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SoLA 2024

한국지질·동맥경화학회 춘계학술대회

2024 Spring Congress on Lipid & Atherosclerosis of KSoLA

2024년 4월 5일(금)~6일(토)

시그니엘 부산

대웅제약

CZ CREZET

크레젯정 [Ezetimibe / Rosuvastatin]

환자 맞춤형
콜레스테롤 관리를 위한
최적의 치료옵션

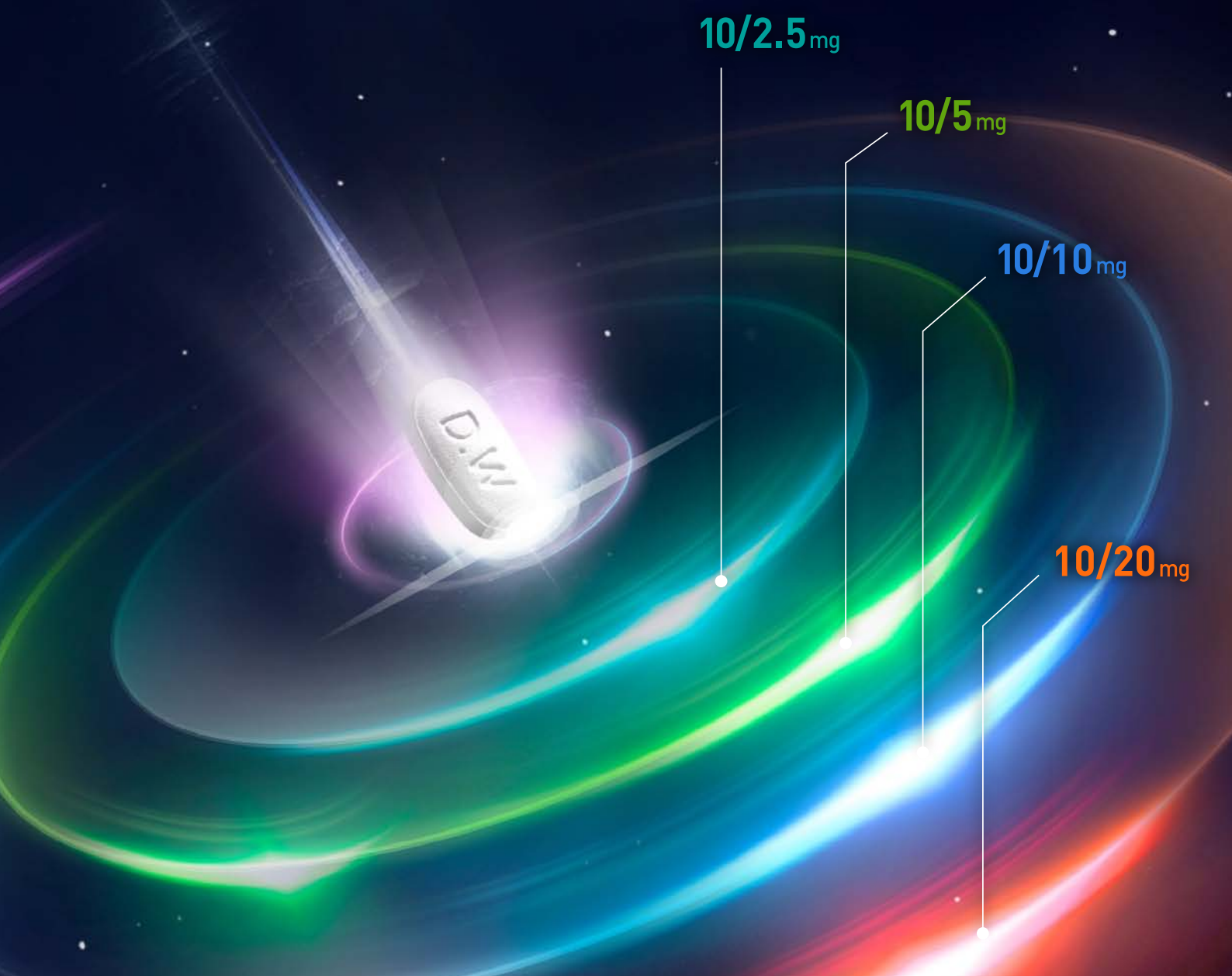
강력하고 안전한 고지혈증 치료제
크레젯으로 시작하세요

콜레스테롤 합성과
흡수 **동시억제**

혈액은 **맑게**
혈관은 **깨끗**하게

효과적인
Dual Action

더욱 **다양해진**
치료옵션



ICoLA 2024

The 13th International Congress on Lipid & Atherosclerosis

New Perspectives in Lipid and Atherosclerosis: Exploring Innovations

September 26(Thu)-28(Sat), 2024

CONRAD Seoul, Republic of Korea

• Key Speakers •

Paul Ridker
Harvard University, USA

Joerg Heeren
University of Hamburg, Germany

Scott Wright
Mayo Clinic, USA

Børge Nordestgaard
Copenhagen University, Denmark

Jaetaek Kim
Chung-Ang University, Republic of Korea

Sung Nim Han
Seoul National University, Republic of Korea

ICoLA by KSoLA International Congress on Lipid & Atherosclerosis

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SoLA 2024

한국지질·동맥경화학회 춘계학술대회

2024 Spring Congress on Lipid & Atherosclerosis of KSoLA

2024년 4월 5일(금)~6일(토)

시그니엘 부산

조직위원회

◑ 한국지질·동맥경화학회 임원진

직책(국문)	성함(국문)	소속(국문)
2024 회장	정익모	이화의대 순환기내과
2024 부회장	한 진	인제의대 생리학교실
	김정선	국립암센터
이사장	김재택	중앙의대 내분비내과
총무이사	이왕수	중앙의대 순환기내과
부 총 무	김제상	부천세종병원 심장내과
	김진화	조선의대 내분비내과
	손장원	가톨릭의대 내분비내과
	전재한	경북의대 내분비내과
	조준환	중앙의대 순환기내과
	홍준화	을지의대 내분비내과
재무이사	전성완	순천향의대 내분비내과
기획이사	이병완	연세의대 내분비내과
학술이사	이우제	울산의대 내분비내과
간행이사	강 현	중앙의대 마취통증의학과
홍보이사	안지현	한국의학연구소 내분비내과
대외협력이사	홍순준	고려의대 순환기내과
보험법제이사	박재형	고려의대 순환기내과
윤리이사	문민경	서울의대 내분비내과
교육이사	김병진	성균관의대 순환기내과
진료지침이사	이상학	연세의대 심장내과
임상연구이사	이승환	가톨릭의대 내분비내과
기초연구이사	박용식	경희의대 미생물학교실
식품영양이사	김오연	동아대 식품영양학과
특임이사	박영미	이화의대 분자의과학교실
무임소이사	박경우	서울의대 순환기내과
	박철영	성균관의대 내분비내과
	이은정	성균관의대 내분비내과
	최성희	서울의대 내분비내과
감사	권혁상	가톨릭의대 내분비내과
	김상현	서울의대 순환기내과

◑ 한국지질·동맥경화학회 학술위원회

직책(국문)	성함(국문)	소속(국문)
학술이사	이우제	울산의대 내분비내과
학술간사	정창희	울산의대 내분비내과
	윤종찬	가톨릭의대 순환기내과
	김병규	인제의대 심장내과
학술위원	김남훈	고려의대 내분비내과
	김범준	울산의대 신경과
	김보경	부산대 식품영양학과
	김진화	조선의대 내분비내과
	남궁준	연세원주의대 생화학교실
	박영미	이화의대 분자의과학교실
	양여리	가톨릭의대 내분비내과
	오규철	가톨릭의대 순환기내과
	윤민재	서울의대 순환기내과
	이상은	이화의대 순환기내과
	이용호	연세의대 내분비내과
	이중희	연세의대 심장내과
	이호규	연세의대 예방의학교실
	임현정	경희대 의학영양학과
	장영우	가천의대 심장내과
	전재한	경북의대 내분비내과
	전지은	경희의대 내분비내과
	정수명	성균관대 생명과학과
	조동혁	고려의대 순환기내과
	조은희	강원의대 내분비내과
	조준환	중앙의대 순환기내과
	최재훈	한양대 생명과학과
	허지혜	한림의대 내분비내과
	홍준화	을지의대 내분비내과
자문위원	강유미	Brigham and Women's Hospital/Harvard Medical School, USA
	김현창	연세의대 예방의학교실
	박철영	성균관의대 내분비내과
	이상학	연세의대 심장내과
	한창엽	University of Washington, USA

Program at a Glance

2024년 4월 5일(금)			
	Room 1	Room 2	Room 3
11:30–	Registration		
12:30–14:00	**Research Group Session 1** 심혈관질환예측모형 연구TFT *Current Status and Challenges in the Development of Cardiovascular Disease Prediction Models*	**Research Group Session 2** LP(a)/TRL 연구TFT *Decoding the Shadows: Exploring the Frontiers of Residual Lipid Risk in Cardiovascular Health*	
14:00–15:20	**Committee Session 1** 지질영향 식품연구 TFT *심혈관 위험관리를 위한 식사요법은 어디에 초점을 맞추어야 하는가?*	**Committee Session 2** 간행위원회 *좋은 논문으로 가는 Big Step*	**Committee Session 3** 기초연구위원회 *Advances in Metabolic Regulation: Foundations for Therapeutic Insights in Metabolic Diseases and Beyond*
15:20–15:30	Break		
15:30–17:00	**Young Investigator Session 1**	**Young Investigator Session 2**	**Young Investigator Session 3**
17:10–18:00	**Mini-Oral Presentation 1**		
18:10–19:30	Welcome Reception		

2024년 4월 6일(토)				
	Room 1	Room 2	Room 3	Room 4
07:30–08:30	**Breakfast Symposium 1**	**Breakfast Symposium 2**	**Breakfast Symposium 3**	**Breakfast Symposium 4**
08:30–10:00	**Symposium 1** *Recent Hot Trials in Cardio-Metabolic Disorders*	**Symposium 2** *Cutting-Edge Insights in Lipid and Atherosclerosis Research*	**Symposium 3** *Dietary Factors and Cardio-Metabolic Disease*	
10:00–10:15	Break			
10:15–10:20	Opening Address			
10:20–11:00	**Plenary Lecture 1** *Heart-Immune-Brain network in the pathogenesis of cardio-cerebrovascular disease*			
11:10–12:20	**Mini-Oral Presentation 2**			
12:20–13:20	**Luncheon Symposium 1**	**Luncheon Symposium 2**	**Luncheon Symposium 3**	
13:30–15:00	**Symposium 4** (KSoLA-KDA-KSSO Joint Symposium) *Multidisciplinary Approach to Prevent Cardiovascular Disease*	**Symposium 5** *Ferroptosis & Vascular Inflammation*	**Symposium 6** *Current Status and Perspectives in Nutrition Management in Cardiovascular Disease*	
15:00–15:20	Break			
15:20–16:00	**Plenary Lecture 2** *Cyclase-associated protein 1 (CAP1) binds to Resistin or PCSK9, standing at the nodal point of metabolic diseases*			
16:10–17:40	**Symposium 7** *How Can We Manage Dyslipidemia in Special Populations?*	**Symposium 8** *Frontiers in Omics Technology*	**Symposium 9** (KSoLA & KNS Joint Symposium) *Intermittent Fasting and Cardiovascular Disease (Debate Session)*	
17:40–	Closing Ceremony			

행사 개요

◑ **행사명**
- 국문: 2024 한국지질·동맥경화학회 춘계학술대회
- 영문: 2024 Spring Congress on Lipid and Atherosclerosis of KSoLA (SoLA 2024)

◑ **일자:** 2024년 4월 5일(금)~6일(토)

◑ **장소:** 시그니엘 부산

◑ **주최/주관:** 한국지질·동맥경화학회

◑ **개최방식:** 오프라인 학술대회

◑ **웹사이트:** https://sola.or.kr/

◑ **평점**
- 대한의사협회 연수교육 평점: 4월 5일(금) 최대 2평점 / 6일(토) 최대 6평점
- 한국영양교육평가원 임상영양사 전문연수교육 (CPD) 최대 5평점

평점안내

구분		4월 5일(금)	4월 6일(토)
의사	대한의사협회 연수평점	최대 2평점	최대 6평점
영양사	임상영양사 전문연수교육(CPD)	5평점 * 2일 모두 수강하셔도 최대 5평점 승인됩니다.	

※ 2016년부터 연수교육 평점에 대한 관리가 엄격히 시행됨에 따라 아래와 같이 안내드리오니 평점 인정기준을 숙지해 주시길 부탁드립니다.

ⓥ 본인 신분증 필히지참

ⓥ 출결 관리 강화: 모든 교육기관에서 교육 수강 시작 전, 교육 수강 종료 후 출석여부확인을 의무화하고 2번의 확인이 없는 경우 평점을 부여하지 않습니다.

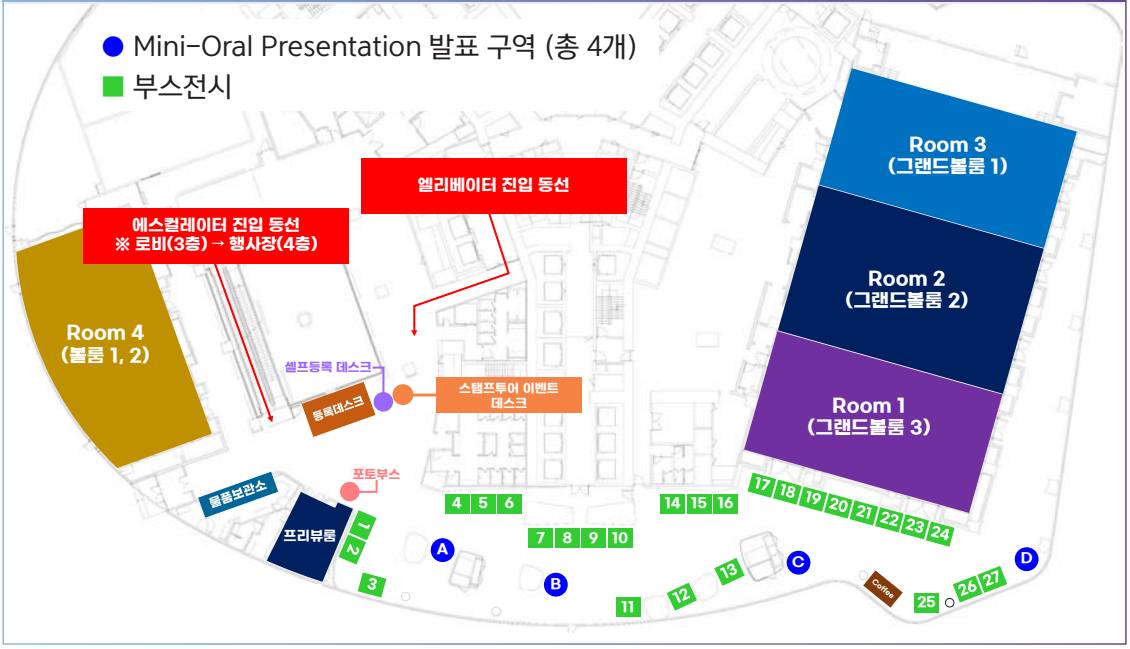
ⓥ 부분평점 인정기준

1시간 미만	평점 없음
1시간 이상~2시간 미만	1평점
2시간 이상~3시간 미만	2평점
3시간 이상~4시간 미만	3평점
4시간 이상~5시간 미만	4평점
5시간 이상~6시간 미만	5평점
6시간 이상	6평점 (최대)

※ 2번의 출석 확인이 되지 않는 경우에는 평점 부여가 되지 않습니다. 반드시 확인하시어 불이익이 없도록 하시길 부탁드립니다.

행사장 안내

Floor Plan (시그니엘 부산 4층)



부스번호	후원사명
1	동아ST
2	
3	한독
4	JLA 부스
5	셀트리온제약
6	한국다이이찌산쿄
7	JW중외제약
8	대웅제약
9	유영제약
10	한미약품
11	제일약품
12	보령
13	일동제약
14	유한양행

부스번호	후원사명
15	종근당
16	LG화학
17	대원제약
18	
19	안국약품
20	
21	한림제약
22	GC녹십자
23	비씨월드제약
24	동국생명과학
25	알보젠코리아
26	대웅바이오
27	

SoLA 이벤트 안내

첫번째 이벤트! 스탬프투어 이벤트

SoLA 2024 행사장 로비에 설치된 전시부스를 방문하시고 스탬프를 80% 이상 적립하시면 스타벅스 기프트카드(3만원권)를 증정해드립니다!

[참여방법]

① 등록 후 받으신 키트에 스탬프투어 이벤트 용지가 포함되어 있습니다.
② 전시부스에 방문하여 스탬프를 적립해주세요. 80%(19개) 이상 적립!
③ 등록데스크 부근에 위치한 스탬프투어 이벤트 데스크를 방문해주세요.
④ 스타벅스 기프트카드를 받아가세요!

두번째 이벤트! 럭키드로우 이벤트

웰컴 리셉션, 조찬 심포지엄, 폐회식에서 경품 추첨이 진행됩니다!

웰컴 리셉션 | 4월 5일(금) 18:00~ | Room 4
***사전 등록 시 신청하신 분들만 참여 가능합니다.**


1등 (1명)
Apple 아이폰 15 프로 256GB


2등 (2명)
LG전자 시네빔 프로젝터


3등 (3명)
Apple 에어팟 프로 2세대

조찬 심포지엄 | 4월 6일(토) 07:30~08:00 | Room 1~4


1등 (1명)
Apple 애플워치 9


2등 (1명)
Apple 에어팟 프로 2세대


3등 (3명)
스타벅스 기프트 카드

폐회식 | 4월 6일(토) 17:40 | Room 1,2,3


1등 (1명)
삼성전자 갤럭시 S24
울트라 5G 256GB


2등 (1명)
Apple 아이패드 프로
12.9형 6세대 M2 128GB


3등 (1명)
삼성전자 비스포크
공기청정기


4등 (3명)
삼성전자 갤럭시 워치6

[참여방법]

① 웰컴 리셉션, 조찬 심포지엄, 폐회식에 참여해주세요. (단, 웰컴 리셉션은 사전 등록 시 신청하신 분들만 참여 가능!)
② 명찰에 삽지된 경품 응모권 중 '제출용' 응모권을 추첨함에 넣어주세요.
③ 경품 추첨 응모함은 각 행사장의 입구에 비치될 예정입니다.

SoLA 이벤트 안내

세번째 이벤트! 포토제닉 이벤트

SoLA 2024 행사장 어디서든 같은 소속 연구실, 동료분들과 사진을 촬영해주세요!
가장 많은 인원이 촬영된 사진을 제출하시면 스타벅스 e-기프트카드를 증정해드립니다!
(1등 20만원권 / 2등 15만원권 / 3등 10만원권)

[참여방법]

① 행사장에서 동료분들과 단체사진을 촬영해 주세요.
② 우측 QR코드로 SoLA 2024 카카오톡 채널에 접속해주세요.
③ 카카오톡 채널에 사진을 제출해주세요.
④ 학술대회 종료 후 공지사항을 통해 선정 결과가 공지됩니다.

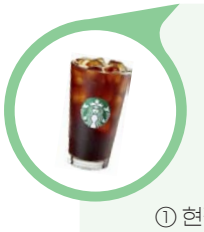
네번째 이벤트! 학회 참관기

SoLA 2024 참석에 대한 소감을 자유롭게 작성하고, 사진과 함께 보내주세요!
선정되신 분께는 백화점 상품권(20만원)을 증정해드립니다!

[참여방법]

① 워드/한글에 자유로운 형식으로 SoLA 2024 참석에 대한 후기, 소감 등을 적어주세요. (1,000자 이내)
② SoLA 2024 행사장에서 촬영한 본인의 사진을 함께 저장해주세요.
③ SoLA 2024 준비사무국 이메일(sola2024@planbear.co.kr)로 학회 참관기를 제출해주세요.
④ 학술대회 종료 후 공지사항을 통해 선정 결과가 공지됩니다.

다섯번째 이벤트! 현장참석 설문조사



SoLA 2024 현장 참석자 분들 중 설문조사에 참여하신 분들을 대상으로
추첨을 통해 선정되시면 스타벅스 아이스 아메리카노 쿠폰을 증정해드립니다!

[참여방법]

① 현장에서 안내되는 설문조사 참여 링크를 확인해주십시오. (브레이크 타임, 폐회식 전 등
강의장에서 설문조사 링크를 확인하실 수 있고, 추후 문자로 링크를 보내드릴 예정입니다.)
② 설문조사를 작성 및 제출해주십시오.
③ 설문조사 종료 후 추첨을 통해 선정되신 분들께 커피 쿠폰을 보내드립니다.

Program in Detail

Plenary Lecture 1

4월 6일(토) 10:20-11:00 | Room 1, 2, 3

좌장 : 김재택(중앙의대 내분비내과)

10:20-11:00 **Heart-Immune-Brain network in the pathogenesis of cardio-cerebrovascular disease** / 38

오구택(이화여대 생명과학과)

Plenary Lecture 2

4월 6일(토) 15:20-16:00 | Room 1, 2, 3

좌장 : 정익모(이화의대 순환기내과)

15:20-16:00 **Cyclase-associated protein 1 (CAP1) binds to Resistin or PCSK9, standing at the nodal point of metabolic diseases** / 42

김효수(서울의대 순환기내과)

Symposium 1

4월 6일(토) 08:30-10:00 | Room 1

Recent Hot Trials in Cardio-Metabolic Disorders

좌장 : 이상학(연세의대 심장내과), 문민경(서울의대 내분비내과)

패널 : 김병규(인제의대 심장내과), 김진화(조선의대 내분비내과), 윤민재(서울의대 순환기내과)

심혈관 및 대사 질환 발생의 예방을 위해 전방위적인 치료 접근이 연구되고 있고 나날이 발전하고 있습니다. 본 세션에서는 지난 한해 동안 발표된 이 분야의 대규모 연구를 살펴보는 시간을 마련하였으며, 이 세션을 통해 지질 분야 및 심부전의 새로운 치료 접근법에 대해서 최신 지견을 넓힐 수 있는 시간이 될 것으로 기대합니다.

08:30-08:50 **The role of bempedoic acid in patients with high cardiovascular risk and statin intolerance** / 46

김학령(서울의대 순환기내과)

08:50-09:10 **Semaglutide: beyond STEP trials, toward obesity & diabetocardiology** / 48

윤종찬(가톨릭의대 순환기내과)

09:10-09:30 **Lessons from clinical trials of Inclisiran: a first-in-class siRNA therapy against PCSK9** / 50

전재한(경북의대 내분비내과)

09:30-10:00 **Panel Discussion**

Symposium 2

4월 6일(토) 08:30-10:00 | Room 2

Cutting-Edge Insights in Lipid and Atherosclerosis Research

좌장 : 김치대(부산의대 약리학교실), 한진(인제의대 생리학교실)

패널 : 권유욱(서울의대 의생명연구원), 김경진(인하의대 의생명학교실),
김규호(가톨릭의대 내분비내과), 최재훈(한양대 생명과학과)

비만, 동맥경화 등의 질환에 대한 기초연구에서 조직의 정상 기능과 질병의 병태생리를 이해하는 것은 매우 중요합니다. 본 세션에서는 지질 및 동맥경화 연구 분야의 최신 지견을 제공하여 관련 질환들에 대한 새로운 시각을 제공할 것으로 기대합니다.

08:30-08:50	**Dissecting adipose tissue at single-cell resolution during metabolic disease development** **문준호**(서울의대 내분비내과)	/ 54
08:50-09:10	**Monocyte priming and macrophage reprogramming in atherosclerosis and diet-induced obesity** **안용주**(POSTECH IT융합공학과)	/ 56
09:10-09:30	**RXX regulates balance of skeletal muscles and adipose tissues** **국현**(전남의대 약리학교실)	/ 58
09:30-10:00	**Panel Discussion**	

Symposium 3

4월 6일(토) 08:30-10:00 | Room 3

Dietary Factors and Cardio-Metabolic Disease

좌장 : 박용순(한양대 식품영양학과), 김은정(대구가톨릭대 식품영양학과)

패널 : 곽정현(인제대 식품영양학과), 김오연(동아대 식품영양학과), 조동혁(고려의대 순환기내과)

식이성 요인은 심혈관계질환 및 대사성질환의 예방과 관리에 있어서 매우 중요한 요인 중 하나입니다. 이에 본 세션은 최근 관심사를 중심으로 대사기전, 임상연구 및 활용 등을 다루고자 합니다. 첫번째 연자인 김보경 교수님은 식이성 콜레스테롤의 체내 흡수와 대사기전에 대해 강연할 예정입니다. 두번째 연자인 김민주 교수님은 이차성 이상지질혈증 발생과 식이성 요인에 대한 최신 임상연구 및 근거에 대해 소개할 예정입니다. 마지막 연자인 김유리 교수님은 기능성 감미료가 대사성질환에 미치는 효과와 대사기전에 대해 강연할 예정입니다.

08:30-08:50	**Trust your gut for cholesterol metabolism** **김보경**(부산대 식품영양학과)	/ 62
08:50-09:10	**Current evidence regarding dietary causes of secondary dyslipidemia** **김민주**(한남대 식품영양학과)	/ 64
09:10-09:30	**Functional sweeteners and metabolic diseases: effect and mechanism** **김유리**(이화여대 식품영양학과)	/ 66
09:30-10:00	**Panel Discussion**	

Symposium 4 (KSoLA-KDA-KSSO Joint Symposium)

4월 6일(토) 13:30-15:00 | Room 1

Multidisciplinary Approach to Prevent Cardiovascular Disease

좌장 : 박철영(성균관의대 내분비내과), 이우제(울산의대 내분비내과)

패널 : 손장원(가톨릭의대 내분비내과), 이상은(이화의대 순환기내과), 진상만(성균관의대 내분비내과)

동맥경화성 심혈관 질환 예방을 위해서는 어느 하나의 접근이 아닌 다학제적인 접근이 매우 중요합니다. 이러한 시대적 흐름에 발맞추어 이번 SoLA 2024 에서는 한국지질·동맥경화학회, 대한당뇨병학회, 대한비만학회와 공동 심포지움을 준비하였는데, 각 학회의 대표 연사 분들께서 지질분야의 unmet needs, 디지털 치료 기반 당뇨병 관리 및 항비만 치료의 심혈관계 질환 발생에 미치는 영향에 대해서 다루어 줌으로써 상호 이해를 넓히고 향후 공동으로 실행해 나갈 방향을 모색하고자 합니다.

13:30-13:50	**KSoLA – Deciphering lipid enigmas: exploring TRL, triglycerides, and remnant cholesterol as residual lipid risks and treatment targets** **김병진**(성균관의대 순환기내과)	/ 70
13:50-14:10	**KDA – Role of continuous glucose monitoring in diabetic patients at high cardiovascular risk** **이준엽**(가톨릭의대 내분비내과)	/ 72
14:10-14:30	**KSSO – CVOTs using non-GLP-1 based anti-obesity medications and bariatric surgery** **홍준화**(을지의대 내분비내과)	/ 74
14:30-15:00	**Panel Discussion**	

Symposium 5

4월 6일(토) 13:30-15:00 | Room 2

Ferroptosis & Vascular Inflammation

좌장 : 박용식(경희의대 미생물학교실), 강은석(연세의대 내분비내과)

패널 : 김영국(전남의대 생화학교실), 최동욱(고려대 생명공학부), 한주희(우석대 약학과), 허경선(충남대 약학과)

대사질환의 병태생리에서 ferroptosis 및 염증반응이 주목을 받고 있습니다. 본 세션에서는 해당 분야를 전문으로 하시는 연구자분들의 최신 연구 결과들을 공유하는 자리를 준비하였습니다.

13:30-13:50	**Role of metabolic reprogramming in chorioretinal diseases** **박동호**(경북의대 안과)	/ 78
13:50-14:10	**Roles of E3 ligases involved in NASH development** **송재환**(연세대 생화학과)	/ 80
14:10-14:30	**Potential roles of ferroptosis in atherosclerosis** **이은우**(한국생명공학연구원 대사제어연구센터)	/ 82
14:30-15:00	**Panel Discussion**	

Symposium 6

4월 6일(토) 13:30-15:00 | Room 3

Current Status and Perspectives in Nutrition Management in Cardiovascular Disease

좌장 : 김은미(강북삼성병원 영양팀), 김오연(동아대 식품영양학과)

패널 : 백진경(을지대 식품영양학과), 전지은(경희의대 내분비내과), 최미옥(동아대 식품영양학과)

본 세션에서는 심혈관계질환의 영양관리 현황과 전망을 주제로 하여 임상영양치료와 활용방안을 다루고자 합니다. 첫번째 연자인 강현희 임상영양사님은 심혈관계질환의 영양치료 전략에 대해 강연할 예정입니다. 두번째 연자인 임현정 교수님은 심혈관계질환 예방 및 관리에 있어서 식생활 중재의 효과연구에 대해 소개할 예정입니다. 마지막 연자인 이보경 교수님은 심혈관계질환 위험관리에 있어서 최근 관심사인 프로바이오틱스의 역할에 대해 강연할 예정입니다.

13:30-13:50	**Nutrition therapy strategy for cardiovascular management** **강현희**(창원경상대병원 영양팀)	/ 86
13:50-14:10	**Impact of lifestyle modifications in preventing and managing cardiovascular diseases** **임현정**(경희대 의학영양학과)	/ 90
14:10-14:30	**Role of probiotics in cardiovascular risk management** **이보경**(동아대 식품영양학과)	/ 92
14:30-15:00	**Panel Discussion**	

Symposium 7

4월 6일(토) 16:10-17:40 | Room 1

How Can We Manage Dyslipidemia in Special Populations?

좌장 : 김상현(서울의대 순환기내과), 이은정(성균관의대 내분비내과)

패널 : 오규철(가톨릭의대 순환기내과), 정창희(울산의대 내분비내과), 조준환(중앙의대 순환기내과)

임상에서 이상지질혈증을 치료하는 가운데 고민스러운 상황이 종종 있고, 논란이 되는 특정 집단이 있습니다. 특히 노인환자, 만성콩팥병을 동반한 환자 및 청소년이나 젊은 성인에서 이상지질혈증을 과연 치료해야 하는 지 치료한다면 어느 정도를 해야 하는 지 등 혼란스러운 경우가 있습니다. 본 세션에서는 이러한 특정 집단에서 이상지질혈증 관리에 대한 최신 지견을 제공하여 줄 것입니다.

16:10-16:30	**Dyslipidemia management in elderly patients** **최성훈**(한림의대 순환기내과)	/ 96
16:30-16:50	**Dyslipidemia in chronic kidney disease** **유태현**(연세의대 신장내과)	/ 98
16:50-17:10	**Dyslipidemia in adolescents and young adults** **이은영**(가톨릭의대 내분비내과)	/ 100
17:10-17:40	**Panel Discussion**	

Symposium 8

4월 6일(토) 16:10-17:40 | Room 2

Frontiers in Omics Technology

좌장 : 배순식(부산의대 약리학교실), 남궁준(연세원주의대 생화학교실)

패널 : 김정한(가톨릭의대 생화학교실), 김형석(충남의대 생리학교실), 류홍열(경북대 생명과학부)

Omics 기술의 발달은 이전에는 힘들었던 분자수준의 병태생리를 이해하는데 도움을 주고 있습니다. 본 세션은 질병 연구에서의 최신 기술의 적용 및 그 활용에 대해 논의하는 시간이 될 것입니다.

16:10-16:30	**Exploring the role of somatic mutations in human brain diseases** **김준호**(성균관대 생명과학과)	/ 104
16:30-16:50	**Integration of multiomics for understanding nanotoxicity** **이광**(아주의대 생리학교실)	/ 106
16:50-17:10	**Dysfunctional adipocytes promote tumor progression through YAP/TAZ-dependent cancer-associated adipocyte transformation** **이한웅**(연세대 생화학과)	/ 108
17:10-17:40	**Panel Discussion**	

Symposium 9 (KSoLA & KNS Joint Symposium)

4월 6일(토) 16:10-17:40 | Room 3

Intermittent Fasting and Cardiovascular Disease (Debate Session)

좌장 : 김혜영(용인대 식품영양학과), 정인경(경희의대 내분비내과)

패널 : 신민정(고려대 바이오시스템의과학부), 이수용(부산의대 순환기내과), 홍경희(동서대 식품영양학과)

본 세션은 한국영양학회(KNS)와 한국지질·동맥경화학회(KSoLA)가 공동으로 개최하는 debate 세션으로, 최근 들어 많은 관심을 받고 있는 간헐적 단식이 심혈관계질환 예방 및 관리에 미치는 효과에 대해 찬반으로 나누어 발표하고 토론하는 형식으로 구성되었습니다. 찬성편의 발표자는 송윤주 교수님으로 첫번째로 발표할 예정이며, 뒤이어서 장영우 교수님이 반대편에서 발표할 예정입니다.

16:10-16:30	**The effects of intermittent fasting on cardiometabolic health: Pros** **송윤주**(가톨릭대 식품영양학과)	/ 112
16:30-16:50	**The effects of intermittent fasting on cardio metabolic health: Cons** **장영우**(가천의대 심장내과)	/ 114
16:50-17:40	**Panel Discussion**	

Research Group Session 1. 심혈관질환예측모형 연구TFT

4월 5일(금) 12:30-14:00 | Room 1

Current Status and Challenges in the Development of Cardiovascular Disease Prediction Models

좌장 : 배장환(충북의대 심장내과), 김현창(연세의대 예방의학교실)

패널 : 구유정(서울의대 내분비내과), 김응규(인제의대 신경과), 박경민(울산의대 심장내과), 신지애(강원대 빅데이터메디컬융합학과), 이혁희(연세의대 예방의학교실), 천대영(한림의대 순환기내과)

심혈관질환 발생 위험도를 정확하게 예측하는 것은 예방적 중재 대상자들을 선별하고, 최적의 치료 전략을 세우는데 필수적인 요소입니다. 국내외에서 다양한 심혈관질환 예측 모델이 개발되고 있지만, 아직 임상현장에서 활용도는 높지 않습니다. 본 세션에서는 심혈관질환 예측 모델 개발 현황을 파악하고, 우리나라의 대표적인 연구성과들을 리뷰한 이후에 심혈관질환 예측모형 연구 발전 방향에 대한 종합 토론을 진행합니다. 이를 통하여 심혈관질환 예측모형 연구TFT의 활동 방향을 설정할 것입니다.

12:30-12:50	**CVD prediction model in Korea: current status and challenge** **김현창**(연세의대 예방의학교실)	/ 118
12:50-13:10	**Cardiovascular risk prediction model based on prospective cohort studies** **이호규**(연세의대 예방의학교실)	/ 120
13:10-13:30	**Cardiovascular risk prediction model based on health insurance claims data** **박상우**(울산의대 심장내과)	/ 122
13:30-14:00	**Panel Discussion**	

Research Group Session 2. LP(a)/TRL 연구TFT

4월 5일(금) 12:30-14:00 | Room 2

Decoding the Shadows: Exploring the Frontiers of Residual Lipid Risk in Cardiovascular Health

좌장 : 정명호(광주보훈병원 순환기내과), 김병진(성균관의대 순환기내과)

패널 : 박경우(서울의대 순환기내과), 정인경(경희의대 내분비내과),
최성훈(한림의대 순환기내과), 최성희(서울의대 내분비내과)

본 세션은 잔존지질위험 분야에서의 새로운 탐구와 발견을 강조하며 심혈관건강에 대한 깊은 이해와 향상된 관리방법을 탐색하는 것을 목표로 합니다. 'Decoding the Shadows'는 아직 완전히 이해되지 않은 잔존위험의 영역을 탐색하고 그 해답을 찾아가는 과정을 암시합니다. 이는 심혈관 질환 관리와 예방에 있어 주요 잔존지질 위험인자 중 LPa/TRL에 대한 최신 연구와 통찰을 제공하겠다는 세션의 목표를 반영합니다.
강의 1. 임상의가 LDLC RC 및 LP(a)검사방법에 대해 올바르게 이해하고 검사 시 주의해야 할 사항과 결과 해석 시 주의사항에 대한 중요한 통찰을 제공하겠다는 목적입니다. 'Deciphering lipid tests'는 이러한 검사 방법들을 정확히 이해하고 적용하는데 필요한 지식을 제공하며, 'critical insights for clinicians'는 임상의들이 실제 환자 진료에 있어서 이러한 검사 결과를 올바르게 해석하고 적용하는 데 중용한 정보와 지침을 받을 것을 강조합니다.
강의 2. 중성지방 연구에서 나타난 한국 빅데이터 분석과 최근 RCTs결과사이의 불일치를 해결하고, 이 두가지 접근 방법에서 얻은 통찰을 통합하는 과정에 초점을 맞춥니다. 'Unraveling the triglycerides puzzle'은 중성지방에 관한 연구의 복잡성을 풀어내고, 더 깊은 이해를 추구하며, 'unifying insights and addressing discrepancies'는 두 연구 방법의 결과 사이에 존재하는 차이점을 인정하고, 이를 조화롭게 해석하려는 의지를 반영합니다.
강의 3. LP(a)에 대한 연구가 잔존위험 평가에 어떠한 중요한 의미를 가지며, 한국에서의 연구와 데이터 수집에서 겪고 있는 도전과 부족한 점들을 탐구하겠다는 의도를 명확하게 전달합니다. 'quest for clarity'는 이해의 깊이를 더하고, 잔존위험 연구 분야에서의 명확성을 추구하는 과정을 강조하며, 'unveiling the residual risk and the gaps in Korea's data landscape'은 국내 데이터 현황과 그 한계를 직접적으로 다루겠다는 계획을 반영합니다.

12:30-13:00	**Deciphering lipid tests: critical insights into LDLC, RC, and LP(a) measurements for clinicians** **김솔잎**(울산의대 진단검사의학과)	/ 126
13:00-13:20	**Unraveling the triglycerides puzzle: unifying insights and addressing discrepancies between Korean big-data and RCT findings** **김남훈**(고려의대 내분비내과)	/ 128
13:20-13:40	**Lipoprotein(a) and the quest for clarity: unveiling the residual risk and the gaps in Korea's data landscape** **장영우**(가천의대 심장내과)	/ 130
13:40-14:00	**Panel Discussion**	

Committee Session 1. 지질영향 식품연구 TFT

4월 5일(금) 14:00-15:20 | Room 1

심혈관 위험관리를 위한 식사요법은 어디에 초점을 맞추어야 하는가?

좌장 : 박은주(경남대 식품영양학과), 박영미(이화의대 분자의과학교실)

패널 : 김오연(동아대 식품영양학과), 김희동(순천향의대 심장내과),
이규환(대상건강연구소), 임현정(경희대 의학영양학과)

섭식은 동물의 생존이자 인류의 문화며 또한 투약의 경로입니다.
식사 후 생체반응은 너무나 뚜렷해서 별도의 배움이 없어도 누구든 효과를 봅니다. 자연스럽게 동서고금 모든 사회에 다양한 식사 치료법이 존재했고 현재도 개발되고 있습니다. 다만, 현대의학은 수많은 식사 치료법 중 효과와 안전성이 확실한 치료법만 선별 적용할 의무가 있습니다. 본 세션에서는 범람하는 정보를 해석하는 기준과 최신 현장을 둘러보고 향후 식사 치료의 전망을 토론합니다.

14:00-14:15	**심혈관 위험 관리에서 식사의 보건학적 관점** **김미경**(한양의대 예방의학교실)	/ 134
14:15-14:30	**특수의료용도식품의 활용** **이인석**(경희의료원 영양팀)	/ 136
14:30-14:45	**심혈관 건강기능식품 산업 및 활용 현황** **이상길**(부경대 식품영양학과)	/ 138
14:45-15:00	**건강보조식품의 허와 실** **이시훈**(가천의대 내분비내과)	/ 140
15:00-15:20	**Panel Discussion**	

Committee Session 2. 간행위원회

4월 5일(금) 14:00-15:20 | Room 2

좋은 논문으로 가는 Big Step

좌장 : 강현(중앙의대 마취통증의학과), 오형철(중앙의대 소화기내과)

최근의 연구 환경은 끊임없는 변화를 맞이하고 있으며, 이에 따라 논문 작성 환경 또한 다양한 도구들의 도입으로 진화하고 있습니다. 본 세션은 연구자와 저자들에게 최신 정보를 제공하여 논문 작성의 효율성을 높이고자 합니다. 연구 결과를 효과적으로 시각화하는 방법, graphical abstract, 그리고 최신 AI 도구의 활용에 중점을 두고 다룰 예정입니다. 우선 연구자와 디자이너의 시각에서 데이터 시각화를 살펴보며, 연세의대 예방의학교실 이호규 교수님과 인권앤파트너스 황인권 대표님의 발표가 이루어집니다. 논문 작성에서 주요한 쟁점인 graphical abstract 및 chat GPT 활용에 대해서도 논의하며, Medical Illustration & Design 장동수 대표님과 컴팩스의 황윤희 대표님이 발표를 맡아주실 예정입니다. 이 세션을 통해 저자와 연구자 들은 논문 작성과 표현에 대한 새로운 가능성 및 현대 연구의 동향에 대한 새로운 시각을 가질 수 있을 것입니다. 또한 연구 결과를 표현하고, 전달하는 방법에 대해 심도 있게 고민해보는 의미 있는 시간이 될 것입니다.

14:00-14:20	**의학연구를 위한 데이터 시각화 길라잡이** **이호규**(연세의대 예방의학교실)	**/ 144**
14:20-14:40	**데이터 시각화의 최신 트렌드와 디자이너가 바라본 메디컬 인포그래픽스** **황인권**(인권앤파트너스)	**/ 146**
14:40-15:00	**좋은 논문의 완성, Graphical Abstract 만들기** **장동수**(Medical Illustration & Design)	**/ 148**
15:00-15:20	**인공지능이 연구 논문 작성을 어떻게 변화시키는가: 가능성과 한계** **황윤희**(컴팩스)	**/ 150**

Committee Session 3. 기초연구위원회

4월 5일(금) 14:00-15:20| Room 3

Advances in Metabolic Regulation: Foundations for Therapeutic Insights in Metabolic Diseases and Beyond

좌장 : 김형규(인제의대 심혈관대사질환센터), 조계원(순천향대 의생명연구원)

패널 : 김용숙(전남대 의생명연구원), 송주현(전남의대 해부학교실), 이준엽(울산의대 안과), 정수명(성균관대 생명과학과)

본 세션은 지질대사 연구회가 주최하는 심포지엄으로 오믹스, 시스템 생물학, 단백질 대사를 포함하는 다양한 연구방법을 기반으로 한 최신 대사 연구내용들로 심포지엄을 준비했습니다. 서울대학교 강윤표 교수님은 지질대사와 밀접한 관련이 있는 세포사멸 모델인 페롭토시스의 대사적 기전을, GIST 이선재 교수님은 시스템 생물학적 접근법을 바탕으로 한 생체 대사 모델링 연구에 대해 발표할 예정입니다. 전남대학교 윤소미 교수님은 당뇨병을 포함한 대사병리에서 중요한 역할을 하는 번역 후 단백질 변형 기전에 대해서 발표할 예정입니다.

14:00-14:20	**Understanding and targeting of cysteine metabolism in cancer** **강윤표**(서울대 약학과)	/ 154
14:20-14:40	**Systems biology of human metabolism – multi-omics and modeling approach** **이선재**(GIST 생명과학부)	/ 156
14:40-15:00	**Nitric oxide-induced protein modifications in disease** **윤소미**(전남대 약학부)	/ 158
15:00-15:20	**Panel Discussion**	

Breakfast Symposium 1

4월 6일(토) 07:30-08:30 | Room 1

좌장 : 김명아(서울의대 순환기내과)

패널 : 구유정(서울의대 내분비내과), 김병규(인제의대 심장내과)

07:30-07:50	**The latest clinical trial of Atorvastatin, LODESTAR study** **정창희**(울산의대 내분비내과)	/ 162
07:50-08:00	**Panel Discussion**	

Breakfast Symposium 2

4월 6일(토) 07:30-08:30 | Room 2

좌장 : 한기훈(울산의대 순환기내과)

패널 : 김희동(순천향의대 심장내과), 양여리(가톨릭의대 내분비내과)

07:30-07:50	**Benefits of Rosuvastatin and Ezetimibe combination therapy beyond lipid lowering effect in T2DM** **문준성**(영남의대 내분비내과)	/ 164
07:50-08:00	**Panel Discussion**	

Breakfast Symposium 3

4월 6일(토) 07:30-08:30 | Room 3

좌장 : 박경수(서울의대 내분비내과)

패널 : 이은영(가톨릭의대 내분비내과), 조동혁(고려의대 순환기내과)

07:30-07:50 Combination of DPP-4i and SGLT-2i: new insights from recent clinical trials / 166
김진화(조선의대 내분비내과)

07:50-08:00 Panel Discussion

Breakfast Symposium 4

4월 6일(토) 07:30-08:30 | Room 4

좌장 : 최동훈(연세의대 심장내과)

패널 : 배재현(고려의대 내분비내과), 윤민재(서울의대 순환기내과)

07:30-07:50 The lower the better: atorvastatin's next option for dyslipidemia management / 168
최강운(영남의대 심장내과)

07:50-08:00 Panel Discussion

Luncheon Symposium 1

4월 6일(토) 12:20-13:20 | Room 1

좌장 : 김덕경(성균관의대 순환기내과)

패널 : 김민지(경북의대 내분비내과), 이상은(이화의대 순환기내과)

12:20-12:40 Individual treatment strategy for dyslipidemia in high risk patients / 170
조준환(중앙의대 순환기내과)

12:40-12:50 Panel Discussion

Luncheon Symposium 2

4월 6일(토) 12:20-13:20 | Room 2

좌장 : 이인규(경북의대 내분비내과)

패널 : 오규철(가톨릭의대 순환기내과), 정경혜(충남의대 내분비내과)

12:20-12:40 A paradigm shift in dyslipidemia treatment: case review / 172
홍준화(을지의대 내분비내과)

12:40-12:50 Panel Discussion

Luncheon Symposium 3

4월 6일(토) 12:20-13:20 | Room 3

좌장 : 장학철(서울의대 내분비내과)

패널 : 김경수(차의대 내분비내과), 이수용(부산의대 순환기내과)

12:20-12:40	**Cutting edge care of pitavastatin with ezetimibe combination therapy** **박경일**(동아의대 순환기내과)	**/ 174**
12:40-12:50	**Panel Discussion**	

Young Investigator Sessions

'지질동맥경화 관련 임상, 기초, 중개, 영양, 운동 등 다양한 분야의 젊은 연구자분들이 이번 SoLA 2024에 본인들이 그 동안 진행했던 우수한 연구 초록을 제출하여 주었습니다. 전체 제출된 초록들 중에 엄격한 심사를 거쳐 좀 더 깊이 있게 발표를 들어볼만한 40세 이하의 젊은 연구자분들의 초록을 선정하여 Young Investigator Session을 마련하였습니다.

Young Investigator Session 1

4월 5일(금) 15:30-17:00 | Room 1

Young Investigator Session 2

4월 5일(금) 15:30-17:00 | Room 2

Young Investigator Session 3

4월 5일(금) 15:30-17:00 | Room 3

Mini-Oral Presentations

전통적인 포스터 전시 및 발표의 형식에서 벗어나 심사를 통과한 모든 초록에 대해서 짧지만 임팩트 있는 Mini 구연의 형식을 SoLA 2024 에서 마련하였습니다. 금, 토 양 일에 4개의 트랙에서 동시에 많은 초록들이 Q&A 포함 각 5분의 시간 동안에 발표될 예정입니다.

Mini-Oral Presentation 1

4월 5일(금) 17:10-18:00 | 행사장 로비 (그랜드볼룸 포이어) | Mini-Oral Presentation A~D

Mini-Oral Presentation 2

4월 6일(토) 11:10-12:20 | 행사장 로비 (그랜드볼룸 포이어) | Mini-Oral Presentation A~D

SoLA 2024

한국지질·동맥경화학회 춘계학술대회

2024 **S**pring Congress **o**n **L**ipid & **A**therosclerosis of KSoLA

Plenary Lecture 1

4월 6일(토) 10:20-11:00 | Room 1, 2, 3

좌장 : 김재택(중앙의대 내분비내과)

10:20-11:00 **Heart-Immune-Brain network in the pathogenesis of cardio-cerebrovascular disease**

오구택(이화여대 생명과학과)

CURRICULUM VITAE

오구택

이화여자대학교 생명과학과

[학력]

1990-1993	서울대학교 수의과대학 박사
1987-1989	서울대학교 수의과대학 석사
1981-1987	서울대학교 수의과대학 학사

[경력]

1995-1997	미국 노스캐롤리나 의과대학 Research Associate Fellow
1988-2004	한국생명공학연구원 연구원(선임, 책임)
2004-현재	이화여자대학교 생명과학과 부교수, 교수
2012-2021	한국연구재단 리더연구자지원사업(창의적연구) 혈관·면역세포 네트워크 연구단 단장
2020-2029	한국연구재단 기초연구사업(리더연구) 심장-면역-뇌 세포 네트워크 연구단 단장

[관심분야]

Myocardial Infarction, Stroke and Inflammation, Atherosclerosis, ROS and mitochondria

[논문]

1. TK Kim, S Jeon, et al. 2'-5' oligoadenylate synthetase-like 1 (OASL1) protects against atherosclerosis by maintaining endothelial nitric oxide synthase mRNA stability. Nat Commun. 2022 Nov 4;13(1):6647
2. S Jeon, et al. Anti-inflammatory actions of soluble Ninjurin-1 ameliorate atherosclerosis. Circulation 2020 Nov 3;142(18):1736-1751
3. TJ Yun, et al. Indoleamine 2,3-Dioxygenase-Expressing Aortic Plasmacytoid Dendritic Cells Protect against Atherosclerosis by Induction of Regulatory T Cells. Cell Metabolism. 2016 May10;23(5):852-866
4. JH Choi, C Cheong, et al. Flt3 signaling-dependent dendritic cells protect against atherosclerosis. Immunity 2011 Nov23;35(5):819-831
5. HJ Jeon, JH Choi, I Jung, et al. CD137(4-1BB) Deficiency Reduces Atherosclerosis in Hyperlipidemic Mice. Circulation 2010 Mar 9;121(9):1124-1133

Heart-Immune-Brain network in the pathogenesis of cardio-cerebrovascular disease

Goo Taeg Oh

Heart-Immune-Brain Network Research Center, Department of Life Science, Ewha Womans University, Korea

This lecture amalgamates insights from the studies to unveil the intricacies of bidirectional interactions within this physiological axis and their profound implications for cardio-cerebrovascular diseases. The research investigates the dynamic interplay between the cardio-immune and neuro-immune systems, unravelling their roles in heart-to-brain and brain-to-heart interactions. Employing a mouse model of myocardial infarction, the study elucidates how cardiac dysfunction instigates changes in the cardio-immune system, subsequently leading to alterations in brain function. Conversely, in a stroke model, the study explores the neuro-immune system's influence on cardiac function, shedding light on connections between stroke-induced macrophage production and cardiac performance. Additionally, the analysis of extracellular vesicles (EVs) secreted in response to cardiac dysfunction provides novel insights into the communication between the heart and brain.

The studies focus on myeloid cell heterogeneity, delineating the transition from local tissue-resident macrophage proliferation to circulating cell recruitment and phenotypic plasticity. Through single-cell RNA sequencing, a distinct microglia type, denoted as stroke-associated microglia (SAM), emerges. SAM exhibits enhanced antioxidant function and unique molecular markers, proving crucial in mitigating reactive oxygen species (ROS) damage during ischemia/reperfusion (I/R) in the brain. The indispensable role of Peroxiredoxin-1 (Prdx1) in SAM's antioxidative capacity is highlighted, influencing ROS defense genes and promoting stroke-protective molecules.

This comprehensive exploration of the heart-immune-brain network provides a paradigm shift in understanding cardio-cerebrovascular diseases. The elucidation of molecular and functional immune cell mechanisms not only facilitates the development of innovative therapeutic strategies but also introduces potential targets for intervention. By unraveling the complexities of this network, this lecture contributes to groundbreaking research and the cultivation of future researchers poised to lead advancements in science and technology.

SoLA 2024

한국지질·동맥경화학회 춘계학술대회

2024 **S**pring Congress **o**n **L**ipid & **A**therosclerosis of KSoLA

Plenary Lecture 2

4월 6일(토) 15:20-16:00 | Room 1, 2, 3

좌장 : 정익모(이화의대 순환기내과)

15:20-16:00 **Cyclase-associated protein 1 (CAP1) binds to Resistin or PCSK9, standing at the nodal point of metabolic diseases**

김효수(서울의대 순환기내과)

CURRICULUM VITAE

김효수

서울의대 순환기내과

[Education]

1987-1994	Ph.D. in Medical Science, Postgraduate School, Seoul National University
1985-1987	Master of Medical Science, Postgraduate School, Seoul National University
1980-1984	M.D., Seoul National University College of Medicine
1978-1980	Premedical Course, College of Liberal Arts & Science, Seoul National University

[Research Interests]

Basic research field: "stem cell biology & its application"

[Recent 5 major papers (with impact factor 〉 10) as the first or corresponding author]

1. Dasom Shin, Soungchan Kim, Hwan Lee, Hyun-Chae Lee, Jaewon Lee, Hyun-woo Park, Mina Fukai, EunByule Choi, Subin Choi, Bon-Jun Koo, Ji-Hoon Yu, Gyurae No, Sungyoon Cho, Chan Woo Kim, Dohyun Han, Hyun-Duk Jang, and Hyo-Soo Kim (corresponding). PCSK9 stimulates Syk, PKCδ, and NF-κB, leading to atherosclerosis progression independently of LDL receptor. Nature Communications 2024 (in press).[IF=16.6]
2. Han-Mo Yang, Joonoh Kim, Baek-Kyung Kim, Hyun Ju Seo, Ju-Young Kim, Joo-Eun Lee, Jaewon Lee, Jihye You, Sooryeonhwa Jin, Sahmin Lee, Yoo-Wook Kwon, Hyun-Duk Jang, Hyo-Soo Kim (corresponding). Resistin Regulates Inflammation and Insulin Resistance in Humans via the Endocannabinoid System. Research 2024 (in press). [IF=11.0]
3. Han-Mo Yang; Joonoh Kim; Hyun-Duk Jang; Dasom Shin; Ju-Young Kim; Jihye You; Hyun-Chae Lee; Sahmin Lee; Hyo-Soo Kim (corresponding). Resistin Impairs Mitochondrial Homeostasis via Cyclase-associated Protein 1-mediated Fission, Leading to Obesity-induced Metabolic Diseases. Metabolism 2023 Jan;138:155343. [IF=13.9]
4. Yoo-Wook Kwon, Jeong-Hwan Chae, Hyo-Soo Kim (corresponding). A subset of macrophages and monocytes in the mouse bone marrow express atypical chemokine receptor-1. Cell Stem Cell, Volume 29, Issue 7, 7 July 2022, Pages 1016-1017. [IF=25.7]
5. Jin-Woo Lee; Jin Hur; Yoo-Wook Kwon; Cheong-Whan Chae; Jae-Il Choi; Injoo Hwang; Ji-Yeon Yun; Jin-A Kang; Young-Eun Choi; Young Hyun Kim; Sang Eun Lee; Cheol Lee; Dong Hyun Jo; Heeyoung Seok; Byong Seung Cho; Sung Hee Baek; Hyo-Soo Kim (corresponding). KAI1(CD82) is a key molecule to control angiogenesis and switch angiogenic milieu to quiescent state. Journal of Hematology & Oncology 2021 [IF=17.34]

Cyclase-associated protein 1 (CAP1) binds to Resistin or PCSK9, standing at the nodal point of metabolic diseases

Hyo-Soo Kim

Strategic Center of Cell & Bio Therapy, Seoul National University Hospital, Seoul, Korea

<Resistin-Cap1 biology>

Resistin is an adipose-secreted cytokine first identified as a mediator of insulin resistance in obese mice. In human, however, peripheral blood mononuclear cells and macrophages are the primary source of resistin. We demonstrated that resistin is a causal factor to aggravate atherosclerosis by stimulating monocytes and inducing vascular inflammation **(J Am College Cardiology 2011)[IF=27.2]**.

We identified adenylyl cyclase-associated protein 1 (CAP1) as a novel functional receptor for human resistin and clarified its intracellular signaling pathway to modulate inflammatory action of monocytes **(Cell Metabolism 2014)[31.4]**. We found that human resistin directly binds to CAP1 in human monocytes to mediate up-regulation of intracellular cAMP concentration, PKA activity and NF-kB related transcription of inflammatory cytokines such as IL-6, TNFα.

Resistin impairs mitochondrial function, leading to obesity-induced metabolic diseases. We analyzed the mitochondrial function in skeletal muscle in two mice model. A high-fat diet in humanized resistin mice increased fragmented and shorter mitochondria in the skeletal muscle, whereas resistin-knockout mice had healthy normal mitochondria. Moreover, our newly developed biomimetic selective blocking peptide could repress human resistin-mediated mitochondrial dysfunction. **(Metabolism 2023 press)[IF=13.9]**

Human Resistin is an Effector of the Endocannabinoid System and Induces Inflammation and Insulin Resistance. In human atheromatous plaques, cannabinoid 1 receptor (CB1R)-positive macrophage was colocalized with the resistin expression. In addition, resistin was exclusively expressed in the sorted CB1R-positive cells from human PBMCs. In CB1R-positive cells, endocannabinoid ligands induced resistin expression via the p38-Sp1 pathway. The regulation of resistin via the CB1R could be a potential therapeutic strategy for cardiovascular diseases by improving obesity-related inflammation and insulin resistance. **(Research 2024 in press)[IF-11.0]**.

<PCSK9-CAP1 biology>

To further substantiate the role of CAP1 in metabolism in vivo, we made TALEN-mediated CAP1 knock-out mouse. CAP1 homogenous knock-out mouse was lethal. The viable heterozygous CAP1 knock-out mice had higher protein levels of LDLR in the liver and lower LDL-C levels in the plasma, than the control mice. Mechanistic analysis revealed that PCSK9-induced endocytosis and lysosomal degradation of LDLR were mediated by caveolin but not by clathrin, which were dependent on binding

between CAP1 and caveolin-1 (**European Heart J 2019)[IF=39.3]**.

Another interesting biology here was uncovered by the observation that PCSK9 induced inflammation even in monocytes from *Ldlr*$^{-/-}$ mice. Systemic administration of AAV-PCSK9 aggravated atherosclerosis of the carotid artery in *Ldlr*$^{-/-}$ mice. We identified the pivotal role of PCSK9 in inducing inflammation and atherosclerosis independently of LDLR. Interaction of PCSK9 and CAP1 followed by activation of the SYK/PKCδ pathway may be a promising therapeutic target for inflammation-mediated disease. (**Nature Communications 2024) [IF=16.6]**

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춘계학술대회
2024 Spring Congress on Lipid & Atherosclerosis of KSoLA

Symposium 1

Recent Hot Trials in Cardio-Metabolic Disorders

4월 6일(토) 08:30-10:00 | Room 1

좌장 : 이상학(연세의대 심장내과), 문민경(서울의대 내분비내과)

패널 : 김병규(인제의대 심장내과), 김진화(조선의대 내분비내과), 윤민재(서울의대 순환기내과)

08:30-08:50	**The role of bempedoic acid in patients with high cardiovascular risk and statin intolerance** **김학령**(서울의대 순환기내과)
08:50-09:10	**Semaglutide: beyond STEP trials, toward obesity & diabetocardiology** **윤종찬**(가톨릭의대 순환기내과)
09:10-09:30	**Lessons from clinical trials of Inclisiran: a first-in-class siRNA therapy against PCSK9** **전재한**(경북의대 내분비내과)
09:30-10:00	**Panel Discussion**

CURRICULUM VITAE

김학령

보라매병원 순환기내과

[학력]

2003	전남의대졸업
2011	성균관대학교 경영학과 석사 졸업
2013	서울대학교의과대학 내과학 박사 졸업

[경력]

2004-2008	서울대병원 내과 전공의
2011-2012	서울대병원 순환기내과 전임의
2012-현재	보라매병원 순환기내과, 현재 부교수

[관심분야]

일차예방요법, 성차의학, 동맥경직도, 사회경제적수준과 심혈관질환

[논문]

1. Association between arterial stiffness and autonomic dysfunction in participants underwent treadmill exercise testing: a cross-sectional analysis. Sci Rep. 2024 Feb 13;14(1):3588.
2. Influence of Socioeconomic Status on the Presence of Obstructive Coronary Artery Disease and Cardiovascular Outcomes in Patients Undergoing Invasive Coronary Angiography Healthcare 2024, 12, 228.
3. Association between invasively measured central aortic pulse pressure and diameter of ascending aorta. Sci Rep 2023;13:21152.
4. The Prognostic Value of Arterial Stiffness According to Socioeconomic Status. J Clin Med 2023 Nov 6;12(21):6943.
5. Prognostic value of brachial-ankle pulse wave velocity according to subjects' clinical characteristics: data from analysis of 10,597 subjects. J Korean Med Sci 2023 Dec 25;38(50):e414.

The role of bempedoic acid in patients with high cardiovascular risk and statin intolerance

Hack-Lyoung Kim

Division of Cardiology, Department of Internal Medicine, Bormae Medical Center,
Seoul National University College of Medicine, Seoul, Korea

Cardiovascular disease (CVD) stands as the foremost cause of mortality globally. Atherosclerosis plays a central role in CVD development, with high levels of low-density lipoprotein cholesterol (LDL-C) being a key factor in its onset and progression. In response, the HMG-CoA reductase inhibitor, known as statins, was developed, demonstrating substantial efficacy in lowering LDL-C and significantly reducing cardiovascular risk. Present guidelines, underscored by robust evidence of statin effectiveness, advocate for the employment of high-intensity statins to diminish LDL-C and cardiovascular risk, particularly among individuals at high risk. Nevertheless, the application of high-intensity statins in real-world settings is often hampered by side effects, including muscle symptoms, hepatotoxicity, and the onset of diabetes mellitus. The low tolerance for statins frequently leads to reduced adherence to therapy, resulting in less than ideal cholesterol management. Ezetimibe, in combination with statins, provides an added LDL-C reduction effect, leading to its increasingly widespread use. Yet, there is a continuing need for more effective and tolerable treatment alternatives. Although PCSK9 inhibitors are significant for their additional LDL-C lowering capabilities, their invasive nature and high cost restrict their broad application. Bempedoic acid, an oral medication, targeting the ATP citrate lyase enzyme crucial in cholesterol synthesis, offers a liver-specific action, thereby avoiding muscle-related side effects. In high-risk cardiovascular disease patients who are on the maximum tolerated statin dosage, adding bempedoic acid as opposed to a placebo has shown to significantly reduce LDL-C levels and the rate of major adverse cardiac events (MACE). Bempedoic acid is particularly beneficial for patients who are unable to tolerate statins due to adverse effects such as muscle pain.

CURRICULUM VITAE

윤종찬

가톨릭의대 서울성모병원 순환기내과

[학력 및 경력]

연세대학교 의과대학 의학과 학사, 석사, 박사
연세대학교 세브란스병원 인턴, 내과 레지던트 수료
연세대학교 세브란스병원 심장내과 강사
KAIST 의과대학원, 면역 및 감염 질환 연구실, Post-Doc Research Fellow
연세대학교 세브란스병원 심장내과 임상조교수
한림대학교 동탄성심병원 순환기내과 부교수
미국 LA Cedars-Sinai Medical Center, Advanced Heart Disease 연수
現 가톨릭대학교 서울성모병원 순환기내과 교수

[가입 학회 및 활동]

대한심장학회 학술간사, 편집위원, 간행위원, 심장종양학연구회 총무위원장, KCJ Assistant Editor,
대한심부전학회 연구이사, 디지털헬스연구회 총무위원장, IJHF Associate Editor, 대한내과학회 순환기분과 간사,
한국지질·동맥경화학회 학술 간사, 대한심뇌혈관질환예방학회 정책기획이사, 학술위원, 심장대사증후군학회 윤리이사,
연구위원, 대한혈관학회 국내교류이사, 대한이식학회 의료심사위원, 세계심폐이식학회, 대한고혈압학회

[수상경력]

2007	연세의대 내과학교실 우수 연구전공의상 수상
2013	대한고혈압학회 최우수 젊은 연구자상 수상
2013	The Taiwan Society of Cardiology, International Young Investigator Award
2015	ISHLT, Transplant Registry Early Career Award
2016	대한심장학회 KCJ 우수심사위원상
2016	Asian Pacific Society of Hypertension Young Investigator Award
2017	대한이식학회 한국아스텔라스 젊은연구자연구비
2017	대한심장학회 심부전연구회 The Best Research Achievement Award
2018	ISHLT, International Travelling Scholarship Award

Semaglutide: beyond STEP trials, toward obesity & diabetocardiology

Jong-Chan Youn

Division of Cardiology, Department of Internal Medicine, Seoul St. Mary's Hospital, College of Medicine, The Catholic University of Korea

Obesity is a chronic, relapsing disease associated with multiple complications and a substantial morbidity, mortality and health care burden. There is a clear need to redouble efforts to target obesity-related cardiometabolic risk as a strategy for combating cardiovascular disease. However, until recently, few pharmacologic tools were available to safely and effectively lower body weight. Medications that include glucagon-like peptide-1 (GLP-1) receptor agonist components have now been shown to produce substantial weight loss - similar to that associated with bariatric surgery - and to lower the risk of cardiovascular disease, specifically in persons with diabetes. Semaglutide was first approved to treat diabetes mellitus and obesity. GLP-1 agonism seems to be particularly beneficial when patients with insulin resistance have low endogenous levels of GLP-1. However, GLP-1 agonism has broad effects - it redistributes fat, decreases inflammation, inhibits glucagon production, and delays gastric emptying. Recently the SELECT trial provides evidence of improved cardiovascular disease outcomes with GLP-1 receptor agonists in the absence of diabetes. For patients with atherosclerotic cardiovascular disease and overweight or obesity, GLP-1 receptor agonist therapy with semaglutide joins the list of established therapies that form the basis of our pharmacologic strategies for reducing the risk of cardiovascular disease. Improvements in cardiometabolic risk factors, including high blood pressure, atherogenic lipids and benefits on physical function and quality of life were seen with semaglutide. The safety profile of semaglutide was consistent across trials, primarily gastrointestinal adverse events. The magnitude of weight loss reported in the STEP trials offers the potential for clinically relevant improvement for individuals with obesity-related diseases.

CURRICULUM VITAE

전재한

경북의대 내분비내과

[학력]

2005 경북의대 의학사
2014 경북의대 내과학 박사

[경력]

2013-2017 경북대학교병원 내분비대사내과 전임의/임상교수
2017-현재 칠곡경북대학교병원 내분비대사내과 조교수/부교수

[관심분야]

Diabetic complications, mitochondria, immunometabolism

[논문]

1. Comprehensive overview of the role of mitochondrial dysfunction in the pathogenesis of acute kidney ischemia-reperfusion injury: a narrative review. J Yeungnam Med Sci. 2024.
2. Mitochondrial dysfunctions in T cells: focus on inflammatory bowel disease. Front Immunol. 2023 Sep 22;14:1219422.
3. Inhibition of pyruvate dehydrogenase kinase 4 ameliorates kidney ischemia-reperfusion injury by reducing succinate accumulation during ischemia and preserving mitochondrial function during reperfusion. Kidney Int. 2023 Oct;104(4):724-739.
4. Upregulation of the ERRγ-VDAC1 axis underlies the molecular pathogenesis of pancreatitis. Proc Natl Acad Sci U S A. 2023 May 16;120(20):e2219644120.
5. Lee H, Jeon JH, Lee YJ, Kim MJ, Kwon WH, Chanda D, Thoudam T, Pagire HS, Pagire SH, Ahn JH, Harris RA, Kim ES, Lee IK. Inhibition of Pyruvate Dehydrogenase Kinase 4 in CD4+ T Cells Ameliorates Intestinal Inflammation. Cell Mol Gastroenterol Hepatol. 2023;15(2):439-461.

Lessons from clinical trials of Inclisiran: a first-in-class siRNA therapy against PCSK9

Jae-Han Jeon

Department of Internal Medicine, School of Medicine, Kyungpook National University

Despite the progress in lipid-lowering treatments, such as statins, a notable number of patients remain unable to reach optimal low-density lipoprotein cholesterol (LDL-C) levels due to reasons like statin intolerance, insufficient response, or severe hypercholesterolemia. This gap in effective management has led to the exploration of new therapeutic avenues, notably targeting the PCSK9 (proprotein convertase subtilisin/kexin type 9) pathway, which plays a vital role in modulating hepatic LDL receptor turnover and cholesterol balance. Inclisiran, a pioneering siRNA (small interfering RNA) therapy, stands out in this new class of drugs by utilizing RNA interference to selectively suppress PCSK9 gene expression in the liver, thus enhancing LDL receptor availability and promoting the removal of LDL-C from the circulation.

The distinct mechanism of action of inclisiran, which allows for prolonged reductions in PCSK9 and LDL-C levels, offers a viable supplement or alternative to conventional lipid-lowering strategies. The efficacy and safety of inclisiran have been thoroughly validated in the ORION clinical trials, which have shown substantial LDL-C reduction with a well-tolerated safety profile and the practicality of bi-annual dosing. These findings emphasize inclisiran's potential to enhance adherence and patient outcomes in managing hypercholesterolemia.

This presentation seeks to detail the biological rationale behind the therapeutic effects of inclisiran, examine the critical data from the ORION trials highlighting its efficacy and safety, and consider its role in the broader context of cardiovascular risk management. Through the introduction of inclisiran, a novel and effective treatment option is now available, marking a significant advance in the effort to alleviate the impact of hypercholesterolemia and, by extension, reduce the global burden of cardiovascular disease. The incorporation of findings from the ORION studies into our understanding of inclisiran's profile underscores its potential as a transformative element in the lipid management landscape.

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Symposium 2

Cutting-Edge Insights in Lipid and Atherosclerosis Research

4월 6일(토) 08:30-10:00 | Room 2

좌장 : 김치대(부산의대 약리학교실), 한진(인제의대 생리학교실)

패널 : 권유욱(서울의대 의생명연구원), 김경진(인하의대 의생명학교실)
김규호(가톨릭의대 내분비내과), 최재훈(한양대 생명과학과)

08:30-08:50	**Dissecting adipose tissue at single-cell resolution during metabolic disease development** **문준호**(서울의대 내분비내과)
08:50-09:10	**Monocyte priming and macrophage reprogramming in atherosclerosis and diet-induced obesity** **안용주**(POSTECH IT융합공학과)
09:10-09:30	**RXX regulates balance of skeletal muscles and adipose tissues** **국현**(전남의대 약리학교실)
09:30-10:00	**Panel Discussion**

CURRICULUM VITAE

문준호

서울의대 분당서울대학교병원 내분비대사내과 조교수

[학력]

2005-2016 서울대학교 의과대학
2016-2020 카이스트 의과학대학원

[경력]

2011-2016 서울대학교병원/분당서울대병원 전공의, 임상강사
2022- 서울의대/분당서울대학교병원 내분비대사내과 조교수

[관심분야]

지방조직, 베타세포

[논문]

1. Moon JH et al. Nonalcoholic fatty liver disease and sarcopenia additively increase mortality: A Korean nationwide survey, J Cachexia Sarcopenia Muscle, 2021, 12: 965-972.
2. Kim MN and Moon JH et al. SGLT2 inhibition reduces cellular senescence in the diabetic kidney by promoting ketone body-induced NRF2 activation, Diabetes Obes Metab, 2021, 23:2561-2571. DOI: 10.1111/dom.14503
3. Moon JH et al. Lactation improves pancreatic β cell mass and function through serotonin production, Sci Transl Med, 2020 Apr 29; 12: eaay0455 (2020). DOI: 10.1126/scitranslmed.aay0455.
4. Moon JH et al. Serotonin regulates adult β cell mass by stimulating perinatal β cell proliferation, Diabetes, 2020 Feb; 69(2): 205-214. DOI: 10.2337/db19-0546.
5. Moon JH et al. Weight Gain and Progression to Type 2 Diabetes in Women with a History of Gestational Diabetes Mellitus, J Clin Endocrinol Metab, 2015, 100(9):3548-3555.

Dissecting adipose tissue at single-cell resolution during metabolic disease development

Joon Ho Moon[1], Jisu Jung[1], Eun-Seo Park[2], Jong-kyung Kim[2], Sung Hee Choi[1]

[1]Seoul National University Bundang Hospital, Seongnam, Gyeonggi-do, South Korea,
[2]POSTECH (Pohang University of Science and Technology), Pohang, Gyeongsangbuk-do, South Korea

Visceral adipose tissue (VAT), with its significant heterogeneity, plays a crucial role in the development of metabolic diseases like obesity and type 2 diabetes. Recent advancements in single-cell RNA technology made it possible to understand the complexity of VAT on pathophysiological changes. However, recent studies have focused on Western populations, primarily Caucasians. In this study, we present a detailed analysis of VAT within the context of metabolic diseases in East Asian, Korean population.

Our comprehensive study categorized individuals into three groups: those with a normal body weight (BMI<23 kg/m^2, n=3), no metabolic disease, individuals with morbid obesity (BMI>30 kg/m^2, n=5), and individuals with obesity and diabetes (BMI>30 kg/m^2, n=5). We used single nuclei analysis to scrutinize a total of 67,391 cells (26,876 from normal-weight subjects, 16,243 from those with morbid obesity, and 24,466 from individuals with obesity and diabetes). Our analysis unveiled notable changes in specific cell types associated with disease progression.

The upregulation of a specific pro-inflammatory macrophage population in obese diabetic patients was observed. Adipose stem cells (ASC) showed distinct cell fate decisions in individuals with obesity and it was further discriminated by the presence of diabetes. Trajectory analysis indicated a divergence in ASC development, with a particular cluster exhibiting fibrotic and pro-inflammatory traits significantly upregulated in both obese and obese diabetic patients.

Furthermore, we observed a specific cluster of adipocytes that was enriched in individuals with obesity and diabetes. This cluster shared fibrotic and pro-inflammatory characteristics observed in ASCs and displayed a suppressed response to insulin signals.

Our investigation of cell-to-cell communication revealed specific clusters, including ASCs, adipocytes, and immune cells, as key participants in intercellular communication. As our next step, we are working to identify the key factors responsible for the development and progression of metabolic diseases in this East Asian population.

CURRICULUM VITAE

안용주

포항공과대학교 융합대학원 의과학, IT융합공학과

[학력]

2000	경희대학교 의학
2006	경희대학교 대학원 약리학 석사
2021	경희대학교 대학원 기초의학과 박사

[경력]

2004-2008	경희의료원 소아청소년과 레지던트
2009-2014	Research Fellow, Brigham and Women's Hospital, Massachusetts General Hospital, USA
2015-2017	Assistant Professor, University of Texas Health Science Center at San Antonio, USA
2017-2022	Assistant Professor, Wake Forest School of Medicine

[관심분야]

The role of monocytes and macrophages in atherosclerosis, Myeloid reprogramming in Diet-induced obesity, Maternal-to-fetal metabolic reprogramming

[논문]

1. Ahn YJ, Wang L, Kim S, Eber MR, Salerno AG, Asmis R. Macrophage-restricted overexpression of glutaredoxin 1 protects against atherosclerosis by preventing nutrient stress-induced macrophage dysfunction and reprogramming. Atherosclerosis. 2023 Dec;387:117383.
2. Ahn YJ, Wang L, Tavakoli S, Nguyen HN, Short JD, Asmis R. Glutaredoxin 1 Controls Monocyte Reprogramming During Nutrient Stress and Protects Mice Against Obesity and Atherosclerosis in a sex-specific manner. Nat. Commun. 2022 Feb 10;13(1):790.

Monocyte priming and macrophage reprogramming in atherosclerosis and diet-induced obesity

Yong Joo Ahn

Convergence IT Engineering, Medical Science Engineering, Pohang University of Science and Technology, Pohang, Korea

Atherosclerosis begins in early life and develops subclinically for decades, and may cause cardiovascular and cerebrovascular diseases in adulthood. Chronic inflammation is a risk factor for later cardiovascular disease. Diet-induced obesity and the development of low-grade inflammation can accelerate the development of atherosclerosis. The nutrient stress-induced reprogramming of blood monocytes gives rise to dysregulated, obesogenic, proatherogenic monocyte-derived macrophages. The primed blood monocytes by a high-calorie diet increased chemotactic activity *in vitro* and *in vivo* in response to metabolic stress gradient. These monocytes and the recruitment of monocyte-derived macrophages into the site of inflammation play a key role in atherogenesis. MKP-1 is a key functional regular of monocyte and macrophage phenotypes and can be used as the biomarker of atherosclerosis. We found that dietary supplemental 23-hydroxy ursolic acid, a phytochemical, protects mice monocytes against nutrient-stressed induced weight gain, adipose tissue inflammation, and atherogenesis.

Oxidative stress plays key roles in monocyte and macrophage biology in atherogenesis, from the formation of early fatty streaks to the development of advanced plaques. Glutaredoxin 1 (Grx1) mediates the deglutathionylation of proteins induced by H2O2. Grx1 protects monocytes from nutrient-stress-induced inactivation of MKP-1, a master regulator of monocyte adhesion and chemotaxis, and macrophage functions and plasticity. This new macrophage-dependent mechanism may contribute to the well-established differences in cardiovascular risk between men and women and to the elevated obesity and cardiovascular risk among postmenopausal women.

CURRICULUM VITAE

국 현

전남의대 약리학교실

[학력]

1992	전남대학교 의과대학 학사
1994	전남대학교 석사 (의학)
1996	전남대학교 박사 (의학, 약리학)

[경력]

1999	일본 교토대학교 제2내과 방문교수 (mentor: Prof. Itoh Hiroshi / Prof. Kazuwa Nakao)
2001	미국 University of Pennsylvania 의대 심장내과 방문교수 (mentor: Jonathan Epstein)
2011	미국 University of Pennsylvania 의대 심장내과 방문교수 (mentor: Jonathan Epstein)

[관심분야]

심혈관 및 골격근육 리모델링의 후생성 조절

[논문]

1. Circular RNA circSMAD4 regulates cardiac fibrosis by targeting miR-671-5p and FGFR2 in cardiac fibroblasts Mol Therapy - Nucleic Acids, 34: 102071, 2023
2. Circular RNA circSmac1-2 regulates vascular calcification by acting as miR-874-3p spongy in vascular smooth muscle clells. Mol Therapy - Nucleic Acids, 27: 645, 2022
3. Regulation of MDM2 E3 ligase-dependent vascular calcification by MSX1/2. Experimental and Molecular Medicine 53: 1181, 2021
4. The roles of non-coding RNAs in vascular calcification and opportunities as therapeutic targets. Pharmacology & Therpeutics 218: 107675, 2021
5. S-nitrosylation of histone deacetylase 2 by neuronal nitric oxide synthase as a mechanism of diastolic dysfunction. Circulation 143: 1912, 2021

RXX regulates balance of skeletal muscles and adipose tissues

Hyun Kook

Department of Pharmacology, Chonnam National University Medical School, Republic of Korea

The RXX, also identified as TRIM-XX, plays multifunctional roles, notably as an oncoprotein and a regulator in various biological processes. Our laboratory has been at the forefront of exploring RXX's involvement in skeletal muscle diseases and its broader biological implications. RXX functions as a pro-tumorigenic gene, integrating into tumor suppressor genes, and displays dual functionality as both a transcription regulator and an E3 ligase. A significant breakthrough came when we identified RXX's interaction with the enhancer of polycomb 1 (EPC1), a transcription activator. This interaction modifies the transcriptional activity of the EPC1-containing protein complex, shedding light on the intricacies of gene expression regulation. Further investigations revealed RXX's high expression in the satellite cells of undifferentiated skeletal muscles and during early muscle development stages, indicating a pivotal role in muscle differentiation. RXX obstructs skeletal muscle differentiation by extracting EPC1 from the serum response factor (SRF), essential for initiating muscle differentiation. Moreover, RXX instigates the degradation of MyoD, a key muscle differentiation protein, in an E3 ligase-dependent manner, underscoring its intricate involvement in muscle physiology. Our research also extended to RXX's impact on adipose tissue, revealing that RXX knockout mice subjected to a high-fat diet exhibited less weight gain and improved metabolic health compared to wild-type counterparts. This was evident in the reduced weight increase in both inguinal and epididymal white adipose tissues, alongside normalized blood glucose levels and improved glucose and insulin tolerance. The generation of adipose-tissue-specific knockout mice further corroborated these findings, with inhibited adipocyte differentiation observed in isolated stromal vascular cells from RXX knockout mice. These studies unveiled RXX's interference with PPAR-γ function, suggesting its role in promoting body fat accumulation by enhancing adipogenic differentiation. This multifaceted research highlights RXX's significant implications in muscle differentiation, adipogenesis, and potential therapeutic targets in related diseases.

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Symposium 3

Dietary Factors and Cardio-Metabolic Disease

4월 6일(토) 08:30-10:00 | Room 3

좌장 : 박용순(한양대 식품영양학과), 김은정(대구가톨릭대 식품영양학과)

패널 : 곽정현(인제대 식품영양학과), 김오연(동아대 식품영양학과), 조동혁(고려의대 순환기내과)

08:30-08:50	**Trust your gut for cholesterol metabolism** **김보경**(부산대 식품영양학과)
08:50-09:10	**Current evidence regarding dietary causes of secondary dyslipidemia** **김민주**(한남대 식품영양학과)
09:10-09:30	**Functional sweeteners and metabolic diseases: effect and mechanism** **김유리**(이화여대 식품영양학과)
09:30-10:00	**Panel Discussion**

CURRICULUM VITAE

김보경

부산대학교 식품영양학과

[학력]

2009.02 부산대학교 식품영양학과 박사
2002.02 부산대학교 식품영양학과 석사
2000.02 부산대학교 식품영양학과 학사

[경력]

2022.03- 부산대학교 식품영양학과 부교수
2018.03-2022.02 부산대학교 식품영양학과 조교수
2017.05-2018.02 전북대학교 식품영양학과 연구원
2016.11-2017.03 연세대학교 내과학교실 연구원
2010.10-2015.05 University of Connecticut Postdoctoral Scholar

[관심분야]

심혈관질환, 비알콜성지방간염, 섬유화, 에너지표현형

[논문]

1. Inhibitory Effects of Ginsenoside Compound K on Lipopolysaccharide-Stimulated Inflammatory Responses in Macrophages by Regulating Sirtuin 1 and Histone Deacetylase 4. H. Kang, S. Kim, J-Y. Lee, B. Kim. Nutrients. 2023 15(7): 1625.
2. Bioactive Compounds as Inhibitors of Inflammation, Oxidative Stress and Metabolic Dysfunctions via Regulation of Cellular Redox Balance and Histone Acetylation State. H. Kang and B. Kim. Foods 2023 12(5): 925.
3. DGKB mediates radioresistance by regulating DGAT1-dependent lipotoxicity in glioblastoma. H. Kang, H. Lee, K. Kim, E. Shin, B. Kim, JH Kang, B. Kim, J. S. Lee, J-M Lee, HS. Youn, BH Youn. Cell Rep Med 17: 4(1):100880.
4. The protective effects of Aster yomena (Kitam.) Honda on high-fat diet-induced obese C57BL/6J mice. M. J. Kim, J. H. Kim, S. Lee, B. Kim, H. Y. Kim. Nutr Res Pract. 2022 16(1): 46.
5. The Effects of Anthocyanin-Rich Bilberry Extract on Transintestinal Cholesterol Excretion. J. Hong, M. Kim, B. Kim. Foods 2021 10(11), 2852.

Trust your gut for cholesterol metabolism

Bohkyung Kim

Department of Food Science and Nutrition, Pusan National University, South Korea

Hypercholesterolemia is one of the primary and modifiable risk factors for cardiovascular disease (CVD). Disrupted cholesterol homeostasis is highly associated with hypercholesterolemia. Cholesterol metabolism is regulated by de novo synthesis, uptake, efflux and excretion. The interplay between the liver and the intestine maintains cholesterol homeostasis. The liver, a primary site for cholesterol metabolism, contributes to de novo cholesterol synthesis, uptake of lipoprotein-derived cholesterol, and conversion of cholesterol to bile acids for biliary cholesterol secretion. Therefore, most of the studies about cholesterol homeostasis have been focused on the liver [4,5]. The absorption of dietary and biliary cholesterol is the primary role of the intestine in cholesterol balance. Cholesterol excretion is critical in human cholesterol metabolism, as there is a lack of the enzymes responsible for the degradation of the cholesterol ring. Classically, hepatobiliary cholesterol excretion mediated by high-density lipoprotein (HDL)-driven reverse cholesterol transport has been accepted as the only way to remove cholesterol from the body. In this pathway, biliary cholesterol is subsequently excreted as fecal-neutral sterols. Transintestinal cholesterol excretion (TICE) is the nonbiliary cholesterol excretion, the second major pathway for cholesterol elimination from the body. In the alternative pathway to cholesterol removal, enterocytes directly uptake circulating lipoprotein-derived cholesterol from plasma for subsequent removal into the intestinal lumen for cholesterol excretion. The underlying mechanisms of TICE remain largely unknown. However, several studies reported that 30-40% of fecal sterol excretion is from TICE in both mice and humans under normal conditions. Recent studies support that the role of the intestine in cholesterol net balance has been underestimated. Therefore, dynamics of the alteration of intestinal cholesterol metabolism provide attractive intestine-specific nutritional strategies to lower hypercholesterolemia for protection against CVD. The recent insights into intestinal cholesterol metabolism and stimulation of TICE through nutrition will be reviewed and discussed.

CURRICULUM VITAE

김민주

한남대학교 식품영양학과

[학력]

2010.02 연세대학교 식품영양학과 이학사
2014.08 연세대학교 식품영양학과 이학박사

[경력]

2024.04-현재 한남대학교 식품영양학과 부교수
2020.03-2024.03 한남대학교 식품영양학과 조교수
2017.04-2020.02 연세대학교 심바이오틱라이프텍연구원 노화과학연구센터 연구교수

[관심분야]

- Clinical Nutrition (Personalized Nutrition)
- Medical Nutrition Therapy in Metabolic Diseases (diabetes mellitus, obesity, coronary vascular disease, dyslipidemia, hypertension, metabolic syndrome, liver disease, etc.)
- Omics: Metabolomics (Nutrition-Metabolite interaction), Genomics (Nutrigenetics and Nutrigenomics)
- Lipid Metabolism
- Oxidative Stress
- Nutrition and Aging
- Age and Disease-Related (Early) Biomarkers
- Disease Early Prediction
- Clinical Trials
- Nutritional Support or Supplementations
- Epidemiological Study: Cohort Study

[논문]

1. Cho D, Huang X, Han Y, Kim M. NPC1L1 rs217434 A>G as a Novel Single Nucleotide Polymorphism Related to Dyslipidemia in a Korean Population. Biochem Genet. 2024 Jan. DOI: 10.1007/s10528-023-10649-6. (corresponding author)
2. Han Y, Yoo HJ, Kim Y, Huang X, Lee JH, Kim M. Changes in Lp-PLA2 are associated with elevated alanine aminotransferase levels: a nested case-control study in a three-year prospective cohort. Scr Med. 54(4):353-361, 2023. (corresponding author)
3. Han Y, Jang K, Kim U, Huang X, Kim M. The possible effect of dietary fiber intake on the metabolic patterns of dyslipidemia subjects: Cross-sectional research using nontargeted metabolomics. J Nutr. 153(9):2552-2560, 2023. (corresponding author)
4. Huang X, Han Y, Jang K, Kim M. Early prediction for prediabetes and type 2 diabetes using the genetic risk score and oxidative stress score. Antioxidants. 11(6):1196, 2022. (corresponding author)
5. Lee S-Y, Kim TY, Hong JY, Kim GJ, Oh J, Kim M, Apostolidis E, Lee J-Y, Kwon Y-I. Anti-Obesity and Anti-Adipogenic Effects of Administration of Arginyl-Fructose-Enriched Jeju Barley (Hordeum vulgare L.) Extract in C57BL/6 Mice and in 3T3-L1 Preadipocytes Models. Molecules. 27(10):3248, 2022.

Current evidence regarding dietary causes of secondary dyslipidemia

Minjoo Kim

Department of Food and Nutrition, College of Life Science and Nano Technology, Hannam University, Daejeon, Korea

Dyslipidemia is a global issue with an increasing prevalence, including in South Korea. Dyslipidemia can be classified into primary and secondary forms, with secondary dyslipidemia accounting for approximately 30-40 percent of all cases. There is limited research and information available on specific details, but poor dietary habits are recognized as a significant contributing factor. This type of disease is primarily caused by unhealthy lifestyle choices and acquired medical conditions.

There are limited studies investigating secondary dyslipidemia that have been conducted over a long period of time. As a result of the limitations inherent in cross-sectional or case-control studies, existing evidence is inconclusive. Randomized controlled trials cannot establish causation as they assess how dietary interventions can manage dyslipidemia rather than how it develops due to nutritional factors.

In addition, there is a lack of research specifically focused on the Korean population. In response to this, healthcare practitioners, including medical practitioners and clinical nutritionists, tend to emphasize disease management more than prevention. It is possible, however, to significantly reduce dyslipidemia prevalence and effectively manage blood lipid profiles by identifying and managing the dietary causes before they develop.

In order to address the issue of dyslipidemia, we intend to offer an overview of the dietary factors that contribute to this condition, including nutrients, food choices, and meal patterns, based on the existing research. Furthermore, we will provide a brief summary of expert consensus on effective strategies for managing cases of dyslipidemia.

CURRICULUM VITAE

김유리

이화여자대학교 식품영양학과

[학력]

1992	이화여자대학교 식품영양학과, 학사
1999	The Ohio State University, Human Nutrition, 석사
2005	Tufts University, Nutritional Biochemistry and Metabolism, 박사

[경력]

현재	이화여자대학교 식품영양학과, 교수
현재	이화여자대학교 글로벌미래평생교육원장, 이화리더십개발원장, 문화예술교육원장
2017-2018	Yale University, School of Medicine, 교환교수

[관심분야]

여러 기능성 물질 (비타민 A, 감미료, phytochemicals)의 항암, 항비만, 항당뇨 효과 및 분자적 기전

[논문]

1. Y. Kim, Y. Oh, Y.S. Kim, J-H Shin, Y.S. Lee, Y. Kim (2024) β-carotene attenuates muscle wasting in cancer cachexia by regulating myogenesis and muscle atrophy Oncology Rep 51:9
2. Y Kim, H. Han, Y. Oh, H. Shin, G. Park, S. Park, J.A. Manthey, Y. Kim, Y. Kim (2024) A combination of rebaudoside A and neohesperidin dihydrochalcone suppressed weight gain by regulating visceral fat and heaptic lipid metabolism in ob/ob mce. Food Sci Biotechnol 33:913-923
3. M Kwon, Y Kim, J Lee, J.A. Manthey, Y Kim, Y Kim (2022) Neohesperidin dihydrochalcone and neohesperidin dihydrochalcone-O-glycoside attenuate subcutaneous fat and lipid accumulation by regulating PI3K/AKT/mTOR pathway in vivo and in vitro. Nutrients 14:1087
4. NY Kim, Y. Kim, YS Kim, J-H Shin, LP Rubin, Y Kim. (2020) β-carotene exerts anti-colon cancer effects by regulating M2 macrophage and activated fibroblasts. J Nutr Biochem 82:108402
5. E Kim, Y Kim, J Lee, J-H Shin, PR Seo, Y Kim (2020) Leucrose, a natural surcrose isomer, suppresses dextran sulfate sodium (DSS)-induced colitis in mice by regulating macrophage polarization via JAK1/STAT6 signaling. J Funct Foods 74:104156

Functional sweeteners and metabolic diseases: effect and mechanism

Yuri Kim

Dept of Nutritional Science and Food Management, Ewha Womans University, Republic of Korea

Metabolic disorders are diseases that disrupt normal metabolism and include obesity, dyslipidemia, type 2 diabetes mellitus (T2DM), and non-alcoholic fatty liver diseases (NAFLD). Type 2 diabetes is characterized as a metabolic disorder involving glucose and fat metabolism that are compromised by hyperglycemia and insulin resistance. Patients with type 2 diabetes also have a higher risk of atherosclerotic cardiovascular diseases due to dyslipidemia. The liver is largely responsible for metabolizing lipids, and dysregulation of lipid metabolism can lead to lipid accumulation and the development of metabolic disorders, such as obesity, diabetes, and hyperlipidemia. In addition, obesity is a major risk factor for these diseases. Evidence shows the association between overconsumption of added sugars with the development of insulin resistance caused by dysregulation of insulin action, dyslipidemia, and fat accumulation in adipocytes and the liver. Sugar reduction strategies often rely on the use of alternative sugars that are designed to substitute sugar and mimic its sensory profile, but also exert beneficial effects on obesity-related metabolic disorders. Consequently, numerous studies have been conducted to develop new types of sugars and sugar substitutes that have nutritional and beneficial effects that can help manage metabolic diseases. Xylobiose (XB) is a dimer of D-xylose and is a major component of xylooligosaccharides (XOS). The beneficial effects reported for XOS have included an ability to revitalize the growth of intestinal bifidobacteria, to activate the immune system, and to suppress colon cancer. Phylloduclin is one of isocoumarin derivatives and a well-known natural sweetener. It is known to be 400-800 times sweeter than sucrose. The beneficial effects reported for phyllodulcin have included an ability for anti-fungal, anti-ulcer, and anti-diabetic effects. Neohesperidin dihydrochalcone (NHDC) is one of intense sweeteners. It is extracted and processed from hesperidin, its parent flavanone. The relative sweetness of NHDC is 250-2000 times higher than sucrose solution. We reported anti-diabetic and anti-obesity effects of these sweeteners by regulating hepatic and adipocytic metabolism. Results from these studies suggested that various sweeteners could be potential functional sweeteners for preventing diabetic and obesity-related metabolic disorders.

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Symposium 4
(KSoLA-KDA-KSSO Joint Symposium)

Multidisciplinary Approach to Prevent Cardiovascular Disease

4월 6일(토) 13:30-15:00 | Room 1

좌장 : 박철영(성균관의대 내분비내과), 이우제(울산의대 내분비내과)

패널 : 손장원(가톨릭의대 내분비내과), 이상은(이화의대 순환기내과)
진상만(성균관의대 내분비내과)

13:30-13:50	KSoLA - Deciphering lipid enigmas: exploring TRL, triglycerides, and remnant cholesterol as residual lipid risks and treatment targets **김병진**(성균관의대 순환기내과)
13:50-14:10	KDA - Role of continuous glucose monitoring in diabetic patients at high cardiovascular risk **이준엽**(가톨릭의대 내분비내과)
14:10-14:30	KSSO - CVOTs using non-GLP-1 based anti-obesity medications and bariatric surgery **홍준화**(을지의대 내분비내과)
14:30-15:00	Panel Discussion

CURRICULUM VITAE

김병진

성균관의대 강북삼성병원 순환기내과

[학력]

1993년, 1995년, 2003년	부산대학교 의과대학 학사 석사 박사
2001년-2003년	성균관의대 삼성서울병원 순환기내과 임상강사
2003년, 2008년, 2014년	성균관의대 강북삼성병원 순환기내과 조교수, 부교수, 정교수

[경력]

2007년-2008년	UCSD, Preventive Medicine and Epidemiology 연수
2023년-	심장대사증후군학회 학술이사
2021년-	한국지질·동맥경화학회 교육이사

[관심분야]

Dyslipidemia-Residual Risk, Lipoprotein(a), Hypertension, Primary Prevention, Lifestyle Modification

[논문]

1. Lipoprotein(a)-related cardiovascular and all-cause mortalities in Korean adults. Eur J Prev cardiol 2023.
2. Comparison of Office Blood Pressure, Automated Unattended Office Blood Pressure, Home Blood Pressure, and 24-Hour Ambulatory Blood Pressure Measurements. J Korean Med Sci 2023.
3. Association between Low-Density Lipoprotein Cholesterol Level and Cardiovascular Outcomes in Korean Adults: A Nationwide Cohort Study. Diabetes Metab J 2023.
4. Association of environmental tobacco smoke exposure with metabolic syndrome: A longitudinal Cohort Study of 71,055 never smokers. Nutr Metab Cardiovasc Dis 2022.
5. 2022 Consensus statement on the management of familial hypercholesterolemia in Korea. Korean J Intern Med 2022.

KSoLA - Deciphering lipid enigmas: exploring TRL, triglycerides, and remnant cholesterol as residual lipid risks and treatment targets

Byung Jin Kim

Division of Cardiology, Department of Internal Medicine,
Kangbuk Samsung Hospital, Sungkyunkwan University School of Medicine, Korea

Lipoprotein particles in the blood can be broadly classified into six types: chylomicron, VLDL, IDL, LDL, and lipoprotein(a), except HDL, which contains one apolipoprotein B (apoB) per particle and is referred to as "ApoB-containing lipoprotein". LDLC has traditionally been the primary lipid risk factor for atherosclerotic cardiovascular disease (ASCVD). However, recent studies have highlighted the role of triglycerides and remnant cholesterol in addition to LDLC. Triglycerides (TGs)-rich lipoprotein (TRL) refers to lipoprotein particles with high TGs content. The cholesterol present in TRL particles is called TRL-cholesterol. Remnant cholesterol refers to the cholesterol in TRL-remnant particles, excluding chylomicrons and very large VLDL. However, it is typically considered to be the cholesterol in VLDL and IDL particles. In clinical practice, remnant cholesterol can be calculated by subtracting HDLC and LDLC from total cholesterol or by using the Friedewald formula to calculate TGs divided by 5 if TGs are not high.

Previous epidemiological and Mendelian randomization studies have shown that TGs and remnant cholesterol are risk factors for ASCVD morbidity and mortality, suggesting that they are as important as LDLC in terms of residual risk.

However, there is still debate regarding whether TGs can serve as a therapeutic target in patients at high risk of ASCVD. Currently, it remains unclear whether fibrates and omega-3 fatty acids are effective in reducing the risk of ASCVD events. This uncertainty may be due to differences in the study population and control groups, drug type and dose, statin use, and outcomes on other lipid markers in recent randomized clinical trials. It is generally suggested that treatment aimed at lowering TGs with fibrates or omega-3 fatty acids act on lipoprotein particle remodeling rather than clearance. Therefore, we need to see the results of recent major trials of agents targeting apoCIII or ANGPTL3.

This lecture will cover what TRL and remnant cholesterol are and their metabolism and impact on ASCVD. It will also comment on the relevance of TGs as a marker of cardiovascular risk and as a therapeutic target based on the results of recent studies.

CURRICULUM VITAE

이준엽

가톨릭의과대학 서울성모병원 내분비내과

[학력]

2006-2012	가톨릭의과대학 학사
2015-2017	가톨릭의과대학 내과학 석사
2017-2021	한국과학기술원 의과학대학원 박사

[경력]

2012-2013	가톨릭중앙의료원 수련의
2013-2017	가톨릭중앙의료원 전공의
2017-2021	한국과학기술원 의과학대학원
2021-2023	서울성모병원 내분비내과 전임의
2023-2024	서울성모병원 내분비내과 임상진료조교수
2024-	서울성모병원 내분비내과 조교수

[관심분야]

췌도부전, 디지털 헬스케어, 비만

[논문]

1. Prediction of Cardiovascular Complication in Patients with Newly Diagnosed Type 2 Diabetes Using an XGBoost/GRU-ODE-Bayes-Based Machine-Learning Algorithm. Endocrinol Metab (Seoul) 2023; doi: 10.3803/EnM.2023.1739
2. Multiparity increases the risk of diabetes by impairing the proliferative capacity of pancreatic β cells. Experimental & Molecular Medicine 2023;55:2269-2280.
3. Risk of Cause-Specific Mortality across Glucose Spectrum in Elderly People: A Nationwide Population-Based Cohort Study. Endocrinol Metab (Seoul) 2023; doi: 10.3803/EnM.2023.1765
4. Opening the Precision Diabetes Care through Digital Healthcare. Diabetes Metab J 2023; doi: 10.4093/dmj.2022.0386
5. PRMT1 Is Required for the Maintenance of Mature β-Cell Identity. Diabetes. 2020;69(3):355-68.

KDA - Role of continuous glucose monitoring in diabetic patients at high cardiovascular risk

Joonyub Lee

Division of Endocrinology and Metabolism, Department of Internal Medicine, Seoul St. Mary's Hospital, College of Medicine, The Catholic University of Korea, Seoul, Republic of Korea

Glycemic homeostasis is an important factor in the development and prognosis of cardiovascular diseases. Patients with diabetes are at an increased risk of cardiovascular disease, which can often lead to fatal outcomes. Fluctuations in blood glucose levels, including hyperglycemia, hypoglycemia, and glycemic variability, are associated with adverse cardiovascular outcomes. Continuous Glucose Monitoring (CGM) systems, as wearable devices, provide real-time, high-quality glycemic data, offering a novel approach to managing diabetes. The adoption of CGM among diabetic patients has rapidly increased recently, driven by its potential to significantly reduce hyperglycemia, hypoglycemic episodes, and glycemic variability. Despite the growing evidence supporting CGM's benefits in glycemic management, its impact on diabetic patients with a high risk of cardiovascular events remains to be fully elucidated. This talk aims to explore the latest evidence on the effectiveness of CGM in managing patients with diabetes who are at high cardiovascular risk. By examining recent clinical studies and outcomes, we will discuss whether CGM can offer benefits in reducing cardiovascular risk among this high-risk patient population.

CURRICULUM VITAE

홍준화

대전을지대학교병원 부교수

[학력]

2004	을지의대 학사
2008	을지의대 석사
2015	을지의대 내과 박사

[경력]

2014	충남대학교병원 전임의
2016	경북대학교병원 임상교수
현재	대전을지대학교병원 부교수

[관심분야]

비만, 당뇨병, 이상지질혈증, 갑상선, 골다공증, 부신

[논문]

1. Comparison of therapeutic efficacy and safety of sitagliptin, dapagliflozin, or lobeglitazone adjunct therapy in patients with type 2 diabetes mellitus inadequately controlled on sulfonylurea and metformin: third agent study. Diabetes Res Clin Pract. 2023 Aug 11;110872. doi: 10.1016/j.diabres.2023.110872.
2. Comparison of the effects of gemigliptin versus glimepiride on cardiac function in patients with type 2 diabetes uncontrolled with metformin: The gemi-heart study. Diabetes Obes Metab. 2023 Aug;25(8):2181-2190. doi: 10.1111/dom.15095. Epub 2023 May 3.
3. A randomized, active-controlled, parallel, open-label, multicenter, phase 4 study to compare the efficacy and safety of pregabalin sustained release tablet and pregabalin immediate release capsule in type II diabetic patients with peripheral neuropathic pain. Medicine (Baltimore). 2023 Apr 25;102(17):e33701.
4. Effects of Virtual Reality Exercise Program on Blood Glucose, Body Composition, and Exercise Immersion in Patients with Type 2 Diabetes. Int. J. Environ. Res. Public Health 2023, 20(5), 4178.
5. SGLT-2 inhibitors and GLP-1 receptor agonists in metabolic dysfunction-associated fatty liver disease: Trends Endocrinol Metab.

KSSO - CVOTs using non-GLP-1 based anti-obesity medications and bariatric surgery

Jun Hwa Hong

Division of Endocrinology, Internal medicine, Daejeon Eulji Medical Center, Eulji University, Daejeon, Korea

심혈관 질환(CVD)은 전 세계적으로 주요 사망 원인이며, 세계보건기구에 따르면 2016년 약 1,800만 명의 환자가 사망했습니다. 여러 원인이 심혈관 사망률에 기여하지만 비만을 근간으로 하는 다양한 질환의 영향이 큽니다. 비만 유병률이 지난 4년간 3배 증가했습니다. 비만은 제2형 당뇨병, 고혈압, 이상지질혈증의 심혈관 위험요소의 변화를 초래하고, 더 나아가 심혈관 질환 및 뇌혈관 질환, 악성 종양의 발생과 연관성이 높다. 하지만, 현재는 퇴출되었던 비만 약제들은, 체중을 감량하지만 심혈관 위험도를 높이거나, 악성 종양의 발생 위험성이 있어 시장에서 퇴출되었다. 비만과 다양한 만성 질환 및 심뇌혈관 질환과의 연관성도 중요하지만, 비만 약제의 체중 감량 효과에 따른 안정성에도 관심이 높아지고 있습니다.

현재 국내에서 사용하고 있는 Liriglutide 3.0 mg은 심혈관 위험도에 대한 근거 자료를 동반하고 있으며, 아직 국내에 출시되지는 않았지만 Semaglutide는 이미 심혈관 위험도를 낮추는 임상 결과를 보유하고 있다. GLP1을 근간으로 하는 항비만 약제들은 심혈관 위험도에 대한 다양한 연구들이 진행되거나 계획되고 있어서, 긍정적인 결과를 기대할 수 있습니다.

하지만 국내에서 사용하고 있는 GLP1 근간이 아닌 항비만 약제들은 대규모 심혈관질환 발생에 대한 전향적인 연구들이 부족하고, 지금 시점에서 대규모 연구를 진행하기에는 한계가 있습니다.

따라서, 몇몇 관련된 연구들의 자료를 바탕으로 Non-GLP1 항비만 약제와 대사 수술에 따른 심혈관 질환과의 연관성을 유추해 보고자 합니다.

SoLA 2024

한국지질·동맥경화학회 춘계학술대회

2024 **S**pring Congress **o**n **L**ipid & **A**therosclerosis of KSoLA

Symposium 5

Ferroptosis & Vascular Inflammation

4월 6일(토) 13:30-15:00 | Room 2

좌장 : 박용식(경희의대 미생물학교실), 강은석(연세의대 내분비내과)

패널 : 김영국(전남의대 생화학교실), 최동욱(고려대 생명공학부)
한주희(우석대 약학과), 허경선(충남대 약학과)

13:30-13:50	**Role of metabolic reprogramming in chorioretinal diseases** **박동호**(경북의대 안과)
13:50-14:10	**Roles of E3 ligases involved in NASH development** **송재환**(연세대 생화학과)
14:10-14:30	**Potential roles of ferroptosis in atherosclerosis** **이은우**(한국생명공학연구원 대사제어연구센터)
14:30-15:00	**Panel Discussion**

CURRICULUM VITAE

박동호

경북대학교 의과대학 안과학교실

[학력]

2018.02	Massachusetts Eye and Ear Infirmary, Harvard Medical School, US, Postdoc fellowship
2011.08	경북대학교 의과대학 안과학교실, Ph.D.

[경력]

2023-	경북대학교병원 진료협력센터장
2022-	경북대학교 의과대학 기획실장, 한국망막학회 회무이사,
2021-	경북대학교 의과대학 안과학교실 교수

[관심분야]

미토콘드리아 대사 제어에 의한 맥락막망막질환 치료

[논문]

1. Kim SY, Yoon NG, Im JY, Lee JY, Kim JH, Jeon YJ, Choi YJ, Lee JH, Uemura A, Park DH, Kang BH. Targeting the mitochondrial chaperone TRAP1 alleviates vascular pathologies in ischemic retinopathy, Advanced Science, 2024, 11(2):1-16
2. Do JR, Park SJ, Kim JY, Shin JP, Park DH. Risk Factors for Pupillary Optic Capture Following Sutureless Flanged Intraocular Lens Fixation for Intraocular Lens Dislocation. Retina, 2023, 43(6):964-971
3. Park SJ, Hwang JM, Park YJ, Shin JP, Park DH. Comparison of Surgeon Muscular Properties between Standard Operating Microscope and Digitally Assisted Vitreoretinal Surgery Systems. Retina. 2022, 42(8), 1583-1591
4. Sim H, Lee W, Choo S, Park EK, Baek MC, Lee IK, Park DH (co-corresponding author), Bae JS. Sulforaphane Alleviates Particulate Matter-Induced Oxidative Stress in Human Retinal Pigment Epithelial Cells Frontiers in Medicine. 2021, 8:1-9
5. Kim JH, Kim JH, Do JY, Lee JY, Yanai R, Lee IK, Suk K, Park DH. Key Role of Microglial Matrix Metalloproteinases in Choroidal Neovascularization. Frontiers in Cellular Neuroscience 2021/2/26. 15: 1-10

Role of metabolic reprogramming in chorioretinal diseases

Dong Ho Park

Department of Ophthalmology, School of Medicine, Kyungpook National University, Republic of Korea

Purpose

To assess the therapeutic effects of the small compounds on choroidal neovascularization (CNV) by its modulation of inflammation and metabolic reprogramming in the retinal pigment epithelium (RPE).

Methods

The anti-angiogenic effects of the small compounds were assessed by measuring vascular leakage and CNV lesion size in the laser-induced CNV mouse model. Inflammatory responses were evaluated by qPCR, Western blot, and ELISA in both CNV eye tissues and primary human RPE (hRPE) cells under inflammatory cytokine mixture (ICM) treatment or hypoxia. Mitochondrial respiration was assessed by measuring oxygen consumption in primary hRPE cells treated with ICM±the drug.

Results

In laser-induced CNV, the small compounds significantly decreased vascular leakage and lesion size, together with choroidal and retinal inflammatory cytokines, including *Il-1β*, *Il-6*, *Il-8*, and *Tnf-α*. Furthermore, the small compounds decreased proinflammatory cytokine secretion in ICM-treated primary hRPE cells. Interestingly, the small compounds significantly enhanced mitochondrial respiration in the ICM-treated primary hRPE cells.

Conclusions

Our findings show that the small compounds are a viable putative therapeutic for neovascular AMD by modulating the inflammatory response and metabolic reprogramming by enhancing mitochondrial respiration in the RPE.

CURRICULUM VITAE

송재환

연세대학교 생화학과

[학력]

1993	연세대학교 생화학과 학사
1995	연세대학교 생화학과 석사
2000	Northwestren University, USA 박사

[경력]

2000-2002	Northwestern University, Post-Doctoral Fellow
2002-2010	성균관대학교 생명공학과 조/부교수
2010-Present	연세대학교 생화학과 부/정교수

[관심분야]

세포사멸, 암, 물질대사

[논문]

1. Choosing-Sil Lee, Gyuho Hwang, Young Woo Nam, Chi Hyun Hwangm, Jaewhan Song. IKK-mediated TRAF6 and RIPK1 interaction stifles cell death complex assembly leading to the suppression of TNF-α-induced cell death (2023) Cell Death Differ. 2023 Jun:30(6): 1575-1584.
2. Jinho Seo, Young Woo Nam, Seongmi Kim, Doo-Byoung Oh & Jaewhan Song, Necroptosis molecular mechanisms: Recent findings regarding novel necroptosis regulators (2021) Experimental & Molecular Medicine volume 53, pages1007-1017.
3. Jinho Seo, Daehyeon Seong, Young Woo Nam, Chi Hyun Hwang, Seung Ri Lee, Choong-Sil Lee, Young Jin, Han-Woong Lee, Doo-Byoung Oh, Peter Vandenabeele, Jaewhan Song, Beclin 1 functions as a negative modulator of MLKL oligomerisation by integrating into the necrosome complex, Cell Death & Differentiation (2020) 27:3065-3081.
4. Daehyeon Seong, Manhyung Jeong, Jinho Seo, Ji-Yoon Lee, Chi Hyun Hwang, Ho-Chul Shin, Jeong Yoon Shin, Young Woo Nam, Jeong Yeon Jo, Haeseung Lee, Hye-Jung Kim, Hwa-Ryeon Kim, Ji Hoon Oh, Sang-Jun Ha, Seung Jun Kim, Jae-Seok Roe, Wankyu Kim, June-Won Cheong, Kwang-Hee Bae, Sang Chul Lee, Andrew Oberst, Peter Vandenabeele, Dong Hoon Shin, Eun-Woo Lee, and Jaewhan Song, Identification of MYC as an antinecroptotic protein that stifles RIPK1-RIPK3 complex formation, PNAS August 18, 2020 117 (33) 19982-19993.
5. Soyeon Shin, Kyungeun Kim, Hwa-Ryeon Kim, Kris Ylaya, Sung-Im Do, Stephen M. Hewitt, Hee-Sae Park, Jae-Seok Roe, Joon-Yong Chung & Jaewhan Song, Deubiquitylation and stabilization of Notch1 intracellular domain by ubiquitin-specific protease 8 enhance tumorigenesis in breast cancer, Cell Death & Differentiation volume 27, pages1341-1354(2020).

Roles of E3 ligases involved in NASH development

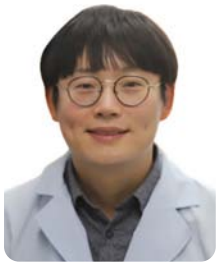
Jaewhan Song

Department of Biochemistry, Yonsei University

Non-alcoholic fatty liver disease (NAFLD) comprises a spectrum of diseases in the liver with steatosis without substantial alcohol consumption or competing etiologies for the hepatic steatosis. NAFLD symptoms could be typically subdivided into non-alcoholic fatty liver (NAFL) and non-alcoholic steatohepatitis (NASH) depending on disease severity. NASH, the severe form of NAFLD, is usually diagnosed when there is more than 5% hepatic steatosis and inflammation with hepatocellular injury in the presence or absence of fibrosis. Currently, the NASH creates a great clinical and economic burden on healthcare systems worldwide due to hepatic and extrahepatic comorbidity and liver transplant. Here we report E3 ligases involved in NASH progression and discuss how the regulation of these enzymes could lead to regression of NASH.

CURRICULUM VITAE

이은우



한국생명공학연구원 대사제어연구센터

[학력]

2007-2011 박사, 성균관대학교 식품생명공학과
2005-2007 석사, 성균관대학교 식품생명공학과
2000-2005 학사, 성균관대학교 식품생명공학과

[경력]

2021-현재 부교수, 과학기술연합대학원대학교 기능유전체학과
2016-현재 전임연구원/선임연구원, 한국생명공학연구원 대사제어연구센터
2011-2016 박사후연구원, 연세대학교 생화학과

[관심분야]

Ferroptosis, Lipid Metabolism, ROS, Senescence, Athersclerosis

[논문]

1. Oh M. et al., Darapladib, an inhibitor of Lp-PLA2, sensitizes cancer cells to ferroptosis by remodeling lipid metabolism. Nat Commun. 14(1):5728 (2023).
2. Kim J. et al., FSP1 confers ferroptosis resistance in KEAP1 mutant non-small cell lung carcinoma in NRF2-dependent and -independent manner. Cell Death Dis, 14(8):567 (2023).
3. Kim J. et al., An integrated view of lipid metabolism in ferroptosis revisited via lipidomic analysis. Exp Mol Med. 55(8):1620-1631 (2023).
4. Lee JY. et al., Polyunsaturated fatty acid biosynthesis pathway determines ferroptosis sensitivity in gastric cancer. PNAS, 117(51): 32433-32442 (2020).
5. Park TJ. et al., Quantitative proteomic analyses reveal that GPX4 downregulation during myocardial infarction contributes to ferroptosis in cardiomyocytes. Cell Death Dis. 10, 835 (2019).

Potential roles of ferroptosis in atherosclerosis

Eun-Woo Lee

Metabolic Regulation Research Center, Korea Research Institute of Bioscience & Biotechnology (KRIBB), Korea

Ferroptosis is a form of regulated cell death that relies on iron and is triggered by lipid peroxidation of polyunsaturated fatty acids (PUFAs) in membrane phospholipids. Extensive in vitro and in vivo studies have underscored the crucial roles of ferroptosis in various human diseases, including neurological disorders, ischemia-reperfusion injury, kidney damage, and blood disorders. Notably, cancer cells, particularly those resistant to chemotherapy, are highly susceptible to ferroptosis inducers, presenting a promising avenue for anti-cancer therapy. We previously reported that arachidonic acid (AA; C20:4) and adrenic acid (C22:4) biosynthesis from linoleic acid (C18:2), the predominant fatty acid in the blood, by fatty acid desaturases (FADSs) and elongases (ELOVLs) is critical for ferroptosis sensitivity. I will introduce that the cell-autonomous recycling of AA facilitated by lipoprotein-associated phospholipase A2 (Lp-PLA2) is a rapid mechanism influencing the abundance of AA-containing phosphatidylethanolamine (PE) species, contributing to ferroptosis resistance. Inhibition of Lp-PLA2 with darapladib, which failed in phase III clinical trials for acute and chronic coronary diseases, can sensitize cells to ferroptosis both in vitro and in vivo, offering a novel therapeutic strategy for enhancing ferroptosis in cancer treatment. Additionally, I will explore the initiation and propagation of lipid peroxidation to the plasma membrane, causing membrane damage and ferroptosis. Our recent findings indicate that a specific protein translocates to the plasma membrane within 15 minutes of ferroptotic stimuli. Cells lacking this protein exhibit lipid peroxidation signals solely in the cytoplasm or subcellular organelles, not at the plasma membrane, resulting in ferroptosis resistance. Finally, I will propose that inhibiting ferroptosis by targeting this protein or using a lipophilic antioxidant could represent a potential therapeutic approach for atherosclerosis.

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2024 **S**pring Congress **o**n **L**ipid & **A**therosclerosis of KSoLA

Symposium 6

Current Status and Perspectives in Nutrition Management in Cardiovascular Disease

4월 6일(토) 13:30-15:00 | Room 3

좌장 : 김은미(강북삼성병원 영양팀), 김오연(동아대 식품영양학과)

패널 : 백진경(을지대 식품영양학과), 전지은(경희의대 내분비내과), 최미옥(동아대 식품영양학과)

13:30-13:50	**Nutrition therapy strategy for cardiovascular management** **강현희**(창원경상대병원 영양팀)
13:50-14:10	**Impact of lifestyle modifications in preventing and managing cardiovascular diseases** **임현정**(경희대 의학영양학과)
14:10-14:30	**Role of probiotics in cardiovascular risk management** **이보경**(동아대 식품영양학과)
14:30-15:00	**Panel Discussion**

CURRICULUM VITAE

강현희

창원경상국립대학교병원 영양팀

[학력]

1997년 2월	경상대학교 식품영양학과 학사 졸업
1999년 2월	경상대학교 식품영양학과 석사 졸업
2009년 8월	경상대학교 식품영양학과 박사 졸업

[경력]

2002년-2015년	경상대학교병원 영양실
2016년-	창원경상국립대학교병원 영양팀

[관심분야]

Medical Nutrition therapy

[논문]

1. Oral Nutritional Supplements Reduce Body Weight Loss after Gastrectomy in Patients with Gastric Cancer :A Systematic Review and Meta-Analysis of Randomized Controlled Trials
2. Biological activities of Korean Berries.
3. Physicochemical characteristic of four different berries cultivar in Korea.
4. Antioxidant Activity and Identification of Anthocyanins Fractionated from Korean Berries
5. Determination of Bio:ogical Activities of Korean Berries and their anthocyanin Identjfication.

Nutrition therapy strategy for cardiovascular management

Hyun-hi Kang

Nutrition team, Gyeongsang National University Changwon Hospital, Changwon, Korea

심혈관건강과 식이사이의 관계는 점점 진화되고 있습니다. 식이요법의 변화를 포함한 생활 방식 수정은 심장 대사 위험요인을 관리하는 주요 접근법입니다. 다양한 식이요법과 심혈관건강에 미치는 영향에 대해 이해하는 것은 심혈관질환의 1, 2차 예방을 위한 중요 사항입니다. 그럼에도 불구하고 심장건강에 좋은 식단을 채택하는 대에는 많은 장벽과 한계가 있습니다.


01 올바른 식사원칙

고혈압, 당뇨, 고지혈증, 비만, 음주, 운동 부족 등은 심장질환의 위험요인입니다. 이는 심장질환의 회복 및 재발 방지를 위해 반드시 관리가 필요합니다. 위험인자를 낮추기 위해 잘못된 식생활 습관을 개선하여 건강한 생활습관을 갖는 것이 중요합니다.

1 매일 일정한 시간에 하루 3끼 규칙적으로 식사합니다.
규칙적인 식사는 과식을 방지하고, 당뇨가 있는 경우 저혈당을 예방하는데 도움이 됩니다.

2 나에게 맞는 알맞은 양으로 골고루 식사합니다.
과식하거나 폭식을 하는 경우 체중증가 뿐 아니라 당뇨병이 있는 경우 혈당 상승이 원인이 될 수 있습니다. 탄수화물, 단백질, 지방과 더불어 필요한 각종 비타민, 무기질을 섭취하기 위해 다양한 식품을 골고루 먹습니다.

3 정상체중 및 허리둘레를 유지합니다.
비만하거나 복부비만을 가지고 있는 경우, 심장질환 재발가능성이 높아집니다. 비만한 경우는 체중조절 이 필요합니다.

4 지나치게 짠 음식은 피하고, 간은 싱겁게 먹습니다.
소금으로 나트륨(체액의 균형을 조절하는 무기질)을 많이 섭취하면 체액의 수분을 많이 끌어당겨 혈액의 양이 많아져 혈압이 높아집니다. 그러므로 염분섭취를 줄이면 혈압 조절에 도움을 주고 심장의 부담을 덜어줍니다.

5 기름이 많은 음식은 삼가합니다.
고지방섭취가 많아지면 열량섭취가 높아져 비만을 유발하고, 혈중 지질 농도를 증가시키므로 주의합니다.

6 단 음식은 제한합니다.
당질은 탄수화물의 일종으로 과다 섭취시, 중성지방 수치가 올라가게 되며, 복부비만의 원인이 될 수 있 습니다.

7 식이섬유소를 충분히 섭취합니다.
식이섬유소는 콜레스테롤 흡수를 저해하고 포만감을 주어 체중조절에 도움이 됩니다. 단, 항응고제(와파 린, 쿠마딘)를 복용하는 경우 즙이나 엑기스 등(녹즙) 섭취는 제한합니다.

8 술은 피하는 것이 좋습니다.
과도한 음주는 혈압과 혈청지질 농도를 상승시켜 심장질환의 위험을 높이고 체중증가 및 복부비만을 일으킬 수 있습니다.

9 민간요법은 피합니다.
과학적으로 검증되지 않은 각종 달인물, 즙, 엑기스 등의 민간요법은 간수치 상승 등의 부작용이 발생할 수 있습니다.

4

<창원경상국립대학교병원 심장질환과 식이요법 리플릿 발췌>

또한, 우리나라는 경제 발달과 식생활의 서구화 등으로 인해 일부 식이 요인에서 급격한 섭취량 추이 변화를 겪고 있습니다. 여러 식이 요인 가운데 이상지질혈증과 심혈관건강에 대한 관심도가 증가하고 있습니다.

이에 따라 가당 음료를 제한하고, 바람직한 단백질, 지질 섭취에 대해 보다 구체적으로 심혈관질환을 예방하기 위한 다음의 식사원칙과 심장 보호식품을 권장하고 있습니다.

과일, 야채, 콩류, 통곡물, 저지방 단백질이 풍부한 식단을 섭취하고 가공식품, 트랜스지방, 설탕이 첨가된 음료를 최소화하는 것이 예방의 지침으로 권장됩니다.

<심장보호성분의 효능 및 함유식품>

CoQ10	심장근육 세포에서 ATP 생산증가 강력한 항산화 효과 내피 기능 개선 지질 프로필 개선	지방이 많은 생선, 콩, 시금치, 견과류
오메가-3	염증표지자의 수준을 낮춤 혈관 내피기능 개선 CVD발병 위험감소 TG수치 낮춤 혈압감소 혈소판 응집감소	기름진 바다 생선, 해조류, 아미씨, 치아씨드
시토스테롤	지질 프로필개선 항산화효과	야채 및 과일, 식물성기름, 견과류, 콩과 식물
비타민E	항산화효과 혈소판 응집감소 CVD발병 위험 감소	식물성 기름, 견과류
폴리페놀	혈압강하, 내피기능개선, 지질 프로필개선 CVD발병위험감소	과일, 식물성 기름

또한, 식이 관리는 임상지침에서 1차, 2차 예방에 중요한 것으로 널리 인식되고 있으며, 영양교육은 심장재활의 중요한 요소입니다. 이에 다수의 식이 패턴이 보조수단이 아닌 치료관리방법으로 다루어져야 합니다.

<심혈관질환에서 선택된 식이 모델의 건강상의 이점>

지중해 식단	· 심혈관 질환 위험 감소 · 심혈관 질환으로 인한 사망 위험 감소 · 지질 프로파일 개선	· 혈압 감소 · 염증 감소
DASH 다이어트	· 심혈관 질환으로 인한 사망 위험 감소 · 혈압 감소	· LDL 콜레스테롤 수치 감소 · 제2형 당뇨병 위험 감소
마인드 다이어트	· 산화 스트레스 감소 · 대사기능 개선	· 뇌졸중 후 인지 저하 예방
채식주의 식단	· 심혈관 질환으로 인한 사망 위험 감소 · 허혈성 및 출혈성 뇌졸중의 위험을 감소.	· LDL 콜레스테롤 수치 감소
북유럽 식단	· LDL 콜레스테롤 수치 감소 · 대사기능 개선 · 인슐린에 대한 세포의 감작	· CRP 농도 감소 · 혈압 감소
저탄수화물 다이어트	· 체중 감소 · CRP 농도 감소 · 트리글리세리드 수치 감소	· 혈관 내피 기능 개선 · 혈압 감소

그러나, 영양분야는 상충되는 정보로 인해 지나치게 복잡해지는 경우가 많습니다. 그러한 예로 소금을 들 수 있으며 최근 보고에 따르면 소금제한의 이점은 이미 고혈압이 있는 사람들에게 가장 크며 낮은 섭취와 높은 섭취 모두 사망률이 증가한 것과 관련이 있을 수 있겠습니다.

본 강좌의 목적은 심혈관 관련 영양분야를 검토하고 심혈관 건강에 있어 확립된 식단과 새로운 식단에 대한 포괄적인 최신개요를 제공하여 실무자가 환자의 건강한 식이요법을 결정할 수 있도록 제안하고자 합니다. 아울러, 다양한 다이어트의 효과에 대한 논의를 하며, 영양상담을 통해 환자가 심장에 건강한 식사를 채택하도록 돕고자 합니다.

심장질환 환자는 동반질환이 많아지면서 점점 더 복잡해지고 있습니다. NACR (National Adult of Cardiac Rehabilitation)데이터에 의하면 고혈압이 가장 흔한 동반질환(49.9%)이고, 고콜레스테롤 혈증/이상지질혈증(31.7%), 당뇨병(24.5%)이 있고, 이들은 잘못된 식습관이 동반질환에서 수정가능한 중요한 위험요소이며, 생활방식을 다루는데 중점을 두면 개선될 가능성이 높다고 보고하고 있습니다.

따라서, 환자들이 가지고 있는 사회 경제적 부담 등을 포함한 생활문화에 맞는 식이 권장 사항을 구현하는데 있어서 영양전문가의 역할을 포함한 다학제의 팀을 기반으로 하는 접근 방식의 필요성에 대해 고민해 보아야 하겠습니다.

MEMO

CURRICULUM VITAE

임현정

경희대학교 의학영양학과

[학력]

2002	경희대학교 생활과학대학 식품영양학과/ 호텔관광대학 조리과학과 학사
2004	경희대학교 동서의학대학원 의학영양학과 석사
2009	경희대학교 동서의학대학원 의학영양학과 박사

[경력]

2010-2013	Johns Hopkins University, Center for Human Nutrition, Research fellow
2023-현재	한국영양학회, 총무이사
2023-현재	한국임상영양학회, 사업이사
2021-현재	한국지질·동맥경화학회, 식품영양간사, 학술위원, 홍보위원, 간행위원
2021-현재	대한비만학회, 학술위원, 연구위원, 연수위원, IT 융합 대사증후군 치료위원회 위원

[관심분야]

- Development and application of nutritional intervention protocol for chronic diseases caused by dietary factors
- Epidemiological study of the relationship between dietary risk factors and non-communicable diseases
- Evaluation of nutritional status of inpatients by disease, customized nutrition management, research on nutritional intervention methods
- Development of a precision nutritional care platform

[논문]

1. Sun X, Yon DK, Nguyen TT, Tanisawa K, Son K, Zhang L, Shu J, YangY, Branca F, Wahlqvist ML, Lim H, Wang Y. Influence of dietary and other lifestyle factors on non-communicable diseases in the Western Pacific region and policy implications. The Lancet Regional Health - Western Pacific 43:100842, 2024
2. Bae JH, Lim H, Lim S. The Potential Cardiometabolic Effects of Long-Chain Omega-3 Polyunsaturated Fatty Acids: Recent Updates and Controversies. Advances in Nutrition 14(4): 612-628, 2023
3. Park S, Kim HJ, Kim S, Rhee SY, Woo HG, Lim H, Cho W, YonDK. National trends in physical activity among adults in South Korea before and during the COVID-19 pandemic, 2009-2021. JAMA Network Open 6(12): e2349249, 2023
4. Lim H, Son K, Lim H. Association between Skeletal Muscle Mass-to-Visceral Fat Ratio and Dietary and Cardiometabolic Health Risk Factors among Korean Women with Obesity. Nutrients 15(7): 1574, 2023
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Impact of lifestyle modifications in preventing and managing cardiovascular diseases

Hyunjung Lim

Department of Medical Nutrition, Kyung Hee University, Republic of Korea

Cardiovascular diseases (CVDs) constitute a major global health challenge, contributing significantly to morbidity and mortality. This presentation delves into the critical role of lifestyle modifications as potent tools in both preventing and managing CVDs. A comprehensive review of current literature reveals compelling evidence supporting the efficacy of lifestyle interventions in reducing the risk factors associated with cardiovascular ailments.

First of all, dietary modifications, emphasizing a balanced and heart-healthy nutrition plan, play a pivotal role in reducing cholesterol levels, controlling blood pressure, and preventing the development of atherosclerosis. Furthermore, engaging in regular physical activity emerges as a cornerstone of preventive measures, promoting optimal cardiovascular health by enhancing blood circulation, managing weight, and improving overall fitness. Smoking cessation is identified as a paramount lifestyle change, with its profound impact on lowering the risk of coronary artery disease and stroke. Additionally, stress management techniques contribute significantly to mitigating the detrimental effects of chronic stress on the cardiovascular system.

Public health initiatives aimed at promoting awareness and education on the importance of lifestyle modifications are crucial in fostering sustainable behavioral changes. These initiatives empower individuals to make informed choices and embrace heart-healthy habits throughout their lives. The presentation highlights the need for tailored interventions considering cultural, socioeconomic, and individual factors to maximize the adoption of these lifestyle changes. In addition, by emphasizing the transformative potential of lifestyle modifications, this presentation underscores their integral role in the comprehensive prevention and management of cardiovascular diseases.

CURRICULUM VITAE

이보경

동아대학교 식품영양학과

[학력]

2008-2014	Ph.D in Food Science, University of California, Davis
2006-2008	석사, 이화여자대학교 공과대학 식품공학과
2001-2006	학사, 이화여자대학교 자연과학대학 화학과

[경력]

2019-	동아대학교 식품영양학과 조교수
2015-2019	Postdoctoral researcher: Center for comparative medicine, UCDavis

[관심분야]

장내미생물, 프로바이오틱스, 프리바이오틱스, 건강증진

[논문]

1. Sodedji KAF, Assogbadjo AE, Lee B, Kim HY (2024) An Integrated Approach for Biofortification of Carotenoids in Cowpea for Human Nutrition and Health. Plants 13, 412. https://doi.org/10.3390/plants13030412.
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Role of probiotics in cardiovascular risk management

Bokyung Lee

Department of Food Science and Nutrition, Dong-A University, South Korea

Cardiovascular disease (CVD) is the leading cause of death worldwide, including heart disease, cerebrovascular disease, and rheumatic heart disease. The treatments for CVDs typically involve pharmacotherapy and surgery although pharmacotherapy may induce liver and kidney damage and other side effects. Thus, there is a need for efficient and alternative approaches to treat or prevent CVDs. Previous studies have shown that potential probiotics may reduce the incidence of CVD by regulating the body's metabolism, lowering blood glucose and lipids, and controlling blood pressure. Moreover, probiotic intake plays a crucial role in promoting the growth of beneficial bacteria, decreasing the risk of chronic diseases such as CVDs. Clinical studies also supported the cardiovascular benefits of probiotic supplementation. In conclusion, probiotics might be a promising way for mitigating the risk factors associated with CVDs.

SoLA 2024

한국지질·동맥경화학회 춘계학술대회

2024 **S**pring Congress **o**n **L**ipid & **A**therosclerosis of KSoLA

SoLA 2024
한국지질·동맥경화학회
춘계학술대회
2024 Spring Congress on Lipid & Atherosclerosis of KSoLA

Symposium 7

How Can We Manage Dyslipidemia in Special Populations?

4월 6일(토) 16:10-17:40 | Room 1

좌장 : 김상현(서울의대 순환기내과), 이은정(성균관의대 내분비내과)

패널 : 오규철(가톨릭의대 순환기내과), 정창희(울산의대 내분비내과), 조준환(중앙의대 순환기내과)

16:10-16:30 **Dyslipidemia management in elderly patients**
최성훈(한림의대 순환기내과)

16:30-16:50 **Dyslipidemia in chronic kidney disease**
유태현(연세의대 신장내과)

16:50-17:10 **Dyslipidemia in adolescents and young adults**
이은영(가톨릭의대 내분비내과)

17:10-17:40 **Panel Discussion**

CURRICULUM VITAE

최성훈

한림대강남성심병원 순환기내과

[학력]

1991-1997 연세의대 졸업

[경력]

2004-2005 세브란스병원 심장혈관병원 내과 전임의
2006- 한림대강남성심병원 순환기내과 교수
2015-2016 HARVARD BIDMC visiting schollarship

[관심분야]

Atherosclerosis, lipoprotein metabolism

[논문]

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2. Statement on chronotherapy for the treatment of hypertension: consensus document from the Korean society of hypertension. Park S, Ihm SH, Cho IJ, Kim DH, Park JH, Chung WB, Choi S, Lee HY, Kim HC, Sohn IS, Lee EM, Kim JH, Kim KI, Cho EJ, Sung KC, Shin J, Pyun WB
3. KSHF Guidelines for the Management of Acute Heart Failure: Part II. Treatment of Acute Heart Failure. Lee JH, Kim MS, Yoo BS, Park SJ, Park JJ, Shin MS, Youn JC, Lee SE, Jang SY, Choi S, Cho HJ, Kang SM, Choi DJ.
4. The Potential Role of Biomarkers Associated with ASCVD Risk: Risk-Enhancing Biomarkers. Choi S.

Dyslipidemia management in elderly patients

Seonghoon Choi

Cardiology, Internal Medicine, Kangnam Sacred Heart Hospital, Hallym University, Seoul, Republic of Korea

한국은 65세 이상을 노인으로 정의하는 세계보건기구 정의를 사용하더라도 전세계에서 가장 빠른 노령화 사회에 이미 진입하였다. 사망 원인과 관련하여 신생물질환의 경우 초고령(80세 이상)에서 감소하나 심혈관 질환 사망은 지속적으로 증가한다. 따라서 죽상경화성 심혈관질환의 위험도는 죽종의 발생이 유발되는 30대 중반이후 지속적 위험도가 증가함으로 심혈관질환의 예방을 위한 지질조절이 필요하다. 노인에서 스타틴 사용은 60194명을 대상으로 체계적 검토 및 메타분석에서 일차예방 목적 투여는 관상동맥질환은 21%, 심근경색증을 55% 감소시킴이 확인되었다. 또한 심뇌혈관질환을 포함한 이차예방 연구에서 스타틴을 투여한 노인에서 총사망 20%, 심혈관사망 32%, 관상동맥질환 32%, 심근경색증 32% 및 뇌경색증을 22% 감소시킨다. 따라서 2차예방을 위한 LDL cholesterol 감소를 위한 스타틴 치료는 연령에 제한없이 사용이 필요하다. 다만 고강도 스타틴의 사용과 관련하여 주의가 필요하며 이미 스타틴을 사용하고 있는 경우 안정성과 효과가 확인된 경우 지속 사용을 권고하나 75세 이상에서 새롭게 스타틴을 사용하는 경우에는 중강도 스타틴으로 시작하여 이상반응과 LDL cholesterol 강하 정도에 따른 용량의 점진적 증량을 고려한다(대사, 동반약물, 신기능 고려). 75세 이상의 노인에서 일차예방목적으로 스타틴 투여는 현재까지 무작위배정연구는 진행된 바 없으며 심혈관 보호효과 역시 명확한 이득이 증명되지 않고 있다. 따라서 고령을 이유로 일차예방 목적으로 스타틴을 투여하는 것을 권고하지 않으나 개별 환자의 동반질환, 안정성, 심혈관 위험도를 평가하여 스타틴 약물 투여의 이익이 충분한 경우 환자와 심도 깊은 논의를 통한 결정이 권고된다. 오메가3지방산의 경우 일차예방 목적으로 노인에서 투여시 명확한 이익은 확립되지 않았으며 에케티미브의 경우 75세 이상 관상동맥질환이 없는 환자에서 심혈관 임상사고가 감소(EWTOPIA 75) 되었다는 일부 연구가 있으나 무작위배정 연구가 아니었다는 점에서 제한적이며 이를 근거로 일관적인 사용을 근거가 부족하다. 마지막으로 스타틴 중강도 용량과 에제티미브 약제 조합은 기존의 심혈관질환 환자에서 약물 수용성을 증대시키고 임상사고 발현은 고강도 스타틴 대비 비열등하며 이는 노인에서도 유지된다는 연구(RACING) 결과가 최근 소개된 바, 심뇌혈관질환 노인의 2차예방에서 이상반응 감소 및 순응도 향상이란 점에서 약물 치료 전략을 다양화 시킬 수 있는 가능성이 확인되었다. 다만 하위분석 연구에 해당하며 일차예방에 대한 연구 결과는 부족하다. 결론적으로 이차예방 목적으로 사용되는 스타틴은 지속적인 사용을 권고하며 노인의 다양한 임상조건을 고려하여 약제 사용시 주의점을 잘 확인하는 것이 권고된다, 일차예방 목적하 지질강하요법은 지질강하 치료를 통한 임상적 이익이 있는 고위험군에서 적절한 환자-의사 협의를 통하여 결정하는 것이 필요하겠다.

CURRICULUM VITAE

유태현

연세대학교 세브란스 병원

[학력]

1995년 연세대학교 의과대학 졸업
2005년 연세대학교 내과학 박사

[경력]

2008년 연세대학교 신장내과 조교수
2018년- 연세대학교 신장내과 교수

[관심분야]

당뇨병성 신증, 급성 신손상

[논문]

1. Park, C. H., et al. (2024). "Nocturnal systolic blood pressure dipping and progression of chronic kidney disease." Hypertens Res 47(1): 215-224.
2. Kim, C. H., et al. (2023). "Soluble receptors for advanced glycation end-products prevent unilateral ureteral obstruction-induced renal fibrosis." Front Pharmacol 14: 1172269.
3. Jhee, J. H., et al. (2023). "Short-term Blood Pressure Variability and Incident CKD in Patients With Hypertension: Findings From the Cardiovascular and Metabolic Disease Etiology Research Center-High Risk (CMERC-HI) Study." Am J Kidney Dis 81(4): 384-393.e381.
4. Lee, J. Y., et al. (2022). "Association of blood pressure with cardiovascular outcome and mortality: results from the KNOW-CKD study." Nephrol Dial Transplant 37(9): 1722-1730.
5. Kim, S., et al. (2021). "Exosome-based delivery of super-repressor IκBα ameliorates kidney ischemia-reperfusion injury." Kidney Int 100(3): 570-584.

Dyslipidemia in chronic kidney disease

Tae-Hyun Yoo

Department of Internal Medicine, Yonsei University College of Medicine, Seoul

As the average age increase, hypertension and diabetes has been gradually increasing and the prevalence of chronic kidney disease patients is rapidly increasing. Hypertension, along with diabetes, is a representative complication of chronic kidney disease (CKD) and the increases in the risk of cardiovascular disease, thereby increasing mortality. In addition, with continued decline in renal function, there is a high possibility of progression to end stage kidney failure requiring dialysis or transplantation. Dyslipidemia is known to be the common cause of CVD in diabetes and hypertension. In CKD, dyslipidemia is closely related to high risk in CVD and mortality. Thoroughly, controlling lipid profiles has been found to be a very important treatment policy not only in the general population but also in patients with diabetes and hypertension, so it is important to control dyslipidemia and set appropriate cholesterol targets. However, there was controversy over setting cholesterol targets in CKD population. Therefore, in this session, the prognostic roles and optimal lipid targets in CKD will be presented.

CURRICULUM VITAE

이은영

가톨릭의대 서울성모병원

[학력]

2006	연세의대 학사
2011	연세의대 석사
2014	연세의대 박사

[경력]

2006-2010	연세의대 세브란스병원 인턴, 레지던트
2011-2014	연세의대 세브란스병원 임상강사, 임상연구조교수
2015-	가톨릭의대 서울성모병원 임상조교수, 조교수, (현) 부교수

[관심분야]

당뇨병, 췌도이식, 인슐린저항성, 대사증후군, 당뇨합병증

[논문]

1. Risk of Incident Dementia According to Glycemic Status and Comorbidities of Hyperglycemia: A Nationwide Population-Based Cohort Study. Kim WJ, Lee SJ, Lee E, Lee EY*, Han K*. Diabetes Care 2022; 45:134-141.
2. Generation of iPSC-derived insulin-producing cells from patients with type 1 and type 2 diabetes compared with healthy control. Kim MJ*, Lee EY*, You YH, Yang HK, Yoon KH, Kim JW. Stem Cell Res. 2020 Oct;48:101958.
3. Exposure-weighted scoring for metabolic syndrome and the risk of myocardial infarction and stroke: a nationwide population-based study. Lee EY, Han K, Kim DH, Park YM, Kwon HS, Yoon KH, Kim MK, Lee SH. Cardiovasc Diabetol. 2020 Sep 29;19(1):153.
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Dyslipidemia in adolescents and young adults

Eun Young Lee

The Catholic University of Korea, Korea

Atherosclerotic cardiovascular disease (ASCVD) stands as a significant global cause of mortality. While clinical manifestations typically arise in middle age and beyond, atherosclerosis may commence in childhood, persisting into adulthood and contributing to cardiovascular disease (CVD) progression. Thus, early intervention through evidence-based measures such as screening, education, lifestyle adjustments, and guideline-directed medical therapy holds promise for this demographic. Given that atherosclerosis is fueled by atherogenic lipoproteins and inflammation, dyslipidemia emerges as a pivotal risk factor for ASCVD development. Lifestyle modifications represent a primary strategy for managing dyslipidemia in adolescents and young adults. In critical scenarios, pharmacological interventions, complementing lifestyle changes, may yield significant dyslipidemia alleviation. Initial dyslipidemia management entails a low-fat diet enriched with water-soluble fiber, plant stanols, and plant sterols, coupled with weight management and physical activity. Considering adolescents with a positive family history of premature CVD and low-density lipoprotein cholesterol levels exceeding 160 mg/dL, drug therapy involving HMG-CoA reductase inhibitors, bile acid sequestrants, and cholesterol absorption inhibitors could be warranted. Such dietary and pharmaceutical approaches demonstrate safety and efficacy, offering potential in retarding atherosclerosis progression.

SoLA 2024

한국지질·동맥경화학회 춘계학술대회

2024 **S**pring Congress **o**n **L**ipid & **A**therosclerosis of KSoLA

Symposium 8

Frontiers in Omics Technology

4월 6일(토) 16:10-17:40 | Room 2

좌장 : 배순식(부산의대 약리학교실), 남궁준(연세원주의대 생화학교실)

패널 : 김정한(가톨릭의대 생화학교실), 김형석(충남의대 생리학교실), 류홍열(경북대 생명과학부)

16:10-16:30 **Exploring the role of somatic mutations in human brain diseases**
김준호(성균관대 생명과학과)

16:30-16:50 **Integration of multiomics for understanding nanotoxicity**
이광(아주의대 생리학교실)

16:50-17:10 **Dysfunctional adipocytes promote tumor progression through YAP/TAZ-dependent cancer-associated adipocyte transformation**
이한웅(연세대 생화학과)

17:10-17:40 **Panel Discussion**

CURRICULUM VITAE

김준호

성균관대학교 생명과학과

[학력]

2009.09-2014.08 박사과정, KAIST 바이오및뇌공학과
2008.03-2009.08 석사과정, KAIST 바이오및뇌공학과
2004.03-2008.02 학사과정, KAIST 바이오및뇌공학과

[경력]

2021.09-현재 조교수, 성균관대학교 생명과학과
2017.04-2021.08 박사후연구원, Boston Children's Hospital / Harvard Medical School
2014.09-2017.03 박사후연구원, 연세대학교 의과대학

[관심분야]

생명정보학, 유전체학, 체성돌연변이 분석

[논문]

1. In Seok Yang, Insu Jang, Jin Ok Yang, Jinhyuk Choi, Min-Seo Kim, Ka-Kyung Kim, Byung-Joon Seung, Jae-Ho Cheong, Jung-Hyang Sur, Hojung Nam, Byungwook Lee§, Junho Kim§, and Sangwoo Kim§, CanISO: a database of genomic and transcriptomic variations in domestic dog (Canis lupus familiaris), BMC genomics, 2023, 24.1: 613.
2. Junho Kim, August Yue Huang, Shelby L. Johnson, Jenny Lai, Laura Isacco, Ailsa M. Jeffries, Michael B. Miller, Michael A. Lodato, Christopher A. Walsh§ and Eunjung Alice Lee§, Prevalence and mechanisms of somatic deletions in single human neurons during normal aging and in DNA repair disorders, Nature Communications, 2022, 13.1: 5918.
3. Michael B. Miller, August Yue Huang, Junho Kim, Zinan Zhou, Samantha L. Kirkham, Eduardo A. Maury, Jennifer S. Ziegenfuss, Hannah C. Reed, Jennifer E. Neil, Lariza Rento, Steven C. Ryu, Chanthia C. Ma, Lovelace J. Luquette, Heather M. Ames, Derek H. Oakley, Matthew P. Frosch, Bradley T. Hyman, Michael A. Lodato§, Eunjung Alice Lee§ and Christopher A. Walsh§, Somatic genomic changes in single Alzheimer's disease neurons, Nature, 2022, 604.7907: 714-722.
4. Junho Kim, Boxun Zhao, August Yue Huang, Michael B. Miller, Michael A. Lodato, Christopher A. Walsh§ and Eunjung Alice Lee§, APP gene copy number changes reflect exogenous contamination, Nature, 2020, 584.7821: E20-E28.
5. Junho Kim, Dachan Kim, Jae Seok Lim, Ju Heon Maeng, Hyeonju Son, Hoon-Chul Kang, Hojung Nam, Jeong Ho Lee§ and Sangwoo Kim§, The use of technical replication for detection of low-level somatic mutations in next-generation sequencing, Nature Communications, 2019, 10.1: 1047.

Exploring the role of somatic mutations in human brain diseases

Junho Kim

Department of Biological Sciences, Sungkyunkwan University, South Korea

With advances in next-generation sequencing (NGS) technologies, somatic mutation analysis has made great strides in establishing a comprehensive description of genomic changes in cancer over the past decade. International collaborative efforts such as The Cancer Genome Atlas (TCGA) and International Cancer Genome Consortium (ICGC) have been made to construct the landscape of somatic mutations in various cancer types, leading to unprecedented knowledge of the somatic mutations and their underlying mechanisms.

Recent analyses of somatic mutations have begun to be made in non-cancerous diseases, especially in brain diseases. Unlike cells in other tissues, differentiated neurons in the brain are rarely replaced and regenerated during the course of a person's life, so that somatic mutations in those cells may critically affect the function of neurons and even brain circuits. Recent studies have demonstrated that somatic mutations occurring during brain development do actually cause multiple neurodevelopmental diseases, supporting a new pathogenesis of brain disorders. However, due to the lack of specialized bioinformatic tools for detecting rare somatic mutations in tissues without clonal expansion, somatic mutations in brain have not been comprehensively explored yet.

In this talk, the speaker will discuss the difficulties associated with analyzing somatic mutations in the brain, as well as the current efforts aimed at tackling these challenges. The talk will cover different sequencing approaches and developed bioinformatic methods used to detect rare somatic mutations in low cell population or even in a single cell.

CURRICULUM VITAE

이광

아주대학교 의과대학 생리학교실

[학력]

1983-1991	학사 (부산대학교)
1991-1993	석사 (부산대학교)
1994-1998	박사 (동경대학교)

[경력]

1996-1997	동경대학교 의과학연구소 조교
1999-2001	NINDS, NIH, USA

[관심분야]

통합오믹스, 기계학습, 나노독성

[논문]

1. Unveiling Local and Global Conformational Changes and Allosteric Communications in SOD1 Systems using Molecular Dynamics Simulation and Network Analysis. Basitha S, Manavalanb B, Lee G. Comput Biol Med. 2024 Jan;168:107688
2. Comparative analysis of machine learning-based approaches for identifying therapeutic peptides targeting SARS-CoV-2. Manavalan B, Basith S, Lee G. Brief Bioinform. 2022 Jan 17;23(1):bbab412.
3. Silica-coated-magnetic-nanoparticle-induced cytotoxicity is reduced in microglia by glutathione and citrate discovered using integrated omics analysis. Shin TH, Manavalan1 B, Lee DY, Basith S, Seo C, Paik MJ, Kim SW, Seo H, Lee JY, Kim JY, Kim AY, Chung JM, Baik EJ, Kang SH, Choi DK, Kang Y, Mouradian MM, Lee G. Part Fibre Toxicol. 2021 Nov 25;18(1):42.
4. Silica-coated magnetic nanoparticles activate microglia and induce neurotoxic D-serine secretion. Shin TH, Lee DY, Manavalan B, Basith S, Na YC, Yoon C, Lee HS, Paik MJ, Lee G. Part Fibre Toxicol. 2021 Aug 12;18(1):30.
5. Machine intelligence in peptide therapeutics: A next-generation tool for rapid screening of various diseases. Basith S, Manavalan B, Hwan Shin T, Lee G. Med Res Rev. 2020;40:1276-1314.

Integration of multiomics for understanding nanotoxicity

Gwang Lee[1,2]

[1]Department of Physiology, Ajou University School of Medicine, Suwon, Republic of Korea,
[2]Department of Molecular Science and Technology, Ajou University, Suwon, Republic of Korea

Exposure to nanoparticles leads to their accumulation in the brain, but drug development to counteract this nanotoxicity remains challenging. Here we assessed the effect of silica-coated-magnetic nanoparticles containing the rhodamine B isothiocyanate dye [MNPs@SiO2(RITC)] on microglia through integration of transcriptomics, proteomics, and metabolomics. Intracellular reactive oxygen species production, an inflammatory response, and morphological activation of cells were greater, but glucose uptake was lower in MNPs@SiO2(RITC)-treated BV2 microglia and primary rat microglia. Expression of 121 genes, and levels of 45 proteins and 17 metabolites related to the above phenomena changed in MNPs@SiO2(RITC)-treated microglia. We integrated the three omics datasets and generated a single network using a machine learning algorithm. We screened 19 compounds and predicted their effects on nanotoxicity within the triple-omics network. A combination of glutathione and citrate attenuated nanotoxicity induced by MNPs@SiO2(RITC) and ten other nanoparticles in vitro and in the murine brain, protecting mostly the hippocampus and thalamus.

CURRICULUM VITAE

이한웅

연세대학교 생화학과

[학력]

1977.3-1983.2 B.S., M.S., 연세대학교 이과대학 생화학과
1991.7-1994.1 M.S. in Microbiology and Immunology, Albert Einstein Colloge of Medicine
1991.7-1997.1 Ph.D. in Molecular Genetics, Albert Einstein College of Medicine

[경력]

1986.2-1991.6 Memorial Sloan-Kettering Cancer Center 생화학면역유전학연구실 테크니션
1997.1-1998.2 Albert Einstein College of Medicine 미생물면역학교실, 박사후연구원, 전임강사
1998.3-1999.2 서울대학교 의과대학 생화학분자생물학교실 조교수
1999.9-2006.2 성균관대학교 의과대학 생화학분자생물학교실 조교수, 부교수
2006.3-현재 연세대학교 생명시스템대학 생화학과 부교수, 교수

[관심분야]

Mouse molecular genetics, lung/breast cancer, aging, cancer drug development, Telomerase biology, mouse models for human diseases.

[논문]

1. Y. Song, ···, H. -W. Lee. Dysfunctional Adipocytes Promote Tumor Progression Through YAP/TAZ-dependent Cancer Associated Adipocyte Transformation. Nature Communications in press, 2024.
2. Na, H., Y. Song, H. -W. Lee. Emphasis on adipocyte transformation: anti-Inflammatory agents to prevent the development of cancer-associated adipocytes. Cancers 15(2): 502, Jan. 2023.
3. Jin, Young, ·····, H. -W. Lee. Depletion of adipocyte Becn1 leads to lipodystrophy and metabolic dysregulation. Diabetes 70(1): 182-195, Jan. 2021.
4. Kim, H. J., S. E. Lee, H. Na, J. -S. Roe, J. Roh, H. -W. Lee. Divergence of the PIERCE1 expression between mice and humans as a p53 target gene. PLoS One 15(8):e0236881, Aug 2020.
5. Roh, J., ·····., H. -W. Lee. Impaired AKT and tumorigenesis by PIERCE1 ablation in KRAS mutant non-small cell lung cancer. Oncogene 39(36):5876-5887, Jul 2020.

Dysfunctional adipocytes promote tumor progression through YAP/TAZ-dependent cancer-associated adipocyte transformation

Han-Woong Lee

Department of Biochemistry, Yonsei University, Republic of Korea

Obesity has emerged as a prominent risk factor for the development of malignant tumors. However, the existing literature on the role of adipocytes in the tumor microenvironment (TME) to elucidate the correlation between obesity and cancer remains insufficient. Here, we aimed to investigate the formation of cancer-associated adipocytes (CAAs) and their contribution to tumor growth using mouse models harboring dysfunctional adipocytes. Specifically, we employed adipocyte-specific BECN1 KO (BaKO) mice, which exhibit lipodystrophy due to dysfunctional adipocytes. Our results revealed active YAP/TAZ signaling in both CAAs and BECN1-deficient adipocytes, which induced adipocyte dedifferentiation to form a malignant TME for breast and colon cancer progression. Additional deletion of YAP/TAZ from BaKO mice significantly restored the lipodystrophy and inflammatory phenotypes, leading to tumor regression. Furthermore, mice fed a high-fat diet (HFD) exhibited decreased BECN1 and increased YAP/TAZ expression in their adipose tissues. Treatment with the YAP/TAZ inhibitor, verteporfin, suppressed tumor progression in BaKO and HFD-fed mice, highlighting its efficacy against mice with metabolic dysregulation. Overall, our findings provide insights into the key mediators of CAA and their significance in developing a TME, thereby suggesting a viable approach targeting adipocyte homeostasis to suppress cancer growth.

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2024 **S**pring Congress **o**n **L**ipid & **A**therosclerosis of KSoLA

Symposium 9
(KSoLA & KNS Joint Symposium)

Intermittent Fasting and Cardiovascular Disease
(Debate Session)

4월 6일(토) 16:10-17:40 | Room 3

좌장 : 김혜영(용인대 식품영양학과), 정인경(경희의대 내분비내과)

패널 : 신민정(고려대 바이오시스템의과학부), 이수용(부산의대 순환기내과)
홍경희(동서대 식품영양학과)

16:10-16:30	**The effects of intermittent fasting on cardiometabolic health: Pros** **송윤주**(가톨릭대 식품영양학과)
16:30-16:50	**The effects of intermittent fasting on cardio metabolic health: Cons** **장영우**(가천의대 심장내과)
16:50-17:40	**Panel Discussion**

CURRICULUM VITAE

송윤주

가톨릭대학교 식품영양학과

[학력]

1991-1995	서울대학교 식품영양학과 학사
1995-1997	서울대학교 식품영양학과 석사
1997-2001	서울대학교 식품영양학과 박사

[경력]

2002-2003	미국 샌디애고 주립대학 지역건강및역학연구소(C-BEACH) 박사후연구원
2006-2007	미국 시애틀 Fred Hutchinson Cancer Research Center 연구원
2007-2009	중국 쿤밍 Kunming Medical College 외국인교수

[관심분야]

Exploring the association between time-restricted eating, meal cycles, low-carb diets, dietary sugar, carb quality and their associations with obesity, metabolic syndrome, and type 2 diabetes

[논문]

1. Song J, Oh TJ, Song Y. Individual postprandial glycemic responses to meal types by different carbohydrate levels and their associations with glycemic variability using continuous glucose monitoring. Nutrients 2023, 15, 3571.
2. Kim J, Song Y. Early time-restricted eating reduces weight and improve glycemic response in young adults: a pre-post single-arm intervention study. Obesity Facts 2023;16:69-81.
3. Park S, Yang J, Song Y. The effect of four weeks dietary intervention with 8-hour time-restricted eating on body composition and cardiometabolic risk factors in young adults. Nutrients 2021,13, 2164.
4. Ha K, Nam K, Song Y. A moderate-carbohydrate diet with plant protein is inversely associated with cardiovascular risk factors: the Korea National Health and Nutrition Examination Survey 2013-2017.Nutrition Journal 2020;19:84.
5. Ha K, Song Y. Association of meal timing and frequency with obesity and metabolic syndrome among Korean adults. Nutrients 2019, 11, 2437.

The effects of intermittent fasting on cardiometabolic health: Pros

YoonJu Song

Department of Food Science & Nutrition, The Catholic University of Korea, Korea

Intermittent fasting was initially studied among individuals fasting for religious reasons like Ramadan fasting or shift workers. Recently, it has expanded to the general population as a new strategy for weight loss. Major types have emerged, including the 5:2 method, which designates fasting days weekly; alternate-day fasting (ADF) where fasting occurs every other day; and time-restricted eating (TRE), where a daily eating window is designated. The mechanism underlying intermittent fasting involves synchronizing eating and fasting periods with the body's circadian rhythm to improve metabolism efficiency. Various studies have reported weight loss effects for each type, with recent meta-analyses supporting these findings. Although some studies did not observe significant weight loss, favorable effects in certain cardiovascular risk factors were reported. Among the types of intermittent fasting, TRE has been the most extensively studied recently. A major issue in TRE are the length and timing of the eating window. The most common form involves fasting for 16 hours and eating within an 8-hour window (16:8). However, there is no conclusion on whether shortening the eating window maximizes health benefits. Additionally, another issue is whether starting the eating window early in the morning (early TRE) yields better effects on blood glucose control and cardiovascular risk factors compared to starting later (late TRE). Although research varies in terms of when the eating window starts, optimizing the eating window is essential for individuals to maximize the benefits. Other issues include the applicability of intermittent fasting in populations with underlying conditions like type 2 diabetes. A few studies reported efficacy of TRE, suggesting its applicability in those population. In summary, intermittent fasting positively impacts cardiovascular health through weight loss and improvements in cardiovascular risk factors. However, it is crucial to understand that individualized approaches tailored to each person's characteristics to maximize intermittent fasting's benefits.

CURRICULUM VITAE

장영우

가천대학교 길병원 조교수

학력 및 경력

School of Medicine, Gachon University, Doctor of Medicine
School of Medicine, Gachon University, Master of Medical Science
Cardiovascular Institute, Stanford Medicine, Postdoctoral Research Associate
Department of Cardiology, Gachon University, Gil Medical Center, Clinical Assistant Professor
Department of Cardiology, Gachon University, Gil Medical Center, Assistant Professor

관심 연구 분야

Cardiovascular intervention, Atherosclerosis, Acute myocardial infarction, Angina and heart failure, Pulmonary hypertension, Atrial fibrillation

주요 논문 및 저서

1. Jang AY, Lee HH, Lee H, Kim HC and Chung WJ. Epidemiology of PAH in Korea: An Analysis of the National Health Insurance Data, 2002-2018. Korean Circ J. 2023;53:313-327.
2. Jang AY, Kim M; Oh, PC, Suh SY, Lee K, Kang WC, and Han SH, Long-term Clinical Outcomes and Its Predictors Between 1- and 2-stent Strategy in Coronary Bifurcation Lesions: A Baseline Clinical and Lesion Characteristic Matched Analysis, Circ J. 2022 Aug 25;86(9):1365-1375. doi: 10.1253/circj.CJ-22-0163.2.
3. Jang AY, Kim B-G, Kwon S, Seo J, Kim HK, Chang H-J, et al. (2020) Prevalence and clinical features of bone morphogenetic protein receptor type 2 mutation in Korean idiopathic pulmonary arterial hypertension patients: The PILGRIM explorative cohort. PLoS ONE 15(9): e0238698. Sept. 2020. https://doi.org/10.1371/journal.pone.0238698.
4. Jang AY, Scherer PE, Kim JY, Lim S, and Koh KK. Adiponectin and cardiometabolic trait and mortality: where do we go? Cardiovasc Res. 2021 Jun 12;cvab199. doi: 10.1093/cvr/cvab199.
5. Oh S, Jang AY (co-first author), Chae S, Hwang D, Byun K, and Chung WJ. Comparative Analysis on the Anti-inflammatory/immune Effect of Mesenchymal Stem Cell Therapy for the Treatment of Pulmonary Arterial Hypertension. Sci Rep. 2021 Jan 21;11(1):2012. PMID: 33479312 PMCID: PMC7820276 DOI: 10.1038/s41598-021-81244-1.

The effects of intermittent fasting on cardio metabolic health: Cons

Youngwoo Jang

Gachon University, Gil Medical Center, Incheon, Korea

This lecture explores the potential negative impacts of intermittent fasting (IF) on cardiometabolic health, questioning its acclaimed benefits. It delves into how IF might negatively influence metabolic regulation, lipid profiles, and cardiovascular risk factors, potentially causing stress responses and disrupting circadian rhythms. The variability in individual responses to IF, suggesting the need for personalized dietary approaches, will be discussed. Additionally, the presentation addresses the lack of long-term data on IF's cardiometabolic effects. This concise overview aims to provide healthcare professionals with a critical perspective on IF, aiding in informed decision-making for those considering this dietary regimen.

SoLA 2024

한국지질·동맥경화학회 춘계학술대회

2024 **S**pring Congress **o**n **L**ipid & **A**therosclerosis of KSoLA

Research Group Session 1.
심혈관질환예측모형 연구TFT

Current Status and Challenges in the Development of Cardiovascular Disease Prediction Models

4월 5일(금) 12:30-14:00 | Room 1

좌장 : 배장환(충북의대 심장내과), 김현창(연세의대 예방의학교실)

**패널 : 구유정(서울의대 내분비내과), 김응규(인제의대 신경과), 박경민(울산의대 심장내과)
신지애(강원대 빅데이터메디컬융합학과), 이혁희(연세의대 예방의학교실)
천대영(한림의대 순환기내과)**

12:30-12:50	**CVD prediction model in Korea: current status and challenge** **김현창**(연세의대 예방의학교실)
12:50-13:10	**Cardiovascular risk prediction model based on prospective cohort studies** **이호규**(연세의대 예방의학교실)
13:10-13:30	**Cardiovascular risk prediction model based on health insurance claims data** **박상우**(울산의대 심장내과)
13:30-14:00	**Panel Discussion**

CURRICULUM VITAE

김현창

연세대학교 의과대학 예방의학교실

[학력]

1996	연세대학교 의과대학 의학사
1999	연세대학교 보건학석사
2003	연세대학교 보건학박사

[경력]

2004-현재	연세대학교 의과대학 전임강사/조교수/부교수/교수
2010-현재	연세대학교 의과대학 내과학교실 심장내과 겸무교수
2022-현재	연세대학교 디지털헬스케어혁신연구소 소장

[관심분야]

심혈관질환 역학 및 예방, 질병예측 모형, 건강형평성

[논문]

1. Hoang MT, Jung SJ, Lee H, Kim HC. Parent-Offspring Associations of Ideal Cardiovascular Health Metrics: Findings From the 2014 to 2021 Korea National Health and Nutrition Examination Survey. J Am Heart Assoc. 2024:e030995.
2. Lee HH, Lee H, Bhatt DL, Kang D, Youn JC, Shin DW, Cho J, Kim HC. Changes in physical activity and incident cardiovascular events in cancer survivors. Eur Heart J. 2023;44(47):4997-5000.
3. Lee HH, Lee H, Bhatt DL, Lee GB, Han J, Shin DW, Kang D, Youn JC, Guallar E, Cho J, Kim HC. Smoking habit change after cancer diagnosis: effect on cardiovascular risk. Eur Heart J. 2023:ehad199.
4. Rim TH, Lee CJ, Tham YC, Cheung N, Yu M, Lee G, Kim Y, Ting DSW, Chong CCY, Choi YS, Yoo TK, Ryu IH, Baik SJ, Kim YA, Kim SK, Lee SH, Lee BK, Kang SM, Wong EYM, Kim HC, Kim SS, Park S, Cheng CY, Wong TY. Deep-learning-based cardiovascular risk stratification using coronary artery calcium scores predicted from retinal photographs. Lancet Digit Health. 2021;3(5):e306-e316.
5. Kim HC, Greenland P, Rossouw JE, Manson JE, Cochrane BB, Lasser NL, Limacher MC, Lloyd-Jones DM, Margolis KL, Robinson JG. Multimarker prediction of coronary heart disease risk: the Women's Health Initiative. J Am Coll Cardiol. 2010;55(19):2080-91.

CVD prediction model in Korea: current status and challenge

Hyeon Chang Kim

Department of Preventive Medicine, Yonsei University College of Medicine, Korea

Research into cardiovascular disease (CVD) prediction models has long been a vibrant area of study in Western countries. These models have become essential components of guidelines for managing dyslipidemia, diabetes, hypertension, and the prevention of CVD. In Korea, interest in CVD prediction research has surged since the 2000s. However, the integration of CVD prediction models into clinical guidelines and their active application in clinical settings has been limited. This limitation is partly due to the scarcity of models that are developed and validated across diverse and representative cohorts. Additionally, the relatively lower incidence of CVD in Korea compared to Western populations diminishes the efficiency of these models in pinpointing individuals at high risk who would benefit from treatment.

There is an urgent need for the development of CVD prediction models tailor-made for the Korean population. Critical to this endeavor is the research and discussion necessary to establish cutoff levels for intervention based on the CVD risk estimated by these models. Determining such thresholds is vital for the models' effective application in clinical practice, ensuring that interventions are appropriately targeted at individuals at significant risk.

Furthermore, advancements in CVD prediction performance are necessary. Future models should account for the visit-to-visit variability in data from periodic health examinations and integrate novel biomarkers, while also adopting state-of-the-art methodologies, such as artificial intelligence. By addressing these challenges, we can enhance the predictive accuracy of CVD risk assessments and facilitate the identification of high-risk individuals. This progress will lead to a better integration of predictive models into clinical practice and targeted interventions, ultimately reducing the burden of CVD in Korea.

CURRICULUM VITAE

이호규

연세의대 예방의학교실

[학력]

2004-2008	미국 UC Berkeley 졸업 (BSc)
2009-2013	연세대학교 의과대학 졸업 (MD)
2016-2022	연세대학교 대학원 의학과 졸업 (PhD, 예방의학)

[경력]

2013-2018	세브란스병원 인턴, 내과 레지던트 (내과전문의)
2018-2024	연세대학교 의과대학 예방의학교실 강사, Physician-Scientist, 조교수
2024-현재	연세대학교 의과대학 예방의학교실 부교수
현재	한국지질·동맥경화학회 학술위원
현재	대한고혈압학회 역학연구회 부회장
현재	대한고혈압학회 학술위원, 연구위원, 편집위원, 진료지침위원
현재	대한심부전학회 데이터운영위원
현재	한국역학회 Epidemiology & Health 부편집인

[관심분야]

임상역학, 심장대사질환

[논문]

1. Lee HH, Lee HA, Kim EJ, Kim HY, Kim HC, Ahn SH, Lee H, Kim SU. Metabolic dysfunction-associated steatotic liver disease and risk of cardiovascular disease. Gut 2024;73(3):533-540.
2. Lee HH, Lee H, Bhatt DL, Kang D, Youn JC, Shin DW, Cho J, Kim HC. Changes in physical activity and incident cardiovascular events in cancer survivors. Eur Heart J 2023;44(47):4997-5000.
3. Kaneko H, Yano Y, Lee H, Lee HH, Okada A, Suzuki Y, Itoh H, Matsuoka S, Fujiu K, Michihata N, Jo T, Takeda N, Morita H, Nishiyama A, Node K, Kim HC, Yasunaga H. Blood Pressure Classification Using the 2017 ACC/AHA Guideline and Heart Failure in Patients With Cancer. J Clin Oncol 2023;41(5):980-990.
4. Lee HH, Lee H, Cho SMJ, Kim DW, Park S, Kim HC. On-treatment Blood Pressure and Cardiovascular Outcomes in Adults with Hypertension and Left Ventricular Hypertrophy. J Am Coll Cardiol 2021;78(15):1485-1495.
5. Lee H, Yano Y, Cho SMJ, Park JH, Park S, Lloyd-Jones DM, Kim HC. Cardiovascular Risk of Isolated Systolic or Diastolic Hypertension in Young Adults. Circulation 2020;141(22):1778-1786.

Cardiovascular risk prediction model based on prospective cohort studies

MEMO

CURRICULUM VITAE

박상우

울산대학교병원 심장내과

[학력]

2010	인제대학교 의학과 학사
2015	울산대학교 의학과 석사
2023	울산대학교 의학과 박사

[경력]

2011년-2015년	서울아산병원 전공의
2015년-2018년	육군 군의관
2018년-2019년	서울아산병원 심장내과 임상강사
2019년-현재	울산대학교병원 심장내과 조교수

[관심분야]

동맥경화증, 심혈관 위험인자, 심혈관 위험 예측

[논문]

1. Park S, Chang J, Hong SP, Jin ES, Kong MG, Choi HY, Kwon SS, Park GM, Park RW. Impact of Trimetazidine on the Incident Heart Failure Following Coronary Artery Revascularization. J Cardiovasc Pharmacol. 2023 Oct 1;82(4):318-326.
2. Park S, Jeon YJ, Ann SH, Kim YG, Lee Y, Choi SH, Han S, Park GM. Comprehensive Prediction of Subclinical Coronary Atherosclerosis in Subjects Without Traditional Cardiovascular Risk Factors. Am J Cardiol. 2023 Jul 1;198:64-71.
3. Park S, Kim YG, Ann SH, Cho YR, Kim SJ, Han S, Park GM. Prediction of the 10-year risk of atherosclerotic cardiovascular disease in the Korean population. Epidemiol Health. 2023;45:e2023052.
4. Park S, Park SJ, Park DW. Percutaneous Coronary Intervention Versus Coronary Artery Bypass Grafting for Revascularization of Left Main Coronary Artery Disease. Korean Circ J. 2023 Mar;53(3):113-133.
5. Park S, Park SJ, Park DW. Percutaneous Coronary Intervention for Left Main Coronary Artery Disease: Present Status and Future Perspectives. JACC Asia. 2022 Mar 15;2(2):119-138.

Cardiovascular risk prediction model based on health insurance claims data

박상우

울산대학교병원 심장내과

심혈관질환은 전세계적으로 질병부담 및 사망의 주요한 원인이 되고 있으며, 적절한 심혈관 위험도 평가는 증가된 위험을 갖는 환자를 식별하고 예방적 중재를 시행하기 위해 필요하다. 하지만, 한국에서는 한국인 인구집단을 대상으로 개발되고 널리 검증된 심혈관질환 위험도 평가도구가 현재까지 없는 실정으로, 현재 국내진료지침에서는 특정 위험예측모델들을 진료지침에 채택하여 그 활용을 권고하고 있지 않다. 국내진료지침에서는 고위험군, 초고위험군을 시사하는 질병의 유무, 주요 심혈관위험인자의 개수를 바탕으로 심혈관질환 위험도를 분류하여 치료 전략 수립에 활용하고 있다. 이와 같은 방법은 활용하기에 직관적이고 편리하나, 환자의 잠재적인 위험을 지나치게 단순화하여 위험을 과대평가 또는 과소평가할 수 있다는 한계점을 갖는다.

일반적으로 위험예측모델은 전향적 코호트에서 수집된 자료를 바탕으로 개발되지만, 한국에서는 위험예측모델 개발에 활용가능한 현대 한국인 인구집단에 대한 대표성을 지닌, 잘 유지되고 있는 전향적 코호트 자료가 부족한 실정이다. 또한, 개발된 위험예측모델의 성능은 시간의 흐름에 따라 점차 저하될 수 있으므로, 위험예측모델에 대한 주기적인 재보정이 필요할 수 있다. 한국에서는 전국민을 대상으로 국민건강보험이 적용되고 있으므로, 국민건강보험공단 데이터를 이용한 위험예측모델의 개발이 위와 같은 문제를 해결하기 위한 대안이 될 수 있다. 실제 본 연구진들이 국민건강보험공단 표본 코호트에 포함된 건강검진 데이터와 이후 약 10년 간의 의료이용 데이터를 활용하여 한국형 동맥경화성 심혈관질환 위험예측모델(Korean ASCVD risk prediction model [K-CVD])을 개발한 바 있어, 이를 간단히 소개한다. K-CVD 모델의 개발을 위해서 심혈관질환의 병력이 없는 20-80세의 325,934명의 대상자를 선택하여 남성, 여성 코호트로 구별하여 모델을 개발하였다. 동맥경화성 심혈관질환 사건은 심혈관 원인에 의한 사망, 심근경색 및 뇌졸중의 발생으로 정의하였다. 개발된 K-CVD 모델은 한국 인구집단을 대상으로 우수한 예측성능(AUC=0.846 [0.828-0.864], χ^2 4.73, goodness-of-fit p=0.32)을 보여주었으며, 특히 예측된 위험과 관찰된 사건 사이의 일치를 나타내는 Calibration 측면에서 매우 우수한 성능을 보여주었다. 기존 위험예측모델의 개발을 위해 널리 사용되고 있는 전통적인 코호트 기반 방법론에 비교해서, K-CVD 모델의 개발에 이용된 건강보험 청구자료 기반의 방법론은 전체인구집단에 대한 대표성을 갖는 표본 코호트에서 시간의 흐름에 따라 자동적으로 축적되는 의료정보를 이용함으로써 기존 방법론이 갖는 제한점을 극복하고, 주기적인 재보정을 용이하게 하여 인구집단의 변화에 따라 장기적인 모델성능을 유지하는데 기여할 수 있을 것으로 생각된다.

SoLA 2024

한국지질·동맥경화학회 춘계학술대회

2024 **S**pring Congress **o**n **L**ipid & **A**therosclerosis of KSoLA

SoLA 2024
한국지질·동맥경화학회
춘계학술대회
2024 Spring Congress on Lipid & Atherosclerosis of KSoLA

Research Group Session 2.
LP(a)/TRL 연구TFT

Decoding the Shadows: Exploring the Frontiers of Residual Lipid Risk in Cardiovascular Health

4월 5일(금) 12:30-14:00 | Room 2

좌장 : 정명호(광주보훈병원 순환기내과), 김병진(성균관의대 순환기내과)

패널 : 박경우(서울의대 순환기내과), 정인경(경희의대 내분비내과)
최성훈(한림의대 순환기내과), 최성희(서울의대 내분비내과)

12:30-13:00	**Deciphering lipid tests: critical insights into LDLC, RC, and LP(a) measurements for clinicians** **김솔잎**(울산의대 진단검사의학과)
13:00-13:20	**Unraveling the triglycerides puzzle: unifying insights and addressing discrepancies between Korean big-data and RCT findings** **김남훈**(고려의대 내분비내과)
13:20-13:40	**Lipoprotein(a) and the quest for clarity: unveiling the residual risk and the gaps in Korea's data landscape** **장영우**(가천의대 심장내과)
13:40-14:00	**Panel Discussion**

CURRICULUM VITAE

김솔잎

울산의대 서울아산병원

[학력]

2004	고려대학교 의과대학 (학사)
2008	울산대학교 의과대학 대학원 (석사)
2012	울산대학교 의과대학 대학원 (박사)

[경력]

2005.03-2009.02	서울아산병원 진단검사의학과 전공의
2009.03-2011.02	삼성서울병원/서울아산병원 진단검사의학과 임상강사
2011.03-2022.08	인제대학교 일산백병원 진단검사의학과 조교수/부교수
2022.09-현재	울산의대 서울아산병원 진단검사의학과 부교수
2022.01-현재	Ann Lab Med (SICE, IF 4.9) 부편집장(임상화학)
2022.07-현재	진단검사의학재단 표준화사업부장
2023.01-현재	대한임상화학회 총무이사
2023.01-현재	보건의료표준화추진단 핵심교류데이터 실무위원
2023.12.-현재	IFCC-EFLM EuroMedLab Brussels 2025 학술위원
2024.01-현재	IFCC EBLM committee member (국제임상화학회 근거중심진단검사의학 위원회 위원)
2024.01-현재	APFCB PBRTQC WG member (아태임상화학회 환자기반실시간정도관리 실무위원)

[관심분야]

임상화학, 진단검사 표준화, 질량분석검사, AI의 검사실 적용

[논문]

1. Machine learning-based delta check method for detecting misidentification errors in tumor marker tests. Clin Chem Lab Med. 2023
2. Interpreting changes in consecutive laboratory results: clinician's perspectives on clinically significant change.
3. Status of Pre-analytical Quality Management of Laboratory Tests at Primary Clinics in Korea. Ann Lab Med. 2023
4. Practical delta check limits for tumour markers in different clinical settings. Clin Chem Lab Med. 2023
5. Proposed Model for Evaluating Real-world Laboratory Results for Big Data Research. Ann Lab Med. 2023

Deciphering lipid tests: critical insights into LDLC, RC, and LP(a) measurements for clinicians

Sollip Kim

Department of Laboratory Medicine, Asan Medical Center, University of Ulsan College of Medicine, South Korea

한국인의 심뇌혈관질환의 가장 중요한 위험요인은 혈압, 흡연, 이상지질혈증, 당뇨병으로 알려져 있다. 한 사람이 여러 가지 위험요인을 가지고 있을 수 있으며, 위험요인 노출 정도가 다르기 때문에 여러 위험요인의 노출 정도를 계량화하여 개인의 질병 발생 확률을 계산하는 위험도 평가가 임상 의사결정에서 매우 중요하다. 대표적인 심혈관질환 역학연구인 Framingham Heart Study에서는 개인의 연령과 성, 총콜레스테롤(TC), 고밀도지단백 콜레스테롤(HDLC), 혈압, 당뇨병, 흡연 정보를 이용하여 위험도 평가 모형을 처음 개발하였고, 이후 여러가지 변형 모형이 개발되고 있다. 우리나라에서도 여러 심혈관질환 위험도 평가 도구가 개발 및 발표되고 있다.

위험도 평가 및 진단/치료 방침 결정에 TC, HDLC, 저밀도지단백 콜레스테롤(LDLC), 중성지방(TG) 등 혈액 지질검사 수치가 사용되므로 이들 검사 결과의 신뢰도 확보가 필수적이다. JCTLM (Joint Committee for Traceability in Laboratory Medicine) 및 CRMLN (Cholesterol Reference Method Laboratory Network) 주도의 지질검사 표준화 활동을 통해 현재 TC, HDLC, LDLC, TG 네 항목에 대해서는 표준검사법 및 표준물질을 바탕으로 검사시스템(기기-시약-칼리브레이터)에 대한 국제 품질인증을 시행하고 있으며, 우리나라에서도 질병관리청과 대한진단검사의학회가 협력하여 제품 인증 사업을 시행하고 있다.

최근에 이상지질혈증이 아포지단백, 아포지단백 수용체, 지단백 대사 관련효소 유전자 변이 등의 요인에 의하여 발생할 수 있음이 밝혀짐에 따라 고전적 지질검사 4종(TC, HDLC, LDLC, TG) 외에 다양한 지질검사에 대한 관심이 높아지고 있다. NCEP에서는 심혈관 질환 발생과 관련한 추가 지질성 위험인자로 TG, Remnant cholesterol (RC), lipoprotein(a) (Lp(a)), small LDL particles, HDL subclass, apolipoprotein B, apolipoprotein A-I, TC/HDLC ratio, 추가 비지질성 위험인자로 homocysteine, 혈전/지혈 인자, hsCRP, 공복혈당장애 등을 제시하였다. 이들 검사들은 아직 표준화가 잘 이루어져 있지 않고, 임상검사실에서 수행하고 있는 각 검사시스템의 특성에 대한 정보도 부족한 상황이다.

이번 연제에서는 고전적 지질검사인 TC, HDLC, LDLC, TG와 최근 관심이 증대되고 있는 Lp(a) 및 RC에 대해 검사원리 및 표준화 현황을 다루고자 한다. 임상검사실에서 실제 이루어지고 있는 지질 검사의 효용과 한계를 이해함으로써, 이들 검사를 바탕으로 한 위험도 평가 도구 및 치료지침 개발에 도움이 될 것이다. 아울러 국내 지질 검사 표준화를 위해 전문학회 간 협업도 가능하기를 기대해 본다.

CURRICULUM VITAE

김남훈

고려대학교 안암병원, 고려대학교 의과대학

[학력]

2004	고려대학교 의과대학 학사
2009	고려대학교 의과대학 석사
2015	고려대학교 의과대학 박사

[경력]

2023-현재	한국지질·동맥경화학회 간행위원회 간사
2023-현재	한국지질·동맥경화학회 학술위원회 위원
2024-현재	대한당뇨병학회 학술위원회 간사
2021-현재	대한내분비학회 기획위원회 간사

[관심분야]

Lipid metabolism, diabetes therapeutics, obesity

[논문]

1. Kim JY, Kim NH. Initial Combination Therapy in Type 2 Diabetes. Endocrinol Metab (Seoul). 2024 Feb;39(1):23-32.
2. Kim NH, Kim JY, Choi J, Kim SG. Associations of omega-3 fatty acids vs. fenofibrate with adverse cardiovascular outcomes in people with metabolic syndrome: propensity matched cohort study. Eur Heart J Cardiovasc Pharmacother. 2024 Feb 23;10(2):118-127.
3. Kim JY, Choi J, Kim SG, Kim NH. Comparison of on-Statin Lipid and Lipoprotein Levels for the Prediction of First Cardiovascular Event in Type 2 Diabetes Mellitus. Diabetes Metab J. 2023 Nov;47(6):837-845.
4. Kim KJ, Son S, Kim KJ, Kim SG, Kim NH. Weight-adjusted waist as an integrated index for fat, muscle and bone health in adults. J Cachexia Sarcopenia Muscle. 2023 Oct;14(5):2196-2203.
5. Kim JY, Choi J, Kwon Y, Park S, Kim SG, Kim NH. Serum fibroblast growth factor 1 and its association with pancreatic beta cell function and insulin sensitivity in adults with glucose intolerance. Front Endocrinol (Lausanne). 2023 May 22;14:1198311.

Unraveling the triglycerides puzzle: unifying insights and addressing discrepancies between Korean big-data and RCT findings

Nam Hoon Kim

Department of Internal Medicine, Korea University Anam Hospital, Korea University College of Medicine, Korea

We have observed the results from RCTs and CVOTs of TG lowering therapies, which mostly failed to prove cardioprotective effects as a single drug. However, several cohort studies within a real world setting, in contrast, consistently showed that Tg lowering therapies, including fenofibrate and omega-3 fatty acids, had a beneficial role in the prevention of atherosclerotic diseases or cardiovascular outcomes. First of all, there might be publication bias, but considering that, differences evidently are likely to exist between RCTs and RWEs of this topic. I will summarize and discuss about the reasons about that, including the real meaning of hyperTG, impact of TG variability on cardiovascular risk, different strategies to reduce hyperTG, and ethnic difference.

CURRICULUM VITAE

장영우

가천대학교 길병원 조교수

학력 및 경력

School of Medicine, Gachon University, Doctor of Medicine
School of Medicine, Gachon University, Master of Medical Science
Cardiovascular Institute, Stanford Medicine, Postdoctoral Research Associate
Department of Cardiology, Gachon University, Gil Medical Center, Clinical Assistant Professor
Department of Cardiology, Gachon University, Gil Medical Center, Assistant Professor

관심 연구 분야

Cardiovascular intervention, Atherosclerosis, Acute myocardial infarction, Angina and heart failure, Pulmonary hypertension, Atrial fibrillation

주요 논문 및 저서

1. Jang AY, Lee HH, Lee H, Kim HC and Chung WJ. Epidemiology of PAH in Korea: An Analysis of the National Health Insurance Data, 2002-2018. Korean Circ J. 2023;53:313-327.
2. Jang AY, Kim M; Oh, PC, Suh SY, Lee K, Kang WC, and Han SH, Long-term Clinical Outcomes and Its Predictors Between 1- and 2-stent Strategy in Coronary Bifurcation Lesions: A Baseline Clinical and Lesion Characteristic Matched Analysis, Circ J. 2022 Aug 25;86(9):1365-1375. doi: 10.1253/circj.CJ-22-0163.2.
3. Jang AY, Kim B-G, Kwon S, Seo J, Kim HK, Chang H-J, et al. (2020) Prevalence and clinical features of bone morphogenetic protein receptor type 2 mutation in Korean idiopathic pulmonary arterial hypertension patients: The PILGRIM explorative cohort. PLoS ONE 15(9): e0238698. Sept. 2020. https://doi.org/10.1371/journal.pone.0238698.
4. Jang AY, Scherer PE, Kim JY, Lim S, and Koh KK. Adiponectin and cardiometabolic trait and mortality: where do we go? Cardiovasc Res. 2021 Jun 12;cvab199. doi: 10.1093/cvr/cvab199.
5. Oh S, Jang AY (co-first author), Chae S, Hwang D, Byun K, and Chung WJ. Comparative Analysis on the Anti-inflammatory/immune Effect of Mesenchymal Stem Cell Therapy for the Treatment of Pulmonary Arterial Hypertension. Sci Rep. 2021 Jan 21;11(1):2012. PMID: 33479312 PMCID: PMC7820276 DOI: 10.1038/s41598-021-81244-1.

Lipoprotein(a) and the quest for clarity: unveiling the residual risk and the gaps in Korea's data landscape

Youngwoo Jang

Gachon University, Gil Medical Center, Incheon, Korea

This lecture addresses the role of Lipoprotein (a) [Lp(a)] in cardiovascular disease (CVD), focusing on its contribution to residual risk and the lack of comprehensive data in Korea. Lp(a) is an independent risk factor for CVD, yet its mechanisms and impact on the Korean population are not well understood. We will explore Lp(a)'s biochemistry, its role in atherogenesis and thrombosis, and highlight the need for ethnic-specific research due to the scarcity of studies in Korea. The presentation emphasizes the importance of advanced genetic screening and tailored epidemiological studies to clarify Lp(a)'s role in CVD risk. By addressing these gaps, we aim to improve cardiovascular risk assessment and therapy, enhancing strategies to combat CVD in diverse populations.

SoLA 2024

한국지질·동맥경화학회 춘계학술대회

2024 **S**pring Congress **o**n **L**ipid & **A**therosclerosis of KSoLA

SoLA 2024
한국지질·동맥경화학회
춘계학술대회
2024 Spring Congress on Lipid & Atherosclerosis of KSoLA

Committee Session 1. 지질영향 식품연구 TFT

심혈관 위험관리를 위한 식사요법은 어디에 초점을 맞추어야 하는가?

4월 5일(금) 14:00-15:20 | Room 1

좌장 : 박은주(경남대 식품영양학과), 박영미(이화의대 분자의과학교실)

패널 : 김오연(동아대 식품영양학과), 김희동(순천향의대 심장내과)
이규환(대상건강연구소), 임현정(경희대 의학영양학과)

14:00-14:15	**심혈관 위험 관리에서 식사의 보건학적 관점** 김미경(한양의대 예방의학교실)
14:15-14:30	**특수의료용도식품의 활용** **이인석**(경희의료원 영양팀)
14:30-14:45	**심혈관 건강기능식품 산업 및 활용 현황** **이상길**(부경대 식품영양학과)
14:45-15:00	**건강보조식품의 허와 실** **이시훈**(가천의대 내분비내과)
15:00-15:20	**Panel Discussion**

CURRICULUM VITAE

김 미 경

한양대학교 의과대학 예방의학교실

[학력]

1989	한양대학교 이학사
1992	한양대학교 이학석사
1996	한양대학교 이학박사

[경력]

1990-1996	한양대학교 의과대학 예방의학교실 연구원
1996-1998	한양대학교 의과대학 예방의학교실 박사후
1998-2001	지역사회연구소 연구교수
2001-2003	Research fellow, Harvard School of Public Health

[관심분야]

Diet and Cardiometabolic condition; systems epidemiologic approach; precision public health

[논문]

1. Prospective Associations between Cumulative Average Intake of Flavonoids and Hypertension Risk in the CArdioVascular Disease Association Study (CAVAS). Nutrients 2023,15:1186
2. Empirically identified dietary patterns and metabolic syndrome risk in a prospective cohort study: The Cardiovascular Disease Association Study. Clinical Nutrition 2022, 41:2156-2162
3. Development and validation of dietary atherogenic index using common carotid artery-intima-media thickness: A food frequency questionnaire-based longitudinal study in Korean adults. Nutrition Research 2022, 104:55-65
4. Genome-wide gene and serum ferritin interaction in the development of type 2 diabetes in adults aged 40 years or older. NMCD 2022, 32:231-240
5. Sex-specific associations of habitual intake of soy protein and isoflavones with risk of type 2 diabetes. Clinical Nutrition 2021, 40:127-136

심혈관 위험 관리에서 식사의 보건학적 관점

김미경

한양대학교 의과대학 예방의학교실

세계보건기구는 비감염성질환에 대한 글로벌 액션플랜 2013-2020을 수립, 2025년까지 만성질환으로 인한 조기 사망률 25% 감소를 목표로 설정하고 회원국 대상 국가 단위 정책 수립 및 성과지표 관리를 촉구해 왔다. 또한 2015년 UN은 지속가능발전을 위한 2030 Agenda를 채택하고 17개 목표, 169개 세부목표를 설정하여 발표하였다. 17개 목표 중 3번째 목표인 "Good Health and Well-being" 소목표에는 비감염성질환(심혈관질환, 암, 당뇨, 만성호흡기질환)으로 인한 조기 사망률 1/3을 감소시킨다는 내용이 포함되어 있다. 이에 따라 질병관리청에서는 매년 '만성질환 현황과 이슈'에서 심뇌혈관질환을 포함하여 질병 부담이 높은 주요 만성질환의 현황과 이슈를 분석 발표하고 있다. 통계청에서 발표한 2022년 1년간 한국인 주요 사망원인 중 순환계통 질환 사망률은 인구 10만명당 134.7명이었으며, 심장 질환(65.8명), 뇌혈관 질환(49.6명), 고혈압성 질환(15.1명) 순이었다. 2021년 대비 모두 증가 결과를 보였지만 World health statistics 결과에 따르면 2012년 대비 2019년 30-70세에서 사망률은 21.5% 감소한 결과를 보였다. 질병관리청에서 발표한 2019년 한국인의 조기사망으로 인한 질병부담의 주요 기여 질환의 1순위는 뇌혈관질환 질환이었고 허혈성 심질환 또한 4위를 차지했고 조기사망과 장애로 인한 질병부담에 식이위험은 위험요인 중 흡연, 고혈당, 알코올에 이어 4위를 차지한다고 발표했다. 따라서 심혈관질환 발생 이전 단계에서 수정가능한 효과적 개입 도구를 찾고 적용하는 것은 적극적 예방전략으로 가치가 높다.

식이는 이러한 적극적 예방전략 도구로 적합하고 서구인구집단에서 심혈관질환발생과 연관된 식이요인에 대한 많은 연구결과가 발표되었지만 첫째, 식생활은 하나의 문화로 표현될 만큼 문화적 특성이 강하여 한국인에 직접 적용하기 어렵고, 둘째, 식이 요인들이 서로 강한 상관이 있기 때문에 식이 자체의 복잡성(dietary complexity)을 고려한 분석이 필요하며, 또한, 이에 더하여 평균적 대상자에 국한하지 않는 정밀 보건학으로의 관점의 전환 또한 필요하다. 이 강좌는 이러한 내용을 포함하여 심혈관질환 예방을 위한 식이요인을 다룬다.

CURRICULUM VITAE

이인석(Inseok Lee)

경희의료원 영양팀(Kyung Hee University Medical Center, nutrition team)

[학력]

2003	경희대학교 생활과학대학 식품영양학과 졸업
2005	경희대학교 동서의학대학원 의학영양학과 석사
2009	경희대학교 동서의학대학원 의학영양학과 박사

[경력]

2010	New York obesity nutrition research center에서 post-doctoral follow
2020-2024	한양여자대학교 식품영양과 겸임교수
2011-현재	경희의료원 영양팀 임상영양사
2024	한국임상영양학회 정보이사

[관심분야]

임상영양, 영양지원, 신장질환, 비만

[논문]

1. J Lipid Atheroscler. 2020 Sep;9(3):474-475.
2. Nutrients. 2020 Oct 20;12(10):3203.
3. Pediatr Gastroenterol Hepatol Nutr. 2013 Sep;16(3):143-52.
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특수의료용도식품의 활용

이인석

경희의료원 영양팀

특수의료용도식품(foods for special medical purposes)은 정상적인 섭취, 소화, 흡수 또는 대사 기능이 제한되거나 질병, 수술 등의 임상적 상태로 인해 일반인과 생리적으로 특별히 다른 영양 요구량을 가지는 사람에게 필요한 영양소를 공급하거나 특정 영양 성분을 제한 또는 보충하기 위해 식사의 일부 또는 전부를 대신할 목적으로 제조·가공된 식품입니다. 현재 특수의료용도식품은 크게 표준형 영양조제식품, 맞춤형 영양조제식품, 그리고 식단형 식사관리식품으로 분류됩니다.

식품산업의 발전과 건강에 대한 관심 증가로 환자 대상으로 개발된 특수의료용도식품들이 일반인 대상으로도 확대 발전하고 있지만, 일반 식품과 달리 특정 질환(당뇨병, 신장병 등)을 대상으로 하는 특수의료용도식품은 의사 및 임상영양사와 상담 후 섭취해야 합니다. 특히, 모든 식사를 전부 대신하기 위해 사용하는 경우 섭취량에 따라 부족할 수 있는 영양소들이 발생하기 때문에 이를 모니터링하고 추가로 보충하는 것이 중요합니다. 그뿐 아니라 영양보충식품(oral nutritional supplements)으로 사용 시에도 대상자의 부족한 영양소를 파악하여 적절한 식품을 제공할 수 있는 것도 중요합니다.

한국사회가 빠르게 고령화 되면서 심혈관 질환을 동반한 영양불량 환자가 증가하고 있습니다. 이러한 이유로 특수의료용도식품은 영양 불량인 심혈관 질환 환자에게 다양한 형태로 사용이 가능합니다. 그러나 올바른 사용을 위해 의사 및 임상영양사 등과 같은 전문가의 관리가 반드시 필요합니다.

CURRICULUM VITAE

이상길

국립부경대학교 식품영양학전공 부교수

[학력]

2007	경희대학교 식품생명공학과, 이학사
2009	경희대학교 식품생명공학과, 이학석사
2015	University of Connecticut, Department of Nutritional Science, 이학박사

[경력]

2015-2017	North Carolina A&T State University 박사후 연구원
2018-2022	국립부경대학교 식품영양학전공 조교수
2022-현재	국립부경대학교 식품영양학전공 부교수

[관심분야]

해조류 유래 천연물의 항산화 및 항염증 기능을 기반으로 한 노인성 질환 개선

[논문]

1. The Role of Sargahydroquinoic Acid and Sargachromenol in the Anti-Inflammatory Effect of Sargassum yezoense. Y Park, L Cao, S Baek, S Jeong, HJ Yun, MB Kim, SG Lee Marine Drugs 2024
2. Sargahydroquinoic acid from Sargassum macrocarpum attenuates TNF-α and UV-induced skin aging in human dermal fibroblasts, L Cao, B Lee, BH Lee, S Lee, HR Kim Algal Research, 2024
3. Suppression of Pro-Inflammatory M1 Polarization of LPS-Stimulated RAW 264.7 Macrophage Cells by Fucoxanthin-Rich Sargassum hemiphyllum, Sanggil Lee Seungjin Jeong, Mi-Bo Kim, Suhyeon Baek, Joowon Lee, Hyeju Lee, Bei Cao, Yongeun Kim, Lei Cao, Marine Drug 2023
4. The Anti-Muscle Atrophy Effects of Ishige sinicola in LPS-Induced C2C12 Myotubes through Its Antioxidant and Anti-Inflammatory Actions, Mi-Bo Kim, Hyeju Lee, Chaehyeon Lee, Yuqing Tan, Sang Gil Lee, Applied Science, 2023
5. Embedded 3D printing of abalone protein scaffolds as texture-designed food production for the elderly, HJ Yun, WK Jung, HW Kim, S Lee Journal of Food Engineering, 2023

심혈관 건강기능식품 산업 및 활용 현황

Sang-Gil Lee

Department of Food and Nutrition, Pukyong National University, USA

심혈관 건강기능식품은 심혈관 질환 예방과 관리에 도움을 주는 기능성을 갖춘 제품으로, 특정 영양성분이나 항산화 물질 등의 생리활성 물질들을 함유하여 심혈관 건강을 촉진합니다. 국내 시장에서는 인구 고령화 및 식생활의 변화와 함께 심혈관 질환에 대한 관심이 높아지면서 심혈관 건강기능식품의 수요가 증가하고 있습니다. 현재 다양한 종류의 심혈관 건강기능식품이 출시되고 있으나, 소비자들은 전문가의 조언을 고려하여 심혈관 건강기능식품을 선택하는 것이 중요합니다. 본 연구는 심혈관 건강기능식품의 개발, 출시 및 소비 현황에 대해 논의하고자 합니다.

CURRICULUM VITAE

이시훈

가천의대 내과

[학력]

1992-1998	연세대학교 의과대학 졸업 (MD)
2000-2005	연세대학교 대학원 졸업 (PhD)
2006-2008	미국 국립보건원 (NIH) (Postdoc)

[경력]

1998-2003	세브란스병원 내과 전공의
2004-2005	일본 동경대학 대학원 연구원
2005-2006	연세대학교 의과대학 내과학교실 강사
2008-현재	가천대학교 의과대학 내과학 교수
2016	일본 동경대학 대학원 GPLLI 초빙교원
2016-2017	미국 하버드 의과대학/MGH 초빙부교수
2023-현재	미국 마운트사이나이 의과대학 약리학 겸임교수

[관심분야]

희귀유전질환, (부)갑상선호르몬, 탈요오드화효소, 재생의료, 정밀의료, 유전자편집

[논문]

1. Association of blood pressure with cardio-renal events and mortality in Type 2 DM: a national health insurance database. J Clin Endocrinol Metab 109:227-236, 2024
2. In-depth proteomic signature of parathyroid carcinoma. Eur J Endocrinol 188:385-394, 2023
3. Inherited Disorders of Thyroid Hormone Metabolism Defect caused by Selenoprotein Dysregulation. Front Endocrinol 12:803024. doi: 10.3389/fendo.2021.803024, 2022
4. Rare PTH Gene Mutations Causing Parathyroid Disorders: A Review. Endocrinol Metab 35:64-70, 2020
5. Incidence of hypoparathyroidism after thyroid cancer surgery in South Korea, 2007-2016. JAMA 322:2441-2443, 2019

건강보조식품의 허와 실

이시훈

가천의대 내과

당뇨병 및 심혈관질환을 비롯한 만성대사성 질환 환자 진료 시 영양상담은 매우 중요한 부분을 차지한다. 생활습관의 가장 많은 부분을 차지하는 것이 음식물 섭취이기 때문인데, 3분 진료로 대표되는 현재 우리 나라의 대학병원 시스템에서 생활습관 교정, 즉, 운동 및 식이 습관에 대한 교육 및 처방을 함께 진행하기란 여간 어려운 일이 아니다. 어떤 조성의 음식을 어느 정도 섭취하느냐가 매우 중요한 관건일텐데, 자주 환자에게서 이러저러한 음식 및 식품을 섭취하는 것은 어떻냐는 질문을 받을 때, 꽤나 곤혹스러울 때가 많다. 워낙 많은 환자들이 오랜 기간 동안 이환되어 있다 보니 민간에서 예전부터 구전되는 전통요법도 많고, 신문지상이나 인터넷 상에도 효과가 좋다는 정체 불명의 약제(?)에 대한 정보도 넘쳐나고 있다. 환자에게 최적의 처방과 교육으로 건강하게 천수를 누릴 수 있게 도와주는 일이 의사의 사명이라고 한다면, 환자가 궁금해하는 보조식품 섭취에 대한 정보도 제공해야 하는 게 당연한 도리인데, 도무지 그 근거를 찾을 수가 없고, 개인적인 경험에 의거하여 제대로 된 정보를 도출하기도 쉽지가 않은 실정이다. 이러한 시대적 요구와 요청에 부응하고자 1999년 전 세계 의생명연구의 중심이라 할 수 있는 미국 국립보건원(NIH)의 산하에 국립보완대체의학센터(NCCAM)을 설립하였고, 보완대체의학이란 미명하에 시행되는 수많은 검증되지 않은 약제 및 의료행태들에 대한 과학적인 검증 과정을 통해 가치를 평가하고 제대로 된 정보를 제공할 수 있도록 하였다. 본인은 2006년부터 2008년까지 이 곳에서 Dr. Michael Quon의 지도하에 박사 후 과정을 수행하면서 연구에 참여할 수 있었다. 보완대체의학이란 우리가 의과대학 및 부속병원에서 배우고 가르치면서 실제로 진료실에서 이루어지는 과학적 배경과 근거 중심의 정통의학의 범주에는 들어가지 않지만, 수 천 년간 경험적으로 수행되어 왔거나 구전된 각종 전통 혹은 민간의료, 그리고 대규모의 임상시험이 수행되지 않은 채 소규모의 제한적인 효과를 입증한 의료 행태로서 추가적인 안전성이나 보편적 효과의 입증을 필요로 하는 것들을 일컫는 말로, 우리의 전통의학이나 민간요법도 이 범주에 포함될 수 있고, 연구의 대상이 될 수 있다. 우리는 주로 침술이나 명상 등 객관화하거나 수치화하기가 어려운 대상은 배제하였고, 당뇨병 및 그 합병증, 혹은 심혈관계 에 효과가 예상되는 건강보조식품이나 기능성 식품에 대한 연구를 주로 수행하였다. 이를 위해서 Dr. Quon이 강조한 부분은 기존의 정통의학에 대한 깊은 이해와 성찰뿐만 아니라 보완대체의학에 대한 맹목적 신뢰 및 편견을 배제하고 그 숨어있는 가치를 최대한 도출하고, 혹시나 있을 수 있는 위해를 찾아내기 위한 깊은 상호 이해 및 높은 표준 수준(higher standard)이었다. Dr. Quon은 당뇨병 및 인슐린 저항성, 혈관내피세포 기능부전에 대해 이미 세계적 석학 반열에 오른 분이었고, 이에 대한 식견과 이해도 높고 깊었는데, 새로운 보조식품에 대해 매우 신중하고, 조심스럽게 접근하는 것을 가까이서 보고 배울 수 있었다. 그리고, 어느 보조식품에 대한 가치의 검증은 실험실적 연구에만 국한하는 것이 아니고, 동물실험과 인슐린 클램프법을 이용한 임상시험으로 연결되어 일관되고 신뢰성 있는 결과를 도출하여 전 세계 연구자들 및 임상가들에게 정확한 지식과 정보를 제공한다는 데에 깊은 자긍심을 갖고 있었다.

실제로 당뇨병에 뽕나무가 좋은지 홍삼이 좋은지, 오메가-3가 혈관건강에 좋은지에 대해 정확한 정보를 줄 수 없기에 답답한 면이 없지 않지만, 분명한 근거와 축적된 많은 경험이 부족한 보조식품은 피하는 것이 좋다고 권유한다. 그리고 정말 효과가 기대되는 보조식품이 있으면, 시간과 비용이 걸리더라도 정확한 연구와 임상시험을 통한 근거를 마련하는 것이 느리지만 정도(正道)라고 감히 말씀드린다.

SoLA 2024

한국지질·동맥경화학회 춘계학술대회

2024 **S**pring Congress **o**n **L**ipid & **A**therosclerosis of KSoLA

SoLA 2024
한국지질·동맥경화학회
춘계학술대회
2024 Spring Congress on Lipid & Atherosclerosis of KSoLA

Committee Session 2. 간행위원회

좋은 논문으로 가는 Big Step

4월 5일(금) 14:00-15:20 | Room 2

좌장 : 강현(중앙의대 마취통증의학과), 오형철(중앙의대 소화기내과)

14:00-14:20	**의학연구를 위한 데이터 시각화 길라잡이** **이호규**(연세의대 예방의학교실)
14:20-14:40	**데이터 시각화의 최신 트렌드와 디자이너가 바라본 메디컬 인포그래픽스** **황인권**(인권앤파트너스)
14:40-15:00	**좋은 논문의 완성, Graphical Abstract 만들기** **장동수**(Medical Illustration & Design)
15:00-15:20	**인공지능이 연구 논문 작성을 어떻게 변화시키는가: 가능성과 한계** **황윤희**(컴팩스)

CURRICULUM VITAE

이호규

연세의대 예방의학교실

[학력]

2004-2008	미국 UC Berkeley 졸업 (BSc)
2009-2013	연세대학교 의과대학 졸업 (MD)
2016-2022	연세대학교 대학원 의학과 졸업 (PhD, 예방의학)

[경력]

2013-2018	세브란스병원 인턴, 내과 레지던트 (내과전문의)
2018-2024	연세대학교 의과대학 예방의학교실 강사, Physician-Scientist, 조교수
2024-현재	연세대학교 의과대학 예방의학교실 부교수
현재	한국지질·동맥경화학회 학술위원
현재	대한고혈압학회 역학연구회 부회장
현재	대한고혈압학회 학술위원, 연구위원, 편집위원, 진료지침위원
현재	대한심부전학회 데이터운영위원
현재	한국역학회 Epidemiology & Health 부편집인

[관심분야]

임상역학, 심장대사질환

[논문]

1. Lee HH, Lee HA, Kim EJ, Kim HY, Kim HC, Ahn SH, Lee H, Kim SU. Metabolic dysfunction-associated steatotic liver disease and risk of cardiovascular disease. Gut 2024;73(3):533-540.
2. Lee HH, Lee H, Bhatt DL, Kang D, Youn JC, Shin DW, Cho J, Kim HC. Changes in physical activity and incident cardiovascular events in cancer survivors. Eur Heart J 2023;44(47):4997-5000.
3. Kaneko H, Yano Y, Lee H, Lee HH, Okada A, Suzuki Y, Itoh H, Matsuoka S, Fujiu K, Michihata N, Jo T, Takeda N, Morita H, Nishiyama A, Node K, Kim HC, Yasunaga H. Blood Pressure Classification Using the 2017 ACC/AHA Guideline and Heart Failure in Patients With Cancer. J Clin Oncol 2023;41(5):980-990.
4. Lee HH, Lee H, Cho SMJ, Kim DW, Park S, Kim HC. On-treatment Blood Pressure and Cardiovascular Outcomes in Adults with Hypertension and Left Ventricular Hypertrophy. J Am Coll Cardiol 2021;78(15):1485-1495.
5. Lee H, Yano Y, Cho SMJ, Park JH, Park S, Lloyd-Jones DM, Kim HC. Cardiovascular Risk of Isolated Systolic or Diastolic Hypertension in Young Adults. Circulation 2020;141(22):1778-1786.

의학연구를 위한 데이터 시각화 길라잡이

MEMO

CURRICULUM VITAE

황인권

인권앤파트너스

[학력]

1997 침례신학대학교 신학과
2011 홍익대학교 국제디자인대학원 IDAS Digital Media Design M.Des.

[경력]

2021- 숙명여자대학교 문헌정보학과 겸임교수
2017 국제백신연구소 브랜드 자문
2016 국세균 홍보 자문

[관심분야]

- Medical Branding
- Data Visualiazation

[논문]

1. 대한의학회 리브랜딩 2015
2. 세브란스 어린이병원 리브랜딩 2016
3. 국제백신연구소 IVI 리브랜딩 2017
4. 대한내과학회 리브랜딩 2017
5. 대한당뇨병학회. 대한고혈압학회. 한국지질·동맥경화학회 등의 팩트시트 작업

데이터 시각화의 최신 트렌드와 디자이너가 바라본 메디컬 인포그래픽스

황인권

인권앤파트너스, 서울

2013년 퓰리처상을 수상한 New York Times의 Snow Fall은 인포그래픽 기사의 유행을 일으켰다. 66개의 모션 그래픽과 1.7만자의 텍스트로 이루어 진 심층 보도 기사는 전세계에 영향을 끼쳐, 한국도 많은 신문에서 영상과 이미지, 스토리텔링을 중심으로 한 인포그래픽 기사들을 내고 있다. 2020년 중앙일보는 55년 기획 기사, '기후재앙 눈앞에 보다'를 VR 동영상과 입체 사운드 등으로 구성하여 '한국디지털저널리즘 어워드'와 '2020 과학언론상-올해의 의과학취재상' 등을 수상하는 등의 높은 평가를 받았다.

인포그래픽은 인류의 역사와 함께 해 왔으나 현대적인 구성의 인포그래픽이 등장하는 것은 1700년대가 되어서였다. 1769년 Joseph Priestley는 A New Chart of History에서 오늘날 쓰이는 인포그래픽과 유사한 형태의 미려한 스토리텔링을, 1857년 Florence Nightingale은 The Causes of Mortality in the Crimean War에서 'Polar Area Diagram'라는 새로운 스타일을 제안하여 수백만 명의 생명을 구하고, 의학 인포그래픽의 새로운 장을 열었다.

한국은 2020년 인포그래픽 전문 스튜디오 '스튜디오 203(대표 장성환)'에서 세계적으로 권위있는 '말로피에 어워드'를 수상, 국제적인 수준의 인포그래픽 역량을 보유하고 있는 국가가 되었다.

오늘날 인포그래픽은 사회 모든 분야에서 사용되고 있으며 Google Data Studio, Flourish, Tableau와 같이 온라인 툴을 통해 인터렉티브 스토리텔링, 실시간 데이터 인포그래픽을 구현하는 경우가 많으나, 아직까지 의학에서는 조금 더 보수적인 접근이 이루어지고 있다.

한국 의학의 인포그래픽은 인포그래픽 전문 회사나 디자인 스튜디오, 메디컬 일러스트 전문 회사에서 다양하게 생성되고 있는데, 한국 의학 데이터 시각화는 어디서부터 시작되었다고 볼 수 있을까?

국제 학술지의 표지 등에 사용하는 메디컬 일러스트레이션이 오래 전부터 간간히 있어 왔으나, 복잡한 의학 정보/숫자를 이용한 데이터 시각화의 지속적인 사례는 대한당뇨병학회에서 2012년 시작되었다. 이후 다양한 대중 커뮤니케이션을 해야 하는 대한비만학회, 대한고혈합학회 등을 중심으로 그래픽을 사용한 Visual Factsheet들이 학회 차원에서 발행되고, 점점 분량과 숫자가 늘어나고 있다.

최근 팩트시트의 경향은 다음과 같다. 하나는 논문이나 신문 기사 등에 삽입하는 추세를 볼 수 있는 그래프 모음들이다. 다른 하나는 복합적인 정보가 구조화 되는 인포그래픽이다.

소셜 미디어 시대가 되면서 많은 의학 논문들이 Graphical Abstract와 같이 한 장의 이미지를 통해 인스타그램 같은 SNS에서 논문을 알릴 필요가 증가되고 있어, 인포그래픽에 대한 의학 분야의 깊이 있는 접근이 점점 요구 되고 있다.

CURRICULUM VITAE

장동수

연세대학교 의과대학 연구부 그래픽지원실

[학력]

2020 홍익대학교 미술대학원 조형예술학과 미술학박사
2007 홍익대학교 미술대학원 조소전공 석사

[경력]

2008-현재 연세대학교 의과대학 연구부 그래픽지원실 / Medical Illuarator 총괄
현재 대한간학회, 대한당뇨병학회, 대장항문외과학회, trd, YMJ,/ Graphic Editor
2022-현재 대한메디컬아티스트 총무이사

[관심분야]

Medical visualization, Scientific visualization

[논문]

1. 2022 / Initial Experiences With Robotic Single-Site Thoracic Surgery for Mediastinal Masses / The Annals of Thoracic Surgery
2. 2020 / Baha Attract Implantation Using a Small Incision: Initial Report of Surgical Technique and Surveillance. / Clin Exp Otorhinolaryngol
3. 2020 / Glioblastoma Cellular Origin and the Firework Pattern of Cancer Genesis from the Subventricular Zone. / Korean Neurosurg Soc
4. 2019 / Cleaved Cochlin Sequesters Pseudomonas aeruginosa and Activates Innate Immunity in the Inner Ear / Cell Host Microbe

좋은 논문의 완성, Graphical Abstract 만들기

장동수

Research Affairs, Yonsei University College of Medicine

연구의 복잡한 내용을 직관적으로 전달하는 Graphical Abstract는 학술 커뮤니케이션의 핵심으로, 복잡한 데이터를 명료하게 시각화하여 독자의 이해와 기억을 극대화합니다. 점점 더 많은 저널이 Graphical Abstract의 제출을 요구하며, 이는 연구의 본질을 효과적으로 요약하고, 독자들의 학문적 호기심을 자극하는 중요한 수단이 되었습니다. 이러한 시각적 자료의 전파력은 소셜 미디어의 확산을 통해 강화되며, 학계와 일반 대중 간의 소통 다리 역할을 합니다. Adobe Illustrator를 포함한 다양한 그래픽 디자인 도구의 활용은 Graphical Abstract 제작에 있어 효과적입니다. 그러나, 프로그램 사용 능력 못지않게 중요한 것은 학술위원회의 명확한 가이드라인 제공과 연구자의 이를 통한 교육입니다. 이는 연구자가 자신의 연구를 효과적으로 시각화하고 전달할 수 있도록 돕고, 연구의 국제적 소통을 촉진합니다. 연구자는 투고 목적의 저널 가이드라인을 반드시 확인해야 하며, 이는 Graphical Abstract 제작과 투고 과정에서의 핵심 요소입니다. 학계의 지속적인 혁신과 발전은 연구 결과의 시각화를 통해 학술 연구의 미래를 형성하는 중요한 요소로, 연구 커뮤니티의 지원과 연구자들의 노력이 결합될 때 더 큰 영향을 미칠 것입니다..

Keywords

Medical illustration, Medical illustrator, Medical visualization, Graphical abstract, Cover design

CURRICULUM VITAE

황윤희

(주)컴팩스

[학력]

1987	MEd. 영어교육(TESOL), Boston University
2001	PhD. 응용언어학, Boston University

[경력]

2015-현재	컴팩스 대표
2002-2015	서울대학교 기초교육원 강의교수
2007-2012	서울대학교 English Writing Center 운영실장

[관심분야]

영어논문작성법, 영어논문 심사의견서 작성법, AI를 활용한 영어논문작성, 영어교수법

[논문]

1. Whang, Y., "ChatGPT for editors: enhancing efficiency and effectiveness" Science Editing 2024; 11(1): 84-90 http://doi.org/10.6087/kcse.332
2. Whang, Y. and Wendler-Shaw, P., “Writing letters and emails in English: correspondence for the editorial office” Science Editing 2021;8(2):186-192 http://doi.org/10.6087/kcse.254
3. Whang, Y., “Reviewing a journal article with clarity and politeness: key language tips for non-native English-speaking reviewers” Science Editing 2020;7(2):204-208 http://doi.org/10.6087/kcse.220

인공지능이 연구 논문 작성을 어떻게 변화시키는가: 가능성과 한계

황윤희

응용언어학, (주)컴팩스, 대한민국

본 발표에서는 영어로 논문을 작성하는 과정에 인공지능(AI) 기술이 어떠한 변화를 가져오고 있는지를 소개합니다. 특히 비원어민 연구자들이 AI 도구를 활용하여 어떻게 논문 작성의 효율성을 향상시킬 수 있는지에 대한 실질적인 팁을 제공하고자 합니다. 이를 위해, AI 기술(예를 들어 자동화된 문헌 검토 도구나 언어 생성 모델 등)이 연구 아이디어의 생성, 논문 초안 작성, 그리고 문헌 검토 과정에서 어떠한 역할을 할 수 있는지 실제 사례를 살펴보고자 합니다. 본 발표에서는 AI 기반 도구가 문헌 검토/정리 속도를 향상시키고, 논문 초안 작성을 가속화하는 등, 연구논문 작성과정의 다양한 단계에서 연구자들의 작업 효율을 향상시키는 사례들을 제시합니다. 구체적으로 SciSpace, Perplexity, Scholar AI, AskYourPDF 등 다양한 도구들과 언어 생성 모델(ChatGPT 등)을 어떻게 활용할 수 있는지에 대한 전략을 논의합니다. ChatGPT의 경우에서는 "Customize ChatGPT, GPTs, Plugins, GPT Vision, GPT Mentions" 와 같은 기능과 프롬프트 엔지니어링을 어떻게 잘 활용해야 원하는 맞춤형의 답변을 얻을 수 있는지 살펴봅니다. 또한 ChatGPT를 번역과 교정의 도구로 사용하고자 할 때, 양질의 결과를 만들어내는 방법과 유의할 점도 살펴봅니다. 마지막으로 AI 도구를 사용할 때 발생할 수 있는 문제점들이 어떠한 것들이 있는지 알아봅니다. 예를 들어, 정확성, 편향, 환각현상, 기계풍의 글쓰기 스타일, 용어사용 및 번역/교정의 결과 관련 일관성 부재 등과 관련된 문제점은 여전히 보이고 있습니다. 본 발표에서는 AI가 연구논문 작성과정에 어떠한 변화를 가져오고 있는지에 대한 통찰력을 제공하며, 연구자들이 이러한 도구들을 효과적으로 활용할 수 있도록 실질적인 가이드를 제공합니다. 또한 AI 기술의 가능성을 최대한 활용하면서도 그 한계를 인식하고 극복하는 방안을 모색하는 것의 중요성도 강조합니다.

Keyword

인공지능 기반 도구, 논문작성, ChatGPT

SoLA 2024

한국지질·동맥경화학회 춘계학술대회

2024 **S**pring Congress **o**n **L**ipid & **A**therosclerosis of KSoLA

SoLA 2024
한국지질·동맥경화학회
춘계학술대회
2024 Spring Congress on Lipid & Atherosclerosis of KSoLA

Committee Session 3. 기초연구위원회

Advances in Metabolic Regulation: Foundations for Therapeutic Insights in Metabolic Diseases and Beyond

4월 5일(금) 14:00-15:20| Room 3

좌장 : 김형규(인제의대 심혈관대사질환센터), 조계원(순천향대 의생명연구원)

패널 : 김용숙(전남대 의생명연구원), 송주현(전남의대 해부학교실)
이준엽(울산의대 안과), 정수명(성균관대 생명과학과)

14:00-14:20	**Understanding and targeting of cysteine metabolism in cancer** **강윤표**(서울대 약학과)
14:20-14:40	**Systems biology of human metabolism – multi-omics and modeling approach** **이선재**(GIST 생명과학부)
14:40-15:00	**Nitric oxide-induced protein modifications in disease** **윤소미**(전남대 약학부)
15:00-15:20	**Panel Discussion**

CURRICULUM VITAE

강윤표

서울대학교 약학대학

[학력]

2015 Ph.D., College of Pharmacy, Seoul National University
2009 B.S., College of Pharmacy, Seoul National University

[경력]

2021 Assistant Professor, College of Pharmacy, Seoul National University
2016 Postdoctoral Fellow, H. Lee. Moffitt Cancer Center
2015 Postdoctoral Fellow, Research Institute of Pharmaceutical Science, Seoul National University

[관심분야]

Ferroptosis, Cysteine metabolism, Cancer metabolism

[논문]

1. Nguyen, C. T. N., Kim, S. M., and Kang, Y. P. Mass spectrometry-based approaches to explore metabolism regulating ferroptosis. BMB Reports, 55, 9, 2022.
2. Kang, Y. P., Mockabee-Macias, A., Jiang, C., Falzone, A., Prieto-Farigua, N., Stone E., Harris, I. S., and DeNicola, G. M. Non-canonical glutamate-cysteine ligase activity protects against ferroptosis. Cell Metabolism, 33, 1. 2021.
3. Kang, Y. P., Falzone, A., Liu, M., González-Sánchez, P., Choi, B.-H., Coloff, J. L., Saller, J. J., Karreth, F. A., & DeNicola, G. M. PHGDH supports liver ceramide synthesis and sustains lipid homeostasis. Cancer & Metabolism, 8, 6. 2020.
4. Kang, Y. P., Torrente, L., Falzone, A., Elkins, C. M., Liu, M., Asara, J.M., Dibble, C.C. & DeNicola, G. M. Cysteine dioxygenase 1 is a metabolic liability for non-small cell lung cancer. Elife. e45572. 2019.
5. Kang, Y. P., Ward, N. P. & DeNicola, G. M. Recent advances in cancer metabolism: a technological perspective. Experimental & molecular medicine, 50, 31. 2018. Review.

Understanding and targeting of cysteine metabolism in cancer

Yun Pyo Kang

College of Pharmacy, Seoul National University, South Korea

CDO1 is an enzyme oxidizing cysteine and produce cysteine-sulfinic acid (CSA). CDO1 is generally depleted in NSCLC cells and tumors due to epigenetic silencing. We found that CDO1 restoration in NSCLC cells depleted cysteine, impaired cell growth and increased sensitivity to oxidative stress. Mechanistically, CDO1 expression led to a significant accumulation of sulfite due to GOT1-mediated transamination of CSA. Further, sulfite reacted with cystine, accelerating cysteine depletion and toxicity. Our results demonstrate that CDO1 is a metabolic liability for NSCLC cells through toxic sulfite production and cysteine depletion. Cystine deprivation induces ferroptosis in cancer cells, which provides therapeutic potential. Here, we show that cystine deprivation induced both GSH depletion and glutamate accumulation to accelerate ferroptosis. Surprisingly, GCLC directly catalyzes gamma-glutamyl peptide synthesis in cysteine starved cells. gamma-glutamyl peptide production reduced intracellular glutamate accumulation, thereby preventing the glutamate-mediated oxidative stress and ferroptosis induction. Therefore, the combinatorial approach of cystine deprivation and GCLC inhibition will provide better therapeutic potential to treat NSCLC.

CURRICULUM VITAE

이선재

광주과학기술원 생명과학부

[학력]

2006	KAIST 바이오시스템학과 학사
2008	KAIST 바이오및뇌공학과 석사
2015	KAIST 바이오및뇌공학과 박사 (생명정보학 전공)

[경력]

2015-2018	스웨덴 KTH 왕립공과대학 박사후연구원
2018-2020	영국 킹스컬리지런던 박사후연구원
2020-현재	광주과학기술원 생명과학부 조교수

[관심분야]

Bioinformatics, Metabolism, Microbiome, Systems Biology

[논문]

1. Vishal Patel*, Sunjae Lee* et al., “Rifaximin reduces gut-derived inflammation and mucin degradation in cirrhosis and encephalopathy: RIFSYS Randomised-Controlled Trial”, J Hepatology (2022)
2. Byong-Sop Song et al., "Mitoribosomal defects aggravate liver cancer via aberrant glycolytic flux and T cell exhaustion", BMJ Journal for Immunotherapy of Cancer (2022)
3. Chang-Hyun Kim*, Sang-Moo Park*, Sunjae Lee* et al., “NSrp70 is a lymphocyte-essential splicing factor that controls thymocyte development”, Nucleic Acids Research (2021)
4. Sara Omenetti et al., “The intestine harbours functionally distinct homeostatic tissue-resident and inflammatory Th17 cells”, Immunity (2019)
5. Sunjae Lee et al., “Integrated Network Analysis Reveals an Association between Plasma Mannose Levels and Insulin Resistance”, Cell Metabolism (2016)

Systems biology of human metabolism - multi-omics and modeling approach

Sunjae Lee

School of Life Sciences, Gwangju Institute of Science and Technology, Korea

In public health sector, chronic diseases, including cardiovascular disease, obesity, diabetes, and liver disease, are the most life-threatening diseases, projected to account for 85% of all deaths. Unlike infectious disease, it is not curable by vaccines or medication, and even worse, people of chronic diseases are likely to have multiple chronic conditions, impeding proper therapeutic developments.

Since the recent advance in high throughput technology, molecular profiling of chronic disease patients has been provided - transcriptome, epigenome, proteome, metabolome, and metagenome (Sunjae Lee et al., 2016, Cell Metabolism; Mardinoglu at al., 2017, Cell Metabolism; Brian D. Piening et al., 2018, Cell Systems; Nathan Price et al., 2017, Nature Biotechnology) - guiding us to understand its pathophysiology based on extensive scale of molecular evidence.

For instance, in insulin resistant patients, decreased uptake of mannose in liver tissue and thereby increased blood mannose was identified from multi-omics clues, such as transcriptome, epigenome and metabolome (Sunjae Lee et al., 2016, Cell Metabolism). In addition, metabolic dysfunctions of cardiovascular diseases and liver diseases were identified by multi-omics and modeling approaches, providing metabolic map that helps the design of therapeutic interventions (Stephen Doran et al., Briefings in Bioinformatics, 2021).

In this talk, the speaker will present recent systems biology-based approaches to understand health and disease, especially focusing on understanding human metabolism of chronic diseases. Such paradigm shift in future medicine will make a new promise of proactive medicine based on data-driven approaches.

CURRICULUM VITAE

윤소미

전남대학교 약학대학

[학력]

2002-2006 전남대학교 약학사
2006-2008 전남대학교 약학석사
2012-2015 전남대학교 의과학박사

[경력]

2015-2022 유전자제어 의과학센터 (전남대학교)

[관심분야]

심부전(Heart Failure preserved Ejection Fraction), 번역후수식화(Acetylation, Nitrosylation)

[논문]

1. Kim M, Kim YS, Ahn Y, Eom GH, Yoon S*. PSME4 determines mesenchymal stem cell fate towards cardiac commitment through YAP1 degradation. Korean J Physiol Pharmacol. 2023 Jul 1; 27(4): 407-416.
2. Kim YS, Kim M, Cho DI, Yoon S* et al. PSME4 Degrades Acetylated YAP1 in the Nucleus of Mesenchymal Stem Cells. Pharmaceutics. 2022;14(8):1659.
3. Yoon S, Kim M, Lee H, Kang G, Bedi K, Margulies KB, et al. S-Nitrosylation of Histone Deacetylase 2 by Neuronal Nitric Oxide Synthase as a Mechanism of Diastolic Dysfunction. Circulation. 2021; 143(19):1912-1925.
4. Yoon S, Kim M, Min HK, Lee YU, Kwon DH, Lee M, et al. Inhibition of heat shock protein 70 blocks the development of cardiac hypertrophy by modulating the phosphorylation of histone deacetylase 2. Cardiovascular research. 2018.
5. Yoon S, Kook T, Min HK, Kwon DH, Cho YK, Kim M, et al. PP2A negatively regulates the hypertrophic response by dephosphorylating HDAC2 S394 in the heart. Experimental & molecular medicine. 2018;50(7):83.

Nitric oxide-induced protein modifications in disease

Somy Yoon

College of Pharmacy, Chonnam National University, South Korea

Nitric oxide (NO) is known as a vasodilator, regulating vascular tone in smooth muscles, and traditionally considered cardioprotective in heart disease. However, recent investigations have uncovered a paradoxical involvement of NO in heart failure pathogenesis. Heart failure with preserved ejection fraction (HFpEF) presents a significant clinical challenge, with current therapeutic strategies, including NO donors, often proving ineffective. Despite the heterogeneous nature of HFpEF, diastolic dysfunction (DD) emerges as a prominent feature. Within DD, we explore the role of neuronal nitric oxide synthase (nNOS) in inducing S-nitrosylation of histone deacetylase 2 (HDAC2). Animal models, including SAUNA and mild transverse aortic constriction mice, revealed impaired diastolic function and exercise tolerance, accompanied by increased S-nitrosylation levels. Enhanced nNOS expression and NO production were observed in heart samples from both mice and patients with left ventricular hypertrophy. In vivo interventions targeting nNOS or HDAC2 S-nitrosylation ameliorated DD development, with nNOS knockout mice exhibiting resistance to SAUNA stress. HDAC2 S-nitrosylation was confirmed at specific cysteine residues (C262 and C274), and HDAC2 C262A/C274A mice maintained diastolic function under DD stimuli. Moreover, gene delivery of NRF2 or pharmacological denitrosylation with dimethyl fumarate attenuated DD in vivo. These findings unveil a novel mechanism underlying DD pathophysiology mediated by nNOS-induced HDAC2 S-nitrosylation, providing insights into the limitations of conventional NO enhancement therapies for HFpEF. Importantly, they propose reducing NO levels or promoting HDAC2 denitrosylation as a promising therapeutic strategy for refractory.

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Breakfast Symposium 1

4월 6일(토) 07:30-08:30 | Room 1

좌장 : 김명아(서울의대 순환기내과)

패널 : 구유정(서울의대 내분비내과), 김병규(인제의대 심장내과)

07:30-07:50	**The latest clinical trial of Atorvastatin, LODESTAR study** **정창희**(울산의대 내분비내과)
07:50-08:00	**Panel Discussion**

CURRICULUM VITAE

정창희

울산의대 서울아산병원 내과 교수

[학력]

1996-2002	고려대학교 의과대학
2010-2012	울산대학교 의과대학, 석사
2012-2014	울산대학교 의과대학, 박사

[경력]

2023-현재	울산의대 서울아산병원 내과 교수
2018-2020	University of Virginia, VA, USA, Visiting Scholar
2017-2023	울산의대 서울아산병원 내과 부교수
2013-2017	서울아산병원 내과 임상조교수
2010-2012	서울아산병원 내과 임상강사
2003-2007	서울아산병원 내과 전공의

[관심분야]

Diabetes, Obesity, Adipose Tissue, Therapeutics, Vascular complications

[논문]

1. Cho YK, Jung HN, Kim EH, Lee MJ, Park JY, Lee WJ, Kim HK, Jung CH: Association between. sarcopenic obesity and poor muscle quality based on muscle quality map and abdominal computed tomography. Obesity (Silver Spring) 2023.:31:1547-1557.
2. Kim HS, Lee J, Kim EH, Lee MJ, Bae IY, Lee WJ, Park JY, Kim HK, Jung CH: Association of Myosteatosis with Nonalcoholic Fatty Liver Disease, Severity, and Liver Fibrosis Using Visual Muscular Quality Map in Computed Tomography. Diabetes Metab J 2023, 47(1):104-117.
3. Lee SM, Lee JW, Kim I, Woo DC, Pack CG, Sung YH, Baek IJ, Jung CH, Kim YH, Ha CH: Angiogenic adipokine C1q-TNF-related protein 9 ameliorates myocardial infarction via histone deacetylase 7-mediated MEF2 activation. Sci Adv 2022, 8(48):eabq0898.
4. Jung HN, Cho YK, Kim HS, Kim EH, Lee MJ, Lee WJ, Kim HK, Jung CH: Association between hypertension and myosteatosis evaluated by abdominal computed tomography. Hypertens Res 2023. 46(4):845-855.
5. Jung HN, Kim MJ, Kim HS, Lee WJ, Min SH, Kim YJ, Jung CH: Age-Related Associations of Low Density Lipoprotein Cholesterol and Atherosclerotic Cardiovascular Disease: A Nationwide Population-Based Cohort Study. J Am Heart Assoc 2022, 11(9):e024637.

The latest clinical trial of Atorvastatin, LODESTAR study

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Breakfast Symposium 2

4월 6일(토) 07:30-08:30 | Room 2

좌장 : 한기훈(울산의대 순환기내과)

패널 : 김희동(순천향의대 심장내과), 양여리(가톨릭의대 내분비내과)

07:30-07:50	**Benefits of Rosuvastatin and Ezetimibe combination therapy beyond lipid lowering effect in T2DM** **문준성**(영남의대 내분비내과)
07:50-08:00	**Panel Discussion**

CURRICULUM VITAE

문준성

영남대병원 내분비대사내과

[학력]

2004 의학사, 영남대학교 의학과
2012 석사, 영남대학교 대학원
2019 의학박사, 영남대학교 대학원

[경력]

2024-현재 영남대병원 내분비대사내과 교수
2022-2023 대한당뇨병학회 총무이사
2024-현재 대한당뇨병학회 재무이사

[관심분야]

당뇨병, 췌도생물학, 연속혈당측정기

[논문]

1. Karunakaran U, Elumalai S, Chung SM, Maedler K, Won KC, Moon JS. Mitochondrial aldehyde dehydrogenase-2 coordinates the hydrogen sulfide - AMPK axis to attenuate high glucose-induced pancreatic β-cell dysfunction by glutathione antioxidant system. Redox Biol. 2024 Feb;69:102994.
2. Elumalai S, Karunakaran U, Won KC, Chung SM, Moon JS. Perfluorooctane sulfonate-induced oxidative stress contributes to pancreatic β-cell apoptosis by inhibiting cyclic adenosine monophosphate pathway: Prevention by pentoxifylline. Environ Pollut. 2023 Mar 1;320:120959.
3. Moon JS, Kim NH, Na JO, Cho JH, Jeong IK, Lee SH, Mok JO, Kim NH, Chung DJ, Cho J, Lee DW, Lee SW, Won KC. Safety and Effectiveness of Empagliflozin in Korean Patients with Type 2 Diabetes Mellitus: Results from a Nationwide Post-Marketing Surveillance. Diabetes Metab J. 2023 Jan;47(1):82-91.
4. Moon JS, Hong JH, Jung YJ, Ferrannini E, Nauck MA, Lim S. SGLT-2 inhibitors and GLP-1 receptor agonists in metabolic dysfunction-associated fatty liver disease. Trends Endocrinol Metab. 2022 Jun;33(6):424-442.

Benefits of Rosuvastatin and Ezetimibe combination therapy beyond lipid lowering effect in T2DM

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Breakfast Symposium 3

4월 6일(토) 07:30-08:30 | Room 3

좌장 : 박경수(서울의대 내분비내과)

패널 : 이은영(가톨릭의대 내분비내과), 조동혁(고려의대 순환기내과)

07:30-07:50	**Combination of DPP-4i and SGLT-2i: new insights from recent clinical trials** **김진화**(조선의대 내분비내과)
07:50-08:00	**Panel Discussion**

CURRICULUM VITAE

김진화

조선대학교병원 내분비대사내과

[학력]

1996-2002	조선대학교 의과대학 의학과 (학사)
2004-2006	조선대학교 대학원 의학과 (석사)
2006-2008	조선대학교 대학원 의학과 (박사)

[경력]

2007-2010	조선대학교병원 내분비대사내과 전임의
2014-2015	Massachusetts General Hospital (MGH), Harvard Medical School, Boston, USA Research fellow
2010-2018	조선대학교병원 내분비대사내과 조교수,부교수
2018-현재	조선대학교병원 내분비대사내과 교수

[관심분야]

1. Diabetes epidemiology
2. Diabetes vascular complication

[논문]

1. Kim JH, Lyu YS, Kim BS, Kim MK, Kim SY, Baek KH, Song KH, Han K, Kwon HS. Cardiorenal Outcomes and Mortality after SGLT2 Inhibitor Initiation in Type 2 Diabetes Patients with Prior Percutaneous Coronary Intervention History. Diabetes Obes Metab. 2024, Accepted.
2. Kim JH, Lyu YS, Kim MK, Kim SY, Baek KH, Song KH, Han K, Kwon HS. Repeated detection of non-alcoholic fatty liver disease increases the incidence risk of type 2 diabetes in young adults. Diabetes Obes Metab. 2024 Jan;26(1):180-190.
3. Lyu YS, Pyo JS, Cho WJ, Kim SY, Kim JH. Clinicopathological Significance of Papillary Thyroid Carcinoma Located in the Isthmus: A Meta-Analysis. World J Surg. 2021, 45(9):2759-2768.
4. Kim JH, Lyu YS, Kim SY. Impact of Social Jetlag on Weight Change in Adults: KNHANES2016-2017. Int J Environ Res Public Health. 2020, 18;17(12):4383.
5. Kim JH, Pyo JS, Cho WJ, Kim SY. The Effects of Bariatric Surgery on Type 2 Diabetes in Asian Populations: a Meta-analysis of Randomized Controlled Trials. Obes Surg. 2020, 30(3):910-923.

Combination of DPP-4i and SGLT-2i: new insights from recent clinical trials

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Breakfast Symposium 4

4월 6일(토) 07:30-08:30 | Room 4

좌장 : 최동훈(연세의대 심장내과)

패널 : 배재현(고려의대 내분비내과), 윤민재(서울의대 순환기내과)

07:30-07:50	The lower the better: atorvastatin's next option for dyslipidemia management **최강운**(영남의대 심장내과)
07:50-08:00	Panel Discussion

CURRICULUM VITAE

최강운

영남대학교 의과대학 내과학교실 심장내과 조교수

[학력]

2004-2011 영남대학교 의과대학 의학사
2016-2019 영남대학교 대학원 의학박사

[경력]

2018-2019 연세대학교 세브란스 심장혈관병원 임상연구조교수
2019-2021 동국대학교 경주병원 심장혈관내과 조교수
2022- 영남대학교 의과대학 내과학교실 심장내과 조교수

[관심분야]

심부전, 중환자의학

[논문]

- Relationship between myocardial perfusion and penetrating intramyocardial coronary artery in patients with apical hypertrophic cardiomyopathy

The lower the better: atorvastatin's next option for dyslipidemia management

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Luncheon Symposium 1

4월 6일(토) 12:20-13:20 | Room 1

좌장 : 김덕경(성균관의대 순환기내과)

패널 : 김민지(경북의대 내분비내과), 이상은(이화의대 순환기내과)

12:20-12:40	**Individual treatment strategy for dyslipidemia in high risk patients** **조준환**(중앙의대 순환기내과)
12:40-12:50	**Panel Discussion**

CURRICULUM VITAE

조준환

중앙대학교광명병원 순환기내과 부교수

[학력]

2009	중앙대학교 의학부 학사
2012	중앙대학교 의학부 석사

[경력]

2020.03-2022.02	중앙대학교병원 순환기내과 조교수
2022.03-2024.02	중앙대학교광명병원 순환기내과 조교수
2024.03-	중앙대학교광명병원 순환기내과 부교수

[관심분야]

관상동맥질환

[논문]

1. Rapid Progression of Coronary Atherosclerosis in Patients Taking an Oral Antitumor, Multikinase Receptor Inhibitor
2. Chromosomal abnormalities and atrial fibrillation and ischemic stroke incidence: a nationwide population-based study
3. Neutrophil-Lymphocyte Ratio in Patients with Acute Heart Failure Predicts In-Hospital and Long-Term Mortality
4. Association between obesity type and obstructive coronary artery disease in stable symptomatic postmenopausal women: data from the KoRean wOmen'S chest pain rEgistry (KoROSE)
5. Comparison of Characteristics and 3-Year Outcomes in Patients With Acute Heart Failure With Preserved, Mid-Range, and Reduced Ejection Fraction

Individual treatment strategy for dyslipidemia in high risk patients

Luncheon Symposium 2

4월 6일(토) 12:20-13:20 | Room 2

좌장 : 이인규(경북의대 내분비내과)

패널 : 오규철(가톨릭의대 순환기내과), 정경혜(충남의대 내분비내과)

12:20-12:40	**A paradigm shift in dyslipidemia treatment: case review** **홍준화**(을지의대 내분비내과)
12:40-12:50	**Panel Discussion**

CURRICULUM VITAE

홍준화

대전을지대학교병원 부교수

[학력]

2004 을지의대 학사
2008 을지의대 석사
2015 을지의대 내과 박사

[경력]

2014 충남대학교병원 전임의
2016 경북대학교병원 임상교수
현재 대전을지대학교병원 부교수

[관심분야]

비만, 당뇨병, 이상지질혈증, 갑상선, 골다공증, 부신

[논문]

1. Comparison of therapeutic efficacy and safety of sitagliptin, dapagliflozin, or lobeglitazone adjunct therapy in patients with type 2 diabetes mellitus inadequately controlled on sulfonylurea and metformin: third agent study. Diabetes Res Clin Pract. 2023 Aug 11;110872. doi: 10.1016/j.diabres.2023.110872.
2. Comparison of the effects of gemigliptin versus glimepiride on cardiac function in patients with type 2 diabetes uncontrolled with metformin: The gemi-heart study. Diabetes Obes Metab. 2023 Aug;25(8):2181-2190. doi: 10.1111/dom.15095. Epub 2023 May 3.
3. A randomized, active-controlled, parallel, open-label, multicenter, phase 4 study to compare the efficacy and safety of pregabalin sustained release tablet and pregabalin immediate release capsule in type II diabetic patients with peripheral neuropathic pain. Medicine (Baltimore). 2023 Apr 25;102(17):e33701.
4. Effects of Virtual Reality Exercise Program on Blood Glucose, Body Composition, and Exercise Immersion in Patients with Type 2 Diabetes. Int. J. Environ. Res. Public Health 2023, 20(5), 4178.
5. SGLT-2 inhibitors and GLP-1 receptor agonists in metabolic dysfunction-associated fatty liver disease: Trends Endocrinol Metab.

A paradigm shift in dyslipidemia treatment: case review

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Luncheon Symposium 3

4월 6일(토) 12:20-13:20 | Room 3

좌장 : 장학철(서울의대 내분비내과)

패널 : 김경수(차의대 내분비내과), 이수용(부산의대 순환기내과)

12:20-12:40	**Cutting edge care of pitavastatin with ezetimibe combination therapy** **박경일**(동아의대 순환기내과)
12:40-12:50	**Panel Discussion**

CURRICULUM VITAE

박경일

동아대학교 병원

[학력]

2001년 인제대학교 의과 대학 의학과 학사
2011년 중앙대학교 의과대학 의학과 석사
2017년 중앙대학교 의과대학 의학과 박사

[경력]

2009년-2011년 서울대학교병원 순환기내과 임상 강사
2022년-현재 동아대학교 의과대학 의학과 교수
2023년-현재 동아대학교 의과대학 의학과 대학원 책임교수

[관심분야]

관상동맥질환, 중재시술

[논문]

1. Comparison of 3- to 6-Month Versus 12-Month Dual Antiplatelet Therapy After Coronary Intervention Using the Contemporary Drug-Eluting Stents With Ultrathin Struts: The HOST-IDEA Randomized Clinical Trial. Circulation. 2023;147(18):1358-1368.
2. Hemodynamic Changes in Chronic Liver Disease. Korean J Gastroenterol. 2023;82(5):209-212.
3. Comparison of efficacy and safety between third-dose triple and third-dose dual antihypertensive combination therapies in patients with hypertension. J Clin Hypertens. 2023;25(5):429-439.
4. Prasugrel dose de-escalation in diabetic patients with acute coronary syndrome receiving percutaneous coronary intervention: results from the HOST-REDUCE-POLYTECH-ACS trial. Eur Heart J Cardiovasc Pharmacother. 2023;9(3):262-270.
5. Impact of COVID-19 on Heart Failure Patients in South Korea. Int Heart J. 2021;62(5):1083-1090.

Cutting edge care of pitavastatin with ezetimibe combination therapy

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Young Investigator Sessions

Young Investigator Session 1

☞ 일시: 2024년 4월 5일(금) 15:30~17:00
☞ 장소: Room 1
☞ 발표: YIS1-1 ~ YIS1-6

Young Investigator Session 2

☞ 일시: 2024년 4월 5일(금) 15:30~17:00
☞ 장소: Room 2
☞ 발표: YIS2-1 ~ YIS2-6

Young Investigator Session 3

☞ 일시: 2024년 4월 5일(금) 15:30~17:00
☞ 장소: Room 3
☞ 발표: YIS3-1 ~ YIS3-6

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2024 **S**pring Congress **o**n **L**ipid & **A**therosclerosis of KSoLA

YIS1-1

Establishment of an anti-angiogenic effect and anti-cancer therapeutic adaptation model of KAI1 in lipid rafts

채정환*, 조민국, 윤태훈, 최건, 김유지, 권유욱
서울대학교병원 순환기/심장내과

Objectives: There is limited knowledge regarding the endogenous inhibitors of angiogenic growth factors. In this research, we uncovered a new endogenous anti-angiogenic factor expressed within pericytes, shedding light on its mechanism and clinical implications.
Methods: We observed a significant increase in angiogenesis in Kai1 knockout mice, prompting us to examine the anti-angiogenic function of Kai1 through in vitro and in vivo. KAI1 KO mouse Cell line used Immunofluorescence analysis Angiogenesis assay Spheroid ex vivo analysis.
Results: KAI1 shows anti-angiogenic effects. In Kai1 knockout (Kai1-/-) mice, there was observed an accelerated retinal vascularization compared to wild-type (WT) mice, as evidenced by immunofluorescence images of the retinal vascular. KAI1 localizes at the lipid rafts of Pericytes, which depends on palmitoylation. The expression of Kai1 in the membranous or lipid-raft fraction, being robust in 10T1/2 cells and minimal in MS1 cells. Pericytes exhibited flotillin-enriched planar lipid rafts, while ECs displayed the caveolae type. Inhibition of palmitoylation using 2-bromopalmitate (2-bp) notably hindered Kai1 localization in the lipid raft or membrane of 10T1/2 cells. Through Gene Set Enrichment Analysis (GSEA), Lif levels were markedly decreased in Kai1 knockout primary pericytes. KAI1 has therapeutic potential for inhibiting cancer angiogenesis. spheres with KAI1 knockdown pericytes exhibited the most angiogenic phenotype, while spheres containing wild-type pericytes treated with rhKAI1 displayed the most pronounced anti-angiogenic effects.
Conclusions: Angiogenesis is crucial for devising therapies for cancer and ischemic cardiovascular conditions. Our research validated that Kai1 knockout promoted angiogenesis like neonatal retina, affirming its anti-angiogenic properties. Through RNA-seq analysis, we discovered that KAI1 stimulated the production of LIF (leukemia inhibitory factor). Clinical relevance was affirmed through ex vivo models of cancer angiogenesis, where Kai1 notably inhibited tumor angiogenesis and growth.
Keyword: Angiogenesis, Lipid raft, Tumorigenesis

YIS1-2

TRPC6 loss of function leading to adipogenesis perturbation and metabolic disorder

Phan Anh Nguyen[2,3,4,5,6*], Kyu-Hee Hwang[1,2,3,4,5,6], Duyen Tran Thi Thuy[1,2,3,4,5,6], Kyu-Sang Park[1,2,3,4,5,6], Seung-Kuy Cha[1,2,3,4,5,6]

[1]Department of Physiology, Yonsei University Wonju College of Medicine, [2]Department of Global Medical Science, Yonsei University Wonju College of Medicine, [3]Mitohormesis Research Center, Yonsei University Wonju College of Medicine, [4]Institute of Mitochondrial Medicine, Yonsei University Wonju College of Medicine, [5]Institute of Lifestyle Medicine, Yonsei University Wonju College of Medicine, [6]Yonsei University Wonju College of Medicine, Yonsei University Wonju College of Medicine

Objectives: Metabolic diseases, including obesity and diabetes, commonly involve calcium signaling dysregulation. Our study focuses on the role of transient receptor potential cation channel subfamily C member 6 (TRPC6) in regulating systemic metabolism, mainly adipose tissue functions.
Methods: A whole-body TRPC6 deletion (TRPC6 KO) mouse model has been used to analyze phenotype and physical characteristics compared to control wild-type (WT) mice. The 8 week old (young) mice are carefully monitored in single cages until they reach 20 weeks old (adult). A comprehensive laboratory animal monitoring system (CLAMS) is used to measure food intake, locomotor activities, and respiratory exchange quotient. When reaching the desired age, the mice's body composition is analyzed by nuclear magnetic resonance (NMR) spectroscopy and then challenged with metabolic stress tests to measure glucose and insulin responses. Histological analysis and immunoblotting for key enzyme expression are done in the metabolic active tissues of mice. To further investigate adipogenesis, murine adipose tissue stromal vascular cells are isolated by enzyme digestion of tissue followed by in vitro differentiation.
Results: TRPC6 KO mice exhibit significant body weight gain and an obesity-like phenotype despite reduced food intake. This result suggests that the metabolic disorder is not a consequence of overfeeding. These mice also display glucose intolerance, insulin resistance, and excessive adiposity. In vitro differentiation further revealed diminished adipogenesis.
Conclusions: The loss of TRPC6 leads mice to develop an obese-like phenotype characterized by high adiposity, hypertrophic adipose tissues, and reduced sensitivity to insulin and glucose. Our data suggests that these changes stem from adipose tissue impairment. The perturbation of TRPC6 in white adipose tissue results in defective adipogenesis, leading to fewer mature adipocytes, thus promoting hypertrophic development and metabolic complications. Our findings indicate that TRPC6 could be a promising target for developing new strategies for treating and managing adipose tissues in obesity.
Keyword: Adipogenesis, Lipid metabolism, Obesity, Calcium signaling

YIS1-3

Effects of pressure on macrophages in atherosclerosis

최명렬[1*], 유진희[2], 김형함[2], 안용주[1]

[1]포항공과대학교 기초과학, [2]포항공과대학교 초음파공학

Objectives: Macrophages are one of the most important cells in atherosclerosis, but no much is known about how they are affected by changes in pressure caused by blood flow. The piezo1 channel, one of the most recently recognized mechanosensitive channels affected by pressure, has been the subject of active research, but there is limited understanding of how it changes in macrophages. The aim of this study is to investigate changes in the piezo1 channel by pressure and the changes in its calcium signal.
Methods: Safety-proven and easily controlled ultrasound was used to create pressure changes in macrophage cell line RAW264.7. The ultrasound parameters such as frequency and duty factor were used to find out calcium signal changes. To observe changes in calcium signal within macrophages, the calcium marker Fluo-4 was used. qPCR was used to determine macrophage polarizations in macrophages in response to ultrasound pressure.
Results: The calcium signals were detected within macrophages in response to changes in ultrasound pressure. In RAW264.7, the calcium changes occurred at the minimum pressure in the macrophage cell line. The macrophages showed an increase in the pro-inflammatory markers TNF-alpha and CD86 when pressure was stimulated.
Conclusions: Pressure on the macrophages by using ultrasound drives calcium signal changes. This insight may provide a new way to understand pressure-dependent changes in macrophages and it could possibly be targeted for blood-pressure related atherosclerosis.
Keyword: Macrophage, Ultrasound, Piezo1, Calcium elevation

YIS1-4

HK660S attenuates cardiac fibrosis and mitochondrial dysfunction in isoproterenol-induced mouse heart failure

Mario Albino Sozinho Indarua[1*], Hyoung Kyu Kim[1], Trong Kha Pham[1,2], To Hoai T. Nguyen[1], Hyeong Rok Yun[1], Jin Han[1]

[1]Inje University, Cardiovascular and Metabolic Disease Center,
[2]VNU University, Vietnam National University, Hanoi, Vietnam, Faculty of Biology

Objectives: This study aimed to investigate the potential therapeutic effects of HK660S (β-Lapachone) in isoproterenol - induced hypertrophy and heart failure model.
Methods: Isoproterenol (ISO) was used to induce cardiac hypertrophy and fibrosis in 8 weeks old and male C57BL/6 mice with dose of 100 mg/kg/day for 2 consecutive weeks after one week of HK660S-pre-treatment with 2 different doses of 20 mg/kg/day and 80 mg/kg/day. HK660S were given during ISO injection and maintained until 15 weeks of age. Body weight was checked every week and cardiac function was evaluated by echocardiography. After the treatment, the blood sample was collected, and followed heart tissue analysis.
Results: HK660S treatment alleviated cardiac dysfunction, hypertrophy, and fibrosis mice response to ISO. Blood creatine, AST, and ALT levels were not affected by ISO and HK660S treatment, while they slightly decreased the body weight. In addition, HK660S treatment enhanced mitochondrial function via activating NQO1 and AMPK/NRF2/HO-1 signaling pathway. Moreover, HK660S treatment ameliorated ISO-induced cardiomyocyte apoptosis. Furthermore, HK660S protected ISO-induced heart failure through the activation and phosphorylation of CaMKK2/CaMK4/CREB signaling pathway.
Conclusions: In current study demonstrates that HK660S improves cardiac function in ISO-induced heart failure model by reducing cardiac fibrosis and mitochondrial dysfunction via activating NQO1 and CaMKK2/CaMK4/CREB signaling pathway. These findings suggest that HK660S is expected to be a promising agent against cardiac fibrosis and heart failure.
Keyword: Heart failure, Cardiac fibrosis, Beta-lapachone, Mitochondrial function

YIS1-5

Vascular calcification is attenuated by mitochondrial fission modulation in a murine model

So Hee Kwon[1*], Min-Ji Kim[1], Zerwa Siddique[2], In-Kyu Lee[3], Jae-Han Jeon[1]

[1]Department of Internal Medicine, School of Medicine, Kyungpook National University, Kyungpook National University Chilgok Hospital, Daegu, Republic of Korea, [2]Department of Biomedical Science, Graduate School and BK21 Plus KNU Biomedical Convergence Programs, [3]Department of Internal Medicine, School of Medicine, Kyungpook National University, Kyungpook National University Hospital, Daegu, Republic of Korea

Objectives: Vascular calcification is a pathological consequence of many disease entities including atherosclerosis, diabetes mellitus, and chronic kidney disease. Vascular calcification is not merely a result of hypercalcemia and hyperphosphatemia, but involves an imbalance between osteogenetic signals and anti-calcifying protective mechanisms. Mitochondrial hyperfission in vascular smooth muscle cells (VMSCs) is one of the processes that lead to apoptosis and tissue calcium deposit formation. Thiamine (vitamin B1) is a well-known inhibitor of oxidative damage in nervous tissue, and has been reported to inhibit mitochondrial fission. This study investigated whether modulation mitochondrial function prevents vascular calcification in vitro and in vivo by using inorganic phosphate and cholecalciferol-induced vascular calcification models, respectively.
Methods: An in vivo thoracic aorta calcification model was established by inducing 4-week-old rats with 2.6mM inorganic phosphate and 2mM calcium chloride. 6-week-old male C57BL/6J mice were given a vitamin B1 derivative solution by oral gavage for 13 days, then aortic calcification was induced by subcutaneous injection of 5.5×10-5 IU/kg cholecalciferol for 3 days before sacrifice. Cells and tissues were stained for calcium deposition (van Kossa stain), apoptosis (TUNEL) and mitochondrial morphology (Mitotracker). Western blotting and quantitative real-time PCR were used to assess calcification pathways and mitochondrial function. Tissue calcium content was measured to quantify the extent of calcification. Mitochondrial oxygen consumption ratio (OCR) and mitochondrial membrane potential (JC-1 dye staining) were assessed to analyze quantified mitochondrial function.
Results: Calcium deposition in both in vivo and in vitro models were reduced, with decreased amount of mitochondrial fission and increased mitochondrial function. Restoring mitochondrial function downregulated expression of genes related to calcification and osteogenesis.
Conclusions: Prevention of mitochondrial dysfunction in VSMCs may be a viable therapeutic strategy for treatment of vascular calcification. Further studies on its mechanisms are warranted.
Keyword: Mitochondria, Calcification

YIS1-6

Role of calcium release-activated calcium channel protein 1 in brown and beige adipocytes

김수지*, 남궁준

원주연세대학교 의과대학 기초과학

Objectives: This study aimed to investigate the role of Calcium release-activated calcium channel protein 1 (Orai1) in brown adipose tissue (BAT) metabolism and its implications in thermogenesis and obesity.
Methods: Orai1 BKO mice were generated to specifically lack Orai1 in brown adipocytes. These mice were compared with wild-type (WT) controls in terms of body weight, fat mass, and metabolic rate using oxygen consumption measurements. Cold exposure tests were conducted to assess thermogenic response, with body temperature monitoring and thermal imaging. Histological analysis via H&E staining of BAT, along with protein expression levels of thermogenic (including Ucp1) and lipolytic (including HSL) genes, were evaluated. PKA phosphorylation status, cAMP levels, and the expression of adenylyl cyclases (with a focus on Ac3) were also measured.
Results: Orai1 BKO mice exhibited increased body weight and fat mass, along with a significant reduction in metabolic rate compared to WT mice. Cold exposure revealed impaired thermogenesis in Orai1 BKO mice, as evidenced by reduced body temperatures and decreased fat combustion in BAT. There was a notable reduction in the protein expression of thermogenic and lipolytic genes, as well as decreased PKA phosphorylation. cAMP levels failed to increase upon cold exposure in Orai1 BKO mice, and a significant downregulation of Ac3 expression was observed. Treatment with Forskolin restored metabolic rates in Orai1 BKO mice, suggesting that Orai1, in conjunction with Ac3, plays a critical role in transporting calcium to elevate cAMP levels, thereby activating PKA.
Conclusions: The findings suggest that Orai1 is integral to maintaining metabolic homeostasis and thermogenic capacity in brown adipose tissue, possibly through a mechanism involving calcium-mediated cAMP production and PKA activation. Further investigation into the Orai1-cAMP-PKA pathway could provide deeper insights into the regulation of energy expenditure and adiposity.
Keyword: Obesity, Calcium release-activated calcium channel protein 1

YIS2-1

Differential impacts of physical activity types on non-alcoholic fatty liver disease, sarcopenia and cardiovascular disease risk

So Ra Kim[1*], Eugene Han[2], Byung-Wan Lee[1,3], Eun Seok Kang[1,3], Bong-Soo Cha[1,3], Yong-ho Lee[1,3,4]

[1]Yonsei University College of Medicine, Department of Internal Medicine,
[2]Keimyung University Scool of Medicine, Department of Internal Medicine, [3]Yonsei University College of Medicine, Institute of Endocrine Research, [4]Yonsei University, Institute for Innovation in Digital Healthcare

Objectives: Although physical activity (PA) is recommended to improve metabolic health, the differential impacts of aerobic and muscle strengthening PA on non-alcoholic fatty liver disease (NAFLD), and sarcopenia and cardiovascular risks has not been elucidated.
Methods: We conducted a cross-sectional study using nationally representative data from 2007-2020 Korean National Health and Nutrition Examination Surveys (n=66,201). Aerobic PA (A-PA) was defined as ≥ moderate-intensity 150 min/week or high-intensity 75 min/week, and muscle strengthening PA (MS-PA) was defined as ≥ 2 days/week of muscle strength training. The multicomponent PA includes both APA and MSPA. Framingham Steatosis Index and NAFLD liver fat score were applied to determine NAFLD. High atherosclerotic cardiovascular disease (ASCVD) risk was defined as a 10-year ASCVD risk score >10%. Skeletal muscle was assessed by dual-energy X-ray absorptiometry, and sarcopenia was defined as the lowest quintile sarcopenia index value (0.876 for men and 0.585 for women).
Results: The prevalence of NAFLD was decreased in individuals with all types of PA compared to inactive individuals (24.7% vs. 23.2% vs. 23.4% vs. 21.0% for physically inactive, A-PA, MS-PA, and multicomponent PA, all P<0.05). Multivariate logistic regression analyses showed that the risk of NAFLD was lowest in people with multicomponent PA, compared to those with A-PA or MS-PA (OR=0.92, 95% CI=0.91-0.92 for A-PA, OR=0.82, 95% CI=0.81-0.82 for MS-PA, OR=0.75, 95% CI=0.74-0.75 for multicomponent PA). Among individuals with NAFLD, multicomponent PA was associated with lower ASCVD risk compared to other groups (OR=0.74, 95% CI=0.73-0.75 for A-PA, OR=0.70, 95% CI=0.68-0.64 for MSPA, OR=0.62, 95% CI=0.61-0.64 for multicomponent PA). The sarcopenia risk was also decreased among physically active individuals with NAFLD (OR=0.77, 95% CI=0.76-0.77 for A-PA, OR=0.97, 95% CI=0.96-0.98 for MS-PA, OR=0.57, 95% CI=0.57-0.58 for multicomponent PA).
Conclusions: Individuals with PA have lower risk of NAFLD, sarcopenia and ASCVD. The risk reduction was more prominent in individuals with multicomponent PA.
Keyword: Physical activity, Non-alcoholic fatty liver disease, Sarcopenia

YIS2-2

Effect of bifidobacterium lactis supplementation on lipid profiles of obese women: a 12-week randomized controlled trial

강민지[1,2*], 강연지[1,2], 이미지[1,2], 임현정[1,2]

[1]경희대학교 동서의학대학원 의학영양학과, [2]경희대학교 임상영양연구소

Objectives: Evidence on the efficacy of Bifidobacterium lactis (B. lactis) in improving lipid profiles in obese women is limited despite its proposed potential as a probiotic intervention. This study aims to investigate the effects of B. lactis supplementation on lipid profiles in obese women.
Methods: This randomized controlled trial was conducted at Kyung Hee University Medical Center from March 2022 to January 2023. 99 participants aged between 20 and 65 years with a body mass index (BMI) ranging from 25 to 30 kg/m^2 were enrolled, with a total of 93 completing the trial, comprising a test group of 46 participants (receiving over 5.0*109 CFU per day) and a placebo group of 47 participants. Subjects adhered to their normal dietary and physical activity routines throughout the study. Evaluations of serum triglyceride (TG), total cholesterol (TC), high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), and the calculation of the atherogenic index of plasma (AIP) were conducted at baseline and after 12 weeks.
Results: Significant reductions in TG and AIP were observed in the test group compared to the placebo group (TG: -15.09±63.30 mg/dL vs. 12.74±60.07 mg/dL, p=0.0088; AIP: -0.04±0.23 vs. 0.07±0.22, p=0.0145), although AIP decreased in the placebo group as well (from 0.27±0.31 to 0.33±0.30, p=0.0473). However, no significant differences were observed in TC, HDL-C, or LDL-C between the two groups. Subgroup analysis focusing on postmenopausal women indicated greater reductions in TG and AIP in the test group compared to placebo (TG: -18.52±68.37 mg/dL vs. 11.67±58.27 mg/dL, p=0.0152; AIP: -0.05±0.23 vs. 0.07±0.22, p=0.0131).
Conclusions: B. lactis may have a beneficial effect on lipid profiles, particularly in reducing serum TG concentration and AIP level in obese women.
Keyword: B. lactis, Obesity, Women

YIS2-3

Weight cycling accelerates nonalcoholic fatty liver disease (NAFLD) progression through activation of IGFBP7

Shindy Soedono[1,2*], Hoang Nguyet Dan Vo[1], Jiyeon Chang[1], Yuri Song[2], Vivi Julietta[1], Yuha Joo[1], Hadia Nawaz[2], Maria Averia[1], Yeonwoo Choi[2], Okgyu Kim[2], Kae Won Cho[1,2]

[1]순천향대학교 의생명융합학과, [2]순천향대학교 의생명연구원

Objectives: Weight loss intervention is the current standard for MAFLD/MASH treatment. However, combatting obesity-associated MAFLD and atherosclerosis can lead to large fluctuations in body weight, referred to as weight cycling or yo-yo dieting. Recent studies implicated weight cycling as an independent factor to hasten MASH and worsened metabolic status, but the underlying mechanisms of MASH development are not well understood.
Methods: We utilized a diet-switch model involving normal diet (ND) and high-fat diet (HFD) to induce obesity, weight loss (WL), re-challenged HFD (RCHFD), short-term HFD (STHFD), or long-term HFD (LTHFD). Metabolic parameters were assessed through biochemical and histological analysis, and liver macrophages were quantified with flow cytometry.
Results: While body weights were comparable between the HFD-challenged (STHFD) and HFD re-challenged (RCHFD) groups, RCHFD displayed aggravated metabolic and liver profiles, including higher blood glucose levels, glucose intolerance, increased hepatic TG accumulation, and greater collagen deposition. RCHFD had higher numbers of pro-inflammatory liver macrophages, which were induced by HFD and persisted in WL periods. In another MASH model using CCl4 injection, we also observed worsened liver fibrosis with hastened collagen deposition and elevated pro-inflammatory and macrophage activation markers in RCHFD CCl4 compared to STHFD CCl4 group. Notably, co-culture of liver macrophages isolated from WL and HFD group with hepatic stellate cells (HSCs) LX-2 resulted in increased smooth muscle actin expressions, a marker of liver fibrosis. Additionally, we found that liver macrophages from formerly obese mice produced higher amounts of IGFBP7, and treatment of HSCs with IGFBP7 led to the up-regulation of smooth muscle actin.
Conclusions: These results indicate that worsened MASH progression during weight cycling could be attributed in part to the sustained liver macrophage activation and their product IGFBP7, which induces HSCs activation. Targeting liver macrophage IGFBP7 may serve as a promising biomarker and early therapeutic intervention for metabolic diseases associated with obesity.
Keyword: Obesity, Weight cycling, Weight loss, MASH, Liver macrophage

YIS2-4

Fenofibrate use is associated with reduced risk of heart failure outcomes in patients with type 2 diabetes treated with statins: a propensity-matched cohort study

김지윤[1*], 김남훈[2], 이지윤[2], 김신곤[2]

[1]삼성서울병원 내분비내과, [2]고려대학교 안암병원 내분비내과

Objectives: To investigate the association of fenofibrate use with heart failure (HF) outcomes in type 2 diabetes (T2D) patients on statin therapy within a real-world setting.
Methods: In a nationwide cohort database (2008-2021) in South Korea, patients with T2D (≥30 years) receiving statin therapy were matched up to 1:3 by propensity score into the statin plus fenofibrate group (n=46,771) and the statin only group (n=115,866). The primary outcome was a composite of hospitalization for HF or cardiovascular death. Cox proportional hazard model was used to assess the association of treatments with HF outcomes.
Results: During a median of 56.5 months, the incidence rate per 1000 person-years of the primary outcome was 6.27 in the statin plus fenofibrate group and 6.91 in the statin only group (adjusted HR, 0.83; 95% CI, 0.74-0.93; p=0.002). Adjusted HRs of hospitalization for HF and cardiovascular death were 0.85 (0.75-0.97), and 0.66 (0.49-0.88), respectively, favoring statin plus fenofibrate over statin only. The benefit of fenofibrate for the primary outcome was prominent in patients with a prior history of HF (HR, 0.70; 95% CI, 0.56-0.88; p=0.003), and those with atherogenic dyslipidemia (HR, 0.46; 95% CI, 0.34-0.62; p<0.001).
Conclusions: In this propensity-weighted cohort study, the addition of fenofibrate to statin was associated with a significantly lower risk of HF outcomes in patients with T2D, suggesting an additional cardioprotective role of fenofibrate.
Keyword: Fenofibrate, Statin, Heart failure, Cardiovascular death, Type 2 diabetes

YIS2-5

COVID-19 vaccination-related myocarditis, pericarditis and myopericarditis : an umbrella review of systematic reviews and updated meta-analyses

정재원[1*], 최근주[2], 김재택[1], 강현[2]
[1]중앙대병원 내분비내과, [2]중앙대병원 마취통증의학과

Objectives: Vaccination was a major modality for mitigating the COVID-19 pandemic. However, adverse effects following vaccination have been reported in the cardiovascular system such as thromboembolism or vaccination-related myocarditis, pericarditis and myopericarditis (VMP). Since the incidence of VMP has been limited to case reports or small-cohort studies, we conducted umbrella review of systematic reviews and updated meta-analyses to investigate the risk of myo/pericarditis in mRNA-vaccinated individuals.
Methods: A systematic search was performed of ovid-MEDLINE, ovid-EMBASE, and Scopus from inception to the end of February of 2024 and updated on July 1st. The overall confidence of the included systematic reviews using A MeaSurement Tool to Assess systematic Reviews (AMSTAR). The quality of evidence for each pooled outcome from the included systematic reviews using the Grading of Recommendations, Assessment, Development, and Evaluation (GRADE) system.
Results: The pooled incidence of VMP was 0.018/1000 doses. Where the incidence was reported in terms of number of people, sensitivity analyses were performed assuming that these people were vaccinated twice, and the pooled incidence was 0.016/1000 doses. Of note, incidence of VMP was decreased with increase of age. COVID-19 VPM incidence was 0.021/1000 after the first vaccination and 0.075/1000 after the second dose. The incidence of VMP in male and female was 0.088/1000 doses and 0.019/1000 doses, respectively. The incidence of VMP was statistically significantly increased in after COVID-19 vaccination group compared with control group.
Conclusions: Despite the substantial heterogeneity among studies, our study has reported that age and sex have a significant impact on the incidence of COVID-19 VMP. More adequately powered studies are needed.
Keyword: COVID-19 vaccination, Myocarditis, Pericarditis

YIS2-6

HK660S (β-lapachone) ameliorates diabetic cardiomyopathy by enhancing mitochondrial fuction, antioxidant capacity and calorimetries through activation of NQO1

Bui Van Nam[1,2*], Hyoung Kyu Kim[1], Pham Trong Kha[1], Jin Han[1]
[1]Cardiovascular and Metabolic Disease Center, Smart Marine Therapeutic Center, Department of Physiology, College of Medicine, Inje University, Busan, South Korea,
[2]Department of Stroke, 103 Hospital, Vietnam Millitary Medical University, Hanoi, Vietnam

Objectives: This study aimed to investigate the effects of the drug HK660S, a natural compound and newly developed β-lapachone analogue that increases mitochondrial function, mitochondrial intergrity, energy expenditure and antioxidant capacity in various tissues, on DCM and explore its underlying mechanisms.
Methods: To evaluate the effect of HK660S on mitochondrial function in the in vitro treatment: mitochondrial isolation and fiber permeabilization from heart tissue of C57BL/6 eight weeks male mice and four types of intact cell A549, HepG2, AC16, H9C2 were treated with different dose of drug to measure mitochondrial respiratory capacity then checked and compaired the NQO1 expression in different cell types and heart tissue. In the in vivo treatment: C57BL/6 seven weeks male mice were used high-fat diet (HFD) and low-dose streptozotocin 40 mg/kg/day (STZ) to established diabetic mouse modal. Mice were randomly divided into six groups: WT (wild type mice), WT+HK80, DM (diabetic mouse), DM+HK20, DM+HK80 and DM+Met (mice were fed HK660S 20mg/kg/day (HK20), 80mg/kg/day (HK80) and Metformin 200 mg/kg/day (Met) combined with HFD treatment for 10 weeks, STZ intraperitoneal (ip) injection for 5 consecutive days after 2 weeks of treatment to study the protective effect of HK660S on DCM.
Results: In the in vitro treatment, HK660S ameliorates mitochondrial function. In the in vivo models, HK660S-treated DM mice reduced heart and body weight, food and water intake, blood glucose levels and HbA1C, enhanced cardiac function and Insulin resistance (IR). In addition, treated-DM mice showed increased mitochondrial respiratory capacity and calorimetries. Furthermore, HK660S reversed the decrease in phosphorylated AMPK expression, altered the levels of proteins associated with mitochondrial biogenesis, increased mitochondrial content and antioxidant ability.
Conclusions: These data suggest that HK660S is at least partially cardioprotective. Mitochondrial function is important for cardiomyocyte survival in DCM, and mitochondrial dysfunction is a critical factor in DCM. HK660S restored impaired mitochondrial biogenesis and improved mitochondrial activity, content, and function in cardiomyocytes. Therefore, HK660S has the potential to serve as a novel therapeutic agent for the prevention and treatment of DCM.
Keyword: Diabetic mellitus, Diabetic cardiomyopathy, Mitochondria function, Beta-lapachone

YIS3-1

Prolactin improves insulin sensitivity by expanding adipose tissue reservoir capacity during and after lactation

Na Keum Lee*, Jisu Jung, Jung-Jae Lee, Sung Hee Choi, Joon Ho Moon

Department of Endocrionology, Seoul National University Bundang Hospital,
Seoul National University College of Medicine, Seongnam, Gyeonggi-do, South Korea

Objectives: Pregnancy imposes a substantial metabolic burden on women. Lactation is known to reduce postpartum type 2 diabetes mellitus and we have reported than lactation improves pancreatic β cell mass and function in postpartum women (Science Translational Medicine, 2020), but little is known on the mechanism how lactation can improve insulin sensitivity. We assessed metabolic phenotypes of lactating and previously lactated mice.
Results: Lactating and post-lactation mice at 3 and 6 weeks postpartum respectively demonstrated improved glucose tolerance and enhanced insulin sensitivity, despite exhibiting higher levels of adiposity. Through our exploration, we administered prolactin treatment in primary preadipocytes and observed a significant induction of proliferation. RNA-sequencing analysis of prolactin-treated 3T3-L1 cells revealed a marked increase in the expression of genes associated with the E2F pathway and G2M checkpoint genes, hinting at potential mechanisms underlying cellular proliferation.
Conclusions: Currently, our ongoing research is dedicated to unraveling the intricate molecular pathways triggered by prolactin, leading to the proliferation of preadipocytes. Initial findings suggest the involvement of the PRLR-JAK2-SRC-PDGFR-E2F pathway, a cascade potentially responsible for the observed proliferation in preadipocytes induced by prolactin. This study provides significant insights, proposing that the increased capacity of adipose tissue reservoirs triggered by prolactin might underlie the sustained enhancement of insulin sensitivity observed during lactation.
Keyword: Prolactin, JAK/STAT5, Adipocyte, Cell cycle

YIS3-2

RUNX3 negatively regulates agonists induced cardiac fibroblasts differentiation

Thi Van Trang Luong[1*], 이왕수[2], 양선부[1], 옥상미[1], 김재택[1]

[1]중앙대학교 의과대학 내분비내과, [2]중앙대학교 의과대학 순환기/심장내과

Objectives: Runt-related transcription factors (RUNX) genes encode transcription factors that bind DNA as components of the core-binding factor complex which activates and represses transcription of key regulators of growth, survival and differentiation pathways. In mammals, there are three RUNX family members: RUNX1, RUNX2 and RUNX3. While RUNX1-deficient mice were protected against adverse cardiac remodeling after myocardial infarction, the role of RUNX3 in myocardial remodeling has not been directly evaluated.
Methods: We performed cardiac pressure overload by infusion of angiotensin II (AngII) to C57BL6 male mice via osmotic minipump for 14 days. Cardiomyocytes and cardiac fibroblasts were isolated using retrograde Langendorff perfusion.
Results: Mice subjected to AngII infusion had increased perivascular and interstitial fibrosis compared to vehicle-infused mice. In addition, Tgfβ1, Tgfβ2, Tgfβ3, collagen I, and collagen III mRNA expression levels were significantly higher in AngII-infused hearts. Interestingly, expressions of RUNX3 in the hearts was increased after AngII infusion in parallel with αSMA expressions. RUNX3 expression was significantly increased in cardiac fibroblasts isolated from AngII-infused mice compared with those isolated from vehicle-infused mice. By contrast, cardiomyocytes isolated from vehicle-infused or AngII-infused mice expressed relatively low levels. To explore the role of RUNX3, we stimulated adult rat cardiac fibroblasts with TGFβ2 for 24 hr. TGFβ2 significantly increased RUNX3 protein levels accompanied by the upregulation of αSMA. To verify whether αSMA is a downstream target of RUNX3, we transfected RUNX3 siRNA to cells and treated them with TGFβ2 up to 24 hr. As a consequence of the knockdown of RUNX3, TGFβ2-induced upregulation of αSMA was not observed confirming that TGFβ2 regulates αSMA expression through RUNX3. In addition, RUNX3 knockdown represses TGFβ2 induced CTGF, collagen I, and collagen III expressions.
Conclusions: Our findings demonstrate that RUNX3 is a critical regulator of cardiac fibrosis and could be a potential therapeutic agent for the management of AngII-associated cardiac fibrosis.
Keyword: RUNX3, AngII, Fibrosis, Cardiomyocyte

YIS3-3

Urolithin A, a gut metabolite: alleviator for cardiac dysfunction in heart failure

송한결*, 김영민, 윤차현, 오창명

광주과학기술원 의생명공학과

Objectives: 이번 연구는 박출량 보존 심부전(HFpEF)에서 Urolithin A의 심장 기능 저하와 다양하게 나타나는 phenotype에 대한 효과에 연구 목적을 가진다.
Methods: 이 연구에서 박출량 보존 심부전(HFpEF)을 모방하기 위한 마우스 모델을 제작하고, Urolithin A (UA)의 효과를 평가하는 것을 목표로 하였다. 마우스 모델 제작을 위해 BL6J mouse를 사용하여 고지방식이 (60% fat/kCal)와 L-NAME (0.5 g/L)을 통해 대사적 스트레스와 고혈압 스트레스를 동시에 유도하였다. 이후 HFpEF 마우스 모델에서 UA 처리의 효과를 확인하기 위해 UA (250 mg/kg)를 고지방식이와 혼합한 특수 사료를 12주간 투여하였다. 최종적으로 UA에 대한 심장에 대한 유익한 효과를 확인하기 위해서 심장 초음파를 사용하여 심장의 수축 및 이완기능을 평가하였고, Dual energy x-ray absorptiometry를 사용하여 체성분의 변화를 평가하였다. 또한 조직 검사를 통해 심부전의 일반적인 phenotype인 fibrosis, 심장 비대(heart weight/tibia length)와 폐부종(wet lung/dry lung weight)의 변화를 확인하였으며, 전자현미경을 사용하여 미토콘드리아의 구조적 변화를 관찰하였다. 더불어 qPCR을 통해 UA 처리에 따른 mRNA 수준의 변화를 분석하였다.
Results: HFpEF 모델에서 Urolithin A (UA) 처리 후에 이완 기능 지표인 E/A 비율이 5.98에서 3.13으로 유의하게 감소하였다. 또한, UA 처리 그룹에서 좌심실 내부 직경과 벽 두께가 각각 2.76 mm에서 2.22 mm, 1.6 mm에서 1.4 mm로 감소하는 결과를 나타냈다. 심장 비대 지표인 heart weight/tibia length 값이 12.56에서 9.98로, 폐부종 지표인 wet/dry lung ratio가 9.66에서 5.18로 크게 감소하였다. 심장의 fibrosis의 경우에도 앞선 결과와 마찬가지로 UA를 처리한 그룹에서 22.38%에서 11.38%로 유의하게 감소하였고 dexa 분석을 통해 측정된 체성분 변화에서 body mass content, Bone area, Bone volume이 유의하게 증가하는 결과를 보였다. BIo-TEM 결과에서는 UA를 처리한 그룹에서 미토콘드리아의 섹션 당 면적이 감소하고, 미토콘드리아의 수가 증가한 것으로 나타났습니다. 또한 mRNA 수준에서 변화를 보면 심장 비대 및 lipotoxicity 관련 마커들의 유의하게 감소한 것으로 확인되었다.
Conclusions: 본 연구를 통해 Urolithin A 처리가 HFpEF 모델에서 심장 기능을 개선하고 심장 비대, 폐부종, fibrosis 등의 개선 효과를 보였다. 또한 체성분 변화와 미토콘드리아 구조의 변화도 관찰되었다. 추후 연구에서는 Urolithin A와 미토콘드리아의 관계성에 집중하여 mitophagy와 미토콘드리아 기능 개선에 따른 Urolithin A의 잠재적인 분자 매커니즘을 연구를 하고자 한다.
Keyword: Heart failure, Urolithin A

YIS3-4

6'-sialyllactose inhibits LPS-induced macrophage inflammation via regulating Nrf-2-mediated oxidative stress and inflammatory signaling pathways

Hami Yu[1*], Yujin Jin[1], Lila Kim[2], Kyung-Sun Heo[1]

[1]충남대학교 약학대학 약리학과, [2]GeneChem Inc. 회사

Objectives: Macrophages are central to the onset of cardiovascular diseases, including atherosclerosis, as they accumulate in vessel walls and trigger sustained local inflammatory responses characterized by the release of chemokines, cytokines, and matrix-degrading enzymes. Emerging reports have suggested that 6'-sialyllactose (6'-SL) has the potential to reducing the inflammatory response by regulating the immune system. In this study, we investigated the role of 6'-SL in lipopolysaccharide (LPS)-stimulated acute inflammatory responses using RAW 264.7 cells and a mouse acute inflammation model.
Methods: In vivo, ICR mice were pretreated with 6'-SL at 100 mg/kg for 2 h, followed by intraperitoneal injection of LPS at 10 mg/kg for 6 h. In vitro, RAW 264.7 cells were pretreated with 6'-SL followed by LPS stimulation. Molecule mechanisms were explored by western blotting, qRT-PCR, and immunofluorescence assays.
Results: LPS-stimulated p38 and Akt phosphorylation as well as p65 nuclear translocation were effectively inhibited by 6'-SL treatment. We further discovered that 6'-SL has an inhibitory effect on tissue damage marker MMP9 and inflammatory cytokine and chemokine, including IL-1ß and MCP-1 among the transcription targets, as a result of the regulation of NF-κB activation stimulated by LPS. 6'-SL reduced dihydroethidium-detected reactive oxygen species (ROS) via the activation of p38 and Akt signaling pathways. On the other hand, 6'-SL restored LPS-downregulated nuclear factor erythroid 2-related factor 2 (Nrf2) and cell apoptosis, similar to 10 μM specific inhibitors for p38 MAPK, SB203580 and Akt, LY294002. Consistent with the in vitro data, 6'-SL suppressed the expression of oxidative stress, MMP9, and MCP-1 in a mouse endothelium with macrophage activation induced by LPS.
Conclusions: Together, our findings suggest that 6'-SL is a promising agent for alleviating atherosclerosis by suppressing LPS-induced acute macrophage inflammation.
Keyword: 6'-sialyllactose, Acute inflammation, Macrophage, Oxidative stress

YIS3-5

LDL-콜레스테롤의 약물타깃 유전자와 관상동맥질환과의 관련성: drug target mendelian randomization study

지용호[1*], 신종원[2], 송태진[3]

[1]이화여자대학교 서울병원 첨단의생명연구원, [2]서울아산병원 진단검사의학과, [3]이화여자대학교 의과대학 신경과

Objectives: 높은 LDL-콜레스테롤(LDL-C)은 관상동맥질환의 잘 알려진 위험요인이다. LDL-C 저하와 관련 있는 약물 유전자(lipid lowering drug genes)로서 HMGCR, ACLY, NPC1L1, PCSK9, LDLR 등이 보고되어 있다. 최근, 서양인을 대상으로 LDL-C과 관련된 유전자기반 모방약(mimic drug)과 관상동맥질환과의 관련성 연구가 발표된 바 있으나(Ference et al., NEJM 2019) 아시아인을 대상으로 한 연구는 드물다. 이 연구는 한국인과 일본인 자료를 통해 LDL-C의 약물타깃 유전자와 관상동맥질환과의 관련성을 two sample Mendelian Randomization (MR) 방법을 통해 알아보았다.
Methods: 이전 연구에서 Ference 등이 보고한 LDL-C 관련 약물 타깃 gene 중 HMGCR 내 6개 Single Nucleotide Polymorphism (SNP), ACLY 내 9개 SNP, NPC1L1 내 5개 SNP, PCSK9 내 7개 SNP, LDLR 내 3개 SNP을 한국인의 LDL-C자료와 일본인의 관상동맥질환자료를 사용하여 MR 분석을 하였다. LDL-C은 관련 Genome-wide association studies (GWAS)데이터는 한국인 유전체역학연구(Korean Genome and Epidemiology Study, KoGES)를 통해, 관상동맥질환 자료는 일본 Biobank(Biobank of Japan, BBJ) 부터 summary statistics를 다운로드 받아 사용하였다. 이 연구에 사용된 도구변수는 lipid lowering 약물타깃에 위치하고 있는 SNP를 사용하였으므로 SNP 들 간에 높은 Linkage disequilibrium (LD)를 형성하고 있었다. 따라서 독립적인 SNP들을 션별하고자 r2<0.01을 조건으로 LD clumping 후 남은 SNP를 도구변수로서 활용하였다. 유전자 기반 LDL-C risk score의 감소와 관상동맥질환 위험도와의 MR 분석은 1) Wald ratio 방법을 사용하여 회귀계수(beta)와 표준오차(SE)를 산출하였고, 역분산가중치(inverse variance method, IVW) 방법을 적용하여 통합된 관련성을 추정하였다.
Results: HMGCR gene내 6개 SNP, PCSK9 gene내 SNP중 4개, LDLR gene 중 2개 SNP 가 LDL-C을 유의하게 감소했다. HMGCR gene 내 SNP들에 의해 LDL-C 이 10mg/dl 감소마다 관상동맥질환의 위험도는 약 11% 감소했다 (OR=0.898, 95% CI: 0.855-0.943). PCSK9 gene에 속한 SNP들에 의해 LDL-C 이 10mg/dl 감소마다 관상동맥질환의 위험을 약 20% 줄였다 (OR=0.802, 95% CI: 0.748-0.861). LDLR gene에 속한 SNP들에 의해 LDL-C 이 10mg/dl 감소마다 관상동맥질환의 위험도는 약 27% 감소하였다 (OR=0.737, 95% CI: 0.659-0.824).
Conclusions: LDL-C 저감과 관련된 약물 타깃 gene내 SNP들에 의해 추정된 LDL-C 감소는 관상동맥질환의 위험도를 유의하게 감소시켰다. 이 연구는 LDL-C 저감 약물을 직접 처방하는 임상시험의 대체 (proxy) 연구설계로 활용법을 제시한다.
Keyword: Low density lipoprotein cholesterol, Drug targeting, Mendelian randomization analysis

YIS3-6

Unveiling bone marrow macrophages: the unique population expressing ACKR1(DARC) in blood components

채정환, 최건, 윤태훈, 조민국, 김유지, 권유욱

서울대학교병원 순환기/심장내과

Objectives: The Duffy antigen receptor, also referred to as ACKR1 or DARC, is a protein consisting of seven transmembrane domains found within a specific subset of red blood cells, vascular endothelial cells, and epithelial cells. Recent scientific inquiries have revealed that DARC interacts with CD82 in hematopoietic stem cells (HSCs) sourced from human umbilical cord blood. This finding underscores CD82 role as a functional surface marker that defines long-term hematopoietic stem cells (LT-HSCs). The interaction between CD82 and macrophages expressing DARC has been observed in both mice and humans, playing a crucial role in maintaining the quiescent state of LT-HSCs and enhancing their ability for self-renewal.
Methods: The "ImageStream" represents an advanced approach within flow cytometry analysis, renowned for its exceptional resolution and the fusion of imaging capabilities with conventional flow cytometry techniques. With this methodology, it captures precise images of each individual cell, facilitating a more comprehensive scrutiny of cellular characteristics in comparison to traditional flow cytometry methods.
Results: We performed "ImageStream and found DARC, Cox2 and a-SMA Triple positive macrophages. These macrophages were found to play a crucial role in regulating the cell cycle of LT-HSC. Remarkably, only a very small fraction, constituting just 2% of the macrophage population in the bone marrow, were found to interact with and control the quiescence of LT-HSC. These triple-positive macrophages acted to prevent the differentiation of LT-HSC and served as a protective shield for the HSC population, safeguarding them from the harmful effects like reactive oxygen species attack.
Conclusions: We identified notable differences in the levels of ACKR1 mRNA expression across various subsets of macrophages, especially suggesting diverse potential functional roles of DARC+a-SMA+COX2+ triple positive macrophages. Our study provides a solid groundwork for future research aimed at unraveling the precise functions carried out by DARC within these specific cellular contexts.
Keyword: Blood components, Macrophage, Bone marrow

SoLA 2024

한국지질·동맥경화학회 춘계학술대회

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Mini-Oral Presentations

Mini-Oral Presentation 1

☞ **일시: 2024년 4월 5일(금) 17:10~18:00**
☞ **장소: 행사장 로비 (그랜드볼룸 포이어)**
Mini-Oral Presentation A~D

- Mini-Oral Presentation 1-1
  - 발표구역: A
  - 발표: MOP1-1-01 ~ MOP1-1-10
- Mini-Oral Presentation 1-2
  - 발표구역: B
  - 발표: MOP1-2-01 ~ MOP1-2-09
- Mini-Oral Presentation 1-3
  - 발표구역: C
  - 발표: MOP1-3-01 ~ MOP1-3-09
- Mini-Oral Presentation 1-4
  - 발표구역: D
  - 발표: MOP1-4-01 ~ MOP1-4-09

Mini-Oral Presentation 2

☞ **일시: 2024년 4월 6일(토) 11:10~12:20**
☞ **장소: 행사장 로비 (그랜드볼룸 포이어)**
Mini-Oral Presentation A~D

- Mini-Oral Presentation 2-1
  - 발표구역: A
  - 발표: MOP2-1-01 ~ MOP2-1-14
- Mini-Oral Presentation 2-2
  - 발표구역: B
  - 발표: MOP2-2-01 ~ MOP2-2-14
- Mini-Oral Presentation 2-3
  - 발표구역: C
  - 발표: MOP2-3-01 ~ MOP2-3-13
- Mini-Oral Presentation 2-4
  - 발표구역: D
  - 발표: MOP2-4-01 ~ MOP2-4-13

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2024 **S**pring Congress **o**n **L**ipid & **A**therosclerosis of KSoLA

MOP1-1-01 *Mini-Oral Presentation 1-1*

Proton pump inhibitors use in low-risk patients for upper gastrointestinal bleeding with ischemic stroke on DAPT: a nationwide cohort study

Minyoul Baik*, Jimin Jeon, Jinkwon Kim, Joonsang Yoo

Department of Neurology, Yongin Severance Hospital,
Yonsei University College of Medicine, Yongin-si, Gyeonggi-do, South Korea

Objectives: Current guidelines lack recommendations regarding the use of proton pump inhibitors (PPIs) for preventing upper gastrointestinal (UGI) bleeding among patients at low risk for UGI bleeding treated with dual antiplatelet therapy (DAPT) for ischemic stroke (IS). We aimed to evaluate the efficacy of PPIs in reducing the risk of severe UGI bleeding in this patient group.
Methods: This retrospective cohort study included patients at low risk for UGI bleeding admitted for IS between 2014 and 2018 and treated with DAPT. The study used a nationwide health claims database in Korea. The primary outcome was severe UGI bleeding within 1 year after IS. To evaluate the risk of severe UGI bleeding according to PPI use, we performed a multivariable Cox regression analysis. Propensity score matching (PSM) analysis and subgroup analyses were conducted for validation.
Results: Among 96,722 patients with IS at low risk for UGI bleeding on DAPT (mean age, 67.0 years; male: 63.0%), 16,084 (16.6%) were treated with PPIs. Within 1 year post-IS, 325 patients experienced severe UGI bleeding, and 479 experienced any UGI bleeding. PPI use was associated with a reduced severe UGI bleeding risk (hazard ratio: 0.64; 95% confidence interval: 0.46-0.90; P=0.010). This association was consistent in the subgroup and PSM analyses.
Conclusions: In IS patients receiving DAPT, PPI use reduced the risk of severe UGI bleeding by 36%, even among low-risk patients. However, the use of PPIs in this patient group was limited, highlighting the need for additional prospective studies.
Keyword: Ischemic stroke, Dual-antiplatelet therapy, Proton pump inhibitor, Upper gastrointestinal bleeding

MOP1-1-02 *Mini-Oral Presentation 1-1*

Cardiovascular health by life's essential 8 and subsequent coronary artery calcium among Korean adults: a prospective cohort study

안효은[1*], 전주은[2], 이혁희[2], 심지선[2], 김현창[2], 이호규[2]

[1]연세대학교 의과대학 보건학과, [2]연세대학교 의과대학 예방의학과

Objectives: The American Heart Association's (AHA) "Life's Essential 8" (LE8) is an updated method for quantifying cardiovascular health (CVH), with focus on the primordial prevention of cardiovascular disease (CVD). We investigated the association of CVH by LE8 with subsequent coronary artery calcium (CAC) in Korean adults.
Methods: We studied 1,242 healthy participants of the Cardiovascular and Metabolic Diseases Etiology Research Center (CMERC) prospective cohort study, with a baseline visit in 2013-2018 and a follow-up visit in 2022-2023. The LE8 CVH score was calculated as the average score of 8 metrics: diet, physical activity, nicotine exposure, sleep health, body mass index, blood lipids, blood glucose, and blood pressure, each scaled 0 to 100 points at baseline. The CVH scores of 80 to 100 were categorized as high CVH, 50 to 0) at the follow-up visit. Associations were evaluated based on odds ratios (OR) and 95% confidence intervals (CI) via multivariable logistic regression.
Results: At baseline, participants had median age 56 years, and 65.5% were female. After a median follow-up of 7 years, 503 participants had CAC>0. When the low CVH group was the reference, the multivariable-adjusted odds ratio (OR (95% CI) for follow-up CAC>0 was 0.37 (0.22-0.60) in the moderate CVH group and 0.29 (0.17-0.52) in the high CVH group (Table). Each 10-point higher LE8 CVH score was associated with 24% lower odds of having CAC>0 at follow-up (Table). The association of the LE8 CVH score and CAC was consistent across pre-specified subgroup and sensitivity analyses.
Conclusions: Higher CVH by LE8 score was associated with a lower risk of developing non-zero CAC among healthy middle-aged Koreans, suggesting the importance of promoting ideal CVH for the primordial prevention of CVD.
Keyword: Cardiovascular health, Life's essential 8, Coronary artery calcium, Primordial prevention

MOP1-1-03 *Mini-Oral Presentation 1-1*

Differential statin intensity and outcomes in patients following myocardial infarction with very low low-density lipoprotein cholesterol

오석[1*], 조경훈[1], 김민철[1], 심두선[1], 홍영준[1], 안영근[1], 정명호[1,2], 김주한[1]

[1]Department of Cardiovascular Medicine, Chonnam National University Hospital,
[2]Cardiovascular Center, Gwangju Veterans Hospital

Objectives: Despite increasing evidence on the benefits of statin therapy for acute myocardial infarction (AMI), differential outcomes in accordance with statin intensity have not been evaluated in patients with AMI and low-density lipoprotein cholesterol (LDL-C) levels <55 mg/dL. Therefore, this study aimed to compare the clinical outcomes of high- and moderate-intensity statin therapy in this population.
Methods: A total of 752 participants with AMI and LDL-C levels <55 mg/dL from a Korean nationwide multicenter observational cohort (2016-2020) were included and categorized into two groups: high-intensity statin group (n=384) and moderate-intensity statin group (n=368). The primary outcome was 1-year major adverse cardiac and cerebrovascular events (MACCEs). Propensity score matching (PSM) and Cox models were used to determine whether statin intensity independently influenced the primary outcome.
Results: Compared to the moderate-intensity statin group, the high-intensity statin group had a comparable risk of MACCE in all Cox models and PSM-adjusted analyses. The cumulative incidence of MACCE was comparable between the two groups (Table 1).
Conclusions: Statin intensity did not affect the clinical outcomes of patients with AMI and LDL-C levels <55 mg/dL. These results underscore the need for further investigations aimed at refining treatment strategies for this specific patient cohort, potentially reducing treatment-related burdens without compromising clinical effectiveness.
Keyword: Comparative study, LDL cholesterol, Myocardial infarction, Statins

MOP1-1-04 *Mini-Oral Presentation 1-1*

Global trends in clinical trials of dyslipidemia

김정국*

연세대학교 의과대학 의생명정보학교실

Objectives: Dyslipidemia remains a significant global health challenge, contributing to the increasing prevalence of cardiovascular diseases and impacting patient quality of life worldwide. In light of this, our study aims to provide a comprehensive analysis of the recent trends in clinical trials for dyslipidemia. Specifically, the research focuses on identifying patterns in annual study counts, geographical distribution, types of interventions tested, and the primary conditions under investigation. This analysis seeks to understand the evolving landscape of dyslipidemia research and to highlight areas of growing interest and potential gaps in current studies.
Methods: Data from ClinicalTrials.gov was extracted, covering studies first posted from January 1, 2021, to December 31, 2023, related to dyslipidemia. The analysis included categorizing studies by their posting year, geographical location, type of intervention, and condition addressed. Trends were identified based on the annual number of studies, predominant locations, intervention strategies, and primary focus conditions.
Results: The analysis revealed a stable interest in dyslipidemia research, with approximately 140 to 144 studies posted annually. Geographical distribution showed significant activity in Korea and the United States, particularly in Florida, Texas, and California. Pharmaceutical interventions dominated the research landscape, with 190 studies focused on drug therapies, followed by dietary supplements (53 studies) and behavioral interventions (35 studies). The conditions most frequently addressed were Dyslipidemias and Hypercholesterolemia, highlighting the primary focus on lipid-related disorders. Secondary emphasis was noted on associated conditions such as Hypertension, Obesity, and Familial Hypercholesterolemia.
Conclusions: The global research efforts in dyslipidemia are consistent, with a strong emphasis on pharmacological solutions. The geographical spread indicates a worldwide concern and research investment in addressing dyslipidemia. While drug therapies remain predominant, there is a noticeable shift towards exploring dietary and behavioral interventions, reflecting an integrated approach to managing dyslipidemia and its related conditions.
Keyword: Dyslipidemia, Clinical trials, Global trends

MOP1-1-05 *Mini-Oral Presentation 1-1*

Current status and clinical characteristics of familial hypercholesterolemia patients in Korea: a single center, real world experience

Moon-kyung Jung[1*], Kyung An Kim[1,2], Dongwoo Kim[3], Joonseok Kim[3], Jong-Chan Youn[1]

[1]Department of Cardiology, Seoul St. Mary's Hospital, The Catholic University of Korea, Seoul, Republic of Korea, [2]Department of Cardiovascular Medicine, Incheon St. Mary's Hospital, The Catholic University of Korea, Incheon, Republic of Korea, [3]College of Medicine, The Catholic University of Korea, Seoul, Republic of Korea

Objectives: Early diagnosis and initiation of lipid-lowering treatment is key to lowering cardiovascular risk in familial hypercholesterolemia (FH), but FH is frequently underdiagnosed. We aimed to investigate the current status of FH diagnosis and treatment in a large tertiary hospital in Korea.
Methods: Patients with either a diagnosis of FH (ICD-10 code: E7800) or had undergone testing for LDLR, APOB, or PCSK9 mutations were considered for inclusion. A total of 115 patients were retrospectively identified, and their demographic and laboratory characteristics as well as pharmacologic treatment patterns were analyzed.
Results: The mean age at diagnosis was 49.9 years, and 32 (27.8%) patients had a history of percutaneous coronary intervention or ischemic stroke. Coronary artery disease related symptoms (32.2%) and dyslipidemia (29.6%) were the most common reasons for initial visit. The majority of patients received their first diagnosis at the cardiology department (63.5%), followed by endocrinology (20.0%) and pediatrics (6.1%). The initial low-density lipoprotein cholesterol (LDL-C) level was 165.7±77.6 mg/dL (conversion to treatment-naïve LDL-C: 291.5±156.8 mg/dL), which decreased to 96.6±39.3 mg/dL after one year. Pathogenic mutations in the LDLR gene were found in 21 patients, and these patients had significantly higher maximal (p=0.046) and treatment naïve (p=0.015) LDL-C levels, lower platelet count (p=0.001), and higher total bilirubin (p=0.013). A high proportion of patients were treated with statins (92.9%) and ezetimibe (64.3%), but the use of PCSK9 inhibitors was low (5.4%). No major adverse cardiovascular events were noted during a mean follow-up duration of 19.1 months.
Conclusions: FH is frequently diagnosed late after the cumulative effect of hypercholesterolemia become evident. Although there remains room for improvement, current lipid-lowering therapy is effective in both lipid-lowering and cardiovascular event reduction, which again underlines the need for proper screening and identification of patients with FH.
Keyword: Familial hypercholesterolemia

MOP1-1-06 *Mini-Oral Presentation 1-1*

Age at menopause and risk of type 2 diabetes: a nationwide cohort study

남가은*

고대구로병원 가정의학과

Objectives: There has been limited evidence on the association between age at menopause and incident type 2 diabetes. We examined the association between age at menopause and premature menopause and type 2 diabetes development among Korean postmenopausal women.
Methods: Totally 1,125,378 postmenopausal women who underwent the health examination by the Korean National Health Insurance Service in 2009 were included. Multivariable Cox proportional hazards models were performed to determine the hazard ratios (HRs) and 95% confidence intervals (CIs) of incident type 2 diabetes.
Results: After a median follow-up of 8.4 years, 113,864 cases (10.1%) were newly ascertained as type 2 diabetes. Women with premature menopause had HR of 1.13 (95% CI 1.08-1.18) after adjusting for socioeconomic, cardiovascular, and reproductive risk factors. As age at menopause was younger, the risk of type 2 diabetes was increased (P for trend <0.002).
Conclusions: Women with a history of premature menopause or early menopausal age may have an increased risk of type 2 diabetes. Those with these reproductive factors require public attentions to prevent or delay type 2 diabetes.
Keyword: Type 2 diabetes, Age at menopause, Premature menopause

MOP1-1-07 *Mini-Oral Presentation 1-1*

Association of metabolic dysfunction-associated steatotic liver disease and handgrip strength with cardiovascular disease risk

So Ra Kim[1*], Eugene Han[2], Byung-Wan Lee[1,3], Eun Seok Kang[1,3], Bong-Soo Cha[1,3], Yong-ho Lee[1,3,4]

[1]Yonsei University College of Medicine, Department of Internal Medicine,
[2]Keimyung University School of Medicine, Department of Internal Medicine, [3]Yonsei University College of Medicine, Institute of Endocrine Research, [4]Yonsei University, Institute for Innovation in Digital Healthcare

Objectives: Although metabolic dysfunction-associated steatotic liver disease (MASLD) and handgrip strength (HGS) are associated with an unfavorable long-term prognosis, their association has not been studied. This study investigated the influence of MASLD and HGS on the risk of atherosclerotic cardiovascular disease (ASCVD) in the general population.
Methods: We used data from 20,611 individuals from the 2014-2019 Korea National Health and Nutrition Examination Survey. Steatotic liver disease was defined as the Framingham steatosis index ≥23 or simple NAFLD score ≥ 8. Low HGS was defined as <26 kg in men and <16 kg in women. The 10-year ASCVD risk score was assessed and a high-probability ASCVD was defined as >10%.
Results: When the Framingham steatosis index was used to define MASLD, the prevalence of MASLD and low HGS was 34.0% and 3.8%, respectively. Individuals with both MASLD and low HGS were at greater risk for high-probability ASCVD (OR=6.55, 95% CI=6.44-6.67, P<0.001) than those without MASLD. The impact of low HGS on high-probability ASCVD was higher when MASLD was accompanied by advanced liver fibrosis (OR=14.32, 95% CI=13.44-15.26, P<0.001). In the MASLD population, ASCVD risk was lower in individuals who had low HGS but engaged in regular exercise than in individuals with neither low HGS nor regular exercise (P<0.001). Similar findings were observed when a simple NAFLD score was used to define MASLD.
Conclusions: Individuals with MASLD and low HGS had a significantly higher risk of ASCVD than those without. Concurrent advanced liver fibrosis further increases the ASCVD risk among individuals with MASLD.
Keyword: Metabolic dysfunction-associated steatotic liver disease, Handgrip strength

MOP1-1-08 *Mini-Oral Presentation 1-1*

The changes of diagnostic rate of metabolic syndrome according to the lipid test intervals

Jihyun Ahn[3*], Chang-Ho Jihn[1], Hyeon Jin Jeon[2], Wang-Soo Lee[4]

[1]Kyung Hee University, Department of Industrial and Management Systems Engineering,
[2]Kyung Hee University, Department of Software Convergence, [3]Korea Medical Institute, Department of Internal Medicine, [4]College of Medicine, Chung-Ang University, Department of Internal Medicine

Objectives: With the extension of the lipid profile in national health screening interval from 2 to 4 years, the evaluation of the 5 components and 3 components of metabolic syndrome will alternate biennially. This study aimed to understand the impact of these changes on the diagnosis rates of metabolic syndrome.
Methods: Data from the National Health Insurance records from 2009 to 2017 were extracted, focusing on health screening tables. The analysis included the annual diagnosis rates of metabolic syndrome and the diagnosis rates when all 3 components (waist circumference, blood pressure, glucose), excluding triglycerides and HDL-cholesterol, were met for each year.
Results: Among the cumulative examinees from 2009 to 2017, 26.0% were identified as having metabolic syndrome. There was a rising trend from 24.6% in 2009 to 29.1% in 2017. However, when excluding the lipid profile, the proportion of individuals meeting all 3 of the other criteria for metabolic syndrome remained at 32% of actual cases with metabolic syndrome.
Conclusions: By extending the interval for lipid screening test, only about one-third of actual cases of metabolic syndrome could be diagnosed. Therefore, it is recommended that lipid test be conducted biennially in the national health screening, alongside other tests.
Keyword: Dyslipidemia, Metabolic, Screening

MOP1-1-09 *Mini-Oral Presentation 1-1*

Nationwide population-based cohort study indicates a decrease in pancreatic cancer incidence with the use of SGLT2 inhibitors

조윤경[1*], 김세희[2], 김명진[1], 이우제[1], 김예지[2], 정창희[1]

[1]서울아산병원 내분비내과, [2]서울아산병원 의학통계학과

Objectives: We aimed to investigate whether sodium-glucose cotransporter 2 inhibitors (SGLT2i) decrease the risk of gastrointestinal (GI) cancers in patients with type 2 diabetes (T2D).
Methods: Using an active-comparator, new-user cohort study design, and drawing from nationwide data spanning September 2014 through June 2020, we analyzed 79,423 new users of SGLT2i and 294,707 users of other glucose-lowering medications (oGLMs). We calculated hazard ratios (HRs) and 95% confidence intervals (CIs) for the incidence of gastrointestinal (GI) cancers, encompassing stomach, colorectal, liver, and pancreatic cancers, in addition to all-cause mortality rates. Sensitivity analyses were performed within a propensity score-matched (PSM) sample using the Cox proportional hazards model.
Results: Throughout the observation period, a total of 7,794 GI cancers and 10,726 deaths were identified. The use of SGLT2is was associated with a significant reduction in the incidence of GI cancers, with a multivariable-adjusted hazard ratio (HR) of 0.88 (95% confidence interval [CI]: 0.82, 0.95). Upon further examination of the risk for individual cancers, it was observed that only the incidence of pancreatic cancer was significantly lower in SGLT2i users compared to non-users, with a multivariable-adjusted HR of 0.70 (95% CI: 0.56, 0.88). In the PSM cohort, the cause-specific HR for pancreatic cancer was 0.72 (95% CI: 0.55, 0.92), confirming a reduced risk of pancreatic cancer among SGLT2i users.
Conclusions: For T2D patients, SGLT2i use was associated with a diminished pancreatic cancer risk. Future studies should ascertain the potential protective effect of SGLT2i against pancreatic cancer.
Keyword: Clinical science, Epidemiology, Clinical diabetes, Oral hypoglycemic agents

MOP1-1-10 *Mini-Oral Presentation 1-1*

Relationship between epicardial adipose tissue and metabolic syndrome

김보경[1*], 정주혜[2], 정유지[1], 김세홍[1]

[1]성빈센트병원 가정의학과, [2]여의도성모병원 가정의학과

Objectives: Epicardial adipose tissue (EAT) is closely related to the obesity-associated complications similarly to the characteristics of abdominal visceral adipose tissue (VAT). However, the association between EAT and metabolic syndrome (MS) is unclear. The purpose of our study was to evaluate the association of EAT with the cardiometabolic risk factors and MS.
Methods: Cardiac and abdominal computed tomography (CT) images were obtained in 256 asymptomatic subjects (110 subjects with MS and 146 without MS). Pixels with a threshold range of -190 to -30 Hounsfield units was identified as EAT and VAT. The concentrations of serum inflammatory cytokines and adipokines were also measured.
Results: The MS group had significantly lower adiponectin levels but significantly higher levels of resistin, leptin, tumor necrosis factor-alpha (TNF-α), interleukin-6 (IL-6), intercellular adhesion molecule (ICAM), monocyte chemotactic protein-1 (MCP-1), and oxLDL than the control group ($p<0.05$). EAT was significantly associated with MS in both men and women (OR 2.371; $p<0.001$) whereas the ORs between SAT and MS were not significant ($p=0.597$). The age-adjusted ORs between EAT and MS (OR of 8.359 in men and 3.183 in women, $p<0.001$) were higher than those of VAT (OR of 7.941 in men and 2.570 in women, $p<0.05$).
Conclusions: We demonstrated that EAT was associated with increased inflammation and oxidative stress, suggesting that EAT is an important determinant of MS. Therefore, EAT should be considered as a distinct a contributing factor of metabolic syndrome (MS) and coronary artery disease (CAD).
Keyword: Epicardial adipose tissue, Visceral adipose tissue, Subcutaneous adipose tissue, Metabolic syndrome, Inflammatory cytokine

MOP1-2-01 *Mini-Oral Presentation 1-2*

Mitochondria-associated membrane complex protein Ei24 modulate stored-operated calcium entry via STIM1 interaction

Duyen Tran Thi Thuy[1,2,3,4*], Phan Anh Nguyen[1,2,3,4], Subo Lee[1,2,3,4], Kyu-Hee Hwang[1,3,4], Ji-Hee Kim[5], Seung-Kuy Cha[1,2,3,4]

[1]Yonsei University Wonju College of Medicine, Department of Physiology, [2]Yonsei University Wonju College of Medicine, Department of Global Medical Science, [3]Yonsei University Wonju College of Medicine, Mitohormesis Research Center, [4]Yonsei University Wonju College of Medicine, Institute of Mitochondrial Medicine, [5]Soonchunhyang University College of Medical Science, Department of Occupational Therapy

Objectives: Store-operated calcium entry (SOCE) is the most important for maintaining calcium homeostasis by inducing calcium entry after Ca2+ store depletion in the ER in non-excitable cells. Stromal interaction molecules (STIM1), acting as ER Ca2+ sensors, and Orai1 proteins, form the structure of calcium release-activated calcium (CRAC) channels in the plasma membrane. Mitochondria-associated membrane (MAM) complex component Ei24 (etoposide-induced gene 2.4 kb) protein has been reported to directly interact with ER membrane Ca2+ channels. It remains unclear whether Ei24 has an impact on plasma membrane Ca2+ channels, such as the CRAC channel. Therefore, in this study, we examined the role of Ei24 in modulating SOCE by Ei24 by interacting with STIM1.
Methods: Besides basic molecular research techniques such as cell culture, exogenous gene overexpression, Western Blot, and quantitative PCR, we used CRISPR/Cas9 to generate an Ei24 knockout HeLa cell line and examined the physiological features of Ei24 using whole-cell patch-clamp, co-immunoprecipitation (Co-IP), and fluorescence recovery after photobleaching (FRAP).
Results: The CRAC channel currents were shown to be compromised by Ei24 overexpression, according to patch-clamp experiments. Furthermore, HeLa cells with the Ei24 knockout showed a notable increase in the SOCE response, and this effect can be reversed by rescue of Ei24. Co-immunoprecipitation studies demonstrated that STIM1 and Ei24 physically interact at the CRAC activation domain (CAD). Furthermore, it was shown by FRAP assays that Ei24 overexpression limits the kinetics of STIM1 mobilization.
Conclusions: Our findings not only shed light on the role of Ei24 in directing the Ca2+ sensor STIM1 but also underscore its crucial role in the optimal regulation of SOCE. Therefore, this research contributes significantly to understanding calcium homeostasis in non-excitable cells.
Keyword: CRAC channel, Orai1, FRAP assay

MOP1-2-02 *Mini-Oral Presentation 1-2*

Anti-obesity effect of Kimchi in differentiated T37i brown adipocytes by thermogenesis

윤예랑*

세계김치연구소 김치기능성연구단

Objectives: Kimchi is well-known to have a various health functionality including antioxidant, anti-inflammation, and anti-obesity effects. Previously, the anti-obesity effect of kimchi has been exhibited in 3T3-L1 adipocytes and high-fat diet-induced obese mice by measuring obesity-related gene expression. In recent days, the anti-obesity effect has been verified by thermogenesis. Herein, this study aimed to investigate the anti-obesity effect of kimchi by measuring the thermogenic effect in differentiated T37i brown adipocytes. This is the first study on the anti-obesity effect of kimchi by controlling thermogenesis.
Methods: Initially, the antioxidant activities of kimchi were examined by total antioxidant capacity (TAC) and ferric reducing antioxidant power (FRAP) level. Cell toxicity of kimchi with various concentrations (0, 50, 100, 250, 500, 1000, and 2500 μg/mL) was measured by cell counting-8 kit. After establishing the optimal differentiation condition for T37i adipocytes, intracellular triglyceride (TG) content was investigated. Lipid accumulation was observed and quantified in oil-red O (ORO) image. Thermogenesis- and obesity-related gene expression were investigated by quantitative real-time PCR. Lastly, thermogenesis-related protein was also investigated.
Results: As antioxidant activities, the TAC and FRAP levels of kimchi dose-dependently increased. The viability of kimchi in T37i adipocytes was over 90% until 1,000 μg/mL, showing low toxicity. In the differentiation condition, T37i preadipocytes efficiently differentiated in differentiation media containing 10% calf serum, 2 nM T3, and 100 nM insulin. Kimchi significantly suppressed intracellular TG levels and lipid accumulation compared to the control. In addition, kimchi significantly increased the thermogenesis-related gene/protein expression, whereas it significantly decreased the obesity-related gene expression.
Conclusions: Consequently, kimchi could prevent obesity by controlling obesity and thermogenesis in T37i brown adipocytes.
Keyword: Anti-obesity, Thermogenesis, Kimchi, Obesity

MOP1-2-03 *Mini-Oral Presentation 1-2*

Phosphate interferes with calcium-dependent filtration mechanism in podocyte

Dang Thi Ngoc Bao[1,2,3,4,5*], Nguyen Phan Anh[1,2,3,4,5], Seung-Kuy Cha[1,2,3,4,5]

[1]Department of Physiology, Yonsei University Wonju College of Medicine, [2]Department of Global Medical Science, Yonsei University Wonju College of Medicine, [3]Mitohormesis Research Center, Yonsei University Wonju College of Medicine, [4]Institute of Mitochondrial Medicine, Yonsei University Wonju College of Medicine, [5]Institute of Lifestyle Medicine, Yonsei University Wonju College of Medicine

Objectives: The dysregulated intracellular Ca2+ signaling in podocyte leads to disruption of the actin cytoskeleton and subsequent slit diaphragm malfunction, resulting in proteinuria - a hallmark of early kidney diseases. Alongside the well-established role of Trpc5/6 channels, emerging evidence suggests that Orai1-mediated store-operated Ca2+ entry (SOCE) contributes to maintaining Ca2+-dependent filtration integrity and mitigating podocyte injury. In diabetic nephropathy, increased podocyte Ca2+ levels due to reactive oxygen species (ROS) are observed. This ROS is elevated in chronic kidney disease (CKD), where hyperphosphatemia can be one of the causes for such kidney impairment. However, the precise mechanisms by which excessive inorganic phosphate (Pi) impacts SOCE-mediated Ca2+ signaling, podocyte actin dynamics, and filtration function, leading to proteinuria in CKD, remain incompletely understood.
Methods: Mouse podocyte, HEK293ft cell, GFP-Orai1/m-Cherry-Stim1 HEK293ft inducible cell; Generation of podocyte-specific *Orai1* knockout mouse model (Npsh2-CreOrai1$^{fl/fl}$); Western Blot; Quantitative real-time polymerase chain reaction (qPCR); Mitochondrial ROS measurement; Fura-2 measurement; Flexstation JC-1 mitochondria membrane potential.
Results: Our findings indicate that Pi enhanced mitochondrial Ca2+ uptake and disrupted membrane potential, potentially inducing mitochondrial ROS generation. Additionally, Pi promoted Akt-dependent exocytosis of Orai1 channels, increasing their surface expression. Consequently, dysregulated cytosolic calcium or ROS might interfere the actin cytoskeleton and reduced synaptopodin expression, impairing podocyte morphology and increasing albumin permeability. Notably, inhibition of Orai1 by GSK7975A partially restored actin cytoskeleton integrity and prevented synaptopodin dissolution. In vivo, podocyte-specific Orai1-deletion (Nphs2;Orai1fl/fl) mice administered with Pi showed less albuminuria compared to wild-type mice. Furthermore, short-term Pi exposure increased expression of GDF15, a stress marker that might provide negative feedback to mitigate the deleterious effects of ROS and intracellular Ca2+ dysregulation. However, prolonged Pi exposure irreversibly damaged the actin cytoskeleton, compromising podocyte viability and slit diaphragm integrity, ultimately leading to proteinuria.
Conclusions: In summary, our findings highlight the complex interplay of Pi in podocyte function, particularly in the context of Ca2+ regulation and filtration integrity.
Keyword: SOCE, Orai1, Calcium signaling, Actin cytoskeleton, Proteinuria

MOP1-2-04 *Mini-Oral Presentation 1-2*

Oxidative stress-mediated feedforward upregulation of TRPC6 initiates hepatic stellate cell activation and fibrosis

Kyu-Hee Hwang[1,3*], Phan Anh Nguyen[1,3], Ji-Hee Kim[2], Kyu-Sang Park[1,3], Seung-Kuy Cha[1,3]

[1]Yonsei University Wonju College of Medicine, Department of Physiology, [2]Soonchunhyang University, Department of Occupational Therapy, [3]Yonsei University Wonju College of Medicine, Department of Global Medical Science

Objectives: Hepatic fibrosis is primarily initiated by the activation of hepatic stellate cells (HSCs). This event begins with the disruption of Ca2+ signaling driven by reactive oxygen species (ROS), influenced by the fibrosis mediators TGFb and endothelin-1 (ET-1), which are known to induce oxidative stress. Our study aimed to elucidate the mechanism by which injury-produced ROS initiates Ca2+-dependent HSC activation.
Methods: We established in vivo model using bile duct ligation (BDL) and thioacetamide (TAA) administration to induce physical and chemical hepatic fibrosis. Subsequently, we employed DCFDA and MitoSOX to measure cytosolic and mitochondrial-derived ROS.
Results: This study identifies TRPC6 as the predominant Ca2+ channel and unravels the Ca2+ signaling mechanism during HSC activation. Our findings revealed an increase in the expression of TRPC6 and fibrosis markers, such as aSMA, during HSC activation. Notably, H2O2 directly increased TRPC6 current density and Ca2+ influx. Meanwhile, ET-1-mediated TRPC6 activation led to the depolarization of the mitochondrial membrane's potential, followed by the generation of mitochondrial ROS. Inhibiting TRPC6 reduced the expression of fibrosis markers in primary and cultured HSCs. These results underscore the critical role of ROS and TRPC6 in the pathogenesis of hepatic fibrosis.
Conclusions: In summary, our findings shed light on the critical role of ROS-mediated TRPC6 upregulation in initiating HSC activation, thereby establishing a feedforward signaling loop that exacerbates hepatic fibrosis. This insights into the hepatic fibrosis pathogenesis opens up potential therapeutic avenues targeting cirrhosis.
Keyword: ROS, TGFb

MOP1-2-05 *Mini-Oral Presentation 1-2*

Differential expression of SOCE-related genes in renal cell carcinoma subtypes revealed by transcriptome analysis

오지연[1,2*], 안보영[3], 황규희[1,2,4], 이태식[4], 차승규[1,2,4], 김지희[5]

[1]Yonsei University Wonju College of Medicine, Department of Physiology, [2]Yonsei University Wonju College of Medicine, Department of Global Medical Science, [3]University of California, Berkeley, USA, Department of Integrative Biology, [4]Yonsei University Wonju College of Medicine, Department of Convergence Medicine, [5]Soonchunhyang University, Department of Occupational Therapy

Objectives: In clear cell renal cell carcinoma (ccRCC), regulation of calcium ion transport is essential for cell migration and proliferation, primarily orchestrated by store-operated calcium entry (SOCE) that is well documented to involve ORAI1 and STIM1. While the impact of SOCE on tumor growth and metastasis has been extensively studied, the significance of SOCE in different kidney cancer subtypes remains elusive. Our findings reveal differences in gene expression and the pathophysiological relevance of SOCE-related genes across various types of RCC.
Methods: Transcriptome analysis was performed using two RNA-sequencing profiling sets of data obtained from The Cancer Genome Atlas Program (TCGA)'s Kidney Renal Clear Cell Carcinoma (KIRC) and Kidney Renal Papillary Cell Carcinoma (KRIP) projects. We used ORAI channels' small interfering RNA (siRNA) to observe cascade of SOCE by causing functional knockdown.
Results: By analyzing TCGA datasets, we identified differentially expressed SOCE genes, including ORAI channels (ORAI1~3) and STIM1/STIM2. Notably, between normal and cancerous states, the majority of SOCE-associated genes, except ORAI2, exhibited significant upregulation in ccRCC. On the other hand, papillary renal clear cells (pRCC) displayed substantial upregulation of ORAI channels (ORAI1-3) and STIM1, coupled with deregulation of STIM2. Moreover, distinct expression profiles of ORAI1 and ORAI3 emerged in RCC cell lines, with ORAI3 dominating in Caki-2 (pRCC primary cell line) and ORAI1 in Caki-1 (ccRCC metastatic cell line). The upregulation of SOCE due to 2-APB indicated ORAI3 as the major calcium channel for Caki-2, whereas reduced SOCE in Caki-1 suggested a prevalence of ORAI1. Subsequently, the imperative roles of ORAI1 and ORAI3 were confirmed through functional knockdown resulting in the disruption of cell migration in both cell lines.
Conclusions: In conclusion, our findings uncover that SOCE-related genes are differentially expressed within subtypes of RCC, which may provide important guidance in addressing the issue of kidney cancer metastasis.
Keyword: ORAI, STIM

MOP1-2-06 *Mini-Oral Presentation 1-2*

Regulation of autophagy via the lysosomal TRPML1 channel by WNK kinase

Subo Lee[1,2*], Kyu-sang Park[1,2], Seung-kuy Cha[1,2]

[1]Department of Physiology Yonsei University Wonju College of Medicine, Wonju, Gangwon-do, Republic of Korea, [2]Department of Global Medical Science Yonsei University Wonju College of Medicine, Wonju, Gangwon-do, Republic of Korea

Objectives: Autophagy is a crucial cellular degradation pathway that maintains cellular physiology and facilitates adaptation to metabolic stress. The TRPML1 lysosomal Ca2+ release channel plays a pivotal role in initiating autophagy. WNK kinases are known regulator of multiple ion channel/transporter homeostasis, particularly Na+ and K+. WNK1 have been implicated in autophagy inhibition through the suppression of the class Ⅲ phosphoinositide-3-kinase complex. However, the precise contribution of WNK signaling to TRPML1 regulation in context of autophagy remains unclear. In this study, we discovered that WNK kinases suppressed TRPML1, resulting in autophagy inhibition.
Methods: Through experiments conducted on HEK293 cells or HeLa cells expressing GCaMP3-labelled TRPML1, we observed that the overexpression of WNK1 or 4 led to the suppression of TRPML1-mediated peri-lysosomal Ca2+ release, indicating that multiple WNK kinases function as TRPML1 regulators.
Results: Notably, the suppression of Ca2+ release and subsequent nuclear translocation of TFEB by WNK1 were rescued by forced expression of a catalytically inactive mutant of WNK1 (kinase-dead mutant, K233M), emphasizing the crucial role of catalytic activity of WNK1 in TRPML1 activation. Furthermore, insulin, as an endogenous WNK1 activator, suppressed TRPML1-mediated Ca2+ release. This inhibition was effectively reversed by pretreatment with WNK463, an WNK inhibitor or diC-16-PI(3,5)P2. These results provide further support for the notion that WNK1 inhibits TRPML1 activity via suppression of class Ⅲ phosphoinositide-3-kinase.
Conclusions: Overall, our findings offer novel insights into the role of WNK kinase signaling in autophagy regulation, specifically targeting TRPML1-mediated Ca2+ regulation and lysosomal biogenesis.
Keyword: TFEB, Lysosomal biogenesis

MOP1-2-07 *Mini-Oral Presentation 1-2*

WNK1 is a novel culprit for hepatic stellate cell activation and the progression of hepatic fibrosis

Boyeong An[1*], Ji-Yeon Oh[2,3,4], Seung-Kuy Cha[2,3,4], Kyu-Hee Hwang[2,3,4]

[1]University of California, Berkeley, Department of Integrative Biology, [2]Yonsei University Wonju College of Medicine, Department of Global Medicine Science, [3]Yonsei University Wonju College of Medicine, Department of Physiology, [4]Yonsei University Wonju College of Medicine, Mitohormesis Research Center

Objectives: Gain-of-function mutations in the human WNK (With-No-Lysine[K]) gene have been identified as the cause of a specific type of arterial hypertension. The progression of hepatic fibrosis and the onset of portal hypertension, often associated with Na+ retention, are primarily instigated by the activation of hepatic stellate cells (HSCs). While WNK1 is recognized for its critical roles in renal ion transport, vascular tone, and angiogenesis, the precise engagement of WNK1 and its downstream effectors in HSC activation remains elusive. This study investigates the expression and function of WNK1 and its downstream regulators, including SPAK, OSR1, NCX1, and NKCC1, in the context of hepatic fibrosis and HSC activation.
Methods: Differential gene expression analysis (limma and DESeq2) was complemented by an inverse variance meta-analysis (META) to scrutinize the prevalence of WNK1 and the WNK-SPAK/OSR1-NKCC-NCX signaling pathway in hepatic fibrosis cases. Employing a thioacetamide mouse model and immortalized human hepatic stellate cells (hHSCs), we induced hepatic fibrosis and conducted a comprehensive array of in vivo and in vitro experiments.
Results: Significant expression of WNK1 was observed in activated hHSCs. Analysis of RNASeq data from public databases, along with corroborative wet lab experiments, revealed WNK1 upregulation and its signaling pathway components in fibrotic liver tissues and activated HSCs. Silencing and inhibiting WNK1 in hHSCs led to reduced fibrosis markers, such as α-smooth muscle actin, whereas WNK1 overexpression resulted in its exacerbation. Additionally, we explored the role of the WNK-SPAK/OSR1-NKCC-NCX signaling pathway in hepatic fibrosis by inhibiting NKCC and NCX.
Conclusions: These findings unveil the intricate role of WNK1 and its signaling molecules in hepatic fibrosis, providing new insights into the molecular mechanisms of the disease and suggesting potential therapeutic interventions.
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Keyword: Hypertension, Vasoconstriction, Angiogenesis

MOP1-2-08 *Mini-Oral Presentation 1-2*

Beneficial effects of Lobeglitazone, a new PPAR-γ agonist, on atherosclerosis and valve inflammation

박신희[1*], 박상은[1], 박규성[1], 안효석[2], 최재훈[1]

[1]한양대학교 기초과학, [2]가톨릭대학교 의정부성모병원 순환기/심장내과

Objectives: Lobeglitazone, one of the thiazolidinediones (TZDs), is an activator of PPAR-γ and is used as an oral medication for Type 2 diabetes. Although activated PPAR-γ, known for its anti-inflammatory properties, suggests that lobeglitazone could be effective in various diseases, its therapeutic effects on advanced atherosclerosis have not been confirmed. Therefore, this study investigated the effects of lobeglitazone on established atherosclerosis and valve inflammation in mice induced with hyperlipidemia.
Methods: The ApoE (-/-) mouse model of atherosclerosis was subjected to a high-fat, high-cholesterol Western diet from 7 weeks of age to induce atherosclerosis for a duration of 16 weeks after reaching adulthood. Following this, while maintaining the Western diet, the mice were divided into two groups: 1) control group receiving saline and 2) group receiving Lobeglitazone (10 mg/kg) administered orally once every other day for 8 weeks.
Results: Histological analysis of the aortic root in hypercholesterolemic mice following Lobeglitazone administration revealed a reduction in lipid content and macrophage infiltration in the lesions and plaques. Additionally, an increase in collagen content in the lesions due to Lobeglitazone treatment was observed. These findings suggest that Lobeglitazone administration contributes to the stabilization of lesions, indicating its potential for mitigating atherosclerotic progression.
Conclusions: This study revealed through histological examinations that Lobeglitazone administration reduces lipid accumulation and inflammatory cell infiltration within the lesions, while also showing an increase in the proportion of collagen, which plays a crucial role in lesion stability. While previous research has demonstrated that activation of PPAR-γ leads to increased expression of LXRα, ABCA1, ABCG1, and CD36, thereby enhancing oxLDL uptake or cholesterol efflux associated with decreased lipid deposition, further studies are needed to understand the mechanisms underlying the increase in collagen content.
Keyword: Atherosclerosis, PPARg agonist

MOP1-2-09 *Mini-Oral Presentation 1-2*

Creatine kinase tyrosine phosphorylation: a novel mechanism for heart protection in ischemic cardiomyopathy

Maria Victoria Faith Garcia[1*], Nammi Park[1], Jubert Marquez[1,4], Jeong Rim Ko[1], Hyoung Kyu Kim[1,2], Jin Han[1,2]

[1]Inje University, Cardiovascular and Metabolic Disease Core Research Center, [2]Inje University, Department of Physiology, College of Medicine, [4]De La Salle University, College of Science, Biology Department

Objectives: Despite being a major contributor to heart failure deaths globally, ischemic cardiomyopathy (ICM) can potentially be alleviated through ischemic preconditioning (IPC). This method involves short episodes of oxygen deprivation followed by a sustained period of oxygen deprivation and reperfusion. Recognizing the crucial role of mitochondria in heart disease progression, we investigated the influence of mitochondrial creatine kinase (CKMT2) during simulated ischemic injury in this study.
Methods: Sprague-Dawley rat hearts were used to simulate normal perfusion, I/R and IPC conditions via ex vivo Langendorff system. Isolated cardiomyocytes were analyzed using phosphoproteomics to determine candidate mitochondrial protein targets. Human cardiomyocyte cell line AC16 was used for in vitro study to define the cardioprotective role of CKMT2. This was done by determining mitochondrial cell viability, membrane potential, and ATP production of normoxic and hypoxic AC16 as well its related mitochondrial protein expression using western blot analyses. CKMT2 mutation was also examined to determine its effect under hypoxic and normoxic conditions of AC16 cells.
Results: Analysis of protein phosphorylation revealed that CKMT2 in the mitochondria is dephosphorylated during simulated ischemia/reperfusion but ischemic preconditioning maintains CKMT2's phosphorylated state. Interestingly, increasing the levels of CKMT2 protected heart cells from oxygen deprivation and reoxygenation by promoting cell survival, maintaining energy levels within the mitochondria, and reducing harmful free radicals. However, altering a specific part of CKMT2 (Y368) abolished this protective effect. Furthermore, increased CKMT2 levels enhanced mitochondrial function through a specific pathway, which was significantly blocked by the Y368 alteration. CKMT2 overexpression increased cell viability and mitochondrial ATP level against hypoxia/reoxygenation. Conversely, CKMT2 phosphomutation decreased cell viability and increased ROS during H/R. Increased mitochondrial function via the PGC-1α/ERRα pathway was observed upon CKMT2 overexpression.
Conclusions: Our results highlight the potential of targeting CKMT2 expression and phosphorylation as a unique strategy for future therapies against ischemic cardiomyopathy.
Keyword: Creatine kinase, Ischemia/reperfusion, Cardioprotection, Mitochondria, Phosphorylation

MOP1-3-01 *Mini-Oral Presentation 1-3*

Association between Korean healthy diet and dyslipidemia prevalence among Korean adults

김수현[1*], 정지나[2], 윤예진[1], 정효지[1,2]

[1]서울대학교 보건학과, [2]서울대학교 보건환경연구소

Objectives: This study aimed to investigate the association between adherence to a Korean healthy diet and the prevalence of dyslipidemia among Korean adults.
Methods: The Korean healthy diet was defined through literature reviews, statistical analyses, and expert consultations. A scoring system was developed to quantify adherence, assigning 1 point for adherence and 0 points for non-adherence to 13 dietary components: carbohydrates, sugar, fiber, protein, total-fat, saturated-fat, sodium, calcium, mixed grains, meat·fish·eggs·beans, vegetables, fruits, and dairy products. The sum of these scores represented the overall adherence level to the Korean healthy diet. The association between the adherence score and dyslipidemia prevalence was analyzed using data from the 24-hour dietary recall surveys of the 2016-2021 Korea National Health and Nutrition Examination Survey. Dyslipidemia was defined according to the National Cholesterol Education Program Adult Treatment Panel Ⅲ criteria, which includes total cholesterol, triglyceride, HDL-cholesterol, and LDL-cholesterol levels. Individuals meeting one or more of these criteria were identified as having dyslipidemia. Logistic regression analysis was conducted to examine the association using SAS.
Results: The analysis included 20,219 participants (8,577 men, 11,642 women), with 30.1% identified as having dyslipidemia. The average adherence score to the healthy diet was 6.21±1.71. The odds ratios (ORs) for dyslipidemia were calculated at 0.97 (95% CI, 0.95-0.99). Regarding specific lipid abnormalities, significant associations were observed for hypo-HDL-cholesterolemia and hypertriglyceridemia, with ORs of 0.97 (0.94-0.99) and 0.95 (0.92-0.98) respectively. In contrast, hypercholesterolemia and hyper-LDL-cholesterolemia showed no significant associations. Gender-stratified analysis highlighted that men exhibited a more pronounced association with ORs for dyslipidemia and hypertriglyceridemia at 0.97 (0.94-1.00) and 0.95 (0.92-0.99). However, women demonstrated a significant association only for hypertriglyceridemia, with an OR of 0.94 (0.90-0.99).
Conclusions: This investigation demonstrated that a higher adherence score to the Korean healthy diet is associated with a lower prevalence of dyslipidemia.
Keyword: Healthy diet, Dyslipidemia

MOP1-3-02 *Mini-Oral Presentation 1-3*

Association of dietary guideline adherence and lifestyle disease risks in Korean elderly

임영숙*, 오지수, 김혜영

용인대학교 식품영양학과

Objectives: This study aimed to investigate how compliance with dietary guidelines using a recently developed assessment tool (J Nutr Health 57(1):1-15, 2024) is associated with the risk of lifestyle disease in Korean elderly.
Methods: We surveyed 800 subjects aged 65 or older (400 men, 400 women) residing in five regions across the country to assess their adherence to Korean dietary guidelines. Data were analyzed to identify the mean and quartiles of dietary guideline adherence (DGA) scores. Differences in DGA scores between those with hypertension, diabetes, and obesity and those without were compared, and the risk of hypertension, diabetes, and obesity according to DGA quartiles was analyzed using multivariate logistic analysis adjusted for confounding factors.
Results: The average age of survey participants was 70.8 years. The prevalence of hypertension was 57.1% (58.5% men, 55.8% women), diabetes prevalence was 24.0% (23.5% men, 24.5% women), and obesity prevalence was 31.8% (27.5% men, 36.0% women). In both men and women, those with hypertension and diabetes had significantly lower DGA scores than those without, with obese women having significantly lower DGA scores than those without (58.2 vs. 61.0). The odds ratio for hypertension in the first quartile of DGA was 2.25 times higher than that in the fourth quartile, and the odds ratio for diabetes and obesity in the first quartile were 2.06 times and 1.91 times higher than those in the fourth quartile.
Conclusions: The dietary guideline adherence among the Korean elderly appears to be linked to the prevention of lifestyle-related hypertension, diabetes, and obesity. Funding Sources: This study was conducted by a research grant from the Rural Development Administration, Republic of Korea.
Keyword: Aged, Diet, Elderly

MOP1-3-03 *Mini-Oral Presentation 1-3*

심혈관계 예방에 도움이 되는 케일 분말을 활용한 기능성 양갱의 품질특성

최효경[1*], 강혜미[1], 백진경[2]

[1]을지대학교 임상영양전공, [2]을지대학교 식품영양학과

Objectives: 현대 사회에서 식생활의 서구화로 인해 곡물 및 채소 섭취량이 감소하고 지방, 육류 및 가공식품의 섭취가 증가하고 있으며, 이로 인한 심혈관계질환 발생 위험이 높아지고 있다. 이를 예방하기 위해서는 다양한 영양소가 풍부한 자연의 과일과 채소를 다양하게 섭취하는 것이 권장되고 있으며, 심혈관계질환 예방에 도움이 되며 페놀화합물,플라보노이드,식이섬유,비타민C,카로티노이드,비타민K,칼슘,인,철 등의 영양소를 풍부하게 함유한 케일을 활용하여 제조된 양갱의 품질특성 및 항산화 활성 평가를 통해 기능성 간식으로서의 가능성을 평가하고자 한다.
Methods: 물리·화학적 특성과 기호도를 평가하기 위해 케일,백앙금,프락토올리고당,설탕,한천,소금을 사용하여 케일과 백앙금의 비율을 0~8%로 조절하여 제조하였다. 제조된 양갱의 수분함량,pH,당도,색도,물성,항산화능,기호도평가를 진행하였으며, 모든 실험 결과는 SPSS를 이용하여 기호도평가를 제외한 실험 결과의 통계적 유의성은 일원배치분산으로 분석하여 평균±표준오차로 나타냈으며, $p<0.05$ 수준에서 LSD를 이용하여 시료 간의 사후검정을 진행하였다.
Results: 케일 첨가량에 따른 양갱의 수분함량과 당도는 유의적으로 증가하였고, pH는 유의적으로 감소하였다. 색도의 경우 명도와 적색도는 유의적으로 감소하고 황색도는 유의적으로 증가하였다. 물성의 경우 점착성,응집성,검성,씹힘성 모두 유의적으로 증가하였다. 항산화능의 경우 총 플라보노이드 함량, 총 폴리페놀 함량, ABTS 라디칼 소거능 모두 케일 첨가량이 증가할수록 유의적으로 증가하였다. 마지막으로 기호도 평가에서는 색,단맛,쓴맛,입안에서의촉감,입안에서의부드러움,전반적기호도가 4%까지 증가하다가 감소하는 경향을 나타냈다.
Conclusions: 케일은 십자화과 채소 중에서 식이섬유가 매우 풍부한데, 이러한 식이섬유는 수분결합력이 높아 양갱 내부의 수분 유출을 억제한다. 케일 첨가량이 증가할수록 양갱의 수분함량도 증가하는데 이러한 성질이 영향을 미친 것으로 사료되며 이는 부드럽고 촉촉한 식감을 제공하여 소비자 만족도를 높이는데 긍정적인 영향을 줄 것으로 예상된다. 케일은 구연산과 말산이 풍부하며, 이는 케일 첨가량이 증가할수록 양갱의 pH를 낮추는데 영향을 미친 것으로 판단된다. 이로 인해 양갱의 pH는 미생물 성장에 억제하는 범위로 조절되어 저장성을 향상시키는데 긍정적인 영향을 미칠 것으로 판단된다. 또한, 케일 첨가량이 증가할수록 양갱의 색은 점점 어두운 녹색으로 나타났다. 이러한 녹색으로 인한 식욕 자극이 높아져 소비자들에게 긍정적인 영향을 줄 것으로 예상된다. 케일 첨가량이 증가하면 양갱에 함유된 총 플라보노이드, 총 폴리페놀, ABTS 등의 항산화능이 증가하였다. 이러한 결과는 케일이 양갱의 부재료로 활용 시 기능성이 향상되고, 더 나아가 케일을 활용한 기능성 간식의 개발이 가능할 것으로 기대된다. 마지막으로 소비자의 기호도 평가를 통해 케일을 4%까지 첨가하는 것이 가장 이상적일 것으로 판단된다.
Keyword: Kale, Antioxidant, Functional, Yanggaeng

MOP1-3-04 *Mini-Oral Presentation 1-3*

브로콜리 새싹 분말을 활용한 건강한 머핀의 항산화 효과

강혜미[1*], 최효경[1], 백진경[2]

[1]을지대학교 식품영양학과 임상영양전공, [2]을지대학교 식품영양학과

Objectives: 한국인의 아침식사 결식률은 2022년 기준 34%로 그중 20대의 결식률은 59.2%로 가장 높았다. 아침식사 결식은 혈압, 이상지질혈증 및 심혈관질환 등의 유병률을 증가시키는 것으로 밝혀져 있다. 머핀은 부드러운 식감을 지닌 베이커리 제품으로, 간편식을 선호하는 젊은 소비자들에게 아침 및 간식 대용으로 많이 이용되고 있다. 브로콜리 새싹은 설포라판으로 전환될 수 있는 글루코시놀레이트가 다량 함유되어 있다. 설포라판은 항암, 항산화 등 생리활성이 우수한 기능성 물질로 동맥경화의 위험성을 줄이고 혈압 조절에 도움을 준다. 브로콜리 새싹은 브로콜리보다 높은 라디칼 소거능을 지녔음에도 불구하고 이를 이용한 식품 개발 연구는 미비한 실정이다. 이에 본 연구에서는 브로콜리 새싹 분말의 첨가가 머핀의 품질 및 항산화 활성에 미치는 영향을 확인하고자 한다.
Methods: 머핀은 중력분, 브로콜리 새싹 분말, 버터, 설탕, 달걀, 우유, 베이킹파우더를 이용해 제조하였으며, 샘플은 대조군인 0%와 브로콜리 새싹 분말 2%, 4%, 6%, 8% 첨가군으로 설정하였다. 제조된 머핀의 수분함량, pH, 당도, 색도, 조직감, 항산화 활성(polyphenol, flavonoid, DPPH, ABTS)을 측정하여 비교 분석하였다.
Results: 머핀의 수분함량과 pH는 브로콜리 새싹 분말의 첨가 비율이 증가함에 따라 유의하게 감소하였다. 수분함량은 시료의 수분 친화성에 따라 차이를 나타내는데, 브로콜리 새싹 분말이 머핀의 수분함량에 영향을 미친 것으로 사료된다. pH의 경우 브로콜리 새싹 분말 자체의 pH는 4.77로 분말 첨가량이 증가할수록 분말 자체 pH가 영향을 주어 낮아진 것으로 사료된다. 머핀 내부의 색도 측정 결과, 명도는 유의하게 감소하였고 적색도와 황색도는 유의하게 증가하였다. 머핀 내부의 조직감은 부착성, 경도, 탄력성, 씹힘성이 유의하게 감소하였다. 항산화 활성의 경우 폴리페놀의 범위는 525.77~888.23mg/mL로 분말 첨가 비율에 따라 증가하였으나 샘플 간에 통계적으로 유의한 차이를 보이지 않았고, 플라보노이드, DPPH, ABTS는 모두 유의하게 증가하였다.
Conclusions: 결과적으로 브로콜리 새싹 분말의 첨가는 머핀의 수분 및 pH를 감소시켰으며, 부착성, 경도, 탄력성, 씹힘성과 같은 머핀의 조직감에 영향을 미쳤다. 또한, 브로콜리 새싹 분말의 첨가는 머핀의 항산화 활성을 증가시키는 결과를 보였다. 이를 통하여 브로콜리 새싹 분말의 첨가가 머핀의 품질과 항산화 활성에 미치는 영향을 확인할 수 있었고, 높은 항산화 활성을 지닌 아침식사 대체 브로콜리 새싹 머핀의 개발이 가능할 것으로 기대된다.
Keyword: Broccoli sprouts, Muffin, Antioxidant, Characteristic

MOP1-3-05 *Mini-Oral Presentation 1-3*

Association between dietary fat intake and the lung function and metabolic related parameters among Korean men from a nationwide study

황수민[1*], 박지현[1], 김효진[1], 오수민[1], 김오연[2]

[1]Clinical Nutrition, Dept. of Health Science, Graduate School, Dong-A University,
[2]Dept of Food Science and Nutrition, Graduate School, Dong-A University, Busan, Korea

Objectives: Dietary pattern and nutrients composition are one of important factors affecting metabolic status. We investigated the relationship between dietary fat intake level and the risk of chronic obstructive pulmonary disease (COPD) using data from the Korean National Health and Nutrition Examination Survey (KNHANES) IV.
Methods: Among the 22,948 participants, 1604 males who met the criteria were finally included in the analysis. Data for basic information, anthropometric, biochemical and lung function parameters and nutrient intake information were collected. Subjects were divided into 9 groups according to their energy and fat intake levels (normal/normal, normal/low, normal/high, low/normal, low/low, low/high, high/normal, high/low, and high/high) based on the Dietary Reference Intake for Koreans.
Results: Compared with people consuming normal energy/high fat (15~30% of energy) (NN group), those consuming high energy/high fat (≥30%) (HH group) showed a significantly higher odds ratio (OR) of COPD before [OR:2.969, confidence intervals (CIs):1.661-9.486, P=0.002], and after the adjustment of confounding factors (OR:4.661, CIs:1.273-17.068, P=0.02). When HH group have metabolic syndrome (MetS), the risk of COPD became much higher than NN group without MetS (OR:11.069, CIs:1.601-76.537, p=0.015).
Conclusions: High energy consumption with high fat intake may increase the risk of COPD, which suggest the importance of energy intake with proper macronutrient composition for the metabolic disease and COPD.
Keyword: Dietary fat, Energy intake, Lung function, Metabolic

MOP1-3-06 *Mini-Oral Presentation 1-3*

Beneficial effect of short-term oligonol consumption on fatigue and oxidative stress response during maximal exercise test among healthy young men

김효진[1*], 박지현[1], 황수민[1], 오수민[1,2], 김오연[2]

[1]Clinical Nutrition, Dept. of Health Science, Graduate School, Dong-A University,
[2]Dept. of Food Science and Nutrition, Dong-A University

Objectives: We investigated whether the consumption of oligonol (OG), low-molecular weight polyphenols affect metabolic status indicating fatigue and oxidative stress response during maximal exercise test among healthy young men.
Methods: This study was a cross-over and blinded design with three phase by at least 2-week interval [placebo, single consumption of OG (S-OG) and 5-day consumption of OG (5-OG)]. Among the volunteers, 10 people who met the criteria were finally enrolled in the study. They were measured for the exercise ability, fatigue related metabolic parameters, and oxidative stress markers before, and immediately after the maximal exercise test, and also at the 30-min rest after the exercise. Particularly, heart rate and lactate, the fatigue related parameter were also measured at 1, 2, 3, 4, and 5 min immediately after the exercise.
Results: Anthropometric parameters and Exercise ability were not significantly different among the phases. Blood lactate levels at 30 min rest after the exercise test were significantly reduced in both S-OG and 5-OG groups compared with the placebo group. Increased malondialdehyde levels immediately after the exercise were significantly recovered close to the baseline level at 30-min in the OG groups, particularly in the S-OG group than in the placebo group.
Conclusions: Short term consumption of OG may improve fatigue and oxidative stress response during the maximal exercise test.
Keyword: Oligonol, Fatigue, Oxidative stress

MOP1-3-07 *Mini-Oral Presentation 1-3*

Carbohydrate intake levels and the risk of metabolic syndrome in Korean populations

박경[*]

영남대학교 식품영양학과

Objectives: In many Asian nations, including Korea, the traditional dietary patterns characterized by high carbohydrate intake, primarily from rice and other grains, have raised significant concerns regarding their potential implications on metabolic health. These concerns are particularly centered around metabolic syndrome (MetS) and its associated health complications, which include a cluster of conditions such as elevated blood pressure, high blood sugar levels, excess body fat around the waist, and abnormal cholesterol or triglyceride levels.
Methods: Our study involved analyzing data from 7,902 individuals enrolled in the Korean Association Resource (KARE) study, specifically those without prevalent MetS. Nutrient intakes, including carbohydrates and fiber, were assessed through a validated semi-quantitative food frequency questionnaire. This approach facilitated the calculation of the percentage of total energy derived from carbohydrates (P_CARB), and the carbohydrate-fiber ratio, which was used to determine carbohydrate quality. Blood samples from participants were collected after a fasting period of at least eight hours for laboratory analysis. The Cox proportional hazards model was utilized to estimate hazard ratios (HRs) and 95% confidence intervals (CIs), focusing on the relationship between P_CARB and the risk of developing MetS and its individual components, while controlling for carbohydrate quality.
Results: In the fully adjusted model, which accounted for carbohydrate quality as a covariate, individuals in the highest percentile of carbohydrate intake (P_CARB) showed a significantly increased risk of MetS, hypertriglyceridemia, hypo-HDL cholesterolemia, and high blood pressure, compared to those in the lowest P_CARB group. Spline curve analyses indicated that the risks for MetS and its components consistently escalated with increasing P_CARB, with all p-values for nonlinearity exceeding 0.05.
Conclusions: Our research suggests that higher P_CARB levels may elevate the risk of MetS and its associated conditions, such as hypertriglyceridemia, low HDL cholesterol, and high blood pressure, emphasizing the need for dietary awareness and potential modifications in populations with high carbohydrate.
Keyword: Metabolic syndrome, Cohort, Carbohydrate

MOP1-3-08 *Mini-Oral Presentation 1-3*

Study on the relationship between dietary intake and cataract incidence among Koreans aged 60 and above: focused on macronutrients -The Korea National Health and Nutrition Examination Survey 2015~2017-

최지영*, 박은주

경남대학교 식품영양학과

Objectives: Cataracts are a major cause of visual impairment and blindness, as the lens becomes cloudy, presenting a significant health concern in an aging society. Several recent reports have indicated that moderate carbohydrate consumption is associated with a lower risk of atherosclerosis and cataract formation. Additionally, apolipoprotein E (APOE) genotypes have been associated with the risk of developing cataracts. This study aimed to investigate the relationship between dietary intake of macronutrients and the incidence of cataracts.
Methods: We conducted a cross-sectional analysis using nationally representative samples of the elderly aged ≥60 years (N=1,619) from the Korean National Health and Nutrition Examination Survey (KNHANES, 2015-2017). The study utilized health and examination surveys along with a 24-hour food recall for dietary assessment. Variables such as sex, age, income, education, BMI, smoking and drinking habits, physical activity, and various health factors were analyzed and categorized into quartiles for uniform sample distribution. Nutrient intake was evaluated using the 24-hour recall method, focusing on energy, carbohydrates, proteins, and fats.
Results: The cataract incidence among Koreans aged 60 and above was found to be 51.8% of participants having cataracts (838), compared to 48.2% in the control group (781), with higher incidence rates observed in females compared to males. Results from the intake of macronutrients indicated a higher risk of cataracts with increased carbohydrate intake as a proportion of energy consumption. However, a higher proportion of protein and fat intake in energy consumption showed a lower risk of cataract development.
Conclusions: Reducing carbohydrate intake and increasing protein and fat intake may be a viable strategy for lowering cataract risk, particularly in aging populations. The study suggests a potential dietary approach to mitigate the prevalence of cataracts, highlighting the need for further research to explore the mechanisms behind these associations and to develop targeted nutritional guidelines for cataract prevention.
Keyword: Cataract, Food habits, Atherosclerosis

MOP1-3-09 *Mini-Oral Presentation 1-3*

Association between dietary inflammatory index and mortality from cardiovascular disease in patients with metabolic disorders: a population-based prospective cohort study

Dahyun Park[1,2*], HeeJu Jun[2,3], Garam Jo[4]

[1]Research and Management Center for Health Risk of Particulate Matter, Seoul, South Korea, [2]Department of Integrated Biomedical and Life Sciences, Graduate School, Korea University, Seoul, South Korea, [3]Interdisciplinary Program in Precision Public Health, Graduate School of Korea University, Seoul, South Korea, [4]Institute for Bio Materials, Korea University, Seoul, Korea

Objectives: The Dietary Inflammatory Index (DII), an extensively researched and literature-based tool, is utilized to quantify the impact of diet on markers of inflammation. While a correlation between DII scores and mortality has been established in numerous investigations, prospective research focusing on Korean cohorts is notably scarce. We investigate the prospective association between DII score and risk of cardiovascular disease (CVD) mortality using nationally representative repeated cross-sectional surveys.
Methods: This analysis included 40,596 participants (mean±standard deviation of age at baseline: 49.5±16.3 y) who were enrolled in the Korea National Health and Nutrition Examination Survey 2007-2015 and agreed to mortality follow-up through 31 December, 2019. Those diagnosed with cancer or cardiovascular disease at baseline, participants who were pregnant, and those who died within the first 2 years of follow-up were excluded from the analysis. The DII was calculated based on a 24-h dietary recalls. Cox proportional hazards models were used to estimate HRs and 95% CIs for mortality according to DII tertile.
Results: During ≤8.2 y of follow-up, 454 CVD deaths were identified. DII (OR for highest compared with lowest tertiles: 1.41; 95% CI: 1.23-1.61) were positively associated with CVD mortality. DII was associated with a statistically significant increase in CVD mortality in both woman (OR: 2.29; 95% CI: 1.51-3.47) and in men (OR: 1.65; 95% CI: 1.14-2.41). This association was stronger in patients with metabolic disorders such as hypertension (OR: 1.99; 95% CI: 1.42-2.80), Diabetes Mellitus (OR: 1.90; 95% CI: 1.09-3.33), Hypercholesterolemia (OR: 2.37; 95% CI: 1.13-4.97), Low HDL Cholesterolemia (OR: 1.85; 95% CI: 1.13-3.03).
Conclusions: In Korean adults, especially those with metabolic disorders, High DII levels are associated with higher CVD mortality.
Keyword: Diet, Inflammatory, Mortality, Cardiovascular disease

MOP1-4-01 *Mini-Oral Presentation 1-4*

Flow shear stress-associated KLF4 dysregulation: a pathogenic mechanism and potential therapeutic target in diabetic eye diseases

김수진[1,2*], 김유림[1,2], 최상욱[3], Hanjoong Jo[4], 이준엽[1,2]

[1]서울아산병원 안과, [2]울산대학교 의과대학, [3]중앙대학교병원 안과,
[4]Georgia Institute of Technology and Emory University, Department of Biomedical Engineering

Objectives: The choroid in the eye, supplying the retina and being the most vascularized tissue in the body, highlights the critical role of flow shear stress (FSS) in maintaining homeostasis. Although research on FSS has been concentrated on larger vessels, such as the aorta, to understand conditions associated with atherosclerosis, the small vasculatures of the choroid present a unique area for investigation. Notably, our recent findings indicate altered angiographic patterns in the choroids of diabetic patients, pointing towards diminished FSS as a possible pathological factor in these choroidal vasculatures.
Methods: We conducted ultra-widefield ICGA angiography in diabetic mice to confirm the changes in choroidal vascular density and hyperpermeability. In vitro experiments with choroid endothelial cells (CECs) were performed to modulate FSS with a rotating shaker. Kenpaullone, a small molecule KLF4 inhibitor, and APTO253, a small molecule KLF4 inducer, were utilized in vivo and in vitro to manipulate KLF4 expressions.
Results: Our in vivo studies showed a marked decrease in the expression of endothelial KLF4 and VEGFR3 in diabetic mice, indicative of impaired FSS. This was corroborated by ultra-widefield ICGA angiography, which revealed increased vascular density and hyperpermeability in the diabetic choroid. In vitro experiments showed that VE-cadherin, an adherens junction molecule, increased with FSS. Kenpaullone disturbed this dynamic, adversely affecting vascular stability and permeability. Furthermore, inhibition of KLF4 led to increased ICAM1 expression in CECs under FSS conditions, aligning with increased leukostasis observed in the diabetic choroid. Intravitreal administration of Kenpaullone mimicked the angiographic features of the diabetic choroid, whereas APTO253, a KLF4 inducer, appeared to alleviate these abnormalities.
Conclusions: Our research underscores the significance of FSS disruptions as a novel pathway for vascular dysfunction and disturbed homeostasis of diabetic eye diseases. Targeting endothelial KLF4 under abnormal FSS conditions emerges as a promising therapeutic strategy.
Keyword: Shear stress, KLF4, Flow, Diabetic eye

MOP1-4-02 *Mini-Oral Presentation 1-4*

Effects of time-restricted feeding on hepatic lipidomic profiles in middle-aged mice with long-term induced obesity

한예지[*], 권수진, 정자용

경희대학교 식품영양학과

Objectives: Time-Restricted Feeding (TRF) is a dietary approach that typically limits food availability to less than 10 hours per day, and has many metabolic benefits in obesity. However, few studies have investigated the effects of TRF on hepatic lipidomic profile changes in mice. In the present study, we employed untargeted lipidomics approaches to systematically identify changes in lipid mediators induced by long-term obesity and TRF.
Methods: C57BL/6J male mice were fed high-fat (HF) diet for 15 months, followed by HF diet ad libitum (HF-AL) or HF-TRF, 8 hours during the dark phase for 12 weeks. Control group was fed low-fat (LF) diet for the same period of study.
Results: The results showed that TRF significantly reduced body weight, fat mass and hepatic TG but significantly increased % lean mass without affecting food intake. In the principal component analysis (PCA) of hepatic TG, Control and HF-TRF groups overlapped, but separated from the HF-AL group. However, all three groups overlapped in the PCAs of different phospholipids. Lipidomic analyses of hepatic TG showed that 133 of the 187 TGs (71.1%) were significantly different among three groups, with 113 of them (85.0%) being higher in HF-AL group. When analyzing fatty acid composition in the TG, the major fatty acids were palmitate, oleate, linoleate, arachidonate, and docosahexaenoic acid (DHA), all of which were significantly higher in HF-AL compared to the control and HF-TRF groups. However, when expressed as a percentage of total fatty acid content, TRF diet significantly increased the content of DHA, an omega-3 fatty acid with anti-inflammatory effects, compared to LF diet. Interestingly, TRF increased the percentage of ether containing TG (TG ether), which are known to have antioxidant effects, over HF-AL diet.
Conclusions: In summary, TRF resulted in a different hepatic lipid profile, with increased beneficial fatty acids in middle-aged mice with long-term induced obesity.
Keyword: Time-restricted feeding, Lipidomics, Obesity

MOP1-4-03 *Mini-Oral Presentation 1-4*

On bacterial cellulose, β2 integrins (CD11/18) are important for the chemosensory migration and adhesion of PMN (polymorphonuclear leukocytes)

정택승*, 김도윤, 박용식

경희대학교 의과대학 기초의과학과

Objectives: To studies on the effect of leukocytes on BC(bacterial cellulose) as a prosthetic vascular graft, we investigated the contribution of the individual β2 integrin family in adhesion and migration of leukocytes under fMLP-induced vascular inflammation-like conditions on bacterial synthesized cellulose in vitro using PMN.
Methods: We cultured Gluconacetobacter xylinus to produce BC cells, and PMN was isolated from human peripheral blood. After incubating the separated PMN with various concentrations of fMLP, flow cytometry for CD11 and CD18 was conducted, and experiments for adhesion and migration of PMN to BC cells were conducted.
Results: Our findings demonstrate that Mac-1 (CD11b/CD18) plays a key role in the process of chemokinetic adhesion of PMN to BC. Also CD18 may be directly related to PMN adhesion and migration on BC. Despite the fact that expression of the α subunit is induced by chemokinetic stimulation, neither CD11a nor CD11c affected PMN migration. It has been shown that only CD11b can affect the migration of PMNs.
Conclusions: Our findings support the finding that the β2 integrin family(CD11/CD18) mediates chemokinetic attachment and migration of BC-adherent PMNs. Certain β2 integrins (α subunits) appear to mediate the adherent of PMNs to BC when stimulated with fMLP, but, chemokinetic shift was affected only by CD11b but not by CD11a or CD11c. Finally, CD18 play a essential role in both fMLP-induced adhesion and migration of PMN on BC surfaces. Furthermore, CD18 cross-linking may be have various functions of PMN, including adhesion, migration, and cytotoxicity.
Keyword: Gluconacetobacter xylinus, Bacterial cellulose (BC), β2 integrins (CD11/CD18), Polymorphonuclear leukocytes (PMN), N-formylmethionyl-leucyl-phenylalanine (fMLP)

MOP1-4-04 *Mini-Oral Presentation 1-4*

The role of small leucine zipper protein in prostate cancer progression

조성찬*, 김정한

Department of Biochemistry, The Catholic University of Korea College of Medicine, Seoul, South Korea

Objectives: Androgen and the androgen receptor (AR) have important roles in prostate cancer (PCa) development, and androgen ablation has been the main therapeutic option for the treatment of PCa. However, the transition mechanism from androgen-dependent to independent PCa after androgen depletion remains unclear. We investigated the distinct roles of small leucine zipper (sLZIP) in proliferation of androgen-dependent and indeproteinpendent PCa cells.
Methods: We investigated the expression of CyclinD3 mediated by sLZIP and the transcriptional activity of sLZIP in the CyclinD3 promoter. The binding of sLZIP to the Androgen Receptor (AR) and their colocalization within cells were confirmed. Furthermore, the expression and role of sLZIP in androgen-independent prostate cancer cells were validated using an animal model.
Results: Cyclin D3 is known to interact with AR and attenuates the ligand-dependent function of AR in PCa cells. sLZIP regulates the transcription of cyclin D3 by binding directly to the AP-1 region in the cyclin D3 promoter. sLZIP represses AR transcriptional activity by interaction with AR that is phosphorylated by cyclin D3/cyclin-dependent kinase11(p58), leading to the suppression of androgen-dependent proliferation of PCa cells. The expression level of sLZIP is elevated in androgen-independent PCa cells and advanced human prostate tumors. Knockdown of endogenous sLZIP suppresses proliferation of androgen-independent PCa cells. LNCaP cells transformed to androgen-independent PCa cells exhibit increased expressions of sLZIP and cyclin D3. Tumor formation is inhibited in nude mouse xenografts from two androgen-independent PCa cells that are stably transfected with sh-sLZIP.
Conclusions: Our findings indicate that sLZIP negatively regulates AR transactivation in androgen-dependent PCa cells and functions as a positive regulator in tumor progression of androgen-independent PCa. sLZIP contributes to the malignant phenotype of PCa and constitutes a novel therapeutic target for human PCa.
Keyword: Prostate cancer, Androgen receptor, sLZIP

MOP1-4-05 *Mini-Oral Presentation 1-4*

Role of placenta-derived exosomes on pancreatic beta cell

조예원[1*], 강효은[1], 이주희[2], 이민아[3], 김형석[1]

[1]충남대학교 의과대학 생화학과, [2]충남대학교병원 내분비내과, [3]충남대학교병원 산부인과

Objectives: The placenta plays an important role in orchestrating maternal adaptation to pregnancy. The placenta releases exosomes containing membranous and cytoplasmic substances such as proteins and miRNAs into the maternal circulation. Exosomal miRNAs are involved in the pathogenesis of gestational diabetes. In addition, exosomal miRNAs modulate beta cell function and viability. However, the role of placenta-derived exosomes on pancreatic beta cells is still unclear.
Methods: Exosomes from placenta chorionic villi explant culture media and plasma were isolated using ultracentrifugation and density gradient centrifugation. Exosomes were characterized by nanoparticle tracking analysis(NTA) and western blot. Exosomes were treated to human beta cell line(EndoCβH3). Cell proliferation was measured by immunofluorescence staining for KI67. Expression of marker genes was analyzed by qPCR. miRNA sequencing was performed to find candidate miRNAs that regulate beta cell proliferation.
Results: The isolated exosomes had diameters ranging from 50 nm to 200 nm and average peak diameter was 160nm. These small extracellular vesicles carried the exosome-specific markers CD63 and TSG101. Exosome-treated EndoC βH3 showed increased K67 positive cells, but this increase was not statistically significant. Expression of beta cell marker genes was not affected by exosome treatment. miRNA sequencing data showed that exosomal miRNAs were mainly originated from chromosome 19.
Conclusions: In this study, placenta-derived exosomes did not alter beta cell identity but increased proliferation in EndoCβH3. These data suggest that exosomes derived from the placenta could affect the compensatory proliferation of beta cell during pregnancy.
Keyword: Exosome, Beta cell, miRNA

MOP1-4-06 *Mini-Oral Presentation 1-4*

Role of serotonin in hepatic endoplasmic reticulum stress

이은지[1*], 황인선[3], 김형석[1,2]

[1]충남대학교 의과대학 의과학과, [2]충남대학교 의과대학 생화학교실, [3]대전보건대학교 바이오의약과

Objectives: Endoplasmic reticulum (ER) stress is caused by the accumulation of unfolded or misfolded proteins and highly associated with liver diseases such as metabolic dysfunction associated fatty liver disease (MAFLD). Previous studies have reported that ER stress plays a key role in progression of MAFLD. Serotonin, a neurotransmitter that regulates central and peripheral functions, has been found to affect development and progression of MAFLD. However, the role direct mechanism of serotonin in hepatic ER stress remains to be elucidatedunclear.
Methods: In this study, we found that gut specific deletion of Tph1 ameliorates hepatic ER stress in ethanol binge drinking mice model. Next, we induced ER stress by tunicamycin administration to both wild type (WT) mice and gut-specific Tph1 knockout (Tph1 GKO) mice, comparing ER stress levels in the liver.
Results: Ethanol binge drinking increases ER stress in the liver and production of 5-HT in the gut. 5-HT production is completely abrogated in gut specific Tph1 knock mice. Tunicamycin induced-ER stress is not improved in gut-specific Tph1 knockout mice liver under 1mg/kg administration condition.
Conclusions: In the present study, we have treated tunicamycin in Tph1 GKO mice, but it did not improve tunicamycin-induced ER stress. Thus, it is necessary to optimize the hepatic ER stress model and conduct further investigation.
Keyword: Serotonin, Endoplasmic reticulum, Hepatocyte

MOP1-4-07 *Mini-Oral Presentation 1-4*

Dipeptidyl peptidase-4 inhibitor evogliptin attenuates cardiac fibrosis and lipid accumulation in db/db mice

Trong Kha Pham[1,2*], Hyoung Kyu Kim[1], To Hoai T. Nguyen[1], Hyeong Rok Yun[1], Vu Thi Thu[2], Jin Han[1]

[1]Inje University, Cardiovascular and Metabolic Disease Center,
[2]VNU University of Science, Vietnam National University, Hanoi, Vietnam, Faculty of Biology

Objectives: This study aimed to investigate the potential therapeutic effects of evogliptin® (EVO), a dipeptidyl peptidase-4 (DPP-4) inhibitor used in the treatment of type 2 diabetes mellitus, on diabetic cardiomyopathy (DCM). The objective was to determine whether EVO could protect against DCM and elucidate its underlying mechanisms.
Methods: Eight-week-old diabetic and obese db/db mice were chosen as the experimental model. EVO was administered to the mice at a dose of 100 mg/kg/day through daily oral gavage for a duration of 12 weeks. Control groups included db/db mice receiving the vehicle and wild-type (WT) mice (C57BLKS/J) receiving equal amounts of the vehicle. Various parameters, including blood glucose, HbA1c levels, body weight, and blood lipid profile, were monitored. Echocardiography was used to evaluate both systolic and diastolic functions. Molecular analyses were conducted to investigate the impact of EVO on cardiac hypertrophy, fibrosis, lipotoxicity, and mitochondrial damage.
Results: EVO treatment resulted in significant reductions in blood glucose and HbA1c levels, improved insulin sensitivity, and ameliorated both systolic and diastolic functions in db/db mice. Additionally, EVO-treated diabetic mice exhibited attenuated cardiac hypertrophy and fibrosis. Mechanistically, EVO prevented cardiac lipotoxicity by suppressing the expression of key lipid metabolism genes (CD36, ACSL1, FABP3, PPARgamma, and DGAT1) and enhancing the phosphorylation of FOXO1. Furthermore, EVO positively influenced mitochondrial function and mitigated damage through the activation of PGC1a/NRF1/TFAM, promoting mitochondrial biogenesis. RNA seq analysis of the whole heart confirmed that EVO primarily affected differentially expressed genes (DEGs) related to lipid metabolism.
Conclusions: In conclusion, our study demonstrates that EVO improves cardiac function in diabetic mice by mitigating lipotoxicity and reducing mitochondrial injury. These findings suggest that EVO holds promise as a potential therapeutic option for treating diabetic cardiomyopathy, addressing an unmet need in current clinical practice.
Keyword: Dipeptidyl peptidase-4, Evogliptin, Diabetic cardiomyopathy, Cardiac fibrosis

MOP1-4-08 *Mini-Oral Presentation 1-4*

Characterization of diabetic nephropathy in early-onset type 2 diabetes mellitus using mouse model

이예지[1*], 윤재승[1], 안유배[1], 문민경[2], 고승현[1], 김규호[1]

[1]가톨릭대학교 내분비내과, [2]서울대학교 내분비내과

Objectives: Early-onset type 2 diabetes mellitus (T2DM) has a more rapid deterioration of kidney function than is seen in late-onset T2DM. However, exact mechanisms of rapid deterioration of kidney function in early-onset T2DM have yet to be elucidated. The aim of the study is to investigate mechanisms of rapid deterioration of kidney function in early-onset T2DM using mouse model of diabetic nephropathy.
Methods: Eight-week-old (early-onset) or 38-week-old (late-onset) male C57BL/6J mice received intraperitoneal injection of streptozotocin (STZ, 100 mg/kg) 3 times. From 2 weeks after STZ injection, mice were fed with high-fat diet for 6 weeks. Mice were sacrificed at 16 week or 46 weeks of age.
Results: Early-onset T2DM mice had significantly smaller kidneys compared with late-onset T2DM mice. Both early-onset and late-onset T2DM mice exhibited albuminuria, but there was no significant difference between 2 groups. Early-onset T2DM mice exhibited significantly higher level of urine 8-OHdG, but lower level of IL-6 compared with late-onset T2DM mice.
Conclusions: These findings suggested a critical role of oxidative stress in aggressive feature of diabetic nephropathy in early-onset T2DM.
Keyword: Type 2 diabetes mellitus (T2DM), High-fat diet, Diabetic nephropathy

MOP1-4-09 *Mini-Oral Presentation 1-4*

The function of adenylyl cyclase-associated protein 1 in vascular inflammation and atherosclerosis

김유지*, 조민국, 채정환, 윤태훈, 최건, 권유욱
서울대학교병원 순환기/심장내과

Objectives: Adenylyl cyclase-associated protein1 (CAP1), a human resistin receptor, mediates monocyte actions and links chronic inflammation to cardiometabolic diseases. However, the mechanisms of CAP1 and adenylyl cyclase (AC) in the regulation of the action of resistin have not been clarified and the role of CAP1 in endothelial cells is uncertain.
Methods: We used siRNA, adenoviral transfection, and CAP1+/- mice to examine the role of CAP1 in endothelial cells. The precise location of AC isoforms was determined by membrane fractionation experiment. We analyzed the role of CAP1 in trans-endothelial migration of monocytes in vitro as well as in vivo models.
Results: We demonstrated that CAP1 acts as a functional receptor of human resistin in human endothelial cells. AC isoforms 3 and 6 were predominant in endothelial cells and were localized in the lipid-raft layer of the plasma membrane along with caveolae, which is distinguished from that in monocytes. CAP1 in endothelial cells formed a complex with AC and caveolin to suppress AC in the quiescent state; upon resistin treatment, the CAP1-AC-caveolin assembly was disorganized so that AC was de-suppressed, while resistin directly activated AC through CAP1 in monocytes. Activation of the cAMP-PKA-NF-κB signaling pathway induced the expression of ICAM-1 and VCAM-1 in endothelial cells. In animal models, CAP1 heterozygosity in endothelial cells resulted in the increased trans-endothelial migration of the circulating monocytes and local inflammation.
Conclusions: Our findings reveal the molecular mechanism on how CAP1 activates AC in endothelial cells after binding with resistin and consequently contributes to trans-endothelial monocyte infiltration.
Keyword: Caveolin, Inflammation, Endothelial cell, Resistin, Adenylyl cyclase

MOP2-1-01 *Mini-Oral Presentation 2-1*

Beneficial effect of statin and ezetimibe on insulin resistance

홍준화*
대전을지대학교병원 내분비내과

Objectives: LDL lowering treatment is essential to prevent atherosclerotic cardiovascular disease. Although statin is first choice to lower LDL cholesterol, new onset diabetes is nettlesome, especially to prediabetes. Ezetimibe shows additive LDL lowering effect in combination with statin and also improves insulin resistance. In this study, we investigated the change of insulin resistance with atorvastatin and ezetimibe in prediabetic patients.
Methods: The participants are people of prediabetes and dyslipidemia with LDL over 130 mg/dl. We analyzed the differences of the metabolic parameters and HOMA-IR before treatment and 3 months after treatment of atorvastatin and ezetimibe.
Results: 113 patients with prediabetes and dyslipidemia were enrolled. Baseline LDL cholesterol level was 159.97±21.90 mg/dl. After 3 months with atorvastatin and ezetimibe combination therapy, LDL cholesterol level was lowered to 72.65±23.24 mg/dl (54.58%). HbA1c level didn't show significant change (5.69±0.27%, 5.67±0.33, p=0.556). Fasting glucose level was lowered from 104.05±9.83 mg/dl to 101.08±11.75 mg/dl (p=0.014). HOMA-IR was also lowered from 2.54±2.23 to 1.95±1.74 (p=0.017). Additionally, the improvement of insulin resistance was more prominent to patients with good responder of LDL cholesterol lowering (> 50%) and patients with higher insulin resistance (HOMA-IR > 2).
Conclusions: The combination therapy of atorvastatin and ezetimibe showed high LDL cholesterol lowering effect and improvement of insulin resistance. This treatment modality may be helpful to overcome the side effect of new onset diabetes with statin.
Keyword: Insulin resistance, Ezetimibe

MOP2-1-02 *Mini-Oral Presentation 2-1*

Association between long-term exposure to air pollutants, smoking status, and vitamin D deficiency with hypercholesterolemia in Korean adults: a cross-sectional study from the 2008-2014 Korea National Health and Nutrition Examination Survey

곽정현[1*], 김현자[2]

[1]인제대학교 식품영양·식품공학부, [2]강릉원주대학교 식품영양학과

Objectives: Air pollutants directly block exposure to ultraviolet B photons and indirectly reduce outdoor activities, resulting in vitamin D deficiency (VDD). In addition, smoking increases oxidative stress in the body and accelerates skin aging, thereby reducing the concentration of vitamin D in the body. Therefore, air pollution and smoking may trigger VDD. Previous study reported that VDD increases total cholesterol levels by reducing vitamin D receptor activity.
Methods: This cross-sectional study aimed to investigate the association between long-term exposure to air pollutants such as PM10, PM2.5, NO2, SO2, CO, and O3, smoking status, VDD, and their combination with hypercholesterolemia in the representative Korean men and women aged ≥19 years, using data from the 2008-2014 Korea National Health and Nutrition Examination Survey (KNHANES). We used linked data from the KNHANES to the daily moving average of air pollution data from 730 days before the examination date, using the participants' addresses in latitude and longitude coordinates. We included 28,134 adults with data on serum vitamin D, cholesterol concentrations, smoking status, and air pollutants levels. Main results were analyzed using a survey logistic regression model for complex sample analyses.
Results: After adjusting for potential covariates, adults with exposure to high concentrations of air pollutants and ever smokers showed a significantly higher risk of VDD (odds ratio [OR], 1.70; 95 % confidence interval [CI], 1.44-2.00). In the stratified group with high exposure to air pollutants, adults with low vitamin D status and ever smokers had a significantly higher risk of hypercholesterolemia than adults with high vitamin D status and never smokers (OR, 1.55; 95 % CI, 1.09-2.19). We found that high exposure to air pollutants, ever smokers, and VDD may increase hypercholesterolemia prevalence in Korean adults.
Conclusions: To reduce hypercholesterolemia risk, adults living in areas with high exposure to air pollution may need adequate vitamin D intake.
Keyword: Hypercholesterolemia, Total cholesterol, Air pollutants, Vitamin D deficiency, Smoking

MOP2-1-03 *Mini-Oral Presentation 2-1*

Association of lipoprotein(a) with NAFLD and MAFLD

강정규[*], 김병진

강북삼성병원 순환기/심장내과

Objectives: Recent studies have produced mixed results regarding the relationship between lipoprotein(a) (LP(a)) and non-alcoholic fatty liver disease (NAFLD). Furthermore, limited research is available on the connection between LP(a) and metabolic associated fatty liver disease (MAFLD). This study aims to clarify the relationship between LP(a) and both NAFLD and MAFLD through a comprehensive cross-sectional analysis.
Methods: Between March 2015 and December 2019, a total of 109,248 participants enrolled in the Kangbuk Samsung Health Study were included in this study. High LP(a) levels were defined as LP(a) >120 nmol/L using a latex particle-enhanced immunoturbidimetric assay, and MAFLD was defined based on abdominal ultrasound examinations and the international expert consensus statement's definition.
Results: In the overall population, the median LP(a) levels were 18.0 nmol/L (IQR: 8.6-41.5). The prevalence of MAFLD was 24.3% (n=26,576), with the isolated NAFLD rate of 1.9% (n=2,109). LP(a) levels were significantly higher in the MAFLD(+) group than in the MAFLD(-) group (15.8 nmol/L in the MAFLD(-) group vs 18.7 nmol/L in the MAFLD(+) group, p<0.001), with no significant difference in NAFLD status (18.7 nmol/L in the NAFLD(-) group vs 18.2 nmol/L in the NAFLD(+) group). Multivariable regression analyses revealed that LP(a) levels were significantly associated with decreased odds ratio(OR) for MAFLD (OR[95% CI, 0.94[0.92, 0.96], p<0.001). This association was consistent across the high LP(a) group as well (0.83[0.76, 0.91], p<0.001). However, LP(a) did not show a significant association with NAFLD (0.97[0.92, 1.01], p=0.132 for LP(a) levels; 0.93[0.77, 1.13], p=0.467 for the high LP(a) group).
Conclusions: This study demonstrates a clear negative association between LP(a) and MAFLD, but this correlation is not observed in groups with only NAFLD. Further longitudinal studies are necessary to clarify the distinct roles of LP(a) in MAFLD and NAFLD.
Keyword: Lipoprotein(a), NAFLD, MAFLD

MOP2-1-04 *Mini-Oral Presentation 2-1*

Association of smoking status and urinary cotinine levels with lipoprotein(a)

김병진*

성균관대학교 강북삼성병원 순환기/심장내과

Objectives: Lipoprotein(a) (LP(a)) concentrations are largely determined by genetic factors, with lifestyle changes having no effect on modifying levels. There is a paucity of studies on the relationship between LP(a) concentrations and smoking, particularly the biomarker cotinine. This study aims to evaluate the association between smoking status and cotinine levels, a biomarker of smoking, with LP(a) concentrations.
Methods: This study included 146,129 participants (age 37+/-6.7 years, men 59.2%) of the Kangbuk Samsung Health Study from 2011 to 2013. LP(a) levels were measured using the immunoturbidimetric assay, while urinary cotinine was quantified using the DRI cotinine assay. Cotinine-verified smoking was defined as a urinary cotinine level exceeding 50 ng/mL, and the high LP(a) group was defined as having LP(a) levels of 120 nmol/L or higher.
Results: The median LP(a) level was 55.9 nmol/L (IQR: 32.2-94.1 nmol/L). The prevalence of high LP(a) and cotinine-verified smokers was 16% and 24.4%, respectively. The prevalence of high LP(a) was lower in the cotinine-verified smokers (13.8%) compared to non-smokers (16.6%, p<0.001). Similarly, the prevalence of high LP(a) decreased progressively from never-smokers to current-smokers (16.9% for never-smokers, 15.9% for former-smokers, and 13.8% for current-smokers, p<0.001). Multivariable analysis showed a negative association between cotinine and LP(a) levels (beta=-0.346, p<0.001), with cotinine-verified smoking reducing the odds of high LP(a) (OR[95%CI], 0.94 [0.90, 0.98], p=0.005). The negative association between LP(a) and smoking became more pronounced with increasing self-reported daily smoking amount and pack-years. There was no gender interaction in the association between LP(a) levels and smoking.
Conclusions: This study demonstrates a negative association between LP(a) levels and cigarette smoke, especially cotinine. However, further longitudinal studies are needed for a more detailed analysis of their association and causality.
Keyword: Lipoprotein(a), Cotinine, Smoking

MOP2-1-05 *Mini-Oral Presentation 2-1*

Relationship between the length of diabetes mellitus and the onset of dementia in individuals after stroke: a comprehensive cohort study

이진화[1*], 한경도[3], 이민우[2], 천대영[1]

[1]한림대학교 동탄성심병원 순환기/심장내과, [2]한림대학교성심병원 신경과, [3]숭실대학교 정보통계보험수리학과

Objectives: To investigate the association between the duration of diabetes mellitus (DM) and the risk of developing dementia, including Alzheimer's disease (AD) and vascular dementia (VaD), among patients with a history of ischemic stroke.
Methods: A retrospective cohort study was conducted using data from the Korean National Health Insurance Database, encompassing 118,790 stroke survivors without prior dementia diagnoses from 2009 to 2018. Patients were categorized based on their glycemic status into normoglycemia, impaired fasting glucose, newly diagnosed DM, and established DM with durations of less than 5 years and 5 years or more. The primary endpoint was the incidence of dementia, with adjustments for demographic and clinical factors.
Results: The study cohort consisted of 118,790 eligible patients, with a mean age of 64.3 years and 48.00% being male. Over an average follow-up of 7.3 years, 16.7% of participants developed dementia. Patients were divided into five categories according to their glycemic status, with the following distribution: 50.82% non-diabetic, 25.15% with IFG, 1.27% newly diagnosed with DM, 10.63% with DM for less than 5 years, and 12.14% with DM for 5 years or more. Patients with both DM of less than 5 years (aHR 1.267, 95% CI 1.210-1.327) and DM of 5 years or more (aHR 1.466, 95% CI 1.408-1.527) significantly increased risk of all-cause dementia even after rigorous adjustments. Also the risk for developing AD and VaD was 43.4% and 51.4% higher, respectively, in those with DM duration exceeding 5 years.
Conclusions: The study demonstrates a significant association between prolonged DM duration and increased risk of all-cause dementia, AD, and VaD in stroke survivors. These findings underscore the importance of rigorous DM management and proactive dementia prevention strategies, particularly for patients with longstanding DM. The study advocates for integrated care approaches to mitigate dementia risk among stroke survivors with diabetes.
Keyword: Diabetes mellitus, Dementia, Stroke survivors

MOP2-1-06 *Mini-Oral Presentation 2-1*

Prediction of diabetic peripheral neuropathy via machine learning analysis of foot radiograph

Chae Won Chung[1*], Yong Eun Jang[2], Minjun Kwon[2], Gwang Lee[2], Jaetaek Kim[1]

[1]Division of Endocrinology and Metabolism, Department of Internal Medicine, College of Medicine, Chung-Ang University, Seoul, Republic of Korea, [2]Department of Physiology and Department of Molecular Science and Technology, Ajou University School of Medicine, Suwon, Republic of Korea

Objectives: Diabetic neuropathy is probably the most common complication of diabetes.Neuropathic ulcers or fractures may develop due to nerve damage in the foot.Thus all patients with diabetes should be screened for nerve fiber functions at the time of diagnosis of type 2 diabetes. However, optimal cost-effective strategy has not yet been established. Deep learning algorithms have rapidly become a novel methodology for analyzing medical images. In this study, we demonstrate the potential of utilizing deep learning models to predict diabetic neuropathy by combining foot radiography and patients' medical records.
Methods: We collected a dataset consisting of foot radiography images and medical records from 133 patients diagnosed with type 2 diabetes. A pre-trained ResNet-34 model, adapted to output both binary classification (presence of neuropathy) and regression (severity of nerve damage based on age and HbA1c levels), was trained using a custom loss function. The dataset was subjected to K-Fold cross-validation to ensure model robustness. Post-processing techniques were applied to refine model predictions.
Results: The model achieved an AUROC of 0.71 in detecting neuropathy, with post-processing improvements elevating this to 0.85. For the regression tasks, initial MAEs were 0.5 (age) and 0.6 (HbA1c), which improved to 0.4 and 0.4, respectively, after post-processing. These results underscore the model's high precision and the value of combining imaging with clinical data for neuropathy screening.
Conclusions: In this study, we developed a Deep learning algorithms that can accurately predicts patients with diabetic neuropathy from simple foot radiographs.
Keyword: Diabetic neuropathy, Machine learning, Artificial intelligence, Foot

MOP2-1-07 *Mini-Oral Presentation 2-1*

Exploration of factors related to suboptimal adherence for dyslipidemia using the KNHANES 2010 to 2021

신지혜[1*], 조상용[2], 강주성[1], 손민국[3]

[1]동아대학교 의과대학, [2]동아대학교 의과대학 순환기/심장내과, [3]동아대학교 의과대학 기초과학

Objectives: While dyslipidemia is no longer a rare chronic disease, lack of treatment and suboptimal adherence remain to be the problem. However, only limited studies exist regarding the suboptimal adherence of dyslipidemia. This study aims to identify potential clinical variables on suboptimal adherence for dyslipidemia.
Methods: The data were extracted from the Korean National Health And Nutrition Examination Survey(KNHANES) from 2010 to 2021. We performed two analyses: (1) dyslipidemia in general, including hyper-LDL-cholesterolemia, hypertriglyceridemia, and hypo-HDL-cholesterolemia, and (2) hyper-LDL-cholesterolemia in specific. Multi-variable adjusted logistic regression analysis was performed to investigate the factors associated with treatment and adherence.
Results: For dyslipidemia in general, the adherent, non-adherent, and non-treatment groups were 6,356 (20.2%), 336 (1.1%), and 24,708 (78.7%), respectively. In the adjusted model, age, sex, education level, income level, residential region, dyslipidemia diagnosis duration, dyslipidemia family history, hypertension, diabetes, thyroid diseases, ischemic heart diseases, depression, ex-smoking, alcohol drinking, regular exercise, fasting blood glucose, hemoglobin, and glomerular filtration rate (GFR) were associated with non-treatment. Meanwhile, age, sex, dyslipidemia diagnosis duration, hypertension, diabetes, stroke, ischemic heart diseases, and fasting blood glucose were associated with non-adherence. For hyper-LDL-dyslipidemia, the adherent, non-adherent, and non-treatment group were 6,356 (53.9%), 336 (2.8%), and 5,098 (43.2%), respectively. In the adjusted model, age, sex, residential region, dyslipidemia diagnosis duration, hypertension, diabetes, thyroid diseases, stroke, ischemic heart diseases, ex-smoking, regular exercise, fasting blood glucose, hemoglobin, and GFR were associated with non-treatment. The adherent and non-adherent groups were not different from general dyslipidemia.
Conclusions: This study shows which factors could be associated with the suboptimal adherence for dyslipidemia in general and hyper-LDL-cholesterolemia in specific. This study recommends further consideration to increase the treatment and adherence rate to reach the target lipid levels.
Keyword: Dyslipidemia, Hyper-LDL-cholesterolemia, KNHANES, Treatment, Adherence

MOP2-1-08 *Mini-Oral Presentation 2-1*

The effect of fenofibrate and omega-3 fatty acid based on baseline remnant cholesterol levels: an analysis of the National Health Insurance Service-National Sample Cohort, 2002-2015

장영우*

가천대학교 길병원

Objectives: Lipid-lowering therapy such as statins consistently demonstrates stronger cardiovascular (CV) benefit in patients with higher risk. However, there is limited data on whether fenofibrate or omega-3 fatty acid (O3FA) has stronger benefit on higher remnant cholesterol (RC) levels. Herein, we analyzed the Korean National Health Insurance Service - National Sample Cohort to investigate the effect of fenofibrate and O3FA based on baseline RC levels between 2010 and 2015.
Methods: The cohort was mainly divided into subjects administered with (statin (+)) and without statin (statin (-)). Each group was subsequently classified according to baseline RC levels (R≥24 and R<24 group). Propensity score matching (PSM) was performed between subjects who did or did not take fenofibrate or O3FA. The primary endpoint was major adverse cardiac and cerebrovascular event (MACE), a composite of CV death, stroke, and incident coronary artery disease.
Results: Fenofibrate or O3FA did not show CV benefit in the statin(-) group regardless of RC levels. However, fenofibrate was associated with reduction of MACE (HRadj: 0.58; 95% CI: 0.440-0.778) in the RC≥24 group. O3FA was not lined with CV benefit regardless of statin intake or baseline RC category. Fenofibrate only showed MACE reduction in the 4th quartile of RC group, although there was a dose-dependent decreased in HRadj with increasing levels of RC. We then evaluated the benefits of both drugs based on low-density lipoprotein cholesterol (LDL-C) and RC levels. Unlike O3FA, fenofibrate was consistently associated with reduced MACE in patients with both elevated LDL-C and RC levels, regardless of the cutoff values (Table).
Conclusions: Fenofibrate was linked to CV risk reduction in statin-taking patients with high RC and LDL-C levels.
Keyword: Remnant cholesterol, Fenofibrate, Triglyceride

MOP2-1-09 *Mini-Oral Presentation 2-1*

Trends in hepatic steatosis over 15 years: a comprehensive age-period-cohort study

Garam Jo[1*], Dahyun Park[2], Hee Ju Jun[3], Hae Jin Lee[3]

[1]Institute for Bio Materials, Korea University, Seoul, Korea,
[2]Research and Management Center for Health Risk of Particulate Matter, Seoul, South Korea,
[3]Interdisciplinary Program in Precision Public Health, Graduate School, Korea University, Seoul, Korea,

Objectives: Metabolic Dysfunction-associated Steatotic Liver Disease (MASLD) is emerging as a worldwide pandemic. A comprehensive overview of its long-term trends will help to improve public awareness. We aimed to track changes in hepatic steatosis over a 15-year period through an Age-Period-Cohort (APC) approach and to define the birth-cohort effect on hepatic steatosis levels in Korea.
Methods: We conducted the APC analysis to examine trends in MASLD steatosis levels using data from the Korea National Health and Examination Survey 2007-2021. The hepatic steatosis index (HSI) and Framingham steatosis index (FSI) were proposed as non-invasive tools for assessing advanced liver steatosis. They were calculated from age, body mass index, triglyceride, aspartate aminotransferase, alanine aminotransferase, diabetes history, and hypertension status. In addition, APC analyses were performed after stratification by concurrent conditions such as obesity, hypertension, diabetes, hypercholesterolemia, and hypertriglyceridemia.
Results: Our results indicated that both HSI and FSI remained stable overall during study period, but an increasing trend was observed in men. When diagnosing MASLD with HSI ≥36 or FSI ≥23, both men and women exhibited a recent upward trend in MASLD prevalence. Cohort effect showed a U-shaped pattern with the lowest risk in individuals born in the 1960s, while recent cohorts showed an increasing risk. The estimated mean of two hepatic steatosis indices was greater in individuals with obesity or hypertriglyceridemia than other people. Notably, the most substantial increases were observed in individuals having more comorbidities, emphasizing a distinct cohort effect.
Conclusions: In conclusion, our study highlights an increasing trend in hepatic steatosis, particularly in younger birth cohorts. The interplay of comorbidity amplifies the risk, showing the highest risk in individuals having obesity and hypertriglyceridemia. Understanding these trends is essential to develop targeted healthcare strategies with a goal to reduce a disease burden for future generations.
Keyword: Steatosis, MASLD, Age-period-cohort, HSI, FSI

MOP2-1-10 *Mini-Oral Presentation 2-1*

Association between physical activity and mortality among dyslipidemia patients in Korea (KNHANES 2007-2013)

노진원*, 최영환, 김연수

서울대학교 스포츠의학

Objectives: To determine the association between physical activity and the risk of all-cause and cardiovascular disease (CVD) among dyslipidemia patients in Korea.
Methods: This study included 17,395 adults with dyslipidemia (mean age: 51.6, women: 64.1%) from the Korea National Health and Nutrition Examination Survey, mean average follow-up period being 9.1 years. Dyslipidemia was defined as having at least one of the following conditions: total cholesterol ≥240 mg/dL, triglycerides ≥200 mg/dL, low-density lipoprotein cholesterol ≥160 mg/dL, high-density lipoprotein cholesterol <40 mg/dL for men and <50 mg/dL for women, or being on lipid-lowering medications. Physical activity level was assessed using self-reported questionnaire and categorized into four groups: inactive (0 metabolic equivalents(METs)-min/wk), insufficiently active(1-499METs-min/wk), sufficiently active(500-999METs-min/wk), and highly active (≥1,000METs-min/wk). Cox proportional hazard models calculated hazard ratios for all-cause and CVD mortality associated with dyslipidemia, followed by a gender-stratified analysis.
Results: There was a significant reduction in all-cause and CVD mortality with increased physical activity levels. 1,109 all-cause deaths and 260 CVD deaths occurred in this study. For all-cause mortality, the hazard ratio (HR) of insufficiently active (HR: 0.82, 95% CI(Confidence Interval): 0.68-0.99), sufficiently active (HR: 0.75, 95% CI: 0.62-0.92), and highly active (HR: 0.75, 95% CI: 0.64-0.88) were reduced. For CVD mortality, the HR of sufficiently active (HR: 0.64, 95% CI=0.43-0.95) and highly active (HR: 0.60, 95% CI: 0.43-0.81) exhibited significant reductions. After conducting gender-stratified analysis, results were only significant in women. For all-cause mortality, the sufficiently active (HR: 0.70, 95% CI: 0.53-0.91) and the highly active (HR: 0.68, 95% CI: 0.54-0.84), and for CVD mortality, only highly active (HR: 0.54, 95% CI:0.36-0.83) showed reductions.
Conclusions: Engaging in the recommended amount of physical activity reduces all-cause and CVD mortality risk in dyslipidemia patients in Korea, especially in women who participate in recommended physical activity guideline for all-cause mortality and highly active for CVD mortality.
Keyword: Physical activity, Dyslipidemia, Mortality

MOP2-1-11 *Mini-Oral Presentation 2-1*

Current status of lipid management and subsequent cardiovascular events after acute coronary syndrome in Korea: real world findings from the observation and survey studies

Jong-Young Lee[1*], Chang-Hwan Yoon[2], Jin-Yong Hwang[3], Jung-Sun Kim[4], Kwang Soo Cha[5], Doo-Il Kim[6], Jin-Bae Lee[7], Seung-Ho Hur[8], Jung-Hee Lee[9], Kiyuk Chang[10], Seok Kyu Oh[11], Jung Ho Heo[12], Seong-Ill Woo[13], Kyung Kuk Hwang[14], Sang-Ho Jo[15], Seung-Jae Joo[16], Soo-Joong Kim[17], Tae Hoon Ahn[18], Won Young Jang[19], So-Yeon Choi[20], Byung-Ryul Cho[21], Suk-Hwan Kim[22], Sang-Hyun Kim[23], Min-Jung Kang[24], Dae-Woo Lee[24], In-Ho Chae[2], Myung Ho Jeong[25]

[1]Department of Cardiology, Kangbuk Samsung Hospital, University of Ulsan College of Medicine, Seoul, [2]Department of Cardiology, Seoul National University Bundang Hospital, Seongnam, [3]Department of Internal Medicine. College of Medicine, Gyeongsang National University and Hospital, Jinju, [4]Department of Cardiology, Yonsei University Severance Hospital, Seoul, [5]Department of Cardiology, Pusan National University Hospital, Busan, [6]Department of Internal Medicine, Haeundae Paik Hospital, Busan, [7]Department of Cardiology, Daegu Catholic University Medical Center, Daegu, [8]Department of Cardiology, Keimyung University Dongsan Hospital, Daegu, [9]Department of Cardiology, Wonju Serverance Christian Hospital, Wonju, [10]Deparment of Cardiology, Seoul St. Mary's Hospital, The Catholic University of Korea, Seoul, [11]Department of Cardiovascular Medicine, Regional Cardiocerebrovascular Center, Wonkwang University Hospital, Iksan, [12]Department of Internal Medicine, Kosin University Gospel Hospital, Kosin University College of Medicine, Busan, [13]Department of Cardiology, Inha University Hospital, Incheon, [14]Department of Cardiology, Chungbuk National University Hospital, Cheongju, [15]Department of Internal Medicine, Hallym University Sacred Heart Hospital, Anyang, [16]Department of Internal Medicine, Jeju National University School of Medicine, Jeju, [17]Department of Cardiology, College of Medicine, Kyung Hee University, Seoul, [18]Department of Internal Medicine, Naeun hospital, Incheon, [19]Department of Cardiology, St. Vincent's Hospital, College of Medicine, The Catholic University of Korea, Seoul, [20]Department of Cardiology, Ajou University School of Medicine, Suwon, [21]Department of Internal Medicine, Kangwon National University Hospital, Kangwon National University School of Medicine, Gangwon-do, [22]Department of Cardiology, Gimpo woori hospital, Gyeonggi-do, [23]Department of Internal Medicine, Boramae Medical Center, Seoul National University College of Medicine, Seoul, [24]Medical Department, Sanofi Korea, Seoul, [25]Department of Cardiology, Chonnam National University Hospital, Gwangju, Republic of Korea

Objectives: The Observation study of Lipid management and risks in patients with Acute coronary syndrome (ACS) in Korea (OLA-K) and ACS KoreaPath Survey (K-PATH) were designed to get real world data about the lipid management strategy after ACS and the association of lipid-lowering therapy with major cardiovascular events (MACE).
Methods: OLA-K study was a 24-months prospective, multi-center, observational study wherein association between lipid lowering therapies and MACE rate along with LDL-C goal achievement was assessed. In K-PATH survey, a questionnaire was designed to collect information regarding post-ACS lipid management strategy and 35 investigators from 24 study sites completed the questionnaire.
Results: A total of 1053 ACS patients were enrolled and followed up for 24 months between 2018 and 2022. The mean age was 63.8 years and 25.2% were female. At baseline, 6.6% had previous ACS, 39.8% had diabetes and 74.8% had hypertension. The mean LDL-C at baseline was 108.7mg/dL. Overall, at month 24, 103(9.8%) patients (incidence rate 5.2/100 patient-years) had experienced a MACE with a median time to event of 7.6 (6.3-10.8) months. MACE incidence rate was increasing from month 1(0.8%) to month 24(9.8%). No difference observed in MACE incidence rate between patients on high intensity and medium intensity statin. At months 24, only 29.8% of patients reached the LDL-C goal. In the survey, investigators reported that a median of 80% of their ACS patients would expect to achieve LDL-C goal. However only 25.5% and 29.1% at 6 and 12 months respectively, reached the LDL-C goal in the study.
Conclusions: This study showed that at the 24-month post-event point, two-thirds of ACS patients did not achieve the LDL-C goal, suggesting an unmet need for improving lipid management strategies. In addition, the gap between OLA-K and K-PATH in terms of LDL-C goal achievement has been identified.
Keyword: Observational study, Lipid management, Acute coronary syndrome, Physician survey

MOP2-1-12 *Mini-Oral Presentation 2-1*

Real-world application of evolocumab among hyperlipidemia patients in Korea: a multicenter prospective study

임용환[1*], 김민철[1], 이승헌[1], 안준호[1], 박경일[2], 김충기[3], 안종화[4], 정진선[6], 이호준[6], 강웅철[5]

[1]전남대학교병원 순환기/심장내과, [2]동아대병원 순환기/심장내과, [3]이대서울병원 순환기/심장내과, [4]창원경상대병원 순환기/심장내과, [5]가천대길병원 순환기/심장내과, [6]암젠코리아 의학부

Objectives: Elevated low-density lipoprotein cholesterol (LDL-C) is a major residual risk factor among patients with acute coronary syndrome (ACS). In the absence of sufficient real-world evidence, this observational study investigated the effectiveness and safety of evolocumab in patients with hypercholesterolemia treated with evolocumab for recent ACS in the clinical practice setting in Korea.
Methods: Between January 2022 and February 2023, patients from 10 hospitals in Korea who initiated evolocumab within 24 weeks of an ACS event were enrolled. Data collected at visit 1 (evolocumab initiation) included patients' characteristics, comorbidities, and lipid-lowering therapies. LDL-C reduction from visit 1 (week 0) to visit 2 (week 8) was assessed. The primary outcome was the proportion of patients who achieved LDL-C <55 mg/dL at follow up; the secondary outcome was the proportion who achieved LDL-C <70 mg/dL at follow up.
Results: Overall, among 142 patients enrolled in the study, 89 were included in the efficacy analysis. Mean age was 59.6 years and most patients were male (87.6%). ST-segment elevation myocardial infarction (STEMI) was the most common ACS (39.3%), followed by NSTEMI (32.6%), and 61 patients received ezetimibe combination therapy (68.5%). Median [Q1, Q3] LDL-C at week 0 was 98 [77, 115] mg/dL and after evolocumab treatment at week 8 it was 49 [29, 67] mg/dL, resulting in a mean 50.9% reduction and a mean 55.1 mg/dL absolute reduction from week 0. LDL-C goals of <55mg/dL and <70mgl/dL at follow up were achieved by 55.1% and 78.7% of patients, respectively. No adverse or serious adverse drug reactions were reported.
Conclusions: Evolocumab treatment was associated with significant LDL-C lowering and favorable safety and guideline-recommended LDL-C goal achievement rates among very high-risk ACS patients in the real-world clinical practice setting in South Korea.
Keyword: Atherosclerotic cardiovascular disease, Acute coronary syndrome, Low-density lipoprotein cholesterol, Evolocumab, Real-world evidence

MOP2-1-13 *Mini-Oral Presentation 2-1*

Comparison of the stroke patients with atherosclerotic and non-atherosclerotic occlusions successfully treated with thrombectomy

이승재[*], 이재상

순천향 부천병원 신경과

Objectives: Emergent atherosclerotic occlusions of brain vessels are more difficult to treat with thrombectomy than non-atherosclerotic occlusion (e.g. cardioembolism). This study attempts to compare acute stroke patients with atherosclerotic and non-atherosclerotic occlusions successfully recanalized by thrombectomy.
Methods: We analyzed a total of 176 anterior circulation stroke patients successfully recanalized by thrombectomy. Using the baseline CT angiographies, collateral status was dichotomized as poor (≤50% filling) or good (>50% filling). Using the CT performed within 24 hours after thrombectomy, the presence or absence of contrast accumulation (CA) was confirmed. Early neurologic deterioration (END) was defined as ≥4-point increase in the score of the National Institutes of Health Stroke Scale (NIHSS) in the first 72 hours after thrombectomy.
Results: Among the 176 recanalized patients, 46 (26.1%) had atherosclerotic occlusions. Compared to the patients with non-atherosclerotic occlusions, patients with atherosclerotic occlusions had higher prevalence of male (76.1% vs 55.4%, p=0.013), hypertension (82.6% vs. 63.8%, p=0.018), diabetes (41.3% vs. 18.5%, p=0.002), and current smoking (30.4% vs. 8.5%, p<0.001). In addition, the patients had lower score of initial NIHSS (stroke severity; median [interquartile range] 10.5 [7-16] vs. 16 [11-19], p<0.001) with a statistical trend toward higher frequency of good collateral status (77.3% vs. 62.5%, p=0.076), and also had a longer procedural time (minutes, median [interquartile range]: 60.0 [38.5-84.0] vs. 38.0 [24.0-55.0], p=0.001) and higher number of passes (≥4; 17.4% vs 6.2%, p=0.027). However, there was no statistical difference between the groups in the clinical outcomes including CA, END, symptomatic intracranial hemorrhage, malignant stroke, poor functional outcome (3 month modified Rankin scale >2), and mortality.
Conclusions: Our data suggests that atherosclerotic occlusions may need a longer procedural time for successful recanalization. However, the recanalized patients may have clinical outcomes comparable to the patients with non-atherosclerotic, embolic occlusion.
Keyword: Stroke, Atherosclerosis, Thrombectomy, Recanalization

MOP2-1-14 *Mini-Oral Presentation 2-1*

Association between estimated glucose disposal rate and subclinical coronary atherosclerosis

김명진[1*], 조윤경[1], 김은희[1,2], 이민정[1,2], 이우제[1], 김홍규[1,2], 정창희[1]

[1]서울아산병원 내분비내과, [2]서울아산병원 건강의학과

Objectives: The estimated glucose disposal rate (eGDR) is an easily accessible clinical parameter for assessing insulin resistance in patients with diabetes mellitus. In this study, we aimed to investigate the link between eGDR and subclinical coronary atherosclerosis in an asymptomatic middle-aged Korean population.
Methods: This study involved 4,004 subjects who underwent routine health checkups with coronary multidetector computed tomography at Asan Medical Center from 2007 to 2011, among whom 913 were included in a follow-up analysis through 2014. The eGDR was derived using the following formula: 21.16 - (0.09 * waist circumference [cm]) - (3.41 * hypertension) - (0.55 * glycated hemoglobin [%]). Patients were categorized into three groups according to the tertiles of eGDR. Subclinical coronary atherosclerosis was defined by significant coronary stenosis (equal or greater than 50%), presence of plaques, coronary artery calcification (CAC) score calculated using the Agatston scoring method, and its progression.
Results: A higher eGDR level was associated with lower prevalence of significant coronary stenosis, plaques, moderate to severe CAC, and CAC progression. Compared to those in the highest eGDR tertile, those in the lowest eGDR tertile had an adjusted odd ratio (95% confidence interval) of 2.54 (1.79-3.61) for moderate to severe CAC and 2.09 (1.30-3.36) for CAC progression. Baseline eGDR demonstrated a consistent negative association with annualized changes in CAC scores (β=-0.051, p<0.001).
Conclusions: Decreased eGDR values were significantly associated with higher subclinical coronary atherosclerosis burdens in an asymptomatic middle-aged Korean population. Our study findings suggest that eGDR could be a preferential predictor for future cardiovascular events.
Keyword: Insulin resistance, Estimated glucose disposal rate, Coronary artery calcification

MOP2-2-01 *Mini-Oral Presentation 2-2*

Preventive effect of isocaloric restriction on high-fat diet-induced metabolic disturbances

은성진[1*], 정은지[1], 채서연[1], 이선혜[2]

[1]Department of Applied Biological Sciences, Sun Moon University, [2]Division of Food Science, Sun Moon University

Objectives: Overconsumption of a high-fat (HF) diet can cause gut microbiota dysbiosis, which increasing gut permeability, causing gut inflammation, and subsequently facilitating the translocation of components of Gram-negative bacteria, lipopolysaccharide (LPS), into circulation. The increased plasma LPS level alters vagal gut-brain communication, impairing the sensitivity to anorexigenic hormones such as cholecystokinin (CCK) and leptin, promoting hyperphagia, consequently leading to metabolic disturbances. Thus, we aimed to investigate the effect of controlling energy overconsumption via isocaloric restriction on HF diet-induced metabolic complication.
Methods: Male C57BL/6 mice (n=8/group; 6 weeks old) were divided into three groups and fed on their respective diet for 12 weeks; a low-fat (LF; 10% fat, n=8), HF (45% fat, n=16), and HF with isocaloric restriction (pair-fed to the LF group as a model for the prevention effect; IR, n=8). After 12 weeks on their respective diet, general phenotypes, gut-brain signaling, and inflammatory and lipid profiles were examined.
Results: Isocaloric restriction suppressed body weight gain and decreased fasting blood glucose levels. The HF-induced impairment in the sensitivity to CCK, as a proxy for gut-brain signaling, was prevented by isocaloric restriction. The IR group showed upregulated expression of leptin in the ileum, duodenum, and visceral fat, indicating an appetite-suppressing effect. Furthermore, the gene expression of cluster of differentiation 14, acting as a pivotal receptor for LPS, was significantly downregulated along with monocyte chemoattractant protein-1, a marker of macrophage infiltration, consequently leading to suppression of subsequent inflammatory response.
Conclusions: Taken together, these data show that isocaloric restriction can improve general phenotypes, glycemic control, gut-brain signaling, and inflammatory profiles, supporting potential application of isocaloric restriction for the regulation of metabolic disturbances induced by HF feeding.
Keyword: Isocaloric restriction, High-fat diet, Lipopolysaccharide, Gut-brain signaling, Metabolic disturbances

MOP2-2-02 *Mini-Oral Presentation 2-2*

Multimodal analysis of human thrombus

Hyeonji Mun[1*], Joo Young Kweon[1], Dougho Park[2], Yong Joo Ahn[3]
[1]포항공과대학교 융합대학원 의과학전공, [2]에스포항병원 재활의학과, [3]포항공과대학교 IT융합공학과

Objectives: Endovascular thrombectomy (EVT) is a standard treatment for a large vessel occlusion in ischemic stroke. The investigation of the relationship between thrombus composition and stroke etiology by histopathologic analysis could give appropriate insight into the treatment options and patient outcomes. Thrombus forms a complex composition consisting of various cells and molecules as well as the origin of thrombi. EVT trial times and type of thrombectomy (aspiration or retrieval) depend on the composition of the thrombus. Therefore, in this study, we aimed to the analysis of human thrombus and its correlation with clinical features.
Methods: To visualize the 3D structure and composition of the whole thrombus, the samples from acute stroke patients were cleared and followed by the staining with DAPI, CD45, P-selectin, Fibrin, and Citrullinated (CitH3), and imaged using light-sheet microscopy. H&E staining and immunostaining on frozen sectioned samples were used for confocal imaging, and holotomography imaging. To investigate the genomic architecture, spatial genomics was performed. Along with multimodal thrombus analysis results, the patient's CBCs, chemistry and the underlying medical conditions were analyzed.
Results: The whole 3D structure and composition of the thrombus and the fibrin structure according to blood flow were observed from light-sheet microscope analysis. The general morphologic feature of the thrombus was confirmed through H&E. In the holotomography results, the presence of heterogeneous cell populations were confirmed using DAPI staining combined with refractive index (RI) measurements. Additionally, CitH3+ neutrophils exhibited high RI values in their granules.
Conclusions: The identification of the cells compose the thrombi using multimodal approaches not only discover therapeutic strategies but also stroke prevention.
Keyword: Thrombus, Thrombectomy, Stroke, Multimodal analysis

MOP2-2-03 *Mini-Oral Presentation 2-2*

Ubxn4 deficiency aggravates hepatic steatosis in high-fat diet-fed mice

양선부*, 김재택
중앙대학교 의과대학 내분비내과

Objectives: UBX domain-containing protein 4 (ubxn4) is a discrete protein domain that binds p97/valosin-containing protein (VCP), a molecular chaperone involved in endoplasmic-reticulum-associated protein degradation (ERAD). Ubxn4 is highly expressed in the liver and loss of ubxn4 resulted in activation of ER stress in worms. We therefore hypothesized that deficiency of ubxn4 in mice would be susceptible to the development of non-alcoholic fatty liver disease (NAFLD).
Methods: We generated whole-body ubxn4 knockout (UBXN-/-) mice. Male WT and UBXN-/- mice at the age of 6 - 8 weeks were fed with standard chow or high-fat diet (HFD) for 8 weeks.
Results: Ubxn4 gene expression was upregulated in HFD-fed mouse livers. UBXN-/- mice exhibited impaired glucose tolerance compared to WT mice, and it was observed that ALT levels significantly increased concurrently with a notable increase in lipid droplet accumulation, indicating exacerbated hepatic steatosis. In addition, ubxn4 deletion enhanced HFD-induced hepatic expression of AKT and mTOR proteins. On the other hand, the gene expressions contributing to lipid and energy metabolism was found to be lower in HFD-UBXN-/- compared to HFD-WT livers.
Conclusions: This study revealed that ubxn4 deficiency led to enhancement of lipid accumulation in diet-induced NAFLD mouse model and might be considered as a target for the prevention and treatment of NAFLD.
Keyword: UBXN4, Steatosis, NAFLD, Glucose tolerance, Lipid metabolism, Metabolism

MOP2-2-04 *Mini-Oral Presentation 2-2*

Induction of COX-2 expression by the effect of crotonaldehyde in human endothelial cells (HUVECs)

김도윤*, 정택승, 박용식

경희대학교 기초과학

Objectives: Cyclooxygenase-2 (COX-2) is an inducible isoform protein and regulates various biological mechanisms in vascular pathophysiology. COX-2 is induced in response to diverse stimuli, which results in the production of prostaglandins (PGs), associated with inflammation. Crotonaldehyde (CRA), extremely toxic α, β-unsaturated aldehyde, is a main compound found in cigarette smoke. α, β-Unsaturated aldehyde in cigarette smoke mediates inflammation and vascular dysfunction. We demonstrated that whether CRA stimulates the induction of COX-2 and PGE2 synthesis in the human endothelial cell.
Methods: The effects of CRA stimulation on COX-2 expression were performed in human umbilical vein endothelial cells (HUVECs). We evalulated the expression of protein, mRNA level, amount of prostaglandin E2 (PGE2) using Western blot analysis, Reverse transcriptase polymerase chain reaction (RT-PCR), PGE2 EIA kit.
Results: COX-2 induction by stimulation of Crotonaldehyde (CRA) was accompanied by enhanced p38 phosphorylation and PGE2 generation. However, the production induced by CRA was reduced by pretreatment with an inhibitor of p38 MAPK. Thus, we determined that CRA, a highly reactive α, β-unsaturated aldehyde found in cigarette smoke, stimulates the induction of COX-2 expression and PGE2 synthesis via the p38 MAPK-CREB pathway.
Conclusions: These findings suggest that CRA might play a significant role in the development of vascular diseases through an inflammatory response associated with COX-2 expression.
Keyword: Cyclooxygenase-2, Crotonaldehyde, Cigarette smoke, Endothelial cells, Oxidative stress

MOP2-2-05 *Mini-Oral Presentation 2-2*

Systems genetics analysis to identify candidate genes for fat distribution in BXD mice

김나영*, 강윤원, 김재영, 오창명

Gwangju Institute of Science and Technology, Department of Biomedical Science and Engineering

Objectives: Obesity is defined as the accumulation of excess fat in the body. Although obesity is the main cause of metabolic disorders, not all obese people have metabolic disorders. Body fat distribution has been proposed as a possible explanation for this discrepancy.
Methods: To investigate the regulation of body fat distribution, we performed a genetic analysis using the phenotype and subcutaneous fat microarray data from the BXD mouse genetic reference population. Our analysis identified H2-Ke6 (Hsd17b8), encoding hydroxysteroid 17-beta dehydrogenase, as a candidate target for adipose tissue plasticity.
Results: To further explore its role, we employed multiple approaches to reveal the effect of Hsd17b8 inhibition. First, lipid accumulation was attenuated in 3T3-L1 where Hsd17b8 was inhibited by CRISPER/Cas9. Furthermore, we confirmed that the expression of adipogenesis-related transcription factor as well as mature adipocyte marker was decreased in 3T3-L1. We additionally verified a decrease in estrogen receptor expression, which impacts the transcription factor. Consistently, in C. elegans, we used RNA interference to inhibit the expression of the Hsd17b8 ortholog, dhs-25, and observed a reduction in lipid accumulation. All these data suggest that the reduction in lipid accumulation is attributed to the suppression of the adipogenesis process. On top of that, to determine whether Hsd17b8 influences body fat distribution in vitro, we utilized IngWAT cell line. In contrast to the previous findings, we observed an increase in lipid accumulation of IngWAT when Hsd17b8 was inhibited. Surprisingly, the elevation of lipid accumulation in sWAT, as seen in gynoid obesity, has been revealed to be indicative of robust metabolic health compared to android.
Conclusions: Collectively, these data suggest that Hsd17b8 may represent a novel therapeutic target for the treatment of obesity. Our study provides valuable insights into the mechanisms underlying metabolic health in obesity and highlights the potential of Hsd17b8 as a therapeutic target for modulating adipose tissue plasticity.
Keyword: Obesity, Adipose tissue plasticity, Body fat distribution, Metabolic syndrome, Systems genetics

MOP2-2-06 *Mini-Oral Presentation 2-2*

Small leucine zipper protein regulates mesenchymal stem cell differentiation via transcriptional modulation of PPARγ2

하지명*, 김정한

Department of Biochemistry, The Catholic University of Korea College of Medicine, Seoul, South Korea

Objectives: Differentiation of mesenchymal stem cells (MSCs) into either adipocytes or osteoblasts is transcriptionally regulated by the two key transcription factors PPARγ2 and Runx2. PPARγ2 is highly expressed during adipocyte differentiation and regulates expression of genes involved in adipogenesis. Although transcriptional modulation of PPARγ2 has been investigated in adipogenesis, the underlying molecular mechanisms to control the balance between adipogenesis and osteogenesis in MSCs remain unclear. In this study, the role of sLZIP in regulation of PPARγ2 transcriptional activation and sLZIP involvement in differentiation of multipotent MSCs into adipocytes and osteoblasts were investigated.
Methods: We have examined the interaction between sLZIP and PPARγ2, and analyzed the role of sLZIP in the promoter region of FABP4 to determined the transcriptional activity of PPARγ2. The extent of adipogenesis and osteogenesis was quantified in sLZIP transgenic mice.
Results: sLZIP induces recruitment of co-repressors of PPARγ2, and enhances formation of complexes between PPARγ2 and HDAC3, resulting in down-regulation of PPARγ2 transcriptional activity. sLZIP prevents adipocyte differentiation and expression of PPARγ2 target genes in vitro and in vivo. sLZIP also up-regulates Runx2 transcriptional activity and osteoblast differentiation, leading to enhanced bone development. However, sLZIP does not affect chondrogenesis and osteoclastogenesis.
Conclusions: These results indicate that sLZIP functions as a key regulator to control the balance between adipogenesis and osteogenesis in MSC differentiation.
Keyword: Mesenchymal stem cell, PPARγ2, Runx2, Adipogenesis

MOP2-2-07 *Mini-Oral Presentation 2-2*

Adipocyte specific deficiency of A20 enhances energy homeostasis and lipid metabolism in diet-induced obesity

이예린*, 이상헌, 김소연, 김효주, 김민주, 최이주, 박성실, 정수명

성균관대학교 생명과학과

Objectives: Many studies on ubiquitin editing enzyme A20 have been extensively centered on its roles in inflammation and immune diseases; less understood are in vivo functions of A20 in other physiological/pathophysiological contexts, particularly metabolism. Adipose tissue is defined by the presence of specialized lipid processes that function in storing energy for maintaining energy homeostasis. Moreover, unhealthy adipose tissue is highly related to metabolic maladaptation such as obesity, cardiovascular and metabolic diseases. Which is why regulating adipose tissue lipid metabolism takes center stage in maintaining whole-body conditioning.
Methods: For in vivo experiments, we have generated adipose tissue specific A20 knock-out mice(Adipoq-Cre; A20fl/fl (A20FATKO)) mice by interbreeding Adipoq-Cre and A20 floxed mice. With A20FATKO mice, we fed a high-fat diet (HFD) for up to 14 weeks and sacrificed them for further investigation. For in vitro experiments, we have generated immortalized preadipocytes wherein loss of A20 by CRISPR/Cas9 vector systems.
Results: Conditionally deleting A20 in pan-adipocytes protects mice from the high-fat diet-induced metabolic disease at least in part through preventing adipose tissue hypertrophy and hepatic steatosis. Gene expression profiling of Subcutaneous White Adipose Tissue (SAT) reveals inhibiting adipocyte A20 reduces adipose tissue inflammation and reprograms SAT lipid metabolism to favor catabolism. Such reprogramming may associate with our in vitro results using immortalized preadipocytes wherein loss of A20 by CRISPR/Cas9 promotes b-adrenergic signaling-induced lipid oxidation and thermogenesis. Our findings provide unexpected roles for A20 in adipose tissue's lipid handling and systemic energy homeostasis.
Conclusions: Conditional knockout of A20 in pan-adipocyte protects mice from high-fat diet-induced weight gain with alleviated metabolic disorders. A20 deletion in subcutaneous white adipose tissue (SAT) reprograms lipid metabolism catabolically like beige fat, such that reprogramming may be due to increased expression of beta-adrenoreceptor (β-AR) signaling target genes which encode oxidative metabolism.
Keyword: Adipose tissue, Lipid metabolism, Obesity

MOP2-2-08 *Mini-Oral Presentation 2-2*

Comparison of adipose tissue and hippocampus transcriptome profile of IGF-1 injected Alzheimer's disease mouse

안서연[1,2*], 최서윤[1,2], 송주현[1,2]

[1]전남대학교 의과대학 해부학교실, [2]전남대학교 Biomedical Science Graduate Program (BMSGP)

Objectives: Alzheimer's disease (AD) as the most prevalent neurodegenerative disorder is characterized by a multitude of pathological manifestations, prominently marked by the aggregation of amyloid beta. Current studies reported a compelling association between excessive adiposity and glial activation, further correlating with cognitive impairments. In addition, changes in levels of insulin-like growth factor 1 (IGF-1) have been reported in individuals with metabolic conditions accompanied by memory dysfunction. Hence, our research endeavors to comprehensively explore the impact of IGF-1 on the hippocampus and adipose tissue in the context of Alzheimer's disease.
Methods: To address this, we have conducted an in-depth analysis utilizing APP/PS2 transgenic mice, recognized as a well-established mouse model for Alzheimer's disease. Upon administering IGF-1 injections to the APP/PS2 mice, we observed notable alterations in their behavioral patterns, prompting us to undertake a comprehensive transcriptomic analysis of both the hippocampal and adipose tissues.
Results: Our data unveiled significant modifications in the functional profiles of these tissues. Specifically, in the hippocampus, we identified changes associated with synaptic activity and neuroinflammation. Concurrently, the adipose tissue displayed shifts in processes related to fat browning and cell death signaling. In addition to these findings, our analysis enabled the identification of a collection of long non-coding RNAs and circular RNAs that exhibited significant changes in expression subsequent to the administration of IGF-1 injections. Furthermore, we endeavored to predict the potential roles of these identified RNA molecules within the context of our study.
Conclusions: To sum up, we suggest valuable transcriptome data for hippocampal and adipose tissues within an Alzheimer's disease model and posits a significant role for IGF-1 within both the hippocampus and adipose tissue.
Keyword: Alzheimer's disease, IGF-1, Adipose tissue

MOP2-2-09 *Mini-Oral Presentation 2-2*

CircTmcc1 modulates the astrocytic inflammation in the hyperammonemia induced brain

최서윤[1,2*], 안서연[1,2], 송주현[1,2]

[1]전남대학교 의과대학 해부학교실, [2]전남대학교 Biomedical Science Graduate Program (BMSGP)

Objectives: Hepatic encephalopathy-induced hyperammonemia changes astrocytic glutamate metabolism in the brain, which is involved in cognitive decline.
Methods: To assess specific therapeutic strategies for the treatment of hepatic encephalopathy, various molecular signaling studies, such as non-coding RNA functional study, have been conducted. However, despite several reports of circular RNAs (circRNAs) in the brain, few studies of circRNAs in hepatic encephalopathy-induced neuropathophysiological diseases have been conducted. In this study, we performed RNA sequencing to identify whether the candidate circRNA cirTmcc1 is specifically expressed in the brain cortex in a bile duct ligation (BDL) mouse model of hepatic encephalopathy.
Results: Based on transcriptional and cellular analysis, we investigated the circTmcc1-dysregulation-induced changes in the expression of several genes that are associated with intracellular metabolism and astrocyte function. We found that the circTmcc1 binds with the NF-κB p65-CREB transcriptional complex and regulates the expression of the astrocyte transporter EAAT2. In addition, circTmcc1 contributed to the secretion of proinflammatory mediators and glutamate metabolism in astrocytes and subsequently modulated an improvement in spatial memory by mediating neuronal synaptic plasticity.
Conclusions: To sum up, circTmcc1 may be a promising circRNA candidate for targeted interventions to prevent and treat the neuropathophysiological complications that occur due to hepatic encephalopathy.
Keyword: circTmcc1, Hyperammonia, Astrocyte, Neuroinflammation

MOP2-2-10 *Mini-Oral Presentation 2-2*

Lyso-globotriaosylsphingosine induces endothelial dysfunction via autophagy-dependent regulation of necroptosis

황애랑*, 우창훈

영남대학교 약리학과

Objectives: Fabry disease is a lysosomal storage disorder characterized by the lysosomal accumulations of glycosphingolipids in a variety of cytotypes, which include endothelial cells. The disease is inherited and originates from an error in glycosphingolipid catabolism caused by insufficient α-galactosidase A activity, which causes uncontrolled progressive storage of intracellular globotriaosylceramide (Gb3) in the vasculature and extracellular accumulation of lyso-Gb3 (a deacetylated soluble form of Gb3). Necrosis can lead to inflammation, which exacerbates necrosis and creates a positive feedback loop that triggers necroinflammation. However, the role played by necroptosis, a form of programmed necrotic cell death, in the cell-to-cell inflammatory reaction between epithelial and endothelial cells is unclear. Thus, the present study was undertaken to determine whether lyso-Gb3 induces necroptosis and whether necroptosis inhibition protects endothelial dysfunction against lyso-Gb3 inflamed retinal pigment epithelial cells.
Methods: MLKL oligomerization, Real time quantitative RT-PCR, TEM, Immunofluorescence analysis, SA-beta-Gal staining for cellular senescence.
Results: We found lyso-Gb3 induced necroptosis of a retinal pigment epithelial cell line (ARPE-19) in an autophagy-dependent manner and that conditioned media (CM) from ARPE-19 cells treated with lyso-Gb3 induced the necroptosis, inflammation, and senescence of human umbilical vein endothelial cells. In addition, a pharmacological study showed CM from lyso-Gb3 treated ARPE-19 cells induced endothelial necroptosis, inflammation, and senescence were significantly inhibited by an autophagy inhibitor (3-MA) and by two necroptosis inhibitors (necrostatin and GSK-872), respectively.
Conclusions: These results demonstrate lyso-Gb3 induces necroptosis via autophagy and suggest that lyso-Gb3 inflamed retinal pigment epithelial cells trigger endothelial dysfunction via the autophagy-dependent necroptosis pathway. This study suggests the involvement of a novel autophagy-dependent necroptosis pathway in the regulation of endothelial dysfunction in Fabry disease.
Keyword: Autophagy, Glycosphingolipids, Necroptosis, Cellular senescence, Endothelial dysfunction

MOP2-2-11 *Mini-Oral Presentation 2-2*

Melatonin alleviates experimental autoimmune myocarditis-mediated myocardial inflammation

양선부[1*], 이왕수[2], Thi Van Trang Luong[1], 김재택[1]

[1]중앙대학교 의과대학 내분비내과, [2]중앙대학교 의과대학 순환기/심장내과

Objectives: Melatonin attenuates inflammation and cardiac dysfunction in myocardial infarction. However, whether melatonin exerts any effect on myocardial inflammation in the autoimmune myocarditis is unclear. In the present study, we investigated whether melatonin regulates myocardial inflammation in mice with experimental autoimmune myocarditis (EAM).
Methods: Male A/J mice received subcutaneous immunization with MyHC-α peptide on days 0 and 7 to establish the EAM model. Melatonin was administered daily via intraperitoneal route for 21 days and mice were euthanized on day 21.
Results: EAM mice exhibited increased Th17 cell activation, TNF, IL-1beta, IL-6, and F4/80 expressions. However, malatonin significantly suppressed these changes.
Conclusions: Our results suggest that malatonin effectively prevents the development of myocarditis.
Keyword: Myosin heavy chain alpha, EAM, Melatonin, Myocarditis, Th17 cell, Inflammation

MOP2-2-12 *Mini-Oral Presentation 2-2*

PDGFR-β signaling mediates MCP-1 expression in vascular smooth muscle cells with repeated mechanical stress

김지원*, 김주연, 배희은, 김치대
부산대학교 융합의과학과

Objectives: Vascular smooth muscle cells (VSMCs) undergoing biophysical stress play an active role in the progression of vascular inflammation, however, the precise mechanisms are unclear. Thus, the cellular expression of monocyte chemoattractant protein 1 (MCP-1) and its related mechanisms were investigated using cultured rat aortic VSMCs stimulated with mechanical stretch (MS, equibiaxial cyclic stretch, 60 cycles/min).
Methods: When cells were stimulated with 10% MS, MCP-1 expression was markedly increased compared to those in cells stimulated with low intensity of MS (3% or 5%). In an ELISA analysis, HMGB1 released into culture media was increased in cells stimulated with 10% MS, compared to those in cells stimulated with 3% MS. By pretreatment with glycyrrhizin, an inhibitor for HMGB1, MCP-1 expression was markedly attenuated in cells stimulated with 10% MS, suggesting a pivotal role of HMGB1 on MCP-1 expression. In Western blot, an increased expression of both PDGFR-α and PDGFR-β was demonstrated in cells stimulated with 10% MS compared to 3% MS-stimulated cells. In cells deficient of PDGFR-β using siRNA, but not PDGFR-α, HMGB1 released into culture media was significantly attenuated in 10% MS-stimulated cells. Likewise, MCP-1 expression induced in 10% MS-stimulated cells was also attenuated in cells deficient of PDGFR-β.
Conclusions: Taken together, the PDGFR-β signaling plays a pivotal role on the increased expression of MCP-1 in VSMCs stressed with 10% MS. Thus, targeting PDGFR-β signaling in VSMCs might be a promising therapeutic strategy for vascular complications in the vasculatures undergoing excessive biophysical stress.
Keyword: MCP-1, HMGB1, PDGFR

MOP2-2-13 *Mini-Oral Presentation 2-2*

Differential regulatory effects of exercise and hypocaloric diet on adipose thermogenesis and inflammation in obese mice

Vivi Julietta[1*], Shindy Soedono[1,3], Eun Bi Ma[2], Yuha Joo[1], Dan Vo Hoang Nguyet[3], Maria Averia[1], Hadia Nawaz[3], Okgyu Kim[3], Yeonwoo Choi[3], Byeong Chul Oh[4], Chan Hee Lee[5], Joo Young Huh[2], 조계원[1,3]
[1]순천향대학교 의생명융합학과, [2]Chonnam National University, College of Pharmacy, [3]순천향대학교 순천향의생명연구원(SIMS), [4]Gachon University, Department of Physiology, [5]Hallym University, Department of Biomedical Science

Objectives: Adipose tissue (AT) inflammation and thermogenesis are critical regulatory factors contributing to obesity-associated metabolic dysregulation. While diet and exercise are known to attenuate obesity, the impacts of a hypocaloric diet and exercise on weight loss-associated AT metabolism and their underlying mechanisms remain unelucidated. Here, we investigate the effects of equivalent weight loss induced by either exercise or calorie reduction on metabolic dysregulation, AT inflammation, and thermogenesis in obese mice.
Methods: Obese mice fed high-fat diets (HFD) were exercise trained (EX, n=8) or weight-matched to EX via caloric reduction (CR, n=8), and compared with ad libitum HFD-fed mice (Con, n=8). Metabolic parameters were assessed upon 8 weeks of exercise, and inflammatory indicators were examined using flow cytometry, histological analysis, and biochemical assays.
Results: EX and CR both reduced adiposity and improved glucose tolerance and insulin sensitivity. While EX and CR both reduced macrophage accumulation in AT, CR, but not EX, decreased circulating neutrophil and monocyte numbers. Gene expression analysis revealed that only EX significantly increased the expression of anti-inflammatory genes Adipoq and Ym1 in visceral AT. EX also enhanced the expression of fat oxidation-related genes in visceral AT, including Ppara, Pgc1a, and Acox1. Additionally, EX upregulated thermogenesis genes in subcutaneous AT, including Ucp1, Cidea, and Prdm16.
Conclusions: Both EX and CR reduced AT inflammation, however, EX led to more robust changes in anti-inflammatory gene expressions, increased fat oxidation, and enhanced indices of thermogenesis function. Our findings indicate that exercise uniquely regulates AT function, which may be attributed to the metabolic benefits of exercise.
Keyword: Adipose tissue, Exercise, Calorie reduction, Inflammation, Thermogenesis

MOP2-2-14 *Mini-Oral Presentation 2-2*

Nitric oxide releasing nanofiber stimulates revascularization in response to ischemia via cGMP-dependent protein kinase

김원*

경희대학교병원

Objectives: Nitric oxide (NO) promotes angiogenesis via various mechanisms; however, the effective transmission of NO in ischemic diseases is unclear. Herein, we tested whether NO-releasing nanofibers modulate therapeutic angiogenesis in an animal hindlimb ischemia model.
Methods: Male wild-type C57BL/6 mice with surgically-induced hindlimb ischemia were treated with NO-releasing 3-methylaminopropyltrimethoxysilane (MAP3) -derived nanofiber or control fiber, by applying them to the wound for 20 min, three times every two days. The amount of NO from the nanofiber into tissues was assessed by NO fluorometric assay. The activity of cGMP-dependent protein kinase (PKG) was determined by western blot analysis. Perfusion ratios were measured 2, 4, and 14 days after inducing ischemia using laser doppler imaging. On day 4, Immunohistochemistry (IHC) with F4/80 and gelatin zymography were performed. IHC with CD31 was performed on day 14. To determine the angiogenic potential of NO-releasing nanofibers, aorta-ring explants were treated with MAP3 or control fiber for 20 min, and the sprout lengths were examined after 6 days.
Results: As per either LDPI (Laser doppler perfusion image) ratio or CD31 capillary density measurement, angiogenesis in the ischemic hindlimb was improved in the MAP3 nanofiber group; further, the total nitrate/nitrite concentration in the adduct muscle increased. The number of macrophage infiltrations and matrix metalloproteinase-9 (MMP-9) activity decreased. Vasodilator-stimulated phosphoprotein (VASP), one of the major substrates for PKG, increased phosphorylation in the MAP3 group. MAP3 nanofiber or NO donor SNAP (s-nitroso-n-acetyl penicillamine)-treated aortic explants showed enhanced sprouting in an ex vivo aortic ring assay, which was partially abrogated by KT5823, a potent inhibitor of PKG.
Conclusions: These findings suggest that the novel NO-releasing nanofiber, MAP3 activates PKG and promotes therapeutic angiogenesis in response to hindlimb ischemia.
Keyword: Nitric oxide

MOP2-3-01 *Mini-Oral Presentation 2-3*

HK660S (β-lapachone) prevents diabetic cardiomyopathy by regulating cardiac inflammation, fibrosis, apoptosis and lipotoxicity in high fat diet-streptozotocin-induced diabetic mice

Bui Van Nam[1,2*], Hyoung Kyu Kim[1], Pham Trong Kha[1], Jin Han[1]

[1]Cardiovascular and Metabolic Disease Center, Smart Marine Therapeutic Center, Department of Physiology, College of Medicine, Inje University, Busan, South Korea,
[2]Department of Stroke, 103 Hospital, Vietnam Millitary Medical University, Hanoi, Vietnam

Objectives: This study aimed to investigate the effects of HK660S, a newly developed β-lapachone analogue that modulated the inflammation, fibrosis, apoptosis and cardiac lipotoxicity, and boosted antioxidant defenses on DCM and explore its underlying mechanisms.
Methods: C57BL/6 seven weeks male mice were used HFD and low-dose STZ to established DM mouse modal. Mice were randomly divided into six groups: WT (wild type mice), WT+HK80, DM, DM+HK20, DM+HK80 and DM+Met mice were fed with HK660S 20 mg/kg/day (HK20), 80mg/kg/day (HK80) and Metformin 200 mg/kg/day (Met) combined with HFD treatment for 10 weeks, STZ intraperitoneal (ip) injection for 5 consecutive days after 2 weeks of treatment to study the protective effect of HK660S on DCM.
Results: Treatment of diabetic mice with HK660S showed significant decreases in blood glucose, HOMA-IR, cardiac function markers and blood lipid profile values, while insulin levels increased. In addition, HK660S also improved the cardiac function of diabetic mice. The activities of creatine kinase MB, LDH and AST in the serum were significantly reduced, and ameliorated hypertrophy and fibrosis by reducing heart weight, LVDd, IVSd, LVPWd, and protein expression of Cola1, TGF-β1, NF-κB, elastin, IGFBP7, p-Smads, and Smads in the heart tissue. Oral administration of HK660S significantly protected against increases in NF-kB p65 and pro-inflammatory cytokine levels ten weeks after induction. Furthermore, HK660S blocked the apoptotic pathway by increasing Bcl-2 levels and reducing Bax and caspase-3 levels and prevented cardiac lipotoxicity by reducing the accumulation of lipid droplets in the myocardium by inhibiting CD36, ACSL1, FABP3, and PPAR-gamma and enhancing FOXO1 phosphorylation.
Conclusions: HK660S demonstrated its hypoglycemic and antidyslipidemic effects, improved cardiac function in DM mice, reduced hypertrophy, anti-inflammatory potential, anti-apoptotic effects and enhanced antioxidant defense. Therefore, these results indicate that HK660S has a protective effect on the heart of diabetic patients, and it is recommended as a supplement for diabetic patient.
Keyword: Diabetic mellitus, Diabetic cardiomyopathy, Beta-lapachone, Lipids, Apoptosis, Inflammation, Fibrosis

MOP2-3-02 *Mini-Oral Presentation 2-3*

Downregulation of TRPA1 decreases fibrosis markers in TGFβ1-treated mouse cardiac fibroblast

Flores Jessa[2*], Nammi Park[1], Marquez Jubert[1], Garcia Maria Victoria Faith[1], Jeongrim Ko[1], Hyoung Kyu Kim[1,2], Jin Han[1,2]

[1]Cardiovascular and Metabolic Diseases Center, Inje University Busan, [2]Department of Physiology, Inje University Busan

Objectives: This study aims to evaluate the regulation of Transient Receptor Potential Ankyrin 1 (TRPA1) and subsequently determine its role in the physiology of cardiac fibrosis in vitro.
Methods: Western blot was performed to check the TRPA1 protein levels in vitro (mouse cardiac fibroblasts) fibrotic model. RT-PCR analysis was used to measure gene expression while immunocytochemistry was used to visualize the protein expression in cells. To create an in vitro fibrotic model, mouse cardiac fibroblasts (MCF) were treated with rhTGFβ1 (5 ng/mL). On the other hand, to create a TRPA1 knockdown model, siRNA targeting TRPA1 was treated to the cells.
Results: MCF treated with rhTGFβ1 recorded increased levels of TRPA1 as well as upregulated protein levels of the fibrotic markers: α-SMA and Col1a1. Meanwhile, knocking down of TRPA1 using siRNA reveresed the aforementioned upregulation of fibrotic markers. Further exploration of possible molecular pathway to explain these results, phosphorylation of SMAD2 and ERK1/2 proteins were also checked. It was observed that decreased levels of TRPA1 under fibrotic conditions attenuated the phosphorylation of SMAD2 and ERK1/2 which eventually led to decreased fibrotic markers.
Conclusions: The findings of this study showed that inhibition of TRPA1 is a potential strategy in addressing against TGF-β1-induced cardiac fibrosis. The study also provided evidence that TRPA1 affects the progression of fibrosis through the fibrotic ERK1/2 signaling pathway.
Keyword: TRPA1, Cardiac fibrosis, TGFβ1

MOP2-3-03 *Mini-Oral Presentation 2-3*

Evaluation of bioresorbable vascular scaffold in a pig coronary artery model

Dae Sung Park[1,2,3*], Yu Jeong Jin[1,2], Mi Hyang Na[1,2], Jung Ha Kim[1,2], Young Joon Hong[1,2,4], Doo Sun Sim[1,2,4], Kyung Hoon Cho[1,2,4], Dae Young Hyun[1,2,4], Seok Oh[1,2,4], Jung Hoon Kim[1,2,4], Myung Ho Jeong[1,2,4]

[1]The Korean Cardiovascular Stent Research Institute, Jangsung, Republic of Korea, [2]The Cardiovascular Convergence Research Center of Chonnam National University Hospital Designated by the Korean Ministry of Health and Welfare, Gwangju, Republic of Korea, [3]The Research Institute of Medical Sciences, Chonnam National University Gwangju, Republic of Korea, [4]Department of Cardiology, Chonnam National University Hospital, Gwangju, Republic of Korea

Objectives: The drug-eluting stents developed so far are permanently implanted, leaving the risk of stent fracture, local inflammation, and stent thrombosis. Therefore, it is very necessary to develop a new bioresorbable vascular scaffold (BVS) that can replace it. The aim of this study was to compare the safety and efficacy of drug-free bioresorbable vascular scaffold (BVS) with that of sirolimus eluting BVS in vascular treatment of porcine coronary arteries.
Methods: An drug-free BVS [poly(L-lactide) (PLLA) scaffold backbone 3.0×18 mm, n=3] and sirolimus-eluting BVS [poly(L-lactide) (PLLA) scaffold backbone with drug sirolimus and polymer poly(D,L-lactide) (PDLLA) (SE-BVS) 3.0×18 mm, n=3] was implanted in pig coronary arteries. The stents were implanted in the coronary artery at a 1.1:1 stent to artery ratio. Three stented coronary arteries in each group were finally analyzed using X-ray angiography, optical coherence tomography (OCT), and histopathologic evaluation 1 months after stenting.
Results: In the 4-week follow-up angiography and OCT examination, the minimal luminal diameter was smaller and diameter stenosis was more severe in the drug-free BVS group. Histomorphological findings showed no significant differences in internal elastic lumen (3.61±0.535 vs. 3.70±0.750, p=NS) and injury score (1.1±0.21 vs. 1.0±0.06, p=NS) between drug-free BVS and SE-BVS. Neointima area (2.57±0.786 vs. 1.38±0.407, p<0.001), percent stenosis (72.2±20.47 vs. 38.8±12.94, p<0.001), and Inflammation score (1.8±0.71 vs. 1.1±0.07, p<0.001) were significantly higher in the drug-free BVS group. The fibrin score (0.5±0.69 vs. 2.5±0.51, p<0.001) was higher in the SE-BVS group.
Conclusions: Therefore, the SE-BVS in this study can be used as a promising stent for the treatment of lipid atherosclerosis in coronary artery disease.
Keyword: Bioresorbable vascular scaffold, Drug-free, Sirolimus

MOP2-3-04 *Mini-Oral Presentation 2-3*

The positive effects of fermented momordica charantia with leuconostoc mesenteroides MKSR on metabolic disorders in C57BL/6 mice fed a diet high in fat and cholesterol

장현수*, 문희원, 조민서, 한은비, 이지수, 하정헌, 김미숙

단국대학교 식품영양학과

Objectives: This study aimed to investigate the beneficial effects of Momordica charantia (MC) extract and MC fermented with Leuconostoc mesenteroides MKSR (FMC) on metabolic complications arising from the consumption of a high-fat and high-cholesterol diet.
Methods: Male C57BL/6 mice, aged 5 weeks, were divided into six groups subjected to different dietary interventions: a normal diet group (ND), a high-fat and high-cholesterol diet group (HFCD), and four groups fed the HFCD supplemented with either 1% or 4% MC extract (HFCD + 1M and HFCD + 4M, respectively) or 1% or 4% fermented MC (HFCD + 1F and HFCD + 4F, respectively).
Results: Over a 12-week experimental period, the inclusion of MC fermented with L. mesenteroides MKSR in the diet significantly reduced weight gain in white adipose tissues (specifically in the epididymal and retroperitoneal white adipose tissues), lowered serum alanine aminotransferase levels, and decreased hepatic triglyceride and total cholesterol levels. Furthermore, FMC improved glucose tolerance, insulin sensitivity, and facilitated cholesterol elimination in the experimental mice through feces. It also increased hepatic mRNA expressions related to cholesterol efflux, indicating a mechanism to counterbalance the enhanced cholesterol elimination. FMC modified genes linked to both the breakdown and synthesis in white adipose tissue, including sterol regulatory element-binding protein 1c, lipoprotein lipase, peroxisome proliferator-activated receptor alpha, and adiponectin.
Conclusions: The results strongly suggest that FMC, through fermentation with the probiotic L. mesenteroides MKSR, enhances the protective effects of MC against metabolic complications associated with a high-fat and high-cholesterol diet.
Keyword: Momordica charantia, Leuconostoc, Metabolic syndrome

MOP2-3-05 *Mini-Oral Presentation 2-3*

The role of exercise-induced cereblon for metabolism

서대윤*, 한진

인제대학교 기초과학

Objectives: Cereblon (CRBN) serves as a substrate receptor of the E3 ubiquitin ligase complex and plays a critical role in regulating AMPK activity. Exercise is known to be a key modulator of AMPK. However, the precise cellular mechanisms governing CRBN regulation during exercise training remain.
Methods: In this study, we investigated the role of CRBN during exercise in both animal and human models. We conducted aerobic exercise experiments in STZ-induced animal models and examined its impact on CRBN and AMPK levels in skeletal muscle. Additionally, we evaluated the effects of a 12-week aerobic exercise regimen on CRBN serum levels in pre-diabetic patients and its relationship with aerobic exercise capacity. Resistance exercise was also studied for its impact on CRBN serum levels. Furthermore, we conducted experiments involving moderate acute exercise in college students and explored its influence on CRBN serum levels.
Results: Our findings indicate that aerobic exercise leads to a decrease in CRBN levels and an increase in AMPK levels in the skeletal muscle of STZ-induced animal models, resulting in improved glucose metabolism. Additionally, a 12-week aerobic exercise program was associated with decreased CRBN serum levels in pre-diabetic patients. Notably, the reduction in CRBN serum levels was negatively correlated with aerobic exercise capacity in these patients. In contrast, resistance exercise did not induce changes in CRBN serum levels. Furthermore, moderate acute exercise resulted in decreased CRBN serum levels in college students, while acute high-intensity exercise exhibited a trend towards increased CRBN serum levels.
Conclusions: Our study suggests that aerobic exercise has a significant impact on reducing CRBN levels in both serum and skeletal muscle. Moreover, our results indicate that different types of exercise may regulate CRBN levels, potentially making CRBN a target for exercise-induced metabolic alterations. These findings contribute to a better understanding of the intricate cellular mechanisms involved in exercise-induced adaptations.
Keyword: Exercise, Cereblon

MOP2-3-06 *Mini-Oral Presentation 2-3*

IKKε involves in pathological alterations of macrophages in response to cardiac injury

조향희*, 김용숙, 조동임, 전주희, 강보경, 유수지, 조미영, 유진, 안영근

전남대학교병원 순환기/심장내과

Objectives: Myocardial infarction (MI) is a leading cause of heart failure globally, triggering chronic inflammation and cardiac fibrosis. Inhibitor of NF-kB kinase ε (IKKε) is an inflammation modulator in various diseases, and its pathophysiological role in the heart remains unclear. In this study, the involvement of IKKε in cardiac inflammation and fibrosis was examined in a mouse MI model.
Methods: MI was induced in IKKε knockout (KO) mice by coronary artery ligation, and macrophages were isolated from the bone marrows or heart tissues for further studies by Western blot, PCR, and flow cytometry. Cell phenotypes were identified by immunofluorescence staining.
Results: MI resulted in a poor survival rate, heightened inflammatory responses, pronounced cardiac fibrosis, and low ejection fraction in the IKKε KO compared to the wild type group. Non-myocyte cells were isolated from infarcted heart tissues for single cell RNA sequencing (scRNA-seq) and flow cytometry analysis. We found macrophage-myofibroblast transition (MMT) was accelerated in IKKε KO group. Next, to examine whether MMT-induced macrophages acquire fibrotic features, the collagen 1 and fibrotic markers were measured in isolated cardiac macrophages. Especially M2 macrophages, rather than M1, exhibit fibrotic features under MI conditions. Cell phenotypic change with MMT features was also identified in the human failing hearts. We performed a phosphorylated protein array in macrophages to find the responsible mediator for enhanced inflammation and MMT in IKKε KO macrophages. We found that the level of phosphorylated p38 (p-p38) was lower in IKKε KO macrophages than in WT macrophages, and inflammation and MMT were accelerated in p38 inactivated macrophages. Administration of 5-azacytidine, a cardiac protective reagent, restored p-p38 in macrophages and inhibited MMT in a mouse MI model.
Conclusions: These findings underscore the regulation of inflammation response and macrophage transition by the IKKε-p38 axis, indicating MMT as a promising therapeutic target for ischemic heart disease.
Keyword: Myocardial infarction, IKKε, Macrophage-myofibroblast transtion, Inflammation

MOP2-3-07 *Mini-Oral Presentation 2-3*

Roles of circular RNAs in age-related macular degeneration

류영서[1,2*], 정다희[1,2], 김영국[1,2]

[1]전남대학교 의과대학 생화학교실, [2]전남대학교 Biomedical Science Graduate Program (BMSGP)

Objectives: Age-related macular degeneration (AMD) is a disease that leads to visual impairment in older individuals, primarily caused by the degeneration of the retinal pigment epithelium (RPE). The accumulation of oxidative stress due to aging plays a significant role in the dysfunction of RPE cells, ultimately leading to the development of AMD. In this progressive disease, damaged RPE cells release pro-inflammatory factors into the extracellular space, resulting in macrophage infiltration. In the late stages of AMD, new blood vessels form in the subretinal or sub-RPE area, leading to a deterioration of vision. Recent studies are underway to explore RNA-based drugs that can target protein-coding and non-coding RNAs (ncRNAs) to treat various diseases. However, the role of non-coding RNAs, including circular RNAs (circRNAs), has not been fully elucidated in AMD. CircRNAs are a group of single-stranded transcripts with a closed circular structure typically produced through back-splicing. This study aims to elucidate the role of circRNAs in RPE cells.
Methods: By analyzing total RNA sequencing data from a mouse RPE model with laser-induced choroidal neovascularization, which mimics AMD, we identified a list of differentially expressed circRNAs. We selected five circRNAs from the list for functional analysis: circSNRK, circOXR1, circCEP112, circSTRN3, and circTCF20.
Results: Interestingly, we found that the knockdown of circSNRK in ARPE-19 cells upregulated pro-inflammatory and angiogenic genes (ICAM1, CXCL8, and HIF1A) and increased macrophage migration, suggesting that circSNRK is involved in inflammatory response. Additionally, transcriptome analysis of ARPE-19 cells depleted of circSNRK indicated that the knockdown of circSNRK primarily regulated the expression of cell proliferation-related genes. Furthermore, we confirmed that circSNRK acts as a miRNA sponge, thereby regulating the target genes of each miRNA.
Conclusions: Consequently, this study reveals that circSNRK regulates inflammation and proliferation in RPE cells, offering the potential for circSNRK to serve as a target for new treatments for AMD.
Keyword: Circular RNAs, Retinal pigment epithelium, Age-related macular degeneration

MOP2-3-08 *Mini-Oral Presentation 2-3*

Echinochrome A prevents diabetic nephropathy by enhancing mitochondrial function via AMPKα/NRF2/HO-1 signaling pathway

Nguyen Thi To Hoai[1*], Pham Trong Kha[1,2], Yun Hyeong Rok[1], Vu Thi Thu[2], Luu Thi Thu Phuong[2], Hyoung Kyu Kim[1], Jin Han[1]

[1]Department of Physiology, Cardiovascular and Metabolic Disease Center, Smart Marine Therapeutic Center, College of Medicine, Inje University, Busan, Korea, [2]University of Science, Vietnam National University, Hanoi, Vietnam

Objectives: Echinochrome A (EchA), a natural bioproduct extracted from sea urchins, exerts antioxidant and beneficial effects in various inflammatory disease models. However, its effects on diabetic nephropathy (DN) remain poorly understood.
Methods: In the present study, seven-week-old diabetic and obese db/db mice were injected with EchA (3 mg/kg/day) intraperitoneally for 12 weeks, while db/db control mice and wild-type (WT) mice received an equal amount of sterile 0.9% saline.
Results: EchA improved glucose tolerance and reduced blood urea nitrogen (BUN) and serum creatinine levels but did not affect body weight. Moreover, EchA enhanced AMPK phosphorylation and NRF2/HO-1 signaling contributing to improve mitochondrial function and antioxidant activity. Mechanistically, EchA suppressed oxidative stress and fibrosis by downregulating p53 and c-Jun phosphorylation, and attenuating NADPH oxidase 4 (NOX4) and transforming growth factor-beta 1 (TGFβ1) signaling. In addition, EchA decreased renal malondialdehyde (MDA) and lipid hydroperoxide levels. Histologically, EchA treatment ameliorated renal fibrosis.
Conclusions: Collectively, the findings demonstrated that EchA prevents DN via elevating AMPKα/NRF2/HO-1 signaling pathways in db/db mice, may provide a therapeutic option for DN.
Keyword: Echinochrome A, Diabetic nephropathy

MOP2-3-09 *Mini-Oral Presentation 2-3*

APE1/Ref-1 as a novel biomarker and therapeutic target in ApoE-/- mice on a western diet

Byeong Hwa Jeon[1,2,3*], Yu Ran Lee[1], Hee Kyoung Joo[1], Eun-Ok Lee[1], Sungmin Kim[1,3], Hao Jin[1,3], Cuk Seong Kim[1,3]

[1]충남대학교 의과대학 생리학교실, [2]충남대학교병원 의생명연구원, [5]충남대학교 의과대학 의과학과

Objectives: Apurinic/apyrimidinic endonuclease 1/Redox factor-1 (APE1/Ref-1) is a multifunctional protein involved in base excision DNA repair and transcriptional gene expression regulation. However, the role of APE1/Ref-1 in atherosclerosis is unclear. Herein, we investigated the role of APE1/Ref-1 in atherosclerotic apolipoprotein E (ApoE-/-) mice fed with a Western-type diet.
Methods: In this study, we used 8-week-old male apoprotein E-knockout mice Mice were fed with either a normal diet or a Western-type diet containing 21% fat, 34% sucrose, 19.5% casein, and 0.2% cholesterol for 20 weeks. Plasma levels of APE1/Ref-1 were determined using an APE1/Ref-1 sandwich enzyme-linked immunosorbent assay kit (MediRedox, Daejeon, Korea).
Results: Serologic level of APE1/Ref-1 was strongly correlated with vascular inflammation in these mice. In the ApoE-/- mice fed with a Western-type diet, we observed an increase in the neutrophil/lymphocyte ratio (NLR), endothelial cell/macrophage activation, and atherosclerotic plaque formation, all indicators of atherosclerotic inflammation. APE1/Ref-1 expression was upregulated in the aortic tissues of these mice, and APE1/Ref-1 co-localization suggests its expression specifically in endothelial cells and macrophages, as indicated by positivity for CD31 and galectin-3. Interestingly, APE1/Ref-1 plasma levels in ApoE-/- mice on a Western-type diet were significantly higher compared to those in mice on a normal diet, and levels were suppressed by atorvastatin administration. Correlation analysis revealed a strong correlation between plasma APE1/Ref-1 levels and NLR, a marker of systemic inflammation. This strong correlation underscores the potential of APE1/Ref-1 levels as a biomarker for atherosclerotic inflammation. The cut-off value for APE1/Ref-1 in mice model for predicting atherosclerotic inflammation at 4.903 ng/mL showed a sensitivity of 100% and specificity of 91%.
Conclusions: Our findings confirm that increased APE1/Ref-1 expression in aortic endothelial cells/macrophages and elevated plasma levels can predict atherosclerotic inflammation in atherosclerotic mice.
Keyword: Apurinic/apyrimidinic endonuclease 1/redox factor-1 (APE1/Ref-1), Apoprotein E-knockout mice, CD31, Galectin-3, Biomarker

MOP2-3-10 *Mini-Oral Presentation 2-3*

Comparison of low-density lipoprotein cholesterol estimation methods: analysis of individuals according to current smoking status

배한준*, 정해원

대구가톨릭대학 순환기/심장내과

Objectives: Smoking is associated with elevated low-density lipoprotein cholesterol (LDL-C) levels. However, the effects of smoking on the differences between the LDL formula and direct LDL are unclear. This study aimed to analyze the relationship between smoking and three equations for LDL-C estimation.
Methods: Data on LDL-C and other lipid components were collected from the Korea National Health and Nutrition Examination Survey. Direct LDL-C levels were measured using an enzymatic method in 19,685 participants, while data regarding smoking were available in 15,283 participants. Difference between estimated and direct low-density lipoprotein cholesterol levels by enzymatic method was assessed by various measures for test accuracy correlation coefficient (R2), root mean square error, mean absolute difference, mean absolute error, and median absolute deviation with 95% confidence intervals.
Results: Current smokers had higher mean absolute difference values when triglyceride and direct LDL-C levels were <400 mg/dL and <69 mg/dL, respectively. The median absolute deviation values obtained with the Martin equation were comparatively more accurate when the direct LDL-C level was <40 mg/dL, and the Sampson equation estimate was lower when the direct LDL-C level was <100 mg/dL with smoking than other equations.
Conclusions: This study found that the Sampson equation was appropriate for patients with triglyceride levels >400 mg/dL, while the Martin equation was appropriate for smoking status. If the calculated LDL level is <70 mg/dL, the Martin equation may be more accurate than other equations.
Keyword: Cholesterol, Low-density lipoprotein, Friedewald equation, Martin-hopkins equation

MOP2-3-11 *Mini-Oral Presentation 2-3*

The role of lipogenic pathway SCAP/SREBP in retina angiogenesis and blood-retinal barrier function

양해영[1*], 박현진[1], 최원일[1], 김하일[1], 김인준[1,2]

[1]Graduate School of Medical Science and Engineering, Korea Advanced Institute of Science and Technology,
[2]BioMedical Research Center, Korea Advanced Institute of Science and Technology

Objectives: Sterol regulatory element-binding proteins (SREBPs), activated by SREBP cleavage-activating protein (SCAP), serve as pivotal regulators of lipogenesis across diverse cell types. While endothelial cell (EC) metabolism regarding glucose, and amino acids is widely implicated in vascular development and homeostasis, EC lipid metabolism in vascular regulation remains largely unexplored. In this investigation, we explore the contribution of the endothelial SCAP/SREBP pathway in angiogenesis and blood-retinal barrier (BRB) function.
Methods: Postnatal mouse retina and oxygen-induced retinopathy (OIR) model were used to investigate both developmental and pathological angiogenesis. We generated an inducible deletion of Scap in ECs by crossbreeding Cdh5-CreERT2 mice with Scap floxed mice and injecting tamoxifen, resulting in ScapECKO. We injected the dextran intravenously to evaluate BRB function. To analyze EC mRNA expression profile, we utilized the Ribotag system to enrich EC mRNAs for RNA sequencing (RNA-seq).
Results: SCAP and SREBP1, but not SREBP2, exhibit expression in postnatal retinal ECs. In ScapECKO, postnatal retinal angiogenesis was impaired, showing a similar phenotype after the blockade of the VEGF pathway. Endothelial proliferation was decreased in the retinal EC in ScapECKO. Pathologic neovascularization and leakage decreased in OIR mouse retina after Scap deletion. RNA-seq data showed decreased expression of genes associated with angiogenesis in ScapECKO. Gene set enrichment analysis suggested downregulation of the SREBP1-related pathway in ScapECKO. Some downregulated genes in ScapECKO were also downregulated after VEGF pathway blockade. These commonly downregulated genes were implicated in angiogenesis.
Conclusions: The SCAP/SREBP pathway plays a role in retina angiogenesis and BRB disruption and could be a potential target for blinding diseases.
Keyword: SREBP, SCAP, Angiogenesis, Oxygen-induced retinopathy, Blood-retinal barrier

MOP2-3-12 *Mini-Oral Presentation 2-3*

Cardiac-specific CRBN knockout leads to heart failure via cardiac senescence and fibrosis in 37 weeks mice

Hyeong Rok Yun*, Nguyen Thi To Hoai, Hyoung Kyu Kim, Pham Trong Kha, Jin Han

Inje University, Cardiovascular and Metabolic Disease Center

Objectives: This study aimed to delve into the enduring repercussions of cardiac-specific CRBN knockout on cardiac function and metabolism in mice. By examining the long-term effects, we sought to elucidate the significance of CRBN in sustaining heart health over an extended period.
Methods: In this investigation, we subjected cardiac-specific CRBN knockout mice aged 37 weeks to comprehensive analyses. Through a range of established techniques and assays, we evaluated key parameters including cardiac hypertrophy, systolic function, lipid metabolism, mitochondrial function, cardiac senescence, and fibrosis. These methodologies provided insights into the intricate interplay between CRBN and various aspects of cardiac physiology and metabolism.
Results: The findings revealed compelling evidence of significant cardiac hypertrophy and systolic dysfunction in CRBN knockout mice at 37 weeks. Notably, disruptions in lipid metabolism, characterized by AMPK hyperactivation and subsequent mitochondrial dysfunction, were observed. Moreover, sustained CRBN knockout elicited an increase in cardiac senescence and fibrosis, indicative of progressive cardiac deterioration over time.
Conclusions: In conclusion, our study underscores the critical role of CRBN in maintaining metabolic homeostasis and preserving cardiac function. Prolonged CRBN depletion engenders adverse effects on multiple facets of cardiac physiology, highlighting its indispensability for sustained heart health. These insights contribute to a deeper understanding of the molecular mechanisms governing cardiac metabolism and pave the way for potential therapeutic interventions targeting CRBN-related pathways in cardiovascular diseases.
Keyword: Heart failure, Cereblon, Senescence

MOP2-3-13 *Mini-Oral Presentation 2-3*

Attenuation of atherosclerosis via inhibition of adipocyte differentiation and NF-kB pathway activation in macrophages by hordeum vulgare L. extract

Min Ho Kang*, Min Ho Han, Ha Neul Choi, Jin Woo Kim

Sunmoon University, Department of Food Science

Objectives: Atherosclerosis is cardiovascular disease caused by chronic inflammation due to increased influx of monocyte into vascular endothelial tissue, following elevated accumulation of reactive oxygen species and oxidized low-density lipoprotein. The purpose of this study was to explore the anti-atherosclerotic effects of Hordeum vulgare L. extract (HE) by evaluating its anti-oxidant activity, inhibition of adipocyte precursor differentiation, and suppression of NF-kB pathway activation, with the aim of discovering novel functional materials for promoting vascular health.
Methods: Anti-oxidant substances quantified by measuring polyphenol and flavonoid content, and anti-oxidant activity evaluated via DPPH and ABTS assays. Effects on adipocyte precursor differentiation assessed using Oil Red O staining and gene expression analysis (SREBP-1c, PPARγ, C/EBP-α). Influence of HE on LPS-stimulated macrophages examined by measuring nitric oxide (NO) production and gene expression (NF-kB, iNOS, TNF-α, IL-6).
Results: Anti-oxidant substance content and activity of HE showed 12.1 gallic acid equivalents mg/g dry matter (DM), 7.19 quercetin equivalent mg/g DM, 83.2%, and 82.7% scavenging effect, anticipating Inhibition of lipoprotein oxidation and vascular cell aging. Adipocyte precursors showed 12.3% reduction in lipid droplet formation, with key differentiation genes SREBP-1c, PPARγ, and C/EBP-α decreasing by 19.9 ~ 36.3%, suggesting inhibited intracellular lipid accumulation and adipokine production, reducing vascular endothelial deposition. HE reduced NO in macrophage, suggesting prevention of vascular wall damage by reducing inflammatory signaling, attributed to reduced expression of NF-kB pathway activation genes and cytokine genes, NF-kB, iNOS, TNF-α, IL-1β, with decreases of 42.8 ~ 73.7%.
Conclusions: The superior anti-oxidant activity, ability to inhibit adipocyte differentiation, and capacity to reduce lipid and inflammation of HE demonstrate its potential as a promising natural material for atherosclerosis management. The therapeutic value stemming from decreased lipoprotein oxidation and vascular aging underscores its utility in the food and pharmaceutical industries, supported by its outstanding antioxidant, anti-hyperlipidemic, and anti-inflammatory properties.
Keyword: Atherosclerosis, Anti-oxidant, Adipocyte differentiation, NF-kB pathway

MOP2-4-01 *Mini-Oral Presentation 2-4*

Cytokine-induced apoptosis inhibitor 1 negatively regulates p53 transcription by ROS-mediated nuclear translocation, promoting vascular smooth muscle cell proliferation and migration

이성표*, 한주희

우석대학교 약학과

Objectives: Abnormal vascular smooth muscle cell (VSMC) proliferation and migration lead to neointima formation, ultimately resulting in cardiovascular hyperplastic diseases. Cytokine-induced apoptosis inhibitor 1 (CIAPIN1) has been identified as an anti-apoptotic molecule by inhibiting p53 expression and promoting VSMC proliferation and migration; however, the underlying mechanism is still unclear.
Methods: Here, we used rat aortic SMCs as the primary cell model to evaluate the role of CIAPIN1 in VSMC proliferation and migration. Additionally, loss/gain-of-function approaches were employed to determine how CIAPIN1 regulates p53 transcription in platelet-derived growth factor (PDGF)-BB-stimulated VSMC dysfunction.
Results: Our results show that CIAPIN1 increases in PDGF-BB-stimulated VSMC. CIAPIN1 deficiency significantly induces expression of p53; however, CIAPIN1 overexpression decreases expression of p53 in PDGF-BB-stimulated VSMC. Mechanistically, CIAPIN1 binds to p53 promoter regions and suppresses its transcriptional activity and downstream signaling, promoting VSMC cell cycle progression and migration. We further revealed that reactive oxygen species (ROS) were essential for interactions with CIAPIN1 and p53, which occur via CIAPIN1 translocation into the nucleus.
Conclusions: These findings provide new insights into the mechanism by which CIAPIN1 regulates p53 transcription to promote VSMC proliferation and migration, offering a novel therapeutic opportunity for treating vascular diseases.
Keyword: CIAPIN1, Vascular smooth muscle cell, p53

MOP2-4-02 *Mini-Oral Presentation 2-4*

Role of toll-like receptor 4 pathway in mediating the preventive effects of isocaloric restriction on high-fat diet-induced metabolic disturbances

정은지[1*], 은성진[1], 채서연[1], 이선혜[2]

[1]Department of Applied Biological Sciences, Sun Moon University, [2]Division of Food Science, Sun Moon University

Objectives: Overconsumption of a high-fat (HF) diet can cause gut microbiota dysbiosis, which increasing gut permeability, causing gut inflammation, and subsequently facilitating the translocation of components of Gram-negative bacteria, lipopolysaccharide (LPS), into circulation. The increased plasma LPS level alters vagal gut-brain communication, impairing the sensitivity to anorexigenic hormones such as cholecystokinin (CCK) and leptin, promoting hyperphagia, consequently leading to metabolic disturbances. Thus, we aimed to investigate the effect of controlling energy overconsumption via isocaloric restriction on HF diet-induced metabolic complication. In our previous study, we demonstrated that isocaloric restriction improved HF diet-induced metabolic disturbances. Here, we further investigated whether the interaction of LPS with its pivotal receptor, toll-like receptor 4 (TLR4), mediates the protective effect of isocaloric restriction on HF-induced metabolic complications.
Methods: Male C57BL/6 mice (n=8/group; 6 weeks old) were divided into three groups and fed on their respective diet for 12 weeks; a low-fat (LF; 10% fat, n=8), HF (45% fat, n=16), and HF with isocaloric restriction (pair-fed to the LF group as a model for the prevention effect; IR, n=8). TAK-242 (3 mg/kg), an antagonist for TLR4, was administered throughout the study period to suppress TLR4 expression. After 12 weeks on their respective diet, general phenotypes, gut-brain signaling, and inflammatory cytokines were examined.
Results: Isocaloric restriction suppressed body weight gain and decreased fasting blood glucose levels. Isocaloric restriction prevented the HF-induced impairment in the sensitivity to CCK, as a proxy for gut-brain signaling. The IR group also suppressed HF-induced downregulation of leptin and PYY in the liver, visceral fat, and duodenum, indicating an appetite suppressing effect. Further, the IR group showed significantly downregulated expression of inflammatory cytokines in adipose tissue.
Conclusions: Taken together, these data suggest that isocaloric restriction improves general phenotype, glycemic control, gut-brain signaling, and inflammatory profiles, which was not mediated by LPS-TLR4 pathway.
Keyword: Toll-like receptor 4, Gut-brain axis, Isocaloric restriction, High-fat diet, Lipopolysaccharide

MOP2-4-03 *Mini-Oral Presentation 2-4*

Novel HDAC8 inhibitor YAK577 attenuates vascular calcification in vivo and in vitro

기해진[1*], 정성민[1], Thomas Kurz[3], 정명호[2]

[1]심혈관계융합연구센터 순환기/심장내과, [2]광주 보훈병원 순환기/심장내과, [3]하인리하인대학교 약학과

Objectives: Vascular calcification is associated with advanced age, atherosclerosis, and metabolic diseases such as diabetes and chronic kidney failure. Even though the treatment of vascular calcification is crucial, there is no effective treatment to prevent or reverse the calcification process. Histone deacetylase (HDAC) enzymes have been reported to be involved in the gene regulation. HDAC1, HDAC5, and HDAC6 prevented vascular calcification, whereas HDAC4 and HDAC9 promoted vascular calcification. We hypothesized that HDAC8 may be involved in promoting vascular calcification.
Methods: Vascular calcification was induced by inorganic phosphate plus ascorbic acid treatment in vascular smooth muscle cells (VSMCs) and subcutaneously injection of vitamin D3 in BL6 mice. Calcification was confirmed by Alizarin Red staining, calcium assay, pro-calcification marker gene expression. Overexpression and knockdown of HDAC8 was performed in VSMCs or A10 cells. In vivo experiments, HDAC8 inhibitor YAK577 (10 mg/kg/day) was intraperitoneally injected to vitamin D3-treated mice for 7 days.
Results: HDAC8 inhibitors (PCI34051 and YAK577) treatment reduced inorganic phosphate plus ascorbic acid-induced calcification. Of the two drugs, YAK577 was more effective than PCI34051 in inhibiting calcification. YAK577 treatment attenuated calcification medium-induced upregulation of HDAC8 and pro-calcification marker genes (Bmp2, Runx2, and Msx2). Small interfering RNA for HDAC8 significantly reduced the calcification medium-induced calcium deposition in VSMCs. HDAC8 overexpression increased the pro-calcification genes and decreased the anti-calcification marker genes (Fgf23 and Mgp). YAK577 treatment reversed the HDAC8 overexpression-mediated Mgp downregulation in A10 cells. YAK577 administration suppressed the vitamin D3-induced calcium accumulation in aorta tissues. YAK577 treatment mitigated the vitamin D3-induced pro-calcification genes (Bmp2, Runx2, and Msx2) and Opn expression as well as calcium content.
Conclusions: We suggest that HDAC8 could be new therapeutic target for the treatment of vascular calcification.
Keyword: YAK577, Vascular calcification, HDAC8

MOP2-4-04 *Mini-Oral Presentation 2-4*

Inhibitory effects of human milk oligosaccharide on lipopolysaccharide-induced acute lung injury by suppressing STAT1/NF-κB-mediated inflammation

Lan Phuong Phan[2*], 진유진[1], Thuy Le Lam Nguyen[1], 김리라[3], 허경선[1]

[1]충남대학교 약학대학 약리학과, [2]베트남 하노이 대학교 자연과학대학, [3](주)진켐

Objectives: Acute lung injury (ALI) is primary activator for developing respiratory diseases including chronic obstructive pulmonary disease and pulmonary fibrosis. Mechanistically, sustained and excessive differentiation of macrophages induced lung inflammation by releasing of pro-inflammatory cytokines and its activation was increased mucus secretion and fever. Lipopolysaccharide (LPS) is the component of gram-negative bacteria wall and various studies utilized for inflammation by inducing macrophage differentiation. The present study aimed to investigate the protective effects of human milk oligosaccharides, specifically 3′-sialyllactose (3′-SL) and 6′-sialyllactose (6′-SL), on LPS-induced ALI and elucidate their underlying signaling pathways.
Methods: The inhibitory effects of 3′-SL and 6′-SL on inflammation were evaluated using LPS-treated RAW 264.7 macrophages. To establish the ALI model, mice were treated with 10 mg/kg LPS for 24 h. Histological changes in the lung tissues were assessed using hematoxylin and eosin staining and immunofluorescence.
Results: LPS causes thickening of the alveolar wall infiltration of immune cells in lung tissues and increased serum levels of TNF-α, IL-1β, and GM-CSF. However, these effects were significantly alleviated by 100 mg/kg of 3′-SL and 6′-SL. Consistent with the inhibitory effects of 3′-SL and 6′-SL on LPS-induced pro-inflammatory cytokine secretion in serum, 3′-SL and 6′-SL suppressed mRNA expression of TNF-α, IL-1β, MCP-1, iNOS, and COX2 in LPS-induced RAW 264.7 cells. Mechanistically, 3′-SL and 6′-SL abolished LPS-mediated phosphorylation of NF-κB and STAT1. Bioinformatically, under BioGRID, STAT1 and NF-κB p65 was physically interact with each other. However, fludarabine treatment, a STAT1 inhibitor, did not affect LPS-mediated NF-κB phosphorylation. Finally, fludarabine pretreatment synergically enhanced anti-inflammatory effects of 3′-SL and 6′-SL by suppressing mRNA expression of TNF-α, IL-1β, MCP-1, iNOS, and COX2.
Conclusions: 3′-SL and 6′-SL protect LPS-induced macrophage activation and ALI through the STAT1 and NF-κB signaling pathways.
Keyword: Inflammation, Acute lung injury, Sialyllactose

MOP2-4-05 *Mini-Oral Presentation 2-4*

Transcriptome analysis reveals that a high-iron diet triggers de novo cholesterol synthesis

이지수*, 강다현, 조민서, 윤성진, 장현수, 하정헌
단국대학교 식품영양학과

Objectives: High iron intake stimulates de novo cholesterol synthesis, whereas additional copper intake gradually reduces overall synthesis. This study aimed to investigate the response of hepatic cholesterol metabolism to these changes by analyzing the hepatic transcriptome.
Methods: Four-week-old male Sprague-Dawley rats were given free access to AIN-93G standard diets with varying levels of iron (adequate; AdFe, 100 ppm and high; HFe, 7,000 ppm) and copper (low; LCu, 0.3 ppm, adequate; AdCu, 5 ppm, high; HCu, 165 ppm).
Results: To explore the impact of HFe on lipid profiles and cardiovascular disease risk factors, we analyzed blood lipid and cholesterol levels in experimental animals. The study found that varying the diet with different iron-copper combinations did not significantly affect serum triglyceride levels. However, serum cholesterol levels showed dynamic changes; HFe intake increased total cholesterol (TC), high-density lipoprotein cholesterol (HDL-C), and non-HDL cholesterol levels, while additional copper intake mitigated the rise in cholesterol. To elucidate the effects observed in lipid profiles, we conducted a comparative analysis of the hepatic transcriptome. Notably, changes in the transcriptome of rats consuming HFe/LCu were associated with elevated cholesterol levels during lipid metabolism. HFe/LCu downregulated genes associated with β-oxidation (2,4-DECR, CROT, carnitine O-acetyltransferase) and upregulated genes related to biosynthesis of cholesterol (17β-HSD7, PCSK9, SQLE, IPPI) and fatty acid (FAS, ACSS2). However, providing additional copper to experimental animals normalized all thesse genetic alterations.
Conclusions: In summary, high iron intake affected cholesterol levels, showing varied responses to the addition of copper in the diet. The analysis of the hepatic transcriptome suggests complex interactions with implications for cardiovascular health, underscoring the necessity for further research.
Keyword: High-iron, Transcriptome, Cholesterol synthesis

MOP2-4-06 *Mini-Oral Presentation 2-4*

STAT3-ER stress feedback loop is associated with endothelial to mesenchymal transition in lipopolysaccharide-treated vascular endothelial cell injury

진유진*, 허경선
충남대학교 약학대학 약리학과

Objectives: Endothelial to mesenchymal transition (EndMT) is a process that involves the loss of endothelial cell (EC) phenotype and induced plaque instability during the progression of atherosclerosis. Primarily, EC injury, caused by an accelerated inflammatory signaling pathway, plays a crucial role in activating EC dysfunction by inducing endoplasmic reticulum (ER) stress and disrupting EC junctions. In the present study, we investigated the molecular mechanisms between EndMT and ER stress on STAT3-mdeiated inflammation under LPS-treated human umbilical vein EC (HUVEC).
Methods: Western blot, siRNA transfection, and immunofluorescence assay were employed to evaluate signaling pathways and target proteins. The phenotypic switching of ECs on the endothelium of the mouse aorta after challenging 10 mg/kg LPS was assessed by en face staining.
Results: Deactivation of STAT3 by stattic pretreatment abolished not only LPS-mediated STAT3 phosphorylation, but also VCAM-1 and ICAM-1. In addition, STAT3 inhibition surprisingly reversed LPS-induced mesenchymal markers, including SM22α and vimentin, and the suppression of endothelial marker VE-cadherin. Mechanistically, stattic treatment suppressed LPS-induced EndMT progression by inhibiting protein expression and nuclear translocation of Smad2/3. Furthermore, LPS-induced disrupted EC homeostasis by PERK or IRE-1α/CHOP-mediated ER stress, whereas stattic abolished CHOP expression. Interestingly, depletion of CHOP by siRNA transfection significantly abolished LPS-mediated EndMT progression. Notably, the absence of CHOP significantly suppressed phosphorylation of STAT3 and its nuclear expression as well. Consistent to in vitro data, STAT3 inhibition by stattic injection significantly suppressed LPS-mediated STAT3 activation as well as CHOP expression in the mouse endothelium.
Conclusions: Collectively, the STAT3-CHOP axis may contribute to the loss of EC function and induce mesenchymal differentiation by aggravating chronic inflammation and EndMT.
Keyword: Endothelial to mesenchymal transition, Endoplasmic reticulum stress, STAT3, CHOP, Endothelial cell dysfunction

MOP2-4-07 *Mini-Oral Presentation 2-4*

Taurine mediated cardio protection mechanisms in ob/ob mice

Kainat Ahmed[1,2*], Jung Eun Yim[2]

[1]창원대학교 시니어휴먼에콜로지협동과정, [2]창원대학교 식품영양학과

Objectives: Taurine, a non-essential amino acid, is reported to play vital role in modulation of cardiovascular functions in various mouse models. However, the mechanisms by which taurine causes the effects in ob/ob mice are yet to be explored. This study aimed to verify the beneficial effects of taurine in heart of ob/ob mice and to highlight the mechanisms of the action.
Methods: Five-week-old male C57BL/6-Lepob/ob mice (n=16) and C57BL/6J mice (n=8) as a normal control were fed chow ad libitum for 10 weeks. After 1-week adaptation period, C57BL/6- Lep ob/ob mice were randomly divided into two groups of negative control and taurine group. The taurine group mice were provided orally with taurine (2g/kg bodyweight) for 10 weeks. At sacrifice, the heart tissues were weighed and collected for analysis of genes related to inflammation, fatty acid oxidation and energy metabolism. Total cholesterol (high and low density), Triglycerides, and C- reactive proteins were measured in the serum.
Results: Taurine consumption significantly reduced the final body, liver, total WAT weight, and a decreasing trend in serum triglycerides, C- reactive protein content, and LDL-Cholesterol. Moreover, taurine caused increase in HDL-Cholesterol with significant increase in the expression of hemeoxygenase-1, which is related to anti-oxidation, and PPAR gamma coactivator 1, involved in energy homeostasis, in the heart tissues compared to that of the negative control.
Conclusions: Taurine may have an ameliorating effect on preventing cardiomyopathy by decreasing serum levels of cholesterol and by enhancing anti-oxidation and regulating energy balance in the heart of ob/ob mice.
Keyword: PPAR gamma coactivator 1, Hemeoxygenase-1, Taurine

MOP2-4-08 *Mini-Oral Presentation 2-4*

Angiopoietin-like 4 stabilizes atherosclerotic plaques by modulating the phenotypic transition of endothelial cells and vascular smooth muscle cells

조동임*, 김용숙, 조향희, 강보경, 조미영, 유수지, 전주희, 유진, 안영근

전남대학교 병원 순환기/심장내과

Objectives: Atherosclerosis, the leading cause of death, is a vascular disease of chronic inflammation. We recently showed that angiopoietin-like 4 (ANGPTL4) promotes cardiac repair by suppressing pathological inflammation. Given the fundamental contribution of inflammation to atherosclerosis, we assessed the role of ANGPTL4 in the development of atherosclerosis and determined whether ANGPTL4 regulates atherosclerotic plaque stability.
Methods: We injected ANGPTL4 protein twice a week into atherosclerotic Apoe-/- mice and analyzed the atherosclerotic lesion size, endothelial dysfunction, inflammation, and plaque stability.
Results: In atherosclerotic mice, ANGPTL4 reduced the atherosclerotic plaque size, endothelial dysfunction, and vascular inflammation. Furthermore, ANGPTL4 was associated with maintenance of vascular integrity and attenuated endothelial-to-mesenchymal transition (EndMT) in both HUVEC and atherosclerotic plaque of Apoe-/- mice. In the atherosclerotic lesions and fibrous caps, the number of a-SMA(+), SM22a(+), and SM-MHC(+) cells was higher, while the number of CD68(+) and Mac2(+) cells was lower in the ANGPTL4 group. Most importantly, the fibrous cap was significantly thicker in the ANGPTL4 group than in the PBS group. Smooth muscle cells (SMCs) isolated from atherosclerotic aortas showed significantly increased expression of CD68 and Krüppel-like factor 4 (KLF4), a modulator of the vascular SMC phenotype, along with downregulation of a-SMA, and these changes were attenuated by ANGPTL4 treatment. Furthermore, ANGPTL4 reduced TNFa-induced NADPH oxidase 1 (NOX1), a major source of reactive oxygen species, resulting in attenuation of KLF4-mediated SMC phenotypic changes.
Conclusions: Our results reveal that ANGPTL4 treatment inhibits atherogenesis and suggest that targeting vascular stability and inflammation may serve as a novel therapeutic strategy to prevent and treat atherosclerosis. Even more importantly, ANGPTL4 treatment inhibited the phenotypic changes of SMCs into macrophage-like cells by downregulating NOX1 activation of KLF4, leading to the formation of more stable plaques.
Keyword: Angiopoietin-like 4, Atherosclerosis, Plaque stabilization, Inflammation

MOP2-4-09 *Mini-Oral Presentation 2-4*

Lactoferrin contributes to the development of CRPC by promoting the growth of prostate cancer cells

금혜진*, 하정민, 진서연, 엄채영, 김서영, 정해림, 배순식

부산대학교 약리학과

Objectives: The normal prostate and early-stage prostate cancers (PCa) are stimulated by androgens for growth and survival, and androgen deprivation therapy (ADT) causes them to regress. Although ADT shows significant therapeutic efficacy, almost all the hormone-sensitive prostate cancer (HSPC) patients progress to castration-resistant prostate cancer (CRPC). Mechanisms of castration resistant remain unclear, and it is hard to cure. Therefore, identification and functional analysis of target genes that are involved in the castration resistance may contribute to the therapeutic development of prostate cancer patients.
Results: In the present study, gene expression profile was analyzed in the samples isolated from the same patient with HSPC and CRPC. 61 genes were found to be differentially expressed between HSPC and CRPC tissue. Among 61genes, expression pattern for 14 gene of each upregulated or downregulated target was evaluated using prostate cancer cell lines and tissues from prostate cancer patients. Expression of Lactoferrin (LTF) was consistently upregulated in hormone-sensitive prostate cancer cell lines as well as tissue form CRPC patients. Silencing LTF expression in PC3 cells showed low proliferation rates, whereas, migration was not affected.
Conclusions: These results suggest that LTF play an important role in proliferation and growth of prostate cancer cell and could be a target for the therapeutic development of prostate cancer patients.
Keyword: LTF, HSPC, CRPC, Prostate cancer

MOP2-4-10 *Mini-Oral Presentation 2-4*

The effects of Ferroptosis progress and some related genes in Psoriasis disease

Thien Nguyen Huu*, Jung Eun Seol, Hyoung Kyu Kim, Jin Han

Inje University, College of Medicine

Objectives: Psoriasis is a chronic inflammatory skin disease with a strong genetic predisposition and autoimmune pathogenic traits. It is characterized by sharply demarcated, erythematous, pruritic plaques covered in silvery scales. The pathogenesis of psoriasis relates to immune cells and the increase of proinflammatory cytokines, especially in the IL-23/Th17 axis. Recently, there are many studies found the role of ferroptosis in the pathogenesis of many diseases related to the immune system such as acute kidney injury, intracerebral hemorrhage, and neurodegenerative diseases. So, in this study, we investigate the process of ferroptosis and some related genes in the Psoriasis model.
Methods: We induce the Psoriasis model on mice with 5% Imiquimod cream and induce the ferroptosis model on keratinocyte cells by erastin. Investigate some ferroptosis makers and related genes in these models. Skin parameters were measured and investigating skin lesions by hematoxylin and eosin staining. Western blot was used for protein levels evaluation and mRNA levels were measured by RT-PCR.
Results: There are proliferation and thickening of epidermal keratinocyte cells besides the increase in the water loss index and a significant decrease in the skin hydration index as well melamine index decreased, and the erythema index increased in the skin hydration index in a psoriasis model. Moreover, there are changes in ferroptosis makers such as GPX4, and ACSL4, and the increase of proinflammatory cytokines in vivo Psoriasis model. We also found that keratinocytes are sensitive to ferroptosis inducers and ferroptosis in this cell line is related to some genes that we investigate.
Conclusions: The study shows the role of the ferroptosis process and some related genes in the Psoriasis model.
Keyword: Ferroptosis, Psoriasis, Lipid peroxidation, Cell death program

MOP2-4-11 *Mini-Oral Presentation 2-4*

miR204 induces non-alcoholic fatty liver disease through cpt1 inhibition in hepatocytes

김민수*, Vu Giang Huong, 전소희, 전병화, 김국성
충남대학교 의과대학 생리학

Objectives: Non-alcoholic fatty liver disease (NAFLD) is a liver condition not caused by heavy alcohol consumption; its main characteristic is excess fat storage in liver cells. Imbalance of energy homeostasis causes dysfunction of muscles, liver, and adipocytes, leading to metabolic disease. To remove excess energy, it is necessary to increase metabolic activity via fatty acid βoxidation. Promoting the activation of lipid metabolism in mitochondria is essential, mitochondrial dysfunction causes obesity related disorders, but the underlying mechanisms are unclear.
Methods: We investigate the functional significance of miR204 in the evolution of NAFLD. We analyzed the body weight, epididymal fat-pad weight, lipid droplet in liver, blood parameter and inflammation in IDH2 KO mice feed a normal diet (ND) or HFD.
Results: IDH2 KO mice feed a normal diet (ND) or HFD increased body weight, epididymal fat-pad weight, lipid droplet in liver, blood parameter and inflammation compared to WT mice fed a ND or HFD. Moreover, the expression of miR204 is increased in mice with IDH2 deficiency. Increased miR204 by IDH2 deficiency regulates carnitine palmitoyl transferase 1a (cpt1a) synthesis, which inhibits fatty acid β-oxidation. Inhibition of miR204 prevents the disassembly of two fatty acid-related genes by activating CPT1a expression, which decreases lipid droplet in liver, inflammatory cytokines, epidiymal fat pad weight, blood parameters.
Conclusions: Our findings reveal a novel post-transcriptional mechanism by which miR204 regulates CPT1a expression in the pathogenesis of NAFLD.
Keyword: miR204, NAFLD, IDH2

MOP2-4-12 *Mini-Oral Presentation 2-4*

Echinochrome A inhibits HMGB1-induced osteopontin expression of vascular smooth muscle cell via AP-1 signaling

김주연*, 김지원, 배희은, 김치대
부산대학교 의과대학 융합의과학과 약리학교실

Objectives: Echinochrome A (Ech A), a natural compound isolated from marine organisms, has been identified as a major therapeutic effector for various cardiovascular diseases, but its precise role and mechanisms are still unclear. Thus, this study investigated the role of Ech A on vascular smooth muscle cells (VSMCs) migration induced by high-mobility group box 1 (HMGB1), one of the best characterized damage-associated molecular patterns (DAMPs) mediating the progression of vascular complications.
Results: The cultured A10 cells were stimulated with HMGB1 (100 ng/ml), and then cell migration was measured using a wound-healing assay. Compared to control cells, the migration in HMGB1-stimulated cells was increased in time-course manners for 48 h. In VSMCs stimulated with HMGB1, the expression of OPN mRNA and protein was increased. Also HMGB1-induced VSMC migration was significantly attenuated in cells pretreated with MPIIIB10 (100ng/ml), a neutralizing monoclonal antibody for OPN. In this experiment, the HMGB1-induced OPN expression was significantly attenuated in cells pretreated with Ech A. Likewise, Ech A (3 or 10 μM) inhibited HMGB1-induced VSMC migration in a concentration-dependent manner. In reporter gene assays using OPN promoter-luciferase constructs, the promoter region 538-234 bp of the transcription start site was shown to be responsible for transcriptional activity enhancement by HMGB1, which was significantly inhibited by Ech A.The binding for AP-1 in the promoter region was increased, which was also attenuated by Ech A.
Conclusions: The migration of VSMCs was increased in association with an increased OPN expression in cells stimulated with HMGB1, which were suppressed by Ech A. The inhibitory effect of Ech A on OPN expression in HMGB1-stimulated cells was mediated by AP-1 signaling, and suggested as a key mechanism involved in the effects of Ech A on VSMC migration. These characteristics suggest that Ech A might be a potential candidate drug to alleviate vascular remodeling in the injured vasculatures.
Keyword: Echinochrome A, HMGB1, Osteopontin, VSMC, Migration

MOP2-4-13 *Mini-Oral Presentation 2-4*

Cbl-b E3 ligase-mediated neddylation and activation of PARP-1 induces vascular calcification

권덕화*, 이윤경, 정안나, 임용운, 신세라, 국현
전남대학교 의과대학

Objectives: Vascular calcification (VC) refers to the accumulation of mineral deposits on the walls of arteries and veins, and it is closely associated with increased mortality in cardiovascular disease, particularly among high-risk patients with diabetes and chronic kidney diseases (CKD). Neuronal precursor cell-expressed developmentally downregulated protein 8 (NEDD8) is an ubiquitin-like protein that plays a pivotal role in various cellular functions, primarily through its conjugation to target proteins and subsequent relay of biological signals. However, the role of NEDDylation in VC has not been investigated.
Methods: To undercover which proteins undergo NEDD8-mediated modifications, we conducted immunoprecipitation-based proteomic analysis using an anti-NEDD8 antibody, followed by the affinity purification and liquid chromatography-mass spectrometry. In Pi-treated RVSMCs, NEDD8-conjugated proteins were isolated and the NEDDylation was confirmed through Coomassie blue staining. The gel bands that increased in the Pi lane and decreased in the lane with Pi and MLN4924 were cut out and subjected to LC-MS/MS to identify the NEDDylated proteins.
Results: In our study, we observed that MLN4924, an inhibitor of the NEDD8-activating E1 enzyme, effectively impedes progress of VC. By LC-MS/MS analysis, we identified that poly(ADP-ribose) polymerase 1 (PARP-1) is subjected to NEDD8 conjugation, leading to an increase in PARP-1 activity during VC. Subsequently, we uncovered that the PARP-1 NEDDylation is mediated by the E3 ligase Cbl proto-oncogene B (Cbl-b) and is reversed by the NEDD8-specific protease 1 (NEDP-1) during VC. Furthermore, Cbl-b C373 peptide effectively mitigates the inactive form of E3 ligase activity of Cbl-b, ultimately preventing VC.
Conclusions: These findings provide compelling evidence that the NEDD8-dependent activation of PARP-1 represents a novel mechanism underlying vascular calcification and suggests a promising new therapeutic target for VC.
Keyword: Vascular calcification, NEDD8, PARP-1, Cbl-b, NEDP-1

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[04168] 서울시 마포구 마포대로 68 마포아크로타워 707호

전 화 02-3272-5330

팩 스 02-3272-5331

이메일 ksla@lipid.or.kr

편집제작 플랜베어
[07806] 서울시 강서구 공항대로 220 우성에스비타워II 11층 1101호

전 화 02-6734-1011

팩 스 02-6734-1009

이메일 info@planbear.co.kr

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Aims and Scope

The Journal of Lipid and Atherosclerosis (JLA), an international and peer-reviewed journal, is the official journal of the Korean Society of Lipid and Atherosclerosis. Its abbreviated title is 'J Lipid Atheroscler (JLA)'. The JLA is published 3 times a year (January 25, May 25, and September 25). The JLA aims to advance knowledge of the mechanisms responsible for lipid metabolism and atherosclerosis and enhance insight into prevention, treatment, and ultimate cure for cardiovascular diseases. The JLA covers basic, translational, and clinical research of lipid and atherosclerosis. The JLA also encourages articles on broad aspects of vascular biology, thrombosis, metabolism, nutrition, or cardiovascular health. JLA also features methodological rounds to provide its readers with educational fundamentals and practical implications for the research which investigates lipid metabolism, atherosclerosis, and cardiovascular diseases. Its regional scope focuses mainly in Korea, but it accepts submissions from researchers all over the world. Manuscripts may be submitted as original articles or reviews. The JLA is published online only, and includes keywords indexing with Medical Subject Heading (MeSH) terms. Full text is freely available on the official website (https://www.e-jla.org). The JLA is indexed/tracked/covered by PubMed Central, PubMed, Scopus, KoreaMed, KoreaMed Synapse, KoMCI, Crossref, and Google Scholar.

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No.707, Mapoacro Tower, 68 Mapo-daero, Mapo-gu,
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+82-2-3272-5331
staff@e-jla.org

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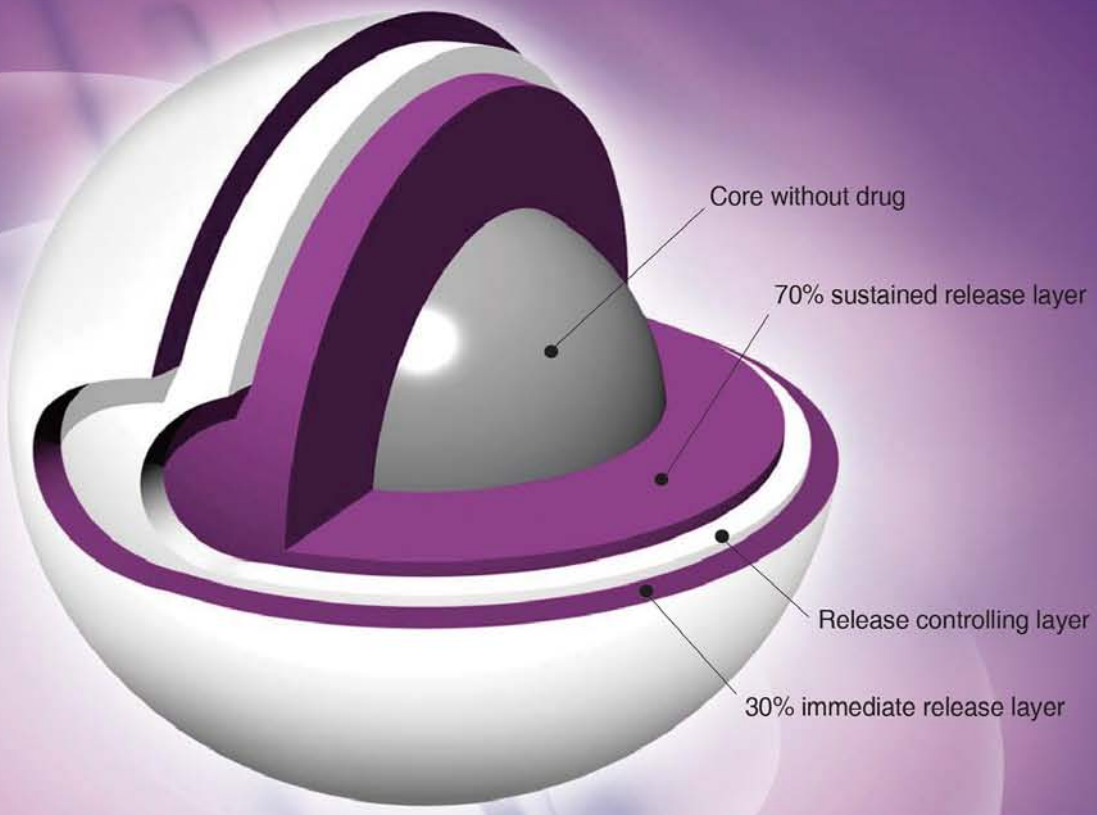

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BIO&CHEMICAL R&D

보건의료전문가용

전문의약품

FENOZETIBE Tab.

Fenofibrate 160mg + Ezetimibe 10mg

새로운 조합의 혼합형 고지혈증 치료제[1]

혼합형 고지혈증 환자의 지질 프로파일 개선[2]

복약순응도 향상을 위한 고정용량 복합제[3,4]

48주 및 52주 장기 임상시험을 통해 양호한 안전성 프로파일 확인[5,6]

References

1. 의약품안전나라 (https://nedrug.mfds.go.kr/) [Acessed on 2022.11.07]. **2.** Farnier M, et al. Eur Heart J. 2005 May;26(9) 897-905. **3.** Divchev D, et al. Cardiol Ther. 2019 Dec;8(2):317-328. **4.** Mach F, et al. Eur Heart J. 2020 Jan 1;41(1):111-188. **5.** S Oikawa et al. J Atheroscler Thromb. 2017 Jan 1;24(1):77-94. **6.** JM McKenney et al. J Am Coll Cardiol. 2006 Apr 18;47(8):1584-7.

[원료약품 및 그 분량] 이 약 1정 중 • 유효성분: 에제티미브(USP) - 10.00 mg 페노피브레이트(EP) - 160.00 mg • 동물유래성분: 스테아르산마그네슘(소-우지), 유당수화물(소-우유) • **기타 첨가제**: 라우릴황산나트륨, 부틸히드록시톨루엔, 오파드라이amb화색(88A180040), 콜로이드성이산화규소, 크로스카멜로오스나트륨, 포비돈, D-만니톨 **[성상]** 흰색의 원형 필름 코팅정 **[효능·효과]** 혼합형 고지혈증 환자의 상승된 총콜레스테롤(total-C), 저밀도지단백 콜레스테롤(LDL-C), 아포지단백 B(Apo B) 및 비-고밀도지단백 콜레스테롤(non-HDL-C)을 감소시키기 위한 식이요법의 보조제로서, 이 약을 투여한다. **[용법·용량]** 성인: 이 약은 1일 1회 1정을 식후 즉시 복용한다. 이 약은 반드시 식이요법과 병행하여 투여한다. 이 약 성분 중 페노피브레이트는 빈속에 흡수가 덜 될 수 있으므로 반드시 식후 즉시 투여한다. ○ 간장애환자에는 투여하지 않는다. ○ 신장애환자는 중등도 ~ 중증 신장애 환자(혈청 크레아티닌치 2.5 mg/dL 이상)의 경우 이 약을 투여하지 않는다. ○ 고령자는 신기능이 감소되지 않은 경우 일반적으로 용량 감량이 필요하지 않다. 에제티미브와 페노피브레이트를 병용으로 복용하고 있는 환자의 경우, 복용의 편리함을 위하여 이 약(개개의 주성분 함량이 동일한 복합제)으로 전환할 수 있다. **[사용상의 주의사항]** 1. 다음 환자에는 투여하지 말 것. 1) 이 약 및 이 약의 구성성분에 과민증이 있는 환자 2) 활성 간질환 환자 혹은 설명되지 않는 혈청 아미노전이효소 수치 증가가 지속되는 환자에게는 이 약과 HMG-CoA 환원효소 억제제를 병용투여하지 않는다. 3) 임부 또는 임신하고 있을 가능성이 있는 여성 및 수유부(6. 임부 또는 수유부에 대한 투여 참조) 4) 간장애환자 5) 중등도 ~ 중증 신장애 환자(혈청 크레아티닌치 2.5 mg/dL 이상)(횡문근융해증이 나타날 수 있다.) 6) 담낭질환 환자(선재성 담낭 질환 환자 포함) 7) 피브레이트 또는 케토프로펜으로 치료하는 동안 광알레르기 또는 광독성을 경험한 환자 8) 소아 9) 담관간경화증 환자 10) 췌장염 환자(중증 고중성지질형증으로 인한 급성 췌장염 제외) 11) 이 약은 유당을 함유하고 있으므로, 갈락토오스 불내성(galactose intolerance), lapp(유당분해효소 결핍증(Lapp lactase deficiency) 또는 포도당-갈락토오스 흡수장애(glucose-galatose-malabsorption) 등의 유전적인 문제가 있는 환자에게는 투여하면 안된다. 2. 다음 환자에게는 신중히 투여할 것 1) 경증 신장애 또는 그 병력이 있는 환자(혈청 크레아티닌치 1.5mg/dL 이상 2.5mg/dL 미만) (횡문근융해증이 나타날 수 있으므로 투여량을 감량 또는 투여간격을 연장하여 사용한다.) 2) 간기능조사에 이상이 있는 환자 또는 그 병력이 있는 환자(간기능 검사값의 이상변동이 나타날 수 있다.) 3) 저알부민혈증(신증후군) 환자 4) 담석의 병력이 있는 환자(담석형성이 보고되었다.) 5) 혈액응고저지제를 투여중인 환자 6) HMG-CoA 환원효소저해제(예, 프라바스타틴, 심바스타틴 등)를 투여중인 환자 7) 고령자 **[포장단위]** 30정/피티피(10정/PTPx3) **[저장방법]** 기밀용기, 실온(1-30℃)에서 보관 **[제조의뢰자]** 알보젠코리아㈜ 경기도 화성시 향남읍 제약공단2길 36 소비자상담번호 : 02-2047-7700 [제조자] 현대약품㈜ 충청남도 천안시 동남구 풍세면 잔다리길 55 **[작성년월일]** 2022-09-15

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SGLT-2, sodium glucose cotransporter 2; TZD, thiazolidinedione.

References. 1. 트루버디정 허가사항, 식품의약품안전처, 의약품안전나라. Accessed on 2023.09.26. 2. 보건복지부 고시 제2023-58호. 요양급여의 적용기준 및 방법에 관한 세부사항.

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[References] 1. DL Tribble et al. Metabolism. 2008 Jun;57(6):796-801. 2. M Farnier et al. Eur Heart J 2005 May;26(9):897-905. 3. SS Kumar et al. Lipids Health Dis. 2009 Dec 17;8:56. 4. Nagula Jayababu. JMSCR. 2019 Oct; 7(10):963-970. 5. A Shinnakasu et al. J Atheroscler Thromb. 2017 Jul 1;24(7):735-748. 6. M Farnier et al. Am Heart J. 2007 Feb;153(2):335.e1-8. 7. M Farnier et al. Diab Vasc Dis Res. 2012 Jul;9(3):205-15. 8. S Oikawa et al. J Atheroscler Thromb. 2017 Jan 1;24(1):77-94. 9. JM McKenney et al. J Am Coll Cardiol. 2006 Apr 18;47(8):1584-7.

[제품요약정보] **제품명** 에제페노 정 **주성분함량** 1정(515mg) 중 에제티미브 10mg 및 페노피브레이트 160mg **성상** 흰색의 원형 필름 코팅정 **효능효과** 혼합형 고지혈증 환자의 상승된 총콜레스테롤(total-C), 저밀도지단백 콜레스테롤(LDL-C), 아포지단백 B(Apo B) 및 비-고밀도지단백 콜레스테롤(non-HDL-C)을 감소시키기 위한 식이요법의 보조제 **용법용량** 성인 : 이 약은 1일 1회 1정을 식후 즉시 복용한다. 이 약은 반드시 식이요법과 병행하여 투여한다. 이 약 성분 중 페노피브레이트는 빈속에 흡수가 덜 될 수 있으므로 반드시 식후 즉시 투여한다. 간장애환자에는 투여하지 않는다. 신장애환자는 중등도~중증 신장애 환자(혈청 크레아티닌치 2.5 mg/dL 이상)의 경우 이 약을 투여하지 않는다. 고령자는 신기능이 감소되지 않은 경우 일반적으로 용량 감량이 필요하지 않다. 에제티미브와 페노피브레이트를 병용으로 복용하고 있는 환자의 경우, 복용의 편리함을 위하여 이 약(개개의 주성분 함량이 동일한 복합제)으로 전환할 수 있다. **사용상의 주의사항(일부)** 1. 다음 환자에는 투여하지 말 것 1) 이 약 및 이 약의 구성성분에 과민증이 있는 환자 2) 활성 간질환 환자 혹은 설명되지 않는 혈청 아미노전이효소 수치 증가가 지속되는 환자에게는 이 약과 HMG-CoA 환원효소 억제제를 병용투여하지 않는다. 3) 임부 또는 임신하고 있을 가능성이 있는 여성 및 수유부 4) 간장애환자 5) 중등도~중증 신장애 환자(혈청 크레아티닌치 2.5 mg/dL 이상)(횡문근융해증이 나타날 수 있다.) 6) 담낭질환 환자(선재성 담낭 질환 환자 포함) 7) 피브레이트 또는 케토프로펜으로 치료하는 동안 광알레르기 또는 광독성을 경험한 환자 8) 소아 9) 담관간경화증 환자 10) 췌장염 환자(중증 고중성지질형증으로 인한 급성 췌장염 제외) 11) 이 약은 유당을 함유하고 있으므로, 갈락토오스 불내성(galactose intolerance), lapp(유당 분해효소 결핍증(Lapp lactase deficiency) 또는 포도당-갈락토오스 흡수장애(glucose-galatose-malabsorption) 등의 유전적인 문제가 있는 환자에게는 투여하면 안된다. 2. 다음 환자에게는 신중히 투여할 것 1) 경증 신장애 또는 그 병력이 있는 환자(혈청 크레아티닌치 1.5mg/dL 이상 2.5mg/dL 미만)(횡문근융해증이 나타날 수 있으므로 투여량을 감량 또는 투여간격을 연장하여 사용한다.) 2) 간기능조사에 이상이 있는 환자 또는 그 병력이 있는 환자(간기능 검사값의 이상변동이 나타날 수 있다.) 3) 저알부민혈증(신증후군) 환자 4) 담석의 병력이 있는 환자(담석형성이 보고되었다.) 5) 혈액응고저지제를 투여중인 환자 6) HMG-CoA 환원효소저해제(예, 프라바스타틴, 심바스타틴 등)를 투여중인 환자 7) 고령자 3. 이상반응: 이 약은 각 단일제인 에제티미브 및 페노피브레이트의 이상반응을 포함한다. **포장단위** 30정/PTP (10정/PTP X3) **저장방법** 기밀용기, 실온(1~30℃) 보관 제품에 대한 자세한 사항은 사용설명서를 참고하시기 바랍니다. 가장 최근 개정된 제품설명서의 내용은 현대약품 홈페이지를 통해 확인하실 수 있습니다. 설명서 작성년월: 2021년 10월

[제조원정보]

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*Moderate-intensity pitavastatin (2-4 mg) vs. moderate-intensity atorvastatin (10-20 mg) and rosuvastatin (5-10 mg).

[References] 1. Choi JY, *et al.* Effect of pitavastatin compared with atorvastatin and rosuvastatin on new-onset diabetes mellitus in patients with acute myocardial infarction. *Am J Cardiol.* 2018;122:922-28. **2.** Data on file. 임상시험 결과보고서. 원발성 고콜레스테롤혈증 환자를 대상으로 AGT, AGZ의 병용요법과 AGT 단일요법의 유효성 및 안전성을 비교평가하기 위한 다기관, 무작위배정, 이중눈가림, 활성 대조, 요인설계 제 3상 임상시험. 2023 (version 2.0).

페바로젯정

[원료약품 및 분량] 1정 중 2/10 mg : 피타바스타틴칼슘(별규) 2 mg, 에제티미브(별규) 10 mg, 4/10 mg : 피타바스타틴칼슘(별규) 4 mg, 에제티미브(별규) 10 mg **[성상]** 2/10 mg : 노란색의 타원형 필름코팅정, 4/10 mg : 분홍색의 타원형 필름코팅정 **[효능/효과]** 원발성 고콜레스테롤혈증 : 원발성 고콜레스테롤혈증(이형접합 가족형 및 비가족형) 또는 혼합형 이상지질혈증 환자의 상승된 총 콜레스테롤(total-C), LDL-콜레스테롤(LDL-C), 아포 B 단백(Apo-B), 트리글리세라이드(TG) 및 non-HDL-콜레스테롤을 감소시키고, HDL-콜레스테롤(HDL-C)을 증가시키기 위한 식이요법의 보조제로서 이 약을 투여한다. **[용법/용량]** 이 약은 식사와 관계없이 1일 1회 투여한다. 이 약을 투여하기 전 또는 투여 중인 환자는 반드시 표준 콜레스테롤 저하식을 지속적으로 해야 한다. 이 약의 투여량은 환자의 LDL-콜레스테롤의 기저치, 권장되는 치료목표치 및 환자의 반응에 따라 조절되어야 한다. 이 약은 초회용량으로 1일 1회 2/10 mg이 권장된다. LDL-콜레스테롤치의 저하효과가 충분하지 않은 경우 1일 최대 4/10 mg까지 증량할 수 있다. LDL-콜레스테롤치, 치료 목표 및 환자의 반응에 따라 4주 또는 그 이상의 간격을 두고 용량을 적절히 조절한다. 피타바스타틴칼슘과 에제티미브를 병용으로 복용하고 있는 환자인 경우, 복용의 편리함을 위하여 이 약(개개의 주성분 함량이 동일한 복합제)으로 전환할 수 있다. **[금기]** 1) 이 약의 구성성분에 과민증이 있거나, 그 병력이 있는 환자 2) 활동성 간질환 환자 또는 원인이 밝혀지지 않은 아미노전이효소수치의 지속적 상승이 있는 환자 3) 중증의 간장애 또는 담도폐쇄가 있는 환자 및 담즙울체 환자 4) 사이클로스포린을 투여중인 환자 5) 근육병증 환자 6) 임부 또는 임신의 가능성이 있는 부인 및 수유부 7) 소아(사용경험이 없다.) 8) 이 약은 유당을 함유하고 있으므로, 갈락토오스 불내성(galactose intolerance), Lapp 유당분해 효소 결핍증(Lapp lactase deficiency) 또는 포도당-갈락토오스 흡수장애(glucose-galactose malabsorption) 등의 유전적인 문제가 있는 환자에게는 투여하면 안된다. **[포장단위]** 30정/병 **[저장방법]** 차광기밀용기, 실온(1~30°C) 보관 **[사용기한]** 제조일로부터 36개월 **[제조자]** 안국약품(주)

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40%의 TG lowering과 20%의 HDL-C raising 효과를 나타내었습니다 Ref.) Clinical Therapeutics/Volume 42, Number 10, 2020(한림제약 스타펜 임상3상)

성분/함량	피타바스타틴칼슘 2mg + 페노피브레이트160mg	용법용량	1일 1회 1캡슐 식사 직후 복용
효능효과	관상동맥심질환(CHD) 고위험이 있는 성인환자에서 피타바스타틴 2mg 단일치료요법시 LDL-콜레스테롤 수치는 적절히 조절되지만 트리글리세라이드 수치는 높고 HDL-콜레스테롤 수치는 낮은 복합형이상지질혈증의 치료	성상	흰색의 원형 필름코팅정과 흰색의 구형 미세과립을 함유 하고 있는 상부 담녹색, 하부 흰색의 경질캡슐
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CREDIBLE LDL-C lowering Effect	BEYOND LDL-C lowering	EXPAND Metabolic Effect
크레더블정 Ezetimibe/Rosuvastatin 10/5, 10/10, 10/20mg	스타펜 Pitavastatin/Fenofibrate 2/160mg	스타젯 Pitavastatin/Ezetimibe 2/10mg, 4/10mg
CV high risk의 Intensive LDL-C lowering을 통한 CV Risk 감소	Mixed Dyslipidemia의 다양한 Lipid Profile 관리를 통한 Residual CV Risk 감소	지질개선 뿐 아니라 긍정적인 Metabolic effect로 안전하고 효과적으로 CV Risk 감소

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다파글리플로진프로판디올메이트
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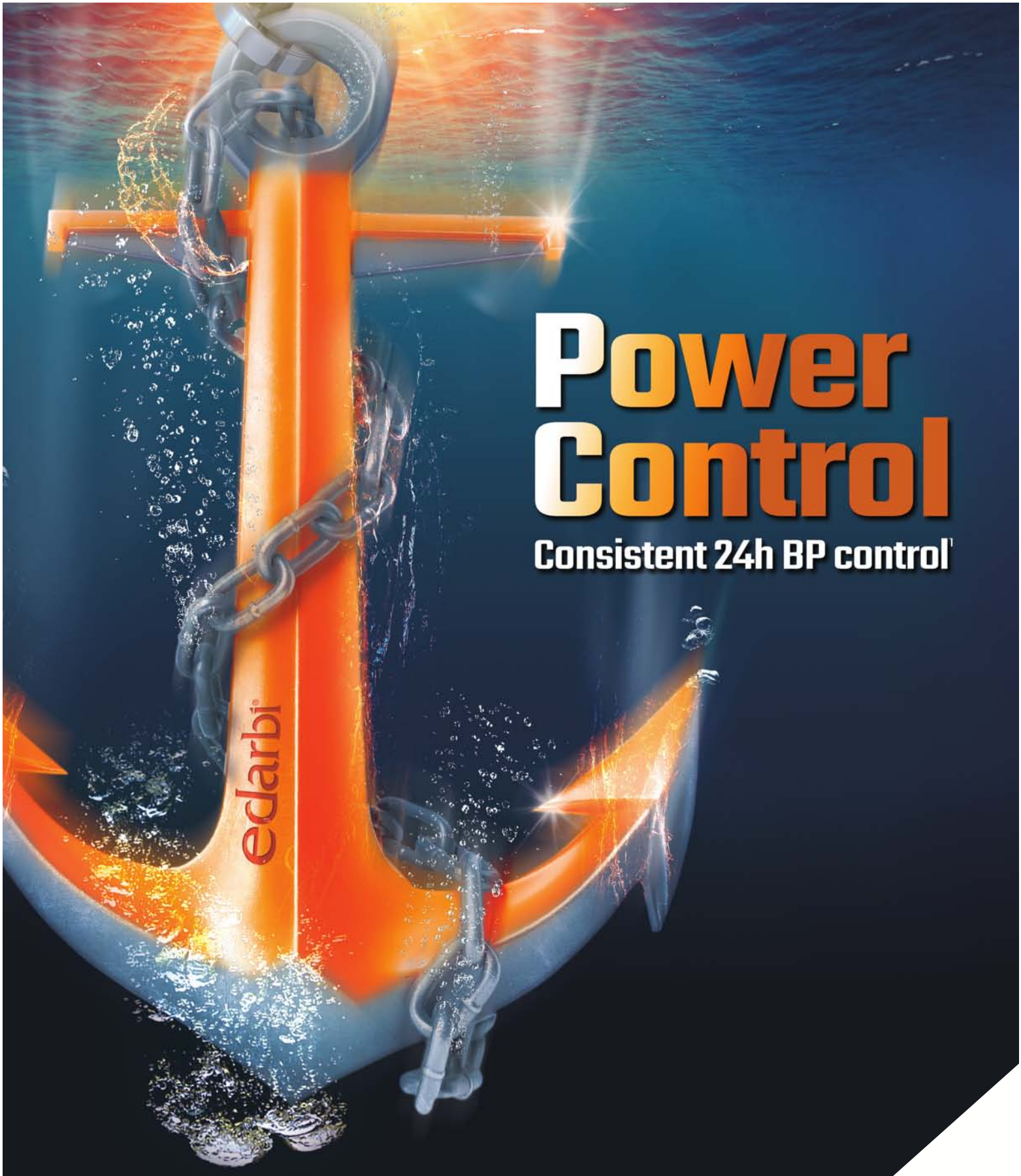

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Reference 1. William B. White, et al. Effects of the Angiotensin Receptor Blocker Azilsartan Medoxomil Versus Olmesartan and Valsartan on Ambulatory and Clinic Blood Pressure in Patients With Stages 1 and 2 Hypertension. Hypertension 2011;57:413–420.

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References 1. 타바로젯 제 3상 임상시험 결과 2. Seo WW, et al. Cardiovasc Diabetol. 2022 May 23;21(1):82.

Drug information

타바로젯정 (피타바스타틴칼슘/에제티미브) 2/10 mg, 4/10 mg [성상] 2/10 mg 노란색의 타원형 필름코팅정, 4/10 mg 분홍색의 타원형 필름코팅정 **[효능·효과]** 원발성 고콜레스테롤혈증 **[용법·용량]** 이 약은 식사와 관계없이 1일 1회 투여한다. **[저장 방법]** 차광기밀 용기, 실온(1~30℃) **[사용기간]** 제조일로부터 36개월 **[포장단위]** 30정/병 ※제품에 대한 자세한 정보는 최신 제품설명서를 참고하시기 바라며, 홈페이지(www.daewonpharm.com)를 통해 확인하실 수 있습니다.

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[References] 1. Endo, Akira. "A historical perspective on the discovery of statins." *Proceedings of the Japan Academy. Series B, Physical and biological sciences* vol. 86,5 (2010): 484-93. doi:10.2183/pjab.86.484
2. Kishida, Y et al. *Yakugaku zasshi : Journal of the Pharmaceutical Society of Japan* vol. 111,9 (1991): 469-87. doi:10.1248/yakushi1947.111.9_469

메바로친®정 5, 10, 20, 40 mg

[주성분] 프라바스타틴 나트륨 **[효능·효과]** 1. 원발성고지혈증: 고콜레스테롤혈증(IIa형), 고콜레스테롤혈증과 고트리글리세라이드혈증의 복합형(IIb형) 2. 고콜레스테롤혈증 또는 복합성고콜레스테롤혈증을 갖고 있는 환자 중 다음의 고위험군 환자에서 심근경색의 초발, 관상동맥심질환성 사망의 위험성 감소 3. 심근경색 또는 불안정성 협심증의 병력이 있는 환자에서 심근경색, 심혈관재관류술의 필요성, 허혈성 뇌졸중, 일과성 허혈발작 질환의 위험성 감소 **[용법·용량]** 치료를 시작하기 전에, 환자는 저콜레스테롤 식이를 시작해야 하고, 치료 중에도 이를 지속하여야 한다. 통상의 개시용량은 10 mg, 20 mg 혹은 40 mg 단일 용량으로 1일 1회이다. 환자의 반응에 따라 최대 40 mg까지 증량할 수 있다. **[사용상의 주의사항]** 1. 다음 환자에는 투여하지 않는다. 1) 이 약에 과민증 또는 그 병력이 있는 환자 2) 활성 간질환 또는 원인이 밝혀지지 않는 트랜스아미나제의 지속적 상승이 있는 환자 3) 임부 또는 임신하고 있을 가능성이 있는 부인, 수유부 4) 소아 5) 중증의 간·신부전 환자 6) 근병증 환자 7) 담즙울체 환자 8) HDL 콜레스테롤 상승이 동반된 hyperalphalipoproteinaemia에 의한 고콜레스테롤혈증 환자 9) 이 약은 유당을 함유하고 있으므로, 갈락토오스 불내성(galactose intolerance), Lapp 유당분해효소결핍증(Lapplactase deficiency) 또는 포도당-갈락토오스 흡수장애(glucose-galactose malabsorption)등의 유전적인 문제가 있는 환자 2. 이상반응 1) 과민증: 발진, 아나필락시스, 혈소판감소, 백혈구 감소, 용혈성 빈혈, 항핵항체(ANA) 양성, 혈액침강속도 증가, 혈관염, 루푸스양증후군, 광과민증, 혈압강하, 혈관부종, 피부근염, 소양증. 2) 소화기계: 설사, 구역, 구토, 변비, 복통, 위부불쾌감, 구내염, 가슴쓰림, 복부팽만감, 식욕부진. 3) 간장: 간기능 이상. 4) 신장: BUN, 혈청 크레아티닌치의 상승. 5) 골격근: 횡문근 융해증, 관절염, 관절통, 근육병변. 6) 정신신경계: 두통, 어지러움, 불면, 말초신경병증, 우울증, 권태감, 피로, 수면장애, 인지장애. 7) 기타: 요산상승, 혈뇨, 부종, 탈모, 발기부전 **[제조원]** HK inno.N **[개정년월일]** 2023년 3월 1일 ※ 본 정보는 요약된 일부의 정보입니다. 따라서 최신 변경 된 허가사항이나, 보다 자세한 내용은 한국다이이찌산쿄 홈페이지(www.daiichisankyo.co.kr)의 제품 설명서나 의약품안전나라(nedrug.mfds.go.kr)을 참고하시기 바랍니다.

P23-136-2025.06-V11

[판매원]
한국다이이찌산쿄주식회사
Daiichi-Sankyo
서울시 중구 을지로5길 26 미래에셋 센터원빌딩 동관 15층 Tel. 02-3453-3300

[공동판매원]
건일제약㈜
KUHNIL www.kuhnil.com
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엑토스® [제품명] 액토스정 15밀리그램(피오글리타존염산염) / 액토스정30밀리그램(피오글리타존염산염) **[효능효과]** 피오글리타존염산염 16.53mg(피오글리타존 15mg) / 피오글리타존염산염 33.06mg(피오글리타존 30mg) **[효능효과]** 이 약은 제2형 당뇨병 환자의 혈당조절을 향상시키기 위해 식사요법 및 운동요법의 보조제로 투여한다 (단독요법, 병용요법) **[용법용량]** 이 약은 식사에 관계없이 1일 1회 경구투여한다. **[사용상의 주의사항]** 1. 경고 1) 이 약을 포함한 티아졸리딘디온계 약물은 일부 환자에서 울혈성심부전을 일으키거나 악화시킬 수 있다. 2) 심부전 환자는 이 약으로의 치료를 시작해서는 안된다. 증후성 심부전 환자에서 이 약의 투여는 권장되지 않는다. 3) 이 약은 다른 티아졸리딘디온계 약물(thiazolidinediones)과 마찬가지로, 단독 또는 인슐린 등의 다른 항 당뇨병 약물과 병용 투여할 때 체액 저류를 일으킬 수 있다. 체액 저류는 심부전증을 유발하거나 악화시킬 수 있다. 4) 치료 중 육안적 혈뇨 또는 배뇨장애나 절박뇨와 같은 기타 증상의 징후가 발현되거나 증가되는 경우, 이러한 증상들은 방광암에 기인한 것일 수 있으므로, 의사에게 즉시 알리도록 환자에게 지시해야한다. 2. 다음 환자에는 투여하지 말 것. 1) 이 약 및 이 약의 구성성분에 대하여 과민반응이 알려진 환자 2)심부전 환자 또는 심부전 병력 환자 3) 활동성 방광암 환자 또는 방광암 병력 환자 4) 간장애 환자 5) 중증 신장애 환자 6) 당뇨병성 케톤산증 환자, 당뇨병성 혼수 및 전 혼수, 제1형 당뇨병 환자 7) 수술 전후, 중증 감염증 환자, 중증 외상 환자 8) 조사되지 않은 육안적 혈뇨 환자 9) 임부 또는 임신하고 있을 가능성이 있는 여성 10) 정제에서 이 약이 유당을 함유하고 있으므로, 갈락토스 불내성, Lapp 유당분해효소 결핍증 또는 글루코스-갈락토스 흡수장애 등 유전적인 문제가 있는 환자 **[저장방법]** 차광기밀용기, 습기를 피하여 25˚C에 보관(15~30˚C)

* 이 내용은 허가사항을 요약한 것으로 자세한 정보는 제품의 첨부문서 또는 http://drug.mfds.go.kr를 확인하십시오.

OT2022-15

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*Original Dapagliflozin을 의미하며, 또 다른 주 성분인 Sitagliptin에는 해당되지 않습니다.
FDC, fixed-dose combination. Reference 1. 시다프비아™정 제품설명서(개정년월일: 2023.06.30).

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KR-144301 Exp. 2025-10 (Prep. 2023-10)

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Zemidapa® Tab.

The only available† FDC of Gemigliptin + Dapagliflozin,
New treatment option for Effective glycemic control.[1-3]

[PIVOTAL STUDY]

Solution II Study
HbA1c reduction of 1.34%†† with Gemi/Dapa dual add-on to Met[3]

Solution I Study
HbA1c reduction of 0.86%†† with Zemiglo® add-on to Met/Dapa[2]

†† Change in HbA1c from baseline at week 24

Met, Metformin; **Gemi,** Gemigliptin; **Dapa,** Dapagliflozin; **HbA1c,** Glycated hemoglobin.

[References] 1. MFDS, Pharmaceutical Integrated intelligence system (https://nedrug.mfds.go.kr). 2. Lee BW et al., *Endocrinol Metab (Seoul).* 2023;38(3):328-337. 3. Data on file, Clinical Phase III trial in Korea (Pivotal Study), LG-GLCL001(2023), LG Chem.

Zemidapa® Tab. (Gemigliptin/Dapagliflozin) 50/10 mg (Product Launch Date: 2023. Apr. 08/ Full product information can be confirmed in the QR code to the right position)

■ **Therapeutic Indication** Zemidapa® is indicated as an adjunct to diet and exercise to improve glycemic control in adults with type 2 diabetes mellitus. ■ **Dosage and Administration** The recommended dose is one tablet once daily in patients who need 50 mg of gemigliptin and 10 mg of dapagliflozin. [Special population] 1) Renal impairment: The efficacy and safety of Zemidapa® is dependent on renal function, and renal function should be evaluated prior to initiation of Zemidapa® and periodically thereafter. For patients with eGFR≥45 mL/min/1.73m2, no dose adjustment is required. For patients with eGFR < 45 mL/min/1.73m2, Zemidapa® is not recommended. 2) Hepatic impairment: No dose adjustment is required for patient with mild or moderate hepatic impairment. The safety and efficacy of Zemidapa® in patients with severe hepatic impairment have not yet been established. ■ **Contraindication** Zemidapa® is contraindicated in patients with/on; 1) hypersensitivity to the active substances or to any of the excipients or a history of serious hypersensitivity reactions, ie., angioedema or anaphylaxis, to another dipeptidyl peptidase-4 (DPP-4) inhibitor or sodium glucose cotransporter (SGLT) 2 inhibitor, 2) type 1 diabetes or diabetic ketoacidosis, 3) rare hereditary problems of galactose intolerance, total lactase deficiency or glucose-galactose malabsorption, and 4) hemodialysis. ■ **Precaution** 1) Gemigliptin; patient taking with medicinal products known to cause hypoglycemia, patient with cardiac impairment, patient with severe hepatic impairment, patient with acute pancreatitis. 2) Dapagliflozin; patient with volume depletion and impaired renal function. ■ **Safety Profile** The most common adverse events reported in ≥ 1.0% in patients treated with Gemigliptin once daily in add-on combination of Metformin and Dapagliflozin in a 24 weeks study (Regardless of Investigator Assessment of Causality); Lipase increased, Chronic gastritis, Dizziness, Urinary tract infection, Cough, Diabetic nephropathy, Gastritis, Gingivitis, Large intestine polyp, Urticaria, Dyspepsia. ■ **Manufacturer** LG Chem.

Zemiglo® Tab. (Gemigliptin) 50 mg (Full product information can be confirmed in the QR code to the right position)

■ **Indication and Usage** Zemiglo® 50 mg is a dipeptidyl peptidase-4 (DPP-4) inhibitor indicated as an adjunct to diet and exercise to improve glycemic control in adults with type 2 diabetes mellitus. Zemiglo® can be administered 1) Monotherapy, 2) Combination therapy ■ **Dosage and Administration** The maximum daily recommended dose of Zemiglo® is 50 mg once daily. Zemiglo® can be taken without regard to food. When used in combination with a sulfonylurea or insulin, a lower dose of the sulfonylurea or insulin may be required to reduce the risk of hypoglycemia. No dosage adjustment is required for patients with impaired renal function. No dosage adjustment is required for patients with mild to severe hepatic impairment function. ■ **Contraindication** Zemiglo® is contraindicated in patients with 1) a history of serious hypersensitivity reactions, i.e., angioedema or anaphylaxis, to another dipeptidyl peptidase-4 (DPP-4) inhibitor or 2) type 1 diabetes or diabetic ketoacidosis. ■ **Precaution** Zemiglo® is used carefully in patients with 1) taking sulfonylurea, 2) cardiac impairment, 3) hepatic impairment, 4) acute pancreatitis, 5) hypersensitive reaction. ■ **Adverse Reactions** 1) Most common adverse reactions reported ≥ 3% of patients treated with Zemiglo® once daily in monotherapy; arthralgia, nasopharyngitis, and bacteriuria, 2) Most common adverse reactions reported in ≥ 3% of patients treated with Zemiglo® once daily in add-on combination therapy; upper respiratory tract infection, nasopharyngitis, blood amylase increased, lipase increased, and pyrexia. ■ **Manufacturer** LG Chem.

※ For more information, please refer to recently updated full prescribing information, including WARNINGS and MEDICATION GUIDE. As approved indications differ by country, consult the local prescribing information available with the manufacture before prescribing this medication.

CDA-PA-0035-1-HCP2

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전문의약품

[조성·성상] 1. 원료약품의 분량 1정 중 유효성분: 리피로우정 10 mg - 아토르바스타틴칼슘삼수화물(별규) 10.85 mg (아토르바스타틴으로서 10 mg), 리피로우정 20 mg - 아토르바스타틴칼슘삼수화물(별규) 21.70 mg (아토르바스타틴으로서 20 mg), 리피로우정 40 mg - 아토르바스타틴칼슘삼수화물(별규) 43.40 mg(아토르바스타틴으로서 40 mg), 리피로우정 80 mg - 아토르바스타틴칼슘삼수화물(별규) 86.80 mg(아토르바스타틴으로서 80 mg), **2.** 성상: 흰색의 달걀형 필름코팅정제 **[효능·효과] 1.** 다음의 심장혈관 질환에 대한 위험성 감소 **2.** 고지혈증 **3.** 식이요법에도 불구하고 여전히 아래의 기준에 해당되는 이형접합 가족형 고콜레스테롤혈증을 가진 10~17세의 소아환자(여성의 경우 초경 이후의 환자)의 총콜레스테롤, LDL-콜레스테롤, 아포-B 단백 수치를 감소시키는 식이요법의 보조제(10, 20 mg에 한함): **가.** LDL-콜레스테롤이 여전히190 mg/dL이상 (≥190 mg/dL)이거나 **나.** LDL-콜레스테롤이 여전히 160 mg/dL이상 (≥160 mg/dL)이고, 조기 심장혈관 질환의 가족력이 있는 경우 또는 해당 소아환자에서 두 가지 이상의 다른 심장혈관 질환의 위험인자가 있는 경우 **[용법·용량] 1.** 고지혈증 환자: 1일 1회 10~80 mg 범위로 투여, - 동형접합 가족형 고콜레스테롤혈증 환자: 1일 1회 10~80 mg, **2.** 이형접합 가족형 고콜레스테롤혈증 소아환자(10~17세): 권장 초회용량 1일 10 mg, 권장 최대용량 1일 20 mg(10, 20 mg에 한함)

※자세한 내용은 제품설명서를 참고하시기 바랍니다.

종근당

LPLU_CKD_202401_338(Expire:2026.02)

유한양행의 **로수바미브**는
한국인 제2형 당뇨병 환자를 대상으로 한 연구*에서
지질 프로파일 개선의 유효성과 안전성을 입증했으며,
국내에서 **연간 100만 건 이상 처방**§되고 있습니다.

* Diabetes Ther. 2020 Apr;11(4):859-871(rosuvastatin 10mg monotherapy 대비 로수바미브 10/5mg의 유효성과 안전성을 확인). § 2022년 유비스트 '로수바미브정' 처방건 수 기준

전문의약품

로수바미브정(에제티미브/로수바스타틴칼슘) 10/5mg, 10/10mg, 10/20mg [원료약품 및 분량] • 로수바미브 10/5mg : 에제티미브(USP) 10.0mg, 로수바스타틴칼슘(별규) 5.2mg(로수바스타틴으로서 5mg) • 로수바미브 10/10mg : 에제티미브(USP) 10.0mg, 로수바스타틴칼슘(별규) 10.4mg(로수바스타틴으로서 10mg) • 로수바미브 10/20mg : 에제티미브(USP) 10.0mg, 로수바스타틴칼슘(별규) 20.8mg(로수바스타틴으로서 20mg) **[성상]** • 로수바미브 10/5mg : 분홍색의 장방형 필름코팅정 • 로수바미브 10/10mg : 노란색의 장방형 필름코팅정 • 로수바미브 10/20mg : 분홍색의 달걀형 필름코팅정 **[효능 · 효과]** 원발성 고콜레스테롤혈증; 원발성 고콜레스테롤혈증(이형접합 가족형 및 비가족형) 또는 혼합형 이상지질혈증 환자의 상승된 총 콜레스테롤(totalC), LDL-콜레스테롤(LDL-C), 아포 B 단백(Apo B), 트리글리세라이드(TG) 및 non-HDL-콜레스테롤을 감소시키고, HDL-콜레스테롤(HDL-C)을 증가시키기 위한 식이요법의 보조제로서 이 약을 투여합니다. 고콜레스테롤혈증에 기인한 동맥경화성 혈관 질환의 위험성이 증가한 환자에게 지질조절약물을 투여할 때에는 많은 위험 인자를 고려해야 합니다. 지질조절약물은 적절한 식이요법(포화지방 및 콜레스테롤 제한을 포함)과 함께 사용하고, 식이요법 및 다른 비약물학적 조치에 대한 반응이 불충분한 경우에 사용해야 합니다. 이 약 투여에 앞서 이상지질혈증의 다른 이차적 원인(예를 들면, 당뇨, 갑상선기능저하증, 폐쇄성 간질환, 만성 신부전, LDL-콜레스테롤을 증가시키는 약물 및 HDL-콜레스테롤을 감소시키는 약물[progestin, anabolic steroid, 및 corticosteroid])을 확인하여야 하며, 필요한 경우 이차적 원인을 치료해야 합니다. 지질 검사시에는 총콜레스테롤, LDL-콜레스테롤, HDL-콜레스테롤 및 트리글리세라이드를 포함해야 합니다. 트리글리세라이드 수치가 400mg/dL 이상(4.5mmol/L 이상)인 경우에는 초원심분리로 LDL-콜레스테롤 농도를 측정해야 합니다. 급성 관상동맥 사고로 입원할 경우에는 입원 시 혹은 입원 후 24시간 이내에 지질을 측정해야 합니다. 환자의 퇴원 전 혹은 퇴원 시에 LDL 저하치료를 시작하는데 있어 이 측정치가 참고가 될 수 있습니다. **[용법·용량]** 이 약은 식사와 관계없이 1일 1회 투여합니다. 이 약을 투여하기 전 또는 투여 중인 환자는 반드시 표준 콜레스테롤 저하식을 지속적으로 해야 합니다. 이 약의 투여량은 환자의 LDL-콜레스테롤의 기저치, 권장되는 치료목표치 및 환자의 반응에 따라 조절되어야 합니다. 원발성 고콜레스테롤혈증: 이 약의 용량범위는 1일 10/5mg~10/20mg입니다. 초회용량으로 1일 10/5mg이 권장됩니다. LDL-콜레스테롤 감소가 더 많이 요구되는 환자의 경우 용량을 조절하여 투여할 수 있습니다. 이 약의 투여를 시작한 후 또는 용량을 적정한 후에는 4주 이상의 간격을 두고 혈중 지질 수치를 확인한 후 그에 따라 용량을 조절하며, 1일 최대 10/20mg까지 증량할 수 있습니다. 에제티미브와 로수바스타틴을 병용으로 복용하고 있는 환자인 경우, 복용의 편리함을 위하여 이 약(개개의 주성분 함량이 동일한 복합제)으로 전환할 수 있습니다. **[사용상의 주의사항]** 1. 다음 환자에는 투여하지 마십시오. 1) 이 약의 주성분 또는 구성성분에 과민반응이 있는 환자 2) 활동성 간질환 환자 또는 혈청 아미노전달효소 수치가 원인불명으로 지속적으로 높은 증상을 수반한 환자('5. 일반적 주의' 참조) 3) 근질환 환자 4) 사이클로스포린 병용투여 환자 5) 중증의 신부전의 신장애 환자(creatinine clearance (CLcr)<30mL/min) 6) 임부 또는 임신하고 있을 가능성이 있는 여성 및 수유부 ('7. 임부 및 수유부에 대한 투여' 참조) 7) 근병증/횡문근융해증에 걸리기 쉬운 환자들에게 로수바스타틴 40밀리그램 용량 투여는 금기입니다. 이러한 인자들은 아래와 같습니다. (1) 중등도의 신장애 (크레아티닌 청소율 < 60ml/min) (2) 갑상선기능저하증 (3) 유전적인 근질환 병력 또는 가족력이 있는 경우 (4) 다른 스타틴계 약물(HMG-CoA 전환효소 저해제) 또는 피브레이트 계열 약물에 대한 근육 독성의 병력이 있는 경우 (5) 알코올 중독 (6) 혈장 농도가 증가할 수 있는 상황 (7) 아시아계 환자 (8) 피브레이트 계열 약물 병용투여 8) 이 약은 유당을 함유하고 있으므로, 갈락토오스 불내성(galactose intolerance), Lapp 유당분해효소 결핍증(Lapp lactase deficiency) 또는 포도당-갈락토오스 흡수장애(glucose-galactose malabsorption) 등의 유전적인 문제가 있는 환자에게는 투여하면 안 됩니다.자세한 내용은 제품설명서 참조) **[저장방법]** 기밀용기, 실온(1~30℃) 보관 **[포장단위]** 30정(PTP), 100정(병) **[개정연월일]** 22.10.21 ※ 제품에 대한 자세한 내용은 최신의 제품설명서 또는 식약처 의약품통합정보시스템 홈페이지(https://nedrug.mfds.go.kr/)를 참조하여 주시기 바랍니다.

제조판매자
유한양행
• 본사 : 서울 동작구 노량진로 74 • 공장 : 충청북도 청주시 청원구 오창읍 연구단지로 219
• 홈페이지 : www.yuhan.co.kr • 소비자상담실 : 080-024-1188 (수신자 요금부담)

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ROS-II-2209-01 HM·Design 2209_01

Reference.
Kim BK, Hong SJ, Lee YJ, *et al.* Long-term efficacy and safety of moderate-intensity statin with ezetimibe combination therapy versus high-intensity statin monotherapy in patients with atherosclerotic cardiovascular disease (RACING): a randomised, open-label, non-inferiority trial. *Lancet.* 2022 Jul 30;400(10349):380-390.

JW 중외제약

효과를 높여보세요

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- 32개국 당뇨병 안전성 공인 [3)]

1), 2) 리바로젯 3상 허가임상 결과 3) 32개 국가 현황 • 유럽(13개국): 영국, 독일, 프랑스, 스페인, 이탈리아, 핀란드, 네덜란드, 스웨덴, 오스트리아, 아일랜드, 포르투갈, 그리스, 노르웨이 • 동유럽(5개국): 러시아, 폴란드, 우크라이나, 조지아, 아르메니아 • 동아시아(5개국): 싱가포르, 대만, 인도네시아, 말레이시아, 카자흐스탄 • 중동(8개국): 사우디아라비아, UAE, 쿠웨이트, 카타르, 요르단, 오만, 레바논, 바레인 • 아프리카(1개국): 모로코