medicine Chungnam National University

S 86 P-09-019 Targeted Therapy and anti-PD-1 treatment synergistically promote antitumor immunity in Hepatocellular Carcinoma
Yanling Wu, Soomi Kim
Department of Physiology Institute for Medical Sciences, Jeonbuk National University Medical School

S 86 P-09-020 Majonoside-R2 active in Vietnamese ginseng has the effect of protecting H9C2 cells against hypoxia/reoxygenation injury via modulating mitochondrial function and biogenesis
Thien Nguyen Huu[1], Thu Vu Thi[2], Yen Ngo Thi Hai[2], Tung Nguyen Huu[2], Hyoung Kyu Kim[1], Jin Han[1]
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S 87 P-09-021 Anticancer effect of verteporfin on non-small cell lung cancer via downregulation of ANO1
Raju Das[1], Yohan Seo[2], JooHan Woo[1,3]
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S 87 P-09-022 Redox function of secreted APE1/Ref-1 downregulates ROS generation and apoptosis in doxorubicin-induced cardiotoxicity
Soo Yeon An[1,2,3], Hee Jeong Seo[3], Yooran Lee[3], Seongmin Kim[1], Byeong Hwa Jeon[1,3], Sun-Ah Jin[2], Jin-Ok Jeong[2]
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S 87 P-09-023 Suppression of TGF-β/Integrin Signaling by Klotho Prevents Transdifferentiation of Hepatic Stellate Cells and Liver Fibrosis
Soo-Jin Kim[1,3], Yangsik Jeong[2,3], Seung-Kuy Cha[1,3], Kyu-Sang Park[1,3]
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S 88 P-09-024 Down-regulation of TASK-3 induces senescence of granulosa cells in the bovine follicular cystic ovary
Chang-Woon Kim[1], Eun-Jin Kim[2], Min Seok Woo[2], Dang Long Cao[2,3], Ji Hyeon Ryu[4], IL-Keun Kong[5], Dong Kun Lee[2,3], Seong-Geun Hong[2], Jaehee Han[2], Dawon Kang[2,3]
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S 88 P-09-025 Isolation and analysis of circulating exosomes in a mouse model of metastatic lung cancer
Kang Minji[1], Lim Jiwoo[1], Ahn Young-Ho[2], Cho Min-Sun[3], Lee Kang Jihee[1], Choi Youn-Hee[1]
[1]Physiology Inflammation-Cancer Microenvironment Research Center, College of Medicine, Ewha Womans University, Seoul, South Korea, [2]Molecular Medicine Inflammation-Cancer Microenvironment Research Center, College of Medicine, Ewha Womans University, Seoul, South Korea, [3]Pathology College of Medicine, Ewha Womans University, Seoul, South Korea

S 88 P-09-026 TREK-1 upregulation promotes the growth of colorectal cancer cells along with PDGFRα activation
Min Seok Woo[1], Young-Tae Ju[2], Eun-Jin Kim[1], Dawon Kang[1]
[1]Department of Physiology and Institute of Health Sciences College of Medicine, Gyeongsang National University, [2]Department of Surgery College of Medicine, Gyeongsang National University

S 88 P-09-027 Drug discovery for overcoming acquired resistance to ALK inhibitors in lung cancer based on a systems approach
Sang-Min Park[1], Haejeong Heo[2,3], Hyun Jung Lim[2,3], Seongwon Cha[4], Mirang Kim[2,3], Haeseung Lee[5]
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P10: Exercise and Integrative physiology

S 89 P-10-001 A comparison of metabolic profile and cardiorespiratory fitness of breast cancer survivors and matched healthy controls.
Jihee Min[1], Eunha Chang[2], Ji Yeong Choi[1], In Deok Kong[1]
[1]Department of Convergence Medicine Wonju College of Medicine, Yonsei University, Wonju, South Korea, [2]Division of Biological Science and Technology Yonsei University, Wonju, South Korea

S 89 P-10-002 Chronic food restriction produces locomotor sensitization to amphetamine
Hyung Shin Yoon[1], Seohyun Lee[2], Jeong-Hoon Kim[1,2]
[1]Physiology Yonsei University College of Medicine, [2]Medical Sciences Yonsei University College of Medicine

S 89 P-10-003 Effects of exercise training on mitochondria dysfunction associated with aging
Minsun Kim[1], Yerim Choi[2], Sieun Park[1], Moonsung Choi[3], Youn-Jung Kim[4], Seung Kyum Kim[1,2]
[1]Department of Sports Science Seoul National University of Science and Technology, [2]Convergence Institute of Biomedical Engineering and Biomaterials Seoul National University of Science and Technology, [3]Department of Optometry Seoul National University of Science and Technology, [4]Department of Basic Nursing Science Kyung Hee University, Seoul, Republic of Korea

S 90 P-10-004 Neddylation attains bone homeostasis by regulating osteoclastogenesis and osteoblastogenesis
Joseung Lee[1], Min Young Lee[1], Yang-Sook Chun[1,2,3]
[1]Department of Biomedical Sciences Seoul National University College of Medicine, Seoul, Korea, [2]Ischemic/Hypoxic Disease Institute Seoul National University College of Medicine, Seoul, Korea, [3]Department of Physiology Seoul National University College of Medicine, Seoul, Korea

S 90 P-10-005 Association between body mass index, domain-specific sedentary behavior, and asthma risk by using Korean Youth Health Risk Behavior Online Survey
Ki-Taek Oh, Jihee Min, In Deok Kong
Convergence Medicine Wonju College of Medicine, Yonsei University

S 90 P-10-006 Novel Function of Jumonji C(JmjC) domain – containing protein in osteoclastogenesis
Joo-Seung Lee[1], Hye-Jin Kim[1], Min Young Lee[1], Seon-Young Kim[1], Do-Won Jeong[1], Jong-Wan Park[1,2,3], Yang-Sook Chun[1,2,3]
[1]Department of Biomedical Sciences Seoul National University, [2]Ischemic/Hypoxic Disease Institute Seoul National University , [3]Department of Physiology Seoul National University College of Medicine, Seoul, Korea

S 90 P-10-007 Exercise training reduces a high-fat diet-induced CXCL12 expression in mouse
Elsayed Mohamed[1,2], Dong-Hwan Kim[3], Bong-Jo Kim[1], Hae-Rahn Bae[1]
[1]Department of Physiology College of Medicine, Dong-A University, Busan, Korea, [2]Department of Genetics Assiut University, Assiut, Egypt, [3]Human Life Research Center Dong-A University, Busan, Korea

P11: Physiomes and Systems Biology

S 91 P-11-001 Simulation of substrate-dependent changes of mitochondrial function using a computational mitochondria model
Ji Yeon Song[1], Seunghak Choi[2], Hyoung Kyu Kim[2], Jin Han[2], Chae Hun Leem[1], Jae Boum Youm[2]
[1]Department of Physiology University of Ulsan College of Medicine/Asan Medical Center, [2]Department of Physiology College of Medicine, Inje University

P12: Others: Drugs, Phytochemicals, Miscellaneous

S 91 P-12-001 Prediction of the Medicinal Mechanisms of Pinellia ternata Breitenbach, a Traditional Medicine for Gastrointestinal Motility Disorders, through Network Pharmacology
Na Ri Choi[1], Jongwon Park[2], Seok-Jae Ko[2,3], Jeong Nam Kim[1], Woogyun Choi[1], Jae-Woo Park[2,3], Byung Joo Kim[1]
[1]Division of Longevity and Biofunctional Medicine School of Korean Medicine, Pusan National University, [2]Department of Clinical Korean Medicine Graduate School of Kyung Hee University, [3]Department of Gastroenterology College of Korean Medicine, Kyung Hee University

S 91 P-12-002 Measuring Pattern Separation in Hippocampus by in Situ Hybridization
Kisang Eom
Dept of Physiology Keimyung univ. School of medicine

S 92 P-12-003 Targeted downregulation of Hipp1 ameliorates tau-induced deficits in Drosophila melanogaster
Sung Yeon Park[1,3], Jieun Seo[2], Seulbee Lee[2], Joohyung Kim[4], Sang Jeong Kim[1,2,3], Seungbok Lee[4], Yang-Sook Chun[1,2,3]
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S 92 P-12-004 A new approach of electrophysiologic efficacy evaluation method for APP/PS1 transgenic mice
YoungHwan Kim[1,2], Ji-Hyun Jeong[1], Ji Woong Ahn[1], Seungsoo Chung[1,2]
[1]BnH Research Co., Ltd. Research Institute, [2]Department of physiology Graduate School of Medical Science, Brain Korea 21 Project, Yonsei University College of Medicine

S 92 P-12-005 Novel marine compound Neopetroside A confers cardioprotection against ischemia/reperfusion injury by inhibiting GSK-3β
Jubert Marquez[1,2], Hyoung Kyu Kim[1,2], Min Kim[1,2], Nikolay Nifantiev[3], Jin Han[1,2]
[1]Cardiovascular and Metabolic Disease Center Inje University, Busan, Korea, Republic of Korea, [2]Department of Health Sciences and Technology Graduate School, Inje University, Busan, Korea, Republic of Korea, [3]GB Elyakov Pacific Institute of Bioorganic Chemistry Far Eastern Branch of the Russian Academy of Science, Vladivostok, Russia

S 93 P-12-006 Mitochondrial creatine kinase tyrosine residue phosphorylation attenuate cardiac hypoxia/reoxygenation injury
Nammi Park[1], Jubert Marquez[1,2], Maria Victoria Faith Garcia[1,2], Ippei Shimizu[3], Jeong Rim Ko[1], Hyoung Kyu Kim[1,2,4], Jin Han[1,2,4]
[1]Cardiovascular and Metabolic Disease Center, Smart Marine Therapeutics Center Inje University, Busan, Korea, [2]Department of Health Sciences and Technology Graduate School of Inje University, Busan, Korea, [3]Department of Cardiovascular Biology and Medicine Niigata University Graduate School of Medical and Dental Sciences, Niigata, Japan, [4]Department of Physiology, College of Medicine Inje University, Busan, Korea

S 93 P-12-007 Chrysosplenol C potently decreases mitochondrial reactive oxygen species independently of protein kinase C
Kim Phuong Luong, Sun-Hee Woo
Department of Physiology Chungnam National University, College of Pharmacy

S 93 P-12-008

Multi-modal effects of Echinochrome A on the activities of ion channels in skin tissue

Sung Eun Kim[1], Elina Da Sol Chung[2], Elena A Vasileva[3], Natalia P Mischchenko[4], Sergey A Fedoreyev[5], Valentin A Stonik[6], Hyoung Kyu Kim[7], Joo Hyun Nam[8], Sung Joon Kim[9]

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S 94 P-12-009

Binding mechanisms of Shikonin derivatives targeting SARS-CoV-2 main protease

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S 94 P-12-010

Vasorelaxant effect of Trachelospermi caulis extract on rat mesenteric resistance arteries

Chae eun Haam, Seonhee Byeon, Sooyeon Choi, Eun Yi Oh, Soo-Kyung Choi, Young-Ho Lee

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S 94 P-12-011

Vascular relaxation induced by vanillin in rat mesenteric resistance arteries

Sooyeon Choi, Chae Eun Haam, Eun Yi Oh, Seonhee Byeon, Soo-Kyoung Choi, Young-Ho Lee

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S 94 P-12-012

Inhibition of lactate dehydrogenase A upregulates mitochondrial proteins and fatty acid oxidation in mouse brown adipocytes

Aye Hsu Lae[1,2], Soo Kyung Lee[1,2], Dat Da Ly[1,2], Jaetaek Kim[3], Chanbae Park[4], Kyu-Sang Park[1,2]

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S 95 P-12-013

Finasteride ameliorates neointima hyperplasia in a rat carotid balloon injury model and suppresses primary cultured rat vascular smooth muscle cell proliferation and migration.

Jung Sook Kim, Akila Cooray, Kyu Pil Lee

Department of Physiology College of Veterinary Medicine, Chungnam National University, Daejeon, Korea

S 95 P-12-014

Peptides derived from voltage-dependent calcium channel beta subunit decrease arterial blood pressure in rats

Dong-ho Youn[1], Jiyeon Jun[1], Hyung Kyu Kim[1], Tae Wan Kim[2]

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S 95 P-12-015

Dietary habits interact with five genetic variants related to dyslipidemia in Korean middle-aged adults

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S 96 P-12-016

Role of lateral hypothalamus-lateral habenula pathway in cocaine-induced psychomotor responses

DanBi Ahn[1], Han Byeol Jang[1,2], Suchan Chang[2], Yeonhee Ryu[3], Hyung Kyu Kim[1,4], Bong Hyo Lee[2], Hee Young Kim[1]

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S 96 P-12-017

Activation of a hypothalamus-habenula circuit by mechanical stimulation inhibits cocaine addiction-like behaviors

Han Byeol Jang[1,2], DanBi Ahn[1], Suchan Chang[2], Hyung Kyu Kim[1,2], Bong Hyo Lee[2], Sang Chan Kim[2], Scott C. Steffensen[4], Kyle B. Bills[5], Hubert Lee[6], Hee Young Kim[1]

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P13: Environmental Physiology

S 96 P-13-001

Coffee consumption may promote sudomotor function activation

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S 97 P-13-002

Effect of exercise intensity on blood irisin, FGF21, adiponectin, DA and 5-HT levels

Ryeo-Won Kwon[1,2], Jin-Sun Park[1], In-Ho Lee[3], Hee-Jin Joo[2], Jeong-Beom Lee[1,2]

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S 97 P-13-003

Effects of caffeine ingestion and thermotherapy on blood orexin circulation in humans

Tae-Hwan Park[1], Jong-In Park[1,3], In-Ho Lee[2], Ji-Sang Jo[3], Sang-Hee Hong[3], Seung-Jea Lee[3], Ryeo-Won Kwon[1,3], Eon-Ah Choo[1], Jeong-Beom Lee[1,3]

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S 97 P-13-004

Psychological and Physiological Effects of Dance Movement Therapy on Depression of Juvenile Adolescents through Cortisol and Serotonin

Eon-Ah Choo[1], Jong-In Park[1,3], In-Ho Lee[2], Ji-Sang Jo[3], Sang-Hee Hong[3], Seung-Jea Lee[3], Ryeo-Won Kwon[1,3], Jeong-Beom Lee[1,3]

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S 97 P-13-005

Heat acclimation affects circulating levels of irisin, orexin and COX-2 in humans

Hye-Jin Lee[1], Jong-In Park[1,3], In-Ho Lee[2], Ji-Sang Jo[3], Sang-Hee Hong[3], Seung-Jea Lee[3], Ryeo-Won Kwon[1,3], Eon-Ah Choo[1], Jeong-Beom Lee[1,3]

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S 98 P-13-006

Sudomotor function evaluated by quantitative direct and axon reflex test in human

In-Ho Lee[1], Tae-Hwan Park[2], Jong-In Park[2,3], Ji-Sang Jo[3], Sang-Hee Hong[3], Seung-Jea Lee[3], Ryeo-Won Kwon[2,3], Eon-Ah Choo[2], Jeong-Beom Lee[2,3]

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S 98 P-13-007

Effects of music therapy as an alternative treatment on depression in children and adolescents with ADHD

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S 98 P-13-008

Thermotherapy as an alternative to exercise for metabolic health in obese postmenopausal women: Focus on circulating irisin level

Seung-Jea Lee[1,3], Tae-Wook Kim[1], Tae-Hwan Park[1], In-Ho Lee[2], Eun-Chul Jang[2], Soon-Chan Kwon[2], Hye-Jin Lee[1], Jeong-Hwan Choi[3], Jeong-Beom Lee[1,3]

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S 99 P-13-010

Firefighters' thermal and immune-inflammatory responses in a hot and humid environment

Hye-Lin Lee, Hyun-Soo Kim, Syifa Salsabila, Cho-Eun Lee, Juhyun Moon, Yujean Kim, Yesung Cho, Minseo Kim, Joo-Young Lee

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S 99 P-13-011

Age-related differences in cutaneous thermal thresholds on the trunk and periphery

Sang-Hyun Roh Roh[1], Joo-Young Lee[1,2,3]

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PL-1

Propulsive colonic contractions are mediated by inhibition-driven post-stimulus responses that originate in interstitial cells of Cajal

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The peristaltic reflex is a fundamental behavior of the gastrointestinal (GI) tract in which mucosal stimulation activates propulsive contractions. The reflex is thought to occur via stimulation of intrinsic primary afferent neurons with cell bodies in the myenteric plexus and projections to the lamina propria, distribution of information by interneurons and activation of muscle motor neurons. The current concept is that excitatory cholinergic motor neurons are activated proximal to and inhibitory neurons are activated distal to the stimulus site. We found that atropine reduced, but did not block colonic migrating motor complexes (CMMCs) in mouse, monkey and human colons, suggesting a mechanism other than one activated by cholinergic neurons is involved in generation/propagation of CMMCs. CMMCs were activated after a period of nerve stimulation in colons of each species, suggesting that the propulsive contractions of CMMCs may be due to the post-stimulus excitation that follows inhibitory neural responses. Blocking nitrergic neurotransmission inhibited post-stimulus excitation in muscle strips and blocked CMMCs in intact colons. Our data demonstrate that post-stimulus excitation is due to increased Ca^{2+} transients in colonic interstitial cells of Cajal (ICC) following cessation of nitrergic, cGMP-dependent inhibitory responses. The increase in Ca^{2+} transients after nitrergic responses activates a Ca^{2+}-activated Cl- conductance, encoded by Ano1, in ICC. Antagonists of ANO1 channels inhibit post-stimulus depolarizations in colonic muscles and CMMCs in intact colons. The post-stimulus excitatory responses in ICC are linked to cGMP-inhibited cAMP phosphodiesterase 3a and cAMP dependent effects. These data suggest novel mechanisms for generation and propagation of CMMCs in the colon.

Acknowledgement: This work was supported by NIH/NIDDK through PO1-41315

Competing interests: None.

Keywords: Colonic motility, Enteric nervous system, Interstitial cells of Cajal, SIP syncytium, Smooth muscle, Rebound excitation

인문학 특강 I

'The Kiss', a Beautiful Atlas of Early Human Development; Embryology embedded in art

유임주

고려의대

20세기 최고 걸작 중 하나로 꼽히며 한국인이 가장 사랑하는 그림 중 하나인 구스타프 클림트(1862~1918)의 <키스>를 의학자의 관점에서 새롭게 분석하여 의학과 예술을 넘나드는 '통섭' 연구가 진행되었습니다. <키스>에 그려진 문양과 상징들을 의학 문헌들과 비교 분석한 결과, 당대에 인류가 꾸준한 연구를 통해 알게 된 인간 발생의 신비를 예술적으로 표현한 작품이라는 사실을 무려 113년 만에 밝혀 내었습니다. 예술가와 의학자들간의 크로스톡을 배경으로 걸작이 그려진 1900년초 비엔나로의 타임슬립 여행에 초대합니다.

인문학 특강 II

혁명과 낭만의 과학, 그리고 과학사 속의 의과학자들

민태기

에스엔에이치기술연구소

인류의 과학기술은 역사적 상황과 밀접하게 연결되어 있으며, 당시의 사회 문화적 배경과 연관되어 발전되어 왔다. 주목할 점은 역사상 중요한 과학적 성과에는 언제나 의과학자들이 있었다. 데카르트의 '생각한다 고로 존재한다'라는 서구 최초의 근대적 철학은 그의 생리학적 관점이 반영된 것이고, 이에 대한 논쟁에서 뉴턴 '프린키피아'의 중력 법칙이 탄생했다. 여기서 촉발된 서구의 과학혁명은 의대 교수였던 베르누이로 이어지며 당대 과학자들과 치열한 경쟁을 하게 된다. 이들 과학혁명의 후계자들은 그들에게 닥친 프랑스 혁명과 나폴레옹 시대와 결코 분리될 수 없는 삶을 살았고, 그들의 업적 역시 당대의 정치적 상황과 절대 무관하지 않았음을 보여준다. 이번 강연에서는 19세기의 산업혁명에도 영향을 미친 의과학자들, 그리고 끊임없이 계속된 과학적 논쟁들이 당시의 역사적 상황 속에 어떻게 의과학자들의 업적과 결합하여 상대성이론과 양자역학 등의 현대 물리학의 기반이 되었는지를 보여준다. 또한, 이러한 잘 알려지지 않았던 의과학자들의 숨겨진 이야기들을 통해 당대의 정치, 경제뿐 아니라 음악, 미술, 문학 등의 문화예술에 미친 영향도 같이 살펴보도록 한다.

연구자와 함께하는 NRF 기초연구사업 간담회

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김성준

한국연구재단 의약학 단장

S-1-1

Non-canonical codes for behavioral sequences in neurodevelopmental diseases

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The start or end of an action sequence is an essential brain function. To do this, each behavioral program must be properly linked to the cognitive process and turned on or off depending on the situation. When they collapse, it causes many severe neurological disorders. Although basal gangalia output regions including thalamus have massive convergence inputs from the various motor system including cortex, cerebellum and basal ganglia, the underlying mechanism with behavioral sequences are largely unknown. Our goal is to unravel the neural circuits and specific cell types that are important to turn a series of actions on and off. Here, we tried to identify the role of basal ganglia output structures in the generation of action using optogenetic tools, deep brain calcium imaging, structural imaging and whole-brain activity mapping. Combining these results, we found novel cell types of output structures that are important to control neuronal ensemble related to action sequences. These suggest that basal ganglia outputs might be a new therapeutic target for neurodevelopmental disorders that show impaired action sequences.

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Keywords: Action, Basal ganglia outputs, Behavioral sequences, Neurodevelopmental disorders

S-1-2

Potent prostaglandin A1 for orphan nuclear receptor Nurr1 as a therapeutic target for Parkinson's Disease

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The orphan nuclear receptor Nurr1 is critical for the development, maintenance, and protection of midbrain dopaminergic (mDA) neurons. Moreover, post-mortem findings revealed that Nurr1 expression is significantly diminished in the substantia nigra of Parkinson's disease (PD) patients, positing Nurr1 as a potential target for developing novel and mechanism-based therapeutics for PD. Here we show that prostaglandin E1 (PGE1) and its dehydrated metabolite, PGA1, directly interact with the ligand-binding domain (LBD) of Nurr1 and stimulate its transcriptional function. We also report the crystallographic structure of Nurr1-LBD bound to PGA1 at 2.05 Å resolution. PGA1 couples covalently to Nurr1-LBD by forming a Michael adduct with Cys566, and induces notable conformational changes, including a 21˚ shift of the helix 12 away from the protein core. Furthermore, PGE1/PGA1 exhibit neuroprotective effects in a Nurr1-dependent manner, significantly enhance expression of Nurr1 target genes in mDA neurons, and improve motor deficits in MPTP-lesioned mouse models of PD. Based on these results, we propose that PGE1/PGA1 represent native ligands of Nurr1 and can exert neuroprotective effects on mDA neurons, via activation of Nurr1's transcriptional function.

Acknowledgement: This study was conducted with Professor Kwang-Soo Kim (Meclean Hospital, Harvard Medical School, USA) and Professor Ho Sup Yoon (Nanyang Technological University, Singapore).

Competing interests: Yongwoo Jang hold the patent applications related to the contents of this work (US20200206309A1).

Keywords: NR4A2, Nurr1, Prostaglandin A1, Neuroprotective effect, Parkinson's disease

S-1-3

Conditional coexpression of AIMP2 and α-synuclein models Lewy body dementia

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Dysfunction of the E3 ubiquitin ligase parkin due to mutations or post-translational modifications contributes to Parkinson's disease (PD). Accumulation of diverse parkin substrates may underlie key pathologic features of PD including progressive loss of dopamine neurons, and formation of Lewy body inclusion that is composed of misfolded α-synuclein. Aminoacyl-tRNA synthetase interacting multifunctional protein-2 (AIMP2) is a parkin substrate that accumulates in mouse brains and postmortem human PD brains of parkin inactivation. Previously, we have reported that aberrant accumulation of AIMP2 is sufficient to stimulate a distinct type of cell death (parthanatos) that is mediated by overactivation of poly (ADP-ribose) polymerase-1 (PARP1) and subsequent production of poly (ADP-ribose). However, the molecular mechanisms by which AIMP2 potentially contributes to Lewy body formation are largely unknown, although it has been reported that AIMP2 can produce aggregate structure in cells and it is present in Lewy body inclusion in human PD brains. In this seminar, I will share our recent findings that showed pathologically active roles of AIMP2 in -synuclein interaction and Lewy body formation. I will also present ongoing development of a novel mouse model of end-stage PD with dementia symptoms by exploiting synergistic AIMP2-α-synuclein interaction.

Acknowledgement: This work was supported by grants from the National Research Foundation of Korea funded by the Korean government (NRF-2017M3C7A1043848).

Competing interests: Y.L. holds the patent related to the contents of this work (Patent no. 10-2198497).

Keywords: Parkin, AIMP2, A-synuclein, Lewy body, Parkinson's disease, Dementia

S-1-4

PET imaging reveals reactive astrocyte-mediated neuronal hypometabolism in Alzheimer's disease patients

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Reactive astrogliosis is a hallmark of Alzheimer's disease (AD). However, a clinically validated neuroimaging probe to visualize the reactive astrogliosis is yet to be discovered. Here, we report that PET imaging with ^{11}C-acetate and ^{18}F-fluorodeoxyglucose (^{18}F-FDG) functionally visualizes the reactive astrocyte-mediated neuronal hypometabolism in the brains with neuroinflammation and AD. Multifaceted approaches including PET imaging, autoradiography, immunohistochemistry, metabolomics, and electrophysiology were used in a neuroinflammation rat model, two AD mouse models, and AD patients. We demonstrate that reactive astrocytes excessively absorb acetate through elevated monocarboxylate transporter-1, leading to aberrant GABA synthesis and release which suppresses neuronal activity and glucose uptake through decreased glucose transporter-3 in the diseased brains. Furthermore, we find a strong correlation between the patients' cognitive function and PET signals of both ^{11}C-acetate and ^{18}F-FDG. We propose the functional PET imaging for astrocytic acetate-hypermetabolism and neuronal glucose-hypometabolism as an advanced diagnostic strategy for AD.

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Competing interests: The authors declare no conflicts of interest.

Keywords: Alzheimer's disease, ^{11}C-Acetate, ^{18}F-Fluorodeoxyglucose, Monocarboxylate Transporter 1 (MCT1), PET imaging, Reactive astrocyte

S-1-5

Hypothalamic neural stem cells in aging

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The hypothalamus is a key neuroendocrine system known to regulate energy homeostasis via the orchestrated actions of neuronal pathways and neuroendocrine hormones that regulate energy balance and nutrient homeostasis. Nutritional status exerts important effects on various types of hypothalamic signaling, such as insulin and leptin pathways, and hypothalamic dysfunction is a critical cause of metabolic syndrome and its related diseases, including aging and immune dysfunction. It has been proposed that hypothalamus helps to control aging. Hypothalamic stem/progenitor cells were observed with a substantial loss of these cells. Conversely, aging retardation and lifespan extension were achieved in mid-aged mice that were locally implanted with healthy hypothalamic stem/ progenitor cells and their microRNAs. Intra-brain delivery of TNF, mimicked bacterial infection, rapidly increase the number of peripheral lymphocytes, especially in the spleen and fat. Hypothalamic induction of lipolysis mediated the brain's action in promoting this increase in the peripheral adaptive immune response. In conclusion, the treatment to hypothalamus with specific factors, including hypothalamic stem/ progenitor cells, TNF, etc., contributes to control metabolic function and disorders.

Keywords: Hypothalamus, Neural stem cells, Exosome, Aging

S-2-1

Role of PRMT1 in NAFLD-associated hepatic fibrosis

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Protein arginine methyltransferase (PRMT) 1 has been shown to be involved in the various metabolic signaling pathways. Previously, we and others have shown that PRMT1 regulates catabolic pathways in the skeletal muscle and white adipose tissues, protecting mice from developments of muscle atrophy or lipodystrophy, respectively. Although nonalcoholic fatty liver disease (NAFLD) has been emerged as one of the most significant health threats in recent years, the involvement of PRMT1 in the NAFLD-associated liver disorders has not been described to date.
In this presentation, we would like to describe the protective role of PRMT1 in reactive oxygen species (ROS)-mediated cellular damage caused by the metabolic insults in the liver, thus preventing the development of hepatic fibrosis in response to methionine/choline deficient diet in mice. We would also like to discuss the role of PRMT1 in the development of NAFLD-associated hepatocellular carcinogenesis both in humans and the mouse model.

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Keywords: Nonalcoholic fatty liver disease, Protein arginine methyltransferase 1, Reactive oxygen species

S-2-2

Loss of SREBP-1c ameliorates iron-induced liver fibrosis via decrease of lipocalin-2

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Sterol regulatory element-binding protein (SREBP)-1c is involved in cellular lipid homeostasis and cholesterol biosynthesis and highly increased in non-alcoholic steatohepatitis (NASH). However, the molecular mechanism and function by which SREBP-1c regulates hepatic stellate cells (HSC) activation in NASH animal model and patients have not been fully elucidated. Here, we found that LCN2 gene expression and secretion were increased in CCl4-induced liver fibrosis mice models and LCN2 gene transcription was regulated by SREBP-1c. Moreover, recombinant LCN2 treatment in primary HSC stimulated intracellular iron accumulation and fibrosis gene expression in WT HSC, and these effects were reduced by treatment with iron chelator, deferoxamine, whereas LCN2-treated intracellular iron accumulation was not increased in SREBP-1c defect HSC, indicating that SREBP-1c-induced LCN2 expression and secretion stimulate HSC activation through iron accumulation in HSC. Also, LCN2 expression level was highly correlated with inflammation as well as fibrosis in the NASH patients. Therefore, those results suggest that deficiency of SREBP-1c ameliorates development of NASH through regulation of LCN2 gene expression in mice and human.

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Keywords: SREBP-1c, LCN2, NASH, Iron, Hepatic stellate cell

S-2-3

Identification of novel targets for pulmonary fibrosis

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Lung fibrosis is one of the chronic diseases caused by various factors such as fine dust, radiation, virus, and chemicals. Idiopathic pulmonary fibrosis (IPF) is a chronic progressive fibrotic disease of unknown etiology that is marked by progressive deposition of extracellular matrix proteins and ultimately failure of the respiratory system and death. The incidence and prevalence of IPF appear to be increasing. Currently, only three pharmacological treatments for IPF, namely pirfenidone (TGF-beta inhibitor), nintedanib (multiple tyrosine kinase inhibitor), and N-acetylcystein are commercially available.
We have previously developed a mouse model simulating clinical SBRT and have used the model to validate the induction of lung fibrosis by high-dose ionizing radiation (IR). The regimen was similar to that used for human therapy reflecting the understanding of the clinically related IR-mediated normal cell damage like fibrosis.
To identify molecular signatures for lung fibrosis, we further examined the fibrosis process at 4 weeks from irradiated region from mouse lung tissues after IR and identified targets (GTSE1 and Cathepsin S) for lung fibrosis. Moreover, we also identified another molecular targets in lung fibrosis development. Hsp27 expression was increased during IR-induced lung fibrosis, and functional inhibition of Hsp27 using a small molecule ameliorated lung fibrosis. While investigating mechanisms of Hsp27 in the development of lung fibrosis, we found that IkBα-NFkB signaling activation by direct interaction of IkBα with Hsp27, is involved in the EMT process that is tightly connected to the development of IR-induced lung fibrosis.

Keywords: Idiopathic pulmonary fibrosis, Novel targets, Hsp27, Cathepsin S, Gtse1

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S-2-4

Kidney fibrosis: is it reversible?

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Kidney fibrosis is structurally identical to fibrosis of other organs, characterized by an increase in the extracellular matrix. Its mechanism of development is known to be similar to that of other organs. However, because the kidney is a complex organ composed of several types of cells, fibrotic lesions can occur in functional compartments such as glomeruli, tubulo-interstitium, and vasculature. Functionally, kidney fibrosis results in a gradual loss of function, resulting in distinct clinical symptoms that differ from other organs. Progressive chronic kidney disease often results in extensive tissue scarring, leading to complete destruction of kidney parenchyma and end-stage kidney failure, a devastating condition requiring dialysis or kidney transplantation.

Characterization of mediators of chronic kidney disease progression and therapeutic goals over the past decade has been a challenge in the scientific community. In the case of kidney disease, regardless of the initial cause, major structural changes occur due to expansion of kidney fibrosis due to dysregulation of profibrotic and antifibrotic factors. In this talk, I discuss the role of key factors promoting kidney fibrosis in both experimental models and human diseases and various therapeutic concepts to inhibit or reverse chronic kidney disease. A better understanding of these issues will not only be essential to elucidate the pathogenic mechanisms of chronic kidney disease, but may also provide new insights for the development of novel therapeutic strategies.

Acknowledgement: This work was supported by grants from the National Research Foundation of Korea funded by the Korean government (NRF-2020R1A2C2003438).

Keywords: Kidney fibrosis, Chronic kidney disease

S-2-5

Targeting autotaxin improves pathophysiologic features of fibrocalcific aortic valve disease

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Fibrocalcific aortic valve disease (FCAVD) accompanies inflammatory cell infiltration, fibrosis, and ultimately calcification of the valve leaflets. We here demonstrated that the lipoprotein(a) - autotaxin (ATX) - lysophosphatidic acid axis involves in the development of fibrotic changes of FCAVD. ATX inhibition significantly decreased osteogenic differentiation and calcification of human valvular interstitial cells *in vitro*, which was accompanied by a reduction in the expression of both osteogenic markers and fibrosis-related gene expression. In addition, ATX administration ameliorated the rate of change in the transaortic peak velocity and mean pressure gradients in *in vivo* rabbit model as assessed by echocardiography. Importantly, ATX administration was also found to suppress the effects of a high-cholesterol diet and of vitamin D2-driven fibrosis in association with a reduction in calcific deposition in aortic valves. Thus, ATX inhibition shows favorable effects to prevent FCAVD progression by inhibiting both fibrosis and calcification, suggesting its potential as a selective therapeutic agent for the inhibition of FCAVD.

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Competing interests: Bridgebio Therapeutics. Inc. hold the patent applications related to the contents of this work.

Keywords: Fibrocalcific aortic valve disease, Aortic valve, Autotaxin, Fibrosis, Calcification

S-3-1

Endothelium-mediated control of vascular contractility in physiological and pathophysiological conditions

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Endothelial cells play an important role in the control of vascular contractility by releasing NO and evoking hyperpolarization of smooth muscle cells. Ca^{2+}-activated K^+ channels ($K_{Ca}2.3$ and $K_{Ca}3.1$) regulate endothelial function via modulating Ca^{2+} influx through Ca^{2+} entry channels. Altered expression of these Ca^{2+}-activated K^+ channels on cell membrane was found in physiological (normal pregnancy, and aging) and pathophysiological (vascular diseases such as pregnancy-induced hypertension) conditions. Endothelial $K_{Ca}2.3$ and $K_{Ca}3.1$ upregulation increased $K_{Ca}3.1$ activation-induced, NO- and prostacyclin-resistant endothelium-dependent relaxation during aging and pregnancy. Growth factor, such as VEGF and TGFβ, estrogen, progesterone, and altered sphingolipid composition upregulated $K_{Ca}2.3$ and $K_{Ca}3.1$ via elevating H_2O_2 signaling, whereas superoxide, oxidized LDL, sFlt-1, and cAMP downregulated $K_{Ca}2.3$ and $K_{Ca}3.1$. $K_{Ca}2.3$ and KCa3.1 upregulation was found in catalase and GPX1 double knock-out mice and ceramide synthase 2 null mice. Estrogen, sphingolipids, and growth factors downregulated H_2O_2-degrading antioxidant enzymes, such as catalase and GPX, thereby elevating H_2O_2 levels. Downregulation of H_2O_2-degrading antioxidant enzymes was found in ceramide synthase 2 null mice. Whereas oxidized LDL or sFlt-1 upregulated NADPH oxidases and downregulated SODs, thereby elevating superoxide levels. The H_2O_2 donor TBHP upregulated $K_{Ca}2.3$ and $K_{Ca}3.1$, whereas the superoxide donor xanthine/xanthine oxidase downregulated $K_{Ca}2.3$ and $K_{Ca}3.1$. Membrane levels of $K_{Ca}2.3$ and $K_{Ca}3.1$ were regulated via clathrin- or caveolae-mediated internalization and Rab5C- and EEA1-mediated transportation. These results suggest that altered redox state plays an important role in the regulation of endothelial function via modulating expression levels of $K_{Ca}2.3$ and $K_{Ca}3.1$ in physiological and pathophysiological condition. Expression levels of $K_{Ca}2.3$ and $K_{Ca}3.1$ may represent endothelial function.

Keywords: Endothelial cells, $K_{Ca}2.3$ and $K_{Ca}3.1$, Endothelial dysfunction, Expression level

S-3-2

Vasodilatory effects and the underlying mechanisms of the medicinal plants extracts in rat mesenteric resistance arteries

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Cardiovascular disease (CVD) is one of the leading causes of morbidity and mortality worldwide. Hypertension is known to be the major risk factor for CVD. Thus, it is substantial to prevent and treat hypertension to reduce the risk of CVD. Although synthetic medications have been widely used to treat and cure patients at various stages of CVD, including hypertension, the adverse effects remain a challenge. In addition to the use of synthetic drugs to treat hypertension, the use of natural product is widely increasing over the decades. In the present study, we investigated the effect of several extracts from medicinal plants in rat mesenteric resistance arteries. Among various plants extract, *phellinus linteus extracts*, *trachelospermi caulis* extract, and *alpinia officinarum* extract induced significant vascular relaxation in rat mesenteric resistance arteries. We found that *phellinus linteus* extract induces vasodilation through opening of intermediate-conductance calcium-activated potassium channel (IK_{Ca}) and causing hyperpolarization of vascular smooth muscle cells, thereby reducing calcium and phosphorylation levels of 20 KDa myosin light chain (MLC_{20}). And we also found that direct inhibition of extracellular Ca^{2+} influx is involved in *trachelospermi caulis* extract- and *alpinia officinarum* extract-induced vascular relaxation. The present study suggests the potential basis of medicinal plants extracts as antihypertensive agents by showing their significant vasodilatory effect.

Acknowledgement: This work was supported by grants from the National Research Foundation of Korea funded by the Korean government (2018R1D1A1B07041820 and 2019R1F1A1061771).

Competing interests: None

Keywords: Vasodilation, Vascular relaxation, Mesenteric resistance artery, Medicinal plant

S-3-3

GI motility – organ level investigation

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Gastrointestinal (GI) motility consists of spontaneous and rhythmic phasic contractions from slow waves and periodic mass movements as migrating motor complexes (MMC) in animal models. Phasic contractions mix materials in the intestine, and MMC propel the materials from the proximal to distal intestine. Slow waves originate from the interstitial cells of Cajal (ICC), especially from the ICC in the myenteric region (ICC-MY) between circular muscle (CM) and longitudinal muscle (LM) in the small bowel. The electrical activities of the ICC, which include changes in membrane potential from the activation of various ion channels, provoke slow waves in smooth muscle cells connected with ICC by gap junctions. Thus, the ICC acts as a pacemaker for the generation of smooth muscle phasic contractions. However, MMC are thought to be controlled by the enteric nervous system and not by the ICC. The MMC have been reported to be subjected to cholinergic agents or neuronal blockers. Rectum is a major organ for continence and defecation. It is the last part of the large intestine, which can serve as fecal reservoir and finally push out the feces through the anus. Rectal compliance affects capacity for the fecal storage and large compliance of the rectum is a unique functional characteristic differentiated from the colon. The main role of the colon is propagation of feces from proximal to distal part. The peristalsis can develop by means of ascending excitation and descending inhibition in enteric nervous system (ENS). When the feces enter the colon, it can be moved by the colonic migrating motor complex (CMMC) with proximal contraction and distal relaxation. However, the rectum reserves the feces first until the storage capacity come to be full and then contract to move the feces out through the anus. The rectal wall can be relaxed as the fecal volume increasing, and the rectal pressure may not be elevated due to the compliance of the rectum. After the rectal distension reach to some threshold volume, the rectum starts to contract for defecation. Abnormal rectal compliance can be presented in many functional gastrointestinal (GI) motility disorders, such as irritable bowel syndrome (IBS), constipation or fecal incontinence. It has been reported that the rectal compliance can be significantly lower in diarrhea-predominant IBS and can be higher in constipation. These abnormal rectal function can make the patients' quality of life worse. The physiologic function of rectal compliance can also be abolished after anterior resection for rectal cancer. Most of the patients suffer from fecal urgency, frequency and incontinence in their lifetime, which is anterior resection syndrome (ARS). Inevitably, it results from removing the rectum and loss of the fecal reservoir. As there has been no specific effective treatment for ARS and decreased compliance, only the symptom based conservative management has been tried. Intrinsic inhibitory neuromuscular transmission can cause the relaxation of the GI smooth muscle. Nitric oxide (NO) is an inhibitory non-adrenergic, non-cholinergic neurotransmitter in the ENS. NO is released from enteric neurons expressing nNOS (NOS1) and can cause outward K+ currents and hyperpolarization of membrane potential at smooth muscle cells. These nitrergic inhibition can be mediated by interstitial cells in GI smooth muscle, as the NOS1+ neurons contact closely with smooth muscle cells, ICC and platelet-derived growth factor receptor α–positive (PDGFRα+) cells, which form SIP syncytium, electrically coupled with each other . The PDGFRα+ cells also have a role of inhibitory regulation of gastrointestinal motility, related to purinergic neurotransmission. The purine can bind G-protein coupled P2Y1 receptor on the membrane of PDGFRα+ cells and activate the small-conductance Ca^{2+}-activated K+ (SK3) channels. The purines can induce outward K+ currents through SK3 channels and the P2Y1 receptor antagonist, MRS2500 blocked this current. Nitrergic and purinergic neurotransmissions have been reported to be present in the rectal smooth muscle, and can inhibit the contractions in the rectum. Large compliance, different from the colonic CMMC, were identified in the murine rectum. Enteric inhibitory neurotransmissions associated with nitric oxide or purine were related to the rectal compliance and ICC or PDGFRα+ cells can control the rectal smooth muscle activities. These electrophysiological and mechanical characteristics of rectal compliance can be used for further studies of gastrointestinal motility disorders.

S-3-4

The role of K_{ATP} channel activation in lymphatic contractile dysfunction associated with metabolic disease

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Lymphatic contractile dysfunction has been implicated in metabolic diseases, including diabetes and obesity. A positive correlation between KATP channel hyperactivity in lymphatic muscle and impaired lymphatic pumping suggests that KATP channel activation is a common cause of lymphatic contractile dysfunction. We investigated the role of KATP channel activation in response to metabolic stressors that mimic metabolic diseases. Exposure of WT lymphatic vessels (LVs) to high glucose or mitochondrial electron transport complex (ETC) inhibitors led to a decrease in the frequency of spontaneous lymphatic contractions that was rescued by the KATP channel inhibitor, GLIB. Contractions of LVs from Kir6.1-/- mice were resistant to inhibition of frequency by high glucose and ETC inhibitors. Antimycin A (a mitochondrial ETC III inhibitor) increased the production of ROS in WT LVs. After pretreatment with ROS scavengers, WT LVs were resistant to the effects of antimycin A, suggesting that KATP channels were activated by ROS production. However, ROS scavengers did not block the inhibitory effect of rotenone or CCCP on pumping, indicating that KATP channels could also be activated by an increased ADP/ATP ratio independent of ROS. To test if KATP channel activation by chronic metabolic stress induced lymphatic dysfunction in animals, leptin receptor-deficient (db/db) mice were

used as a model of metabolic disease. Spontaneous contraction amplitude was blunted in LVs from db/db mice and absent at lower pressures. GLIB restored contraction frequency. In summary, we propose that KATP channels in LVs are activated by the ADP/ATP ratio and/or ROS generation as a result of metabolic stress and contribute to the lymphatic contractile dysfunction associated with metabolic disease.

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Keywords: KATP channels, Lymphatic contractile dysfunction, Metabolic stress, ROS

S-3-5

Increased diphosphorylation of MLC2 is responsible for the impaired relaxation state of pulmonary arteries in the monocrotaline-induced pulmonary arterial hypertension

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Phosphorylation at threonine 18 (T18-P), serine 19 (S19-P) or both (T18/S19-diP) in myosin regulatory light chain (MLC2) is critical for arterial smooth muscle contraction. Other than S19-P, T18-P requires specific condition such as rho A-dependent kinase (ROCK) activation or myosin light chain phosphatase (MLCP, MYPT1) inhibition. The MLCP activity is increased by cGMP-dependent PKG signaling and inhibited by ROCK. While S19-P is easily reversible by MLCP, T18-P and T18/S19-diP are slowly dephosphorylated. In this study, we investigated the speed of relaxation in rat pulmonary arteries (PAs), which showed stark delay of relaxation (half relaxation time(s): 19±2.9 vs. 335±59.5) after a high K+–induced contraction (80K-contraction) in the monocrotaline-induced pulmonary arterial hypertension (PAH-MCT) model. Interestingly, T18/S19-diP was significant in the PAs from PAH-MCT. Consistently, not only soluble guanylate cyclase (sGC) and PKG, but also the MLCP expression was decreased along with the increase of ROCK. The delayed relaxation was almost completely reversed by ROCK inhibitor (Y27632) whereas not significantly affected by membrane permeable 8-Br-cGMP. Different from the contractile response, recovery of increased $[Ca^{2+}]i$ in PA smooth muscle cell was not different between control and PAH-MCT. Furthermore, the delayed relaxation was still observed with L-type Ca^{2+} channel blocker or even with Ca^{2+}-free bath solution. Finally, in the control PAs, the pharmacological inhibition of cGMP production by ODQ induced prominent delay of relaxation and MLC2 diphosphorylation as like the responses of PAH-MCT. In the presence of ODQ, the applications of 8-Br-cGMP or Y27632 largely reversed the delayed relaxation along with the decrease of T18/S19-diP. Taken together, the diphosphorylation of MLC2 accounts for the impaired relaxation of PA in PAH animal via loss of MLCP and elevated ROCK expression.

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Competing interests: Nothing to declare

Keywords: Pulmonary artery, Smooth muscle, Pulmonary Arterial Hypertension, Myosin light chain phosphorylation, Myosin light chain phosphatase, Rho A-dependent kinase

S-3-6

Spontaneous vasomotion in human arteries and their ion channel-based mechanism in the smooth muscle

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Vasomotion is the oscillation of vascular tone which gives rise to flow motion of blood into an organ. It was described in over 100 years ago however the physiological and pathophysiological implications are not well known. Our study was focused to elucidate mechanism and physiological function of vasomotion in in human arteries.

Conventional contractile measuring system, immunohistochemistry, and molecular study were used using human gastric and uterine arteries. Circular muscle of human left gastric artery produced sustained tonic contraction by high K^+ (50mM) which is blocked by nifedipine (2μM), inhibitor of L-type Ca^{2+} channel ($VDCC_L$). Stepwise stretch and high K produced nerve-independent spontaneous contraction(vasomotion). Vasomotion was also produced by application of Bayk 8644(activator of $VDCC_L$), 5-HT, prostagrandins, oxytocin and so an. It was blocked by nifedipine (2μM) and blockers of intracellular Ca^{2+} stores. Inhibitors of Ca^{2+}-activated Cl^- channels (DIDS and/or niflumic acid) and ATP-sensitive K^+ (K_{ATP}) channels inhibited vasomotion reversibly. Metabolic inhibition by application of NaCN and several neuropeptides also regulated vasomotion in a reversible manner too. Finally, in molecular study, we identified Ca^{2+}-activated Cl^- channels, subunits of ATP-sensitive K^+ (K_{ATP}) channels (Kir 6.1/6.2 and sulfonylurea receptor 2B (SUR2B)), and c-Kit positivity in Western blot.

From these results, we found vasomotion which is sensitive to TME-M16A-Ca^{2+}-activated Cl^- channels and metabolic changes in human gastric and uterine arteries. Vasomotion might be play an important for the regulation of microcirculation circulation even in pacemaker-related autonomic contractile organs in human.

Keywords: Human gastroepiploic artery, Human uterine artery, Vasomotion, Ca^{2+}, TMEM16A-sensitive Ca^{2+}-activated Cl^- channels, ATP-sensitive K^+ (K_{ATP}) channel

S-4-1

Role of lysophosphatidylcholine in neutrophil-gated immune response during sepsis

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Sepsis is predominantly initiated by bacterial infection and can cause systemic inflammation accompanied by multiple organ failure, which frequently leads to rapid death of the patient. However, this acute systemic inflammatory response requires further investigation from the perspectives of clinical judgment criteria and early treatment strategies for the relief of symptoms. Lysophosphatidylcholine (LPC) 18:0 may relieve septic symptoms, but the relevant mechanism is not clearly understood. Therefore, we aimed to assess the effectiveness of LPC as a therapeutic treatment for acute inflammation in the lung induced by lipopolysaccharide in mice. We examined whether it alleviated the inflammatory effect of sepsis both *in vitro* and *in vivo*. The results verified that LPC treatment did not influence recruitment of innate immune cells to the lung. However, it altered neutrophil migratory patterns and enhanced phagocytic efficacy in the damaged lung. Moreover, LPC treatment reduced the release of neutrophil extracellular traps (NETs), namely NET formation, which can damage tissues and

exacerbate disease. It also reduced the migration of human neutrophils under septic conditions. Therefore, our results suggest that LPC treatment can alleviate sepsis-induced lung inflammation by regulating the function of neutrophils. These findings provide evidence for the beneficial application of LPC treatment as a potential therapeutic strategy for sepsis.

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Competing interests: All authors declare that the research was conducted by in the absence of any commercial or financial relationships

Keywords: Sepsis, Lysophoshatidylcholine, Inflammation, Neutrophil extracellular trap

S-4-2

In vivo two-photon microscopy imaging of glia-mediated synapse remodeling during chronic pain

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Recent advances in two-photon microscopy, fluorescence labeling techniques and genetically encoded calcium indicators have enabled us to directly observe the structural and functional changes in neurons and glia, and even at synapses, in the brain of living animals. Long-term in vivo two-photon imaging studies have shown that some postsynaptic dendritic spines in the adult cortex are rapidly eliminated or newly generated, in response to altered sensory input or synaptic activity, resulting in experience/activity-dependent rewiring of neuronal circuits. *In vivo* two-photon Ca^{2+} imaging studies have revealed the distinct, input-specific response patterns of excitatory neurons in the brain. These updated *in vivo* approaches are now being widely used for the study of pathophysiological mechanisms of neurological diseases. In this talk, I will introduce my previous and ongoing works in the last decade, focusing on *in vivo* two-photon microscopy imaging of glia-mediated synapse remodeling during chronic neuropathic pain.

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Competing interests: The speaker (SKK) declares that there is no competing interest.

Keywords: Two-photon microscopy, Glia, Synapse remodeling, Chronic neuropathic pain

S-4-3

Intelligence at the nanoscale: super-resolution imaging of brain structure and function

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Brain cells such as neurons and astrocytes exhibit an extremely elaborate morphology, and their functional specializations like synapses and glial processes often fall below the resolution limit of conventional light microscopy. This is a huge obstacle for neurobiologists because the nanoarchitecture critically shapes fundamental functions like synaptic transmission and Ca^{2+} signaling. Super-resolution microscopy can overcome this problem, offering the chance to visualize the structural and molecular organization of brain cells in a living and dynamic tissue context, unlike traditional methods like electron microscopy or atomic force microscopy. In my talk I will review our contributions to developing live-cell super-resolution (STED) microscopy approaches and their application to key problems in cellular neurobiology concerning the structure, function and plasticity of hippocampal synapses and the surrounding extracellular space.

Competing interests: There is no competing interests to declare.

Keywords: Two-photon STED microscopy, Super-resolution shadow imaging, Extracellular calcium imaging, Dendritic spines, Tripartite synapses

S-4-4

Brain micro-anatomy revealed by 2-photon shadow imaging *in vivo*

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Getting an accurate, detailed and physiologically relevant view of brain structure and neuronal circuits is a major goal of modern neuroscience. Current large-scale connectomics efforts rely either on EM or MRI, which are either incompatible with live conditions or do not offer cellular resolution. Fluorescence microscopy allows for live imaging with cellular resolution *in vivo*, but has relied on positively labeling of a sparse set of cells, giving an incomplete and biased view of the anatomical organization of brain tissue. Breaking this impasse, super-resolution shadow imaging (SUSHI) established a new paradigm to visualize tissue anatomy in brain slices with nanoscale resolution in an all-encompassing and panoramic way, based on fluorescence labeling of the ACSF and 3D-STED microscopy. Because of the stringent optical demands of super-resolution microscopy, however, the approach has only been applied to living organotypic brain slices so far.
We have now extended the shadow imaging concept to the mouse brain *in vivo*, based on 2-photon shadow imaging (TUSHI) and labeling of the cerebrospinal fluid with a fluorescent membrane-impermeant dye. We present the optical details of the microscope, the labeling strategy for sufficiently bright and homogeneous inverted cellular contrast, as well as the cranial window technique and anesthesia formula for optically clear and mechanically stable access to superficial layers of the cerebral cortex. Despite the diffraction-limited resolution, the new approach opens a stunning window on the micro-anatomical organization of the brain *in vivo*, where cell bodies, dendritic branches of neurons, perivascular spaces and spatial heterogeneities in the extracellular space become visible. By adding a second fluorescence channel, the shadow imaging approach reveals the diverse and complex anatomical context of positively labeled neurons, astrocytes, microglia and tumor cells.
In summary, our work demonstrates the feasibility of TUSHI *in vivo* to visualize brain structure and context with subcellular resolution. It provides a powerful new investigative tool to monitor dynamical changes of brain structures in vivo under various (patho-physiological) conditions, such as experience-dependent neuronal plasticity, sleep, aging, stroke, tumor invasion & proliferation.

Competing interests: There is no competing interests to declare.

Keywords: 2-photon shadow imaging, Super-resolution shadow imaging (SUSHI), Brain microanatomy, *In vivo* imaging

S-5-1

Structure of epithelial cells in nephron segments

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Renal tubular epithelial cells modulate glomerular filtered primary urine components through reabsorption or secretion. The morphology of tubular epithelial cells is unique to each nephron segment. The proximal tubule (PT) is a simple cuboid type and contains many mitochondria in the cyto-

plasm. The apical membrane of PT has numerous finger-like protrusions called brush border, increasing the surface area. Descending thin limb (DTL) is distinct from PT, in which the epithelium abruptly changes to a flattened form. In pathological conditions (e.g., ischemia-reperfusion injury), PT cells may become difficult to distinguish from DTL due to the flattening of the epithelium and loss of the brush border. Ascending thin limb (ATL) also has a flat epithelial type and is responsible for the urine concentration mechanism based on the countercurrent multiplier system. There are two main types of nephrons: short (cortical) and long (juxtamedullary) loop nephrons. The short loop nephron lacks ATL and its DTL directly leads to the thick ascending limb (TAL). In contrast to the long loop, the DTL of the short loop nephron does not express AQP1 but has UT-A2. The distal tubule (DT) consists of the TAL, the macula densa, and the distal convoluted tubule (DCT). Macula densa is located at the junction of the TAL and DCT. In general, DT cells are characterized by extensive infoldings of the basolateral membrane and their nuclei tend to be positioned to the luminal side. Several nephrons merge to form the collecting duct system via connecting tubules. The collecting duct is subdivided into the cortical collecting duct (CCD), the outer medullary collecting duct (OMCD), and the inner medullary collecting duct (IMCD). Two distinct cell populations exist in the collecting duct: principal cells and intercalated cells. Principal cells are the predominant cell type and have short, sparse microvilli and few organelles. Intercalated cells have developed projections on the apical membrane and contain many cytoplasmic vesicles and mitochondria. The various morphologies of renal epithelial cells reflect their unique functions.

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Competing interests: none

Keywords: Kidney, Epithelium, Cell type, Membrane, Organelle

S-5-2

Single cell transcriptome reveals cell diversity in the kidney

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Our understanding of kidney physiology is limited by an incomplete molecular characterization of the cell types responsible for the organ's multiple homeostatic functions. Whereas prior studies have been able to analyze only the averaged outputs from renal tissue, we now can accurately monitor genome-wide gene expression, regulation, function, cellular history, and cellular interactions in thousands of individual cells in a single experiment. These methods are key drivers in changing our previous morphotype-based organ and disease descriptions to unbiased genomic definitions and therefore improving our understanding of kidney development, homeostasis, and disease. Using unbiased single cell RNA sequencing of healthy mouse kidneys, we identified 16 distinct cell types. Cell trajectory analysis and lineage tracing revealed novel transitional cell type and unexpected plasticity in adult renal collecting duct via Notch mediated interconversion. Furthermore, changes in ratio between the collecting duct cell types were observed in kidney disease conditions. In addition, we created a comprehensive single cell atlas of glomerular cells after isolation of glomerulus. This data provides new insights into parietal epithelial cells and podocyte biology in healthy and diabetic kidneys. In summary, single cell analysis advanced a mechanistic description of kidney diseases by identifying defective homeostatic cell lineages.

Acknowledgement: This work was supported by the Samsung Science and Technology Foundation (SSTF-BA2001-11)

Competing interests: none

Keywords: Single cell RNA sequencing, Kidney cell type, Collecting duct, Glomerulus, Single cell atlas

S-5-3

Regulation of renal aquaporin-2 in kidney collecting duct

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The kidney collecting duct (CD) is the renal tubular segment, in which the osmolality and volume of the final urine are established. This process makes urine concentrated under the arginine vasopressin (AVP) stimulation and contributes to body water homeostasis. AVP binds to the arginine vasopressin receptor 2 (AVPR2) and increases osmotic water permeability of the CD principal cells. The signaling cascade involes the water channel protein aquaporin-2 (AQP2). Specifically, AVP induces the intracellular trafficking of AQP2-expressing vesicles to the apical plasma membrane, thereby, increasing the osmotic water permeability of CD cells. Moreover, AVP stimulates the transcription of the *Aqp2* gene, inducing the AQP2 protein abundance. Moreover, AVP-independent mechanisms for the AQP2 trafficking to the plasma membrane are also present. This can be achieved by bypassing AVPR2 signaling and inducing AQP2 accumulation in the membrane. There are two categories: 1) intracellular cAMP elevation by either activating other GPCRs or inhibiting phosphodiesterases; and 2) cAMP-independent pathways. Endogenously expressed G-protein-coupled receptors (GPCRs) besides AVPR2 are present in the renal CD that naturally couple to GαS to increase cAMP levels and regulate AQP2 expression. There are several potential candidates, including the prostaglandin E receptors (EP2 and EP4), β3-adrenergic receptor (β3-AR), calcitonin receptor, secretin receptor, and TGR5 (bile acid-activated membrane receptor). Alternatively, there are GPCRs that do not couple to GαS and cAMP pathways but regulates AQP2 expression, including frizzled receptor and EGF receptor. In addition, AQP2 protein abundance is regulated by post-translational modification, e.g., ubiquitination or RNA interference. This talk deals mainly with regulation of AQP2, which could provide new insights into the treatment of hereditary nephrogenic diabetes insipidus associated with mutations of either *AVPR2* or *AQP2* gene.

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Competing interests: None

Keywords: Aquaporin, Collecting duct, Vasopressin

S-5-4

Renal Na^+ transporters and salt-sensitive hypertension

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The kidney plays a major role in blood pressure regulation because of its sodium handling. Blood pressure is a function of plasma volume and peripheral vascular resistance, and both are determined by sodium balance. After glomerular filtration, sodium is reabsorbed at four sites of nephron through the respective major Na^+ transporters: NHE3 in the proximal tubule, NKCC2 in the thick ascending limb of Henle's loop, NCC in the distal convoluted tubule, and ENaC in the collecting duct. In particular, the latter two nephron segments are regulated by aldosterone to finely adjust urinary sodium excretion. Many patients with essential hypertension are salt-sensitive, and their polygenic inheritance may be explained by combined variations in the genes of renal sodium handling. In addition, ENaC is localized in the vascular endothelium and exerts vasoconstriction in response to high sodium and aldosterone. Other Na^+ transporters are located in macrophages/monocytes, dendritic cells, and T lymphocytes and release inflammatory cytokines in response to NaCl. Taken together, Na^+ transporters play the piv-

otal role in the pathogenesis of salt-sensitive hypertension via extracellular fluid expansion, vasoconstriction, and proinflammation.

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Competing interests: None.

Keywords: Aldosterone-sensitive distal nephron, Blood pressure, Endothelium, Inflammation, Kidney, Sodium

S-6-1

PSME4 degrades acetylated YAP1 in the nucleus of mesenchymal stem cells to induce cardiac commitment

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Intensive research has focused on minimizing the infarct area and stimulating endogenous regeneration after myocardial infarction. Our group elucidated that apicidin, a histone deacetylase (HDAC) inhibitor, robustly stimulates cardiac commitment of mesenchymal stem cells (MSCs) through acute loss of YAP1. Here we further studied the mechanism of this role of YAP1 in MSCs. We found that acute loss of YAP1 after apicidin treatment resulted in the mixed effects of transcriptional arrest and proteosomal degradation. Subcellular fractionation revealed that YAP1 was primarily localized in the cytoplasm. YAP1 was acutely relocalized into the nucleus and underwent proteosomal degradation. Interestingly, phosphor-S127 YAP1 was shuttled into the nucleus, suggesting that a mechanism other than phosphorylation governed subcellular localization of YAP1. Apicidin successfully induced acetylation and subsequent dissociation of YAP1 from 14-3-3, an essential molecule for cytoplasmic restriction. HDAC6 regulated both acetylation and subcellular localization of YAP1. An acetylation-dead mutant of YAP1 retarded nuclear redistribution upon apicidin treatment. We failed to acquire convincing evidence for polyubiquitination-dependent degradation of YAP1, suggesting that a polyubiquitination-independent regulator determined YAP1 fate. Nuclear PSME4, a subunit of the 26S proteasome, recognized and degraded acetyl YAP1 in the nucleus. MSCs from PSME4-null mice were injected into infarcted heart and aberrant sudden death was observed. Injection of immortalized human MSCs after knocking down PSME4 failed to improve either cardiac function or the fibrotic scar area. Our data suggest that acute ablation of YAP1 in the nucleus by the acetylation-dependent proteasome subunit PSME4 is mandatory for cardiac commitment of MSCs.

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Competing interests: None

Keywords: Mesenchymal stem cell, Regeneration, Myocardial infarction, Cardiac commitment, YAP1, PSME4

S-6-2

Targeting smooth muscle cell phenotypic switching in vascular disease

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Vascular smooth muscle cell (VSMC) phenotypic switching and subsequent VSMC proliferation and migration are major events that are closely associated with progression of cardiovascular diseases. Mitochondria play key roles in regulation of cell function. Recent findings have revealed that mitochondrial fission promotes proliferation and migration of VSMCs. Although a large number of studies have reported the therapeutic effects of natural compounds on vascular-related diseases, recently, emerging data on the therapeutic potential of biocompounds, such as ginsenosides, isolated from ginseng have been reported. Today, I will introduce the key molecular mechanisms and signaling pathways of ginsenosides targeting VSMC phenotypic switching in the prevention and treatment of vascular-related diseases. Therefore, I will try to systematically describe the role of ginsenosides on the vascular dysfunction, which could provide a basis for the clinical application of ginsenosides in the future.

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Competing interests: None

Keywords: Angiotensin II, KLF4, Ginsenosides, Mitochondrial fission, Reactive oxygen species, Vascular Smooth muscle cell

S-6-3

Study of non-coding RNAs in diverse disease models

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There are diverse types of non-coding RNAs (ncRNAs) in human cells, including microRNA (miRNA), long non-coding RNA (lncRNA), and circular RNA (circRNA). They act as important regulators and exhibit a variety of biological functions. We are studying the roles of these ncRNAs in diverse models. These models include the ncRNAs of cardiovascular diseases and neuronal diseases. Among the cardiovascular disease models, we are focusing on the differentiation model of vascular smooth muscle cells and vascular calcification. We identified lncRNAs that are involved in the differentiation of vascular smooth muscle cells, and circRNAs that regulate calcium metabolism in the cells. We also identified candidates lncRNAs and circRNAs differentially expressed in human heart tissues. In this presentation, I will present our recent results about the role of these ncRNAs which will help us understand the roles of ncRNAs in biological processes and find a novel therapeutic strategy for preventing or treating diverse diseases.

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Keywords: Non-coding RNAs, Long non-coding RNAs, Circular RNA, Cardiovascular disease, Vascular smooth muscle cells

S-6-4

Translational and clinical research of diabetic cardiomyopathy

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Diabetes mellitus (DM) is on the rise and has quickly become one of the most common and expensive chronic diseases in the world. DM and cardiovascular disease (CVD), the leading cause of morbidity and mortality in diabetic patients, have a close relationship. Diabetic cardiomyopathy (DCM) is a phenomenon comprising the deterioration in cardiac function and structure independent of coronary artery disease and other causes. We performed the preclinical, translational, and clinical research for investigating the mechanism and therapeutic strategy of diabetic cardiomyopathy.
First, we investigated the cardioprotective effects of pharmacological acti-

vation of angiotensin converting enzyme 2 (ACE2) activator in DCM model by using db/db mice and human embryonic stem cell-derived cardiomyocytes. Second, we investigated the impact of DM on long-term clinical outcomes in patients with moderate diastolic dysfunction and preserved ejection fraction, which is the phenotype of DCM. Finally, for early detection of DCM, we measured the level of several biomarkers and collected clinical and echocardiographic data and analyzed the correlation of these data.

Keywords: Diabetic cardiomyopathy, Angiotensin converting enzyme 2

S-7-1

Myristoylation-dependent palmitoylation of cyclin Y modulates synaptic protein trafficking, LTP, and spatial learning

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Lipid modifications, including palmitoylation and myristoylation, play crucial roles in the subcellular localization and trafficking of proteins. Cyclin Y (CCNY), enriched in the postsynaptic compartment, acts as an inhibitory modulator of functional and structural long-term potentiation (LTP) in the hippocampal neurons. However, cellular and molecular mechanisms underlying CCNY-mediated inhibitory functions in the synapse remain largely unknown. Here, we report that myristoylation located CCNY to the *trans*-Golgi network (TGN), and subsequent palmitoylation directed the myristoylated CCNY from the TGN to the synaptic cell surface. This myristoylation-dependent palmitoylation of CCNY was required for the inhibitory role of CCNY in excitatory synaptic transmission, activity-induced dynamics of AMPA receptors and PSD-95, LTP, and spatial learning. Furthermore, spatial learning significantly reduced palmitoyl- and myristoyl-CCNY levels, indicating that spatial learning lowers the synaptic abundance of CCNY. Our findings provide mechanistic insight into how CCNY is clustered adjacent to postsynaptic sites where it could play its inhibitory roles in synaptic plasticity and spatial learning.

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Competing interests: The author declares no competing interests.

Keywords: Palmitoylation, Myristoylation, Cyclin Y, Long-term potentiation, Spatial learning

S-7-2

Synaptic cell adhesion-like molecule Sy regulates excitatory synaptic density and activity-dependent gene expression

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Sy is a ubiquitously expressed type I transmembrane protein with moderate sequence homology to CD99, and has been shown to play a role in leukocyte infiltration and extravasation through endothelial cells. Although Sy is highly expressed in brain including cerebral cortex, hippocampus, and cerebrum, functional role of Sy in brain remains elusive. Using biochemical approaches combined with confocal imaging technology, we find that Sy is preferentially localized at the excitatory pre- and post- synapses rather than at the inhibitory synapses. We observe a marked increase in excitatory synaptic density by overexpression of Sy in cultured hippocampal neurons, whereas a marked decrease by Sy knockdown or in Sy knockout neurons. By RNA sequencing analysis in Sy knockout brains, we identify increased expression profiles of immediate-early genes (IEGs), such as Arc, Egr1~3, and c-Fos, which are critical for learning and memory-related molecular processes. Furthermore, we find that Sy regulates ERK, CREB, and SRF downstream signaling pathways. Taken together, we provide a model that Sy functions as a synaptic adhesion molecule in neurons by regulating synaptic density and activity-dependent gene expression.

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Competing interests: There are no competing interests to declare.

Keywords: Sy, Immediate early genes, Synaptic adhesion molecule

S-7-3

Investigating physiological and pathophysiological features of neuronal mitochondria using advanced imaging and analysis tools

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Neurons have highly unique structures with distinct morphologies; multiple dendrites and a single axon stem from a soma to transmit information to other cells by generating action potentials. Previous reports unveiled that mitochondria also display strikingly different shapes in dendrites and axons of hippocampal and cortical pyramidal neurons. Dendritic mitochondria are long and tubular, but axonal mitochondria have small and punctate shapes. Mitochondrial morphology is dynamically changed upon environmental stress and neurodegenerative contexts, however the importance of maintaining their shapes in physiological condition has not been studied well.

Neuronal mitochondria play essential roles for various function including synaptic vesicle mobilization and synaptic plasticity by generating ATP and regulating Ca^{2+}. These features have been investigated with recent advances on techniques to probe and interfere with organelle function with high spatial and temporal accuracy. Here, I would like to introduce my key findings based on innovative live Ca^{2+} and synapse imaging related to neuronal mitochondria including: (1) the importance of presynaptic mitochondrial size for Ca^{2+} regulation and axon development, and (2) new functions for ER-mitochondria coupling in the regulation of postsynaptic Ca^{2+} dynamics. In addition, I will show a deep learning-assisted program to expedite the analysis of dendritic and axonal mitochondrial morphology, which is recently developed by tight collaboration. These tools will bring another layer of fundamental neuronal properties based on intracellular organelles and related neurodegenerative diseases.

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Keywords: Neuron, Mitochondria, Calcium imaging, Deep learning, Analysis software

S-7-4

Modulating and monitoring the functionality of corticostriatal circuits using an electrostimulable microfluidic device

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The central nervous system is organized into different neural circuits, each with particular functions and properties. Studying neural circuits is essential to understanding brain function and neuronal diseases. Microfluidic systems are widely used for reconstructing and studying neural circuits but

still need improvement to allow modulation and monitoring of the physiological properties of circuits particularly. In this study, we have built and improved a microfluidic device that electrical modulation of neural circuits with proper support of reassembly. We demonstrated that our microfluidic device provides a system for electrical modulating and monitoring the physiological function of these circuits combined with genetic indicators of synaptic functionality in corticostriatal (CStr) circuits. In particular, our microfluidic device measures activity-driven Ca^{2+} dynamics using Ca^{2+} indicators (synaptophysin-GCaMP6f and Fluo5F-AM), as well as activity-driven synaptic transmission and retrieval using vGlut-pHluorin. Overall, our findings indicate that the improved microfluidic platform described here is an invaluable tool for studying the physiological properties of specific neural circuits.

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Competing interests: There are no competing interests to declare.

Keywords: Microfluidic device, Corticostriatal (CStr) circuit, Synapse, Ca^{2+} dynamics, Action Potential, Synaptic transmission

S-8-1

Mechanoregulation of Endothelial Mitochondrial Phenotype

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Recent studies have greatly advanced our understanding of the central role of mitochondria on endothelial function. We propose a hypothesis that unidirectional laminar (pulsatile) flow and disturbed laminar (oscillatory) flow may differentially modulate mitochondrial phenotypes in the context of their bioenergetic, signaling, and biosynthetic functions, providing novel insights into subcellular mechanisms underlying how exercise benefits the improvement of vascular health.

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Competing interests: None

Keywords: Exercise, Fluid shear stress, Endothelial cell, Mitochondria

S-8-2

Exercise-induced muscle injury, muscle stem cell senescence, and novel therapeutic options

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My talk will describe our most current findings on muscle stem cell senescence during muscle regeneration. Our daily mobility and physical activities are entirely dependent on skeletal muscle. Excessive exercise, paradoxically, may raise the risk of muscular damage. Skeletal muscles' capacity to recover themselves after being damaged via physical exercise and training is widely documented. One of the strategies involved in muscle regeneration is the activation and reproduction of dormant resident muscle stem cells, also known as "satellite cells." Muscle aging is known to be hastened by a decline in the quantity and capacity of muscle stem cells to regenerate. Here, we show that direct muscular injury may lead muscle stem cells to enter senescence, but not cancer cachexia, which can indirectly cause muscle atrophy. My presentation will cover molecular data from in vivo and in vitro experiments confirming the cellular senescence of muscle stem cells in young muscle.

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Competing interests: DR declares that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

Keywords: Muscle aging, Muscle stem cell senescence, Senolytics, Muscle regeneration, Exercise injury

S-8-3

Exercise type and exercise intensity on circulating myokines

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Myokines are exercise-induced hormones regulating various cardiometabolic disorders and related metabolism. Although types and intensity of exercise are implicated as potentially important regulators of various myokines after exercise, the proper stimuli (acute vs. chronic, treadmill vs. swimming, low, moderate vs. high intensity etc) for increasing circulating myokines levels in both experimental animal models and humans remain unclear. In this session, we will explore the effects of exercise type and exercise intensity on circulating myokines including irisin, FSTL-1, and FGF21 etc. which are known as exercise-driven myokines.

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Keywords: Irisin, FSTL-1, FGF21, CathepsinB, Browning

S-8-4

Apelin-AMPK axis in mediating maternal exercise effects on offspring non-shivering thermogenesis

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The obesity rate is rapidly increasing worldwide, which has been attributed to lack of exercise and excessive energy intake, thereby leading to predisposition of their children to obesity and associated metabolic dysfunction. Brown adipose tissue (BAT) and skeletal muscle are metabolically important organs, utilizing excessive energy for generating heat throughout mitochondrial non-shivering thermogenesis (NST). BAT, burning fatty acids and glucose for NST, extensively consumes energy via the UCP1-dependent manner. In the skeletal muscle NST, sarcoendoplasmic reticulum Ca^{2+} ATPase (SERCA) pumps Ca^{2+} ions back into the sarcoplasmic reticulum (SR). Then, sarcolipin (SLN) uncouples Ca^{2+} pumping from ATP hydrolysis by SERCA, triggering futile energy consumption and muscle-based NST, improving muscular and whole-body metabolic homeostasis. Maternal exercise (ME) enhances UCP1-dependent and SLN-dependent NST in BAT and muscle respectively, which protects from maternal obesity (MO)-induced metabolic dysfunction. Furthermore, apelin administration during pregnancy mimicked the benefits of ME on AMPK and respective NST signaling pathways, leading to fetal metabolic development. Together, our finding shows that physically active pregnancy enhances NST and metabolic health of offspring mice, suggesting that the sedentary lifestyle during pregnancy contributes to the obesity epidemic in modern societies.

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land School of Medicine.

Competing interests: The author declares no competing interests.

Keywords: Maternal obesity, Mitochondria, Physical activity, Sarcolipin; UCP1

S-9-1

Update on Alzheimer's disease therapeutics

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In this brief update, I will highlight recent updates of drug trials in Alzheimer's disease (AD). It will mainly include the results of clinical trials targeting beta-amyloid and tau. Potential causes that may have contributed the failure of clinical trials and efforts to troubleshoot the causes will also be discussed. Efforts to detect subjects who have pathology in the brain but not yet have clinical symptoms of AD and treatment trials in those subjects will move the field forward to ultimately understand the pathophysiology and to prevent or halt the pathologic progression of the disease.

Competing interests: None

Keywords: Alzheimer's disease (AD), Therapeutics, Prevention

S-9-2

The activation of lysosomes decreases the tumor growth of colon cancer cells in vivo

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Lysosome is the major machinery of RTK proteolysis. Some of endocytosed RTKs return to the cell membrane after the specific ligand stimulation and signal transduction by recycling endosomes. The other endocytosed RTKs are being digested via lysosomal proteolysis after signaling processes. The ratio of recycling and lysosomal proteolysis depends on the specific receptor. Among RTKs, EGFR is majorly being degraded in lysosomes after the ligand stimulation rather than undergoing to the recycling process. However, this proteolysis is downregulated in hypoxic condition such as the central core of solid tumors. The downregulation of EGFR degradation is because of the suppressed lysosomal activity in hypoxic conditions by the suppress of the nuclear translocation of TFEB.

Here we demonstrate that, in cultured mammalian cells and mouse models, the enhancement of lysosomal activity leads to the degradation of EGFR in colon cancer cells. We overexpressed V-ATPase components in DLD-1 cells that increased the proteolysis of EGFR in lysosomes. The cells were also xeno-transplanted to immune-suppressed nude mice to induce tumors. When the lysosomal activity was enhanced in DLD-1 colon cancer cells, the growth was significantly decreased. Furthermore, when the mice were treated with a tyrosine kinase inhibitor, Osimertinib, the decrease of tumor growth was much more decreased. From this study, we suggest lysosomal activation can be another therapeutic approach for EGFR-mediated cancers including colon cancers. Furthermore, this can be a new combination therapy together with the previous antibody therapies and TKI therapies.

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Competing interests: The author declares not having any conflicts of interest.

Keywords: Lysosome, EGFR, TFEB, RTK

S-9-3

A growth-factor-activated lysosomal K^+ channel regulates Parkinson's pathology

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Lysosomes have fundamental physiological roles and have previously been implicated in Parkinson's disease. However, how extracellular growth factors communicate with intracellular organelles to control lysosomal function is not well understood. Here we report a lysosomal K+ channel complex that is activated by growth factors and gated by protein kinase B (AKT) that we term lysoKGF. LysoKGF consists of a pore-forming protein TMEM175 and AKT: TMEM175 is opened by conformational changes in, but not the catalytic activity of, AKT. The minor allele at rs34311866, a common variant in TMEM175, is associated with an increased risk of developing Parkinson's disease and reduces channel currents. Reduction in lysoKGF function predisposes neurons to stress-induced damage and accelerates the accumulation of pathological α-synuclein. By contrast, the minor allele at rs3488217—another common variant of TMEM175, which is associated with a decreased risk of developing Parkinson's disease—produces a gain-of-function in lysoKGF during cell starvation, and enables neuronal resistance to damage. Deficiency in TMEM175 leads to a loss of dopaminergic neurons and impairment in motor function in mice, and a TMEM175 loss-of-function variant is nominally associated with accelerated rates of cognitive and motor decline in humans with Parkinson's disease. Together, our studies uncover a pathway by which extracellular growth factors regulate intracellular organelle function, and establish a targetable mechanism by which common variants of TMEM175 confer risk for Parkinson's disease.

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Competing interests: The authors declare no competing interests

Keywords: Eletrophysiology, Lysosome, Neurodegeneration

S-9-4

N-terminally truncated hERG channels generated by KCNH2 frameshift mutation (c.453delC) induces LQT phenotype in patient-derived iPSC-CMs

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Patient-specific cardiomyocytes from human induced pluripotent stem

cells (hiPSC-CMs) are valuable for studies in the inherited cardiac diseases. A recent study reported single nucleotide C deletion mutation in the exon 3 of KCNH2 gene (c.453delC-KCNH2, p.151Pfs +15X in hERG) associated with LQT syndrome (Park JK et al., 2013). Since the 453delC-KCNH2 resulted the frameshift of the coding sequences, a premature termination of translation at the N-terminal region was suggested. However, there is an additional initiation codon next to the mutated residue. To elucidate the precise mechanism of LQT phenotype, we performed whole-cell patch clamp and immunoblot assay in 453delC-KCNH2 hiPSC-CMs and HEK293 cells transfected with 453delC-KCNH2. The 453delC-KCNH2 hiPSC-CMs showed significantly prolonged action potential duration (APD) and reduced density of the rapidly activating delayed rectifier K+ current (IKr). The density of IhERG in HEK293 cells transfected with 453delC-KCNH2 was 10 % of the wild type (WT) IhERG. However, voltage dependence of activation, voltage dependence of inactivation, and deactivation kinetics of 453delC-KCNH2 were not significantly different from those of WT. To study the interaction between WT and mutant, the equimolar amounts of WT and 453delC cDNA were transfected into HEK293 cells. The current density of WT/453delC channels was half of that from the WT channel alone, indicating insignificant dominant negative effect. Immunoblot analysis of WT channel showed 150 kDa of core-glycosylated form and 180 kDa of fully-glycosylated channel. Interestingly, 453delC-KCNH2 overexpressed cells showed 135 kDa and 160kDa suggesting that the translation of shorter form, i.e. N-terminal truncated hERG, actually occurred with subsequent glycosylation. Nevertheless, the markedly reduced IhERG and the prolonged APD indicated functionally impaired state of 453delC-KCNH2, consistent with the LQT2 phenotype.

Keywords: Human induced pluripotent stem cells-cardiomyocyte, Long QT syndrome type 2, KCNH2 mutation

S-9-5

A study for red blood cell as physiological marker

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Red blood cell (RBC) is a fundamental cell and blood tests are performed routinely in a clinic setting. However, the study for RBC physiological markers is limited. We aim to discover a clinically meaningful maker for RBC via biomedical engineering approach.

First, for evaluating the effect of anti-diabetic medication, RBC physiological index was investigated. Sodium-glucose cotransporter 2 (SGLT-2) inhibitor is an anti-diabetic drug that improves cardiovascular outcomes. Generally, hematocrit level increases after SGLT-2 inhibitor administration due to the diuretic effect of medication. Although elevated hematocrit increases blood viscosity and risk of cardiovascular disease, SGLT-2 inhibitor has protective effects on the cardiovascular system. A mechanism for this paradoxical phenomenon is unclear. Therefore, hemorheological parameters of diabetic patients were evaluated. As a result, RBC deformability was improved in the SGLT-2 inhibitor group compared with that in the control drug group. This improvement is supposed to have a protective effect on the cardiovascular system.

Second, RBC shape was evaluated as a morphological marker for response to change of solution osmolality. Phosphate-buffered saline (PBS) and Alsever's solution (AS) are frequently used as media in blood-related experiments. Despite frequent use, the effect of these solutions on RBC shape has not been studied. We collected blood samples from 5 healthy adults. RBC shape change was evaluated using three-dimensional refractive index tomography. As a result, sodium chloride alone cannot elicit the biconcave shape of RBC. The biconcave shape could be maintained with the presence of an osmotic pressure-maintaining substance, such as glucose or mannitol.

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Competing interests: No declare.

Keywords: Hemorheology, Blood, Red blood cell, Holotomography

S-10-1

Modeling G2019S-LRRK2 Sporadic Parkinson's Disease in 3D Midbrain Organoids

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Recent advances in generating three-dimensional (3D) organoid systems from stem cells offer new possibilities for disease modeling and drug screening because organoids can recapitulate aspects of in vivo architecture and physiology. In this study, we generate isogenic 3D midbrain organoids with or without a Parkinson's disease-associated LRRK2 G2019S mutation to study the pathogenic mechanisms associated with LRRK2 mutation. We demonstrate that these organoids can recapitulate the 3D pathological hallmarks observed in patients with LRRK2-associated sporadic Parkinson's disease. Importantly, analysis of the protein-protein interaction network in mutant organoids revealed that TXNIP, a thiol-oxidoreductase, is functionally important in the development of LRRK2-associated Parkinson's disease in a 3D environment. These results provide proof of principle for the utility of 3D organoid-based modeling of sporadic Parkinson's disease in advancing therapeutic discovery.

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Keywords: Parkinson's disease, Disease modeling, iPSC, Midbrain, Organoids

S-10-2

Organoid model-based safety test

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Safety/toxicity research is an important area in the drug development process, and drug screening is an essential part of this endeavor. Stem cells are the optimal tools to simulate the human system, and are continuing various developments from 2D culture to 3D organoids. The latest organoids go beyond simple phenotypic differentiation, and feature the maturation of functional mimicry due to spontaneous structural formation, which is drawing attention in the field of research. For the development of functional organoids, this study aims to develop a multi-organoid model in which the heart, blood vessels, and brain are organically connected to each other to maximize their functions. Compared with the existing single organoids, functionally mature multi-organoids exhibit structurally similar characteristics to human organs through actual blood vessel distribution. These functional organoids will have significant implications in future drug development.

Acknowledgement: National Institute of Food and Drug Safety Evaluation

Competing interests: N/A

Keywords: Organoid, Safety test, Heart, Brain, Skin

S-10-3

Generation of human tonsil epithelial organoids as an *ex vivo* model for SARS-CoV-2 infection

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The palatine tonsils (hereinafter referred to as "tonsils") serve as a reservoir for viral infections and play roles in the immune system's first line of defense. The aims of this study were to establish tonsil epithelial cell–derived organ-

oids and examine their feasibility as an ex vivo model for severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection. The tonsil organoids successfully recapitulated the key characteristics of the tonsil epithelium, including cellular composition, histologic properties, and biomarker distribution. Notably, the basal layer cells of the organoids express molecules essential for SARS-CoV-2 entry, such as angiotensin-converting enzyme 2 (ACE2), transmembrane serine protease 2 (TMPRSS2) and furin, being susceptible to the viral infection. Changes in the gene expression profile in tonsil organoids revealed that 395 genes associated with oncostatin M signaling and lipid metabolism were highly upregulated within 72 h after SARS-CoV-2 infection. Notably, remdesivir suppressed the viral RNA copy number in organoid culture supernatants and intracellular viral protein levels in a dose-dependent manner. Here, we suggest that tonsil epithelial organoids could provide a preclinical and translational research platform for investigating SARS-CoV-2 infectivity and transmissibility or for evaluating antiviral candidates.

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Keywords: Organoid, Immuno-oncology drug, Drug screening

S-10-4

Human pluripotent stem cell-derived intestinal organoids and their applications

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The small intestine (SI) is a complex organ with multiple histological and functional structures that promote efficient nutritional absorption, control intestinal microorganisms, and provide protection and defense against pathogens and toxins. The SI has a variety of cell types that can perform multifunctional roles, and in particular, the intestinal epithelium contains four major differentiated cell types, including absorptive enterocytes, goblet cells, secretory enteroendocrine cells, and Paneth cells. Recently, an alternative approach has been demonstrated to successfully generate an *in vitro* model mimicking epithelial dynamics and bioactivity in human intestines. Several studies have shown that a stepwise differentiation process can efficiently produce human intestinal organoids (hIOs), a three-dimensional (3D) structure of epithelial cells derived from human pluripotent stem cells (hPSCs). This experimental model system is also an important tool for studying the differentiation and maintenance of intestinal epithelial cells as well as pathophysiological processes underlying intestinal diseases. Recently, we described an *in vitro* maturation technique for generating adult-like, mature hIOs from hPSCs that closely resemble the *in vivo* tissue structure and cellular diversity. Here, we will discuss the impact of maturation status of hIOs in studying host epithelial–microbiota interactions and in applying regenerative medicine.

Acknowledgement: This work was supported by the Korea Bio Grand Challenge Project (NRF-2018M3A9H3023077, 2021M3A9H3016046) through the National Research Foundation of Korea (NRF) funded by the Ministry of Science, ICT, and Future Planning, the Korean Fund for Regenerative Medicine (KFRM) grant funded by the Korea government (the Ministry of Science and ICT, the Ministry of Health & Welfare, 21A0404L1), and the Technology Innovation Program (No. 20008777) funded by the Ministry of Trade, Industry & Energy (MOTIE, Korea)).

Keywords: Intestinal organoid, Small intestine, Human pluripotent stem cells, Host–microbiota interaction, Regenerative medicine

S-10-5

3D Bioprinting and its Applications

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3D printing technology can create a three-dimensional object using 3D CAD model by printing successive layers of various materials under computer control. The 3D printing technology can be applied to the biomedical field to produce tissue engineered constructs imitating complex organ or tissue structure. 3D bio-printing technology makes it possible to not only fabricate 3D shape, but also precisely place various cell in a single structure. Here, we developed a 3D bioprinting system which can fabricate the 3D polymer scaffold. It serves as a 3D supporting structure for cell adhesion and proliferation. The system has data conversion software which can convert 3D CT/MRI files into the NC code. And the system composes of multi-dispensing modules, high precision multi-axis stages and controller. It can control the shape, size, pore size and porosity of the scaffold. We also developed a cell printing system which can print various types of hydrogels to fabricate 3D hydrogel scaffold. The greatest advantage of the cell printing system is that it can fabricate the cell-laden hydrogel scaffolds. Homogenous encapsulation of the cells can be done using the cell printing system. Both systems can fabricate 3D scaffolds with interconnected porous structures to increase the efficiency of the transport of the nutrient and waste. The mechanical properties of the scaffold, proliferation and differentiation of the cells are compared with different biocompatible polymers and hydrogels.

Acknowledgement: This work was supported by the KRIBB Research Initiative Program.

Competing interests: The authors declare that they have no competing interests.

Keywords: 3D bioprinting, Tissue engineering, Artificial organ, Cell printing

S-11-1

Escherichia coli mimetic gold nanorod-mediated photo- and immunotherapy for treating cancer and its metastasis

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Most cancer-related deaths are due to metastasis or recurrence. Therefore, the ultimate goal of cancer therapy will be to treat metastatic and recurrent cancers. Combination therapy for cancer will be one of trial for effective treating metastasis and recurrence. In this study, Escherichia coli-mimetic nanomaterials are synthesized using Escherichia coli membrane proteins, adhesion proteins, and gold nanorods, which are named E. coli mimetic AuNR (ECA), for combination therapy against cancer and its metastasis. ECA treatment with 808 nm laser irradiation eliminates CT-26 or 4T1 tumors in mice via photothermal effect. ECA with laser irradiation induces activation of dendritic cells and T cells in the tumor-draining lymph nodes. The mice cured from CT-26 or 4T1 tumor by ECA are re-challenged with those cancer in the lung metastatic form, and the results showed that ECA treatment for the 1st CT-26 or 4T1 tumor challenge prevents cancer infiltration to the lung in the 2nd challenge. This preventive effect of ECA against tumor growth in the 2nd challenge is mediated by cancer antigen-specific T cell immunity. Overall, these findings show that ECA is a nanomaterial with dual functions as photothermal therapy for treating primary cancers and as immunotherapy for preventing recurrence and metastasis.

Acknowledgement: This study was supported by the National Research Foundation of Korea (NRF-2019R1C1C1003334 and NRF-2020R1A6A1A03044512).

Keywords: Photothermal therapy, Immunotherapy, Anti-metastasis, Gold nanorod, FimH

S-11-2

Oral microbiota-epithelium crosstalk regulates local and distal carcinogenesis

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Bacteria and fungi, two major components of the microbiota, develop both antagonistic and symbiotic relationships on the host epithelium. However, the crosstalk between epithelium and microbiota on tumorigenesis is poorly understood. Oral mucosa is a well-known habitat for various microorganisms and cancer patients frequently present oral fungal infection. Thus, we investigated whether oral fungal infection can regulate local and distal tumorigenesis, particularly in the context of interaction between oral microbiota and epithelium. IKKα is one of the crucial factors regulating the homeostasis of squamous epithelial tissues. To investigate the crosstalk between epithelium and microbiota, we adopted IKKα conditional knockout mice in epithelial cells of oral mucosa and skin. Then, the mice were orally inoculated with Cladosporium cladosporioidesfungi. Disruption of epithelial homeostasis by IKKα ablation promoted bacterial colonization in oral cavity and skin, oral dysplasia, and skin carcinogenesis in mice. Interestingly, it was further accelerated by oral inoculation with Cladosporium cladosporioides. Moreover, this oral fungal infection induced the fungal-bacterial symbiosis in the oral cavity with broken epithelial homeostasis. However, the mice treated with antibiotics showed reduced incidences in oral dysplasia and skin tumors, implying the involvement of oral fungal-bacterial symbiosis. Disrupted epithelial homeostasis by IKKα loss allows the bacterial-fungal symbiosis in the oral mucosa, which expedites local and distal tumorigenesis. Further investigation is required to address the underlying molecular mechanism.

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Competing interests: None.

Keywords: Oral microbiota, Epithelial homeostasis, Fungal-bacterial symbiosis, IKKα, Skin carcinogenesis

S-11-3

The Emerging role of autophagy-related pathway in immune-driven malignant evolution of tumor cells

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Immune selection drives tumor cells to acquire refractory phenotypes. We previously demonstrated that cytotoxic T lymphocyte (CTL)-mediated immune pressure enriches NANOG+ tumor cells with stem-like and immune-refractory properties that make them resistant to CTLs. Here, we report that the emergence of refractory phenotypes is highly associated with an aberrant macroautophagic/autophagic state of the $NANOG^+$ tumor cells and that the autophagic phenotype arises through transcriptional induction of *MAP1LC3B/LC3B* by NANOG. Furthermore, we found that upregulation of LC3B expression contributes to an increase in EGF secretion. The subsequent hyperactivation of EGFR-AKT signaling rendered $NANOG^+$ tumor cells resistant to CTL killing. The NANOG-LC3B-p-EGFR axis was preserved across various types of human cancer and correlated negatively with the overall survival of cervical cancer patients. Inhibition of LC3B in immune-refractory tumor models rendered tumors susceptible to adoptive T-cell transfer, as well as PDCD1/PD-1 blockade, and led to successful, long-term control of the disease. Thus, our findings demonstrate a novel link among immune-resistance, stem-like phenotypes, and LC3B-mediated autophagic secretion in immune-refractory tumor cells, and implicate the LC3B-p-EGFR axis as a central molecular target for controlling $NANOG^+$ immune-refractory cancer.

S-11-4

T cell's self-recognition: shaping diversity beyond specificity

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Peripheral naïve CD8 T cells under steady state require some degree of self-reactivity for their survival, through their T cell receptor (TCR) interaction with self-ligands. Such TCR self-reactivity is diverse in its strength among each individual naïve T cell and influences their immune responses. For the latter, we have recently demonstrated that relatively high intrinsic self-reactive CD8 T cell populations are phenotypically heterogenous with enhanced gene signatures characteristic of cell proliferation and effector differentiation, and accordingly have augmented cytokine-producing ability and greater anti-viral immune response compared to those of low intrinsic self-reactive populations. Here we now demonstrate total reverse of the afore-mentioned phenomena could occur in non-infectious, different immune contexts presumably where cognate antigen engagements would proceed before fullminent innate immune responses reach a peak. Further details in the underlying mechanism and its physiological insight will be discussed.

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Competing interests: There is no competing interests to declare.

Keywords: Naive $CD8^+$ T cell, T cell receptor, Self-reactivity, Heterogeneity, Viral infection, Inflammation

S-11-5

DDS using Salmonella for treatment of cancer

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Bacterial cancer therapy relies on the fact that several bacterial species are capable of targeting tumor tissue and that bacteria can be genetically engineered to selectively deliver therapeutic proteins of interest to the targeted tumors. However, the challenge of bacterial cancer therapy is the release of the therapeutic proteins from the bacteria and entry of the proteins into tumor cells. This study employed an attenuated *Salmonella typhimurium* to selectively deliver the mitochondrial targeting domain of Noxa (MTD) as a potential therapeutic cargo protein, and examined its anti-cancer effect. To release MTD from the bacteria, a novel bacterial lysis system of phage origin was deployed. To facilitate the entry of MTD into the tumor cells, the MTD was fused to DS4.3, a novel cell-penetrating peptide (CPP) derived from a voltage-gated potassium channel ($K_v2.1$). The gene encoding *DS4.3-MTD* and the phage lysis genes were placed under the control of P_{BAD}, a promoter activated by L-arabinose. We demonstrated that DS4.3-MTD chimeric molecules expressed by the *Salmonellae* were anti-tumoral in cultured tumor cells and in mice with CT26 colon carcinoma.

Keywords: Salmonella, Anticancer protein, Synthetic biology

S-12-1

Physiological and psychological assessments for the Establishment of evidence-based forest healing programs

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This study aimed to establish a health and medical foundation for forest healing programs and provide a basis for developing an evaluation system for such programs. While the number of visitors to forests and interest in forest healing effects are increasing, few studies have examined the various indicators of the persistent changes in forest healing effects. Therefore, this study conducted pre-, post-, and follow-up experiments on 87 health and clinical indicators in a sample of 88 adolescent participants. The relationships between pre-, post-, and follow-up experiment results for each indicator were analyzed. Of the 87 indicators, 46 showed significant changes, including systolic blood pressure, diastolic blood pressure, cholesterol, serotonin, vitamin D, CD16+CD56 count, interferon-γ, resilience, and self-esteem. The findings are significant for studying diverse participants and indicators and lay the foundation for developing forest healing programs by clarifying aspects such as the indicators suitable for short-term observation versus the indicators requiring long-term observation. Based on these analyses, the results of this study are expected to be useful when conducting research to establish an evidence-based forest healing program in the future.

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Keywords: Forest healing, Forest healing program, Forest therapy, Physiological effect, Psychological effect, Follow-up survey, Long-term observation

S-12-2

Splitting up exercise training in morning and afternoon for 14 days in a hot environment: consideration of total body fat and physical fitness

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Conscripted soldiers in Korea are confronted with various heat-related disorders (HRDs) during combat training in summer. To reduce HRDs during heat waves, repetitive short training is preferred to longer training. Korean soldiers regularly march 1-h both in the morning and in the afternoon. We investigated the physiological and perceptual changes from this 2-h daily training, with 14 consecutive days of 1-h exercise in the morning and 1-h exercise afternoon, in a hot and humid environment. Also, the effects of 14-day heat acclimation training on physiological strain was examined through a pre- and post-passive heat stress (HS) test. Eleven males (24 ± 2 y, 172.3 ± 6.1 cm, 72.4 ±18.4 kg) participated in HS test before and after a 14-day heat acclimation program (HA program, 1-h morning and 1-h afternoon exercise at 50%VO_{2max}, 31℃ and 66%RH), while a control group (12 males; 23 ± 2 y, 174.7 ± 2.4 cm, 75.2 ±18.9 kg) participated in the PRE- and POST-HS tests only. The HS test consisted of a 10-min rest followed by 60-min leg immersion in 42℃ water in a climate chamber (30℃, 50%RH). The results showed that on the 14th day, rectal temperature (T_{re}) at the end of exercise was lower (37.7 ± 0.4℃ in the morning and 38.0 ± 0.3℃ in the afternoon) when compared to Tre on the 1st day (38.4 ± 0.5℃ and 38.3 ± 0.4℃) (P<0.05). Heart rate was significantly lower on the 14th day (163 ± 21 bpm and 172 ± 16 bpm in the morning and afternoon, respectively) than HR on the 1st day (144 ± 24 bpm and 145 ± 19 bpm on the 14th day) (P<0.05). However, total sweat rate showed no significant change during the 14-day program, as well as no differences between pre- and post HS tests. Subjects felt less warm and less uncomfortable as days passed, but the improvement was significant in the morning only. Interestingly, responses showing heat acclimation were more significant for subjects who had greater maximal oxygen consumption with less total body fat (%). In summary, T_{re} and HR were effectively reduced through the 14-day heat acclimation program of 1-h morning and 1-h afternoon exercise along with improving thermal perceptions, and body morphology and physical fitness were related to achieve heat acclimation.

Acknowledgement: This work was supported by grants from the Armed Forces Medical Research Institute (AFMRI).

Keywords: Heat acclimation, Rectal temperature, Cardiovascular burden, Heat strain

S-12-3

The role of occupational and environmental medicine in the subsea space creation and utilization technology development project

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In the meantime, Korea has been steadily conducting research on the ecosystem and resource exploration of the seabed, but the need for technology development at the national level has been raised due to difficulties in initial investment such as development and utilization. In 2022, for the first time in Korea, research to utilize the subsea space for various purposes began. In this study, submarine space platform technology such as submarine research space, underwater living space, underwater data center, and underwater chamber technology, and the medical field for the health and safety of residents are included. Ultimately, this study aims to demonstrate the technology by installing a modular underwater structure that can actually stay for 30 days at a depth of 30 meters by three people. The first submarine space in Asia will be built off the coast of Ulsan with the participation and support of Ulsan City. Although it is planned that the internal atmospheric pressure of the subsea structure will be maintained at 1 atm, continuous intervention by the medical management system is required, such as air pressure control in the process of entering and exiting the subsea space, maintenance of the structure, and emergency treatment in case of an emergency in the subsea space. In addition, in the subsea dwelling, the device for removing carbon dioxide and carbon monoxide must always be operated, and in preparation for a temporary increase, it is also necessary to monitor the physiological response of the human body for 24 hours. Various methods such as moonpool and docking system are being considered for the technology of moving from the ground to the seabed, and the decompression procedure and human body monitoring method may vary depending on the method. Diving medicine is a field that has been studied a lot at home and abroad, and in particular, a lot of research has been done on the physiological response, treatment, and decompression procedure in high pressure situations. Projects that have carried out research by installing residential structures in the seabed like this study include "Aquarius reef base" of the US, "Helgoland" of Germany's submarine science base, "Progetto Abissi" of Italy, etc. Although there were many research results on the seabed ecosystem in these projects, the results of follow-up observations, biometric data, and health disorders caused by saturation diving were rare. In Korea, there have been no studies on health disorders when living in the seabed for a long time and living in a saturated diving state under high pressure conditions. Through this study, it is expected that health changes can be observed through physiological monitoring of residents and workers exposed to the subsea environment, and human body data can be collected for the prevention of decompression sickness. Through long-term observational studies, it will contribute to the study of the physiological mechanisms of diseases that can occur under high pressure conditions, such as dysbaric osteonecrosis, where the exact mechanism is unknown.

Acknowledgement: This work was supported by grants from the Ministry of Oceans and Fisheries.

Keywords: Decompression sickness, Diving, Health monitoring

S-12-4

Health monitoring through health assessment and bio-signals of habitat in subsea space

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The technology developed during space development is being used a lot in our daily life, whether we know it or not. Shoes with air cushions were developed to protect the bones of astronauts who returned from living in space for a long time, and hydroponics, water purifiers, and freeze-dried food technologies were developed for survival in space. The economic effects of the space technology developed so far have entered our daily lives, saving about 450,000 lives and creating 19,000 jobs.
The technology for constructing a subsea space is the same as the technology for building a space station, and it is a technology for living and working in extreme conditions, requiring cutting-edge technologies from various disciplines. Therefore, the construction of a subsea platform can be derived from a variety of technologies that can be used in daily life in the future as well as the simple development of subsea technology.
This project is the first phase of the project and aims to develop and demonstrate design, construction, operation, and management technology for the creation and utilization of undersea living space for 5 years from 2022 to 2026. The technical goal is to stay 5 people at a depth of 50 m, and the demonstration target is set to stay for 3 people at a depth of 30 m.
In order to live on the subsea space, in addition to air supply, it is necessary to introduce a technology that can identify and prevent disease occurrence in the subsea environment. Health problems can be caused by changes in pressure and an isolated environment due to the specificity of the subsea space. As a means of psychological stability, facilities that can provide psychological stability and communication facilities that can be connected to the outside are essential. In order to check the impact of sea life on the health of residents, it is necessary to build a health status evaluation system and develop a health monitoring system that can classify health status.

Acknowledgement: This work was supported by grants from the Ministry of Oceans and Fisheries

Keywords: Subsea space, Health, Monitoring

S-12-5

The effect of the program to improve adaptation with the change of living environment

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As social distancing continues due to COVID-19, there are growing concerns that parents and children spend more time together at home, while parents and children may suffer from poor quality-satisfactory interactions. In addition, it is necessary to develop a non-face-to-face counseling program to achieve similar effects to the face-to-face method in a situation where face-to-face counseling, which is recognized as a traditional counseling method, is impossible. To this end, this study developed the CPGT program that integrally applied CPRT and structured game play therapy based on the results of the preliminary study. In order to verify the effectiveness of the developed program, 10 non-face-to-face group counseling, 3 non-face-to-face individual counseling, and a total of 13 programs were applied to 11 parents who have school age children, and self-report evaluation of parent-child relationship was measured postmortem. In addition, the video data that parents interacted with their children in a face-to-face manner was observed by dividing into early, mid, and late stages, and the experience of the study participants was categorized and analyzed based on the researcher's research journal and non-face-to-face interview data. This study developed the CPGT program as a way to alleviate the difficulties of parents and children experienced in daily life and promote their overall development and applied the program in a non-face-to-face manner to verify its effectiveness. It is expected that the detailed program of this study, which consists of specific methods to be applied to the interaction scene of parents and children at home by using game play, can be widely used in education and counseling sites.

Keywords: Child development, Game play therapy, CPRT, CPGT, Non-face-to-face method

S-13-1

Emerging roles of Innate lymphoid cells in airway inflammations

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Innate lymphoid cells (ILCs) are a population of tissue-resident innate lymphocytes that are critical mediators of mucosal immunity in many tissues, including the lung and gut. ILCs are particularly abundant at the mucosal barriers, where they are exposed to allergens, commensal microbes, and pathogens. Within mucosal tissue, ILCs are regulated by complex crosstalk between immune cells and signals received from environmental cues to control infection and restore tissue damage. Here, I will discuss how dysregulation of the ILC response can lead to airway inflammations, such as asthma and COPD (Chronic Obstruction Pulmonary Disease) in humans and experimental models. We have found the dysregulation of the ILC function correlated with the severity and phenotype of diseases. Also, changing the tissue microenvironment affect the sensitivity and features of ILCs. Although many mechanisms and details of the interplay between ILCs and their environment have not yet been elucidated, it is clear that ILCs are a critical player in airway immunity.

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Competing interests: The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

Keywords: Innate lymphoid cells, Airway inflammations, Alarmins, Immune crosstalk

S-13-2

Chronic cough and cough hypersensitivity

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Cough is a vital physiological mechanism to protect the lower airways against the inhalation and aspiration of irritants. However, a dysregulated cough reflex is a significant morbidity. Chronic cough is a globally common condition, affecting about 5-10% of adults in general populations. It is now widely recognized that chronic cough of any etiology, at least in adults, represents a clinical state that deviates by far beyond the protective role of cough reflex. Most patients with chronic cough present with coughing in response to relatively innocuous stimuli, such as cold air, perfume, or talking. The recognition of cough hypersensitivity as the key mechanism underlying chronic cough has opened a new window of opportunity in the management of chronic cough. Particularly in patients with refractory or unexplained chronic cough, clinical trials have shown that cough can be effectively controlled by drugs that modulate the cough reflex pathways. However, it is unlikely that there is a single magic bullet for all chronic cough problems because clinical benefits of a drug may vary with clinical context.

Further understanding of the mechanisms that underlie cough hypersensitivity will contribute to the development of new therapies for chronic cough.

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Keywords: Cough, Neurophysiology

S-13-3

The clinical impact of air pollutants on COPD and its underlying pathophysiology

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Particulate matter (PM) is a global public health concern, and it affects entire cycle of our life. PM increases the risk of preterm death and affects lung development adversely in adolescents. After middle age, PM increases the risk of hospitalization due to COPD, morbidity, mortality, and exacerbation. Consequently, PM exposure shortens our lifespan. The improvement of air quality is the best option, but it is not possible without international collaboration. Therefore, individual strategy to avoid PM exposure should be combined with the improvement of air quality. The most current recommendations about lifestyles are based on experts' opinion without definite evidence. To overcome this limitation, we found six effective lifestyles to decrease the PM exposure, which includes air-filter operation, ventilation though windows and check the air quality forecast. Further studies to intervene lifestyles will elucidate the impact of lifestyles more clearly. The various mechanisms are related to its pathophysiology, and pyroptosis can be suggested as one of the major ones.

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Competing interests: None

Keywords: Particulate matter, COPD, Lifestyles, Air filter, Air pollution

S-13-4

Gut-lung axis in adult asthma

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The gut microbiome affects general health conditions and various diseases in humans via complex communication with the immune system. Because of these interactions, the gut microbiome is becoming an important target for precision medicine in various chronic inflammatory diseases. Asthma is a chronic inflammatory disease of the airways, and growing evidence suggests that the interaction between the gut microbiome and immunity plays an important role in the pathophysiology of asthma. However, to date, treatment guides for adult asthma have not provided effective methods for regulating inflammation by gut microbiota. I recently analyzed the gut microbiome alteration in adult symptomatic eosinophilic asthma patients. Specifically, a decrease in Oscillospiraceae and Lachnospiraceae was observed, and a decrease in Lachnospiraceae was correlated with blood eosinophilia and lung function decline. Here, I will discuss the mechanism by which airway inflammation induces gut microbiome alteration and the effect of the gut microbiome alteration on the control of asthma.
We would like to review the potential of gut microbiota as a treatment or control agent for adult asthma in the studies so far, and review personal research directions that can realize the potential.

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Competing interests: The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

Keywords: Asthma, Eosinophil, Inflammation, Gut microbiota

S-14-1

Targeting the stress support pathways in senescence for healthy aging

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Aging is the most important single risk factor for many chronic diseases, including neurodegenerative disorders, metabolic syndrome, and cancer. Targeting the fundamental process of aging might, therefore, be a better strategy for enhancing human health than targeting each chronic disease individually. Although much should be achieved for completely understanding the biological basis of aging, cellular senescence is considered a crucial contributor to organismal aging via two independent but not mutually exclusive mechanisms: (1) stem cells exhaustion and (2) senescence-associated secretory phenotype (SASP) that causes chronic inflammation and tissue dysfunction. Much effort has been recently made to therapeutically target detrimental effects of senescence, including selectively eliminating senescent cells (senolytics) and modulating a proinflammatory senescent secretome (senostatics or senomorphics). Here, I discuss the current progress and limitations in developing and applying senolytics and senostatics/senomorphics and how to improve these crucial strategies for healthy aging.

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Competing interests: I declare no competing interests.

Keywords: Aging, Cellular senescence, Senescence-associated secretory phenotype (SASP), Senolytics, Senostatics, Senomorphics

S-14-2

Exploring the molecular mechanisms to connect metabolism, DNA damage response, and Aging

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Aging is considered a process to decline organismal function, which results in the collapse of body homeostasis. It increases the susceptibility to age-related diseases including neurodegenerative diseases and cardiovascular diseases. One of the attractions to study the mechanism of aging is to make aging slower. One strategy to appeal is calorie restriction, which is thought to regulate life span based on scientific evidence. However, the detailed mechanism is not clear. Calorie restriction activates Sirt1 and induces autophagy. Autophagy is a self-eating system that degrades intracellular compartments in cells. It degrades protein aggregates and damaged organelles as a defense mechanism when cells are stressed such as starvation, hypoxia, and DNA damage. We showed that Sirt1, aanNAD+-dependent deacetylase interacts with autophagy-related proteins (Atgs) and regulates the acetylation status of several essential Atg proteins such as Atg5,7,8 and 12 in vivo as well as in vitro. In other words, Sirt1 is a positive regulator of autophagy. Both Sirt1 and autophagy are important in preventing aging and age-related diseases such as cancer, neurodegeneration diseases, diabetes,

and so on.
Growing evidence indicates that metabolic signaling pathways are interconnected to DNA damage response (DDR). However, factors that link metabolism to DDR remain incompletely understood. SIRT1, an NAD+-dependent deacetylase that regulates metabolism as well as aging, has been shown to protect cells from DDR. Here we demonstrate that SIRT1 protects cells from oxidative stress-dependent DDR by binding and deacetylating Checkpoint Kinase 2 (CHK2). We first showed that essential proteins in DDR were hyper-acetylated in Sirt1-deficient cells and that among them the level of acetylated CHK2 was highly increased. We found that Sirt1 formed molecular complexes with BRCA1/BRCA2-associated helicase 1 (BACH1), H2AX, Tumor suppressor p53-binding protein 1 (53BP1), and CHK2, which are key factors of DDR. We then demonstrated that CHK2 was normally inhibited by SIRT1 via deacetylation but dissociated with SIRT1 under oxidative stress conditions. This led to acetylation and activation of CHK2, which increased cell death under oxidative stress conditions. Our data also indicated that SIRT1 deacetylated K235 and K249 residues of CHK2, whose acetylation increased cell death in response to oxidative stress. Thus, SIRT1, a metabolic sensor, protects cells from oxidative stress-dependent DDR by deacetylation of CHK2. Our finding suggests a crucial function of SIRT1 that inhibits CHK2 as a potential therapeutic target for cancer treatment.

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Keywords: Metabolism, DDR, Cell death, Aging

S-14-3

The role of senescent tumor cells in cancer progression

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Cellular senescence is featured by an irreversible cell cycle arrest. Various stresses including DNA damage, ER stress, and even shortening of the telomere can lead the cell to the cellular senescence. According to our current insight, senescent tumor cells promote cancer progression rather than suppression in colorectal cancer. We found that colorectal cancer patients who had p16INK4A+ senescent tumor cells showed the decreased number of infiltrated cytotoxic T-cells in the cancer epithelium. It attributes to the CXCL12, a well-known chemokine, being released from senescent tumor cells. The CXCL12 induces internalization of its receptor CXCR4 in cytotoxic T-cells to lose their directionality. Furthermore, senescent tumor cells are involved in macrophage differentiation. The CSF1 released from senescent tumor cells induces monocytes to differentiate into M2 macrophage to promote cancer progression. Recently, other interesting results have been found in single-cell RNA sequencing of colorectal cancer patient tissues. The p16INK4A+ senescent tumor cells showed high correlation with MMP7 expression. Interestingly, consequent immunohistochemistry showed that MMP7+ senescent tumor cells are confined in the invasive front of the cancer tissue rather than the center of it. It suggests that senescent tumor cells are highly involved in local invasion and are heterogenous according to their spatial distribution. Following studies are needed to reveal the subtypes and their physiological role in colorectal cancer progression in keeping with their location. Consequently, it is expected that understanding of heterogeneity of senescent tumor cells can contribute to the advance in precision medicine to inhibit cancer progression and relapse.

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Competing interests: CXCL12 and CSF1 inhibitor hold the patent applications related to the contents of this work (10-2423631 in Korea).

Keywords: Senescent tumor cells, CXCL12, Colorectal cancer

S-14-4

The role of caveolin-2 in age-related neuroinflammation

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Aging is a major risk factor for common neurodegenerative diseases. Although multiple molecular, cellular, structural, and functional changes occur in the brain during aging, the involvement of caveolin-2 (Cav-2) in brain ageing remains unknown. We investigated Cav-2 expression in brains of aged mice and its effects on endothelial cells. The human umbilical vein endothelial cells (HUVECs) showed decreased THP-1 adhesion and infiltration when treated with Cav-2 siRNA compared to control siRNA. In contrast, Cav-2 overexpression increased THP-1 adhesion and infiltration in HUVECs. Increased expression of Cav-2 and Iba-1 was observed in brains of old mice. Moreover, there were fewer Iba1-positive cells in the brains of aged Cav-2 knockout (KO) mice than of wild-type aged mice. The levels of several chemokines were higher in brains of aged wild-type mice than in young wild-type mice; moreover, chemokine levels were significantly lower in brains of young mice as well as aged Cav-2 KO mice than in their wild-type counterparts. Expression of PECAM1 and VE-cadherin proteins increased in brains of old wild-type mice but was barely detected in brains of young wild-type and Cav-2 KO mice. Collectively, our results suggest that Cav-2 expression increases in the endothelial cells of aged brain, and promotes leukocyte infiltration and age-associated neuroinflammation

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Competing interests: I declare no competing interests.

Keywords: Aging, Neuroinflammation, Caveolin-2, Endothelial cell

S-15-1

CXCR4 Regulates Temporal Differentiation of Embryonic Salivary Glands via PRC1 Complex

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CXC-chemokine receptor type 4 (CXCR4), a 7-transmembrane receptor family member, displays multifaceted roles, participating in immune cell migration, angiogenesis, and even adipocyte metabolism. However, the activity of such a ubiquitously expressed receptor in epithelial gland organogenesis has not yet been fully explored. To investigate the relationship between CXCL12/CXCR4 signaling and embryonic glandular organogenesis, we used an ex vivo culture system with live imaging and RNA sequencing to elucidate the transcriptome and protein-level signatures of AMD3100, a potent abrogating reagent of the CXCR4-CXCL12 axis, imprinted on the developing organs. Immunostaining results showed that CXCR4 was highly expressed in embryonic submandibular gland, lung, and pancreas, especially at the periphery of end buds containing numerous embryonic stem/progenitor cells. Despite no significant increase in apoptosis, AMD3100-treated epithelial organs showed a retarded growth with significantly slower branching and expansion. Further analyses with submandibular glands revealed that such responses resulted from the AMD3100-induced precocious differentiation of embryonic epithelial cells, losing mitotic activity. RNA sequencing analysis revealed that inhibition of CXCR4 significantly down-regulated polycomb repressive complex (PRC) components, known as regulators of DNA methylation. Treatment with PRC inhibitor recapitulated the AMD3100-induced precocious differentiation. Our results indicate that the epigenetic modulation by the PRC-CXCR12/CXCR4 signaling axis is crucial for the spatiotemporal regulation of proliferation and differentiation of embryonic epithelial cells during embryonic glandular organogenesis.

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Competing interests: None

Keywords: CXCR4, Epithelial gland, Embryonic submandibular gland, Differentiation, Organogenesis, Polycomb repressive complex (PRC), Epigenetic modulation

S-15-2

Anticancer effect of verteporfin on non-small cell lung cancer via downregulation of ANO1

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Anoctamin 1 (ANO1) is a calcium-activated chloride channel found in various cell types and is overexpressed in non-small cell lung cancer (NSCLC), a major cause of cancer-related mortality. With the rising interest in development of druggable compounds for NSCLC, there has been a corresponding rise in interest in ANO1, a novel drug target for NSCLC. However, as ANO1 inhibitors that have been discovered simultaneously exhibit both the functions of an inhibition of ANO1 channel as well as a reduction of ANO1 protein levels, it is unclear which of the two functions directly causes the anticancer effect. In this study, verteporfin, a chemical compound that reduces ANO1 protein levels was identified through high-throughput screening. Verteporfin did not inhibit ANO1-induced chloride secretion but reduced ANO1 protein levels in a dose-dependent manner with an IC50 value of ~300 nM. Moreover, verteporfin inhibited neither P2Y receptor-induced intracellular Ca^{2+} mobilization nor cystic fibrosis transmembrane conductance regulator (CFTR) channel activity, and molecular docking studies revealed that verteporfin bound to specific sites of ANO1 protein. Confirming that verteporfin reduces ANO1 protein levels, we then investigated the molecular mechanisms involved in its effect on NSCLC cells. Interestingly, verteporfin decreased ANO1 protein levels, the EGFR-STAT3 pathway as well as ANO1 mRNA expression. Verteporfin reduced the viability of ANO1-expressing cells (PC9, and gefitinib-resistant PC9) and induced apoptosis by increasing caspase-3 activity and PARP-1 cleavage. However, it did not affect hERG channel activity. These results show that the anticancer mechanism of verteporfin is caused via the down-regulation of ANO1.

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Keywords: Anoctamin 1, EGFR-STAT3, Verteporfin, Non-small cell lung cancer

S-15-3

Intestinal microphysiological systems for investigating the interactions of the human gut with the gut microbes

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Microphysiological systems aim to recapitulate critical physiological characteristics of the target organ that conventional in vitro model systems cannot reflect. A thoughtfully designed microphysiological system can be a complementary and alternative tool to animal models with its simplicity for testing and screening, flexibility in design, lower cost, and lesser ethical concerns. Here we present the microphysiological systems mimicking some key features of the human and mouse intestines. In particular, we fabricated a simple, easy-to-use intestinal model system with the physiological oxygen gradient across the intestinal epithelium. The microphysiological system was used to coculture individual strains of anaerobic gut bacteria ranging from one of the commensal stains, a potential probiotic strain, to opportunistic pathogen strains with the primary human colon epithelial cells. This simple intestinal microphysiological system with anaerobic gut bacterial coculture capability is a promising model system and a test platform for investigating the interactions between the host and the gut bacteria, as well as screening therapeutics and toxic chemicals.

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Competing interests: NLA holds a financial interest in Altis Biosystems.

Keywords: Microphysiological systems, Intestine, Oxygen gradient, Gut bacterial coculture

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S-15-4

Systems analysis to dissect network mechanisms of drug resistance in cancer

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Targeted drugs aim to treat cancer by directly inhibiting oncogene activity or oncogenic pathways, but drug resistance frequently emerges. To dissect the resistance mechanism of cancer cells, it is necessary to understand the underlying biological system themselves. Biological components are interconnected to form complex networks. These networks consist of multiple pathways linked with various feedback and crosstalk structures that determine phenotype and drug response. From this point of view, disease is now understood as a network disturbance. Complex diseases such as cancer are reinterpreted as dysregulation states of networks. Drug responses are also described as network dynamics. Changes in feedback activity after drug treatment can counteract drug effects, and drug inhibition in one pathway can lead to unexpected activation of another pathway through crosstalk. The proposed systems approach, which integrates omics data analysis, mathematical modeling, and simulation studies, can contribute to the development of therapeutic strategies to control cancer resistance at the network level.

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Keywords: Systems biology, Signaling network, Feedback analysis, Targeted drug, Model simulation, Transcriptomics

Yudang Academic Award

SREBP-1 regulates autophagy and macrophage polarization in metabolic diseases

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Sterol response element binding protein (SREBP)-1a is a key transcriptional regulator of lipogenesis and cell growth and its' properly regulated activity is key to cellular lipid homeostasis. A metabolic imbalance between lipid synthesis and degradation can lead to hepatic lipid accumulation, a characteristic of patients with non-alcoholic fatty liver disease (NAFLD). Here, we review that high-fat-diet-induced sterol regulatory element-binding protein (SREBP)-1c, a key transcription factor that regulates lipid biosynthesis, impairs autophagic lipid catabolism via altered H2S signaling. we demonstrated that SREBP-1c directly upregulated miR-216a which reduces cystathionine gamma-lyase, the enzyme responsible for H2S production. This decreased both circulating and hepatic levels of H2S and blunted sulfhydration dependent activation of Unc-51 like autophagy activating kinase 1 (ULK1), thereby decreasing autophagic flux and lipid droplet turnover. These finding uncover a novel twofold mechanism for SREBP-1c driven lipid accumulation through reciprocal activation and inhibition of lipid biosynthesis and degradation, respectively.

Also, SREBP cleavage-associating protein (SCAP) is a sterol-regulated escort protein that translocates SREBPs from the endoplasmic reticulum to the Golgi apparatus, thereby activating lipid metabolism and cholesterol synthesis. Although SCAP regulates lipid metabolism in metabolic tissues such as the liver and muscle, the effect of macrophage-specific SCAP deficiency in adipose tissue macrophages (ATMs) of metabolic diseases is not completely understood. Here, we demonstrated that fat accumulation increased in high-fat/high-sucrose diet-fed macrophage-specific SCAP knockout mice due to polarization of classical activated macrophages in adipose tissue. SCAP deficiency stimulates M1 macrophage polarization owing to increase in intracellular cholesterol content via suppression of cholesterol efflux by reduction of 25-hydroxycholesterol-dependent LXRα activation in macrophages. Overall, the activation of SCAP-SREBP-1a complex in macrophages may provide a novel therapeutic strategy that ameliorates obesity by controlling cholesterol homeostasis in ATMs.

Young Physiologist Award

Mitochondrial energetic metabolism in Blood brain barrier maintenance

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Cerebral endothelial cells (ECs) require junctional proteins to maintain BBB integrity, restricting toxic substances and peripheral immune cells with a higher concentration of mitochondria than ECs of peripheral capillaries. The mechanism underlying BBB disruption by defective mitochondrial oxidative phosphorylation (OxPhos) is unclear in a mitochondria-related gene-targeted animal model. To assess the role of EC mitochondrial OxPhos function in the maintenance of the BBB, we developed an EC-specific *CR6-interactin factor1* (*Crif1*) deletion mouse. We clearly observed encephalomyelitis-like behavior, myelin damage and leukocyte infiltration caused by BBB disruption in this mice. Furthermore, Loss of *Crif1* led to reorganization of the actin cytoskeleton and a decrease in tight junction-associated protein expression through an ATP production defect in vivo and in vitro. To identify signaling pathways involved in linking EC-specific mitochondrial dysfunction and BBB disruption, we first performed RNA sequencing using isolated cerebral vessels from TEKCRIF1 KO mice and revealed significant changes in Notch1 signaling, a pathway intimately involved in BBB maintenance. We also observed a decrease in Notch1 signaling and expression of the mitochondrial oxidative phosphorylation (OxPhos) complex in the ICH mouse model, which also exhibits BBB disruption. We suggest that mitochondrial OxPhos acts as a source of ATP in cerebral ECs and Notch1 signaling pathway acts as an upstream regulator of DEGs and can be a target to regulate the changes involved with endothelial mitochondrial dysfunction-dependent BBB disruption. Thus, treatment methods that activate Notch1 may be beneficial in acute brain injuries by protecting BBB integrity.