https://sola.or.kr/

SoLA 2026

한국지질·동맥경화학회 춘계학술대회

2026 Spring Congress on Lipid and Atherosclerosis of KSoLA

From Lipids to Life, From Evidence to Action

2026. 4. 3. (금)-4. (토)

시그니엘 부산

리바로하이® 정
(Pitavastatin/Valsartan/Amlodipine)

JW 중외제약 JW 신약

Unlock the power of triple therapy

- **당뇨병 위험 징후 없이** 고혈압, 이상지질혈증 동시 관리[1-4]
- 국내 3상 임상 **혈압 · 지질 동시 개선**[5]
- **6가지 용량** 환자 맞춤형 치료 옵션 제공[3]

리바로하이® 정
(Pitavastatin/Valsartan/Amlodipine)

국내 유일* Pitavastatin 3제 복합제, 당뇨병 위험 징후 없이 혈압 · 지질 동시 개선하는 최적의 Triple Therapy[1-6]

*As of 2025.08.12

References 1. Seo WW, et al. *Cardiovasc Diabetol.* 2022;21(1):82. **2.** NAVIGATOR Study Group. *N Engl J Med.* 2010;362(16):1477-1490. **3.** 의약품안전나라 의약품통합정보시스템 허가사항(리바로하이정2/160/10mg, 리바로하이정2/160/5mg, 리바로하이정2/80/5mg, 리바로하이정4/160/10mg, 리바로하이정4/160/5mg, 리바로하이정4/80/5mg) (Accessed on Sep. 30, 2025). **4.** SmPC of UK. **5.** 리바로하이 3상 임상시험. JW Pharmaceutical Data on file. **6.** 의약품안전나라 의약품통합정보시스템 허가사항(피타바스타틴)(Accessed on Aug. 18, 2025).

* 보다 상세한 제품 정보는 제품설명서를 참고하시기 바랍니다.

JWP393-2511002(EXP.2711)

경기도 과천시 과천대로 7길 38 (갈현동) JW과천사옥, CCM 운영팀: 1588-2675
제품에 대한 상세한 내용은 제품 설명서나 본사 홈페이지를 참조하시기 바랍니다.
www.jw-pharma.co.kr

리바로하이정 2/80/5밀리그램 (피타바스타틴칼슘수화물, 발사르탄, 암로디핀베실산염)

리바로하이정 2/160/5밀리그램 (피타바스타틴칼슘수화물, 발사르탄, 암로디핀베실산염)

리바로하이정 2/160/10밀리그램 (피타바스타틴칼슘수화물, 발사르탄, 암로디핀베실산염)

리바로하이정 4/80/5밀리그램 (피타바스타틴칼슘수화물, 발사르탄, 암로디핀베실산염)

리바로하이정 4/160/5밀리그램 (피타바스타틴칼슘수화물, 발사르탄, 암로디핀베실산염)

리바로하이정 4/160/10밀리그램 (피타바스타틴칼슘수화물, 발사르탄, 암로디핀베실산염)

이상지질혈증 & 고혈압 통합 관리를 위한
Excellent Package Solution

로수젯정
(에제티미브/로수바스타틴)

- **세계 최초** Rosuvastatin + Ezetimibe 복합제의 **CV outcome 발표**[1]
- **국내사 개발** 전문의약품 **최초 2년 연속 원외처방조제액 전체 1위**[2]
- 국내 환자 대상 임상 연구 결과 **20편, SCIE급 국제 학술지 등재** (2025년 기준)
- **한미약품 R&D** 및 **자체 생산**을 통한 Global 진출

10 / 2.5 mg

10 / 5 mg

10 / 10 mg

10 / 20 mg

다양한 이상지질혈증 환자의 맞춤 처방, 로수젯으로 시작하세요!

활성형 Fenofibric acid, 고중성지방혈증 치료제
페노시드® 캡슐
(페노피브릭산)

오메가-3 성분의 고중성지방혈증 치료제
한미 오메가 연질캡슐 1000mg
(오메가-3-산에틸에스테르90)

Statin과 병용 가능한 이상지질혈증 치료제
에제트® 정 10mg
(에제티미브)

아모프렐정
(암로디핀/로사르탄/클로르탈리돈)

(1.67/16.67/4.17mg)

- **세계 최초 초저용량* 3제 항고혈압제**
- **본태성 고혈압 치료 개량신약**
- **국내 환자 대상 3상 임상시험 결과 2건 JACC 등재**[3]
  - 본태성 고혈압 환자에서 Losartan 단독요법 대비 우수한 혈압 강하 효과 입증
  - 국내 환자에서 유효성과 안전성 입증
- **국내 환자 대상 2상 임상시험 결과 2건 SCIE급 국제 학술지 등재**[4),5)]
- **저용량 복합제로 용량 의존적인 이상반응 최소화 기대**[6)]

다양한 원인의 **본태성 고혈압 환자**, **아모프렐**과 함께하세요!

세계 최초 Amlodipine camsylate + Losartan K 복합제
아모잘탄® 정
(암로디핀/로사르탄)

세계 최초 CCB/ARB/Chlorthalidone 3제 복합제
아모잘탄 플러스® 정
(암로디핀/로사르탄/클로르탈리돈)

세계 최초 CCB/ARB/Rosuvastatin 3제 복합제
아모잘탄 큐® 정
(암로디핀/로사르탄/로수바스타틴)

세계 최초 CCB/ARB/Rosuvastatin/Ezetimibe 4제 복합제
아모잘탄 엑스큐® 정
(암로디핀/로사르탄/로수바스타틴/에제티미브)

Hanmi 한미약품

*1/3 용량

CV, cardiovascular; **SCIE,** science citation index expanded; **R&D,** research and development; **CCB,** calcium channel blocker; **ARB,** angiotensin Ⅱ receptor blocker

Ref. 1) Kim BK, et a. *Lancet.* 2022 Jul;400(10349):380-390. **2)** UBIST D1 Sales data. 2024년, 2025년 원외처방조제액 기준. **3)** Sung KC, et al. *J Am Coll Cardiol.* 2026 Feb 3:S0735-1097(25)10559-7. [Epub ahead of print] **4)** Hong SJ, et al. *Drug Des Devel Ther.* 2020 Dec 31;14:5735-5746. Erratum in: *Drug Des Devel Ther.* 2021 Apr 07;15:1477. **5)** Sung KC, et al. *J Clin Hypertens (Greenwich).* 2023 May;25(5):429-439.. **6)** Law MR, et al. *BMJ.* 2003 Jun;326(7404):1427.

CVD-II-2603-01
HM-Design 2511_26

DAEWOONG

보건의료전문가용

LDL-C 목표 달성을 위한[1-6] 다양한 치료 옵션

로수바스타틴(크레젯)[1,2], 피타바스타틴(바로에젯)[3], 아토르바스타틴(리토바젯)[4,5]
3가지 에제티미브/스타틴 복합제[6]로 완성되는 대웅제약의 이상지질혈증 치료 포트폴리오

CREZET Tab.
크레젯정[Ezetimibe / Rosuvastatin] 10/2.5, 10/5, 10/10, 10/20mg

Baroezet® Tab.
바로에젯정 Pitavastatin 1 mg / Ezetimibe 10 mg

Litorvazet® Tab
리토바젯정[Ezetimibe / Atorvastatin]
10/5, 10/10, 10/20, 10/40mg

Ref.> 1. Yang YJ, et al. *Clin Ther.* 2017;39(1):107-117. 2. Data on file. 허가용 임상 3상 연구[DW_DWJ1507301]. 3. Data on file. 허가용 임상 3상 연구[AD-228P3]. 4. Bays HE, et al. *Am J Cardiol.* 2013;112:1885–1895. 5. Leiter LA, et al. *Am J Cardiol.* 2008;102(11):1495–1501. 6. 각 제품별 식약처허가사항. (2026.03.13 확인)

※ 자세한 제품 정보는 제품 설명서 및 QR코드를 참고하시기 바랍니다. ※ 본 의약품은 엄격한 품질관리를 필한 제품입니다. 만약 구입시 사용기한 또는 유효기간이 지났거나 변질, 변패, 오염되었거나 손상된 의약품은 공정거래위원회 고시(소비자 분쟁해결기준)에 의거, 구입한 약국 및 의약품판매업자를 통해 교환 또는 환불받을 수 있습니다. ※ 부작용 피해구제 신청은 한국의약품안전관리원에 할 수 있습니다. [신청방법] 한국의약품안전관리원: 1644-6223, 14-3330 (karp.drugsafe.or.kr) 또는 대웅제약 소비자 센터(수신자 부담전화): 080-550-8308~9 (www.daewoong.co.kr) [신청대상] 의약품 부작용으로 사망, 장애, 질병 피해를 입은 환자 및 유족 [보상범위] 사망일시보상금·장례비, 장애일시보상금, 진료비

CREZET 10/5 BAROEZET 1/10 LITORVAZET 10/10

2026-111
CO.CTZT.BRZT.LTZT.260319.M

PROGOR® ER Capsule

Diltiazem Hydrochloride 120mg 180mg

Extended Release
Know-how Originality

Useful Prevention for HTN & Angina

1 Capsule Daily

푸로골®서방캡슐은 특허기술로 24시간 안정적 관상동맥 관류 유지 및 혈압관리

- ✓ Once Daily 복용으로 환자들의 Compliance 증대
- ✓ 음식과 함께 섭취시에도 Dumping이 없음
- ✓ 약물복용후 초기에 혈중 Peak가 없으며 24시간 최적의 관상동맥 관류 유지

[제품명] 푸로골서방캡슐 **[원료약품 및 그 분량]** 1캡슐 중 · 푸로골서방캡슐120밀리그램: 딜티아젬염산염(EP) 120mg/푸로골서방캡슐180밀리그램: 딜티아젬염산염(EP) 180mg **[성상]** 흰색 내지 거의 흰색의 펠렛이 든 상하부 흰색의 불투명 경질캡슐제 **[효능 · 효과]** 협심증, 본태성고혈압(경증-중등도) **[용법 · 용량]** 1. 성인: 초회량으로 딜티아젬염산염으로서 1일 1회 180㎎을 경구투여한다. 용량은 증상에 따라 2-4주 간격으로 증량할 수 있으며, 통상적인 유지용량은 1일 1회 240-360mg 이다. 1일 최대투여량은 360mg 이다. 2. 노인 및 간·신장애 환자: 초회량으로 1일 1회 120㎎을 투여한다. 투여 중에 심박동수를 측정하여 50회 이하로 저하된 경우에는 증량하지 않는다. **[저장방법]** 기밀용기, 실온보관(1~25℃) **[포장단위]** 28 캡슐/상자(14캡슐/PTP포장 x 2), 56 캡슐/상자(14캡슐/PTP포장 x 4)

[제조원] SMB [수입 및 판매원] 한국유니팜 UNIPHARM KOREA

https://sola.or.kr/

SoLA 2026

한국지질·동맥경화학회 춘계학술대회

2026 Spring Congress on Lipid and Atherosclerosis of KSoLA

From Lipids to Life, From Evidence to Action

2026. 4. 3. (금)-4. (토)

시그니엘 부산

조직위원회

◑ 한국지질·동맥경화학회 임원진

직 책	성 함	소 속
2026 회장	김성래	가톨릭의대 내분비내과
2026 부회장	박영미	이화의대 분자의과학교실
	한성림	서울대 식품영양학과
이사장	김상현	서울의대 순환기내과
총무이사	박재형	고려의대 순환기내과
부 총 무	강선미	순천향의대 내분비대사내과
	강지훈	서울의대 순환기내과
	권오성	가톨릭의대 순환기내과
	김주현	울산의대 심장내과
	배재현	한림의대 내분비내과
	장영우	가천의대 심장내과
	조윤경	울산의대 내분비내과
재무이사	최성훈	한림의대 순환기내과
기획이사	김병진	성균관의대 순환기내과
기획간사	이장훈	경북의대 순환기내과
학술이사	김학령	서울의대 순환기내과
학술간사	김원진	차의대 내분비내과
	김현진	한양의대 심장내과
	양예슬	서울의대 내분비대사내과
	정재훈	동국의대 심장내과
간행이사	정인경	경희의대 내분비대사내과
간행간사	허지혜	한림의대 내분비내과
홍보이사	문민경	서울의대 내분비대사내과
홍보간사	노 은	서울의대 내분비대사내과
대회협력이사	홍순준	고려의대 순환기내과
대외협력간사	차정준	고려의대 순환기내과
국내교류이사	서미혜	순천향의대 내분비대사내과
국내교류간사	한유진	계명의대 내분비대사내과
보험법제이사	이상엽	중앙의대 순환기내과
보험법제간사	김희동	순천향의대 심장내과
의료정보이사	박상민	을지의대 심장내과
의료정보간사	김민정	이화의대 순환기내과

윤리이사	이상학	연세의대 심장내과
윤리간사	원호연	중앙의대 순환기내과
사회공헌이사	손정우	연세원주의대 심장내과
사회공헌간사	정혜문	경희의대 심장내과
교육이사	홍준화	을지의대 내분비내과
교육간사	김병식	한양의대 심장내과
	류영상	조선의대 내분비대사내과
진료지침이사	김현진	한양의대 심장내과
진료지침간사	장영우	가천의대 심장내과
임상연구이사	윤종찬	가톨릭의대 순환기내과
임상연구간사	천대영	한림의대 순환기내과
기초연구이사	김형규	인제의대 생리학교실
기초연구간사	남궁준	연세원주의대 생화학교실
식품영양이사	신민정	고려대 바이오시스템의과학부
식품영양간사	임현정	경희대 의학영양학과
특임(DAWAS)이사	이은정	성균관의대 내분비내과
특임(DAWAS)간사	정창희	울산의대 내분비내과
무임소이사	강 현	중앙의대 마취통증의학과
	권혁상	가톨릭의대 내분비내과
	노정현	인제의대 내분비내과
	박성하	연세의대 심장내과
	박형규	순천향의대 내분비대사내과
	송기호	건국의대 내분비대사내과
	안성균	연세원주의대 심장내과
	안지현	한국의학연구소 내과
	이상록	전북의대 심장내과
	이승환	가톨릭의대 내분비내과
	이왕수	중앙의대 순환기내과
	이준희	한림의대 심장혈관내과
	조상호	한림의대 순환기내과
	최성희	서울의대 내분비대사내과
	한정규	서울의대 순환기내과
	홍영준	전남의대 순환기내과
감사	박철영	성균관의대 내분비내과
	이우제	울산의대 내분비내과

조직위원회

◑ 한국지질·동맥경화학회 학술위원회

직 책	성 명	소 속
학술이사	김학령	서울의대 순환기내과
학술간사	김원진	차의대 내분비내과
	김현진	한양의대 심장내과
	양예슬	서울의대 내분비대사내과
	정재훈	동국의대 심장내과
학술위원	강현주	경북대 식품공학부
	구유정	서울의대 내분비대사내과
	김경진	인하의대 의생명학교실
	김규호	가톨릭의대 내분비내과
	김범준	울산의대 신경과
	김정민	서울의대 신경과
	김혜미	중앙의대 순환기내과
	남궁준	연세원주의대 생화학교실
	류영상	조선의대 내분비대사내과
	박다현	고려대 보건과학대학
	박용현	부산의대 순환기내과
	박현웅	충남의대 심장내과
	배성아	연세의대 심장내과
	배재현	서울의대 내분비대사내과
	서미혜	순천향의대 내분비대사내과
	양여리	가톨릭의대 내분비내과
	오진경	충남의대 심장내과
	우종신	경희의대 심장내과
	유지희	중앙의대 내분비내과

학술위원	이민경	한양의대 내분비내과
	이승헌	전남의대 순환기내과
	이호규	연세의대 예방의학교실
	장정윤	경상의대 순환기내과
	전기현	서울의대 순환기내과
	전재한	경북의대 내분비대사내과
	정미향	가톨릭의대 순환기내과
	정수명	성균관대 생명과학과
	조가람	한남대 식품영양학과
	조익성	연세의대 심장내과
	조현승	서울의대 순환기내과
	차정준	고려의대 순환기내과
	최종한	건국의대 내분비대사내과
	최훈지	한림의대 내분비내과
	홍준화	을지의대 내분비내과
자문위원	김상현	서울의대 순환기내과
	김현창	연세의대 예방의학교실
	김형규	인제의대 생리학교실
	김오연	동아대 식품영양학과
	박용식	경희의대 미생물학교실
	박철영	성균관의대 내분비내과
	신민정	고려대 바이오시스템의과학부
	양한모	서울의대 순환기내과
	윤종찬	가톨릭의대 순환기내과
	이상학	연세의대 심장내과
	이우제	울산의대 내분비내과
	이은정	성균관의대 내분비내과
	정인경	경희의대 내분비대사내과
	최경묵	고려의대 내분비내과

Program at a Glance

Day 1 | 2026년 4월 3일(금)

	Room 1	Room 2	Room 3	Room 4
11:00–	Registration			
11:55–12:00	Opening Address (Room 2)			
12:00–13:20	**Symposium 1** *Metabolic Regulation and Atherosclerosis: Emerging Therapeutic Targets*	**Symposium 2** *Translational Nutrition: From Molecular Pathways to Clinical Practice in Cardiometabolic Diseases*	**Symposium 3** *Clinical Implication of Lipoprotein(a)*	**Oral Presentation 1**
13:20–13:50	Break			
13:50–15:20	**Symposium 4** *Beyond LDL: Targeting Triglyceride-rich Lipoproteins for Residual Cardiovascular Risk*	**Symposium 5** *CVD Risk Prediction in the Context of Clinical Practice Guidance*	**Symposium 6** *Mis- or Disinformation of Statin Therapy in Real World Cardiometabolic Area*	**Symposium 7** *해외 학회 참관기*
15:20–16:50	**Symposium 8** *New Guidelines and New Drugs*	**Symposium 9** *HDL-C: The Higher, the Better? —Revisiting the Myth of Good Cholesterol*	**Symposium 10** **KSS-KSoLA Joint Symposium** *Inflammation and Lipid Crosstalk in Carotid Atherosclerosis*	**Symposium 11 (15:20–16:20)** *AI 시대의 학술 출판 혁신*
17:00–17:50	**Mini-Oral Presentation 1** * 진행 장소: 시그니엘 부산 4층 포이어 (로비) 내 발표구역 A-D			
18:00–19:00	Welcome Reception			

Day 2 | 2026년 4월 4일(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** *교육위원회 세션*
08:30–10:00	**Symposium 12** *Current and Future Perspectives on Lipid-lowering Agents*	**Symposium 13** *AI & Digital Health Technology for Atherosclerosis*	**Symposium 14** **KSCMS-KSoLA Joint Symposium** *Incretin Therapies and Circadian Biology for Cardiometabolic Protection*	**Oral Presentation 2**
10:00–10:10	Break			
10:10–10:40	**Plenary Lecture 1 [ENG]** *Advancing genomic medicine for lipid disorders and atherosclerosis*			
10:50–12:00	**Mini-Oral Presentation 2** * 진행 장소: 시그니엘 부산 4층 포이어 (로비) 내 발표구역 A-D			
12:00–13:00	**Luncheon Symposium 1**	**Luncheon Symposium 2**	**Luncheon Symposium 3**	
13:10–14:40	**Symposium 15** *Updates in the 2026 Korean Dyslipidemia Guidelines: Integrating Global Evidence*	**Symposium 16** *Primary Prevention of Cardiovascular Disease*	**Symposium 17** **KOVAS-KSoLA Joint Symposium** *Unraveling the Pathway of Atherosclerosis through Imaging and Lipid Assessment*	**Symposium 18 (13:40–14:40)** *Dyslipidemia Fact Sheet 2026*
14:40–14:50	Break			
14:50–15:20	**Plenary Lecture 2** *Molecular integration of lipid metabolism and inflammation in cardiovascular disease*			
15:30–17:00	**Symposium 19** *Innovative Technologies and Disease Modeling for Lipotoxicity and Atherosclerosis*	**Symposium 20** *Nutritional Perspectives on Sex Differences in Cardiometabolic Health*	**Symposium 21** **KSIC-KSoLA Joint Symposium** *Targeting Atherosclerotic Vulnerability: Lipoprotein Modulation and Inflammation*	**Oral Presentation 3**
17:00–	Closing Ceremony			

행사 개요

◑ **행사명**
- 국문: SoLA 2026 한국지질·동맥경화학회 춘계학술대회
- 영문: 2026 Spring Congress on Lipid and Atherosclerosis of KSoLA

◑ **일자:** 2026년 4월 3일(금)–4일(토)

◑ **장소:** 시그니엘 부산

◑ **주최/주관:** 한국지질·동맥경화학회

◑ **개최방식:** 오프라인 학술대회

◑ **웹사이트:** https://sola.or.kr/

◑ **평점**
- 대한의사협회 연수교육 평점: 4월 3일(금) 최대 3평점 / 4일(토) 최대 6평점
- 한국영양교육평가원 임상영양사 전문연수교육 (CPD) 최대 5평점

평점 안내

구분		4월 3일(금)	4월 4일(토)
의사	대한의사협회 연수평점	최대 3평점	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

번호	회사명
1	종근당
2	퍼슨헬스케어
3	GC녹십자
4	JLA 부스
5	HK이노엔
6	
7	LG화학
8	셀트리온제약
9	JW중외제약
10	
11	유한양행
12	SK케미칼
13	
14	한국다이이찌산쿄
15	대웅제약
16	대원제약
17	한미약품
18	

번호	회사명
19	한국오츠카제약
20	한국유나이티드제약
21	보령
22	
23	한림제약
24	비씨월드제약
25	신풍제약
26	유영제약
27	동아ST
28	
29	일동제약
30	안국약품
31	
32	한국에자이
33	명문제약
34	건일제약
35	대웅바이오
36	한국팜비오

SoLA 이벤트 안내

첫번째 이벤트! 스탬프 투어 이벤트

SoLA 2026 행사장 로비에 설치된 전시부스를 방문하시고 스탬프를 80% 이상 적립하시면 스타벅스 E-기프트 카드를 증정해드립니다!

※ 스타벅스 E-기프트 카드는 사전등록 시 제출하신 휴대폰번호로 문자 발송하여 드립니다.
응모 전 휴대폰번호가 올바르게 기입되었는지 꼭 확인 부탁드립니다.

[참여방법]

① 등록 시 받으신 스탬프투어 이벤트 용지를 확인해주세요.
② 전시부스에 방문하여 스탬프를 적립해주세요. 80% 이상 적립!
③ 등록데스크 부근에 위치한 스탬프투어 이벤트 데스크를 방문해주세요.
④ 명찰 내 QR코드를 리더기에 태그하여 주시면 학술대회 종료 후 스타벅스 E-기프트 카드를 보내드립니다.

두번째 이벤트! 럭키 드로우 이벤트

조찬 심포지엄, 폐회식에서 경품 추첨이 진행됩니다!

※ 모든 경품은 중복 및 대리 수령이 불가합니다.

조찬 심포지엄 | 4월 4일(토) 07:30-08:00 | Room 1-3

1등 (1명)
갤럭시 워치 8

2등 (1명)
갤럭시 버즈 3 프로

조찬 심포지엄 | 4월 4일(토) 07:30-08:00 | Room 4

1등 (1명)
스타벅스 5만원권

2등 (1명)
스타벅스 3만원권

폐회식 | 4월 4일(토) 17:00 | Room 1,2,3

1등 (1명)
LG전자 그램 프로 16

2등 (1명)
다이슨 에어랩
멀티 스타일러 앤 드라이어

3등 (2명)
에어팟 프로 3

4등 (5명)
스타벅스 5만원권

[참여방법]

① 조찬 심포지엄, 폐회식에 참여해주세요.
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 2026 행사장 어디서든 같은 소속 연구실, 동료분들과 사진을 촬영해주세요!
가장 많은 인원이 촬영된 사진을 제출하시면 스타벅스 E-기프트 카드를 증정해드립니다!

[참여방법]

① 행사장에서 동료분들과 단체사진을 촬영해 주세요.
② 우측 QR코드로 포토제닉 이벤트 참여 링크에 접속해주세요.
③ 이벤트 참여 양식을 작성해주세요.
④ 학술대회 종료 후 공지사항을 통해 선정 결과가 공지됩니다.

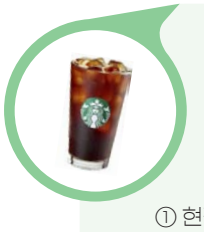
네번째 이벤트! 학회 참관기 이벤트

SoLA 2026 참석에 대한 소감을 자유롭게 작성하고, 사진과 함께 보내주세요!
선정되신 분께는 백화점 상품권을 증정해드립니다!

[참여방법]

① 워드/한글에 자유로운 형식으로 SoLA 2026 참석에 대한 후기, 소감 등을 적어주세요. (1,000자 이내)
② SoLA 2026 행사장에서 촬영한 본인의 사진을 함께 저장해주세요.
③ SoLA 2026 준비사무국 이메일(lipid@planbear.co.kr)로 학회 참관기를 제출해주세요.
④ 학술대회 종료 후 공지사항을 통해 선정 결과가 공지됩니다.

다섯번째 이벤트! 설문조사 참여 이벤트



SoLA 2026 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 4일(토) 10:10–10:40 | Room 1,2,3 (그랜드볼룸 1,2,3)

좌장 : 김성래(가톨릭의대 내분비내과)

10:10–10:40	**Advancing genomic medicine for lipid disorders and atherosclerosis** **Daniel J. Rader** (University of Pennsylvania, USA)	/ 38

Plenary Lecture 2

4월 4일(토) 14:50–15:20 | Room 1,2,3 (그랜드볼룸 1,2,3)

좌장 : 김상현(서울의대 순환기내과)

14:50–15:20	**Molecular integration of lipid metabolism and inflammation in cardiovascular disease** **임승순**(계명의대 생리학교실)	/ 42

Symposium 1

4월 3일(금) 12:00–13:20 | Room 1 (그랜드볼룸 3)

Metabolic Regulation and Atherosclerosis: Emerging Therapeutic Targets

좌장 : 김치대(부산의대 약리학교실), 한진(인제의대 생리학교실)

패널 : 김정한(가톨릭의대 생화학교실), 김형규(인제의대 생리학교실),
남궁준(연세원주의대 생화학교실), 류홍열(경북대 생명공학과)

12:00–12:20	**Redox-dependent regulation of a protein controls lipid peroxidation at the plasma membrane and ferroptosis** **이은우**(한국생명공학연구원 대사제어연구센터)	/ 46
12:20–12:40	**Next generation NAD^+ therapeutics via new modalities** **류동렬**(GIST 의생명공학과)	/ 48
12:40–13:00	**Development of cardiovascular therapeutics through regulation of endothelial dysfunction** **장우철**(부산대 생물교육과)	/ 50
13:00–13:20	**Panel Discussion**	

Symposium 2

4월 3일(금) 12:00–13:20 | Room 2 (그랜드볼룸 2)

Translational Nutrition: From Molecular Pathways to Clinical Practice in Cardiometabolic Diseases

좌장 : 한성림(서울대 식품영양학과), 김정선(국립암센터 암의생명과학과)

패널 : 김오연(동아대 식품영양학과), 박동현(세종대 데이터사이언스학과), 박신옥(눔코리아), 임현정(경희대 의학영양학과)

12:00–12:20	Nutritional modulation of metabolic enzymes and epigenetic approaches for cardiometabolic health **강현주**(경북대 식품공학부)	/ 54
12:20–12:40	Predicting drug–food interactions using AI **김모건**(한국외대 바이오메디컬공학부)	/ 56
12:40–13:00	Shared metabolic switches in obesity treatment: from intermittent fasting to GLP1 based therapy **신민정**(고려대 바이오시스템의과학부)	/ 58
13:00–13:20	Panel Discussion	

Symposium 3

4월 3일(금) 12:00–13:20 | Room 3 (그랜드볼룸 1)

Clinical Implication of Lipoprotein(a)

좌장 : 한기훈(울산의대 심장내과), 박성하(연세의대 심장내과)

패널 : 김웅(영남의대 심장내과), 김지현(가톨릭의대 순환기내과), 박세은(성균관의대 내분비내과)

12:00–12:16	Decoding lipoprotein(a): genetics and metabolism insights **이장훈**(경북의대 순환기내과)	/ 62
12:16–12:32	Lipoprotein(a) epidemiology: insights from Western and Korean populations **장영우**(가천의대 심장내과)	/ 64
12:32–12:48	Unveiling the clinical impact of lipoprotein(a) **주형준**(고려의대 순환기내과)	/ 66
12:48–13:04	Lipoprotein(a) as the next frontier in cardiovascular therapy **위진**(가천의대 심장내과)	/ 68
13:04–13:20	Panel Discussion	

Symposium 4

4월 3일(금) 13:50-15:20 | Room 1 (그랜드볼룸 3)

Beyond LDL: Targeting Triglyceride-rich Lipoproteins for Residual Cardiovascular Risk

좌장 : 김성래(가톨릭의대 내분비내과), 이우제(울산의대 내분비내과)

패널 : 김지윤(성균관의대 내분비대사내과), 류영상(조선의대 내분비대사내과), 박용현(부산의대 순환기내과)

13:50-14:08	TG-rich lipoproteins and residual cardiovascular risk: genetic and mechanistic insights **김병진**(성균관의대 순환기내과)	/ 72
14:08-14:26	Omega-3 fatty acids: from triglyceride reduction to cardiovascular outcomes **임수**(서울의대 내분비대사내과)	/ 74
14:26-14:44	APOC3 metabolism and therapeutic approaches **김남훈**(고려의대 내분비내과)	/ 76
14:44-15:02	ANGPTL3/4/8 inhibition: translational insights and clinical development **정혜문**(경희의대 심장내과)	/ 78
15:02-15:20	Panel Discussion	

Symposium 5

4월 3일(금) 13:50-15:20 | Room 2 (그랜드볼룸 2)

CVD Risk Prediction in the Context of Clinical Practice Guidance

좌장 : 편욱범(이화의대 순환기내과), 김현창(연세의대 예방의학교실)

패널 : 강선미(순천향의대 내분비대사내과), 박광열(중앙의대 신경과), 박상우(울산의대 심장내과)

13:50-14:08	CVD risk prediction in lipid guidelines **김은지**(가천의대 예방의학과)	/ 82
14:08-14:26	CVD risk prediction in diabetes guidelines **구유정**(서울의대 내분비대사내과)	/ 84
14:26-14:44	CVD risk prediction in hypertension guidelines **천대영**(한림의대 순환기내과)	/ 86
14:44-15:02	CVD risk prediction in the context of CKM health **이호규**(연세의대 예방의학과)	/ 88
15:02-15:20	Panel Discussion	

Symposium 6

4월 3일(금) 13:50-15:20 | Room 3 (그랜드볼룸 1)

Mis- or Disinformation of Statin Therapy in Real World Cardiometabolic Area

좌장 : 이준희(한림의대 심장혈관내과), 이상록(전북의대 심장내과)

패널 : 노은(서울의대 내분비대사내과), 박성민(동아일보),
안지현(한국의학연구소 내과), 이재광(창원파티마병원 심장내과)

13:50-14:08	**이상지질혈증 치료 개시 및 유지의 어려움** **김민정**(이화의대 순환기내과)	/ 92
14:08-14:26	**이상지질혈증과 동맥경화치료에 있어서 주요 가짜 정보의 유형 (매체에서 보이는 유형 정리)** **김대영**(인하의대 심장내과)	/ 94
14:26-14:44	**가짜 정보에 의한 환자와 의료진의 피해 현황 (증례와 의료정보위원회 설문조사)** **이봄**(차의대 심장내과)	/ 96
14:44-15:02	**이상지질혈증과 동맥경화 치료 관련 가짜 정보 등에 대한 학회 차원의 대응 대책** **박상민**(을지의대 심장내과)	/ 98
15:02-15:20	**Panel Discussion**	

Symposium 7

4월 3일(금) 13:50-15:20 | Room 4 (볼룸 1,2)

해외 학회 참관기

좌장 : 홍영준(전남의대 순환기내과), 홍순준(고려의대 순환기내과)

패널 : 김민관(연세의대 심장내과), 김봉준(고신의대 순환기내과), 임영효(한양의대 심장내과)

13:50-14:08	**CLVS 2025** **박상민**(을지의대 심장내과)	/ 102
14:08-14:26	**VAS 2025** **김학령**(서울의대 순환기내과)	/ 104
14:26-14:44	**APSAVD 2025** **조상호**(한림의대 순환기내과)	/ 106
14:44-15:02	**AAS 2025** **최훈지**(한림의대 내분비내과)	/ 108
15:02-15:20	**Panel Discussion**	

Symposium 8

4월 3일(금) 15:20-16:50 | Room 1 (그랜드볼룸 3)

New Guidelines and New Drugs

좌장 : 안영근(전남의대 순환기내과), 김철식(연세의대 내분비내과)

패널 : 김재석(연세원주의대 신장내과), 박정환(한양의대 내분비내과), 임홍석(아주의대 순환기내과)

15:20-15:38	Overview of 2025-2026 dyslipidemia guidelines in Europe and US **윤종찬**(가톨릭의대 순환기내과)	/ 112
15:38-15:56	Early and intensive lipid-Lowering after acute coronary syndrome: timing, targets, and real-world challenges **임수빈**(이화의대 순환기내과)	/ 114
15:56-16:14	Managing residual atherosclerotic risk: beyond LDL-C toward precision lipid therapy **이수연**(단국의대 심장혈관내과)	/ 116
16:14-16:32	New and emerging lipid-lowering therapies: what's next **김상현**(서울의대 순환기내과)	/ 118
16:32-16:50	Panel Discussion	

Symposium 9

4월 3일(금) 15:20-16:50 | Room 2 (그랜드볼룸 2)

HDL-C: The Higher, the Better? — Revisiting the Myth of Good Cholesterol

좌장 : 홍은경(한림의대 내분비내과), 박재형(고려의대 순환기내과)

패널 : 유지희(중앙의대 내분비내과), 이희선(서울의대 순환기내과), 진흥용(전북의대 내분비내과)

15:20-15:40	HDL-C in the guidelines: where do we stand today? **정재훈**(동국의대 심장내과)	/ 122
15:40-16:00	HDL-C is cardioprotective – higher HDL-C reduces ASCVD risk **손정우**(연세원주의대 심장내과)	/ 124
16:00-16:20	Extremely high HDL-C may be harmful – function matters more than quantity **양예슬**(서울의대 내분비대사내과)	/ 126
16:20-16:50	Panel Discussion	

Symposium 10 – KSS-KSoLA Joint Symposium

4월 3일(금) 15:20-16:50 | Room 3 (그랜드볼룸 1)

Inflammation and Lipid Crosstalk in Carotid Atherosclerosis

좌장 : 김응규(인제의대 신경과), 서우근(성균관의대 신경과)

패널 : 권형민(서울의대 신경과), 우호걸(경희의대 신경과), 장정윤(경상의대 순환기내과), 조윤경(울산의대 내분비내과)

15:20-15:40	Evaluating and treating atheroinflammation: translating vascular inflammation into clinical practice **김정민**(서울의대 신경과)	/ 130
15:40-16:00	Carotid and cerebral hemodynamics: insights into endothelial dysfunction **정슬기**(메디이미지)	/ 132
16:00-16:20	Systemic atherosclerosis and lipid control: role of carotid IMT and plaque **김범준**(울산의대 신경과)	/ 134
16:20-16:50	Panel Discussion	

Symposium 11

4월 3일(금) 15:20-16:20 | Room 4 (볼룸 1,2)

AI 시대의 학술 출판 혁신

좌장 : 이우제(울산의대 내분비내과), 이상엽(중앙의대 순환기내과)

패널 : 김현진(한양의대 심장내과), 안효석(가톨릭의대 순환기내과), 허지혜(한림의대 내분비내과)

15:20-15:40	AI 도구를 활용한 고품질 Peer Review 전략과 Reviewer Workflow 혁신 **오규철**(가톨릭의대 순환기내과)	/ 138
15:40-16:00	AI 시대의 에디터 역할과 출판 품질관리의 미래: 위기와 기회의 변곡점에서 **유승찬**(연세의대 의생명시스템정보학교실)	/ 140
16:00-16:20	Panel Discussion	

Symposium 12

4월 4일(토) 08:30-10:00 | Room 1 (그랜드볼룸 3)

Current and Future Perspectives on Lipid-lowering Agents

좌장 : 채인호(서울의대 순환기내과), 권혁상(가톨릭의대 내분비내과)

패널 : 김경안(가톨릭의대 순환기내과), 이지은(고려의대 순환기내과), 황유철(경희의대 내분비내과)

08:30-08:48	Bempedoic acid: a non-statin option for LDL-C lowering **차정준**(고려의대 순환기내과)	/ 144
08:48-09:06	The evolving landscape of PCSK9 inhibitors: siRNA, oral agents, and mAbs **조경훈**(전남의대 순환기내과)	/ 146
09:06-09:24	Lp(a)-lowering therapies: olpasiran and other emerging agents **최성훈**(한림의대 순환기내과)	/ 148
09:24-09:42	CETP inhibitors: revisiting an old target with new molecules **홍준화**(을지의대 내분비내과)	/ 150
09:42-10:00	Panel Discussion	

Symposium 13

4월 4일(토) 08:30-10:00 | Room 2 (그랜드볼룸 2)

AI & Digital Health Technology for Atherosclerosis

좌장 : 강석민(연세의대 심장내과), 김병극(연세의대 심장내과)

패널 : 김성은(한림의대 소화기내과), 배성아(연세의대 심장내과), 안지현(한국의학연구소 내과)

08:30-08:50	Updated digital technology for atherosclerosis management **조준환**(중앙의대 순환기내과)	/ 154
08:50-09:10	Multi-modality intracoronary imaging for characterizing atherosclerosis **강동오**(고려의대 순환기내과)	/ 156
09:10-09:30	AI for cardiovascular image in CT **양동현**(울산의대 영상의학과)	/ 158
09:30-10:00	Panel Discussion	

Symposium 14 – KSCMS–KSoLA Joint Symposium

4월 4일(토) 08:30–10:00 | Room 3 (그랜드볼룸 1)

Incretin Therapies and Circadian Biology for Cardiometabolic Protection

좌장 : 김재택(중앙의대 내분비내과), 위진(가천의대 심장내과)

패널 : 김경희(인천세종병원 심장내과), 조은정(중앙의대 순환기내과), 홍상모(한양의대 내분비내과)

08:30–08:48	Circadian rhythm and diabetes/metabolism **김원진**(차의대 내분비내과)	/ 162
08:48–09:06	Incretin signaling and circadian regulation of glucose and energy metabolism **손장원**(가톨릭의대 내분비내과)	/ 164
09:06–09:24	GLP–1/GIP agonists for cardiometabolic protection – one drug fits all? **정창희**(울산의대 내분비내과)	/ 166
09:24–09:42	Safety and sustainability concerns – beyond the GLP–1RA hype **서미혜**(순천향의대 내분비대사내과)	/ 168
09:42–10:00	Panel Discussion	

Symposium 15

4월 4일(토) 13:10–14:40 | Room 1 (그랜드볼룸 3)

Updates in the 2026 Korean Dyslipidemia Guidelines: Integrating Global Evidence

좌장 : 김상현(서울의대 순환기내과), 김현진(한양의대 심장내과)

패널 : 김송이(제주의대 순환기내과), 김치경(고려의대 신경과),
박경택(중앙의대 순환기내과), 제세영(서울시립대 스포츠과학과)

13:10–13:28	Updated risk stratification and treatment pathways in the 2026 Korean guidelines **정재훈**(동국의대 심장내과)	/ 172
13:28–13:46	Optimizing pharmacologic therapy in the 2026 Korean dyslipidemia guidelines **오진경**(충남의대 심장내과)	/ 174
13:46–14:04	Lifestyle interventions in dyslipidemia care: evidence updates and practical recommendations for Korea **조가람**(한남대 식품영양학과)	/ 176
14:04–14:22	Managing dyslipidemia in special populations in the 2026 Korean guidelines **장영우**(가천의대 심장내과)	/ 178
14:22–14:40	Panel Discussion	

Symposium 16

4월 4일(토) 13:10-14:40 | Room 2 (그랜드볼룸 2)

Primary Prevention of Cardiovascular Disease

좌장 : 김명아(서울의대 순환기내과), 이왕수(중앙의대 순환기내과)

패널 : 김효진(고려의대 신장내과), 유지웅(한림의대 순환기내과), 이선화(전북의대 심장내과)

13:10-13:30	Association between cumulative LDL-C exposure during young adulthood and middle age and risk of cardiovascular events **양여리**(가톨릭의대 내분비내과)	/ 182
13:30-13:50	MASLD as a cardiovascular risk enhancer in primary prevention **김경수**(차의대 내분비내과)	/ 184
13:50-14:10	Lipid-lowering therapy for primary prevention in the elderly **이상학**(연세의대 심장내과)	/ 186
14:10-14:40	Panel Discussion	

Symposium 17 - KOVAS-KSoLA Joint Symposium

4월 4일(토) 13:10-14:40 | Room 3 (그랜드볼룸 1)

Unraveling the Pathway of Atherosclerosis through Imaging and Lipid Assessment

좌장 : 정익모(이화의대 순환기내과), 성기철(성균관의대 순환기내과)

패널 : 김학령(서울의대 순환기내과), 이민경(한양의대 내분비내과), 임수빈(이화의대 순환기내과)

13:10-13:30	Coronary artery calcium and imaging-based risk assessment in clinical practice **손정우**(연세원주의대 심장내과)	/ 190
13:30-13:50	Assessment of arterial remodeling and atherosclerosis progression using carotid ultrasound **이종영**(한림의대 순환기내과)	/ 192
13:50-14:10	Pulse wave velocity and ankle-brachial index as indicators of arterial health **권오성**(가톨릭의대 순환기내과)	/ 194
14:10-14:40	Panel Discussion	

Symposium 18

4월 4일(토) 13:40-14:40 | Room 4 (볼룸 1,2)

Dyslipidemia Fact Sheet 2026

좌장 : 유승기(이화의대 건진의학과), 최성훈(한림의대 순환기내과)

패널 : 김병식(한양의대 심장내과), 나진오(고려의대 순환기내과), 배재현(한림의대 내분비내과)

13:40-13:55	Epidemiology of dyslipidemia in Korean adults, 2026 **문민경**(서울의대 내분비대사내과)	/ 198
13:55-14:10	Management of hypercholesterolemia and dyslipidemia: from overall burden to high-risk populations **양예슬**(서울의대 내분비대사내과)	/ 200
14:10-14:25	Health behaviors among people with dyslipidemia **김규호**(가톨릭의대 내분비내과)	/ 202
14:25-14:40	Panel Discussion	

Symposium 19

4월 4일(토) 15:30-17:00 | Room 1 (그랜드볼룸 3)

Innovative Technologies and Disease Modeling for Lipotoxicity and Atherosclerosis

좌장 : 오구택(이화여대 생명과학과), 박영미(이화의대 분자의과학교실)

패널 : 권유욱(서울의대 의생명연구원), 김경진(인하의대 의생명학교실), 송주현(전남의대 해부학교실)

15:30-15:50	From basic discovery to clinical translation: macrophage-targeted imaging and therapy for high-risk plaque **김진원**(고려의대 순환기내과)	/ 206
15:50-16:10	Intracellular Ca^{2+} dysregulation links lipotoxicity to defective autophagy **오병철**(가천의대 생리학교실)	/ 208
16:10-16:30	In vitro modeling of atherosclerosis using iPSC-derived blood vessel organoids **김다현**(성신여대 바이오생명공학과)	/ 210
16:30-17:00	Panel Discussion	

Symposium 20

4월 4일(토) 15:30–17:00 | Room 2 (그랜드볼룸 2)

Nutritional Perspectives on Sex Differences in Cardiometabolic Health

좌장 : 이명숙(성신여대 식품영양학과), 정효지(서울대 보건대학원)

패널 : 박용주(전남대 식품영양학과), 백진경(을지대 식품영양학과), 이동훈(연세대 스포츠응용산업학과)

15:30–15:50	Sex differences in metabolic syndrome and heart **박성미**(고려의대 순환기내과)	/ 214
15:50–16:10	Uncovering FPR2 as a determinant of sex differences in liver fibrosis **정영미**(부산대 생명과학과)	/ 216
16:10–16:30	Sex-dependent microbe-metabolite interactions in cardiometabolic diseases **유현주**(서울대 식품영양학과)	/ 218
16:30–17:00	Panel Discussion	

Symposium 21 – KSIC–KSoLA Joint Symposium

4월 4일(토) 15:30–17:00 | Room 3 (그랜드볼룸 1)

Targeting Atherosclerotic Vulnerability: Lipoprotein Modulation and Inflammation

좌장 : 최동훈(연세의대 심장내과), 남창욱(계명의대 심장내과)

패널 : 김수홍(부산보훈병원 순환기내과), 김희동(순천향의대 심장내과),
양한모(서울의대 순환기내과), 원호연(중앙의대 순환기내과)

15:30–15:48	The role of early PCSK-9 inhibitor in patients with ACS **조윤경**(계명의대 심장내과)	/ 222
15:48–16:06	Lp(a) and atherosclerotic burden: new targets in the post-PCI era **우종신**(경희의대 심장내과)	/ 224
16:06–16:24	The association between coronary artery plaque and ANGPTL4 **한정규**(서울의대 순환기내과)	/ 226
16:24–16:42	Inflammation and atherosclerosis: recent advances in anti-inflammatory therapy **김병식**(한양의대 심장내과)	/ 228
16:42–17:00	Panel Discussion	

Breakfast Symposium 1

4월 4일(토) 07:30-08:00 | Room 1 (그랜드볼룸 3)

좌장 : 신현호(아산충무병원 심장내과), 박철영(성균관의대 내분비내과)

07:30-07:45 Safety and efficacy of moderate-intensity statin with ezetimibe in elderly patients with ASCVD / 232
차정준(고려의대 순환기내과)

07:45-08:00 Benefits of low dose rosuvastatin plus ezetimibe combination therapy / 233
김원진(차의대 내분비내과)

Breakfast Symposium 2

4월 4일(토) 07:30-08:00 | Room 2 (그랜드볼룸 2)

좌장 : 우정택(경희의대 내분비대사내과), 조진만(경희의대 심장혈관내과)

07:30-07:45 Appropriate statin therapy for low- to moderate-risk patients ("The earlier, the better; the longer, the better.") / 236
정창희(울산의대 내분비내과)

07:45-08:00 CKD and lipid management: when guidelines differ, what should we do? / 237
박세훈(서울의대 신장내과)

Breakfast Symposium 3

4월 4일(토) 07:30-08:00 | Room 3 (그랜드볼룸 1)

좌장 : 김명곤(가톨릭관동의대 심장내과), 박헌식(경북의대 순환기내과)

07:30-07:45 Clinical rationale for acid suppressants in cardiology; a multidisciplinary perspective / 240
박상민(을지의대 심장내과)

07:45-08:00 P-CAB treatment strategies: a case-based approach / 241
장영우(가천의대 심장내과)

Breakfast Symposium 4

4월 4일(토) 07:30-08:00 | Room 4 (볼룸 1,2)

교육위원회 세션

좌장 : 백상홍 (가톨릭의대 순환기내과)

07:30-07:45 2026 ACC/AHA 가이드라인, 무엇이 달라졌나요? / 244
홍준화(을지의대 내분비내과)

07:45-08:00 심부전 환자에서 지질관리 / 246
김경희(세종병원 심장내과)

Luncheon Symposium 1

4월 4일(토) 12:00-12:30 | Room 1 (그랜드볼룸 3)

좌장 : 이문규(을지의대 내분비내과), 장학철(서울의대 내분비대사내과)

Luncheon Symposium 2

4월 4일(토) 12:00-12:30 | Room 2 (그랜드볼룸 2)

좌장 : 김치정(중앙의대 순환기내과), 박경수(건국의대 내분비대사내과)

Luncheon Symposium 3

4월 4일(토) 12:00-12:30 | Room 3 (그랜드볼룸 1)

좌장 : 박영배(서울의대 순환기내과), 김효수(서울의대 순환기내과)

Oral Presentations

Oral Presentation 1

좌장 : 국현(전남의대 약리학교실), 김학령(서울의대 순환기내과)

4월 3일(금) 12:00-13:20 | Room 4 (볼룸 1,2)

Oral Presentation 2

좌장 : 박용식(경희의대 미생물학교실), 한정규(서울의대 순환기내과)

4월 4일(토) 08:30-10:00 | Room 4 (볼룸 1,2)

Oral Presentation 3

좌장 : 김대중(아주의대 내분비대사내과), 신미승(가천의대 심장내과)

4월 4일(토) 15:30-17:00 | Room 4 (볼룸 1,2)

Mini-Oral Presentations

Mini-Oral Presentation 1-1

좌장 : 박용현(부산의대 순환기내과)

4월 3일(금) 17:00-17:50 | 발표구역 A (포이어(로비))

Mini-Oral Presentation 1-2

좌장 : 정재훈(동국의대 심장내과)

4월 3일(금) 17:00-17:50 | 발표구역 B (포이어(로비))

Mini-Oral Presentation 1-3

좌장 : 김정민(서울의대 신경과)

4월 3일(금) 17:00-17:50 | 발표구역 C (포이어(로비))

Mini-Oral Presentation 1-4

좌장 : 김원진(차의대 내분비내과)

4월 3일(금) 17:00-17:50 | 발표구역 D (포이어(로비))

Mini-Oral Presentation 2-1

좌장 : 양여리(가톨릭의대 내분비내과)

4월 4일(토) 10:50-12:00 | 발표구역 A (포이어(로비))

Mini-Oral Presentation 2-2

좌장 : 우종신(경희의대 심장내과)

4월 4일(토) 10:50-12:00 | 발표구역 B (포이어(로비))

Mini-Oral Presentation 2-3

좌장 : 정미향(가톨릭의대 순환기내과)

4월 4일(토) 10:50-12:00 | 발표구역 C (포이어(로비))

Mini-Oral Presentation 2-4

좌장 : 전재한(경북의대 내분비대사내과)

4월 4일(토) 10:50-12:00 | 발표구역 D (포이어(로비))

E-Posters

E-Posters

SoLA 2026

한국지질·동맥경화학회 춘계학술대회

2026 Spring Congress on
Lipid and Atherosclerosis of KSoLA

SoLA 2026

한국지질·동맥경화학회 춘계학술대회

2026 Spring Congress on
Lipid and Atherosclerosis of KSoLA

Plenary Lecture 1

4월 4일(토) 10:10–10:40 | Room 1,2,3 (그랜드볼룸 1,2,3)

좌장 : 김성래(가톨릭의대 내분비내과)

10:10–10:40 **Advancing genomic medicine for lipid disorders and atherosclerosis**

Daniel J. Rader (University of Pennsylvania, USA)

CURRICULUM VITAE

Daniel J. Rader

Perelman School of Medicine, University of Pennsylvania, Philadelphia, PA, USA

Education

1981	B.A., Lehigh University (Summa Cum Laude)
1984	M.D., Medical College of Pennsylvania (Summa Cum Laude)

Postgraduate Training and Fellowship Appointments

1984-1985	Internship, Internal Medicine, Yale-New Haven Hospital, New Haven, CT
1985-1987	Residency, Internal Medicine, Yale-New Haven Hospital, New Haven, CT
1987-1988	Chief Resident, Internal Medicine, Yale School of Medicine, New Haven, CT
1988-1991	Medical Staff Fellow, Molecular Disease Branch, National Heart, Lung, and Blood Institute, National Institutes of Health, Bethesda, MD

Academic Appointments

1991-1993	Staff Scientist, Molecular Disease Branch, NHLBI, NIH
1994-2000	Assistant Professor of Medicine, University of Pennsylvania School of Medicine
2000-2005	Associate Professor of Medicine, University of Pennsylvania School of Medicine
2006-2014	Edward S. Cooper, M.D./Norman Roosevelt and Elizabeth Meriwether McLure Professor of Medicine
2006-Present	Professor of Pharmacology, University of Pennsylvania School of Medicine (Secondary)
2013-Present	Professor of Medicine in Genetics, University of Pennsylvania School of Medicine (Secondary)
2014-Present	Seymour Gray Professor of Molecular Medicine, University of Pennsylvania, School of Medicine

Hospital and Administrative Appointments

1994-Present	Director, Preventive Cardiovascular Medicine and Lipid Clinic, University of Pennsylvania Health System
2002	Chair, Experimental Therapeutics Theme Group, Strategic Planning Initiative, University of Pennsylvania School of Medicine
2002-2008	Faculty Advisory Committee, Office of Human Research, Univ. Penn
2003-2012	Chair, Advisory Committee, Molecular Diagnostics and Genotyping Core, University of Pennsylvania School of Medicine
2005-Present	Associate Director, Institute for Translational Medicine and Therapeutics University of Pennsylvania School of Medicine

Advancing genomic medicine for lipid disorders and atherosclerosis

Daniel J. Rader

Perelman School of Medicine at the University of Pennsylvania, USA

Lipid disorders and atherosclerotic cardiovascular disease (ASCVD) have been at the forefront of genomic discovery, which has markedly advanced our understanding of the molecular mechanisms regulating lipid metabolism and influencing atherosclerosis. Multiple new therapeutic targets have been identified through genomics research and are leading to a revolution in our ability to treat lipid disorders and reduce risk of ASCVD. An excellent example is that of triglycerides and triglyceride-rich lipoproteins (TRLs). Extensive mendelian randomization has confirmed that TRLs are causally associated with not only ASCVD, but also peripheral arterial disease (PAD) and abdominal aortic aneurysm (AAA). The lipoprotein lipase (LPL) pathway is central in regulating the metabolism of TRLs and multiple proteins that influence LPL activity are targets for therapies to reduce triglycerides. Whether these approaches will reduce risk of ASCVD (and other cardiometabolic diseases) will be a vitally important question for the next decade. Finally, the field of genomic medicine encompasses the use of genomic information about an individual to optimize their clinical care and prevention of disease. This lecture will also illustrate the applications of genomic medicine to lipid disorders and ASCVD and anticipate the ways in which genomic medicine will transform the practice of medicine in treating and preventing cardiometabolic disease.

SoLA 2026

한국지질·동맥경화학회 춘계학술대회

2026 Spring Congress on
Lipid and Atherosclerosis of KSoLA

SoLA 2026

한국지질·동맥경화학회 춘계학술대회

2026 Spring Congress on
Lipid and Atherosclerosis of KSoLA

Plenary Lecture 2

4월 4일(토) 14:50–15:20 | Room 1,2,3 (그랜드볼룸 1,2,3)

좌장 : 김상현(서울의대 순환기내과)

14:50–15:20 **Molecular integration of lipid metabolism and inflammation in cardiovascular disease**

임승순(계명의대 생리학교실)

CURRICULUM VITAE

임승순

계명대학교 의과대학 생리학교실

[학력 및 경력]

2006	Yonsei University, 박사
2006-2007	Yonsei University College of Medicine, 연구강사
2007-2009	University of California, Irvine, 박사후연구원
2009-2012	Sanford Burnham Medical Research Institute, 선임연구원
2012-Present	Keimyung University School of Medicine, 교수
2018-2019	University of California, San Diego, 방문교수

[관심분야]

Atherosclerosis, Obesity, Lipid metabolism, MASH

[논문]

1. Liver Receptor Homolog-1 Deficiency Impairs Alcohol-Associated Liver Disease Owing to Decrease of Aldehyde Dehydrogenase 1 Family Member B1 Gene Expression. Seo MH, Lee JH, Lee EH, Mukherjee S, Park SY, Bae JH, Song DK, Im SS*. Mol Cell Biol. 2025;45(7):301-314.
2. Loss of SREBP-1c ameliorates iron-induced liver fibrosis by decreasing lipocalin-2. Lee EH, Lee JH, Kim DY, Lee YS, Jo Y, Dao T, Kim KE, Song DK, Seo JH, Seo YK, Seong JK, Moon C, Han E, Kim MK, Ryu S, Shin M, Roh GS, Jung HR, Osborne TF, Ryu D, Jeon TI, Im SS*. Exp Mol Med. 2024 Apr;56(4):1001-1012.
3. SCAP deficiency facilitates obesity and insulin resistance through shifting adipose tissue macrophage polarization. Lee JH, Lee SH, Lee EH, Cho JY, Song DK, Lee YJ, Kwon TK, Oh BC, Cho KW, Osborne TF, Jeon TI, Im SS*. J Adv Res. 2023 Mar;45:1-13.
4. Impairment of ULK1 sulfhydration-mediated lipophagy by SREBF1/SREBP-1c in hepatic steatosis. Nguyen TTP, Kim DY, Im SS*, Jeon TI*. Autophagy. 2021 Dec;17(12):4489-4490.
5. SREBP-1c impairs ULK1 sulfhydration-mediated autophagic flux to promote hepatic steatosis in high-fat-diet-fed mice. Nguyen TTP, Kim DY, Lee YG, Lee YS, Truong XT, Lee JH, Song DK, Kwon TK, Park SH, Jung CH, Moon C, Osborne TF, Im SS*, Jeon TI*. Mol Cell. 2021 Sep 16;81(18):3820-3832.e7.

Molecular integration of lipid metabolism and inflammation in cardiovascular disease

Seung-Soon Im

Department of Physiology, Keimyung University School of Medicine, Republic of Korea

Atherosclerosis as a chronic inflammatory vascular disease underlies most cardiovascular diseases and remains a leading cause of mortality due to its complex etiology and lack of effective pharmacological interventions. The accumulation of lipid-rich plaques within the vascular endothelium plays a pivotal role in atherosclerosis progression, yet the precise molecular mechanisms remain incompletely understood. In particular, the functional role and regulatory mechanisms of Sterol Regulatory Element-Binding Protein 1a (SREBP-1a) in macrophages during atherogenesis are not fully elucidated. In this study, we investigated the role of SREBP-1a in the development of atherosclerosis by utilizing bone marrow-derived cells from SREBP-1a-deficient mice transplanted into lethally irradiated LDL receptor-deficient (LDLR KO) mice to generate atherosclerosis-prone bone marrow transplant (BMT) models. Phenotypic and pathological assessments revealed aggravated atherosclerotic plaque formation and altered lipid metabolic profiles in the SREBP-1a-deficient BMT mice compared to controls. Furthermore, bone marrow-derived macrophages treated with oxidized LDL (oxLDL) exhibited altered expression of lipid metabolism-associated genes and pro-inflammatory mediators under SREBP-1a deficiency, indicating a regulatory role of SREBP-1a in macrophage lipid homeostasis and inflammation. To identify downstream effectors of SREBP-1a, we performed both single-cell RNA sequencing and bulk transcriptome analyses. Among the candidate genes, high mobility group AT-hook 1 and vacuolar protein sorting 37B emerged as key regulators potentially involved in atherogenesis, although their roles in vascular disease are poorly characterized. Our data suggest that SREBP-1a deficiency exacerbates atherosclerosis by modulating the expression of these novel target genes in oxLDL-exposed macrophages. Identification of SREBP-1a-mediated transcriptional targets provide mechanistic insights into macrophage-driven atherogenesis and may offer novel therapeutic strategies for cardiovascular disease.

SoLA 2026

한국지질·동맥경화학회 춘계학술대회

2026 Spring Congress on
Lipid and Atherosclerosis of KSoLA

SoLA 2026

한국지질·동맥경화학회 춘계학술대회

2026 Spring Congress on
Lipid and Atherosclerosis of KSoLA

Symposium 1

Metabolic Regulation and Atherosclerosis: Emerging Therapeutic Targets

4월 3일(금) 12:00-13:20 | Room 1 (그랜드볼룸 3)

좌장 : 김치대(부산의대 약리학교실), 한진(인제의대 생리학교실)

패널 : 김정한(가톨릭의대 생화학교실), 김형규(인제의대 생리학교실)
남궁준(연세원주의대 생화학교실), 류홍열(경북대 생명공학과)

12:00-12:20	**Redox-dependent regulation of a protein controls lipid peroxidation at the plasma membrane and ferroptosis** **이은우**(한국생명공학연구원 대사제어연구센터)
12:20-12:40	**Next generation NAD^+ therapeutics via new modalities** **류동렬**(GIST 의생명공학과)
12:40-13:00	**Development of cardiovascular therapeutics through regulation of endothelial dysfunction** **장우철**(부산대 생물교육과)
13:00-13:20	**Panel Discussion**

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/책임연구원, 한국생명공학연구원 대사제어연구센터
2011-2016	박사후연구원, 연세대학교 생화학과

[관심분야]

Ferroptosis, Lipid metabolism, ROS, Senescence, Athersclerosis

[논문]

1. Kim JW et al., Methionine metabolism is linked with phospholipid and glutamine metabolism to drive ferroptosis, Cell Rep, In press (2026)
2. Cha YJ el al., Limitation of Ferroptosis Inhibitor on the Doxorubicin-Induced Cardiotoxicity, Antioxidants, 15(1), 27 (2026)
3. Kim JW et al., When gut fibroblasts feed epithelial cells to death, Nat Metab. 7(7):1307-1309 (2025)
4. Choi DW et al., "Good" fats, bad news: HDL-delivered vitamin E shields tumors from ferroptosis. Signal Transduct Target Ther. 10(1):284 (2025)
5. 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)

Redox-dependent regulation of a protein controls lipid peroxidation at the plasma membrane and ferroptosis

Eun-Woo Lee

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

Inhibition of GPX4 rapidly induces lipid peroxidation, ultimately leading to ferroptosis after a prolonged period, with specific events during this duration remaining poorly understood. Here, we reveal that a protein X is an early response protein that ensures lipid peroxidation at the plasma membrane during ferroptosis. Genetic ablation or chemical inhibition of X prevents ferroptosis in response to GPX4 inhibition and cysteine deprivation. Remarkably, while lipid peroxidation signals were observed in both the perinuclear region and the plasma membrane in control cells, cells lacking protein X exhibit lipid peroxidation signals solely in the cytoplasm or subcellular organelles, not at the plasma membrane. The reintroduction of wild-type X, but not a mutant with defective membrane localization, restores proper lipid peroxidation and induces ferroptosis. Interestingly, X translocates to the perinuclear region and plasma membrane in response to ferroptotic stimuli, but this process was inhibited by ferrostatin-1, indicating a requirement of lipid peroxidation. The Y Cys to Asp mutant, mimicking cysteine oxidation, displayed a diminished binding capacity to interact with X, consequently losing its ability to prevent the membrane localization of X. Our study suggests that X, released from Y during the early phase of ferroptosis, plays a crucial role in accelerating ferroptotic cell death. Finally, inhibiting ferroptosis by targeting protein X or using a lipophilic antioxidant could represent a potential therapeutic approach for atherosclerosis.

CURRICULUM VITAE

류동렬(Dongryeol Ryu)

GIST 의생명공학과

[학력]

2010	Ph.D. in Molecular Cell Biology, SKKU School of Medicine, South K
2006	M.S. in Molecular Biology, PNU, South Korea
2004	B.S. in Molecular Biology, PNU, South Korea

[경력]

2023-Present	Associate Professor, GIST, Department of Biomedical Science and Engineering
2019-2023	Assistant Professor, SKKU, School of Medicine, Department of Mol Cell Biology
2017-2019	Assistant Professor, PNU, Department of Korean Medical Science

[관심분야]

Aging, Metabolism and their associated diseases

[논문]

1. Zhang Y, et al., Myosteatosis: epidemiological insights, functional decline, and diagnostic advances. Curr. Obes. Rep. 14:83 (2025)
2. Wei S, et al., Detrimental effects of beta2-microglobulin on muscle metabolism. Evidence from in vitro, animal, and human research. J Cachexia Sarcopenia Muscle 16, e13745 (2025)
3. Wei S, et al., Hydrogen peroxide-releasing hydrogel-mediated cellular senescence model for aging research. Biomater Res 29, 0161 (2025)
4. Kim J, et al., A microbiota-derived metabolite 3-phenyllactic acid prolongs healthspan by enhancing mitochondrial function and stress resilience via SKN-1 ATFS-1 in C elegans. Nat Commun 15, 10773 (2024)
5. Kim Y, et al., Inhibition of SIRT7 overcomes sorafenib acquired resistance by suppressing ERK1 2 phosphorylation via the DDX3X-mediated NLRP3 inflammasome in hepatocellular carcinoma. Drug Resist Updates 73, 101054 (2024)

Next generation NAD^+ therapeutics via new modalities

Dongryeol Ryu

Department of Biomedical Science and Engineering, GIST, South Korea

Nicotinamide adenine dinucleotide (NAD^+) is an essential biomolecule that regulates fundamental cellular processes, including energy metabolism, DNA repair, redox balance, and inflammatory responses. NAD^+ depletion is frequently observed during aging, chronic diseases, DNA damage, and persistent inflammatory stress. Despite extensive preclinical evidence supporting the therapeutic potential of NAD^+ boosting, clinical translation has been limited by compound instability, inefficient NAD^+ conversion, and poor delivery to target tissues.

In the first part, we present a classical NAD^+ boosting approach using the precursor nicotinamide riboside (NR). We demonstrate that ritonavir, a drug widely used in HIV and COVID-19 treatment, induces NAD^+ depletion and lipodystrophy, and that oral NR administration confers significant protective effects in mouse models. Mechanistic analyses further suggest that ritonavir-induced NAD^+ loss may be linked to interferon signaling and ferroptosis-related pathways, providing new insight into drug-associated metabolic toxicity.

In the second and third parts, we introduce new modality-based NAD^+ boosting strategies that enable sustained, localized NR delivery using biomaterial platforms. We developed iAngel for myocardial infarction, a biodegradable material with strong tissue adherence and controlled NR release, allowing efficient NAD^+ boosting directly at sites of cardiac injury. In parallel, we designed iDeal for tendinitis, a structurally distinct platform capable of continuous NR release while attached to the Achilles tendon. Together, these approaches address a fundamental limitation of oral NR administration—its inability to achieve therapeutically relevant concentrations in poorly perfused or structurally dense tissues.

In the fourth part, we describe ICoN, a negatively charged, NAD^+-capturing liposomal system that enables direct NAD^+ delivery rather than precursor supplementation. Using in vitro models and ex vivo porcine and human skin tissues, we demonstrate that subcutaneous administration of ICoN effectively transfers and boosts NAD^+ levels within target tissues.

Collectively, these four stories illustrate that next-generation NAD^+ boosting strategies based on innovative delivery modalities can overcome the intrinsic limitations of conventional approaches and open a new translational chapter for NAD^+-based therapeutics.

CURRICULUM VITAE

장우철

부산대학교 생물교육과

[학력]

1994-2002	고려대학교 학사
2002-2005	고려대학교 석사
2005-2009	연세대학교 박사

[경력]

2025-현재	국가신약개발사업단 평가위원
2024-현재	부산대학교 연구부처장
2024-현재	한국분자세포생물학회 대의원
2022-현재	양산부산대병원 의생명융합연구원 연구자문위원
2022-현재	생화학분자생물학회 BMB Reports (SCIE) 편집위원
2022-2025	FAOBMB 2025 Congress 교육위원장
2022-2023	HUPO 2023 Congress 지역위원장
2022-2023	FAOPS 2023 Congress 홍보위원장
2021-2024	한국연구재단 생명과학단 전문위원(RB)
2020-현재	보건복지부 재생의료진흥재단 심사위원
2017-현재	생화학분자생물학회 대의원 및 운영위원
2016-2023	한국줄기세포학회 위원 및 운영위원
2013-현재	부산대학교 생물교육과 교수
2009-2012	Yale University School of Medicine Post Doc.

[관심분야]

Cardiovascular Diseases, Endothelial to Mesenchymal Transition, Stem Cells, Organoid

[논문]

1. Kim R, Kim M, Jeong S, Kim S, Moon H, Kim H, Lee MY, Kim J, Kim HS, Choi M, Shin K, Song BW, Chang W. Melatonin Alleviates Myocardial Dysfunction through Inhibition of Endothelial-to-Mesenchymal Transition via the NF-κB Pathway (2024) Journal of Pineal Research 76(4):12958 (IF=10.2)
2. Kim S, Lee H, Moon H, Kim R, Kim M, Jeong S, Kim H, Kim SH, Hwang SS, Lee MY, Kim J, Song BW, Chang W. Epigallocatechin-3-Gallate Attenuates Myocardial Dysfunction via Inhibition of Endothelial-to-Mesenchymal Transition (2023) Antioxidants. 12(5):1059 (IF=7.675)

Development of cardiovascular therapeutics through regulation of endothelial dysfunction

Woochul Chang

Department of Biology Education, Pusan National University, Republic of Korea

Endothelial to mesenchymal transition (EndMT) is a special type of epithelial to mesenchymal transition. It is a process that is characterized by the loss of features of endothelial cells and acquisition of specific markers of mesenchymal cells. A variety of stimuli, such as inflammation, growth factors, and hypoxia, regulate EndMT through various signaling pathways and intracellular transcription factors. It has been demonstrated that epigenetic modifications are also involved in this process. Recent studies have identified the essential role of EndMT in the cardiovascular system. EndMT contributes to steps in cardiovascular development, such as cardiac valve formation and septation, as well as the pathogenesis of various cardiovascular disorders, such as congenital heart disease, myocardial fibrosis, myocardial infarction and pulmonary arterial hypertension. Thus, comprehensive understanding of the underlying mechanisms of EndMT will provide novel therapeutic strategies to overcome congenital heart disease due to abnormal development and other cardiovascular diseases.

SoLA 2026

한국지질·동맥경화학회 춘계학술대회

2026 Spring Congress on
Lipid and Atherosclerosis of KSoLA

SoLA 2026

한국지질·동맥경화학회 춘계학술대회

2026 Spring Congress on
Lipid and Atherosclerosis of KSoLA

Symposium 2

Translational Nutrition: From Molecular Pathways to Clinical Practice in Cardiometabolic Diseases

4월 3일(금) 12:00–13:20 | Room 2 (그랜드볼룸 2)

좌장 : 한성림(서울대 식품영양학과), 김정선(국립암센터 암의생명과학과)

패널 : 김오연(동아대 식품영양학과), 박동현(세종대 데이터사이언스학과)
박신옥(눔코리아), 임현정(경희대 의학영양학과)

12:00–12:20	**Nutritional modulation of metabolic enzymes and epigenetic approaches for cardiometabolic health** **강현주**(경북대 식품공학부)
12:20–12:40	**Predicting drug–food interactions using AI** **김모건**(한국외대 바이오메디컬공학부)
12:40–13:00	**Shared metabolic switches in obesity treatment: from intermittent fasting to GLP1 based therapy** **신민정**(고려대 바이오시스템의과학부)
13:00–13:20	**Panel Discussion**

CURRICULUM VITAE

강현주

경북대학교 식품공학부

[학력]

2011-2015 연세대학교 식품영양학과 학사
2015-2016 연세대학교 식품영양학과 석사
2017-2020 University of Connecticut, Nutritional Sciences 박사

[경력]

2026-현재 경북대학교 식품공학부 조교수
2022-2026 계명대학교 식품영양학과 조교수
2021-2022 University of Connecticut 박사후연구원

[관심분야]

Immune Regulation, Histone Modification, Bioactive Compound

[논문]

1. J.H. Bae, H. Kang*, Longitudinal study on the association between salty food preference and obesity prevalence, International Journal of Food Sciences and Nutrition (2026), 77, 80.
2. J. Jo, C.Y. Moon, J. Lee, H. Kang*, Fisetin attenuates lipopolysaccharide-induced neuroinflammation by modulating the histone deacetylase 3-nuclear factor erythroid 2-related factor 2 axis, Food Bioscience (2025), 71, 107264.
3. H. Kang, M-.B. Kim, H. Jang, Y. Lee, J. Lee, O. Corvino, A. Kim, Y-.K. Park, J-.Y. Lee. Macrophage Histone Deacetylase 4 Has Sex-Dependent Dimorphic Effects on the Pathogenesis of Alcohol-Associated Hepatitis, Journal of Gastroenterology and Hepatology (2025), 40, 3009.
4. J. Park, C.Y. Moon, J. Jo, H. Kang*, Protective effects of ginsenoside Rd on inflammation and mitochondrial dysfunction in lipopolysaccharide-induced microglial activation through histone deacetylase 5-mediated signaling, Food Bioscience (2025), 66, 106248.
5. C.Y. Moon, J. Park, E.J. Shin, H. Kang*, Nicotinamide riboside abrogates lipopolysaccharide-induced inflammation and mitochondrial dysfunction in microglia through the AMP-activated protein kinase-sirtuin 3 signaling pathway, Food Bioscience (2025), 63, 105799.

Nutritional modulation of metabolic enzymes and epigenetic approaches for cardiometabolic health

Hyunju Kang

School of Food Science and Biotechnology, Kyungpook National University, Daegu, Republic of Korea

The pathogenesis of cardiometabolic diseases is intricately regulated by the dynamic interplay between genetic predispositions and environmental dietary habits. Recent longitudinal analyses reveal that inherent genetic variations associated with sweet and salty taste preferences serve as strong predictors for obesity and metabolic dysregulation. To counteract these inherent phenotypic risks, the targeted nutritional modulation of metabolic enzymes has emerged as a crucial epigenetic defense mechanism. To bridge the gap between these inherent risk factors and molecular nutrition, we detail the protective mechanisms of specific dietary interventions, such as nicotinamide riboside. To mitigate the systemic inflammation and oxidative stress exacerbated by poor dietary habits, these food-derived bioactive compounds actively reprogram cellular energy metabolism, specifically modulating glycolysis and mitochondrial respiration. Furthermore, these nutritional modulators exert profound anti-inflammatory effects by regulating targeted epigenetic enzymes, including specific histone deacetylases (HDACs) and sirtuins (SIRTs), which are NAD^+-dependent deacetylases. Molecular docking simulations demonstrate that these bioactive compounds can directly bind to the targeted enzymes to alter their functional states. Notably, our findings highlight that the epigenetic regulation of these metabolic pathways can exhibit profound sex-dependent dimorphic effects, underscoring the complexity of physiological responses. By integrating genetic taste preference profiling with the precise molecular and epigenetic application of dietary compounds, this research establishes a foundation for precision nutrition, offering highly customized strategies for optimal cardiometabolic health.

CURRICULUM VITAE

김모건

한국외국어대학교 바이오메디컬공학부

[학력]

2018 고려대학교 컴퓨터학과 학사
2023 고려대학교 컴퓨터학과 박사

[경력]

2023.09-2024.08 고려대학교 컴퓨터정보통신연구소 박사후연구원
2024.09- 한국외국어대학교 조교수

[관심분야]

AI and {Precision Nutrition, Drug Discovery, Food Science, Polymer Chemistry}

[논문]

1. Mogan Gim*, Jaewoo Kang, Donghyeon Park**, Minji Jeon**, "ArcDFI: Attention regularization guided by CYP450 interactions for predicting drug-food interactions", PLOS Computational Biology, vol. 21, https://doi.org/10.1371/journal.pcbi.1013055)
2. Junseok Choe*, Hajung Kim*, Yan Ting Chok, Mogan Gim**, Jaewoo Kang**, "Retrosynthetic crosstalk between single-step reaction and multi-step planning", Journal of Cheminformatics, vol. 17, https://doi.org/10.1186/s13321-025-01088-z)
3. Seungheun Baek*, Soyon Park*, Yan Ting Chok, Mogan Gim**, Jaewoo Kang**, "GPO-VAE: Modeling Explainable Gene Perturbation Responses utilizing GRN-Aligned Parameter Optimization", ISMB-ECCB 2025, Jul 20--24, Liverpool, UK (Full Paper Accepted, Oral Presentation, Bioinformatics, vol. 41, https://doi.org/10.1093/bioinformatics/btaf256)
4. Hajung Kim*, Mogan Gim*, Seungheun Baek, Soyon Park, Sunkyu Kim**, Jaewoo Kang**, "BADGER: biologically-aware interpretable differential gene expression ranking model", Bioinformatics Advances, vol. 5, https://doi.org/10.1093/bioadv/vbaf029
5. Seungheun Baek*, Soyon Park*, Yan Ting Chok, Junhyun Lee, Jueon Park, Mogan Gim**, Jaewoo Kang**, "Cradle-VAE: Enhancing Single-Cell Gene Perturbation Modeling with Counterfactual Reasoning-based Artifact Disentanglement", AAAI 2025, Feb 25--Mar 4, Philadelphia, USA (Full Paper Accepted, Poster Presentation), https://doi.org/10.1609/aaai.v39i15.33695

Predicting drug-food interactions using AI

Mogan Gim

Department of Biomedical Engineering, Hankuk University of Foreign Studies, South Korea

Drug-food interactions (DFIs) occur when dietary compounds alter the pharmacokinetic or pharmacodynamic properties of co-administered drugs, potentially leading to therapeutic failure or adverse drug events. Despite their clinical significance, the vast majority of DFIs remain uncharacterized due to the immense combinatorial space of possible drug-food pairs and the cost of experimental validation. This necessitates the development of computational approaches capable of efficiently screening and predicting novel interactions.

In this lecture, we review recent advances in AI-driven DFI prediction, beginning with early graph-based and feature-engineering approaches, and progressing to more biologically informed deep learning frameworks. We highlight ArcDFI, the first DFI prediction model to explicitly incorporate CYP450 isoenzyme interactions into its architecture through an attention regularization mechanism. By modeling compound-enzyme relationships via cross-attention and regularizing attention weights with known drug-CYP450 interaction data, ArcDFI demonstrated improved generalizability for unseen drugs and food compounds while providing interpretable predictions grounded in metabolic biology.

Building upon this foundation, we introduce the concept of multi-mechanistic DFI (mDFI) prediction, which moves beyond binary interaction classification to predict the specific biological mechanisms underlying each interaction. DFIs can be mediated through three principal categories: enzyme-mediated interactions (inhibition or induction of metabolic enzymes), transporter-mediated interactions (blocking or induction of membrane transporters), and pharmacodynamic interactions (synergism or antagonism at shared targets). We present a newly constructed mDFI dataset comprising over 5,000 drug-food compound pairs annotated with six interaction types and 36 protein mediators, along with the Mediated Interaction Attention Block (MIAB), an attention module designed to capture compound-mediator relationships across all mechanistic categories. We also discuss benchmark results evaluating state-of-the-art DDI and DFI models on this task, establishing the first performance baselines for multi-mechanistic DFI prediction. This lecture aims to provide both a comprehensive overview of the field and a forward-looking perspective on biologically grounded AI modeling for drug-food interactions.

CURRICULUM VITAE

신민정

고려대학교 바이오시스템의과학부 & ㈜메디엔진

[학력]

2005.02 연세대학교 대학원, 이학박사

[경력]

2009.03-현재 고려대학교 바이오시스템의과학부, 교수
2021.06-현재 ㈜메디엔진 대표이사

[관심분야]

대사성질환 연구 (From Bench to Bedside to Population)

[논문]

Scholarly output 163, Citation count 94,896, h-index 50
https://koreauniv.pure.elsevier.com/en/persons/min-jeong-shin

Shared metabolic switches in obesity treatment: from intermittent fasting to GLP1 based therapy

Min-Jeong Shin

School of Biosystems and Biomedical Sciences, Korea University; MEDI & GENE Inc.

Obesity is increasingly understood as a disorder of impaired energy homeostasis and reduced metabolic flexibility, rather than simply excess caloric intake. Both non-pharmacological interventions, including dietary restriction and intermittent fasting, and pharmacological approaches such as GLP1 based therapies can induce weight loss, but their long-term effectiveness is limited by physiological compensation.

Intermittent fasting promotes metabolic switching between fed and fasted states, enhancing fat oxidation and metabolic flexibility, yet is constrained by increased appetite and poor long-term adherence. In contrast, GLP1 based therapies effectively suppress appetite and reduce energy intake, but can introduce new therapeutic challenges, including lean mass loss and increased susceptibility to weight regain following discontinuation. These limitations suggest that the two approaches may be complementary.

In this presentation, we discuss an approach in which intermittent fasting-like metabolic switching is induced pharmacologically with GLP1 based therapy, while dietary strategies are used to support peripheral metabolic adaptation. This combined approach may provide a more sustainable framework for long-term weight management.

SoLA 2026

한국지질·동맥경화학회 춘계학술대회

2026 Spring Congress on
Lipid and Atherosclerosis of KSoLA

SoLA 2026

한국지질·동맥경화학회 춘계학술대회

2026 Spring Congress on
Lipid and Atherosclerosis of KSoLA

Symposium 3

Clinical Implication of Lipoprotein(a)

4월 3일(금) 12:00-13:20 | Room 3 (그랜드볼룸 1)

좌장 : 한기훈(울산의대 심장내과), 박성하(연세의대 심장내과)

패널 : 김웅(영남의대 심장내과), 김지현(가톨릭의대 순환기내과)
박세은(성균관의대 내분비내과)

12:00-12:16	**Decoding lipoprotein(a): genetics and metabolism insights** **이장훈**(경북의대 순환기내과)
12:16-12:32	**Lipoprotein(a) epidemiology: insights from Western and Korean populations** **장영우**(가천의대 심장내과)
12:32-12:48	**Unveiling the clinical impact of lipoprotein(a)** **주형준**(고려의대 순환기내과)
12:48-13:04	**Lipoprotein(a) as the next frontier in cardiovascular therapy** **위진**(가천의대 심장내과)
13:04-13:20	**Panel Discussion**

CURRICULUM VITAE

이장훈

경북의대 순환기내과

[학력]

1993.3.2-1999.2.25 경북대학교 의과대학 학사
2008.3.3-2011.2.25 경북대학교 의과대학 석사
2008.3.3-2011.2.25 경북대학교 의과대학 박사

[경력]

2020.10.1-현재 경북대학교병원 순환기내과 교수
2020.10.1-현재 대구경북 권역심뇌혈관센터 심혈관센터장

[관심분야]

Intervention cardiology

[논문]

1. Intravascular modality-guided versus angiography-guided percutaneous coronary intervention in acute myocardial infarction. Kim N, Lee JH, Jang SY, Bae MH, Yang DH, Park HS, Cho Y, Jeong MH, Park JS, Kim HS, Hur SH, Seong IW, Cho MC, Kim CJ, Chae SC; Korea Acute Myocardial Infarction Registry - National Institute of Health Investigators. Catheter Cardiovasc Interv. 2020 Mar 1;95(4):696-703. doi: 10.1002/ccd.28359. Epub 2019 May 27.
2. Usefulness of Calculation of Cardiovascular Risk Factors to Predict Outcomes in Patients With Acute Myocardial Infarction. Kim CY, Lee JH, Jang SY, Bae MH, Yang DH, Park HS, Cho Y, Jeong MH, Park JS, Kim HS, Hur SH, Seong IW, Cho MC, Kim CJ, Chae SC; Korea Acute Myocardial Infarction Registry - National Institute of Health Investigators. Am J Cardiol. 2019 Sep 15;124(6):857-863. doi: 10.1016/j.amjcard.2019.06.010. Epub 2019 Jun 25.
3. Coronary Endothelial Dysfunction and the Index of Microcirculatory Resistance as a Marker of Subsequent Development of Cardiac Allograft Vasculopathy. Lee JH, Okada K, Khush K, Kobayashi Y, Sinha S, Luikart H, Valantine H, Yeung AC, Honda Y, Fearon WF. Circulation. 2017 Mar 14;135(11):1093-1095. doi: 10.1161/CIRCULATIONAHA.116.025268. No abstract available.
4. Coronary Collaterals Function and Clinical Outcome Between Patients With Acute and Chronic Total Occlusion. Lee JH, Kim CY, Kim N, Jang SY, Bae MH, Yang DH, Cho Y, Chae SC, Park HS. JACC Cardiovasc Interv. 2017 Mar 27;10(6):585-593. doi: 10.1016/j.jcin.2016.12.009.
5. Pulling the RIPCORD: FFRCT to Improve Interpretation of Coronary CT Angiography. Fearon WF, Lee JH. JACC Cardiovasc Imaging. 2016 Oct;9(10):1195-1197. doi: 10.1016/j.jcmg.2016.01.037. Epub 2016 Aug 24.

Decoding lipoprotein(a): genetics and metabolism insights

Jang Hoon Lee

Department of cardiology, Kyungpook National University, South Korea

1. Introduction

While LDL-cholesterol (LDL-C) reduction remains the cornerstone of cardiovascular prevention, a significant "residual risk" persists. Lipoprotein(a) [Lp(a)] has emerged as a crucial, genetically determined risk factor. Unlike LDL-C, Lp(a) levels are largely resistant to lifestyle interventions and traditional statin therapy. This presentation aims to decode the complex genetic architecture of the *LPA* locus and the unique metabolic life cycle of Lp(a) that drives its pathogenicity.

2. Genetic Architecture: The *LPA* Locus and KIV-2

The plasma concentration of Lp(a) is 70% to >90% heritable, primarily determined by the *LPA* gene on chromosome 6 (6q26-27).

- Kringle IV Type 2 (KIV-2) Copy Number Variation: The *LPA* gene contains a highly polymorphic number of KIV-2 repeats, ranging from 2 to >40 copies.
- Inverse Correlation: There is a strong inverse relationship between the number of KIV-2 repeats and plasma Lp(a) concentration. Individuals with a low copy number (e.g., <22 repeats) express smaller apo(a) isoforms, which are processed more efficiently by hepatocytes, leading to 2- to 5-fold higher circulating Lp(a) levels compared to those with high copy numbers.
- Mendelian Randomization: Genetic studies confirm that a 100 mg/dL (approx. 200 nmol/L) increase in Lp(a) is associated with a 24-fold increase in the risk of myocardial infarction, establishing a causal link rather than a mere association.

3. Metabolic Insights: Synthesis and Clearance

Lp(a) metabolism deviates significantly from standard VLDL-LDL pathways:

- Hepatocyte Synthesis: The assembly of Lp(a) occurs in a two-step process where apo(a) and apoB-100 are linked by a single disulfide bond. This occurs primarily on the hepatic cell surface.
- Production Rate vs. Fractional Catabolic Rate (FCR): Clinical studies using stable isotope tracers show that plasma Lp(a) levels are predominantly driven by the hepatic production rate rather than the FCR.
- Clearance Challenges: While the LDL receptor (LDLR) plays a role, its affinity for Lp(a) is much lower than for LDL. Consequently, PCSK9 inhibitors only reduce Lp(a) by approximately 20% to 30%, whereas they reduce LDL-C by over 60%, highlighting the need for Lp(a)-specific therapies.

4. Conclusion

Decoding Lp(a) requires a shift from viewing it as a simple lipid parameter to understanding it as a complex genetic trait. With the advent of RNA-based therapeutics capable of reducing Lp(a) by over 90%, we are entering a new era where the residual risk posed by this "silent killer" can finally be addressed.

CURRICULUM VITAE

장영우(Youngwoo Jang)

가천대학교 길병원 조교수

[학력 및 경력]

2008-2012	School of Medicine, Gachon University, Doctor of Medicine
2017-2018	Cardiovascular Institute, Stanford Medicine, Postdoctoral Research Associate
2018-2019	Department of Cardiology, Gachon University, Gil Medical Center, Clinical Fellow
2019-2021	Department of Cardiology, Gachon University, Gil Medical Center, Clinical Assistant Professor
2022-	Department of Cardiology, Gachon University, Gil Medical Center, Assistant Professor

[학회활동]

2023-	심장대사증후군학회 학술간사
2025-	대한지질동맥경화학회 부총무/진료지침 간사

[관심 연구 분야]

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

[주요 논문 및 저서]

1. Jang Y, Lee JH, Lee SG, Jeong IK, Kim BJ. A Position Paper on Lipoprotein(a) from the Lipoprotein(a) Task Force Team of the Korean Society of Lipid and Atherosclerosis: Current Evidence, Clinical Applications, and Future Directions. Journal of Lipid Atherosclerosis and Korean Circ J. 2026 Jan;56(1):9-32.
2. Jang Y, Rhee EJ, Choi SH. Innovative Lipid-Lowering Strategies: RNA-Based, Small Molecule, and Protein-Based Therapies. Endocrinol Metab (Seoul) 2025;40:668-86.
3. Jang Y, Park SD, Lee JP, et al. One-month dual antiplatelet therapy followed by prasugrel monotherapy at a reduced dose: the 4D-ACS randomised trial. EuroIntervention 2025.
4. Kang SH, Pack KY, Kim JH, Jang Y (corresponding author). The effect of sarpogrelate compared to aspirin in high- or very-high-risk diabetes for primary prevention. Sci Rep. 2025 Jan 29;15(1):3616. doi: 10.1038/s41598-025-87868-x.
5. Kang SH, Lee J, Kim JH, Jang Y (corresponding author). Comparative Effectiveness of Clopidogrel Versus Aspirin for Primary Prevention in High-Risk Patients with Type 2 Diabetes: A Nationwide Propensity Score-Matched Cohort Study. Medicina (Kaunas) 2025;61.

Lipoprotein(a) epidemiology: insights from Western and Korean populations

Youngwoo Jang

Dept. Cardiology, Gachon University Gil Medical Center

Lipoprotein(a) [Lp(a)] is an established, genetically determined lipoprotein that confers residual cardiovascular risk independent of low-density lipoprotein cholesterol. Circulating Lp(a) levels show marked inter-individual and inter-ethnic variability, largely driven by differences in LPA gene kringle IV type 2 repeat polymorphisms. Consequently, the epidemiology and clinical implications of elevated Lp(a) differ substantially across populations.

In Western populations, approximately 20-25% of individuals have elevated Lp(a) levels above commonly used risk thresholds, and robust epidemiologic data consistently demonstrate strong associations between high Lp(a) and atherosclerotic cardiovascular disease, calcific aortic valve disease, and premature cardiovascular events. These findings have informed guideline recommendations for at least one-time Lp(a) measurement in adulthood and have driven the development of Lp(a)-lowering therapies.

In contrast, East Asian populations, including Koreans, generally exhibit lower median Lp(a) concentrations and a different distribution of high-risk thresholds. However, recent Korean epidemiologic studies indicate that elevated Lp(a), even at lower absolute levels than in Western cohorts, is independently associated with coronary artery disease, ischemic stroke, and adverse cardiovascular outcomes. Importantly, the relative risk associated with high Lp(a) appears comparable to that observed in Western populations, highlighting the clinical relevance of Lp(a) across ethnicities.

This lecture will review key epidemiologic findings on Lp(a) from Western and Korean datasets, compare population distributions and risk thresholds, and discuss the implications for screening strategies and future risk stratification in the Korean clinical setting.

CURRICULUM VITAE

주형준

고대안암병원 순환기내과

[학력]

1007-2003	고려대학교 의과대학 의학과 학사
2005-2007	고려대학교 의과대하 의학과 석사
2008-2011	한국과학기술원 의과학과 박사

[경력]

2004-2008	고려대학교 의료원 수련의 및 내과전공의
2011-2013	한국과학기술원 박사후 연구원
2021-현재	고려대학교 의과대학 의료정보학교실 겸무교수

[관심분야]

심혈관학, 고혈압, 이상지질혈증, 정보의학

[논문]

1. Sex Differences in Blood Pressure Variability, Office-Home Difference, and Hypertension-Mediated Organ Damage: A Multicenter Analysis. Cha JJ, Oh SW, Cho DH, Kim MN, Park JH, Cho KH, Shin SY, Kim EJ, Joo HJ. Am J Hypertens. 2026 Apr 1;39(4):505-513. doi: 10.1093/ajh/hpaf212.PMID: 41128498
2. Prognostic significance of diastolic hypotensive episodes in patients stabilized after acute heart failure. Joo HJ, Hong SJ, Yu CW, Shin SY, Kim EJ. Am J Med. 2025 Dec;138(12):1680-1688.e4. doi: 10.1016/j.amjmed.2025.06.036. Epub 2025 Jun 28.PMID: 40588056
3. Domain and Language adaptive pre-training of BERT models for Korean-English bilingual clinical text analysis. Jo E, Cho E, Lee Y, Song S, Joo HJ. BMC Med Inform Decis Mak. 2025 Nov 25;25(1):428. doi: 10.1186/s12911-025-03262-7.PMID: 41291648
4. Large Language Model and Knowledge Graph-Driven AJCC Staging of Prostate Cancer Using Pathology Reports. Jo E, Noh TI, Joo HJ. Diagnostics (Basel). 2025 Sep 27;15(19):2474. doi: 10.3390/diagnostics15192474.PMID: 41095693
5. Predictors of lipoprotein(a) variability in clinical practice and their impact on cardiovascular risk. Joo HJ, Yun SG, Park JH, Hong SJ, Yu CW, Shin SY, Kim EJ. Lipids Health Dis. 2025 Jul 23;24(1):250. doi: 10.1186/s12944-025-02666-8.

Unveiling the clinical impact of lipoprotein(a)

Hyung Joon Joo

Department of Cardiology, Korea University Anam Hospital, South Korea

Lipoprotein(a) [Lp(a)] has emerged as a causal, independent risk factor for atherosclerotic cardiovascular disease and calcific aortic valve disease, affecting a substantial proportion of the global population. Large-scale epidemiologic studies and Mendelian randomization data consistently demonstrate a continuous, dose-dependent relationship between Lp(a) concentration and cardiovascular event risk. Current international guidelines recommend at least once-in-a-lifetime Lp(a) measurement, with thresholds of 30 mg/dL and 50 mg/dL widely adopted. However, these cutoffs were largely derived from Western populations, and their applicability to East Asians—who characteristically exhibit lower median Lp(a) concentrations—remains uncertain. Furthermore, the traditional view of Lp(a) as a genetically fixed, temporally stable biomarker is increasingly challenged by evidence of clinically meaningful intra-individual variability and phenotypic heterogeneity.

This presentation includes a series of multicenter studies from three Korea University hospitals, advancing our understanding of Lp(a) across four complementary dimensions. First, in a large Korean patient cohort, a population-specific Lp(a) threshold for predicting major adverse cardiovascular events (MACE) was identified at a level substantially lower than current guideline-recommended cutoffs, consistent with recent proposals for ethnicity-specific thresholds in Asian populations. Second, among patients with serial Lp(a) measurements, a meaningful proportion demonstrated high intra-individual variability associated with adverse cardiometabolic profiles. Notably, more than half of patients in the intermediate "gray-zone" range were reclassified into a different risk category upon repeat measurement, highlighting the importance of serial testing in borderline cases. Third, high longitudinal Lp(a) variability was independently associated with MACE even after adjustment for baseline risk categories, traditional risk factors, and lipid-lowering therapies—suggesting that temporal fluctuation captures a dynamic dimension of residual risk beyond static measurement alone. Fourth, unsupervised clustering among patients with elevated Lp(a) revealed two phenotypically distinct subgroups with markedly different MACE rates despite comparable Lp(a) concentrations, underscoring that Lp(a) level alone is insufficient to characterize cardiovascular risk.

As targeted Lp(a)-lowering therapies approach clinical availability through ongoing late-phase outcome trials, identifying which patients will derive the greatest benefit becomes critical. Our findings collectively advocate for a multidimensional strategy, incorporating population-specific thresholds, selective repeat measurement, and phenotype-guided risk stratification, to optimize therapeutic decision-making in the emerging era of precision Lp(a) management.

CURRICULUM VITAE

위진

가천대 길병원 심장내과

[학력]

2005	연세대학교 의학과 학사
2012	연세대학교 대학원 의학과 석사
2017	연세대학교 대학원 의학과 박사

[경력]

2005-2010	연세대학교 세브란스병원 인턴, 내과 전공의
2010-2013	연세대학교 세브란스병원 심장내과 강사
2013-2019	연세대학교 세브란스병원 심장내과 임상조교수
2019-	가천대 길병원 심장내과 교수

[관심분야]

중증심부전, ECMO, 급성심근경색, 급성심정지, 동맥경화

[논문]

1. Population Pharmacokinetics of Ticagrelor During Veno-Arterial ECMO in Acute Coronary Syndrome: Model-Informed Dosing Simulations. Clinical Pharmacology & Therapeutics. In Press (IF 6.903)
2. Effect of Extracorporeal Membrane Oxygenation Flow Rate on Midazolam Clearance: A Population Pharmacokinetic Study. Anesthesiology 2025;144:485-488 (IF 9.20)
3. Dose Optimization of Meropenem in Patients on Veno-Arterial Extracorporeal Membrane Oxygenation in Critically Ill Cardiac Patients: Pharmacokinetic/Pharmacodynamic Modeling. J Clin Med 2022;11:6621 (IF 5.098)
4. Population pharmacokinetics and dosing optimization of piperacillin/tazobactam in critically ill patients on extracorporeal membrane oxygenation and the influence of concomitant renal replacement therapy. Microbiology Spectrum 2021;9:e0063321 (IF 9.043)
5. Dose Optimization of Cefpirome Based on Population Pharmacokinetics and Target Attainment during Extracorporeal Membrane Oxygenation. Antimicrob Agents Chemother 2020;64:e00249-20 (IF 5.938)

Lipoprotein(a) as the next frontier in cardiovascular therapy

Jin Wi

Division of Cardiology, Gachon University Gil Medical Center, Korea

Despite the successful management of LDL-C with traditional therapies like statins, many patients still face significant cardiovascular events. This persistent "residual risk" has shifted the clinical focus toward Lipoprotein(a) [Lp(a)], an independent and genetically determined risk factor that remains largely unaddressed by current standard-of-care treatments.

Lp(a) is a unique lipoprotein consisting of an LDL-like particle with Apolipoprotein(a) covalently bound to Apolipoprotein B-100. Its pathogenicity is multifaceted, including atherosclerosis, thrombosis, and inflammation.

Unlike LDL-C, which is highly influenced by diet, Lp(a) levels are 80-90% determined by genetics (LPA gene). Consequently, lifestyle changes or statins have minimal impact on its concentration. Clinical guidelines now increasingly recommend that every adult should have their Lp(a) level measured at least once to accurately stratify their lifetime cardiovascular risk.

The "frontier" of therapy lies in precision medicine, specifically targeting the production of Lp(a) at the genetic level. Novel antisense oligonucleotides (e.g., Pelacarsen) and siRNAs (e.g., Olpasiran) have demonstrated the ability to reduce Lp(a) levels by 80% to over 90% in clinical trials. Oral small molecule inhibitors like Muvalaplin are being developed to disrupt the assembly of the Lp(a) particle.

In this lecture, I would like to deal with Lp(a) as the next frontier in cardiovascular therapy and review the future possible clinical applications in detail.

SoLA 2026

한국지질·동맥경화학회 춘계학술대회

2026 Spring Congress on
Lipid and Atherosclerosis of KSoLA

SoLA 2026

한국지질·동맥경화학회 춘계학술대회

2026 Spring Congress on
Lipid and Atherosclerosis of KSoLA

Symposium 4

Beyond LDL: Targeting Triglyceride-rich Lipoproteins for Residual Cardiovascular Risk

4월 3일(금) 13:50-15:20 | Room 1 (그랜드볼룸 3)

좌장 : 김성래(가톨릭의대 내분비내과), 이우제(울산의대 내분비내과)

패널 : 김지윤(성균관의대 내분비대사내과), 류영상(조선의대 내분비대사내과)
박용현(부산의대 순환기내과)

13:50-14:08	**TG-rich lipoproteins and residual cardiovascular risk: genetic and mechanistic insights** **김병진**(성균관의대 순환기내과)
14:08-14:26	**Omega-3 fatty acids: from triglyceride reduction to cardiovascular outcomes** **임수**(서울의대 내분비대사내과)
14:26-14:44	**APOC3 metabolism and therapeutic approaches** **김남훈**(고려의대 내분비내과)
14:44-15:02	**ANGPTL3/4/8 inhibition: translational insights and clinical development** **정혜문**(경희의대 심장내과)
15:02-15:20	**Panel Discussion**

CURRICULUM VITAE

김병진

성균관의대 강북삼성병원 순환기내과

[학력]

1993	부산의대 졸업
1996	부산의대 석사
2003	부산의대 박사

[경력]

2001-2003.02	성균관의대 삼성서울병원 순환기내과 임상강사
2003-	성균관의대 강북삼성병원 순한기내과 조교수, 부교수, 정교수
2007-2008	Dept. of Epidemiology and Preventive Medicine, UCSD, CA, USA

[관심분야]

Lipoprotein(a), Remnant cholesterol, Primary Prevention, Smoking, Epicardial fat

[논문]

1. A Position Paper on Lipoprotein(a) from the Lipoprotein(a) Task Force of the Korean Society of Lipid and Atherosclerosis; Current Evidence, Clinical Applications, and Future Directions. Korean Circ J 2026;56:9-32; J Lipid Atherosclerosis 2026;15:2-25.
2. Remnant cholesterol and cardiovascular and all-cause mortality in Korean adults. J Clin Lipidol 2025;19:477-485.
3. Association of lipoprotein(a) and coronary artery calcium with atherosclerotic cardiovascular disease. J Clin Lipidol 2025;19:521-530.
4. Associations of visit-to-visit lipid variability with coronary artery calcification and cardiovascular event in statin-naïve Koreans. Eur J Prev Cardiol 2025;32:1247-1256.
5. Lipoprotein(a)-related cardiovascular and all-cause mortalities in Korean adults. Eur J Prev Cardiol 2023;30:308-317.

TG-rich lipoproteins and residual cardiovascular risk: genetic and mechanistic insights

Byung Jin Kim

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

Despite substantial reductions in low-density lipoprotein cholesterol (LDL-C) with contemporary lipid-lowering therapies, a considerable residual risk of atherosclerotic cardiovascular disease (ASCVD) persists. Triglyceride-rich lipoprotein (TRLs) and their remnants have emerged as key contributors to this residual risk. TRLs, including very-low density lipoproteins (VLDL) and intermediate-density lipoprotein (IDL), and their cholesterol content, defined as remnant cholesterol (RC), represent atherogenic apolipoprotein B-containing particles distinct from LDL.

Genetic studies provide strong evidence supporting a causal role of TRLs and remnant cholesterol in ASCVD. Variants affecting lipoprotein lipase (LPL), apolipoprotein C-III (APOC3), and angiopoietin-like proteins (ANGPTL3/4) consistently demonstrate that lifelong reductions in TRL levels are associated with lower cardiovascular risk.

Mechanistically, TRL remnants are highly cholesterol-enriched apoB-containing particles that can penetrate and be retained within the arterial wall. Compared with LDL, they carry a greater cholesterol load per particle and contribute directly to foam cell formation, endothelial dysfunction, vascular inflammation, and prothrombotic pathways.

Epidemiological and clinical data consistently show that elevated triglycerides and remnant cholesterol are robust markers of cardiovascular risk, even in patients with well-controlled LDL-C levels. However, randomized controlled trials specifically targeting remnant cholesterol are lacking, and triglyceride-lowering strategies have yielded inconsistent outcomes, particularly when not accompanied by reductions in apolipoprotein B.

In summary, TRLs represent a key causal component of residual cardiovascular risk beyond LDL-C. These findings support the need for improved risk assessment and the development of therapeutic strategies targeting apoB-containing TRL remnants to reduce ASCVD risk.

CURRICULUM VITAE

임수(Soo Lim)

서울대학교 의과대학 분당서울대학교병원 내분비대사내과

[학력]

1990.3-1996.2	서울대학교 의과대학 의학과 학사
2002.3-2004.2	서울대학교 보건대학원 보건학과 석사
2004.3-2006.2	서울대학교 의과대학 내과학 박사
2006.3-2009.2	서울대학교 보건대학원 보건학과 박사

[경력]

1996.3-1997.2	서울대학교병원 인턴 과정 수료
1997.3-2001.2	서울대학교병원 내과전공의 과정 수료
2004.5-2005.7	서울대학교병원 내과 임상강사 (내분비내과)
2007.9-현재	서울의대 분당서울대학교병원 내과 교수
2011.2-2012.8	하버드의대 부속 Massachusetts General Hospital 연수 (Research Fellow)

[관심분야]

비만, 당뇨, 고지혈증 및 대사질환

[논문]

1. Lim S*, Ji L, Tham KW, Misra A, Kadowaki T* (Co-corresponding). Clinical Obesity for Asian People: Bridging the Gap between Adiposity and Disease. Nature Reviews Endocrinology. 2026 [Accepted].
2. Seo JI, Koh A, Lim S (Co-corresponding)*, Yoo HH*, The 3M Roles of the Gut Microbiome in Pharmacotherapy for Diabetes: Mediator, Modifier, Marker. Trends Endocrinol Metab. 2026 [Accepted].
3. Son JW, le Roux CW, Blüher M, Nauck MA, Lim S. Novel GLP-1-Based Medications for Type 2 Diabetes and Obesity. Endocrine Reviews 2025 Oct 7:bnaf036. doi: 10.1210/endrev/bnaf036. Online ahead of print. PMID: 41054801.
4. Lee Y, Lim S* (co-corresponding), Davies MJ*. Cardiometabolic and Renal Benefits of SGLT2 Inhibitors: Mechanisms and Clinical Implications. Nature Reviews Endocrinology. 2025 Dec;21(12):783-798 PMID: 40935880.
5. Lim S (first and corresponding author), Buranapin S, Bao X, Quiroga M, Park KH, Kang JH, Rinnov AR, Suwanagool A. Once-weekly semaglutide 2.4 mg in an Asian population with obesity, defined locally as BMI ≥25 kg/m2 (STEP 11). Lancet Diabetes Endocrinol. 2025 Oct;13(10):838-847. PMID: 40825340.

Omega-3 fatty acids: from triglyceride reduction to cardiovascular outcomes

Soo Lim

Division of Endocrinology and Metabolism, Department of Internal Medicine,
Seoul National University Bundang Hospital, Seoul National University College of Medicine, Seoul, South Korea

Omega-3 fatty acids (ω-3 FAs), particularly eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA), have emerged as important modulators of triglyceride metabolism and cardiovascular risk. Long-chain ω-3 FAs reduce circulating triglycerides by inhibiting hepatic VLDL synthesis and enhancing lipoprotein lipase-mediated clearance. Beyond lipid lowering, ω-3 FAs exert pleiotropic effects, including anti-inflammatory, vascular-protective, and antithrombotic actions, mediated in part via incorporation into cell membranes and production of specialized proresolving mediators. Randomized controlled trials demonstrate that high-dose EPA alone significantly reduces major adverse cardiovascular events, whereas combination EPA/DHA formulations show mixed results, highlighting the importance of compound specificity, dose, and patient population. In the context of obesity and atherogenic dyslipidemia, ω-3 FAs may mitigate residual cardiovascular risk by addressing triglyceride-rich lipoproteins and ectopic fat-related metabolic disturbances. Integrating ω-3 FA therapy into comprehensive cardiometabolic management, alongside lifestyle modification and pharmacotherapy, offers a mechanistically informed approach to improving cardiovascular outcomes in high-risk populations. Future studies are needed to clarify differential effects of EPA versus DHA and to optimize patient-specific treatment strategies.

CURRICULUM VITAE

김남훈(Nam Hoon Kim)

고려대학교 의과대학, 고려대학교 안암병원

[약력]

1997-2004	고려대학교 의과대학 학사
2007-2009	고려대학교 의과대학 석사
2010-2015	고려대학교 의과대학 박사
2012-2014	고려대학교 안암병원 내분비내과 임상강사
2014-2016	고려대학교 안암병원 내분비내과 임상조교수
2016-2018	고려대학교 안암병원 내분비내과 조교수
2019-2020	University of California San Diego, Visiting Scholar
2018-2023	고려대학교 안암병원 내분비내과 부교수
2023-현재	고려대학교 안암병원 내분비내과 교수

[관심분야]

Lipid metabolism, Diabetes therapeutics, Obesity, Diabetic kidney disease, Young-onset type 2 diabetes

[학회활동]

2026-	대한당뇨병학회 당뇨병특성화TF 팀장
2026-	대한당뇨병학회 학술위원회 위원
2026-	대한당뇨병학회 간행위원회 위원
2026-	심장대사증후군학회 학술위원회 위원
2025-	대한내분비학회 미래위원회 이사
2025-	한국지질동맥경화학회 간행위원회 위원
2025-	한국지질동맥경화학회 교육위원회 위원

[수상경력]

2018	대한내분비학회 신진연구자상
2020	대한당뇨병학회 Young Investigator award
2024	대한내분비학회 향설 젊은 연구자상
2025	한국지질동맥경화학회 학술상
2023, 2024, 2025	고려대학교 석탑연구상

APOC3 metabolism and therapeutic approaches

Nam Hoon Kim

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

Apolipoprotein CIII (APOC3) has emerged as a central regulator of triglyceride rich lipoprotein metabolism and a promising therapeutic target for residual cardiovascular risk. APOC3 inhibits lipoprotein lipase activity and delays hepatic clearance of triglyceride rich particles, thereby promoting hypertriglyceridemia and the accumulation of atherogenic remnant lipoproteins. Genetic studies have consistently demonstrated that loss of function variants in APOC3 are associated with lower triglyceride levels and reduced risk of atherosclerotic cardiovascular disease, supporting a causal role of APOC3 in atherogenesis.

Recent advances have refined our understanding of APOC3 metabolism, highlighting its dynamic regulation by nutritional status, insulin signaling, and hepatic lipid flux. In insulin resistant states, increased APOC3 expression contributes to impaired triglyceride clearance and exacerbates dyslipidemia, particularly in individuals with type 2 diabetes and metabolic syndrome. Beyond its metabolic effects, APOC3 may also exert direct pro inflammatory and pro-atherogenic actions on vascular cells.

Therapeutic targeting of APOC3 has rapidly progressed with the development of antisense oligonucleotides and small interfering RNA based approaches. Agents such as volanesorsen, olezarsen, and plozasiran have demonstrated substantial triglyceride lowering effects, often exceeding 60 to 80 percent reductions, along with favorable effects on remnant cholesterol. These therapies have shown particular promise in patients with severe hypertriglyceridemia and familial chylomicronemia syndrome, with ongoing studies evaluating their impact on cardiovascular outcomes.

This lecture will provide an integrated overview of APOC3 biology, its role in triglyceride metabolism and atherosclerosis, and the current landscape of APOC3 targeted therapies.

CURRICULUM VITAE

정혜문

경희의대 심장내과

[학력]

2008	연세대학교 의학과 학사
2013	연세대학교대학원 내과학 석사
2018	연세대학교대학원 내과학 박사

[경력]

2009-2012	강남세브란스병원 내과전공의
2013-2014	연세의료원 심장내과 전임의
2016-2019	경희의료원 임상조교수
2020-현재	경희대학교 의과대학 심장내과 조교수

[관심분야]

심부전, 심장영상

[논문]

1. Age-Dependent Role of Genetics and Renal Function for Atrial Fibrillation Development in Hypertrophic Cardiomyopathy. Korean Circ J. 2025 Nov;55(11):1001-1013.
2. Determinants and effects of microvascular obstruction on serial change in left ventricular diastolic function after reperfused acute myocardial infarction. Front Cardiovasc Med. 2024 Apr 26;11:1338940.
3. Multimodality Imaging in Patients with Hypertrophic Cardiomyopathy and Atrial Fibrillation. Diagnostics (Basel). 2023 Sep 25;13(19):3049.
4. Arterial Stiffness is Associated with False-Positive ST-Segment Depression in Supine Bicycle Exercise Stress Echocardiography. Rev Cardiovasc Med. 2023 Feb 6;24(2):47.
5. Emerging Indicators of Left Atrial Function Evaluation Considering the Unique Characteristics of Hypertrophic Cardiomyopathy. J Cardiovasc Imaging. 2023 Jan;31(1):49-50.

ANGPTL3/4/8 inhibition: translational insights and clinical development

정혜문

경희의대 심장내과

SoLA 2026

한국지질·동맥경화학회 춘계학술대회

2026 Spring Congress on
Lipid and Atherosclerosis of KSoLA

SoLA 2026

한국지질·동맥경화학회 춘계학술대회

2026 Spring Congress on
Lipid and Atherosclerosis of KSoLA

Symposium 5

CVD Risk Prediction in the Context of Clinical Practice Guidance

4월 3일(금) 13:50-15:20 | Room 2 (그랜드볼룸 2)

좌장 : 편욱범(이화의대 순환기내과), 김현창(연세의대 예방의학교실)

패널 : 강선미(순천향의대 내분비대사내과), 박광열(중앙의대 신경과)
박상우(울산의대 심장내과)

13:50-14:08	**CVD risk prediction in lipid guidelines** **김은지**(가천의대 예방의학과)
14:08-14:26	**CVD risk prediction in diabetes guidelines** **구유정**(서울의대 내분비대사내과)
14:26-14:44	**CVD risk prediction in hypertension guidelines** **천대영**(한림의대 순환기내과)
14:44-15:02	**CVD risk prediction in the context of CKM health** **이호규**(연세의대 예방의학과)
15:02-15:20	**Panel Discussion**

CURRICULUM VITAE

김은지

가천의대 예방의학과

[학력]

2021-2024	연세대학교 의과대학, 의학 박사(PhD), 예방의학
2018-2019	University College London, MSc, Global Health and Development
2011-2015	동국대학교 의학전문대학원, 의무석사(MD), 의학

[경력]

2024-현재	가천의대 예방의학, 조교수
2021-2024	연세의대 예방의학, 전공의
2016-2017	연세의대, 인턴

[관심분야]

만성질환, 예방관리, Social determinant of health, 역학

[논문]

1. Kwon J, Shin H, Lee DH, Kim E. Trends in Prevalence of Metabolic Dysfunction-Associated Steatotic Liver Disease: A Nationwide Survey in Korea. Endocrinol Metab (Seoul). 2025 Dec 24.
2. Kim E, Hong J, Shin H, Moon JY, Sunwoo W. Prophylactic antibiotic use in closed basilar skull fractures: A nationwide cohort study. J Trauma Acute Care Surg. 2026 Jan 1;100(1):97-104.
3. Kim E, Lee HH, Kim EJ, Cho SMJ, Kim HC, Lee H. Factors associated with medication adherence among young adults with hypertension. Clin Hypertens. 2025 May 1;31:e18.
4. Kwon J, Kim E. Lifelong impact of elevated blood pressure from childhood to adulthood. Clin Exp Pediatr. 2025 Apr;68(4):278-286.
5. Lim YS, Kim E, Choi WS, Yang HJ, Moon JY, Jang JH, Cho J, Choi J, Woo JH. Non-Contrast Computed Tomography-Based Triage and Notification for Large Vessel Occlusion Stroke: A Before and After Study Utilizing Artificial Intelligence on Treatment Times and Outcomes. J Clin Med. 2025 Feb 15;14(4):1281.

CVD risk prediction in lipid guidelines

Eunji Kim

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

Recent lipid guidelines have increasingly emphasized treatment decisions based on individualized cardiovascular disease (CVD) risk assessment rather than LDL-C levels alone. This approach reflects evidence that the absolute benefit of lipid-lowering therapy depends on a patient's baseline risk of atherosclerotic CVD, particularly in primary prevention settings. The 2026 American Heart Association / American College of Cardiology guideline introduces the PREVENT risk equation as an updated approach to risk estimation, incorporating a broader range of clinical variables to support risk estimation across contemporary populations. The guideline emphasizes risk estimation as a central step in guiding the initiation and intensity of lipid-lowering therapy. In parallel, the European Society of Cardiology guideline updates recommendations for cardiovascular risk estimation, with the implementation of the SCORE2 and SCORE2-OP risk prediction algorithms. These models incorporate regional calibration and age-specific risk estimation and extend beyond traditional 10-year risk assessment to include longer-term risk considerations. In current guidelines, estimated CVD risk informs decisions on treatment initiation, treatment intensity, and the need for additional risk refinement using clinical factors or imaging. This presentation summarizes how CVD risk assessment is integrated into treatment decision-making in recent lipid guidelines.

CURRICULUM VITAE

구유정

서울대학교병원 강남센터 내분비대사내과

[학력]

2008	경북대학교 의과대학 졸업
2013	서울대학교 의과대학 임상의과학과 석사
2021	서울대학교 의과대학 중개의학과 박사

[경력]

2023.8-현재	서울대학교병원 강남센터 부교수
2015.3-2023.7	충북대학교병원 내분비내과 진료임상기금교수
2013.3-2015.2	분당서울대학교병원 내분비내과 임상강사
2009.3-2013.2	서울대학교병원 내과 전공의

[관심분야]

당뇨병, 이상지질혈증, 비만, 심혈관 및 대사질환

[논문]

1. Ku EJ, et al. Deep Learning-Based Adrenal Gland Volumetry for the Prediction of Diabetes. Endocrinol Metab (Seoul). 2025;40(6):991-1001.
2. Ku EJ, et al. Long-term Nonskeletal Complications in Patients With Thyroid Cancer and Hypoparathyroidism Post Total Thyroidectomy. J Clin Endocrinol Metab. 2025;110(12):3538-3545.
3. Ku EJ, et al. Fenofibrate to prevent amputation and reduce vascular complications in patients with diabetes: FENO-PREVENT. Cardiovasc Diabetol. 2024;23(1):329.
4. Yoo WS and Ku EJ, et al. Incidence of Endocrine-Related Dysfunction in Patients Treated with New Immune Checkpoint Inhibitors: A Meta-Analysis and Comprehensive Review. Endocrinol Metab (Seoul). 2023 Dec;38(6):750-759.
5. Ku EJ, et al. Long-Term Effectiveness of Quadruple Combination Therapy with Empagliflozin Versus Basal Long-Acting Insulin Therapy in Patients with Type 2 Diabetes: 3-Year Retrospective Observational Study. Diabetes Ther. 2023 Sep;14(9):1471-1479.

CVD risk prediction in diabetes guidelines

Eu Jeong Ku

Division of Endocrinology and Metabolism, Department of Internal Medicine,
Seoul National University Hospital Healthcare System Gangnam Center, South Korea

Cardiovascular disease (CVD) remains the leading cause of morbidity and mortality in patients with diabetes mellitus. While diabetes was historically simplified as a CVD risk equivalent, the paradigm has shifted toward a more individualized, nuanced approach to risk stratification based on each patient's clinical profile.

This lecture reviews the latest trends in CVD risk prediction strategies across major international and domestic clinical guidelines, comparing their frameworks and practical implications for real-world clinical practice. Major guidelines commonly stratify patients into moderate, high, and very high-risk categories based on the presence of established atherosclerotic cardiovascular disease, target organ damage, duration of diabetes, and comorbid conditions. This risk stratification directly informs key therapeutic decisions, including the intensity of lipid-lowering therapy, selection of antihyperglycemic agents with proven cardiovascular benefit, and blood pressure treatment targets. However, discrepancies remain among guidelines in defining risk categories and treatment thresholds. Furthermore, conventional risk prediction tools have well-recognized limitations in adequately capturing diabetes-specific cardiovascular risk. In response, refined risk stratification strategies incorporating markers of subclinical atherosclerosis, such as the coronary artery calcium (CAC) score, and emerging biomarkers are gaining increasing attention.

This presentation will examine the current landscape and limitations of CVD risk prediction tools from a clinical application perspective, discuss approaches to integrating risk stratification with therapeutic decision-making, and highlight unmet needs and future directions for improving CVD prevention in patients with diabetes.

CURRICULUM VITAE

천대영

한림대학교 동탄성심병원 순환기내과

[학력]

2006-2012 한림대학교 의과대학 학사
2018-2023 한국방송통신대학교 바이오정보.통계학과 석사

[경력]

2012-2017 한림대학교성심병원 인턴, 레지던트
2020-2021 서울대학교 순환기내과 임상강사
2021- 한림대학교 동탄성심병원 임상강사, 임상조교수, (현) 조교수

[관심분야]

심혈관계 중재시술, 고혈압, 죽상동맥경화, 빅데이터 연구

[논문]

1. Dementia risk across blood pressure categories: a South Korean nationwide study, European Heart Journal, 2025.10 (Co-Author)
2. Cardiovascular risk across blood pressure categories defined by the 2024 ESC and 2023 ESH hypertension guidelines: insights from a Korean nationwide cohort study, European Journal of Preventive Cardiology, 2025.9 (1st author)
3. Association between Metabolic Syndrome and Young-Onset Dementia: A Nationwide Population-Based Study. Neurology, 2025.4 (Co-corresponding author)
4. Diabetes status, duration and risk of dementia in patients with myocardial infarction, Diabetology & Metabolic Syndrome, 2025.7 (Co-1st author)
5. Depression and Risk of Stroke and Mortality after Percutaneous Coronary Intervention: A Nationwide Population Study, Journal of Internal Medicine, 2024.09 (Co-1st Author)

CVD risk prediction in hypertension guidelines

천대영

한림대학교 동탄성심병원 순환기내과

심혈관질환(CVD) 위험도 예측은 예방심장학과 고혈압 진료의 핵심 축이며, 최근 주요 가이드라인은 위험도 평가의 중요성을 공통적으로 강조하고 있다. 2025 ACC/AHA 고혈압 가이드라인은 PREVENT equation을 활용한 위험기반 접근을 통해 혈압 치료 결정을 보다 정교화하였고, 2024 ESC 가이드라인은 elevated blood pressure와 hypertension의 분류 및 관리 전략을 새롭게 정비하였다. 2023 ESH 가이드라인은 전통적 혈압 분류를 유지하면서도 표적장기손상과 동반질환을 포함한 총 심혈관 위험 평가를 강조하였으며, 2022 KSH 가이드라인은 한국 임상 환경에서의 정확한 혈압 측정과 고위험군 중심의 목표 혈압 설정을 제시하였다. 본 발표에서는 각 가이드라인이 CVD 위험 예측을 어떠한 방식으로 임상 의사결정에 연결하는지 비교하고, 실제 진료에서의 적용점과 한계를 간략히 고찰하고자 한다.

CURRICULUM VITAE

이호규

연세의대 예방의학교실

[학력]

2004-2008	미국 UC Berkeley 졸업 (BS)
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, 조교수
2024-현재	연세대학교 의과대학 예방의학교실 부교수
현재	한국지질동맥경화학회 학술위원, 진료지침위원
현재	대한고혈압학회 연구이사, 역학연구회 부회장, 진료지침위원
현재	대한심뇌혈관질환예방학회 특임이사, 학술간사
현재	미국심장협회(AHA) Global CKM Committee 위원, 고혈압 분과 학술위원
현재	Circulation 부편집인(Associate Editor)

[관심분야]

예방심장학, 임상역학

[논문]

1. Lee H, Huang X, Khan SS, Son D, Lee HH, Kim EJ, Lloyd-Jones DM, Kim HC, Greenland P. Very High Prevalence of Non-Optimally Controlled Traditional Risk Factors At the Onset of Cardiovascular Disease. J Am Coll Cardiol 2025; 86(14):1017-1029.
2. SCORE2 Asia-Pacific Writing Group on behalf of ESC/EAPC/AFC/APSC. Risk prediction of cardiovascular disease in the Asia-Pacific region: the SCORE2 Asia-Pacific model. Eur Heart J 2025;46(8):702-715.
3. 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.
4. 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.
5. Lee H, Yano Y, Cho SMJ, Lee HH, Kim DW, Lloyd-Jones DM, Kim HC. Associations of Ideal Cardiovascular Health and Its Change During Young Adulthood With Premature Cardiovascular Events: A Nationwide Cohort Study. Circulation 2021;144(1):90-92.

CVD risk prediction in the context of CKM health

이호규

연세의대 예방의학과

SoLA 2026

한국지질·동맥경화학회 춘계학술대회

2026 Spring Congress on
Lipid and Atherosclerosis of KSoLA

SoLA 2026

한국지질·동맥경화학회 춘계학술대회

2026 Spring Congress on
Lipid and Atherosclerosis of KSoLA

Symposium 6

Mis- or Disinformation of Statin Therapy in Real World Cardiometabolic Area

4월 3일(금) 13:50-15:20 | Room 3 (그랜드볼룸 1)

좌장 : 이준희(한림의대 심장혈관내과), 이상록(전북의대 심장내과)

패널 : 노은(서울의대 내분비대사내과), 박성민(동아일보)
안지현(한국의학연구소 내과), 이재광(창원파티마병원 심장내과)

13:50-14:08	**이상지질혈증 치료 개시 및 유지의 어려움** **김민정**(이화의대 순환기내과)
14:08-14:26	**이상지질혈증과 동맥경화치료에 있어서 주요 가짜 정보의 유형 (매체에서 보이는 유형 정리)** **김대영**(인하의대 심장내과)
14:26-14:44	**가짜 정보에 의한 환자와 의료진의 피해 현황 (증례와 의료정보위원회 설문조사)** **이봄**(차의대 심장내과)
14:44-15:02	**이상지질혈증과 동맥경화 치료 관련 가짜 정보 등에 대한 학회 차원의 대응 대책** **박상민**(을지의대 심장내과)
15:02-15:20	**Panel Discussion**

CURRICULUM VITAE

김민정

이화여대 목동병원

[학력]

2011-2015	M/S, Gachon University College of Medicine
2006-2011	B/S, Division of Biotechnology, College of Life Science and Biotechnology, Korea Universit

[경력]

2025-Present	Clinical Assistant Professor, Division of Cardiology, Ewha Woman's University Mokdong Hospital
2022-2025	Clinical Assistant Professor, Division of Cardiology, Myongji Cardiovascular Center, Myongji Hospital, Hanyang University College of Medicine
2020-2022	Fellowship, Severance Hospital, Yonsei University

[관심분야]

심근병증, 유전체학, 심부전, 고지혈증

[논문]

1. Aortic Stenosis in End-Stage Renal Disease: Incidence, Prevalence, and Mortality in a National Korean Cohort. J.Clinical Med. 2025.
2. Risk of retinal vascular occlusive disease according to type and low-density lipoprotein control after acute coronary syndrome. YMJ. 2025.
3. Risk of Retinal Vascular Occlusive Disease in Patients with Aortic Stenosis: A Nationwide Korean Cohort Study. Ophthalmology science. 2025.
4. Effects of genetic mutations on left ventricular myocardial mechanics and fibrosis patterns in hypertrophic cardiomyopathy. Scientific Report. 2025.
5. Differential determinants and prognostic value of aortic valve sclerosis over carotid atherosclerosis. Int J Cardiol. 2025.

이상지질혈증 치료 개시 및 유지의 어려움

김민정

이화여대 목동병원 순환기내과

이상지질혈증은 심혈관질환의 핵심적인 위험인자이며, 스타틴 치료는 심혈관 사건 감소에 있어 가장 근거가 확립되어 있는 전략입니다. 그러나 실제 임상에서는 치료 개시 및 유지 과정에서 상당한 간극이 존재하며, 이는 real-world에서의 환자 인식과 행동에 의해서 크게 영향을 받고 있습니다. 특히 최근에는 다양한 매체를 통해 statin 관련 오해와 정보 왜곡 등이 증가하면서, 환자의 치료 순응도 저하가 중요한 임상적 문제로 대두되고 있습니다. 국내의 이상지질혈증 유병률은 약 40-47%로 매우 높은 수준이며, 치료율은 약 60%에 이르지만 실제 조절률은 약 50%에 머무르고 있습니다. 고위험 환자군에서의 LDL-C 목표 달성률은 약 30% 수준으로 보고되고 있어, 치료에도 불구, 충분한 위험 감소 효과가 달성되지 못하는 "treatment gap"이 명확히 존재하고 있습니다. 이러한 결과는 단순히 약제 선택의 문제만이 아닌, 치료 강도 부족 혹은 순응도 저하 등이 복합적으로 작용한 결과로 생각됩니다.

특히 치료 유지 측면에서 real-world data는 중요한 시사점을 제공합니다. 국내 연구에서 스타틴 치료 시작 후 1-2년 이내 약 40%의 환자가 약물을 중단하는 것으로 보고되었으며, 이는 무작위 임상시험 환경과 비교할 때 현저히 높은 수치입니다. 이러한 차이는 실제 임상 환경에서 환자의 인식, 특히 약물에 대한 불안과 오해가 치료 지속성에 큰 영향을 미칠 수 있다는 점을 시사합니다. 또한 치료 개시 단계에서도 유사한 문제가 관찰됩니다. "증상이 없는데 약이 필요한가?", "생활습관 개선으로 충분하지 않은가?"와 같은 질문은 실제로 임상에 매우 흔합니다. 국내외 가이드라인에서는 위험군에 따라 적극적인 LDL-C 감소 전략을 권고하고 있으나, 실제 진료에서는 이러한 접근이 충분히 반영되지 못하는 경우가 있습니다. 또한 스타틴 관련 부작용에 대한 과장된 인식 역시 치료 유지의 주요 장애 요인입니다. 근육통, 간독성, 당뇨 발생 등에 대한 우려는 환자의 약물 중단으로 이어지며, 실제로는 상당수의 증상들이 약물 자체보다는 nocebo effect와 관련이 있는 것으로 보고되고 있습니다.

따라서 이상지질혈증 치료에서의 주요한 핵심 문제 중 하나로서 real-world에서의 치료 개시 지연, 낮은 순응도, 그리고 statin에 대한 오해에 기반한 치료 중단 등을 고려할 수 있습니다. 본 강의에서는 이상지질혈증 치료에서의 주요한 핵심 문제 중 하나로서 real-world 에서의 치료 개시 지연, 낮은 순응도, 그리고 statin에 대한 오해에 기반한 치료 중단 등의 문제에 대하여 국내 자료를 중심으로 알아보고, 실제 외래에서 흔히 마주하는 문제 상황을 통해 이상지질혈증 치료와 지속에 대한 어려움에 대하여 재조명 하고자 합니다.

References

1. 2019 ESC/EAS Guidelines for the management of dyslipidaemias. Eur Heart J. 2020;41:111-188.
2. AHA/ACC/AACVPR/AAPA/ABC/ACPM/ADA/AGS/APhA/ASPC/NLA/PCNA Guideline on the Management of Blood Cholesterol, Circulation. 2019;139:e1082-e1143.
3. Korean Guidelines for the Management of Dyslipidemia, 5th, Committee of Clinical Practice Guideline of the Korean Society of Lipid and Atherosclerosis
4. Dyslipidemia Fact Sheet in South Korea, 2024, J Lipid Atheroscler. 2025 Sep;14(3):298-311.
5. The nocebo effect in the context of statin intolerance, J Clin Lipidol. 2016;10(4):739-747.

CURRICULUM VITAE

김대영

인하대병원

[학력]

2013 전북대 의학전문대학원 졸업 (석사)

[경력]

2013-2014 순천향대학교 부천병원 인턴
2015-2019 인하대병원 내과 전공의
2019-2021 신촌세브란스병원 심장내과 강사
2021 신촌세브란스병원 심장내과 임상조교수
2022 분당차병원 심장내과 임상조교수
2023- 인하대병원 심장내과 조교수

[관심분야]

심장초음파, 심부전, 고혈압

[논문]

1. Preprocedural Left Atrial Strain as a Predictor of Long-Term Outcomes Following Mitral Valve Interventions in Rheumatic Severe Mitral Stenosis. Journal of the American Heart Association (2025, e043746).
2. Long-Term Follow-Up in Severe Mitral Stenosis With Low Transmitral Diastolic Pressure Gradient: From MASTER Registry. Circulation Cardiovascular Imaging. (2025, e018124).
3. Validation of TRI-SCORE for outcome prediction after isolated tricuspid valve surgery in Asian patients. Journal of the American Heart Association (2024, 13.8:e032929).
4. Outcomes of Severe Mitral Stenosis with the Revised Severity Criteria: Mitral Valve Replacement versus Percutaneous Mitral Valvuloplasty. Canadian Journal of Cardiology (2024, 40.1:100-109).
5. Prognostic Implication of Mitral Valve Disease and Its Progression in East Asian Patients With Hypertrophic Cardiomyopathy. Journal of the American Heart Association (2023, 12.3:e024792).

이상지질혈증과 동맥경화치료에 있어서 주요 가짜 정보의 유형 (매체에서 보이는 유형 정리)

김대영

인하대병원 심장내과

The widespread dissemination of health information through mass media and online platforms has significantly influenced public perception of cardiovascular disease prevention and treatment. However, misinformation related to dyslipidemia and atherosclerosis remains prevalent and may lead to inappropriate health behaviors, delayed treatment, or distrust of evidence-based medicine. In particular, misleading claims about cholesterol, statin therapy, and alternative treatments frequently appear in news media, social media, and commercial advertisements.

This presentation aims to categorize common types of misinformation regarding dyslipidemia and atherosclerosis management that are frequently encountered in public media. We reviewed representative examples of health-related information disseminated through mass media, online articles, and social media platforms (ex. Youtube, Instagram) and analyzed recurring patterns of misleading or inaccurate claims.

Several major categories of misinformation were identified. First, oversimplification of cholesterol concepts, such as portraying all cholesterol as harmful or, conversely, claiming that cholesterol is entirely harmless. Second, exaggerated claims about the risks of statins, including unsupported associations with severe adverse effects, which may discourage appropriate treatment. Third, promotion of unproven alternative therapies, such as dietary supplements or "natural" remedies presented as substitutes for evidence-based lipid-lowering therapy. Fourth, misinterpretation of scientific studies, where preliminary findings or observational results are presented as definitive clinical evidence.

Understanding these patterns of misinformation is important for clinicians and researchers who communicate cardiovascular risk and treatment strategies to the public and patients. Improved collaboration between healthcare professionals, academic societies, and media organizations may help promote accurate health communication and reduce the impact of misinformation on cardiovascular disease prevention and management.

CURRICULUM VITAE

이봄

차의과학대학교 분당차병원

[학력]

2015.02 한림대학교 의과대학 졸업
2020.02 한림대학교 대학원 의학과 수료

[경력]

2015.03-2016.02 한림대학교 춘천성심병원 인턴
2016.03-2020.02 한림대학교 춘천성심병원 내과 전공의
2020.03-2023.02 세브란스병원 심장내과 전임의
2023.03-2024.02 차의과학대학교 분당차병원 심장내과 임상조교수
2024.03- 차의과학대학교 분당차병원 심장내과 조교수

[관심분야]

이상지질혈증

[논문]

1. Moderate-intensity statin plus ezetimibe vs high-intensity statin according to baseline LDL-C in the treatment of atherosclerotic cardiovascular disease: A post-hoc analysis of the RACING randomized trial. Atherosclerosis. 2023;386:117373.
2. Sex Differences in Ticagrelor with or without Aspirin after percutaneous coronary intervention in acute coronary syndrome patients: A post-hoc secondary analysis of TICO Randomized Clinical Trial. Arterioscler Thromb Vasc Biol. 2023;43(6):e218-e226.
3. Stent expansion evaluated by optical coherence tomography and subsequent outcomes: Scientific Reports 2023;13:3781.
4. Escalation of lipid-lowering therapy in patients with vascular disease receiving HIGH-intensity statins: the retrospective POST-HIGH study: Scientific Reports 2021;11:8884.

가짜 정보에 의한 환자와 의료진의 피해 현황 (증례와 의료정보위원회 설문조사)

이봄

Cardiology, CHA University, Korea

실제 임상에서 이상지질혈증의 치료 개시와 유지에는 여전히 많은 어려움이 존재하며, 최근 이러한 문제의 주요 원인으로 가짜 정보(Mis- or Disinformation)가 주목받고 있다. 실제 임상 현장에서 잘못된 의료 정보가 어떠한 형태로 나타나고, 치료 과정에 어떤 영향을 미치는지를 의료진의 관점에서 평가한 자료는 제한적이다.

이에 잘못된 의료 정보가 이상지질혈증 환자의 치료 순응도와 의료진의 진료에 미치는 실질적인 영향을 파악하고자 2026년 2-3월, 한국지질동맥경화학회 소속 의료진을 대상으로 설문 조사를 실시하였다.

응답자는 158명으로, 응답자의 48.1%는 심장내과, 25.3%는 내분비대사내과 전문의이며, 응답자의 58.2%는 대학병원, 22.2%는 의원급 의료기관의 근무자였다. 설문 결과, 응답자의 96.8%가 잘못된 정보로 인해 이상지질혈증의 치료를 중단하거나 거부한 환자를 진료실에서 경험한 것으로 나타났다.

비록 본 조사는 제한된 수의 의료진 응답을 기반으로 한 탐색적 연구라는 한계가 있어, 국내 전체 의료진의 경험을 대표로 한다고 보기는 어렵다. 그러나 본 결과는 진료 현장에서 반복적으로 관찰되는 가짜 정보로 인한 문제를 구체적으로 보여주며, 이상지질혈증 치료의 중요한 장벽으로 작용하고 있음을 시사한다.

본 강의에서는 실제 임상 증례와 함께 설문조사 결과를 제시하여, 가짜 정보가 치료 결정과 환자 순응도에 미치는 영향을 조명하고자 한다.

CURRICULUM VITAE

박상민

을지의대 심장내과

[전문분야]

협심증 및 심근경색, 고혈압, 심부전, 이상지질혈증, 대동맥 및 말초혈관질환, 부정맥

[전문진료]

내과학, 협심증, 심근경색, 고혈압, 심부전, 이상지질혈증, 대동맥 및 말초혈관질환, 부정맥

[주요 진료분야]

내과학, 관상동맥질환 (협심증, 심근경색), 고혈압, 심부전, 이상지질혈증, 대동맥 및 말초혈관질환, 부정맥

[학력]

연세대학교 대학원 의학과 석사
연세대학교 대학원 의학과 박사

[경력]

세브란스 심장혈관병원 임상강사(1998.03-2010.02)
한림대학교 춘천성심병원 순환기내과 부교수(2013.01-2020.06)
현) 노원을지대학교병원 심장내과 부교수

이상지질혈증과 동맥경화 치료 관련 가짜 정보 등에 대한 학회 차원의 대응 대책

박상민

노원을지병원 심장내과

이상지질혈증은 죽상경화성 심혈관질환(atherosclerotic cardiovascular disease, ASCVD)의 핵심적인 위험인자로, 스타틴을 중심으로 한 지질저하 치료는 심혈관 사건 예방에 있어 확립된 근거를 가지고 있다. 그러나 실제 임상 현장에서는 약물의 효과에도 불구하고 장기 치료의 지속성이 낮아, 처방된 지질저하제의 임상적 혜택이 충분히 실현되지 못하는 경우가 흔하다. 다수의 관찰 연구와 실제 진료자료(real-world data)에 따르면, 스타틴 치료를 시작한 환자의 약 40-60%가 1년 이내에 복약을 중단하거나 불규칙한 복용 패턴을 보이는 것으로 보고되고 있으며, 이는 심혈관 사건 및 사망 위험 증가와 직접적으로 연관된다.

복약순응도 저하의 원인은 환자 요인, 의료진 요인, 약물 요인, 그리고 보건의료 시스템 요인이 복합적으로 작용하지만, 최근에는 **스타틴에 대한 잘못된 의료정보(medical misinformation)**가 순응도 저하의 핵심 요인으로 주목되고 있다. 특히 근거가 부족한 근육통, 간독성, 인지기능 저하, 당뇨병 유발 위험에 대한 과장된 정보가 대중매체와 온라인 플랫폼을 통해 확산되면서, 환자의 치료 불안과 자의적 중단을 초래하는 경우가 빈번하다. 이러한 잘못된 정보는 무증상 질환이라는 이상지질혈증의 특성과 결합되어 스타틴 치료의 장기 지속성을 저해하며, 실제 심혈관 사건 예방 효과를 약화시키는 중요한 요인으로 작용한다.

스타틴 관련 잘못된 의료정보로 인한 복약순응도 저하를 극복하기 위해서는 단일 접근법이 아닌 다차원적(multidimensional) 전략이 필요하다. 첫째, **근거 중심의 위험 소통(evidence-based risk communication)**을 통해 스타틴의 절대적 심혈관 사건 감소 효과와 실제 부작용 발생 빈도를 명확히 전달해야 한다. 무작위 임상시험과 메타분석 자료를 활용하여 근육 증상의 상당 부분이 노시보(nocebo) 효과와 연관됨을 설명하는 것은 환자의 불안을 완화하는 데 도움이 된다. 둘째, 의료진은 스타틴에 대한 오해를 선제적으로 언급하고, 환자가 접했을 가능성이 높은 잘못된 정보에 대해 열린 대화를 통해 교정하는 전략을 취해야 한다.

약물 전략 측면에서는 치료의 유연성을 강조하는 접근이 중요하다. 저용량 스타틴 재도전, 간헐적 복용, 또는 ezetimibe 병용 요법 등 개인 맞춤형 치료 전략은 스타틴 불내성에 대한 인식을 완화하고 치료 지속성을 향상시킬 수 있다. 또한 고위험군 환자에서는 PCSK9 억제제와 같은 비스타틴 계열 약제를 적절히 활용함으로써 LDL-콜레스테롤 목표 도달과 순응도를 동시에 개선할 수 있다.

셋째, 디지털 헬스 기술을 활용한 개입 역시 중요한 역할을 한다. 모바일 애플리케이션, 문자 알림, 원격 모니터링은 복약 이행도를 향상시키고 장기 치료 지속성을 높이는 데 기여할 수 있다. 넷째, 다학제적 접근을 통해 간호사, 약사, 코디네이터가 참여하는 팀 기반 관리 모델은 환자 교육과 추적 관리를 강화함으로써 순응도를 유의하게 개선하는 것으로 보고되고 있다.

결론적으로 이상지질혈증 및 동맥경화 치료에서 복약순응도는 단순한 환자 개인의 문제가 아니라 치료 성과를 좌우하는 핵심 요소이다. 효과적인 복약순응도 향상 전략은 근거 중심의 약물 선택, 명확한 의사소통, 치료 단순화, 그리고 시스템 차원의 지원이 통합적으로 이루어질 때 극대화될 수 있으며, 이는 궁극적으로 ASCVD 부담 감소로 이어질 것이다.

Acknowledgement

An AI language model (ChatGPT, GPT-4.1) was used to assist the author(s) in the preparation of this manuscript.

참고문헌

1. Cholesterol Treatment Trialists' (CTT) Collaboration. Efficacy and safety of LDL-lowering therapy among men and women: meta-analysis of individual data from 174,000 participants. Lancet. 2015 Apr 11;385(9976):1397-405.
2. Sabatine MS, Giugliano RP, Keech AC, et al. Evolocumab and clinical outcomes in patients with cardiovascular disease. N Engl J Med. 2017;376:1713-1722.
3. Bradley CK, Wang TY, Li S et al. Patient-Reported Reasons for Declining or Discontinuing Statin Therapy: Insights From the PALM Registry. J Am Heart Assoc. 2019;8:e011765.

SoLA 2026

한국지질·동맥경화학회 춘계학술대회

2026 Spring Congress on
Lipid and Atherosclerosis of KSoLA

Symposium 7

해외 학회 참관기

4월 3일(금) 13:50-15:20 | Room 4 (볼룸 1,2)

좌장 : 홍영준(전남의대 순환기내과), 홍순준(고려의대 순환기내과)

패널 : 김민관(연세의대 심장내과), 김봉준(고신의대 순환기내과)
임영효(한양의대 심장내과)

13:50-14:08	**CLVS 2025** **박상민**(을지의대 심장내과)
14:08-14:26	**VAS 2025** **김학령**(서울의대 순환기내과)
14:26-14:44	**APSAVD 2025** **조상호**(한림의대 순환기내과)
14:44-15:02	**AAS 2025** **최훈지**(한림의대 내분비내과)
15:02-15:20	**Panel Discussion**

CURRICULUM VITAE

박상민

을지의대 심장내과

[전문분야]

협심증 및 심근경색, 고혈압, 심부전, 이상지질혈증, 대동맥 및 말초혈관질환, 부정맥

[전문진료]

내과학, 협심증, 심근경색, 고혈압, 심부전, 이상지질혈증, 대동맥 및 말초혈관질환, 부정맥

[주요 진료분야]

내과학, 관상동맥질환 (협심증, 심근경색), 고혈압, 심부전, 이상지질혈증, 대동맥 및 말초혈관질환, 부정맥

[학력]

연세대학교 대학원 의학과 석사
연세대학교 대학원 의학과 박사

[경력]

세브란스 심장혈관병원 임상강사(1998.03-2010.02)
한림대학교 춘천성심병원 순환기내과 부교수(2013.01-2020.06)
현) 노원을지대학교병원 심장내과 부교수

CLVS 2025

박상민

을지의대 심장내과

CURRICULUM VITAE

김학령

서울특별시 보라매병원 순환기내과

[학력]

2013	전남의대 학사 졸업
2011	성균관대학교 경영학(iMBA) 석사 졸업
2013	서울대학교 의과대학 내과학 박사 졸업

[경력]

2003-2008	서울대병원 인턴/내과 레지던트
2008-2011	육군대위(JSA 경비대대, 국군서울지구병원)
2011-2012	서울대병원 순환기내과 전임의

[관심분야]

동맥경직도, 중심동맥압, 여성심장환

[논문]

1. Hack-Lyoung Kim, Soonil Kwon, Hyun Sung Joh, et al. Invasively measured aortic pulse pressure and long-term prognosis in patients undergoing invasive coronary angiography: a prospective observational study. J Hypertens 2026 Feb 1;44(2):354-359.
2. Hack-Lyoung Kim, Hyun Sung Joh, Sang-Hyun Kim. Body Weight Change and Arterial Stiffness: An Analysis of the Korean National Health and Nutrition Examination Survey. CardioMetab Syndr J 2026 (in press).
3. Hack-Lyoung Kim, Dong-Hoon Kim, Kyueng-Whan Min, et al. Arterial Stiffness and Histologically Confirmed Helicobacter pylori Infection in Young Adults. Helicobacter. 2025 Jul-Aug;30(4):e70067.
4. Hack-Lyoung Kim, Haechan Cho, Soonil Kwon, et al. Sex-Specific Determinants of Arterial Stiffness in Patients with Cardiovascular Risk Factors. Sex Gend Specif Biomed 2026;1:1-11.
5. Hack-Lyoung Kim. Arterial stiffness and pulsatile hemodynamics in cardiometabolic disorders. Cardiometab Syndr J. 2025 Sep;5(2):60-72.

VAS 2025 (My experience at the Vietnam Association of Atherosclerosis (VAS) meeting)

Hack-Lyoung Kim

Division of Cardiology, Boramae Medical Center, Seoul National University College of Medicine, Seoul, Republic of Korea

My visit to Vietnam for the Vietnam Association of Atherosclerosis (VAS) meeting was personally meaningful, as it was my first academic visit to the country, apart from a previous family trip. Because my father was a veteran of the Vietnam War, I grew up hearing many stories about Vietnam and developed a strong interest in its history. Upon arriving in Ho Chi Minh City, I visited the War Remnants Museum. Although it is widely known as a major tourist destination, I was somewhat disappointed by its modest scale and the predominance of photographic exhibits. Nevertheless, seeing original historical photographs, including images of the arrival of Korean troops, evoked complex emotions. As a descendant of a Korean veteran, I felt a sense of reflection and empathy toward the Vietnamese people. The VAS meeting itself was lively and well attended. The registration area was crowded, reflecting the high level of interest in the conference. As a representative of the Korean Society of Lipid and Atherosclerosis, I was warmly welcomed by the organizers. The meeting began with a traditional Vietnamese dance performance, followed by the scientific sessions. Media coverage was also notable, with several reporters documenting the event. I delivered two lectures, focusing on arterial stiffness and cardiovascular disease in women. Although the conference lasted only one day, it was efficiently organized and thoughtfully structured. I also observed cultural similarities between Vietnamese and Korean academic communities, particularly in the respect shown to senior professors and the clear hierarchical organization. The gala dinner began formally, with traditional performances and speeches, but the atmosphere changed dramatically when a famous Vietnamese singer performed, creating a lively and memorable evening. Overall, the VAS meeting was an enriching academic and cultural experience. While most presentations relied heavily on Western data, I hope that more locally generated Vietnamese research will be presented in the future.

CURRICULUM VITAE

조상호(Sang-Ho Jo)

Professor, Div. of Cardiology, Dept. of Internal Medicine, College of Medicine,
Hallym University, Hallym University Sacred Heart Hospital, Gyeonggi-do, Korea

[Education]

1991-1992	Premedical Course, College of Liberal Arts & Science, Seoul National University
1993-1997	Seoul National University College of Medicine (M.D.)
2002-2004	Graduate School, Seoul National University College of Medicine

[Professional Activities]

1998-1999	Internship, Seoul National University Hospital, Seoul, Korea
2000-2004	Residency in Internal Medicine, Seoul National University Hospital, Seoul, Korea
2004-2006	Fellowship in Cardiology, Seoul National University Hospital, Seoul, Korea
2006-2006	Full Time Lecturer, Hallym University Sacred Heart Hospital, Anyang-si, Gyeonggi-do, Korea
2006-Present	Professor, Hallym University Sacred Heart Hospital, Anyang-si, Gyeonggi-do, Korea

[Fields of Interest]

1. Interventional cardiology
2. Myocardial salvage
3. Vascular biology
4. Antioxidant
5. Pathogenesis of atherosclerosis and coronary artery disease
6. Diabetes and vascular biology

[Societies]

Academic activities as editor and reviewer

Editor of the Journal of Korean Medical Science (JKMS) 2012-2015
Editor of the of the Korean Circulation Journal (KCJ)
Editor of the of Journal of the Korean Society of Hypertension
Editor of the Korean Edition of Circulation. Aug 2008
Editor of the Korean Edition of Hypertension. July 2007
Editor of the Korean Edition of Atherosclerosis, thrombosis, vascular biology (ATVB), March 2009
Editor of the Korean Edition of European Heart Journal
Member of Heart Failure Research at Korean Heart Society. May 2008
Editorial members of Journal of the Korean Society of Hypertension

APSAVD 2025

조상호

한림의대 순환기내과

2025년 9월 20일부터 21일까지 대만 타이베이 Taipei International Convention Center (TICC)에서 열린 APSAVD 2025는 아시아-태평양 지역의 죽상경화증과 혈관질환 분야 전문가들이 모여 최신 연구와 임상 경험을 공유하는 뜻깊은 자리였다. 이번 학회는 APSAVD 공식 학회와 대만 관련 학회 안내에 따르면 지질대사, 죽상경화증, 심혈관 예방, 유전학, 생활습관 중재 등 다양한 주제를 폭넓게 다루었으며, 일본·한국·유럽 학회와의 공동 심포지엄, 구연 및 포스터 발표, Young Investigator Session 등도 함께 진행되었다.

이번 학회에 참석하며 가장 인상 깊었던 점은, 동맥경화와 혈관질환을 바라보는 시각이 점점 더 정밀하고 다층적으로 바뀌고 있다는 점이었다. 단순히 LDL-C를 낮추는 접근을 넘어, 잔여위험(residual risk), 중성지방-rich lipoprotein, 염증, 유전적 소인, 그리고 생활습관 교정까지 함께 논의되는 흐름이 매우 뚜렷했다. 특히 아시아 지역은 서구와는 또 다른 임상적 특성과 위험인자를 지니고 있기 때문에, 이러한 국제 학술 교류의 장은 실제 진료에 매우 실질적인 의미를 가진다고 느꼈다. APSAVD 자체도 서구 중심 근거를 그대로 수용하기보다, 아시아-태평양 지역의 질병 양상과 차이를 함께 조명하는 것을 중요한 목표로 두고 있다.

또한 이번 학회는 연구자와 임상의가 서로의 언어를 이해하고 연결하는 장이라는 점에서 의미가 컸다. 기초연구의 새로운 발견이 실제 예방과 치료 전략으로 어떻게 이어질 수 있는지, 반대로 임상 현장에서 제기되는 질문이 어떤 새로운 연구를 촉진하는지를 여러 세션에서 확인할 수 있었다. Young Investigator Session과 포스터 발표를 통해서는 젊은 연구자들의 참신한 아이디어와 에너지도 느낄 수 있었고, 학문의 지속성과 확장 가능성을 다시 생각하게 되었다.

짧은 일정이었지만 APSAVD 2025는 단순한 학술대회 이상의 의미를 남겼다. 빠르게 변화하는 심혈관 예방의학의 흐름 속에서, 최신 지견을 배우는 것뿐 아니라 아시아 지역 전문가들과 문제의식을 공유하고 협력의 가능성을 확인할 수 있었던 의미있는 시간이었다.

CURRICULUM VITAE

최훈지

한림대학교 동탄성심병원

[학력]

2012.03-2016.02 서울대학교 의학대학원
2019.03-2023.02 서울대학교 대학원 내과학 박사

[경력]

2016.03-2017.02 서울대학교병원 수련의
2017.03-2020.02 분당서울대학교병원 내과 전공의
2020.03-2023.02 서울대학교병원 내분비대사내과 임상강사
2023.03-2024.02 한림대학교 동탄성심병원 내분비대사내과 진료조교수
2024.03-2026.02 한림대학교 동탄성심병원 내분비대사내과 임상조교수
2026.03- 한림대학교 동탄성심병원 내분비대사내과 조교수

[관심분야]

당뇨병, 비만, 근감소증, 고중성지방혈증

[논문]

1. Choe HJ, Després JP, Wilding JPH, Ryan DH, Lim S. Clinical and Preclinical Obesity in Korean Adults from 2014 to 2023. Diabetes Metab J. 2026;50(1):127-138.
2. Choe HJ, Han KD, Park JH, Lee J, Kwak MK, Choi YM, Moon SJ, Hong EG. Underweight and Mortality in Type 2 Diabetes: A Nationwide Retrospective Cohort Study. J Cachexia Sarcopenia Muscle. 2025 Dec;16(6):e70145. doi: 10.1002/jcsm.70145. PMID: 41351230; PMCID: PMC12680783.
3. Bae JH, Choe HJ, Yang YS, Seo MH, Choi JH, Kim G, Lyu YS, Han JH, Kang S, Kim WJ, Kim KS, Cho YM, Cha BS; Severe Diabetes Mellitus Task Force of the Korean Diabetes. Defining Severe Diabetes Mellitus: A Consensus Framework for Grading and Staging Diabetes Based on Pathophysiology and Complications. Diabetes Metab J. 2025 Nov;49(6):1141-1154. doi: 10.4093/dmj.2025.0739. Epub 2025 Oct 28. PMID: 41153071; PMCID: PMC12620694.
4. Choe HJ, Lee JS, Park JY, Lee SA, Park YJ, Chung SS, Park KS. SENP2 regulates UCP1-dependent thermogenesis in brown adipocytes via deSUMOylation of ERRα. Exp Mol Med. 2025 Jun;57(6):1283-1293. doi: 10.1038/s12276-025-01458-5. Epub 2025 Jun 27. PMID: 40579429; PMCID: PMC12229480.
5. Choe HJ, Moon JH, Kim W, Koo BK, Cho NH. Steatotic liver disease predicts cardiovascular disease and advanced liver fibrosis: A community-dwelling cohort study with 20-year follow-up. Metabolism. 2024 Apr;153:155800. doi: 10.1016/j.metabol.2024.155800. Epub 2024 Jan 22. PMID: 38266957.

AAS 2025

최훈지

한림대학교 동탄성심병원 내분비대사내과

2025년 10월 29일부터 31일까지 호주 시드니에서 개최된 Australian Atherosclerosis Society Annual Scientific Meeting (AAS 2025)에 연자로 초청되어 학회에 참석하였다. AAS 연례 학술대회는 동맥경화 및 심혈관·대사질환 분야에서 기초부터 임상, 중개연구에 이르기까지 최신 연구 성과와 미래 연구 방향을 공유하는 호주를 대표하는 국제 학술대회이다.

본 학회는 Precision prevention, cardiometabolic crossroads, sex differences in cardiovascular disease, diabetic atherosclerosis, artificial intelligence in cardiovascular innovation 등 현재 심혈관·대사 연구 분야에서 가장 중요한 주제들을 중심으로 구성되었으며, 특히 기초 기전 연구와 임상적 적용을 연결하려는 시도가 다수의 세션에서 강조되었다. 또한 ECR 및 HDR Rising Star 세션을 통해 차세대 연구자들의 혁신적인 연구 성과를 조명하며 학문적 지속 가능성을 도모하였다.

이번 AAS 2025 참관을 통해 최신 심혈관·대사 연구의 국제적 흐름과 연구 패러다임의 변화를 직접 확인할 수 있었으며, 향후 국내 연구 및 진료 현장에 적용 가능한 학문적 통찰을 얻을 수 있었다. 또한 해외 연구자들과의 학술 교류를 통해 향후 국제 공동연구의 가능성을 모색하는 계기가 되었다.

SoLA 2026

한국지질·동맥경화학회 춘계학술대회

2026 Spring Congress on
Lipid and Atherosclerosis of KSoLA

SoLA 2026

한국지질·동맥경화학회 춘계학술대회

2026 Spring Congress on
Lipid and Atherosclerosis of KSoLA

Symposium 8

New Guidelines and New Drugs

4월 3일(금) 15:20-16:50 | Room 1 (그랜드볼룸 3)

좌장 : 안영근(전남의대 순환기내과), 김철식(연세의대 내분비내과)

패널 : 김재석(연세원주의대 신장내과), 박정환(한양의대 내분비내과)
임홍석(아주의대 순환기내과)

15:20-15:38	**Overview of 2025-2026 dyslipidemia guidelines in Europe and US** **윤종찬**(가톨릭의대 순환기내과)
15:38-15:56	**Early and intensive lipid-Lowering after acute coronary syndrome: timing, targets, and real-world challenges** **임수빈**(이화의대 순환기내과)
15:56-16:14	**Managing residual atherosclerotic risk: beyond LDL-C toward precision lipid therapy** **이수연**(단국의대 심장혈관내과)
16:14-16:32	**New and emerging lipid-lowering therapies: what's next** **김상현**(서울의대 순환기내과)
16:32-16:50	**Panel Discussion**

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 학술위원, 간행위원, 윤리위원, 심장종양학연구회 총무위원장, KCJ Assistant Editor
대한심부전학회 연구이사, EHJ-CVP Associate Editor
대한내과학회 학술위원회 간사, 한국지질동맥경화학회 임상연구이사
대한심뇌혈관질환예방학회 학술이사, 심장대사증후군학회 전문가양성위원회 이사
대한혈관학회 국내교류이사, 대한이식학회 학술위원, 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

Overview of 2025-2026 dyslipidemia guidelines in Europe and US

Jong-Chan Youn

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

The 2025 Focused Update of the 2019 ESC/EAS Guidelines for the Management of Dyslipidaemias was released during the ESC Congress 2025 in Madrid. This update, published six years after the original 2019 guideline, represents a substantial revision of lipid management strategies, incorporating newly accumulated evidence from randomized clinical trials and meta-analyses. Importantly, this is not a minor revision but a formal Focused Update issued by the ESC and EAS, reflecting the consensus that emerging evidence is sufficiently robust to influence clinical practice prior to the next full guideline update. The key updates are organized into four major domains: cardiovascular risk assessment, lipoprotein (a), LDL-C-lowering therapies and combination strategies, and triglycerides and nutraceuticals.

The ESC recommendations for lipid-lowering therapy during index hospitalization for acute coronary syndrome (ACS) are closely aligned with the recently published 2025 ACC/AHA ACS guidelines. These guidelines recommend high-intensity statin therapy for all patients with ACS, with consideration of early combination therapy with ezetimibe. In patients receiving maximally tolerated statin therapy who have an LDL-C level ≥70 mg/dL, the addition of nonstatin lipid-lowering agents is recommended. Furthermore, in very high-risk patients, additional intensification of lipid-lowering therapy may be considered even when LDL-C levels are between 55 and <70 mg/dL.

Finally, this lecture will briefly review key updates in the 2026 ACC/AHA dyslipidemia guidelines compared with the 2018 version, including PREVENT equation-based risk prediction, recommendations for lipoprotein(a) measurement, and coronary artery calcium-guided risk reclassification, along with the top 10 take-home messages for clinical practice.

CURRICULUM VITAE

임수빈

이대서울병원

[학력]

2017	고려대학교 의과대학 의학사
2024	고려대학교 의과대학 의학박사

[경력]

2017-2021	고려대학교 의료원 인턴/내과 전공의
2021-2024	고려대학교 안암병원 순환기내과 전임의/임상조교수대우
2024-	이화여자대학교 서울병원 순환기내과 임상조교수

[관심분야]

심혈관중재시술

Early and intensive lipid-Lowering after acute coronary syndrome: timing, targets, and real-world challenges

임수빈

이화의대 순환기내과

CURRICULUM VITAE

이수연(Su Yeon Lee)

단국대학교 병원 심장내과

[학력]

2006.03-2012.02 충남대학교 의학 학사
2016.03-2018.02 성균관대학교대학원 의학 석사
2020.03-2026.02 성균관대학교대학원 의학 박사

[수련]

2012-2013 Internship, Samsung Medical Center
2013-2017 Residency, Internal Medicine, Samsung Medical Center

[경력]

2017-2019 Fellow, Cardiology, Samsung Medical Center
2019-2021 Clinical Assistnat Professor, College of Medicine, Dankook University
2021 Assistant Professor, College of Medicine, Dankook University

[면허, 자격]

2012 License, Korea Medical License (110586)
2017 License, Internal Medicine (17022)
2019 License, Cardiology (2-19-1219)
2019 Certification, Echocardiography

Managing residual atherosclerotic risk: beyond LDL-C toward precision lipid therapy

Su Yeon Lee

Cardiology, Dankook University Hospital, South Korea

스타틴을 포함한 강력한 LDL-C 저하 요법은 죽상경화성 심혈관 질환(ASCVD)의 예방과 치료에 획기적인 전환점을 마련했다. 그러나 최적의 LDL-C 목표치에 도달한 환자 중에서도 약 60~70%는 여전히 주요 심혈관 사건(MACE)을 경험하며, 이는 LDL-C 이외의 '잔류 위험(residual Risk)' 요인을 관리해야 할 필요성을 시사한다. LDL-C 이외에도 아포지단백 B(ApoB)로 반영되는 죽상경화성 입자 부담, 중성지방 풍부 지단백 (TG riched lipoprotein, TRL)과 잔여물 (TRL remnants), 지단백(a) [Lp(a)], 그리고 잔여 염증 활성은 중요한 위험 인자이며, 특히 당뇨병, 대사증후군, 만성콩팥병, 혼합형 이상지질혈증 환자에서 그 중요성이 더욱 크다.

전통적으로 중성지방 자체의 위해성에 대해서는 논란이 있었으나, 최근의 유전학 및 역학 연구는 중성지방 풍부 TRL 및 TRL remnants가 ASCVD의 독립적이고 인과적인 위험 요인임을 명확히 하고 있다. 인슐린 저항성이나 유전적 요인으로 인해 TRL의 과잉 생성 또는 리파아제(LpL)에 의한 지방 분해 저해가 발생하면, 콜레스테롤이 농축된 TRL remnants가 혈중에 축적된다. 이 입자들은 크기가 작아 혈관 내피세포를 통과하여 subendothelial space에 저류되며, LDL보다 입자당 더 많은 콜레스테롤을 함유하여 강력한 죽상경화 유발 효과를 나타낸다. 다만 REDUCE-IT와 PROMINENT의 상반된 결과가 보여주듯 단순한 중성지방 저하만으로는 임상적 이득이 보장되지 않으며, 정확한 병태생리 표적화가 중요하다.

이어서 잔류 염증 위험(residual inflammatory risk) 또한 간과할 수 없는 핵심 요소이다. 지질 수치가 가이드라인 내에 도달했음에도 발생하는 심혈관 사건의 상당수는 혈관 벽 내에 잔류하는 만성적인 염증 활성에 기인한다. CANTOS 연구는 IL-1β를 선택적으로 차단하는 Canakinumab이 지질 프로파일의 변화 없이도 주요 심혈관 사건을 유의하게 감소시킴을 입증함으로써, 지질 저하와 별개로 항염증 접근이 사건 감소에 기여할 수 있음을 임상적으로 보여주었다. 즉 LDL-C가 조절된 상태에서도 hsCRP 수치가 2 mg/L 이상인 환자들에게 잔류 염증 위험 관리가 필수적임을 시사한다. 죽상경화증의 진행에는 IL-1 → IL-6 → CRP로 이어지는 신호 전달 체계가 핵심적인 역할을 하며, 최근에는 Canakinumab뿐만 아니라 저용량 콜히친이 COLCOT 및 LoDoCo2 연구를 통해 강력한 심혈관 보호 혜택을 입증하며 실제 임상 현장에서의 실질적인 대안으로 주목받고 있다.

결국 잔류 심혈관 위험의 관리는 단순히 수치를 목표치보다 낮추는 단계를 넘어, 환자 개별의 병태생리학적 특성에 맞춘 '맞춤형 정밀 의료'를 지향해야 한다. LDL-C 조절 이후에도 잔류하는 TRL remnants의 콜레스테롤 부하와 hsCRP로 대변되는 염증 지표를 면밀히 모니터링하고 적절한 표적 치료를 병행함으로써, 추가적인 ASCVD의 발생을 예방할 수 있다.

CURRICULUM VITAE

김상현

서울의대 순환기내과

[학력]

1985-1991	서울대학교 의과대학 학사
1999-2001	서울대학교 의과대학원 석사
2001-2005	서울대학교 의과대학원 박사

[경력]

1991-1992	서울대학교병원 인턴
1992-1996	서울대학교병원 전공의(내과)
1999-2000	서울대학교병원 임상강사(순환기내과)
2003-2007	서울대학교 의과대학 내과학교실 조교수
2007-2012	서울대학교 의과대학 내과학교실 부교수
2007-현재	서울대학교 의과대학 내과학교실 교수
2000-현재	보라매병원 순환기내과 교수

[관심분야]

Lipid, atherosclerosis, hypertension

New and emerging lipid-lowering therapies: what's next

김상현

서울의대 순환기내과

최근 새로운 이상지질혈증 치료제들이 다수 개발되어 임상연구 진행 중이다. 스타틴, 에제티미브와 같이 경구 chemical 외에도 특정 목표에 대한 유전자 치료 방법들(ASO, siRNA, CRISPR-Cas9)이 다양하게 개발되고 있다. 우선 LDL 콜레스테롤 조절을 위한 PCSK9 억제제의 경우, 투여 간격이 긴 주사제들(Lerodalcibep)이 개발되었고, 경구 약제(MK-0616-Enlicitide, AZD 0780)도 임상연구 진행중이거나 연구완료 후 FDA 심사 중이며, PCSK9 유전자 치료에 관한 연구도 시작되었다. CETP 억제제인 obicetrapib도 surrogate marker 3상연구가 종료되고 임상예후 관련 연구가 진행 중이다. ATP citrate lyase 억제제인 bempedoic acid도 CLEAR OUTCOME 등 기존 연구들의 하위군 분석자료들이 발표되었다. ANGPTL3 억제제로는 현재 미국과 유럽에서 사용을 시작한 항체 evinacumab 외에도, 임상연구가 진행되다가 중단된 ASO (Vupanorsen)와 아직 추가 연구가 계획 중인 siRNA (Zodasiran, Solbinsiran)가 있고, ANGPTL3 유전자 치료에 관한 연구도 시작되었다. 중성지방 관리를 위한 ApoCIII 억제제도 개발되어 ASO (Volanesorsen, Olezarsen) 가 개발되었고, siRNA (Plozasiran)는 FDA 승인을 받았다. Lp(a)에 관한 치료제로는 ASO (Pelacarsen) 3상 연구결과가 올해 하반기 발표될 예정이며, 향후 siRNA (Olpasiran, Zerlasiran, Leopodisiran) 및 경구약 Muvalapin 연구결과들이 발표될 예정이다.

SoLA 2026

한국지질·동맥경화학회 춘계학술대회

2026 Spring Congress on
Lipid and Atherosclerosis of KSoLA

SoLA 2026

한국지질·동맥경화학회 춘계학술대회

2026 Spring Congress on
Lipid and Atherosclerosis of KSoLA

Symposium 9

HDL-C: The Higher, the Better?
— Revisiting the Myth of Good Cholesterol

4월 3일(금) 15:20-16:50 | Room 2 (그랜드볼룸 2)

좌장 : 홍은경(한림의대 내분비내과), 박재형(고려의대 순환기내과)

패널 : 유지희(중앙의대 내분비내과), 이희선(서울의대 순환기내과)
진흥용(전북의대 내분비내과)

15:20-15:40	**HDL-C in the guidelines: where do we stand today?** **정재훈**(동국의대 심장내과)
15:40-16:00	**HDL-C is cardioprotective – higher HDL-C reduces ASCVD risk** **손정우**(연세원주의대 심장내과)
16:00-16:20	**Extremely high HDL-C may be harmful – function matters more than quantity** **양예슬**(서울의대 내분비대사내과)
16:20-16:50	**Panel Discussion**

CURRICULUM VITAE

정재훈

동국대학교 일산병원

[학력]

영남대학교 학사
동국대학교 석사

[경력]

서울대병원 전임의
국립중앙의료원 전문의
동국대학교 일산병원 조교수

[관심분야]

동맥경화

[논문]

1. Chung J, Rhee M-Y, Kim KH, Jang J-S, Kim H-Y. Reassessing home blood pressure thresholds: clinical implications of lowering the diagnostic criteria to 130/80 mmHg. Journal of Hypertension 2026:10.1097.
2. Chung J, Kim H-L, Joh HS, et al. Incremental prognostic value of combined information of arterial stiffness and the result of treadmill exercise test in patients with suspected coronary artery disease. Journal of Human Hypertension 2025;39:566-571.
3. Chung J, Lim W-H, Kim H-L, Joh HS, Seo J-B, Kim S-H, Zo J-H, Kim M-A. Influence of Socioeconomic Status on the Presence of Obstructive Coronary Artery Disease and Cardiovascular Outcomes in Patients Undergoing Invasive Coronary Angiography. Healthcare. 2024; 12(2):228.
4. Chung J, Kim H-L, Lim W-H, et al. New onset diabetes mellitus and cardiovascular outcomes according to statin intensity in patients after drug-eluting stent implantation in Asian patients. Scientific Reports 2023;13:16061.
5. Chung, J., Min, K. W., Son, B. K., Kim, D. H., & Kim, H. L. (2021). Association between histological severity of Helicobacter pylori infection and cardiovascular risk scores in the Korean population. Atherosclerosis, 333, 124-130.

HDL-C in the guidelines: where do we stand today?

정재훈

동국의대 심장내과

CURRICULUM VITAE

손정우

원주세브란스기독병원 심장내과

[학력]

2004 연세대학교 원주의과대학 학사
2026 연세대학교 의학과 석박사통합

[경력]

2016- 원주세브란스기독병원 심장내과 조교수
2014-2015 한림대학교 춘천성심병원 심장내과 임상조교수
2012-2013 세브란스병원 심장내과 전임의
2004-2008 세브란스병원 내과 전공의

[관심분야]

심초음파, 심부전, 이상지질혈증, 고혈압

[논문]

1. Dobutamine Stress Echocardiography for Left Ventricular Reverse Remodeling in Idiopathic Dilated Cardiomyopathy. Int J Heart Fail. 2025 Jul 7;7(3):152-159.
2. Clinical and Echocardiographic Predictors for the Presence of Late Gadolinium Enhancement on Cardiac Magnetic Resonance Imaging in Patients with Carbon Monoxide Poisoning. Diagnostics (Basel). 2023 Dec 27;14(1):60.
3. Epidemiologic Profile of Patients With Valvular Heart Disease in Korea: A Nationwide Hospital-Based Registry Study. J Cardiovasc Imaging. 2023 Jan;31(1):51-61.
4. Temporal trends in heart failure over 11 years in the aging Korean population: A retrospective study using the national health insurance database. PLoS One. 2022 Dec 28;17(12):e0279541.

HDL-C is cardioprotective - higher HDL-C reduces ASCVD risk

손정우

연세원주의대 심장내과

CURRICULUM VITAE

양예슬

서울의대(서울대학교 직장부속의원)

[학력]

2006-2012	한림대학교 의과대학, 학사
2015-2017	서울대학교 융합과학기술대학원, 석사
2018-2024	서울대학교 의과대학, 박사

[경력]

2019-2020	서울의대 서울대학교병원, 내분비내과 진료교수
2020-2022	가톨릭의대 의정부성모병원, 내분비내과 임상진료조교수
2022-	서울의대 의학과 조교수

[관심분야]

당뇨병, 지질대사, 비만

[논문]

1. Physical activity for prevention of cardiovascular disease: consensus statement of Korean Society of Cardio-cerebrovascular Disease Prevention
2. Abdominal obesity and the risk of young-onset dementia in women: a nationwide cohort study
3. Real-World Treatment Patterns according to Clinical Practice Guidelines in Patients with Type 2 Diabetes Mellitus and Established Cardiovascular Disease in Korea: Multicenter, Retrospective, Observational Study
4. Efficacy and safety of monotherapy with enavogliflozin in Korean patients with type 2 diabetes mellitus: Results of a 12-week, multicentre, randomized, double-blind, placebo-controlled, phase 2 trial
5. Lipid Management in Korean People with Type 2 Diabetes Mellitus: Korean Diabetes Association and Korean Society of Lipid and Atherosclerosis Consensus Statement

Extremely high HDL-C may be harmful - function matters more than quantity

Ye Seul Yang

Department of Medicine, Seoul National University, Korea

High-density lipoprotein cholesterol (HDL-C) has traditionally been viewed as a protective factor against atherosclerotic cardiovascular disease (ASCVD), based on its well-known inverse association with cardiovascular risk. However, recent studies have suggested that this relationship may be more complex than previously understood. In particular, observations from large population-based cohorts and clinical trials have raised questions about the clinical significance of HDL-C levels alone, especially at higher ranges.

At the same time, growing attention has been directed toward the functional properties of HDL particles, including their roles in cholesterol efflux, inflammation modulation, and endothelial protection. These insights have led to increasing interest in whether HDL functionality, rather than circulating HDL-C concentration, may provide additional information for cardiovascular risk assessment.

This lecture will review the evolving evidence on HDL-C, highlight the limitations of a quantity-based interpretation, and introduce the concept of HDL functionality. The goal is to provide a balanced overview of current perspectives and to explore how these insights may inform future research and clinical practice.

SoLA 2026

한국지질·동맥경화학회 춘계학술대회

2026 Spring Congress on
Lipid and Atherosclerosis of KSoLA

SoLA 2026

한국지질·동맥경화학회 춘계학술대회

2026 Spring Congress on
Lipid and Atherosclerosis of KSoLA

Symposium 10 –
KSS–KSoLA Joint Symposium

Inflammation and Lipid Crosstalk in Carotid Atherosclerosis

4월 3일(금) 15:20–16:50 | Room 3 (그랜드볼룸 1)

좌장 : 김응규(인제의대 신경과), 서우근(성균관의대 신경과)

패널 : 권형민(서울의대 신경과), 우호걸(경희의대 신경과)
장정윤(경상의대 순환기내과), 조윤경(울산의대 내분비내과)

15:20–15:40	**Evaluating and treating atheroinflammation: translating vascular inflammation into clinical practice** **김정민**(서울의대 신경과)
15:40–16:00	**Carotid and cerebral hemodynamics: insights into endothelial dysfunction** **정슬기**(메디이미지)
16:00–16:20	**Systemic atherosclerosis and lipid control: role of carotid IMT and plaque** **김범준**(울산의대 신경과)
16:20–16:50	**Panel Discussion**

CURRICULUM VITAE

김정민

서울대학교병원 신경과

[학력]

1997-2003	서울대학교 의과대학 학사
2006-2008	서울대학교 의과대학 석사
2011-2013	서울대학교 의과대학 박사

[경력]

2008-2011	공중보건의 (공주치료감호소, 한국보건산업진흥원)
2011-2013	서울대학교병원 임상강사
2013-2020	중앙대학교 의과대학 부교수
2020-현재	서울대학교병원 교수

[관심분야]

뇌졸중, 죽상동맥경화, 혈관성 인지장애

[논문]

1. Kim JM, Lee R, Kim Y, Jeong HB, Seong Lee E, Ryoun Kim H, Park KY, Won Seok J. Impact of Metabolic Activity of Vertebra and Amygdala on Stroke Recurrence: A Prospective Cohort Study. Circ Cardiovasc Imaging. 2023;16:e014544.
2. Yang W, Hong SA, Kim JM, Jeong HB, Nam TK, Choi HH, Kim SM, Park KY, Kim HR. The immunologic phenotype of thrombi is associated with future vascular events after cerebral infarction. J Neurointerv Surg. 2023; jnis-2023-020155.
3. Kim JM, Moon J, Yu JS, Park DK, Jung KH. Therapeutic Target MicroRNA Identification Based on Circular RNA Expression Signature After Intracerebral Hemorrhage. Mol Neurobiol. 2024;61:908-918.
4. Kim SM, Hong SA, Kim JM. Association of immunologic findings of atheromatous plaques with subsequent cardiovascular events in patients with peripheral artery disease. Sci Rep. 2024 Jan 3;14(1):469. doi: 10.1038/s41598-023-50751-8.
5. Yang W, Kim JM, Chung M, Ha J, Kang DW, Lee EJ, Jeong HY, Jung KH, Sung H, Paeng JC, Lee SH. Sodium-Glucose Cotransporter 2 Inhibitor Improves Neurological Outcomes in Diabetic Patients With Acute Ischemic Stroke. J Stroke. 2024 May;26(2):342-346. doi: 10.5853/jos.2023.04056.

Evaluating and treating atheroinflammation: translating vascular inflammation into clinical practice

김정민

서울대학교병원 신경과

염증 반응은 죽상동맥경화증의 시작에서부터 진행에 이르기까지 전 주기에 걸쳐서 동반되는 병리 현상으로 특히 죽상동맥경화반의 파열에 따른 혈관 사건을 초래하는데 중요한 역할을 담당하는 것으로 알려져 있다. 본 강의에서는 염증 반응이 어떻게 죽상동맥경화증을 악화시켜서 혈관 사건을 초래하는 지 전신의 면역 세포 생산 및 공급이란 측면에서, 그리고 특정 혈관의 죽상동맥경화반 불안정성이란 측면에서 연구 결과를 정리하고자 한다. 다양한 최신 분석 기법과 영상 기법의 발전 및 적용을 통해서 죽상동맥경화반의 병태생리에 대한 이해와 그 헐성도 측정이 가능해지고 있다. 아울러 혈관 사건을 억제하기 위해 염증 반응을 제어하는 최신 치료 전략을 적용한 연구 결과를 소개하고 실제 임상 현장에서 적용 가능성을 살펴보고자 한다. 최신 연구 결과들을 토대로 염증 반응을 억제하는 치료 전략의 효과와 한계점에 대한 이해를 토대로 죽상동맥경화증의 진행 및 혈관 사건 발생 기전을 억제하는 치료 전략 연구에 도움이 되리라 예상한다.

CURRICULUM VITAE

정슬기(Seul-Ki Jeong)

Fonder, CEO, MediIMG, Inc

[Education and Training]

1994.02	Chonnam National University Hospital, M.D, Medicine
2004.02	Chonnam National University Hospital, Neurology

[Employment and Position]

2018-Presnet	MediIMG, Inc, CEO
2014-2016	Jeonbuk National University and Hospital, Professor
2009-2014	Jeonbuk National University and Hospital, Associate Professor
2008-2010	Drexel University Mechanical Engineering, Vising Professor

[Important Publications]

1. Lee CH, Jeong SK, Kim HJ, Rosenson RS, Yang W, Jung KH. Arteriopathic effects of hypertension by signal intensity gradient from time-of-flight magnetic resonance angiography. Clin Neuroradiol. 2025 doi: 10.1007/s00062-025-01594-5
2. Lee CH, Jeong SK, Kim HJ, Rosenson RS, Yang W, Jung KH. Association of carotid artery stenosis with cerebral artery signal intensity gradient on time-of-flight magnetic resonance angiography. Front Neurol. 2025;16:1576655.
3. Lee C-H, Lee SH, Kwak H-S, Rosenson RS, Jeong S-K, Jung K-H. Arterial flow volume measurement using signal intensity gradient versus phase contrast. Scientific Reports. 2025;15:44848.
4. Lee J, Jeong SK, Hong JM. Impact of a1 segment asymmetry on hemodynamic conditions around the circle of willis and anterior communicating artery aneurysm formation. Front Neurol. 2024;15:1491247.
5. Lee CH, Lee SH, Kwak HS, Kwak YG, Rosenson RS, Cho YI, Jeong SK. Validation of signal intensity gradient from tof-mra for wall shear stress by phase-contrast mr. J Imaging Inform Med. 2024;37:1248-1258.

[Awards and Honors]

1. 2013; The Best "Poster Award", World Congress of Neurology 2013, Austria
2. 2006; 'Young Researcher', The Korean Stroke Society 2006
3. 2006. 10; 'The Best Poster Award' 2006, The 5th Scientific Meeting of the Asian Chapter of Neurosonology Research Group of the World Federation of Neurology

[Research Interest]

Cerebrovascular disease, Endothelial dysfunction, Peripheral artery disease, Signal intensity gradient, Stroke, TOF-MRA.

Carotid and cerebral hemodynamics: insights into endothelial dysfunction

Seul-Ki Jeong

MediIMG, Inc, Seoul, Korea

Endothelial dysfunction represents a fundamental pathophysiological mechanism underlying cardiocerebrovascular diseases and is closely associated with abnormalities in hemodynamic shear stress. Under physiological conditions, laminar shear stress maintains endothelial homeostasis by promoting nitric oxide bioavailability, anti-inflammatory signaling, and vascular compliance. In contrast, disturbed or reduced shear stress induces pro-inflammatory, pro-thrombotic, and pro-atherogenic endothelial phenotypes. Clinically, endothelial dysfunction has been assessed using flow-mediated dilatation (FMD), which evaluates the vasodilatory response to transient increases in shear stress. However, FMD is limited to peripheral arteries and does not directly reflect intracranial hemodynamics.

Time-of-flight magnetic resonance angiography (TOF-MRA) enables non-invasive evaluation of cerebral arterial flow characteristics. Based on TOF-MRA, the arterial signal intensity gradient (SIG) has been introduced as a surrogate marker of shear stress or shear rate. Validation studies have demonstrated significant correlations between SIG and computational fluid dynamics (CFD)-derived as well as phase-contrast MRI-derived wall shear stress estimates. Clinically, reduced SIG has been associated with regional vulnerability to ischemic stroke, whereas heterogeneous or variable SIG patterns correlate with progression of arteriopathy in moyamoya disease. Moreover, a wide dispersion of SIG values has been reported to predict aneurysmal rupture risk, suggesting its relevance to local hemodynamic instability.

Low SIG has also been linked to systemic conditions such as hypertension and internal carotid artery (ICA) stenosis. In chronic hypertension, elevated arterial pressure may coexist with impaired flow velocity and altered shear profiles, potentially resulting in reduced SIG. Collectively, these findings support SIG as a promising imaging-based surrogate of endothelial dysfunction in carotid and cerebral circulation. Further mechanistic and longitudinal studies are warranted to clarify its biological significance and clinical utility.

CURRICULUM VITAE

김범준(Bum Joon Kim)

Department of Neurology, Asan Medical Center,
University of Ulsan College of Medicine, Seoul, Korea

[학력]

2005 M.D., Department of Medicine, Kyung Hee University, Seoul, Korea
2009 Master of Medicine, Department of Neurology, University of Ulsan College of Medicine, Seoul, Korea
2015 Ph.D. in Medicine, Department of Neurology, University of Ulsan College of Medicine, Seoul, Korea

[경력]

2015-2019 Assistant Professor, Department of Neurology, Kyung Hee University Hospital, Seoul, Korea
2019-2020 Associate Professor, Department of Neurology, Kyung Hee University Hospital, Seoul, Korea
2022-Present Associate Professor, Department of Neurology, Asan Medical Center, Seoul, Korea

[관심분야]

- Acute ischemic stroke
- Intracranial and extracranial atherosclerosis
- Cryptogenic stroke and atrial fibrillation detection
- Stroke imaging and cerebrovascular hemodynamics
- Secondary stroke prevention and antithrombotic therapy

[논문]

1. Genetic and imaging features of CADASIL patients with acute ischemic stroke. Sci Rep. 2025;15:17113.
2. SOLO-ESUS trial rationale and design. Cerebrovasc Dis. 2025.
3. Association between impaired brachial flow-mediated dilation and early neurological deterioration in acute ischemic stroke. BMC Neurol. 2025;25:47.
4. Factors Associated With Silent Brain Infarcts After MCA Stenting or Balloon Angioplasty. J Neuroimaging. 2025;35:e70018.
5. Factors associated with delayed neurologic improvement after complete endovascular reperfusion in anterior and posterior ischemic stroke. Front Neurol. 2025;16:1543743.

Systemic atherosclerosis and lipid control: role of carotid IMT and plaque

Bum Joon Kim

Department of Neurology, Asan Medical Center

Systemic atherosclerosis is a diffuse vascular disorder driven by lipid accumulation and chronic inflammation. Carotid intima-media thickness (IMT), measured by high-resolution ultrasonography, reflects generalized arterial wall remodeling and serves as a surrogate marker of systemic atherosclerotic burden. IMT is preferentially assessed in the common carotid artery (CCA), where laminar flow predominates and turbulence is minimal, enabling reproducible evaluation of lipid-mediated arterial thickening independent of focal plaque formation. Unlike carotid plaque, which represents localized, advanced atherosclerosis with features such as lipid-rich necrotic core or surface irregularity that increase embolic risk, IMT reflects diffuse, early-stage arterial changes associated with cumulative lipid exposure. Increased IMT correlates with cardiovascular risk and responds to intensive lipid-lowering therapy, supporting its role as a non-invasive marker for systemic atherosclerosis and monitoring vascular effects of lipid control.

SoLA 2026

한국지질·동맥경화학회 춘계학술대회

2026 Spring Congress on
Lipid and Atherosclerosis of KSoLA

SoLA 2026

한국지질·동맥경화학회 춘계학술대회

2026 Spring Congress on
Lipid and Atherosclerosis of KSoLA

Symposium 11

AI 시대의 학술 출판 혁신

4월 3일(금) 15:20-16:20 | Room 4 (볼룸 1,2)

좌장 : 이우제(울산의대 내분비내과), 이상엽(중앙의대 순환기내과)

패널 : 김현진(한양의대 심장내과), 안효석(가톨릭의대 순환기내과)
허지혜(한림의대 내분비내과)

15:20-15:40	**AI 도구를 활용한 고품질 Peer Review 전략과 Reviewer Workflow 혁신** **오규철**(가톨릭의대 순환기내과)
15:40-16:00	**AI 시대의 에디터 역할과 출판 품질관리의 미래: 위기와 기회의 변곡점에서** **유승찬**(연세의대 의생명시스템정보학교실)
16:00-16:20	**Panel Discussion**

CURRICULUM VITAE

오규철

가톨릭대학교 서울성모병원 순환기내과

[학력]

2001-2005	서울대학교 공과대학 기계항공공학부(학사)
2005-2009	서울대학교 의과대학 의학과(학사)
2011-2013	서울대학교 의과대학 임상의과학과(석사)
2013-2023	서울대학교 의과대학 내과학(박사)

[경력]

2009-2010	서울대학교병원 인턴
2010-2014	서울대학교병원 내과 전공의
2014-2017	대한민국 육군 군의관(대위)
2017-2020	서울대학교병원 순환기내과 임상강사
2020-현재	가톨릭대학교 서울성모병원 순환기내과 조교수

[관심분야]

이상지질혈증, 관상동맥질환, 중재시술

AI 도구를 활용한 고품질 Peer Review 전략과 Reviewer Workflow 혁신

Gyu Chul Oh

Cardiology, Seoul St. Mary's Hospital, South Korea

AI 시대에 peer review는 단순 평가를 넘어 구조화된 품질 관리 과정으로 진화하고 있다. 본 강의에서는 AI를 활용한 고품질 리뷰 전략과 workflow 혁신을 제시하며, 방법론 검증, 데이터 일관성 확인, 논리 오류 탐지 등 실제 적용 가능한 활용법을 소개한다. 또한 윤리적 고려사항과 reviewer의 새로운 역할을 함께 논의한다. 이 초록 역시 ChatGPT로 작성되었습니다.

CURRICULUM VITAE

유승찬

연세의료원 의생명시스템정보학교실 조교수

[학력]

2005-2011 연세대학교 의과대학 의학 (학사)
2013-2016 연세대학교 의과대학 의학 (석사)
2016-2021 아주대학교 의과대학 의료정보학 (박사)

[경력]

2022.3- 연세의료원 의생명시스템정보학교실 조교수

[관심분야]

Medical informatics medical big data, A.I., and Digital therapeutics

[논문]

1. Kim S, Kim D, ..., You SC†, Yang PS†, Joung B†. The optimal lookback period for estimating incidence and temporal trends in atrial fibrillation. Heart Rythm 22(12): e1115-e1124. 2025.
2. Lee S, ..., You SC†, Kim JH†. Shifts in emergency physicians' attitudes toward large language model-based documentation: a pre- and post-implementation study. Scientific Reports 15(1): 2045-2322. 2025.
3. Kim KJ, Boo D, ..., Oh JS†, You SC†. Comprehensive Evaluation of Treatment Patterns in Postmenopausal Patients with Osteoporosis without Fractures: Insights from Tertiary Care Institutions and Nationwide OMOP-CDM Data. Endocrinology and Metabolism 40(5): 737-747. 2025.
4. Song JW, Park J, Kim JH†, You SC†. Large Language Model Assistant for Emergency Department Discharge Documentation. JAMA Network Open 8(10): e253842. 2025.
5. Kim S, Han CH, ..., You SC†, Kim KW†. Comparative Risk for Neuropsychiatric Events in Leukotriene Receptor Antagonist vs. Inhaled Corticosteroid in Children With Asthma: A Nationwide Observational Study With a Complementary Analysis Using Natural Language. Processing. Pharmacoepidemiology and Drug Safety 34(11): e70254. 2025.

AI 시대의 에디터 역할과 출판 품질관리의 미래: 위기와 기회의 변곡점에서

유승찬

연세대학교 의생명시스템정보학교실

수백 년간 지속되어 온 전통적인 학술 출판 시스템은 현재 심각한 구조적 결함(Flawed)에 직면해 있습니다. 제출부터 출판까지 6개월에서 1년 이상 소요되는 느린 속도(Too Slow), 과도한 오픈 액세스 비용 부담(Too Expensive), 그리고 임팩트 팩터(IF)와 같은 특정 지표에만 매몰된 평가 방식(Too Focused on Metrics) 등은 현대 과학의 빠른 발전 속도를 저해하는 요소로 지적됩니다. 이러한 위기 상황 속에서 등장한 생성형 AI와 대형 언어 모델(LLM)은 언어 장벽을 낮추어 연구의 민주화를 실현할 기회를 제공하는 동시에, 환각(Hallucination), 데이터 조작, 비판적 사고의 저하라는 새로운 위험 요소를 학술 생태계에 던지고 있습니다.

본 발표에서는 먼저 LLM 도입 이후 변화된 논문 작성 트렌드를 실증적으로 분석합니다. 최근 연구에 따르면, AI 활용이 대중화된 2024년 이후 'Professional but concise (전문적이나 간결한)' 톤의 논문이 더 높은 게재 승인율을 보이는 경향이 관찰됩니다. 이어 세계적인 학술지들이 채택하고 있는 상이한 AI 편집 정책을 비교 고찰합니다. 원고 작성 시 AI 사용 공개를 의무화하지 않으며 과학적 소통을 위한 도구 활용을 장려하는 **JACC**의 유연한 입장부터, 투명한 보고와 저자의 전적인 책임을 강조하는 **JAMA** 및 **NEJM AI**의 사례를 통해 저자됨(Authorship)과 책임(Accountability)의 경계를 살펴봅니다.

특히 만성적인 심사자 부족과 심사자 피로도(Reviewer Fatigue) 문제를 해결하기 위한 실질적인 대안으로 'Human-in-the-Loop' 기반의 AI 보조 심사 모델을 제안합니다. AI가 보고 가이드라인 준수 여부 검토나 통계 분석 계획 일치성 확인 등 단순 반복적인 품질 관리(Rote tasks)를 담당하고, 인간 에디터는 연구의 독창성, 임상적 중요성, 윤리적 가치 판단에 집중하는 효율적인 협업 구조의 가능성을 제시합니다. 또한 **Open Evidence**와 같은 AI 기반 증거 합성 플랫폼이 기존의 동료 심사 저널 시스템과 어떻게 상호작용하며 미래의 학술 정보 유통 구조를 재편할 수 있을지 논의합니다.

결국 AI의 도입은 단순한 도구의 변화를 넘어, 기존 출판 시스템의 근본적인 결함을 수선하고 새로운 학술적 가치를 형성해야 하는 과제를 우리에게 안겨주었습니다. AI가 인간의 지적 역량을 보조하는 '지혜로운 동료(Co-PI)'로 자리 잡을 수 있을지, 혹은 인간의 비판적 사고를 침식하는 위협이 될지는 결국 우리의 선택에 달려 있습니다.

SoLA 2026

한국지질·동맥경화학회 춘계학술대회

2026 Spring Congress on
Lipid and Atherosclerosis of KSoLA

SoLA 2026

한국지질·동맥경화학회 춘계학술대회

2026 Spring Congress on
Lipid and Atherosclerosis of KSoLA

Symposium 12

Current and Future Perspectives on Lipid-lowering Agents

4월 4일(토) 08:30-10:00 | Room 1 (그랜드볼룸 3)

좌장 : 채인호(서울의대 순환기내과), 권혁상(가톨릭의대 내분비내과)

패널 : 김경안(가톨릭의대 순환기내과), 이지은(고려의대 순환기내과)
황유철(경희의대 내분비내과)

08:30-08:48	**Bempedoic acid: a non-statin option for LDL-C lowering** **차정준**(고려의대 순환기내과)
08:48-09:06	**The evolving landscape of PCSK9 inhibitors: siRNA, oral agents, and mAbs** **조경훈**(전남의대 순환기내과)
09:06-09:24	**Lp(a)-lowering therapies: olpasiran and other emerging agents** **최성훈**(한림의대 순환기내과)
09:24-09:42	**CETP inhibitors: revisiting an old target with new molecules** **홍준화**(을지의대 내분비내과)
09:42-10:00	**Panel Discussion**

CURRICULUM VITAE

차정준(Jung-Joon Cha)

고려의대 순환기내과

[Education]

- Doctor of Philosophy, Gwangju Institute of Science and Technology Thesis: Study on Aging Process of Cardiovascular Cells Using a Micro-Electrochemical Impedance Spectroscopy
- Master's Degree, Yonsei University College of Medicine Thesis: The association between cardiac involvement and long-term clinical outcomes in patients with Duchenne muscular dystrophy
- M.D., Yonsei University Wonju College of Medicine

[Licensure and Certification]

2021	Fellowship of ESC, European Society of Cardiology
2021	Certified Interventional Cardiologist, Korean Society of Interventional Cardiology
2020	License of Cardiologist, Korean Board of Internal Medicine
2014	License of Internal Medicine, Korean Board of Internal Medicine
2009	License of Medical Doctor (101776), Republic of Korea

[Graduate Training]

Yonsei University Severance Hospital

2019-2020	Interventional Fellowship in Cardiology
2018-2019	General Fellowship in Cardiology

Yonsei University Gangnam Severance Hospital

2010-2014	Residency in Internal Medicine
2009-2010	Internship

[Professional Experience]

Korea University Anam Hospital, Korea University College of Medicine

2024-	Clinical Associate Professor
2020-2024	Clinical Assistant Professor

Bempedoic acid: a non-statin option for LDL-C lowering

Jung-Joon Cha

Interventional Cardiology, Korea University Anam Hospital

Achieving guideline-recommended low-density lipoprotein cholesterol (LDL-C) targets remains challenging in routine practice, particularly among patients who are unable to tolerate statins or who require additional LDL-C reduction despite maximally tolerated therapy. Bempedoic acid is an oral, first-in-class inhibitor of adenosine triphosphate-citrate lyase (ACL), acting upstream of HMG-CoA reductase in the hepatic cholesterol synthesis pathway. As a prodrug primarily activated in the liver (with minimal activation in skeletal muscle), it offers a mechanistic rationale for LDL-C lowering with a relatively low incidence of muscle-related adverse effects, making it a practical option for patients with documented or perceived statin intolerance.

Clinical trials have demonstrated consistent, moderate LDL-C reductions with bempedoic acid, with favorable effects on inflammatory markers such as high-sensitivity C-reactive protein (hsCRP). When used in appropriately selected high-risk patients who cannot take recommended statin therapy, bempedoic acid has also been associated with reductions in major cardiovascular events, supporting its role as an outcomes-supported, oral non-statin strategy within contemporary lipid-lowering algorithms. Combination approaches (e.g., with ezetimibe) can further enhance LDL-C lowering and may be especially useful when an oral regimen is preferred.

Bempedoic acid is generally well tolerated; however, clinicians should monitor for adverse events that appear more frequent than placebo, including hyperuricemia/gout and cholelithiasis, and consider individual risk factors when selecting therapy. Overall, bempedoic acid broadens the therapeutic armamentarium for LDL-C reduction, particularly for statin-intolerant patients or those requiring additional oral non-statin intensification.

CURRICULUM VITAE

조경훈

전남대학교병원

[학력]

2006	전남대학교 의과대학 학사
2009	전남대학교 의과대학 석사
2020	전남대학교 의과대학 박사

[경력]

2006.3-2007.2	전남대학교병원 인턴
2007.3-2011.2	전남대학교병원 레지던트
2015.4-2019.2	전라남도 강진의료원 내과장
2020.3-2023.2	전남대학교병원 임상교수
2024.7-2025.12	호주 모나시대학교 빅토리안 심장 연구소 방문연구원
2023.2-현재	전남대학교 의과대학 부교수

[관심분야]

급성심근경색증, 이상지질혈증, 인공지능의료기술

[논문]

1. 2026 Physician Disagreement With Guidelines as a Critical Barrier to Achieving LDL-C Targets in Very High-Risk Patients: An Implementation Science Study. Cho KH, Cho YR, Lee SR, et al. J Korean Med Sci. 2026 Mar 2;41(8):e74. doi: 10.3346/jkms.2026.41.e74.
2. 2026 Prognostic Value of BARC-Defined Bleeding in East Asian Acute Myocardial Infarction Patients: Evidence From Multicentre Registries in Korea and Japan. Honda S, Cho KH, Jeong MH, Yasuda S. et al. Thrombosis and Haemostasis. 2026 Jan 6. doi: 10.1055/a-2773-5644.
3. 2025 Long-term outcomes according to absolute value vs. percentage reduction in low-density lipoprotein cholesterol levels after acute myocardial infarction. Cho KH. Yang JH, Jeong MH, Kim W. et al. Front Cardiovasc Med. 2025. https://doi.org/10.3389/fcvm.2025.1653447.
4. 2025 Novel Artificial Intelligence Model Using Electrocardiogram for Detecting Acute Myocardial Infarction Needing Revascularization. Cho KH, Ji YH, Jeong MH. et al. Eur Heart J Digit Health. 2025. https://doi.org/10.1093/ehjdh/ztaf049.
5. 2025 Time-Point Clinical Outcomes in Patients with Acute Myocardial Infarction: One Step for Personalized Medicine. Cho KH, Jeong MH; Korea Acute Myocardial Infarction Registry (KAMIR)-National Institutes of Health (NIH) Investigators. Am J Cardiol. 2025 Feb 1:236:64-71.

The evolving landscape of PCSK9 inhibitors: siRNA, oral agents, and mAbs

Kyung Hoon Cho

Department of Cardiology, Chonnam National University Hospital and Medical School, Gwangju, Republic of Korea

Atherosclerotic cardiovascular disease (ASCVD) remains a leading cause of global mortality, with elevated low-density lipoprotein cholesterol (LDL-C) identified as a primary modifiable risk factor. Proprotein convertase subtilisin/kexin type 9 (PCSK9) is a key regulator of lipid metabolism that facilitates the degradation of LDL receptors, thereby limiting the liver's ability to clear LDL-C from circulation. This lecture examines the therapeutic evolution of PCSK9 inhibition, detailing the transition from established injectable therapies to novel oral agents.

The first wave of innovation centers on monoclonal antibodies (mAbs), such as alirocumab and evolocumab. These agents bind directly to circulating PCSK9, delivering a 50%-60% reduction in LDL-C and demonstrating significant cardiovascular risk reduction in large-scale clinical trials. However, their requirement for frequent subcutaneous administration—typically every two to four weeks—presents a barrier for some patients. Small interfering RNA (siRNA) therapy, specifically inclisiran, addresses this through a distinct mechanism that inhibits the hepatic synthesis of PCSK9. Inclisiran offers a simplified twice-yearly dosing schedule following an initial loading phase, maintaining approximately 50% LDL-C lowering.

The landscape is further expanding with the development of oral PCSK9 inhibitors, such as MK-0616. These emerging small-molecule therapies aim to provide the efficacy of injectables with the convenience of daily oral dosing, potentially improving patient adherence and lowering costs. Early clinical data show that oral agents can achieve significant, dose-dependent LDL-C reductions. This session provides a comprehensive overview of these diverse modalities, highlighting their efficacy, safety, and evolving roles in the clinical management of high-risk patients.

CURRICULUM VITAE

최성훈

한림대학교 강남성심병원

[학력]

1997	연세대학교 의학과

[경력]

2002	세브란스병원 내과 전공의
2005	세브란스 심장혈관병원 심장내과 전임의
2006-현재	한림대학교 강남성심병원 순환기내과 교수
2015	Harvard BIDMC Visiting Scholarship

[관심분야]

Inflammation, LDL related secondary prevention

[논문]

1. Spironolactone vs Amiloride for Resistant Hypertension: A Randomized Clinical Trial. JAMA. 2025 Jun 17;333(23):2073-2082.
2. Moderate-Intensity Statin With Ezetimibe Combination Therapy vs High-Intensity Statin Monotherapy in Patients at Very High Risk of Atherosclerotic Cardiovascular Disease: A Post Hoc Analysis From the RACING Randomized Clinical Trial JAMA Cardiol. 2023 Sep 1;8(9):853-858.
3. Lifestyle Modification in the Management of Metabolic Syndrome: Statement From Korean Society of CardioMetabolic Syndrome (KSCMS) Korean Circ J. 2022 Feb;52(2):93-109.
4. The Potential Role of Biomarkers Associated with ASCVD Risk: Risk-Enhancing BiomarkersJ Lipid Atheroscler. 2019 Sep;8(2):173-182.
5. Association between Low-Density Lipoprotein Cholesterol Level and Cardiovascular Outcomes in Korean Adults: A Nationwide Cohort StudyDiabetes Metab J. 2023 Jan;47(1):59-71.

Lp(a)-lowering therapies: olpasiran and other emerging agents

Seonghoon Choi

Cardiology, Kangnam Sacred Heart Hospital, Hallym University, Republic of Korrea

Lipoprotein(a), or Lp(a), is a genetically determined lipid particle that is highly pro-atherogenic and pro-thrombotic. Although PSCK9 mAb may lower some LP(a) level, traditional therapies like statins have negligible effects on it, leading to a surge in the development of targeted RNA-based and small-molecule therapies. Among these, olpasiran is a leading candidate in late-stage clinical development, alongside several other innovative agents. Developed by Amgen, olpasiran is a small interfering RNA (siRNA) designed to silence the LPA gene in the liver, preventing the production of apolipoprotein(a). olpisiran uses the RNA-induced silencing complex (RISC) to degrade LPA messenger RNA, which effectively shuts down the "assembly line" for Lp(a) particles. OCEAN(a)-DOSE trial showed clinical efficacy. In Phase 2 trials, doses of 75 mg or higher administered every 12 weeks reduced Lp(a) levels by over 95%. The effects are exceptionally long-lasting effect. Een a year after the last dose, patients maintained a 40-50% reduction. Generally well-tolerated, with the most common side effect being mild injection-site reactions. Current Status: It is currently being evaluated in the OCEAN(a)-Outcomes Phase 3 trial (enrolling ~7,000-8,000 patients), which will determine if this massive reduction in Lp(a) translates to fewer heart attacks and strokes.9 Results are anticipated around 2026/2027. Other developing agents are listed below.

Agent	Class	Status	Highlights
Pelacarsen	Antisense Oligonucleotide (ASO)	Phase 3	The furthest along in development. Administered monthly; results from the **Lp(a) HORIZON** trial are expected in **early 2026**.
Lepodisiran	siRNA	Phase 3	Notable for its extreme durability. Phase 2 data showed a single dose could keep Lp(a) levels reduced by **94%** for nearly a year.
Muvalaplin	Small Molecule (Oral)	Phase 2/3	The first **oral** agent for Lp(a). Unlike the others, it blocks the physical assembly of the Lp(a) particle rather than silencing a gene.
Zerlasiran	siRNA	Phase 2	Another potent siRNA (by Silence Therapeutics) that has shown $> 90\%$ reduction in early clinical data.

CURRICULUM VITAE

홍준화

대전 을지대학교병원

[학력]

2004	을지의대 학사
2008	을지의대 학사
2015	을지의대 내과 박사

[경력]

2014	충남대학교병원 전임의
2016	경북대학교병원 임상교수
현재	대전을지대학교병원 부교수

[관심분야]

비만, 당뇨병, 이상지질혈증, 갑상선, 골다공증, 부신

[논문]

1. Efficacy and safety of adding a fourth oral antidiabetic drug versus metformin dose escalation in patients with type 2 diabetes inadequately controlled on triple oral combination therapy (EFFORT): A 24-week, randomized, open-label, multicenter trial. Diabetes Obes Metab. 2026;1-12.
2. Letter: Impact of Remnant Cholesterol on the Risk for End-Stage Renal Disease in Type 2 Diabetes Mellitus: A Nationwide Population-Based Cohort Study (Diabetes Metab J 2025;49:1106-15). Diabetes Metab J 2026;50:190-191.
3. Efficacy and Safety of HD-6277, a Novel G Protein-Coupled Receptor 40 Agonist, in Individuals with Type 2 Diabetes Mellitus: A Double-Blind, Randomized, Placebo-Controlled, Parallel-Group, Multicenter Phase 2 Clinical Trial. Diabetes Metab J. 2025 Dec 19. doi: 10.4093/dmj.2025.0528.
4. Efficacy and Safety of Enavogliflozin as Add-on in Adults with Type 2 Diabetes Mellitus Inadequately Controlled with Insulin or Insulin with Other Antidiabetic Drugs. Diabetes Metab J. 2025 Dec 15. doi: 10.4093/dmj.2025.0477.
5. Collaborators: Coadministered Cagrilintide and Semaglutide in Adults with Overweight or Obesity. N Engl J Med. 2025 Aug 14;393(7):635-647. doi: 10.1056/NEJMoa2502081. Epub 2025 Jun 22.

CETP inhibitors: revisiting an old target with new molecules

Jun Hwa Hong

Department of Internal Medicine, Daejeon Eulji Medical Center, Eulji University, Daejeon, Republic of Korea

Cholesteryl ester transfer protein (CETP) inhibitors have long been investigated as a therapeutic strategy for modifying lipid profiles, primarily through increasing high-density lipoprotein cholesterol (HDL-C) and reducing low-density lipoprotein cholesterol (LDL-C). Despite early enthusiasm, first-generation CETP inhibitors such as torcetrapib and dalcetrapib failed to demonstrate cardiovascular benefit, largely due to off-target effects and insufficient efficacy. These setbacks led to skepticism regarding CETP as a viable therapeutic target.

However, renewed interest has emerged with the development of next-generation CETP inhibitors characterized by improved potency, selectivity, and safety profiles. Agents such as anacetrapib and evacetrapib demonstrated substantial lipid-modifying effects, although clinical outcomes have been mixed. More recently, novel compounds like obicetrapib have shown promising results in significantly lowering LDL-C and lipoprotein(a), while maintaining favorable tolerability.

This presentation will revisit the biological role of CETP in lipoprotein metabolism and critically evaluate past clinical trial failures to identify key limitations. It will further highlight emerging evidence supporting the cardiovascular benefits of newer CETP inhibitors, including their potential role in addressing residual cardiovascular risk beyond statin therapy. Finally, we will discuss future directions, including combination strategies and patient populations most likely to benefit from CETP inhibition.

Revisiting CETP inhibition with refined pharmacologic approaches may ultimately reshape its place in lipid-lowering therapy and cardiovascular risk reduction.

SoLA 2026

한국지질·동맥경화학회 춘계학술대회

2026 Spring Congress on
Lipid and Atherosclerosis of KSoLA

SoLA 2026

한국지질·동맥경화학회 춘계학술대회

2026 Spring Congress on
Lipid and Atherosclerosis of KSoLA

Symposium 13

AI & Digital Health Technology for Atherosclerosis

4월 4일(토) 08:30-10:00 | Room 2 (그랜드볼룸 2)

좌장 : 강석민(연세의대 심장내과), 김병극(연세의대 심장내과)

패널 : 김성은(한림의대 소화기내과), 배성아(연세의대 심장내과)
안지현(한국의학연구소 내과)

08:30-08:50	**Updated digital technology for atherosclerosis management** **조준환**(중앙의대 순환기내과)
08:50-09:10	**Multi-modality intracoronary imaging for characterizing atherosclerosis** **강동오**(고려의대 순환기내과)
09:10-09:30	**AI for cardiovascular image in CT** **양동현**(울산의대 영상의학과)
09:30-10:00	**Panel Discussion**

CURRICULUM VITAE

조준환

중앙대학교광명병원

[학력]

2003.3-2009.2 중앙대학교 의학부 학사
2010.9-2012.8 중앙대학교 의학부 석사
2020.9-2022.8 중앙대학교 의학부 박사수료

[경력]

2019.3-2022.2 중앙대학교병원 순환기내과 조교수
2022.3-2024.2 중앙대학교광명병원 순환기내과 조교수
2024.3- 중앙대학교광명병원 순환기내과 부교수

[관심분야]

관상동맥질환, 웨어러블 디바이스, AI

[논문]

1. Prediction of Vasovagal syncope using Artificial Intelligence-enabled Smartwatch Photoplethysmography-derived Heart Rate Variability
2. Residual Cardiovascular Biomarkers After Medical Therapy and Their Prognostic Implications Following Percutaneous Coronary Intervention
3. Smoking Cessation and Incident Cardiovascular Disease
4. Temporal trends in adherence to lifestyle recommendation of patients with hypertension in Korea, 2007-2021
5. Machine Learning Model Using Heart Rate Variability for the prediction of Vasovagal syncope

Updated digital technology for atherosclerosis management

Jun Hwan Cho

Chung-Ang University Gwangmyeong Hospital

죽상경화증 관리는 전통적으로 혈중 지질 수치, 임상 위험인자, 그리고 협착 정도를 중심으로 이루어져 왔으나, 최근에는 디지털 기술의 발전에 따라 위험 예측, 질환 표현형 분석, 치료 순응도 향상, 장기 추적관리의 전 과정이 빠르게 변화하고 있다. 특히 인공지능 기반 영상 분석, 웨어러블 기기, 원격 환자 모니터링, 모바일 헬스, 디지털 치료적 접근은 죽상경화증을 보다 정밀하고 연속적으로 관리할 수 있는 기반을 제공하고 있다.

본 강의에서는 죽상경화증 관리에서 활용 가능한 최신 디지털 기술의 현재와 미래를 정리하고자 한다. 첫째, AI 기반 coronary artery calcium 분석과 coronary CT angiography plaque quantification은 단순 협착 평가를 넘어 전체 plaque burden, non-calcified plaque, high-risk plaque phenotype을 정량화함으로써 보다 정교한 위험도 재분류와 예방전략 수립을 가능하게 한다. 둘째, 웨어러블 디바이스와 가정 기반 생체신호 측정은 혈압, 심박수, 신체활동, 수면, 생활습관 등의 연속적 데이터를 제공하여 죽상경화증의 주요 조절 인자를 일상생활 수준에서 추적할 수 있게 한다. 셋째, 모바일 헬스 및 디지털 플랫폼은 약물 복약 순응도, 생활습관 교정, 환자 교육, 퇴원 후 추적관리를 강화함으로써 예방치료의 지속성을 높일 수 있다. 넷째, 대규모 임상데이터와 전자의무기록, 영상, 웨어러블 데이터를 통합하는 AI 기반 의사결정 지원 시스템은 향후 개인 맞춤형 죽상경화증 관리의 핵심 도구가 될 가능성이 있다.

그러나 이러한 기술의 실제 임상 적용을 위해서는 데이터 표준화, 알고리즘의 재현성과 외부 검증, 해석 가능성, 개인정보 보호, 진료 워크플로우 통합, 보험 및 규제 체계 정비가 필수적이다. 결론적으로, 최신 디지털 기술은 죽상경화증 관리에서 단순한 보조 수단이 아니라, 위험도 평가에서 치료 강화, 장기 추적 및 환자 참여에 이르기까지 관리 패러다임 자체를 변화시키는 핵심 요소로 자리잡고 있다. 향후 죽상경화증 진료는 디지털 기술을 기반으로 한 보다 정밀하고 지속적인 예방의학적 접근으로 발전할 것이다.

CURRICULUM VITAE

강동오(Dong Oh Kang)

고려대학교 구로병원 순환기내과 (Cardiovascular Center, Korea University Guro Hospital)

[학력]

2016.03-2020.02 고려대학교 대학원 의학과 내과학 (순환기학) 박사
2013.03-2015.02 고려대학교 대학원 의학과 내과학 (순환기학) 석사
2004.03-2010.02 고려대학교 의과대학 의학과 학사

[경력]

2024.03-현재 고려대학교 의료원 구로병원 순환기내과 부교수 (現)
2022.09-2024.02 고려대학교 의료원 구로병원 순환기내과 조교수
2020.03-2022.08 고려대학교 의료원 구로병원 순환기내과 임상조교수
2018.05-2020.02 고려대학교 의료원 구로병원 순환기내과 임상강사
2017.05-2018.04 국군의무사령부 국군수도병원 순환기내과 군의관
2010.03-2015.02 고려대학교 의료원 안암병원 수련의 및 내과 전공의

[관심분야]

1) 관상동맥 혈관내 영상 검사 기반 고위험 동맥경화반 조기 정밀 진단
2) 관상동맥 약물풍선 확장술 기반 고위험 동맥경화반 안정화 치료
3) 동맥경화성 심혈관질환의 선행 위험 인자 및 병태생리학적 기전 규명

[논문]

1. Kim S, Nam HS, Kang DO et al. Intracoronary structural-molecular imaging for multi-targeted characterization of high-risk plaque. JAMA Cardiol. 2025;10(7):708-717. (Role: first author)
2. Kang DO et al. Predictors of optimal angiographic lesion outcomes in drug-coated balloon treatment for de novo coronary artery disease. Sci Rep. 2025;15(1):9391. (Role: first author)
3. Lee D-I, Kim S, Kang DO. Exploring the complex interplay between alcohol consumption and cardiovascular health: Mechanisms, evidence, and future directions. Trends Cardiovasc Med. 2025 May;35(4):243-253. (Role: corresponding author)
4. Kang DO et al. Reduced Alcohol Consumption and Major Adverse Cardiovascular Events Among Individuals with Previous Heavy Alcohol Consumption. JAMA Network Open. 2024;7(3):e244013. (Role: first author)
5. Kang DO et al. Stress-associated neurobiological activity is linked with acute plaque instability via enhanced macrophage activity: a prospective serial 18F-FDG-PET/CT imaging assessment. Eur Heart J. 2021;42(19):1883-1895. (Role: first author)
6. Kang DO et al. Cardiovascular and Bleeding Risks Associated With Nonsteroidal Anti-Inflammatory Drugs After Myocardial Infarction. J Am Coll Cardiol. 2020;76(5):518-529. (Role: first author)

Multi-modality intracoronary imaging for characterizing atherosclerosis

Dong Oh Kang

Cardiovascular Center, Korea University Guro Hospital, Republic of Korea

Atherosclerotic plaque vulnerability is determined not only by luminal stenosis but also by complex biological and compositional features, including lipid accumulation, macrophage infiltration, and microcalcification. Accurate identification of high-risk plaques remains a major unmet need in contemporary cardiovascular care. Conventional intravascular imaging modalities, such as intravascular ultrasound (IVUS) and optical coherence tomography (OCT), provide high-resolution structural information. While IVUS offers deeper tissue penetration, OCT enables detailed visualization of superficial plaque morphology. However, both modalities are limited in their ability to fully characterize plaque biology. To address these limitations, multi-modal imaging approaches have been introduced. Techniques such as near-infrared spectroscopy (NIRS)-IVUS and near-infrared autofluorescence (NIRAF)-OCT provide additional information on lipid content and high-risk plaque features. Nevertheless, their ability to comprehensively assess diverse plaque components remains limited.

Fluorescence lifetime imaging (FLIm) is a novel label-free imaging technique that characterizes the intrinsic biochemical properties of atherosclerotic tissue by measuring fluorescence decay signals. Distinct plaque components generate unique fluorescence lifetime signatures, enabling compositional differentiation beyond conventional structural imaging. The integration of FLIm with OCT (FLIm-OCT) allows simultaneous acquisition of high-resolution morphological and bio-compositional information within a single catheter platform. Recent advances have enabled the development of a low-profile, high-speed dual-modal OCT-FLIm system capable of real-time in vivo imaging. Early studies have demonstrated its feasibility and potential for identifying inflammatory and lipid-rich plaques associated with vulnerability. Despite these advances, challenges remain for clinical translation, including technical complexity and data processing requirements. Integration with artificial intelligence-based analytic platforms may further enhance its clinical applicability and enable personalized risk stratification. Taken together, multi-modal intravascular imaging, particularly OCT-FLIm, represents a promising approach for comprehensive plaque characterization and may improve risk assessment and therapeutic decision-making in coronary artery disease.

CURRICULUM VITAE

양동현

울산의대 서울아산병원 영상의학과

[학력]

2008.03-2013.02 울산대학교 영상의학 박사
2002.03-2004.02 울산대학교 의학 석사
1994.03-2000.02 부산대학교 의학 학사

[경력]

2025.01-현재 서울아산병원 빅데이터연구센터 소장, 디지털정보혁신본부 부본부장
2023.03-현재 울산의대 서울아산병원 영상의학과 교수
2017.09-현재 서울아산병원 심장병원 심장혈관영상센터장

[관심분야]

Cardiac CT, 4D flow MRI, Deep learning

[논문]

1. Longitudinal observation of left ventricular inflow reorientation with preserved vorticity after myocardial infarction in a porcine model
2. 4D flow MRI of aortic blood flow parameters in healthy volunteers: Sex- and age-specific analysis
3. Deep Learning Based Automatic Segmentation of the Thoracic Aorta from Chest Computed Tomography in Healthy Korean Adults
4. Artificial Intelligence-Driven Assessment of Coronary Computed Tomography Angiography for Intermediate Stenosis: Comparison With Quantitative Coronary Angiography and Fractional Flow Reserve
5. Flow-Rate-Constrained Physics-Informed Neural Networks for Flow Field Error Correction in Four-Dimensional Flow Magnetic Resonance Imaging

AI for cardiovascular image in CT

Dong Hyun Yang

Radiology, Asan Medical Center, Korea

Artificial intelligence (AI) has rapidly emerged as a transformative technology in cardiovascular imaging, particularly in cardiac computed tomography (CT). This presentation reviews the current landscape of AI applications in cardiac CT, highlighting both technological developments and their clinical implications. Recent evidence indicates that cardiovascular imaging represents one of the most economically successful areas of medical AI implementation, with several AI-driven analysis platforms already incorporated into clinical workflows.

The discussion first outlines the growing role of AI in the entire cardiac CT workflow, ranging from patient selection and image acquisition to automated image analysis, risk stratification, and treatment planning. Advances in deep learning have enabled accurate segmentation and quantification of cardiovascular structures, including epicardial adipose tissue, aortic anatomy, and coronary plaques. These automated measurements allow extraction of quantitative biomarkers from routine CT images, facilitating improved cardiovascular risk prediction and potentially enabling more personalized management strategies.

Several representative applications are highlighted. AI-based epicardial adipose tissue quantification has demonstrated strong associations with cardiovascular outcomes and can enhance risk stratification beyond conventional imaging metrics. Similarly, automated pre-procedural assessment for structural heart interventions such as transcatheter aortic valve replacement (TAVR) has been developed to reduce interobserver variability and improve efficiency. Recent studies have also shown that AI-driven plaque characterization and coronary stenosis quantification can achieve diagnostic performance comparable to expert readers when validated against invasive coronary angiography.

In addition to research developments, commercially available platforms such as HeartFlow and Cleerly illustrate how AI-based quantitative coronary analysis is transitioning into real-world clinical practice. These technologies enable automated plaque quantification, ischemia prediction, and comprehensive coronary disease assessment, supporting more data-driven clinical decision making.

In conclusion, AI applications in cardiac CT are advancing rapidly and are likely to reshape the paradigm of cardiovascular imaging from qualitative interpretation toward comprehensive quantitative analysis. Continued collaboration among clinicians, researchers, industry partners, and academic societies will be essential to fully realize the clinical potential of AI-driven cardiac CT.

SoLA 2026

한국지질·동맥경화학회 춘계학술대회

2026 Spring Congress on
Lipid and Atherosclerosis of KSoLA

SoLA 2026

한국지질·동맥경화학회 춘계학술대회

2026 Spring Congress on
Lipid and Atherosclerosis of KSoLA

Symposium 14 –
KSCMS–KSoLA Joint Symposium

Incretin Therapies and Circadian Biology for Cardiometabolic Protection

4월 4일(토) 08:30–10:00 | Room 3 (그랜드볼룸 1)

좌장 : 김재택(중앙의대 내분비내과), 위진(가천의대 심장내과)

패널 : 김경희(인천세종병원 심장내과), 조은정(중앙의대 순환기내과)
홍상모(한양의대 내분비내과)

08:30–08:48	**Circadian rhythm and diabetes/metabolism** **김원진**(차의대 내분비내과)
08:48–09:06	**Incretin signaling and circadian regulation of glucose and energy metabolism** **손장원**(가톨릭의대 내분비내과)
09:06–09:24	**GLP–1/GIP agonists for cardiometabolic protection – one drug fits all?** **정창희**(울산의대 내분비내과)
09:24–09:42	**Safety and sustainability concerns – beyond the GLP–1RA hype** **서미혜**(순천향의대 내분비대사내과)
09:42–10:00	**Panel Discussion**

CURRICULUM VITAE

김원진

차의과학대학교 강남차병원 내분비내과

[학력]

2008	연세대학교 원주의과대학 학사
2012	연세대학교 의과대학 석사
2023	연세대학교 의과대학 박사

[경력]

2008-2013	연세대학교 신촌세브란스병원 인턴/레지던트(내과)
2013-2015	연세대학교 신촌세브란스병원 내분비내과 임상연구조교수
2015-현재	차의과학대학교 강남차병원 내분비내과 부교수

[관심분야]

당뇨병, 고지혈증, 갑상선, 뇌하수체

[논문]

1. W Kim, MK Seo, YJ Kim, SH Choi, CR Ku, S Kim, EJ Lee, JS Yoon. Role of the suppressor of cytokine signaling-3 in the pathogenesis of Graves' orbitopathy. Front. Endocrinol., 04 March 2025. https://doi.org/10.3389/fendo.2025.1527275
2. KH Chun, HJ Kim, DR Kang, JY Kim, W Kim, YW Jeong, SH Han, KK Koh. Sex-Specific Impact of the COVID-19 Outbreak on the Incidence of Metabolic Syndrome: A Comparative Study of 2018-2019 and 2020-2021. Korean J Intern Med 2025 Mar;40(2):262-274. doi: 10.3904/kjim.2024.288. Epub 2025 Mar 1.
3. HJ Kim, DR Kang, JY Kim, W Kim, YW Jeong, KH Chun, SH Han, KK Koh. Metabolic Syndrome Fact Sheet 2024: Executive Report. Cardiometab Syndr J. 2024;4:e14.
4. W Kim, SK Park, YL Kim. Fetal abdominal obesity in women with one value abnormality on diagnostic test for gestational diabetes mellitus. PLOS ONE 2024, 19(6): e0304875. https://doi.org/10.1371/journal.pone.0304875.
5. W Kim, SK Park, YL Kim. Fetal abdominal obesity and the ensuing adverse perinatal outcomes in older obese pregnant women with or without obesity and with normal glucose tolerance. Scientific Reports. 2023, 13: 16206.

Circadian rhythm and diabetes/metabolism

Wonjin Kim

Division of Endocrinology and Metabolism, Department of Internal Medicine, CHA Gangnam Medical Center, CHA University

Disruption of the circadian timing system has emerged as a key contributor to metabolic disorders, including type 2 diabetes. The circadian clock, coordinated by central (suprachiasmatic nucleus) and peripheral oscillators in metabolic organs, regulates glucose homeostasis through time-dependent modulation of insulin secretion, insulin sensitivity, hepatic glucose production, adipokine release, and mitochondrial function. Experimental models demonstrate that genetic or behavioral circadian misalignment impairs glucose-stimulated insulin secretion and reduces whole-body insulin sensitivity by 20–30%. Human studies further show that late-night eating, irregular sleep patterns, and shift-work are associated with increased glycemic variability and higher diabetes risk.

Behavioral factors that alter circadian alignment—such as meal timing, sleep duration, and chronotype—directly influence metabolic regulation. Early time-restricted eating (eTRE) has been shown to improve insulin sensitivity and lower fasting glucose even in the absence of weight loss. Conversely, evening chronotypes and night-shift workers exhibit impaired glucose tolerance and increased cardiometabolic risk. While pharmacologic chronotherapy in diabetes remains exploratory, optimizing behavioral timing—particularly daytime-concentrated caloric intake, consistent sleep schedules, and individualized strategies for shift workers—offers clinically meaningful benefits.

Understanding circadian biology provides a valuable framework for improving metabolic outcomes beyond traditional pharmacologic approaches. Integrating “time-based” lifestyle interventions into diabetes management may represent an important step toward more personalized and physiologically aligned care.

CURRICULUM VITAE

손장원

가톨릭대학교 부천성모병원 내분비내과

[학력]

1995-2001 중앙대학교 의과대학

[경력]

2017-2018 Visiting researcher, Karolinska institute, Sweden
2021.3-Present 가톨릭대학교 부천성모병원 내분비내과 교수

[관심분야]

당뇨병, 비만, 근육 생리학, 장내미생물

[논문]

1. Son JW, le Roux CW, Blüher M, Nauck MA, Lim S. Novel GLP-1-based Medications for Type 2 Diabetes and Obesity. Endocr Rev. 2026 Mar 11;47(2):159-177.
2. Jang E, Jeong S, Kim J, Jung S, Kim JY, Lee JE, Park S, Son JW; GOMS study group, Korean Society for the Study of Obesity. Comparison of nutrients and ultra-processed food consumption between different phenotypes defined by abdominal obesity and sarcopenia. Front Nutr. 2026 Jan 19;12:1683196. (Corresponding author)
3. Jo J, Ha N, Ji Y, Do A, Seo JH, Oh B, Choi S, Choe EK, Lee W, Son JW, Won S. Association of Multiple-trait Polygenic Risk Score with Obesity and Cardiometabolic Diseases in Korean Population. Genomics Proteomics Bioinformatics. 2025 Oct 28;23(5):qzaf102. (Corresponding author)
4. Lee J, Lee M, Lee SH, Kim MK, Kwon HS, Yun JS, Yang Y, Yoon KH, Cho JH, Ahn SW, Han K, Son JW. Metabolic Syndrome and Risk of Moyamoya Vasculopathy and Subsequent Stroke in Young Adults. J Am Heart Assoc. 2025 Oct 21;14(20):e042852. (Corresponding author)
5. Jo J, Ha N, Ji Y, Do A, Seo JH, Oh B, Choi S, Choe EK, Lee W, Son JW, Won S. Genetic determinants of obesity in Korean populations: exploring genome-wide associations and polygenic risk scores. Brief Bioinform. 2024 Jul 25;25(5):bbae389. (Corresponding author)

Incretin signaling and circadian regulation of glucose and energy metabolism

Jang Won Son

Division of Endocrinology and metabolism, Department of Internal Medicine,
Bucheon St. Mary's hospital, College of Medicine, The Catholic University of Korea

Incretin hormones, including glucagon-like peptide-1 (GLP-1) and glucose-dependent insulinotropic polypeptide (GIP), are traditionally viewed as nutrient-responsive regulators of postprandial glucose metabolism. However, emerging evidence indicates that incretin signaling is tightly integrated with the circadian system, linking gut, pancreatic, and central metabolic pathways within a coordinated chronometabolic network. Both intestinal L cells and pancreatic β-cells possess intrinsic molecular clocks, in which core components such as BMAL1 and CLOCK regulate time-dependent hormone secretion and insulin responsiveness. As a result, incretin effects on glucose homeostasis vary according to time of day, independently of meal composition.

Circadian disruption—driven by irregular meal timing, sleep deprivation, nocturnal light exposure, high-fat diet, and obesity—can impair the rhythmic secretion and action of incretins. These alterations are associated with blunted GLP-1 responses, impaired insulin secretion, and dysregulated appetite control through hypothalamic circuits. In obesity, disrupted circadian signaling further contributes to impaired satiety and energy imbalance, highlighting the role of incretins beyond glycemic regulation.

Clinically, incretin-based therapies such as GLP-1 receptor agonists and dual GIP/GLP-1 agonists have demonstrated profound metabolic benefits, extending into weight reduction and sleep-related disorders such as obstructive sleep apnea. Nevertheless, despite strong mechanistic links, evidence supporting time-specific (chronotherapeutic) optimization of these agents remains limited.

Understanding incretin biology within a circadian framework provides a novel perspective on metabolic disease pathophysiology and suggests that aligning behavioral factors—such as meal timing and sleep—with endogenous rhythms may enhance therapeutic efficacy. Future strategies may integrate pharmacologic and chronobiologic approaches to optimize metabolic health.

CURRICULUM VITAE

정창희

울산의대 서울아산병원 내분비내과

[학력]

1996-2002	고려대학교 의과대학 의학 학사
2010-2012	울산대학교 의과대학 의학 석사
2012-2014	울산대학교 의과대학 의학 박사

[경력]

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

[관심분야]

Diabetes complications, Therapeutics, Obesity, Adipose tissue dysfunction

[논문]

1. Cho YK, Jung CH. Sodium-Glucose Cotransporter 2 Inhibitors as Emerging Anticancer Agents. Diabetes Metab J. 2026;50:1-18.
2. Lee W*, Kim ES, Kim S, Park H, Lee JK, Baek E, Jung CH*, Cho SH. Noninvasive subterahertz glucose monitoring using a communication inspired eye diagram. Sci Rep. 2025;16:1079. (*Co-corresponding author)
3. Cho YK, Kim MJ, Kim EH, Lee MJ, Nam HJ, Lee WJ, Kim HK, Jung CH. Comparison of diagnostic criteria for fatty liver disease in assessing cardiac dysfunction: a cross-sectional study. Hepatol Int.2026;20:81-90.
4. Cho YK, Jung CH. Engineered nutrient-stimulated hormonal multi-agonist for precision targeting of obesity and metabolic disorders. Clin Mol Hepatol. 2025 Nov 26. Online ahead of print.
5. Jang MK, Cho YK, Moon JY, Min SH, Hwang JH, Jung CH. Impact of smart watch mobile application on the risk treatment of type 2 diabetes mellitus (iSMART-DM). Prim Care Diabetes. 2026;20:47-52.

GLP-1/GIP agonists for cardiometabolic protection - one drug fits all?

Chang Hee Jung

Department of Endocrinology & Metabolism, Asan Medical Center, University of Ulsan College of Medicine, Republic of Korea

Cardiometabolic diseases—including obesity, type 2 diabetes, metabolic dysfunction-associated steatotic liver disease (MASLD), and atherosclerotic cardiovascular disease—share interconnected pathophysiologic mechanisms driven by excess adiposity and metabolic dysregulation. Incretin-based therapies have recently emerged as key pharmacologic strategies targeting this cardiometabolic continuum. Glucagon-like peptide-1 receptor agonists (GLP-1 RAs) have demonstrated benefits beyond glycemic control, including substantial weight reduction and significant cardiovascular risk reduction in large randomized clinical trials.

More recently, dual incretin agonists targeting both GLP-1 and glucose-dependent insulinotropic polypeptide (GIP) receptors have been developed. These agents produce greater weight loss and improvements in glycemic control, blood pressure, and lipid profiles, suggesting potential benefits across multiple cardiometabolic conditions. Emerging data also indicate favorable effects on hepatic steatosis and cardiometabolic risk markers.

However, whether a single therapeutic approach can effectively address the heterogeneous spectrum of cardiometabolic disease remains uncertain. Differences in cardiovascular outcome evidence, organ-specific effects, and patient characteristics highlight the importance of individualized treatment strategies.

This lecture will review the current evidence for GLP-1-based and dual GLP-1/GIP agonist therapies in cardiometabolic protection, focusing on mechanisms of action and clinical trial findings. It will also discuss whether dual incretin therapy represents a universal treatment strategy or a key component of precision cardiometabolic medicine.

CURRICULUM VITAE

서 미 혜

순천향대학교 서울병원 내분비대사내과

[학력]

제주대학교 의과대학 의학과 학사
성균관대학교 대학원 의학과 석사
성균관대학교 대학원 의학과 박사

[경력]

성균관의대 강북삼성병원 인턴 및 내과전공의
성균관의대 강북삼성병원 내분비대사내과 임상강사
순천향대학교 구미병원 내분비대사내과 전임강사, 조교수, 부교수
(현) 순천향대학교 서울병원 내분비대사내과 부교수

[관심분야]

Diabetes, Obesity, Dyslipidemia, Cardiovascular disease, Aging

[논문]

1. Seo M, Suh K, Park HK, Cho KW. From bench to bedside: adipose tissue fibrosis in obesity, anti-diabetic therapies, and bariatric surgery. Korean J Intern Med. 2026 Mar;41(2):210-229.
2. Seo M, Cho KW et al. Persistent Adipose Inflammation Despite Metabolic Recovery Reveals Tissue-Specific Immunomodulation by Tirzepatide. Endocrinol Metab (Seoul). 2026 (Accepted January 2026).
3. Kim NH, Seo MH et al. 2023 Diabetic Kidney Disease Fact Sheet in Korea. Diabetes Metab J. 2024 May;48(3):463-472.
4. Seo M et al. Effect of bariatric surgery on circulating and urinary mitochondrial DNA copy numbers in obesity with or without diabetes. BMJ Open Diabetes Res Care. 2020 Oct;8(1):e001372.

Safety and sustainability concerns - beyond the GLP-1RA hype

Mihye Seo

Division of Endocrinology and Metabolism, Department of Internal Medicine,
Soonchunhyang University Seoul Hospital, Soonchunhyang University of College of Medicine

The era of GLP-1 receptor agonist (GLP-1RA)-based therapy has arrived. From clinicians and researchers to the general public, interest in GLP-1RA treatment has grown exponentially. This enthusiasm is driven by compelling evidence across a broadening spectrum of indications — from obesity and type 2 diabetes to emerging data in type 1 diabetes — alongside well-designed cardiovascular and renal outcome trials with liraglutide, semaglutide, tirzepatide, and retatrutide, supported by a growing body of real-world data. Beyond glycemic and weight control, accumulating research continues to uncover pleiotropic effects of GLP-1RAs on inflammation, cardiovascular remodeling, and organ protection.

Yet, amid this therapeutic optimism, critical questions regarding long-term safety and sustainability deserve closer attention. Concerns including lean mass and bone density loss, gastrointestinal tolerability, risk of weight regain following discontinuation, and appropriate patient selection remain incompletely addressed in routine clinical practice.

This lecture reviews the current evidence on GLP-1RA therapy, with a particular focus on safety and sustainability considerations that clinicians should keep in mind when caring for their patients.

SoLA 2026

한국지질·동맥경화학회 춘계학술대회

2026 Spring Congress on
Lipid and Atherosclerosis of KSoLA

SoLA 2026

한국지질·동맥경화학회 춘계학술대회

2026 Spring Congress on
Lipid and Atherosclerosis of KSoLA

Symposium 15

Updates in the 2026 Korean Dyslipidemia Guidelines: Integrating Global Evidence

4월 4일(토) 13:10-14:40 | Room 1 (그랜드볼룸 3)

좌장 : 김상현(서울의대 순환기내과), 김현진(한양의대 심장내과)

패널 : 김송이(제주의대 순환기내과), 김치경(고려의대 신경과)
박경택(중앙의대 순환기내과), 제세영(서울시립대 스포츠과학과)

13:10-13:28	**Updated risk stratification and treatment pathways in the 2026 Korean guidelines** **정재훈**(동국의대 심장내과)
13:28-13:46	**Optimizing pharmacologic therapy in the 2026 Korean dyslipidemia guidelines** **오진경**(충남의대 심장내과)
13:46-14:04	**Lifestyle interventions in dyslipidemia care: evidence updates and practical recommendations for Korea** **조가람**(한남대 식품영양학과)
14:04-14:22	**Managing dyslipidemia in special populations in the 2026 Korean guidelines** **장영우**(가천의대 심장내과)
14:22-14:40	**Panel Discussion**

CURRICULUM VITAE

정재훈

동국대학교 일산병원

[학력]

영남대학교 학사
동국대학교 석사

[경력]

서울대병원 전임의
국립중앙의료원 전문의
동국대학교 일산병원 조교수

[관심분야]

동맥경화

[논문]

1. Chung J, Rhee M-Y, Kim KH, Jang J-S, Kim H-Y. Reassessing home blood pressure thresholds: clinical implications of lowering the diagnostic criteria to 130/80 mmHg. Journal of Hypertension 2026:10.1097.
2. Chung J, Kim H-L, Joh HS, et al. Incremental prognostic value of combined information of arterial stiffness and the result of treadmill exercise test in patients with suspected coronary artery disease. Journal of Human Hypertension 2025;39:566-571.
3. Chung J, Lim W-H, Kim H-L, Joh HS, Seo J-B, Kim S-H, Zo J-H, Kim M-A. Influence of Socioeconomic Status on the Presence of Obstructive Coronary Artery Disease and Cardiovascular Outcomes in Patients Undergoing Invasive Coronary Angiography. Healthcare. 2024; 12(2):228.
4. Chung J, Kim H-L, Lim W-H, et al. New onset diabetes mellitus and cardiovascular outcomes according to statin intensity in patients after drug-eluting stent implantation in Asian patients. Scientific Reports 2023;13:16061.
5. Chung, J., Min, K. W., Son, B. K., Kim, D. H., & Kim, H. L. (2021). Association between histological severity of Helicobacter pylori infection and cardiovascular risk scores in the Korean population. Atherosclerosis, 333, 124-130.

Updated risk stratification and treatment pathways in the 2026 Korean guidelines

정재훈

동국의대 심장내과

CURRICULUM VITAE

오진경

Assistant Professor, Cardiovascular Center,
Chungnam National University Sejong Hospital,
Chungnam National University School of MedicineAsan Medical Center,
Sejong, Republic of Korea

Education

2005.3.2-2011.2.28	충남대학교 의과대학 의학사
2013.8.26-2015.8.25	충남대학교 의과대학 대학원 의학석사
2017.3.1-2019.8.23	충남대학교 의과대학 대학원 의학박사

Medical Training

2013.3.1-2016.2.28	충남대학교병원 전공의
2016.3.1-2018.2.28	충남대학교병원 전임의
2018.3.1-2020.2.28	서울아산병원 임상강사
2020.3.1-2024.2.28	세종충남대학교 병원 심장내과 기금조교수
2024.3.1-	세종충남대학교 병원 심장내과 기금부교수

Optimizing pharmacologic therapy in the 2026 Korean dyslipidemia guidelines

Jin Kyung Oh

Chungnam National University Sejong Hospital

This lecture will review the pharmacologic management of dyslipidemia in the context of the 2026 Korean Dyslipidemia Guidelines, with an emphasis on translating global evidence into practical treatment strategies for Korean patients. The presentation will discuss updated principles for selecting lipid-lowering therapy according to baseline cardiovascular risk, LDL-C levels, treatment response, and comorbid conditions.

Particular attention will be given to statins as first-line therapy and to the appropriate use of combination treatment, including ezetimibe, PCSK9-targeted therapies, and newly incorporated bempedoic acid, in patients who do not achieve recommended lipid targets or who have very high-risk features. The lecture will also address recent evidence supporting earlier treatment intensification, the importance of cumulative LDL-C exposure, and the need to improve long-term adherence and therapeutic persistence in routine practice. By integrating evidence from major international trials with local practice considerations, this session aims to provide a clinically applicable framework for optimizing pharmacologic therapy and improving cardiovascular prevention in Korea.

CURRICULUM VITAE

조가람

한남대학교 생명·나노과학대학 식품영양학과 조교수

[학력]

2021	고려대학교 보건과학과 이학박사
2016	고려대학교 식품영양학과 이학사

[경력]

2026-현재	한남대학교 생명·나노과학대학 식품영양학과 조교수
2025	Visiting Scientist, Department of Nutrition, Harvard T.H. Chan School of Public Health
2023-2026	고려대학교 생물신소재연구소 연구교수
2022-2023	고려대학교 BK21 정밀보건과학융합교육연구단 연구교수
2021-2022	질병관리청 국립보건연구원 심혈관질환연구과 박사후연구원
2018	Visiting Student, Friedman School of Nutrition Science & Policy, Tufts University
2017	Visiting Student, School of Social and Community Medicine, University of Bristol

[관심분야]

영양역학, 정밀영양, 영양유전체, 후성유전체, 대사체, 멀티오믹스, 심장대사질환

[논문]

1. Jo G*, Park D*, Kim S, Jun HJ, Magkos F, Merino J, Sun Q, Kim R, Subramanian SV, Franks W.P, Shin MJ. Obesity transitions across the life course: a population-based application of the Lancet Commission framework using age-period-cohort analysis in Korea and the United States. Diabetes Metab J. 2026 [Accepted].
2. Jun HJ, Kim S, Jo G. Age-Period-Cohort Analysis of dietary sodium, potassium, and sodium-to-potassium ratio in Korea. Epidemiol Health. 2025 Nov 4:e2025062.
3. Oh H, Jo G, Kim OY, Lim H, Song S, Choi JH, Bae JH, Jin ES, Kim R, Lee Y, Jeong IK, Shin MJ. Fact sheet: nationwide trends in dietary intakes among Korean adults, 2013-2022. Korean J Intern Med. 2025; 40(3):427-437.
4. Lee J*, Jo G*, Park D, Jun HJ, Bae JH, Shin MJ. The Association between Advanced Liver Fibrosis and Mortality Is Modified by Dietary Quality among Korean Adults: Results from the Korea National Health and Nutrition Examination Survey with Mortality Data. Nutrients. 2023;15(6):1501.
5. Jo G, Park D, Lee J, Kim R, Subramanian SV, Oh H, Shin MJ. Trends in Diet Quality and Cardiometabolic Risk Factors Among Korean Adults, 2007-2018. JAMA Netw Open. 2022; 5(6):e2218297.

Lifestyle interventions in dyslipidemia care: evidence updates and practical recommendations for Korea

Garam Jo

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

Lifestyle intervention is a fundamental pillar of dyslipidemia management and is recommended for all patients, regardless of pharmacological therapy. Tailored specifically to the Korean population, the Korean Dyslipidemia Guidelines update integrates global evidence into a comprehensive lifestyle intervention, encompassing diet, physical activity, smoking, and alcohol consumption.

Dietary recommendations prioritize limiting saturated and trans fatty acids, replacing them with unsaturated fatty acids, and increasing dietary fiber intake while reducing carbohydrates and added sugars. Evidence-based dietary patterns, including the Mediterranean, DASH, and Portfolio diets, are recommended for their lipid-lowering benefits and adapted to Korean dietary practices centered on whole grains, vegetables, legumes, and fish.

For physical activity, the guidelines recommend a combination of aerobic and resistance, alongside strategies to reduce sedentary behavior, including wearable device utilization. Smoking cessation is strongly recommended given its benefits in increasing HDL-cholesterol and reducing overall cardiovascular risk. For patients with hypertriglyceridemia, strict alcohol abstinence should be prioritized.

This session explores updated lifestyle recommendations on diet, physical activity, smoking, and alcohol, integrating global clinical evidence with the dietary and lifestyle context of the Korean population.

CURRICULUM VITAE

장영우(Youngwoo Jang)

가천대학교 길병원 조교수

학력 및 경력

2008-2012	School of Medicine, Gachon University, Doctor of Medicine
2017-2018	Cardiovascular Institute, Stanford Medicine, Postdoctoral Research Associate
2018-2019	Department of Cardiology, Gachon University, Gil Medical Center, Clinical Fellow
2019-2021	Department of Cardiology, Gachon University, Gil Medical Center, Clinical Assistant Professor
2022-	Department of Cardiology, Gachon University, Gil Medical Center, Assistant Professor

학회활동

2023-	심장대사증후군학회 학술간사
2025-	대한지질동맥경화학회 부총무/진료지침 간사

관심 연구 분야

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

주요 논문 및 저서

1. Jang Y, Lee JH, Lee SG, Jeong IK, Kim BJ. A Position Paper on Lipoprotein(a) from the Lipoprotein(a) Task Force Team of the Korean Society of Lipid and Atherosclerosis: Current Evidence, Clinical Applications, and Future Directions. Journal of Lipid Atherosclerosis and Korean Circ J. 2026 Jan;56(1):9-32.
2. Jang Y, Rhee EJ, Choi SH. Innovative Lipid-Lowering Strategies: RNA-Based, Small Molecule, and Protein-Based Therapies. Endocrinol Metab (Seoul) 2025;40:668-86.
3. Jang Y, Park SD, Lee JP, et al. One-month dual antiplatelet therapy followed by prasugrel monotherapy at a reduced dose: the 4D-ACS randomised trial. EuroIntervention 2025.
4. Kang SH, Pack KY, Kim JH, Jang Y (corresponding author). The effect of sarpogrelate compared to aspirin in high- or very-high-risk diabetes for primary prevention. Sci Rep. 2025 Jan 29;15(1):3616. doi: 10.1038/s41598-025-87868-x.
5. Kang SH, Lee J, Kim JH, Jang Y (corresponding author). Comparative Effectiveness of Clopidogrel Versus Aspirin for Primary Prevention in High-Risk Patients with Type 2 Diabetes: A Nationwide Propensity Score-Matched Cohort Study. Medicina (Kaunas) 2025;61.

Managing dyslipidemia in special populations in the 2026 Korean guidelines

Youngwoo Jang

Dept. Cardiology, Gachon University Gil Medical Center

The management of dyslipidemia in special populations remains a critical challenge in cardiovascular prevention, as these groups often exhibit heterogeneous risk profiles, distinct pathophysiology, and limited representation in randomized clinical trials. The 2026 Korean Dyslipidemia Guidelines aim to integrate global evidence with population-specific data to provide more practical and individualized recommendations for these patients.

Special populations addressed in the updated guidelines include patients with diabetes mellitus, chronic kidney disease, older adults, women of reproductive age, and individuals with inflammatory or metabolic comorbidities. These groups frequently demonstrate discordance between traditional lipid markers and actual cardiovascular risk, underscoring the need for refined risk stratification beyond low-density lipoprotein cholesterol alone. In addition, treatment decisions must carefully balance cardiovascular benefit against safety concerns such as drug-drug interactions, adverse effects, and long-term tolerability.

The updated Korean guidelines emphasize tailored lipid targets, earlier risk assessment, and selective use of non-LDL-based markers in appropriate clinical contexts. Practical recommendations are provided regarding statin intensity selection, combination lipid-lowering therapy, and treatment escalation in patients who fail to achieve risk-based lipid goals. Particular attention is given to real-world considerations relevant to Korean clinical practice, including aging demographics, high prevalence of diabetes, and differences in baseline lipid profiles compared with Western populations.

This lecture will summarize key updates in the 2026 Korean Dyslipidemia Guidelines related to special populations, highlight areas where global evidence has been adapted to local data, and discuss practical strategies for implementing guideline-directed lipid management in daily clinical practice.

SoLA 2026

한국지질·동맥경화학회 춘계학술대회

2026 Spring Congress on
Lipid and Atherosclerosis of KSoLA

SoLA 2026

한국지질·동맥경화학회 춘계학술대회

2026 Spring Congress on
Lipid and Atherosclerosis of KSoLA

Symposium 16

Primary Prevention of Cardiovascular Disease

4월 4일(토) 13:10-14:40 | Room 2 (그랜드볼룸 2)

좌장 : 김명아(서울의대 순환기내과), 이왕수(중앙의대 순환기내과)

패널 : 김효진(고려의대 신장내과), 유지웅(한림의대 순환기내과), 이선화(전북의대 심장내과)

13:10-13:30	Association between cumulative LDL-C exposure during young adulthood and middle age and risk of cardiovascular events	**양여리**(가톨릭의대 내분비내과)
13:30-13:50	MASLD as a cardiovascular risk enhancer in primary prevention	**김경수**(차의대 내분비내과)
13:50-14:10	Lipid-lowering therapy for primary prevention in the elderly	**이상학**(연세의대 심장내과)
14:10-14:40	Panel Discussion	

CURRICULUM VITAE

양여리

가톨릭의대 서울성모병원 내분비내과

[학력]

2004-2010	가톨릭의대 의학 학사
2015-2023	가톨릭의대 내과학 석,박사

[경력]

2010-2015	가톨릭중앙의료원 인턴, 내과 레지던트
2015-2018	가톨릭의대 서울성모병원 내분비내과 임상강사
2018-2023	가톨릭의대 서울성모병원 내분비내과 임상진료조교수
2023-	가톨릭의대 서울성모병원 내분비내과 임상조교수
2019-	가톨릭스마트헬스케어센터 사무국장, 부센터장

[관심분야]

Diabetes, Obesity, Health care, CGM and AID system

[논문]

1. Comparison between a tubeless, on-body automated insulin delivery system and a tubeless, on-body sensor-augmented pump in type 1 diabetes: a multicentre randomised controlled trial. Diabetologia. 2024 Jul;67(7):1235-1244.
2. Risk of developing chronic kidney disease in young-onset Type 2 diabetes in Korea. Rep 2023 Jun 21;13(1):10100.
3. Three-dimensional Multistructural Quantitative Photoacoustic and US Imaging of Human Feet in Vivo. Radiology. 2022 Feb 22;211029.
4. Effect of a Mobile Phone-Based Glucose-Monitoring and Feedback System for Type 2 Diabetes Management in Multiple Primary Care Clinic Settings: Cluster Randomized Controlled Trial. Mhealth Uhealth 2020;8(2):e16266.

Association between cumulative LDL-C exposure during young adulthood and middle age and risk of cardiovascular events

Yeoree Yang[1,2]

[1]Division of Endocrinology, Department of Internal Medicine, Seoul St. Mary's Hospital, The Catholic University,
[2]Catholic Smart Health Care Center, The Catholic University, Korea

Accumulating evidence has demonstrated that cumulative exposure to low-density lipoprotein cholesterol (LDL-C) from early adulthood through midlife plays a crucial role in determining lifetime cardiovascular risk. Large prospective cohorts, including CARDIA, Framingham Offspring, and UK Biobank analyses, consistently show that long-term LDL-C burden is strongly associated with subsequent atherosclerotic cardiovascular disease (ASCVD) events, independent of LDL-C levels measured at a single time point. Recent investigations further suggest that early and sustained LDL-C lowering can attenuate vascular aging and reduce event risk, even when midlife LDL-C values fall within current guideline targets. Collectively, these findings emphasize that cardiovascular prevention should begin much earlier than traditionally practiced, with a focus on lifetime lipid exposure rather than short-term control.

This presentation will summarize current evidence linking cumulative LDL-C exposure to cardiovascular outcomes, review underlying pathophysiological mechanisms, and explore implications for earlier, more aggressive prevention strategies across the life course.

CURRICULUM VITAE

김경수

차의과학대학교 분당차병원 내분비내과

[학력]

1998-2004	차의과학대학교 의학과 학사
2006-2008	차의과학대학교 의학과 석사
2013-2020	차의과학대학교 의학과 박사

[경력]

2014-2020	차의과학대학교 분당차병원 내분비내과 조교수
2020-2025	차의과학대학교 분당차병원 내분비내과 부교수
2025-	차의과학대학교 분당차병원 내분비내과 교수

[관심분야]

당뇨병, 인슐린저항성, 지방간, 임신당뇨병, 지질대사

[논문]

1. Kim KS, Kim B, Han K. Big Data Research for Diabetes-Related Diseases Using the Korean National Health Information Database. Diabetes Metab J 2025;49:13-21.
2. Kim KS, Hong S, Han K, Park CY. Association of non-alcoholic fatty liver disease with cardiovascular disease and all cause death in patients with type 2 diabetes mellitus: nationwide population based study. BMJ 2024;384:e076388.
3. Kim KS, Hong S, Han K, Park CY. Clinical Characteristics of Patients With Statin Discontinuation in Korea: A Nationwide Population-Based Study. J Lipid Atheroscler 2024;13:41-52.
4. Kim KS, Hong S, Ahn HY, Park CY. Metabolic dysfunction-associated fatty liver disease and mortality: a population-based cohort study. Diabetes Metab J 2023;47:220-231.
5. Kim KS, Hong S, Han K, Park CY. Fenofibrate add-on to statin treatment is associated with low all-cause death and cardiovascular disease in the general population with high triglyceride levels. Metabolism. 2022;137:155327.

MASLD as a cardiovascular risk enhancer in primary prevention

김경수

차의과학대학교 분당차병원 내분비내과

대사이상지방간질환(Metabolic dysfunction-associated steatotic liver disease, MASLD)에 대한 관심이 전 세계적으로 높아지고 있다. 대사이상지방간질환은 간과 직접적으로 관련된 간경화, 간암 등의 위험을 높일 뿐만 아니라 2형당뇨병을 비롯한 다양한 대사질환 및 심혈관질환의 위험을 높이기 때문에 적절한 진단과 치료가 중요하다. 본 강의에서는 심혈관질환의 일차예방에 있어 대사이상지방간질환에 대해 살펴보고자 한다.

CURRICULUM VITAE

이상학

연세의대 심장내과

[학력]

1994	연세의대 학사
1999	연세대학교 대학원 석사
2005	연세대학교 대학원 박사

[경력]

2003-2006	한림의대 강남성심병원 전임강사, 조교수
2007-현재	연세의대 세브란스병원 조교수, 부교수, 교수
2010-2011	미국 UC 샌디에고 방문연구원

[관심분야]

지단백 대사, 동맥경화, 예방심장학

[논문]

1. Jin IT, et al. Aspirin and clinical outcomes in individuals with incidentally diagnosed coronary artery stenosis. Am J Med 2025;138:994-1000.
2. Kim J, et al. Statin therapy in individuals with intermediate cardiovascular risk. Metabolism 2024;150:155723.
3. An DB, et al. Hepatic Cdkal1 deletion regulates HDL catabolism and promotes reverse cholesterol transport. Atherosclerosis 2023;375:21-29.
4. Lee CJ, et al. Cardiovascular risk and treatment outcomes in severe hypercholesterolemia: a nationwide cohort study. J Am Heart Assoc 2022;11:3024379.
5. Ann SJ, et al. Role of lncRNA HSPA7 in human atherosclerotic plaque in sponging miR-223 and promoting proinflammatory vascular smooth muscle cell transition. Exp Mol Med 2021;53:1842-1849.

Lipid-lowering therapy for primary prevention in the elderly

Sang-Hak Lee

Cardiology, Yonsei University College of Medicine

As the elderly people have increased in the recent decades, particularly in developed countries, proper cardiovascular prevention became an issue of great interest. Although there is no significant controversy on lipid-lowering therapy (LLT) for secondary prevention even in the elderly, LLT for primary prevention has less strongly recommended in this population by most guidelines.

This policy is based on research data including meta-analysis that could have include fewer clinical trials enrolling elderly people without previous ASCVD. In addition, the characteristics of elderly people such as multi-comorbidities and more frequent drug adverse events, and shorter life expectancy can limit prescription of LLT.

Therefore, it is desirable to consider LLT benefit and shared decision making/personalization (but not expected excessive concern on LLT safety) in this population.

SoLA 2026

한국지질·동맥경화학회 춘계학술대회

2026 Spring Congress on
Lipid and Atherosclerosis of KSoLA

SoLA 2026

한국지질·동맥경화학회 춘계학술대회

2026 Spring Congress on
Lipid and Atherosclerosis of KSoLA

Symposium 17 –
KOVAS-KSoLA Joint Symposium

Unraveling the Pathway of Atherosclerosis through Imaging and Lipid Assessment

4월 4일(토) 13:10-14:40 | Room 3 (그랜드볼룸 1)

좌장 : 정익모(이화의대 순환기내과), 성기철(성균관의대 순환기내과)

패널 : 김학령(서울의대 순환기내과), 이민경(한양의대 내분비내과)
임수빈(이화의대 순환기내과)

13:10-13:30	**Coronary artery calcium and imaging-based risk assessment in clinical practice** **손정우**(연세원주의대 심장내과)
13:30-13:50	**Assessment of arterial remodeling and atherosclerosis progression using carotid ultrasound** **이종영**(한림의대 순환기내과)
13:50-14:10	**Pulse wave velocity and ankle-brachial index as indicators of arterial health** **권오성**(가톨릭의대 순환기내과)
14:10-14:40	**Panel Discussion**

CURRICULUM VITAE

손정우

원주세브란스기독병원 심장내과

[학력]

2004 연세대학교 원주의과대학 학사
2026 연세대학교 의학과 석박사통합

[경력]

2016- 원주세브란스기독병원 심장내과 조교수
2014-2015 한림대학교 춘천성심병원 심장내과 임상조교수
2012-2013 세브란스병원 심장내과 전임의
2004-2008 세브란스병원 내과 전공의

[관심분야]

심초음파, 심부전, 이상지질혈증, 고혈압

[논문]

1. Dobutamine Stress Echocardiography for Left Ventricular Reverse Remodeling in Idiopathic Dilated Cardiomyopathy. Int J Heart Fail. 2025 Jul 7;7(3):152-159.
2. Clinical and Echocardiographic Predictors for the Presence of Late Gadolinium Enhancement on Cardiac Magnetic Resonance Imaging in Patients with Carbon Monoxide Poisoning. Diagnostics (Basel). 2023 Dec 27;14(1):60.
3. Epidemiologic Profile of Patients With Valvular Heart Disease in Korea: A Nationwide Hospital-Based Registry Study. J Cardiovasc Imaging. 2023 Jan;31(1):51-61.
4. Temporal trends in heart failure over 11 years in the aging Korean population: A retrospective study using the national health insurance database. PLoS One. 2022 Dec 28;17(12):e0279541.

Coronary artery calcium and imaging-based risk assessment in clinical practice

손정우

연세원주의대 심장내과

CURRICULUM VITAE

이종영

한림대학교 성심병원 순환기내과

[학력]

1999.2	영남대학교 의과대학 졸업
2011.2	울산대학교 의과대학 석사
2013.2	울산대학교 의과대학 박사

Assessment of arterial remodeling and atherosclerosis progression using carotid ultrasound

Jong-Young Lee

Division of Cardiology, Department of Internal Medicine, Hallym University Sacred Hospital, Republic of Korea

Carotid ultrasound is a powerful, non-invasive tool for monitoring the structural and functional changes in the arterial wall. Here is an assessment of how it evaluates **arterial remodeling and atherosclerosis progression**:

1. Assessment of Arterial Remodeling

Arterial remodeling refers to the compensatory changes in vessel size in response to plaque build-up.

- **Carotid Intima-Media Thickness (CIMT):** A primary marker for subclinical atherosclerosis. An increase in CIMT (typically >0.9 mm) indicates early-stage structural remodeling and heightened cardiovascular risk [1, 2].
- **Remodeling Patterns:**
  - **Positive Remodeling (Outward):** The vessel wall expands outward to maintain the lumen diameter despite plaque growth. While it preserves blood flow, these lesions are often "soft" and prone to rupture [1, 5].
  - **Negative Remodeling (Inward):** The vessel wall shrinks or fails to expand, leading to rapid **luminal narrowing (stenosis)** [1, 3].

2. Monitoring Atherosclerosis Progression

Beyond simple thickness, ultrasound evaluates the "burden" and "behavior" of the disease.

- **Plaque Quantification:** Progression is measured by the increase in **Plaque Area** or **Plaque Volume** (via 3D ultrasound). This is a stronger predictor of future stroke or myocardial infarction than CIMT alone [1, 4].
- **Plaque Vulnerability (Stability):**
  - **Echolucent (Dark) Plaques:** Suggest a lipid-rich necrotic core or intraplaque hemorrhage, indicating high risk (vulnerable plaque) [2, 5].
  - **Echogenic (Bright/Calcified) Plaques:** Indicate fibrous tissue or calcium, suggesting a more stable, chronic lesion [2].
- Surface Irregularity/Ulceration: Identification of craters or "pits" on the plaque surface indicates a high risk of embolization and acute ischemic events [2, 4].

3. Advanced Diagnostic Parameters

- **Contrast-Enhanced Ultrasound (CEUS):** Detects **Intraplaque Neovascularization (IPN)**. The presence of tiny micro-vessels inside the plaque is a hallmark of active inflammation and high rupture risk [3, 5].
- **Strain & Elastography:** Measures the stiffness of the arterial wall. Decreased elasticity (increased stiffness) often precedes visible plaque formation [3].

CURRICULUM VITAE

권오성

가톨릭대학교 은평성모병원

[학력]

2017.3-2020.2 울산대학교 의학과 박사
2009.3-2011.2 울산대학교 의학과 석사
2003.3-2006.2 한림대학교 의과대학 학사

[경력]

2026.3-현재 부교수
2022.3-2026.2 조교수
2021.3-2022.3 임상조교수

[관심분야]

경피적 대동맥 판막 치환술, 관상동맥 중재술, 항혈전치료, 이상지질혈증, 인공지능 예측모델 개발

[논문]

1. Osung Kwon[1,2], Sang-Yeub Lee[3], Bokyoung Kim[4], Kyungdo Han[5], Jihyun Ahn[6]. On behalf of the Committee of Public Relations of the Korean Society of Lipid and Atherosclerosis Dyslipidemia Fact Sheet in South Korea, 2024, J Lipid Atheroscler. 2025 Sep;14(3):298-311.
2. Osung Kwon, MD, PhD, Jong-Hwa Ahn, MD, PhD[3], Jin-Sin Koh, MD, PhD[4], Yongwhi Park, MD, PhD[3], Seok Jae Hwang, MD, PhD[4], Udaya S Tantry, PhD[5], Paul A Gurbel, MD[5], Jin-Yong Hwang, MD, PhD[4], Young-Hoon Jeong, MD, PhD[6,7], Assessment of Platelet-Fibrin Clot Strength and Platelet Reactivity for Predicting Cardiovascular Events After Coronary Intervention, Eur Heart J. 2024 Jul 9;45(25):2217-2231.
3. Osung Kwon, MD, PhD*; Jun-Pyo Myong, MD, PhD*; Yunhee Lee, MS; Yeon-Jik Choi, MD; Jeong Eun Yi, MD, PhD; Suk Min Seo, MD, PhD; Sung-Won Jang, MD, PhD; Pum Joon Kim, MD, PhD; Jung-Min Lee, MD, PhD, Sodium-Glucose Cotransporter-2 Inhibitors After Acute Myocardial Infarction in Patients With Type 2 Diabetes: A Population-Based Investigation, J Am Heart Assoc. 2023;12:e027824.
4. Osung Kwon MD, Wonjun Na2, MS; Heejun Kang, MS; Tae Joon Jun, PhD; Jihoon Kweon, PhD; Gyung-Min Park, MD, PhD; YongHyun Cho, MS; Cinyoung Hur, MS; Jungwoo Chae, BS; Do-Yoon Kang, MD, PhD; Pil Hyung Lee, MD, PhD; Duk-Woo Park, MD, PhD; Jung-Min Ahn, MD, PhD; Soo-Jin Kang, MD, PhD; Cheol Whan Lee, MD, PhD; Seong-Wook Park, MD, PhD; Seung-Whan Lee, MD, PhD; Seung-Jung Park, MD, PhD; Dong Hyun Yang, MD, PhD; Young-Hak Kim, MD, PhD, Electronic Medical Record-Based Machine Learning Approach to Predict the Risk of 30-Day Adverse Cardiac Events After Invasive Coronary Treatment: Machine Learning Model Development and Validation. JMIR MEDICAL INFORMATICS 2022.
5. Osung Kwon MD, Duk-Woo Park, MD Antithrombotic Therapy After Acute Coronary Syndromes or Percutaneous Coronary Interventions in East Asian Populations. JACC Asia 2022.

Pulse wave velocity and ankle-brachial index as indicators of arterial health

Osung Kwon

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

Early identification of individuals at increased cardiovascular risk remains a major challenge in primary prevention, particularly among asymptomatic patients and those classified as having borderline or intermediate risk by traditional risk prediction models. Although coronary artery calcium scoring and carotid imaging are well-established tools for detecting subclinical atherosclerosis, they primarily reflect structural plaque burden and may not fully capture the cumulative functional impairment of the arterial system.

Arterial stiffness, most commonly assessed by pulse wave velocity (PWV), and the ankle-brachial index (ABI) represent non-invasive markers of global arterial health and systemic vascular aging. PWV reflects structural and functional alterations of elastic arteries that precede overt atherosclerotic plaque formation, while ABI identifies both overt and subclinical peripheral arterial disease and integrates diffuse atherosclerotic burden across vascular territories. Accumulating evidence demonstrates that increased arterial stiffness and abnormal ABI are independently associated with cardiovascular morbidity and mortality, even after adjustment for conventional risk factors. Notably, arterial stiffness has been shown to improve risk reclassification in up to 15% of individuals at intermediate risk, whereas a low or borderline ABI identifies a subgroup with substantially elevated cardiovascular risk despite the absence of symptoms.

Despite their prognostic value, the routine use of PWV and ABI in clinical practice remains limited. Methodological heterogeneity, lack of standardized thresholds, and uncertainty regarding treatment strategies guided by these measurements have contributed to their cautious adoption in contemporary guidelines. Nevertheless, these markers offer important complementary information beyond plaque-based imaging, particularly in patients with metabolic risk factors, hypertension, diabetes, or advanced vascular aging where coronary calcification may be absent or underestimated. Their selective use may refine risk stratification in clinically ambiguous populations and support a more personalized approach to cardiovascular prevention.

SoLA 2026

한국지질·동맥경화학회 춘계학술대회

2026 Spring Congress on
Lipid and Atherosclerosis of KSoLA

SoLA 2026

한국지질·동맥경화학회 춘계학술대회

2026 Spring Congress on
Lipid and Atherosclerosis of KSoLA

Symposium 18

Dyslipidemia Fact Sheet 2026

4월 4일(토) 13:40-14:40 | Room 4 (볼룸 1,2)

좌장 : 유승기(이화의대 건진의학과), 최성훈(한림의대 순환기내과)

패널 : 김병식(한양의대 심장내과), 나진오(고려의대 순환기내과)
배재현(한림의대 내분비내과)

13:40-13:55	**Epidemiology of dyslipidemia in Korean adults, 2026** **문민경**(서울의대 내분비대사내과)
13:55-14:10	**Management of hypercholesterolemia and dyslipidemia: from overall burden to high-risk populations** **양예슬**(서울의대 내분비대사내과)
14:10-14:25	**Health behaviors among people with dyslipidemia** **김규호**(가톨릭의대 내분비내과)
14:25-14:40	**Panel Discussion**

CURRICULUM VITAE

문민경

서울대학교 보라매병원

[학력]

1990-1996	서울대학교 의과대학 의학사
2002-2003	서울대학교 의과대학 의학석사
2003-2006	서울대학교 의과대학 의학박사

[경력]

1997-2001	서울대학교병원 내과 전공의
2003-현재	서울특별시 보라매병원 내과 교수
2006-현재	서울대학교 의과대학 내과학교실 교수

[관심분야]

2형당뇨병 합병증, 약물반응성, 이상지질혈증, 내분비교란물질

[논문]

1. Moon MK, Lee I, Lee G, Park S, Choi S, Lee A, Kim MJ, Park YJ, Park J, Choi K. Ethylparaben and consumer chemicals and their associations with metabolic disorders: A multicycle analysis of Korean National Environmental Health Survey (2015-2020). Sci Total Environ. 2026 Jan 10;1011:181128.
2. Park YS, Lee KN, Koo BK, Kwak SH, Han KD, Moon MK. Diabetic retinopathy and chronic kidney disease synergistically increase the risk of incident cardiovascular disease in type 2 diabetes: Insights from two cohort studies. Diabetes Res Clin Pract. 2025 Aug;226:112373.
3. Choi JH, Koo BK, Yang YS, Min SH, Park JS, Rhee SY, Kim HJ, Moon MK. Initial Pharmacological Strategies in People with Early Type 2 Diabetes Mellitus: A Systematic Review and Network Meta-Analysis. Diabetes Metab J. 2025 Nov;49(6):1252-1261.
4. Moon MK, Lee G, Choi S, Lee I, Lee A, Kim MJ, Park S, Cho YH, Park YJ, Oh S, Park J, Cho NH, Choi K. Association of urinary levels of trace metals with type 2 diabetes and obesity in postmenopausal women in Korea: A community-based cohort study. Int J Hyg Environ Health. 2025 Mar;264:114508.
5. Park SS, Koo BK, Park S, Han K, Moon MK. Impact of New-Onset Diabetes after Transplantation on Cardiovascular Risk and Mortality in Korea: A Nationwide Population-Based Study. Diabetes Metab J. 2025 Jan;49(1):117-127.

Epidemiology of dyslipidemia in Korean adults, 2026

Min Kyong Moon

Endocrinology and Metabolism, Seoul National University Boramae Medical Center, Korea

Since 2015, the Korean Society of Lipid and Atherosclerosis has regularly published national dyslipidemia fact sheets, providing concise, population-based snapshots of lipid profiles, prevalence, and management patterns in Korean adults. These serial reports have established a continuous epidemiologic framework that allows temporal comparisons and evaluation of policy and clinical initiatives over time. Building on this foundation, the 2026 edition extends the observation period to 2007-2024 using Korea National Health and Nutrition Examination Survey data, enabling an 18-year perspective on changes in blood lipids and dyslipidemia burden.

Age-standardized mean concentrations of total and LDL cholesterol, as well as HDL cholesterol, increased steadily over 18 years, whereas triglyceride levels peaked around 2016 and then declined, leading to an overall reduction in non-HDL cholesterol. Despite these favorable lipid profile trends, the age-standardized prevalence of hyper-LDL-cholesterolemia rose from 8.8% in 2007 to 26.7% in 2024, and the overall prevalence of dyslipidemia remained high at roughly 40%-45%. Crude prevalence estimates indicated that dyslipidemia and its components were more common in men at younger ages, but after 50 years of age, prevalence in women increased sharply and surpassed that in men, reaching over 60%-70% among those aged 70 years or older. Hypertriglyceridemia and hypo-HDL-cholesterolemia were particularly frequent in men, whereas older women bore a higher burden of hyper-LDL-cholesterolemia. These findings suggest partial success of population-level lipid management but reveal persistent sex- and age-specific gaps, underscoring the need for targeted prevention strategies, especially in peri- and postmenopausal women.

CURRICULUM VITAE

양예슬

서울의대(서울대학교 직장부속의원)

[학력]

2006-2012	한림대학교 의과대학, 학사
2015-2017	서울대학교 융합과학기술대학원, 석사
2018-2024	서울대학교 의과대학, 박사

[경력]

2019-2020	서울의대 서울대학교병원, 내분비내과 진료교수
2020-2022	가톨릭의대 의정부성모병원, 내분비내과 임상진료조교수
2022-	서울의대 의학과 조교수

[관심분야]

당뇨병, 지질대사, 비만

[논문]

1. Physical activity for prevention of cardiovascular disease: consensus statement of Korean Society of Cardio-cerebrovascular Disease Prevention
2. Abdominal obesity and the risk of young-onset dementia in women: a nationwide cohort study
3. Real-World Treatment Patterns according to Clinical Practice Guidelines in Patients with Type 2 Diabetes Mellitus and Established Cardiovascular Disease in Korea: Multicenter, Retrospective, Observational Study
4. Efficacy and safety of monotherapy with enavogliflozin in Korean patients with type 2 diabetes mellitus: Results of a 12-week, multicentre, randomized, double-blind, placebo-controlled, phase 2 trial
5. Lipid Management in Korean People with Type 2 Diabetes Mellitus: Korean Diabetes Association and Korean Society of Lipid and Atherosclerosis Consensus Statement

Management of hypercholesterolemia and dyslipidemia: from overall burden to high-risk populations

Ye Seul Yang

Department of Medicine, Seoul National University, Korea

Dyslipidemia is a major cardiovascular risk factor, especially prevalent in individuals with metabolic disorders. Based on Korean adult data, its prevalence increases markedly with metabolic risk status. Dyslipidemia was found in 28.9% of normoglycemic adults, 55.1% of those with prediabetes, and 70.8% with diabetes. Similarly, prevalence rose from 28.6% in normotensive adults to 44.9% in prehypertension and 63.3% in hypertension.

By obesity status, prevalence was 14.1% in underweight, 29.4% in normal weight, 44.3% in overweight, and 57.0% in obese adults. Central obesity was also strongly linked, with dyslipidemia present in 61.2% compared to 32.3% without abdominal obesity.

LDL-cholesterol levels tended to be lower in individuals with diabetes or hypertension, suggesting treatment effects or altered lipid metabolism.

These findings highlight the high burden of dyslipidemia among metabolic disease populations and the need for integrated management strategies. Early identification and personalized lipid control, combined with overall metabolic risk reduction, are key to preventing future cardiovascular disease in these high-risk groups.

CURRICULUM VITAE

김규호

가톨릭대학교 성빈센트병원

[학력]

2004-2010	영남대학교 의과대학 학사
2010-2015	KAIST 의과학대학원 박사 (석박통합과정)

[경력]

2017-2020	분당서울대학교병원 내과 전공의
2022-2022	분당서울대학교병원 내분비대사내과 임상강사
2024-현재	가톨릭대학교 성빈센트병원 내분비내과 조교수

[관심분야]

당뇨병, 이상지질혈증, 비만, 대사증후군

[논문]

1. Kim K, Lee YJ, Yun JS, Ahn YB, Ko SH. Effects of the FXR agonist GW4064 on metabolic disorders in db/db mice. Lab Anim Res 2026;42:5.
2. Kim K, Ko SH, Yun JS, Lee KW, Kim ES, Jeong IK, Kim JH, Kim SY, Won KC, Kim M, Cha BS, Kim S, Choi SH, Rhee EJ, Kim SG, Kim BH, Park KS, Ju YC, Heo TW, Ahn YB. Efficacy and safety of pioglitazone versus dapagliflozin as an add-on to metformin and alogliptin combination therapy: the EPIDOTE study. Sci Rep 2025;16:1226.
3. Kim K, Kim B, Lee K, Ahn YB, Ko SH, Choi SH, Han K, Yun JS. Older Adults with Diabetes in Korea: Latest Clinical and Epidemiologic Trends. Diabetes Metab J 2025;49:183-93.
4. Kim K, Yun JS, Lee J, Yang Y, Lee M, Ahn YB, Cho JH, Ko SH. Effectiveness of a Social Networking Site Based Automatic Mobile Message Providing System on Glycemic Control in Patients with Type 2 Diabetes Mellitus. Endocrinol Metab (Seoul) 2024;39:344-52.
5. Kim K, Moon JH, Ahn CH, Lim S. Effect of olmesartan and amlodipine on serum angiotensin-(1-7) levels and kidney and vascular function in patients with type 2 diabetes and hypertension. Diabetol Metab Syndr 2023;15:43.

Health behaviors among people with dyslipidemia

김규호

가톨릭의대 내분비내과

최근 국민건강영양조사 자료를 활용하여 이상지질혈증 환자의 식생활행태, 흡연, 음주, 신체활동 등 건강행태를 분석한 결과를 공유하고자 한다. 또한, 과거 분석 결과 및 일반인들의 건강행태 분석 결과와 비교하고자 한다. 이를 통하여 이상지질혈증 환자에서 건강행태 개선할 방법을 모색하고자 한다.

SoLA 2026

한국지질·동맥경화학회 춘계학술대회

2026 Spring Congress on
Lipid and Atherosclerosis of KSoLA

SoLA 2026

한국지질·동맥경화학회 춘계학술대회

2026 Spring Congress on
Lipid and Atherosclerosis of KSoLA

Symposium 19

Innovative Technologies and Disease Modeling for Lipotoxicity and Atherosclerosis

4월 4일(토) 15:30–17:00 | Room 1 (그랜드볼룸 3)

좌장 : 오구택(이화여대 생명과학과), 박영미(이화의대 분자의과학교실)

패널 : 권유욱(서울의대 의생명연구원), 김경진(인하의대 의생명학교실)
송주현(전남의대 해부학교실)

15:30–15:50	From basic discovery to clinical translation: macrophage-targeted imaging and therapy for high-risk plaque **김진원**(고려의대 순환기내과)
15:50–16:10	Intracellular Ca^{2+} dysregulation links lipotoxicity to defective autophagy **오병철**(가천의대 생리학교실)
16:10–16:30	In vitro modeling of atherosclerosis using iPSC-derived blood vessel organoids **김다현**(성신여대 바이오생명공학과)
16:30–17:00	Panel Discussion

CURRICULUM VITAE

김진원(Jin Won Kim)

Cardiology Division, Department of Internal Medicine,
College of Medical School, Cardiovascular Center, Guro Hospital

Education and Training

2005.02	KoreaUniversity, Korea, Ph.D, Medical Science
1999.08	Korea University, Korea, Ms, Medical Science
1995.02	Korea University, Korea, M.D. & Bachelor, Medical Science

Employment and Position

2015-Present	Cardiology Division, Department of Internal Medicine, Korea University Medical Center, Guro Hospital, Seoul, Korea, Professor
2009-2015	Cardiology Division, Department of Internal Medicine, Korea University Medical Center, Guro Hospital, Seoul, Korea, Associate Professor
2009-2011	Cardiovascular Research Center, Harvard Medical School, MGH, Boston, MA, USA, Postdoctoral Research Fellow
2006-2009	Cardiology Division, Department of Internal Medicine, Korea University Medical Center, Guro Hospital, Seoul, Assistant Professor
2004-2006	Cardiology Division, Department of Internal Medicine, Korea University Medical Center, Guro Hospital, Seoul, Clinical Assistant Professor
2003-2004	Cardiology Division, Department of Internal Medicine, Korea University Medical Center, Anam Hospital, Seoul, Fellowship/Clinical Instructor
2000-2003	Director, Cardiology, Capital Military Hospital, Military Service
1996-2000	Korea University Medical Center, Internal Medicine, Residency Training
1995-1996	Korea University Medical Center, Internship Training

Awards and Honors

Jan. 2026	National Academy of Medicine of Korea
Nov. 2025	Commendation by Korea Minister of Health and Welfare
Jun. 2025	KU Hall of Fame
May 2022	SeokTap Research Award
Jan. 2022	Moorok Namgyeongae Medical Grand Prize
May 2021	SeokTap Research Award
May 2018	SeokTap Research Award
Dec. 2017	Commendation by Korea Minister of Health and Welfare
May 2016	SeokTap Research Award
Nov. 2014	Astrazeneca Research Award
Sep, 2012	Yuhan Medical Prize

From basic discovery to clinical translation: macrophage-targeted imaging and therapy for high-risk plaque

Seung Ho Shin, Jin Won Kim

Multimodal Imaging and Theranostic Lab., Cardiovascular Center, Korea University Guro Hospital, Seoul, Korea

High-risk coronary atheroma, characterized by expansive lipid-rich necrotic cores and intense macrophage-mediated inflammation, represents a critical substrate for acute coronary syndromes. Despite advancements in diagnostic modalities, a clinical gap persists in identifying these vulnerable lesions in vivo and inducing durable plaque stabilization. To address the unmet clinical need for comprehensive in vivo plaque characterization and targeted stabilization strategies, we developed an integrated multimodal intravascular imaging and theranostic framework that bridges basic molecular discovery with clinical implementation. This technology enabled high-resolution structural imaging coupled with molecular visualization of macrophage infiltration, specifically identifying inflamed high-risk plaques. Building on this diagnostic foundation, we developed a macrophage mannose receptor-targeted photoactivatable agent that selectively accumulates in inflammatory macrophages to enable simultaneous dual-modal structural-molecular OCT-NIRF (near-infrared fluorescence) imaging and precise photodynamic therapy. In parallel, a Dectin-1-directed laminarin-chlorin e6 (LAM-Ce6) strategy enabled OCT-NIRF-guided intravascular photoactivation, simultaneously localizing macrophage-abundant plaques and enhancing efferocytosis. This dual-track innovation allowed for OCT-NIRF-guided intravascular photoactivation, which simultaneously localized lipid-laden, macrophage-abundant plaques and stabilized them. This approach reduced inflammatory signaling, enhanced efferocytosis, and facilitated plaque remodeling toward a stable, collagen-rich fibrotic phenotype. Furthermore, we recently integrated fluorescence lifetime imaging (FLIm) with OCT to overcome major hurdles for clinical translation. The first-in-human OCT-FLIm studies demonstrated clinical feasibility and safety, providing label-free, multiparametric compositional mapping of coronary plaques in real-time. Notably, this technology revealed heightened inflammatory signatures in patients with acute coronary syndrome compared to those with chronic stable angina, validating its diagnostic accuracy in vivo.

Taken together, these innovations represent a paradigm shift from molecular theranostic discovery to real-world clinical implementation. By bridging structural, molecular, and biological insights, our catheter-based strategy offers a new avenue for the management of high-risk plaque, paving the way for precision medicine in coronary artery disease.

CURRICULUM VITAE

오병철

가천의대 생리학교실

[학력]

2001.08 서울대학교 식품공학과, PhD

[경력]

2002-2006 Joslin Diabetes Center, Harvard Medical School, Post-doc
2006-2008 Chungbuk National University, Research Professor
2007-Present Gachon University, Professor, Vice President

[관심분야]

Metabolism, Fibrosis, Insulin resistance, Autophagy

[논문]

1. Kim OH, Noh SW, Choi JS, Jung Y, Oh BC. The SERCA-PLN-DWORF axis in cardiometabolic disease: mechanisms and therapeutic perspectives. Cardiovasc Diabetol 2025.
2. S.-R. Park, et.al., B.-C. Oh, I.-S. Hong, (2024) Exploring Memory Function Beyond Immune Cells: ANGPTL4-Mediated Memory Functions in Tissue Resident Stem Cells. Adv. Sci., 2307545.
3. Oh, B.-C. (2023). Phosphoinositides and intracellular calcium signaling: novel insights into phosphoinositides and calcium coupling as negative regulators of cellular signaling. Experimental & Molecular Medicine. 10.1038/s12276-023-01067-0.
4. Lee, J.W., et.al., Kim, O.-H., and Oh, B.-C. (2023). Candesartan, an angiotensin-II receptor blocker, ameliorates insulin resistance and hepatosteatosis by reducing intracellular calcium overload and lipid accumulation. Experimental & Molecular Medicine 55, 910-925. 10.1038/s12276-023-00982-6.
5. Kim OH, et al. and Oh, B.-C. (2022) Externalized phosphatidylinositides on apoptotic cells are eat-me signals recognized by CD14. Cell Death Differ 29(7):1423-1432.

Intracellular Ca^{2+} dysregulation links lipotoxicity to defective autophagy

Byung-Chul Oh

Department of Physiology, Lee Gil Ya Cancer and Diabetes Institute, Gachon University College of Medicine, Incheon, Korea

Lipotoxic stress resulting from excessive lipid accumulation is a central driver of metabolic dysfunction, yet the signaling mechanisms linking lipid overload to impaired autophagy remain incompletely defined. Here, we investigated the role of intracellular Ca^{2+} dysregulation as a mechanistic bridge between lipotoxicity and defective autophagic flux. In hepatocyte cell lines, lipid accumulation induced sustained cytosolic Ca^{2+} overload, which was accompanied by pronounced accumulation of the autophagy adaptor p62/SQSTM1 and multiphosphorylation at stress-responsive sites. These molecular alterations were associated with marked suppression of autophagic flux, indicating a failure of cellular quality control under lipotoxic conditions. Importantly, similar patterns of lipid accumulation, Ca^{2+} overload, p62 multiphosphorylation, and autophagy impairment were consistently observed in obese and insulin-resistant animal models, supporting the physiological relevance of this pathway in vivo. Our findings suggest that Ca^{2+} overload is not merely a downstream consequence of lipid excess but functions as an upstream signaling node that amplifies stress responses and disrupts autophagy. Pharmacological intervention targeting Ca^{2+}-dependent stress signaling restored autophagic activity independently of lipid reduction. Collectively, these results identify lipid-driven intracellular Ca^{2+} dysregulation as a critical pathogenic axis linking lipotoxicity to p62 multiphosphorylation and autophagy failure across both cellular and organismal systems.

CURRICULUM VITAE

김다현

성신여자대학교 바이오신약의과학부

[학력]

2015.09-2021.02 서울대학교 수의학과 (수의학박사)
2009.03-2015.02 서울대학교 수의학과 (수의학사)

[경력]

2023.09-현재 성신여자대학교
2022.07-2023.08 서울대학교 수의과학연구소, 연구조교수
2021.03-2022.06 서울대학교 수의과학연구소, 선임연구원

[관심분야]

- Advancing assembloid models by creating organ-specific niche
- Disease modeling with iPSC-derived organoids and discovering new therapeutic targets
- Regenerative therapy using stem cells and organoids

[논문]

1. Therapeutic effects of hypoimmunogenic universal human iPSC-derived endothelial cells in a humanized mouse model of peripheral artery disease, Stem Cell Research & Therapy, 16:430, 2025.
2. In Vitro Modeling of Atherosclerosis Using iPSC-Derived Blood Vessel Organoids, Advanced Healthcare Materials, 14(1):2400919, 2025.
3. Modeling of solar UV-induced photodamage on the hair follicles in human skin organoids, Journal of Tissue Engineering, 15:1-20, 2024.
4. 3D microengineered vascularized tumor spheroids for drug delivery and efficacy testing, Acta Biomaterialia, 165:153-167, 2023.
5. Bioengineered liver crosslinked with nano-graphene oxide enables efficient liver regeneration via MMP suppression and immunomodulation, Nature communications, 14:801, 2023.

In vitro modeling of atherosclerosis using iPSC-derived blood vessel organoids

Da-Hyun Kim

School of Biopharmaceutical and Medical Sciences, Sungshin Women's University, South Korea

Organoids derived from iPSCs through 3D self-organization have gained prominence in disease modeling and tissue therapy due to their remarkable structural similarity to native organs. Induced pluripotent stem cell-derived blood vessel organoids (BVOs) are applicable for modeling vascular diseases, containing multiple cell types, including endothelial and vascular smooth muscle cells self-assembled into a blood vessel structure. As modeling of atherosclerosis requires recapitulating complex interactions with vasculature and immune cells, previous in vitro models have limitations due to their insufficient 3D vascular structures. Thus, we attempt to develop atherosclerotic BVOs by creating a microenvironment associated with atherogenesis, such as shear stress, low-density lipoprotein, pro-inflammatory cytokine, and monocyte co-culture in iPSC-derived BVOs. In atherosclerotic BVOs, representative atherosclerotic phenotypes, including endothelial dysfunction, inflammatory responses, formation of foam cells and fibrous plaque, and moreover, calcification of the plaques are observed. To verify the drug response in this model, it is treated with clinically used lovastatin and confirm phenotype attenuation. Furthermore, the therapeutic efficacy of nano-sized graphene oxides (NGOs) is evaluated on atherosclerosis. Due to their anti-inflammatory effects, NGOs effectively alleviate the pathologic lesions in atherosclerotic BVOs by promoting macrophage polarization toward M2. These results suggest that atherosclerotic BVOs are advanced in vitro models suitable for drug discovery and elucidation of therapeutic mechanisms. From the perspective of precision medicine, this platform using patient-derived BVOs can be further employed for personalized drug screening in the future.

SoLA 2026

한국지질·동맥경화학회 춘계학술대회

2026 Spring Congress on
Lipid and Atherosclerosis of KSoLA

SoLA 2026

한국지질·동맥경화학회 춘계학술대회

2026 Spring Congress on
Lipid and Atherosclerosis of KSoLA

Symposium 20

Nutritional Perspectives on Sex Differences in Cardiometabolic Health

4월 4일(토) 15:30–17:00 | Room 2 (그랜드볼룸 2)

좌장 : 이명숙(성신여대 식품영양학과), 정효지(서울대 보건대학원)

패널 : 박용주(전남대 식품영양학과), 백진경(을지대 식품영양학과)
이동훈(연세대 스포츠응용산업학과)

15:30–15:50	**Sex differences in metabolic syndrome and heart** **박성미**(고려의대 순환기내과)
15:50–16:10	**Uncovering FPR2 as a determinant of sex differences in liver fibrosis** **정영미**(부산대 생명과학과)
16:10–16:30	**Sex-dependent microbe-metabolite interactions in cardiometabolic diseases** **유현주**(서울대 식품영양학과)
16:30–17:00	**Panel Discussion**

CURRICULUM VITAE

박성미

고려대학교 안암병원 순환기내과

[학력]

1997	고려대학교 의과대학 학사
2002	고려대학교 의학과 석사
2005	고려대학교 의학과 박사

[경력]

-현재	고려대학교 안암병원 순환기내과 교수, 로제타홀여성심장센터장
-현재	대한심장대사중후군학회 국내교류이사
-현재	대한심장학회 여성심장질환연구회 부회장, 심장종양학연구회 부회장
-현재	대한성차의과학회 학술이사
-현재	대한심부전학회 홍보이사
-현재	한국심초음파학회 보험이사
-현재	Korean Circulation Journal 부편집장

[관심분야]

심부전, 심장대사증후군, 허혈성심장질환, 여성심장, 성차의학, 심초음파

[논문]

1. Sex differences in diagnosis and treatment of heart failure: toward precision medicine. Lee SY, Park SM. Korean J Intern Med. 2025 Mar;40(2):196-207.
2. Deep learning for predicting rehospitalization in acute heart failure: Model foundation and external validation. Kim MN, Lee YS, Park Y, Jung A, So H, Park J, Park JJ, Choi DJ, Kim SR, Park SM. ESC Heart Fail. 2024 Dec;11(6):3702-3712.
3. Sex differences of sequential changes in coronary blood flow and microvascular function in patients with suspected angina. Kim SR, Kim MN, Cho DH, Kim HD, Bae SA, Kim HL, Kim MA, Hong KS, Shim WJ, Park SM. Clin Res Cardiol. 2024 Dec;113(12):1638-1649.
4. Epicardial Adipose Tissue and Heart Failure, Friend or Foe? Cho DH, Park SM. Diabetes Metab J. 2024 May;48(3):373-384.
5. Early menopause is associated with abnormal diastolic function and poor clinical outcomes in women with suspected angina. Bae S, Park SM, Kim SR, Kim MN, Cho DH, Kim HD, Yoon HJ, Kim MA, Kim HL, Hong KS, Shin MS, Jeong JO, Shim WJ. Sci Rep. 2024 Mar 15;14(1):6306.

Sex differences in metabolic syndrome and heart

박성미

고려대학교 안암병원 순환기내과

Sex differences play a important role in the development, clinical expression, and prognosis of metabolic syndrome and heart disease. Biological factors—including sex hormones, body fat distribution, insulin resistance, and inflammatory pathways—interact with psychosocial and lifestyle determinants to shape cardiometabolic risk differently in women and men. Women more commonly exhibit visceral adiposity-related microvascular dysfunction and heart failure with preserved ejection fraction, whereas men more frequently develop obstructive coronary artery disease. This lecture will review current evidence on sex-specific mechanisms linking metabolic syndrome to cardiovascular remodeling, discuss implications for risk stratification and imaging, and propose tailored preventive and therapeutic strategies.

CURRICULUM VITAE

정영미(Youngmi Jung)

부산대학교 생명과학과(Department of Biological Science, Pusan Nat. Uni.)

[학력]

1992-1996	이화여자대학교 학사 (Ewha Womans Uni. B.S.)
1997-1999	서울대학교 석사 (Seoul Nat. Uni. M.S)
2002-2006	University of Florida, PhD

[경력]

2006-2008	Research Associate at Duke University Medical Center
2008-2010	Assistant Professor at Duke University Medical Center
2010-현재	Assistant, Associate, Full Professor at Pusan National University

[관심분야]

(Non)alcoholic fatty liver disease, liver fibrosis, liver regeneration, somatic stem cells

[논문]

1. Chronic Nanoplastic Exposure Promotes the Development and Progression of Metabolic Dysfunction-Associated Steatotic Liver Disease. Liver International. 2025 Aug;45(8):e70224.
2. Tumor necrosis factor-inducible gene 6 protein and its derived peptide ameliorate liver fibrosis by repressing CD44 activation in mice with alcohol-related liver disease. Journal of Biomedical Science 2024;31:54.
3. Formyl peptide receptor 2 is an emerging modulator of inflammation in the liver. Exp Mol Medicine 2023; 55(2):325-332.
4. Formyl peptide receptor determines sex-specific differences in the progression of nonalcoholic fatty liver disease and steatohepatitis. Nature Communications 2022:13, 578.
5. SEVs from tonsil-derived mesenchymal stromal cells alleviate activation of hepatic stellate cells and liver fibrosis through iR-468-5p. Molecular Therapy 2021:29(4): 1472-1486.

Uncovering FPR2 as a determinant of sex differences in liver fibrosis

Youngmi Jung

Department of Biological Science, Pusan National Univeristy, Korea

Metabolic dysfunction-associated steatotic liver disease (MASLD) is a major global health burden that can progress to steatohepatitis and liver fibrosis. Clinical and epidemiological studies show that disease severity is generally higher in men than in premenopausal women, suggesting the presence of sex-dependent protective mechanisms. Formyl peptide receptor 2 (FPR2) is a G protein-coupled receptor involved in the regulation of inflammatory responses; however, its role in sex-specific susceptibility to liver injury remains unclear.

We previously demonstrated that FPR2 contributes to sex-dependent responses in diet-induced liver injury. In a choline-deficient, L-amino acid-defined high-fat diet (CDAHFD) model, male mice developed more severe liver injury and inflammation than females. Hepatic Fpr2 expression was higher in female mice, and estradiol increased Fpr2 expression in hepatocytes. Genetic deletion of Fpr2 abolished this protection and exacerbated hepatocellular injury in female mice, suggesting that hepatocyte FPR2 contributes to the relative resistance of females to diet-induced liver damage.

To further evaluate the role of FPR2 during liver fibrogenesis, we examined bile duct ligation (BDL) and carbon tetrachloride (CCl_4)-induced models. Consistent with previous reports, male mice exhibited more severe liver injury than females in both models. Deletion of Fpr2 reduced inflammatory responses in both sexes. However, despite reduced inflammation, female Fpr2 knockout mice showed increased liver fibrosis compared with wild-type females, reaching levels comparable to those observed in wild-type male mice. Increased hepatocellular injury was also observed in female Fpr2-deficient mice.

Together, these findings indicate that FPR2 contributes to sex-dependent responses during liver injury and fibrogenesis and suggest that hepatocyte FPR2 may play a role in the relative protection observed in female livers.

CURRICULUM VITAE

유현주

서울대학교 식품영양학과

[학력]

2011	서울대학교 식품영양학과 박사
2003	서울대학교 식품영양학과 석사
2001	서울대학교 식품영양학과 학사

[경력]

2023-현재	서울대학교 식품영양학과 교수
2024-현재	대한지역사회영양학회 이사
2025-현재	한국지질동맥경화학회 식품영양위원, 의료정보위원
2024-현재	International Human Microbiome Consortium LOC 학술부위원장
2026-현재	심장대사증후군학회 연구위원
2026-현재	심장대사증후군학회
2026-현재	한국영양학회
2023-현재	Clinical and Molecular Hepatology, Associate Editor

[관심분야]

생애주기에 걸친 식이-마이크로바이옴 상호작용 및 기전연구, Microbiome Therapeutics, Precision Nutrition, Cardiometabolic diseases, Oral/Gut/Vagina/Skin Microbiome

[논문]

1. You HJ et al. (2023) Bacteroides vulgatus SNUG 40005 restores Akkermansia depletion by metabolite modulation. Gastroenterology. 164(1):103-116.
2. Kang H*, You HJ* et al. (2022) Interaction effect between NAFLD severity and high carbohydrate diet on gut microbiome alteration and hepatic de novo lipogenesis. Gut Microbes.14(1): 2078612.
3. Si J, Kang H, You HJ§, Ko G§. (2022) Revisiting the role of Akkermansia muciniphila as a therapeutic bacterium. Gut Microbes. 14(1): 2078619.
4. Yoon HS, Cho CH, Yun MS, Jang SJ, You HJ, …, Ko G. (2021) Akkermansia muciniphila secretes a glucagon-like peptide-1-inducing protein that improves glucose homeostasis and ameliorates metabolic disease in mice. Nature Microbiology. 6(5):563-573.
5. Lee G*, YouHJ* et al. (2020) Distinct signatures of gut microbiome and metabolites associated with significant fibrosis in non-obese NAFLD. Nature Communications. 5; 11(1): 4982.

Sex-dependent microbe-metabolite interactions in cardiometabolic diseases

Hyun Ju You

Department of Food and Nutrition, Seoul National University;
Research Institute of Human Ecology, Seoul National University, Seoul, Republic of Korea

Emerging evidence indicates that sex-dependent differences in microbial taxa, metabolic pathways, and host-microbe interactions contribute to variability in disease susceptibility, progression, and therapeutic response. Hormonal factors play a central role in modulating these differences. Estrogen has been associated with enrichment of microbial taxa involved in short-chain fatty acid production and anti-inflammatory pathways, supporting metabolic homeostasis and cardiovascular protection. In contrast, male-associated microbiome profiles often show increased capacity for lipid metabolism and production of metabolites such as trimethylamine (TMA), a precursor of TMAO, which has been linked to atherosclerosis and cardiometabolic risk. Testosterone may further influence microbial composition and metabolic outputs, contributing to sex-specific metabolic phenotypes.

These microbe-metabolite interactions are closely linked to key cardiometabolic pathways, including glucose regulation, lipid metabolism, bile acid transformation, and systemic inflammation. Notably, sex-dependent differences in microbial metabolite production—such as short-chain fatty acids, bile acids, and amino acid-derived metabolites—can differentially modulate insulin sensitivity, hepatic lipid accumulation, and vascular inflammation.

Diet further interacts with these sex-specific microbiome features. Responses to dietary interventions, including high-fiber or high-fat diets, are mediated in part by baseline microbial composition and functional capacity, leading to distinct metabolic outcomes between males and females. These findings underscore the need to consider sex as a determinant in microbiome-targeted nutritional strategies.

Understanding sex-dependent microbe-metabolite interactions provides a framework for advancing precision medicine in cardiometabolic diseases. Integrating multi-omics approaches and genome-resolved microbiome analyses will be essential to identify actionable microbial pathways and develop sex-specific therapeutic and nutritional interventions.

SoLA 2026

한국지질·동맥경화학회 춘계학술대회

2026 Spring Congress on
Lipid and Atherosclerosis of KSoLA

SoLA 2026

한국지질·동맥경화학회 춘계학술대회

2026 Spring Congress on
Lipid and Atherosclerosis of KSoLA

Symposium 21 –
KSIC–KSoLA Joint Symposium

Targeting Atherosclerotic Vulnerability:
Lipoprotein Modulation and Inflammation

4월 4일(토) 15:30–17:00 | Room 3 (그랜드볼룸 1)

좌장 : 최동훈(연세의대 심장내과), 남창욱(계명의대 심장내과)

패널 : 김수홍(부산보훈병원 순환기내과), 김희동(순천향의대 심장내과)
양한모(서울의대 순환기내과), 원호연(중앙의대 순환기내과)

15:30–15:48	The role of early PCSK–9 inhibitor in patients with ACS **조윤경**(계명의대 심장내과)
15:48–16:06	Lp(a) and atherosclerotic burden: new targets in the post–PCI era **우종신**(경희의대 심장내과)
16:06–16:24	The association between coronary artery plaque and ANGPTL4 **한정규**(서울의대 순환기내과)
16:24–16:42	Inflammation and atherosclerosis: recent advances in anti–inflammatory therapy **김병식**(한양의대 심장내과)
16:42–17:00	Panel Discussion

CURRICULUM VITAE

조윤경

계명대학교 동산병원

[학력]

1995.3-2001.2 계명대학교 의학과 학사
2004.9-2006.8 계명대학교 의학과 석사
2006.9-2008.8 계명대학교 의학과 박사

[경력]

2012.3-2016.2 계명대학교 동산병원 조교수
2016.3-2022.2 계명대학교 동산병원 부교수
2022.3-현재 계명대학교 동산병원 교수

[관심분야]

Coronary artery disease

[논문]

1. Perfusion Abnormality in Adenosine Stress Myocardial Contrast Echocardiography in Intermediate-Risk Patients With Chest Pain. Kim H, Lee HJ, Kim IC, Lee CH, Cho YK, et al. J Clin Ultrasound. 2025 Nov 16. doi: 10.1002/jcu.70137.
2. Late stent strut apposition and coverage after drug-eluting stent implantation by optical coherence tomography in patients with acute myocardial infarction. Lee CH, Lee S, Hwang J, Kim IC, Cho YK, et al. Coron Artery Dis. 2025 Nov 1;36(7):610-617.
3. Proportion and Clinical Impact of Stent Optimization During Imaging-Guided Percutaneous Coronary Intervention: The OCTIVUS Trial. Kim H, Kang DY, Ahn JM, Kim HJ, Hur SH, Cho YK, et al. JACC Cardiovasc Interv. 2025 May 12;18(9):1089-1099.
4. Comparison of Thick Biolimus A9-Eluting Stent and Thin Zotarolimus-Eluting Stent in Multi-Vessel Percutaneous Coronary Intervention. Lee CH, Lee HJ, Chung TW, Lee S, Hwang J, Kim IC, Cho YK, et al. Korean Circ J. 2025 May;55(5):396-407.
5. Drug-Coated Balloon-Based Treatment of Left Main True Bifurcation Lesion. Her AY, Kim TH, Shin ES, Kim S, Kim B, Kim YH, Choi KH, Cho YK, et al. Catheter Cardiovasc Interv. 2025 Apr;105(5):1024-1031.

The role of early PCSK-9 inhibitor in patients with ACS

Yun-Kyeong Cho

Cardiology, Keimyung University Dongsan Hospital, Republic of Korea

Early use of PCSK9 inhibitors in patients with acute coronary syndrome (ACS) has emerged as a promising strategy to achieve rapid and profound LDL-cholesterol (LDL-C) reduction during a period of extremely high cardiovascular risk. Despite high-intensity statin therapy, many ACS patients fail to reach recommended LDL-C targets promptly, leaving a window of vulnerability for recurrent ischemic events. PCSK9 inhibitors, when initiated early—often during hospitalization or shortly after discharge—can reduce LDL-C by an additional 50-60% within weeks, enabling faster attainment of guideline-recommended levels.

Beyond lipid lowering, early PCSK9 inhibition may provide plaque-stabilizing effects. Imaging studies have demonstrated reductions in lipid core burden and favorable changes in plaque composition, suggesting potential mechanisms for early risk reduction. Clinical data also indicate that initiating therapy soon after ACS is safe, well tolerated, and associated with improved adherence compared with delayed outpatient initiation.

Patients who may benefit most include those with very high baseline LDL-C, prior cardiovascular disease, diabetes, multivessel coronary disease, or statin intolerance. Early combination therapy with statins and ezetimibe plus a PCSK9 inhibitor may be particularly useful in individuals unlikely to achieve targets with stepwise escalation.

However, challenges remain, including cost, access, and uncertainties regarding the magnitude of short-term clinical benefit when started immediately after ACS. Ongoing research continues to evaluate whether ultra-early initiation translates into significant reductions in hard cardiovascular outcomes.

Overall, early PCSK9 inhibitor therapy represents a proactive, risk-based approach in ACS management, aiming to close the treatment gap between hospitalization and long-term secondary prevention while potentially improving both lipid control and cardiovascular prognosis.

CURRICULUM VITAE

우종신

경희의료원 심장내과

[약력]

경희대학교 의과대학 졸업
경희대학교 의과대학 대학원 의학석사/의학박사
경희대학교 의과대학 심장내과 조교수, 부교수
Research Associate, UC Irvine
현) 경희대학교 의과대학 심장내과 교수

[학회 활동]

심장대사증후군학회 총무이사
한국지질동맥경화학회 학술위원, 보험법제위원
대한고혈압학회 학술위원
심근경색증연구회 학술위원, 홍보위원
대한심혈관중재학회 기획위원
대한내과학회 정회원
대한심장학회 정회원
대한심혈관중재학회 정회원
한국심초음파학회 정회원

Lp(a) and atherosclerotic burden: new targets in the post-PCI era

우종신

경희대병원 심장내과

Despite modern PCI, potent antithrombotic therapy, and intensive LDL-C lowering, many patients continue to experience MACE because PCI treats focal culprit lesions while diffuse non-culprit atherosclerosis persists. Lipoprotein(a) [Lp(a)] is a key driver of this residual risk. Lp(a) is an apoB-100 LDL-like particle covalently linked to apolipoprotein(a), and it is a major carrier of oxidized phospholipids, coupling atherogenic lipid delivery with pro-inflammatory signaling and potentially impaired fibrinolysis via apo(a)-plasminogen homology. In post-PCI cohorts, elevated Lp(a) is associated with recurrent ischemic events, repeat revascularization, and long-term stent failure phenotypes such as in-stent restenosis and neoatherosclerosis, often despite LDL-C goal attainment. Imaging with CCTA, IVUS, and OCT supports links between Lp(a) and plaque burden and vulnerability. Guidelines increasingly recommend at least one lifetime Lp(a) measurement and family cascade testing. Emerging ASO/siRNA therapies achieving ~80-95% Lp(a) lowering may enable targeted secondary prevention if outcomes trials confirm event reduction.

CURRICULUM VITAE

한정규(Jung-Kyu Han)

Professor, Division of Cardiology, Department of Internal Medicine,
Seoul National University Hospital, Seoul, South Korea

[Education]

Mar. 2007-Feb. 2012	Postgraduate School, Seoul National University (Ph.D. in Medical Science)
Mar. 2005-Feb. 2007	Postgraduate School, Seoul National University (Master of Medical Science)
Mar. 1998-Feb. 2002	Seoul National University College of Medicine (M.D.)
Mar. 1996-Feb. 1998	Premedical Course, College of Liberal Arts & Science, Seoul National University

[Employment]

Mar. 2021-Current	Professor, Division of Cardiology, Department of Internal Medicine, Seoul National University Hospital, Seoul, Korea
Sep. 2021-Current	Clinical Professor, College of Medicine, Seoul National University, Seoul, Korea
Aug. 2018-Aug. 2021	Clinical Associate Professor, College of Medicine, Seoul National University, Seoul, Korea
Sep. 2015-Feb. 2021	Associate Professor, Division of Cardiology, Department of Internal Medicine, Seoul National University Hospital, Seoul, Korea
Jun. 2019-Nov. 2020	Visiting Scholar, Cardiovascular Institute, Stanford University School of Medicine, Stanford, CA
Dec. 2012-Aug. 2015	Assistant Professor, Division of Cardiology, Department of Internal Medicine, Seoul National University Hospital, Seoul, Korea
May. 2011-Dec. 2012	Clinical Fellowship in Cardiology, Seoul National University Hospital, Seoul, Korea
Jul. 2009-Apr. 2011	Research Fellow, Harvard Medical School, Postdoctoral Research Fellow, Brigham and Women's Hospital, Boston, MA
Oct. 2008-Jul. 2009	Postdoctoral Research fellowship, National Research Laboratory for Cardiovascular Stem Cell, Clinical Research Institute, Seoul National University Hospital, Seoul, Korea
May 2008-Sep. 2008	Public Health Doctor, Director, Department of Internal Medicine, Sorokdo National Hospital, Jeollanamdo, Korea
May 2007-Feb. 2008	Clinical Fellowship in Cardiology, Seoul National University Hospital, Seoul, Korea
Mar. 2003-Feb. 2007	Residency in Internal Medicine, Seoul National University Hospital, Seoul, Korea
Mar. 2002-Feb. 2003	Internship, Seoul National University Hospital, Seoul, Korea

The association between coronary artery plaque and ANGPTL4

한정규

서울의대 순환기내과

CURRICULUM VITAE

김병식

한양대학교 구리병원 심장내과 조교수

[학력]

2009 한양대학교 의과대학 의학과
2022 한양대학교 의학석사
2024 한양대학교 의학박사

[경력]

2020 심장내과 분과전문의
2023 심장혈관중재시술 인증의

[관심분야]

심장내과, 심혈관중재, 고혈압, 이상지질혈증

[논문]

1. Kim BS, Kim HJ, Kim H, et al. Hepatic Steatosis in Cardiovascular-Kidney-Metabolic Syndrome: Fatty Liver Index as a Predictor of Cardiovascular Outcomes. Eur J Prev Cardiol. Published online June 30, 2025. doi:10.1093/eurjpc/zwaf396
2. Kim BS, Kim HJ, Moon S, Kim H, Lee J, Shin JH. Association of the triglyceride-glucose index with cardiovascular outcomes across cardiovascular-kidney-metabolic syndrome stages. Korean J Intern Med. 2025;40(6):961-974.
3. Kim BS, Kim J, Choi N, Kim HJ, Shin JH. Low-density lipoprotein cholesterol and clinical outcomes in patients with liver cirrhosis: a nationwide cohort study. Ann Med. 2025;57(1):2551813. doi:10.1080/07853890.2025.2551813
4. Kim HJ, Kim BS, Kim H, et al. Long-term clinical outcome and risk stratification across stages of cardiovascular-kidney-metabolic syndrome in a nationwide cohort. Korean J Intern Med. 2025;40(6):975-989. doi:10.3904/kjim.2025.194
5. Kim BS, Kim J, Choi N, Kim HJ, Shin JH. Associations Between Low-Density Lipoprotein Cholesterol Levels and Cardiovascular Outcomes in Patients Undergoing Dialysis: A Nationwide Cohort Study. J Clin Med. 2025;14(14):4845. Published 2025 Jul 8. doi:10.3390/jcm14144845

Inflammation and atherosclerosis: recent advances in anti-inflammatory therapy

Byung Sik Kim

Department of Cardiology, Hanyang University Guri Hospital, Korea

Atherosclerosis is a chronic inflammatory disease initiated by endothelial dysfunction and sustained by interactions between lipoproteins and immune pathways. Beyond lipid accumulation, inflammatory processes drive plaque progression, instability, and thrombotic complications. High-sensitivity C-reactive protein (hsCRP) reflects this residual inflammatory risk and provides prognostic information independent of LDL cholesterol.

Recent evidence supports the clinical integration of inflammation into cardiovascular risk assessment. Measurement of hsCRP enables identification of individuals at increased risk, even in the absence of elevated LDL-C, and complements traditional risk stratification. In primary prevention, elevated hsCRP may guide intensification of lifestyle interventions and lipid-lowering therapy. In secondary prevention, persistent inflammation despite optimal lipid control identifies patients at high residual risk and may warrant additional therapeutic strategies.

Anti-inflammatory approaches, particularly low-dose colchicine, have demonstrated reduction in recurrent cardiovascular events when used adjunctively to standard care, although benefits depend on clinical context and timing. Ongoing trials targeting specific inflammatory pathways may further refine treatment strategies.

These findings support a paradigm shift toward integrated management of atherosclerotic cardiovascular disease that addresses both lipid burden and vascular inflammation.

SoLA 2026

한국지질·동맥경화학회 춘계학술대회

2026 Spring Congress on
Lipid and Atherosclerosis of KSoLA

SoLA 2026

한국지질·동맥경화학회 춘계학술대회

2026 Spring Congress on
Lipid and Atherosclerosis of KSoLA

Breakfast Symposium 1

4월 4일(토) 07:30-08:00 | Room 1 (그랜드볼룸 3)

좌장 : 신현호(아산충무병원 심장내과), 박철영(성균관의대 내분비내과)

07:30-07:45	**Safety and efficacy of moderate-intensity statin with ezetimibe in elderly patients with ASCVD** **차정준**(고려의대 순환기내과)
07:45-08:00	**Benefits of low dose rosuvastatin plus ezetimibe combination therapy** **김원진**(차의대 내분비내과)

CURRICULUM VITAE

차정준(Jung-Joon Cha)

고려의대 순환기내과

[Education]

- Doctor of Philosophy, Gwangju Institute of Science and Technology Thesis: Study on Aging Process of Cardiovascular Cells Using a Micro-Electrochemical Impedance Spectroscopy
- Master's Degree, Yonsei University College of Medicine Thesis: The association between cardiac involvement and long-term clinical outcomes in patients with Duchenne muscular dystrophy
- M.D., Yonsei University Wonju College of Medicine

[Licensure and Certification]

2021	Fellowship of ESC, European Society of Cardiology
2021	Certified Interventional Cardiologist, Korean Society of Interventional Cardiology
2020	License of Cardiologist, Korean Board of Internal Medicine
2014	License of Internal Medicine, Korean Board of Internal Medicine
2009	License of Medical Doctor (101776), Republic of Korea

[Graduate Training]

Yonsei University Severance Hospital

2019-2020	Interventional Fellowship in Cardiology
2018-2019	General Fellowship in Cardiology

Yonsei University Gangnam Severance Hospital

2010-2014	Residency in Internal Medicine
2009-2010	Internship

[Professional Experience]

Korea University Anam Hospital, Korea University College of Medicine

2024-	Clinical Associate Professor
2020-2024	Clinical Assistant Professor

Safety and efficacy of moderate-intensity statin with ezetimibe in elderly patients with ASCVD

CURRICULUM VITAE

김원진

차의과학대학교 강남차병원 내분비내과

[학력]

2008 연세대학교 원주의과대학 학사
2012 연세대학교 의과대학 석사
2023 연세대학교 의과대학 박사

[경력]

2008-2013 연세대학교 신촌세브란스병원 인턴/레지던트(내과)
2013-2015 연세대학교 신촌세브란스병원 내분비내과 임상연구조교수
2015-현재 차의과학대학교 강남차병원 내분비내과 부교수

[관심분야]

당뇨병, 고지혈증, 갑상선, 뇌하수체

[논문]

1. W Kim, MK Seo, YJ Kim, SH Choi, CR Ku, S Kim, EJ Lee, JS Yoon. Role of the suppressor of cytokine signaling-3 in the pathogenesis of Graves' orbitopathy. Front. Endocrinol., 04 March 2025. https://doi.org/10.3389/fendo.2025.1527275
2. KH Chun, HJ Kim, DR Kang, JY Kim, W Kim, YW Jeong, SH Han, KK Koh. Sex-Specific Impact of the COVID-19 Outbreak on the Incidence of Metabolic Syndrome: A Comparative Study of 2018-2019 and 2020-2021. Korean J Intern Med 2025 Mar;40(2):262-274. doi: 10.3904/kjim.2024.288. Epub 2025 Mar 1.
3. HJ Kim, DR Kang, JY Kim, W Kim, YW Jeong, KH Chun, SH Han, KK Koh. Metabolic Syndrome Fact Sheet 2024: Executive Report. Cardiometab Syndr J. 2024;4:e14.
4. W Kim, SK Park, YL Kim. Fetal abdominal obesity in women with one value abnormality on diagnostic test for gestational diabetes mellitus. PLOS ONE 2024, 19(6): e0304875. https://doi.org/10.1371/journal.pone.0304875.
5. W Kim, SK Park, YL Kim. Fetal abdominal obesity and the ensuing adverse perinatal outcomes in older obese pregnant women with or without obesity and with normal glucose tolerance. Scientific Reports. 2023, 13: 16206.

Benefits of low dose rosuvastatin plus ezetimibe combination therapy

SoLA 2026

한국지질·동맥경화학회 춘계학술대회

2026 Spring Congress on
Lipid and Atherosclerosis of KSoLA

SoLA 2026

한국지질·동맥경화학회 춘계학술대회

2026 Spring Congress on
Lipid and Atherosclerosis of KSoLA

Breakfast Symposium 2

4월 4일(토) 07:30-08:00 | Room 2 (그랜드볼룸 2)

좌장 : 우정택(경희의대 내분비대사내과), 조진만(경희의대 심장혈관내과)

07:30-07:45	**Appropriate statin therapy for low- to moderate-risk patients ("The earlier, the better; the longer, the better.")** **정창희**(울산의대 내분비내과)
07:45-08:00	**CKD and lipid management: when guidelines differ, what should we do?** **박세훈**(서울의대 신장내과)

CURRICULUM VITAE

정창희

울산의대 서울아산병원 내분비내과

[학력]

1996-2002	고려대학교 의과대학 의학 학사
2010-2012	울산대학교 의과대학 의학 석사
2012-2014	울산대학교 의과대학 의학 박사

[경력]

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

[관심분야]

Diabetes complications, Therapeutics, Obesity, Adipose tissue dysfunction

[논문]

1. Cho YK, Jung CH. Sodium-Glucose Cotransporter 2 Inhibitors as Emerging Anticancer Agents. Diabetes Metab J. 2026;50:1-18.
2. Lee W*, Kim ES, Kim S, Park H, Lee JK, Baek E, Jung CH*, Cho SH. Noninvasive subterahertz glucose monitoring using a communication inspired eye diagram. Sci Rep. 2025;16:1079. (*Co-corresponding author)
3. Cho YK, Kim MJ, Kim EH, Lee MJ, Nam HJ, Lee WJ, Kim HK, Jung CH. Comparison of diagnostic criteria for fatty liver disease in assessing cardiac dysfunction: a cross-sectional study. Hepatol Int.2026;20:81-90.
4. Cho YK, Jung CH. Engineered nutrient-stimulated hormonal multi-agonist for precision targeting of obesity and metabolic disorders. Clin Mol Hepatol. 2025 Nov 26. Online ahead of print.
5. Jang MK, Cho YK, Moon JY, Min SH, Hwang JH, Jung CH. Impact of smart watch mobile application on the risk treatment of type 2 diabetes mellitus (iSMART-DM). Prim Care Diabetes. 2026;20:47-52.

Appropriate statin therapy for low- to moderate-risk patients ("The earlier, the better; the longer, the better.")

CURRICULUM VITAE

박세훈

서울대학교병원 신장내과 임상조교수

[학력]

2007-2013	서울대학교 의과대학 의학과, 의학 학사
2016-2023	서울대학교 의과대학 의과학과 석박통합과정, 의학 박사

[경력]

2014-2018	서울대학교병원 내과 전공의
2018-2019	서울대학교병원 신장내과 임상강사
2019-2022	국군수도병원 내과 군의관, 군복무
2022-2023	서울대학교 의과대학 의과학과 박사과정 연구원, 의사과학자 양성과정 (보건산업진흥원)
2023-2025	서울대학교병원 신장내과 진료조교수
2026-현재	서울대학교병원 신장내과 임상조교수

[학회 활동]

전) 대한신장학회 수련교육위원회 간사
현) 대한신장학회 학술위원회 간사

CKD and lipid management: when guidelines differ, what should we do?

SoLA 2026

한국지질·동맥경화학회 춘계학술대회

2026 Spring Congress on
Lipid and Atherosclerosis of KSoLA

SoLA 2026

한국지질·동맥경화학회 춘계학술대회

2026 Spring Congress on
Lipid and Atherosclerosis of KSoLA

Breakfast Symposium 3

4월 4일(토) 07:30-08:00 | Room 3 (그랜드볼룸 1)

좌장 : 김명곤(가톨릭관동의대 심장내과), 박헌식(경북의대 순환기내과)

07:30-07:45 **Clinical rationale for acid suppressants in cardiology; a multidisciplinary perspective**

박상민(을지의대 심장내과)

07:45-08:00 **P-CAB treatment strategies: a case-based approach**

장영우(가천의대 심장내과)

CURRICULUM VITAE

박상민

을지의대 심장내과

[전문분야]

협심증 및 심근경색, 고혈압, 심부전, 이상지질혈증, 대동맥 및 말초혈관질환, 부정맥

[전문진료]

내과학, 협심증, 심근경색, 고혈압, 심부전, 이상지질혈증, 대동맥 및 말초혈관질환, 부정맥

[주요 진료분야]

내과학, 관상동맥질환 (협심증, 심근경색), 고혈압, 심부전, 이상지질혈증, 대동맥 및 말초혈관질환, 부정맥

[학력]

연세대학교 대학원 의학과 석사
연세대학교 대학원 의학과 박사

[경력]

세브란스 심장혈관병원 임상강사(1998.03-2010.02)
한림대학교 춘천성심병원 순환기내과 부교수(2013.01-2020.06)
현) 노원을지대학교병원 심장내과 부교수

Clinical rationale for acid suppressants in cardiology; a multidisciplinary perspective

CURRICULUM VITAE

장영우(Youngwoo Jang)

가천대학교 길병원 조교수

[학력 및 경력]

2008-2012	School of Medicine, Gachon University, Doctor of Medicine
2017-2018	Cardiovascular Institute, Stanford Medicine, Postdoctoral Research Associate
2018-2019	Department of Cardiology, Gachon University, Gil Medical Center, Clinical Fellow
2019-2021	Department of Cardiology, Gachon University, Gil Medical Center, Clinical Assistant Professor
2022-	Department of Cardiology, Gachon University, Gil Medical Center, Assistant Professor

[학회활동]

2023-	심장대사증후군학회 학술간사
2025-	대한지질동맥경화학회 부총무/진료지침 간사

[관심 연구 분야]

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

[주요 논문 및 저서]

1. Jang Y, Lee JH, Lee SG, Jeong IK, Kim BJ. A Position Paper on Lipoprotein(a) from the Lipoprotein(a) Task Force Team of the Korean Society of Lipid and Atherosclerosis: Current Evidence, Clinical Applications, and Future Directions. Journal of Lipid Atherosclerosis and Korean Circ J. 2026 Jan;56(1):9-32.
2. Jang Y, Rhee EJ, Choi SH. Innovative Lipid-Lowering Strategies: RNA-Based, Small Molecule, and Protein-Based Therapies. Endocrinol Metab (Seoul) 2025;40:668-86.
3. Jang Y, Park SD, Lee JP, et al. One-month dual antiplatelet therapy followed by prasugrel monotherapy at a reduced dose: the 4D-ACS randomised trial. EuroIntervention 2025.
4. Kang SH, Pack KY, Kim JH, Jang Y (corresponding author). The effect of sarpogrelate compared to aspirin in high- or very-high-risk diabetes for primary prevention. Sci Rep. 2025 Jan 29;15(1):3616. doi: 10.1038/s41598-025-87868-x.
5. Kang SH, Lee J, Kim JH, Jang Y (corresponding author). Comparative Effectiveness of Clopidogrel Versus Aspirin for Primary Prevention in High-Risk Patients with Type 2 Diabetes: A Nationwide Propensity Score-Matched Cohort Study. Medicina (Kaunas) 2025;61.

P-CAB treatment strategies: a case-based approach

SoLA 2026

한국지질·동맥경화학회 춘계학술대회

2026 Spring Congress on
Lipid and Atherosclerosis of KSoLA

SoLA 2026

한국지질·동맥경화학회 춘계학술대회

2026 Spring Congress on
Lipid and Atherosclerosis of KSoLA

Breakfast Symposium 4

교육위원회 세션

4월 4일(토) 07:30-08:00 | Room 4 (볼룸 1,2)

좌장 : 백상홍 (가톨릭의대 순환기내과)

07:30-07:45	**2026 ACC/AHA 가이드라인, 무엇이 달라졌나요?** **홍준화**(을지의대 내분비내과)
07:45-08:00	**심부전 환자에서 지질관리** **김경희**(세종병원 심장내과)

CURRICULUM VITAE

홍준화

대전 을지대학교병원

[학력]

2004	을지의대 학사
2008	을지의대 학사
2015	을지의대 내과 박사

[경력]

2014	충남대학교병원 전임의
2016	경북대학교병원 임상교수
현재	대전을지대학교병원 부교수

[관심분야]

비만, 당뇨병, 이상지질혈증, 갑상선, 골다공증, 부신

[논문]

1. Efficacy and safety of adding a fourth oral antidiabetic drug versus metformin dose escalation in patients with type 2 diabetes inadequately controlled on triple oral combination therapy (EFFORT): A 24-week, randomized, open-label, multicenter trial. Diabetes Obes Metab. 2026;1-12.
2. Letter: Impact of Remnant Cholesterol on the Risk for End-Stage Renal Disease in Type 2 Diabetes Mellitus: A Nationwide Population-Based Cohort Study (Diabetes Metab J 2025;49:1106-15). Diabetes Metab J 2026;50:190-191.
3. Efficacy and Safety of HD-6277, a Novel G Protein-Coupled Receptor 40 Agonist, in Individuals with Type 2 Diabetes Mellitus: A Double-Blind, Randomized, Placebo-Controlled, Parallel-Group, Multicenter Phase 2 Clinical Trial. Diabetes Metab J. 2025 Dec 19. doi: 10.4093/dmj.2025.0528.
4. Efficacy and Safety of Enavogliflozin as Add-on in Adults with Type 2 Diabetes Mellitus Inadequately Controlled with Insulin or Insulin with Other Antidiabetic Drugs. Diabetes Metab J. 2025 Dec 15. doi: 10.4093/dmj.2025.0477.
5. Collaborators: Coadministered Cagrilintide and Semaglutide in Adults with Overweight or Obesity. N Engl J Med. 2025 Aug 14;393(7):635-647. doi: 10.1056/NEJMoa2502081. Epub 2025 Jun 22.

2026 ACC/AHA 가이드라인, 무엇이 달라졌나요?

홍준화

대전 을지대학교병원

CURRICULUM VITAE

김경희

인천 세종병원

[학력]

서울대학교 의과대학 석사
서울대학교 의과대학 박사

[경력]

서울대학교 병원 수련의, 전공의, 전임의
세종병원 심장이식 센터장
국제 심폐이식 학회 이사
미국 미네소타 로체스터 메이요 클리닉 연수
한국 장기 조직 기증원 이사

[관심분야]

심부전, 심장이식, 폐동맥 고혈압, 희귀 난치 질환, AI

[논문]

1. The International Society for Heart and Lung Transplantation (ISHLT) guidelines for the care of heart transplant recipients
2. Detection and prognostic stratification of left ventricular systolic dysfunction in left bundle branch block using an artificial intelligence-enabled electrocardiography
3. Inflammation and Heart Failure
4. A multi-ethnic foundation model-based artificial intelligence electrocardiogram for detection and prognostication of elevated left ventricular filling pressure
5. Artificial Intelligence-Enabled Electrocardiography for Detecting Risk of Rehospitalization in patients with Heart Failure

심부전 환자에서 지질관리

김경희

세종병원 심장내과

SoLA 2026

한국지질·동맥경화학회 춘계학술대회

2026 Spring Congress on
Lipid and Atherosclerosis of KSoLA

SoLA 2026

한국지질·동맥경화학회 춘계학술대회

2026 Spring Congress on
Lipid and Atherosclerosis of KSoLA

Luncheon Symposium 1

4월 4일(토) 12:00-12:30 | Room 1 (그랜드볼룸 3)

좌장 : 이문규(을지의대 내분비내과), 장학철(서울의대 내분비대사내과)

12:00-12:15	**Integrated management of hypertension and dyslipidemia for cardiovascular disease prevention** **박상우**(울산의대 심장내과)
12:15-12:30	**Is there a better treatment option for dyslipidemia patients with impaired glucose metabolism?** **김대원**(가톨릭의대 심장내과)

CURRICULUM VITAE

박상우

울산대학교병원

[학력]

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

[경력]

2011-2015	서울아산병원 전공의
2018-2019	서울아산병원 심장내과 임상강사
2019.9-현재	울산대학교병원 심장내과 부교수

[관심분야]

동맥경화, 심혈관 위험인자, 1차 예방, 관상동맥질환

[논문]

1. Temporal modulation of antiplatelet therapy in high-risk patients undergoing complex percutaneous coronary intervention: the TAILORED-CHIP randomized clinical trial. Eur Heart J. 2025 Aug 31:ehaf652.
2. Association of metabolic dysfunction-associated steatotic liver disease and steatosis-associated fibrosis estimator with subclinical coronary atherosclerosis: observation cohort study. Sci Rep. 2025 Jul 10;15(1):24953.
3. Differential impact of lipoprotein(a) on subclinical coronary atherosclerosis in asymptomatic individuals with and without diabetes mellitus. Sci Rep. 2025 Jul 1;15(1):20436.
4. Enhancing quantitative coronary angiography (QCA) with advanced artificial intelligence: comparison with manual QCA and visual estimation. Int J Cardiovasc Imaging. 2025 Mar;41(3):559-568.
5. Association between smoking status and subclinical coronary atherosclerosis in asymptomatic Korean individuals. Epidemiol Health. 2024;46:e2024064.

Integrated management of hypertension and dyslipidemia for cardiovascular disease prevention

CURRICULUM VITAE

김대원

가톨릭대학교 대전성모병원 부교수

[학력 & 경력]

2007.2	전북대학교 졸업
2007.3	의사 면허 취득
2007.3-2008.2	가톨릭 중앙 의료원 인턴
2008.3-2012.2	가톨릭 중앙 의료원 내과 레지던트
2010.9	가톨릭 대학교 석사 과정 시작
2012.3	내과 전문의 취득
2012.4-2014.11	KOICA 국제 협력 의사 - 캄보디아 시엠립 주립 병원 : 시엠립 주립 병원 내과 중환자실 리모델링 현장 사업 (5만불) 진행
2015.8	가톨릭 중앙 의료원 내과학 석사학위 취득 & 대학원 학술상 수상
2015.8	가톨릭 대학교 박사 과정 시작
2015.5-2017.3	대전성모병원 심장내과 임상강사
2017.3	임상 조교수 임용
2017.3	한국 심초음파 인증의 자격증 취득
2017.4.26	TCTAP 2017 best abstract award
2017.9	심장내과 분과 전문의 취득
2018.7	대한내과학회 내과 전공의 심초음파 지도 인증의 취득
2018.7	대한 심혈관중재학회 중재시술 인증의 취득
2018.8.16	가톨릭 중앙 의료원 내과학 박사학위 (제 3468호) 취득 & 대학원 학술상 수상
2019.2.24	헌혈 훈장 금장
2019.3.9	가톨릭 대학교 내과학 교실 올해의 젊은 연구자상 (Young Investigator of 2018)
2019.7	대한심장학회 심장학 연구재단 신진 연구비 선정 : 환자 맞춤형 3D 프린팅 모형을 이용한 시뮬레이션이 심장판막수술 및 경피적 대동맥 판막 치환술 (TAVI, transcatheter aortic valve implantation)에 미치는 임상적 유용성
2020.3.1	전임 조교수 임용
2024.3.1	전임 부교수 임용
2020.9.1-2023.2.28	(연구개발 과제 번호 :RS-2020-NR053328) 과학기술정보통신부 주관 한국연구재단 국책연구과제 (생애 첫 연구) 3년 선정: 인공지능 딥러닝 기술을 이용한 객관적, 정량적 관상동맥 병변 측정방법 개발

Is there a better treatment option for dyslipidemia patients with impaired glucose metabolism?

SoLA 2026

한국지질·동맥경화학회 춘계학술대회

2026 Spring Congress on
Lipid and Atherosclerosis of KSoLA

SoLA 2026

한국지질·동맥경화학회 춘계학술대회

2026 Spring Congress on
Lipid and Atherosclerosis of KSoLA

Luncheon Symposium 2

4월 4일(토) 12:00-12:30 | Room 2 (그랜드볼룸 2)

좌장 : 김치정(중앙의대 순환기내과), 박경수(건국의대 내분비대사내과)

12:00-12:15	**Beyond the Statin, ROSUZET; start early, switch now** **홍준화**(을지의대 내분비내과)
12:15-12:30	**Ultra-low-dose triple FDC in hypertension: a new initial treatment paradigm and the patients who benefit** **이종영**(한림의대 순환기내과)

CURRICULUM VITAE

홍준화

대전 을지대학교병원

[학력]

2004	을지의대 학사
2008	을지의대 학사
2015	을지의대 내과 박사

[경력]

2014	충남대학교병원 전임의
2016	경북대학교병원 임상교수
현재	대전을지대학교병원 부교수

[관심분야]

비만, 당뇨병, 이상지질혈증, 갑상선, 골다공증, 부신

[논문]

1. Efficacy and safety of adding a fourth oral antidiabetic drug versus metformin dose escalation in patients with type 2 diabetes inadequately controlled on triple oral combination therapy (EFFORT): A 24-week, randomized, open-label, multicenter trial. Diabetes Obes Metab. 2026;1-12.
2. Letter: Impact of Remnant Cholesterol on the Risk for End-Stage Renal Disease in Type 2 Diabetes Mellitus: A Nationwide Population-Based Cohort Study (Diabetes Metab J 2025;49:1106-15). Diabetes Metab J 2026;50:190-191.
3. Efficacy and Safety of HD-6277, a Novel G Protein-Coupled Receptor 40 Agonist, in Individuals with Type 2 Diabetes Mellitus: A Double-Blind, Randomized, Placebo-Controlled, Parallel-Group, Multicenter Phase 2 Clinical Trial. Diabetes Metab J. 2025 Dec 19. doi: 10.4093/dmj.2025.0528.
4. Efficacy and Safety of Enavogliflozin as Add-on in Adults with Type 2 Diabetes Mellitus Inadequately Controlled with Insulin or Insulin with Other Antidiabetic Drugs. Diabetes Metab J. 2025 Dec 15. doi: 10.4093/dmj.2025.0477.
5. Collaborators: Coadministered Cagrilintide and Semaglutide in Adults with Overweight or Obesity. N Engl J Med. 2025 Aug 14;393(7):635-647. doi: 10.1056/NEJMoa2502081. Epub 2025 Jun 22.

Beyond the Statin, ROSUZET; start early, switch now

CURRICULUM VITAE

이종영

한림대학교 성심병원 순환기내과

[학력]

1999.2	영남대학교 의과대학 졸업
2011.2	울산대학교 의과대학 석사
2013.2	울산대학교 의과대학 박사

Ultra-low-dose triple FDC in hypertension: a new initial treatment paradigm and the patients who benefit

SoLA 2026

한국지질·동맥경화학회 춘계학술대회

2026 Spring Congress on
Lipid and Atherosclerosis of KSoLA

SoLA 2026

한국지질·동맥경화학회 춘계학술대회

2026 Spring Congress on
Lipid and Atherosclerosis of KSoLA

Luncheon Symposium 3

4월 4일(토) 12:00-12:30 | Room 3 (그랜드볼룸 1)

좌장 : 박영배(서울의대 순환기내과), 김효수(서울의대 순환기내과)

12:00-12:15	**Clinical updates in dyslipidemia: positioning baroezet for optimal patient care** **천대영**(한림의대 순환기내과)
12:15-12:30	**Strategies for high-risk dyslipidemia patients focusing on ASCVD** **배장환**(부산좋은삼선병원 순환기내과)

CURRICULUM VITAE

천대영

한림대학교 동탄성심병원 순환기내과

[학력]

2006-2012 한림대학교 의과대학 학사
2018-2023 한국방송통신대학교 바이오정보.통계학과 석사

[경력]

2012-2017 한림대학교성심병원 인턴, 레지던트
2020-2021 서울대학교 순환기내과 임상강사
2021- 한림대학교 동탄성심병원 임상강사, 임상조교수, (현) 조교수

[관심분야]

심혈관계 중재시술, 고혈압, 죽상동맥경화, 빅데이터 연구

[논문]

1. Dementia risk across blood pressure categories: a South Korean nationwide study, European Heart Journal, 2025.10 (Co-Author)
2. Cardiovascular risk across blood pressure categories defined by the 2024 ESC and 2023 ESH hypertension guidelines: insights from a Korean nationwide cohort study, European Journal of Preventive Cardiology, 2025.9 (1st author)
3. Association between Metabolic Syndrome and Young-Onset Dementia: A Nationwide Population-Based Study. Neurology, 2025.4 (Co-corresponding author)
4. Diabetes status, duration and risk of dementia in patients with myocardial infarction, Diabetology & Metabolic Syndrome, 2025.7 (Co-1st author)
5. Depression and Risk of Stroke and Mortality after Percutaneous Coronary Intervention: A Nationwide Population Study, Journal of Internal Medicine, 2024.09 (Co-1st Author)

Clinical updates in dyslipidemia: positioning baroezet for optimal patient care

CURRICULUM VITAE

배장환

좋은삼선병원

[학력]

1988-1994	충북대학교 의예과 의학과 (의학사)
1995-1997	충북대학교 의과대학원 내과학 석사
1998-2004	충북대학교 의과대학원 내과학 박사

[경력]

2002-2004	서울대학교병원 순환기내과 전임의
2004-2005	경희대학교 부속병원 순환기내과 임상조교수
2005-2024	충북대학교병원 순환기내과 교수
2024-현재	은성의료재단 좋은삼선병원 순환기내과, 심혈관중재시술연구소장

[관심분야]

순환기내과, 심혈관중재, 이상지질혈증

[논문]

1. Physician Disagreement With Guidelines as a Critical Barrier to Achieving LDL-C Targets in Very High-Risk Patients: An Implementation Science Study. J Korean Med Sci. 2026 Mar 2;41(8):e74. (공저자)
2. Shared Decision-Making in Chronic Stable Angina and Severe Aortic Stenosis. Korean Circ J. 2025 Nov 18. doi: 10.4070/kcj.2025.0261. (교신저자)
3. Metabolic Regulatory Mechanism of Gastric Motility by Acetylcholine. Gastroenterology Res. 2025 Nov 17;18(6):286-298. (교신저자)
4. Intravascular imaging-guided percutaneous coronary intervention for acute myocardial infarction according to ACC/AHA lesion classification. Rev Esp Cardiol (Engl Ed). 2025 Dec 4:S1885-5857(25)00345-7. (공동저자)
5. The Clinical Impact of Intravascular Imaging-Guided Percutaneous Coronary Intervention in Acute Myocardial Infarction Patients with High Thrombus Burden. Am J Cardiol. 2026 Jan 1:258:54-62.doi: 10.1016/ (공동저자)

Strategies for high-risk dyslipidemia patients focusing on ASCVD

SoLA 2026

한국지질·동맥경화학회 춘계학술대회

2026 Spring Congress on
Lipid and Atherosclerosis of KSoLA

SoLA 2026

한국지질·동맥경화학회 춘계학술대회

2026 Spring Congress on
Lipid and Atherosclerosis of KSoLA

Oral Presentations

Oral Presentation 1

☞ **일시: 2026년 4월 3일(금) 12:00-13:20**
☞ **장소: Room 4 (볼룸 1,2)**

- 좌장 : 국현(전남의대 약리학교실), 김학령(서울의대 순환기내과)
- 발표 : OP1-1 ~ OP1-5

Oral Presentation 2

☞ **일시: 2026년 4월 4일(토) 08:30-10:00**
☞ **장소: Room 4 (볼룸 1,2)**

- 좌장 : 박용식(경희의대 미생물학교실), 한정규(서울의대 순환기내과)
- 발표 : OP2-1 ~ OP2-5

Oral Presentation 3

☞ **일시: 2026년 4월 4일(토) 15:30-17:00**
☞ **장소: Room 4 (시그니엘 부산 4층 볼룸 1,2)**

- 좌장 : 김대중(아주의대 내분비대사내과), 신미승(가천의대 심장내과)
- 발표 : OP3-1 ~ OP3-6

SoLA 2026

한국지질·동맥경화학회 춘계학술대회

2026 Spring Congress on
Lipid and Atherosclerosis of KSoLA

OP1-1 *2. Basic Science of Atherosclerosis*

Discovery therapeutic miRNAs in LPS-treated mouse endothelium

Lan Phuong Phan*, Yujin Jin, Kyung-Sun Heo

College of Pharmacy, Chungnam National University, Daejeon, Republic of Korea

Objectives: Endothelial cell (EC) dysfunction plays a critical role in the development of cardiovascular diseases (CVDs), triggered by inflammatory stimuli such as lipopolysaccharide (LPS). Signal transducer and activator of transcription 3 (STAT3), which is known to mediate vascular inflammation and remodeling, has recently been linked to EC-cardiomyocyte communication. While microRNAs (miRNAs) have been recognized as regulators of EC function, their role in STAT3-mediated EC-cardiomyocyte crosstalk remains poorly understood. This study aims to investigate the interaction between STAT3 signaling and miRNA regulation in EC-mediated cardiomyocyte function.
Methods: Mice were treated under four conditions: control, LPS, Stattic (a STAT3 inhibitor), and LPS combined with Stattic. Extracted RNA from mouse aortic EC was then subjected to small RNA sequencing to identify novel miRNAs. Candidate miRNAs were subsequently validated with RT-qPCR. Five databases (DIANA micro-T, miRmap, TargetScan, PicTar, and miRDB) were utilized for target prediction. In addition, promoter regions were analyzed using FIMO and JASPAR2024 to identify potential transcription factor binding sites.
Results: Extracted RNA from mouse aortic EC was subjected to small RNA sequencing, revealing 248 differentially expressed miRNAs. Cross-referencing with known endothelial miRNA datasets identified 7 novel LPS-induced miRNAs including mir-219-1-3p, mir-875-5p, mir-547-5p, mir-713-3p, mir 7218-5p, mir-201-3p, mir-3544-5p. Target prediction identified established regulator of EC dysfunction including Rock2, Fzd7, and Tsc1. Notably, the analysis also highlighted shared downstream genes such as Luzp1 and Zbtb20, both of which are highly expressed in cardiomyocytes, suggesting their roles in EC-cardiomyocyte signaling. Although STAT3 was not predicted as a direct miRNA target, promoter analysis (5 kb upstream and 500 bp downstream of the transcription start sites) revealed potential STAT3 binding sites in the promoters of several candidate miRNAs, indicating potential regulatory link.
Conclusions: These findings suggest that STAT3 may modulate a subset of inflammation-responsive miRNAs in aortic ECs, potentially affecting cardiomyocyte behavior via EC-cardiomyocyte.
Keyword: miRNAs, LPS

OP1-2 *5. Others*

New role of the carboxyl terminus of Hsc70-interacting protein in angiotensin II-induced aortic aneurysm

Thuy Le Lam Nguyen[1*], Diem Thi Ngoc Huynh[1], HaeSeung Lee[2], Kyung-Sun Heo[1]

[1]College of Pharmacy, Chungnam National University, Daejeon,
[2]College of Pharmacy, Pusan National University, Busan, Republic of Korea

Objectives: Excessive proliferation and migration of vascular smooth muscle cells (VSMCs) contribute to vascular diseases such as aortic aneurysm (AA). The carboxyl terminus of Hsc70 interacting protein (CHIP), a U-box-type chaperone-associated E3 ligase, plays an important role in protein degradation; however, its function in VSMCs remains poorly understood. This study aimed to investigate the role of CHIP in VSMC dysfunction, focusing on angiotensin II (Ang II)-induced AA.
Methods: Single-nucleus RNA sequencing (snRNA-seq) data (GSE207784) from 7 control subjects and 6 patients with thoracic aortic aneurysm (TAA) were analyzed to identify key molecular changes. In vivo, AA was induced in wild-type (WT) and CHIP heterozygous (CHIP+/-) male mice by Ang II infusion (1000 ng/kg/min) and β-aminopropionitrile (BAPN) for 14 days. In vitro, treated VMSCs were subjected to wound healing assay, SRB assay, western blotting to examine cell proliferation and migration.
Results: Analysis of human snRNA-seq data revealed that VSMCs from TAA patients exhibited elevated expression of CHIP, KLF4, and cyclin D1 compared with controls, suggesting a role for CHIP in mitochondrial regulation and VSMC phenotypic switching. In vivo AA model, we found that CHIP heterozygosity significantly attenuated Ang II/BAPN-induced aortic dilatation, elastin degradation and calcification as indicated by H&E, EVVG and Alizarin red S staining, respectively. In vitro, CHIP deficiency significantly suppressed Ang II-induced VSMC migration and proliferation. Consistently, si-CHIP silencing reduced Ang II-induced KLF4-mediated synthetic switching markers vimentin.
Conclusions: CHIP is involved in Ang II-induced aortic aneurysm, suggesting that CHIP is a potential therapeutic target for treatment of vascular diseases.
Keyword: CHIP, Aortic aneurysm, VSMC dysfunction

OP1-3 5. Others

Tyrosine phosphorylation of CKMT2 confers cardioprotection during hypoxia/reoxygenation

Jed Allyn Hernandez[1,4*], Jubert Marquez[1], Nammi Park[1], Maria Victoria Faith Garcia[1], Ippei Shimizu[3], Sung Ryul Lee[1], Hyoung Kyu Kim[1,2,4], Jin Han[1,2,4]

[1]Cardiovascular and Metabolic Disease Center, Smart Marine Therapeutics Center, Inje University, Busan, [2]Department of Health Sciences and Technology, Graduate School of Inje University, Busan, Republic of Korea, [3]Department of Cardiovascular Biology and Medicine, Niigata University Graduate School of Medical and Dental Sciences, Niigata, Japan, [4]Department of Physiology, College of Medicine, Inje University, Busan, Republic of Korea

Objectives: Ischemic cardiomyopathy (ICM) is the primary cause of heart failure worldwide. Ischemic preconditioning (IPC) has been found to protect the heart against ICM by regulating mitochondrial function as evidenced in proteomic and genomic studies. However, regulation of protein expression in IPC occurs rapidly given the brief nature of IPC induction underscoring the critical role of post-translational modifications in modulating protein activity. As such, this mainly aimed to identify and characterize novel phosphorylated mitochondrial proteins as potential therapeutic targets for ischemia/reperfusion (I/R) injury.
Methods: Sprague-Dawley rat hearts were subjected in normal perfusion, I/R, and IPC conditions through an ex vivo Langendorff system and were subsequently processed for phosphoproteomic analysis. To further investigate the cardioprotective role of CKMT2 overexpression and site-directed mutagenesis of putative CKMT2 phosphorylation sites (Y159A, Y255A, and Y368A) were done in human cardiomyocyte AC16 cells. CKMT2 enzymatic activity, mitochondrial function, and associated protein expression changes were subsequently evaluated.
Results: Phosphoproteomic analysis demonstrated that mitochondrial creatine kinase (CKMT2) is dephosphorylated during ischemia and I/R, whereas its phosphorylated state is preserved during IPC. Overexpression of CKMT2 led to increased cell viability and mitochondrial ATP, preserved mitochondrial membrane potential, and reduced reactive oxygen species (ROS) production thereby promoting cardioprotection against hypoxia/reoxygenation (H/R). On the other hand, phosphomutations, particularly in Y368, abolished cardioprotection significantly decreasing cell viability and elevating ROS levels during HR. Additionally, CKMT overexpression improved mitochondrial function through mediating proliferator-activated receptor γ coactivator-1α/estrogen-related receptor-α pathway, an effect largely attenuated by Y368A mutation.
Conclusions: These findings suggest that modulation of CKMT2 expression levels and phosphorylation at Y368 represents a potential therapeutic approach for ICM.
Keyword: Hypoxia, Reoxygenation, Phosphorylation, CKMT2, Mitochondria

OP1-4 5. Others

Impact of severe hypoglycemia on the heart in type 2 diabetes mellitus

이예지*, 김규호, 김지원, 이지영, 고승현

가톨릭대학교 내분비내과

Objectives: Severe hypoglycemia (SH) is increasingly recognized as an important contributor to adverse cardiovascular outcomes in type 2 diabetes mellitus (T2DM). However, the direct impact of SH on cardiac function in T2DM remains insufficiently characterized. This study aimed to evaluate the effects of SH on cardiac function in T2DM mouse models.
Methods: T2DM was induced in 38-week-old C57BL/6J mice by intraperitoneal injection of streptozotocin (50 mg/kg for 3 consecutive days), followed by a 45% high-fat diet for 12 weeks. At 50 weeks of age, SH was induced by daily insulin injections (250 IU/kg) for 5 consecutive days. Cardiac function was assessed by echocardiography before and after SH induction.
Results: Fasting blood glucose levels were significantly elevated in the DM group (434.2±26.6 mg/dL) compared with the non-DM group (110.2±6.8 mg/dL) ($p<0.05$). Following SH induction, the DM-SH group demonstrated a significant reduction in ejection fraction (from 70.1±2.1% to 63.5±1.2%, $p<0.05$) and fractional shortening (from 34.6±1.6% to 29.4±0.8%, $p<0.05$). There were no significant differences in echocardiographic parameters between the DM and non-DM groups.
Conclusions: Recurrent SH markedly impaired cardiac dysfunction in in T2DM mouse models. These findings suggest the importance of preventing hypoglycemia to ensure cardiovascular safety in diabetes management.
Keyword: Severe hypoglycemia, Type 2 diabetes mellitus, Cardiac dysfunction, Echocardiography, Cardiovascular risk

OP1-5 *5. Others*

Cardioprotective effects of Echinochrome A on cardiac dysfunction and remodeling in db/db mice

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

[1]Department of Physiology, Cardiovascular and Metabolic Disease Core Research Support Center, Inje University, Republic of Korea, [2]Department of Physiology and Human Biology, VNU University of Science, Vietnam National University-Hanoi, Vietnam

Objectives: To evaluate whether Echinochrome A (EchA) protects against diabetic cardiomyopathy in db/db mice.
Methods: Seven-week-old diabetic and obese db/db mice were treated with EchA (3 mg/kg/day, intraperitoneal injection) for 12 weeks. Age-matched db/db controls and wild-type (WT) mice received an equal volume of sterile 0.9% saline. Cardiac function was assessed by echocardiography, and cardiac remodeling (hypertrophy, fibrosis) and molecular indices of oxidative stress, mitochondrial dysfunction, and apoptosis were analyzed in heart tissue.
Results: EchA treatment significantly improved both systolic and diastolic function in db/db mice compared with saline-treated db/+ controls. EchA also attenuated cardiac hypertrophy and fibrosis. Diabetic hearts exhibited increased oxidative stress, mitochondrial dysfunction, and apoptosis, whereas these alterations were markedly reversed by EchA.
Conclusions: EchA preserves cardiac function and limits pathological remodeling in db/db mice, likely through mitigation of mitochondrial dysfunction, oxidative stress, and apoptosis, supporting its potential as a therapeutic candidate for diabetic cardiomyopathy.
Keyword: Echinochrome A, Diabetic cardiomyopathy

OP2-1 *4. Clinical Vascular Disease & Nutrition*

Composite indices of social determinants of health and coronary artery calcium in Korean

Jooeun Jeon[1,3*], Hyoeun An[2], Hyeok-Hee Lee[2,4], Hansol Choi[2,3], Kyoung Hwa Ha[2,3], Jee-Seon Shim[2,3], Hae Won Chung[5], Ji-Hye Kim[5], Yoosik Youm[6], Sungha Park[7], Dae Jung Kim[8], Hokyou Lee[2,3], Hyeon Chang Kim[2,3]

[1]Institute of Medical and Convergence, Ewha Womans University Mokdong Hospital, Ewha Womans University College of Medicine, Seoul, [2]Department of Preventive Medicine, Yonsei University College of Medicine, [3]Yonsei Institute of Digital Health, Yonsei University, Republic of Korea, [4]Richard A. and Susan F. Smith Center for Outcomes Research in Cardiology, Beth Israel Deaconess Medical Center, Harvard Medical School, USA, [5]Department of Health Promotion, Severance Health Check-Up, Yonsei University Health System, [6]Department of Sociology, Yonsei University College of Social Sciences, [7]Division of Cardiology, Severance Cardiovascular Hospital and Cardiovascular Research Institute, Yonsei University College of Medicine, [8]Department of Endocrinology and Metabolism, Ajou University School of Medicine, Republic of Korea

Objectives: Social determinants of health (SDoH) are critical in identifying populations at risk for atherosclerotic cardiovascular disease (ASCVD). Despite their importance, standardized measurement tools tailored to specific regions remain scarce. This study aimed to develop a composite SDoH framework for the Korean population and evaluate its association with coronary artery calcification (CAC), a key marker of subclinical atherosclerosis.
Methods: We analyzed 2,045 participants (aged 30-79) from the Cardiovascular and Metabolic Diseases Etiology Research Center (CMERC) cohort (2013-2018). CAC was quantified using the Agatston method via computed tomography, with presence defined as a score >0. A composite SDoH index was constructed from eight items across three domains: socioeconomic status (education, occupation, income), social support (marital status, network size, intimacy, frequency), and the area deprivation index (ADI). Adverse SDoH composite scores ranged from 0 to above 4. Multivariable logistic regression adjusted for traditional risk factors examined the associations.
Results: During mean 8.4 years of follow-up, CAC was detected in 41% of participants. Correlation between SDoH items was low (all $r<0.5$), indicating independent contributions. After multivariable adjustment, the highest adverse SDoH group (score above 4) was significantly associated with CAC presence (HR 1.62, 95% CI 1.00-2.67; p-trend=0.01) compared to the score 0 group. In sex-stratified analysis, this association remained significant among males (p-trend=0.01) but was not observed in females (p-trend=0.13).
Conclusions: The composite SDoH index is significantly associated with subclinical atherosclerosis in the Korean general population. These findings highlight the importance of integrating multidimensional social determinants when evaluating subclinical atherosclerosis in Korean.
Keyword: Risk factor, Cohort study, Coronary artery calcium, Social determinants of health

OP2-2 *4. Clinical Vascular Disease & Nutrition*

Regional variation in 30-day case fatality after myocardial infarction in Korea, 2003-2023

Seojeong Shin[1*], Sojeong Shin[2], Hyeon Chang Kim[1,2], Hokyou Lee[1,2]

[1]Digital Health, Yonsei Institute for Digital Health, Yonsei University, Seoul,
[2]Department of Preventive Medicine, Yonsei University College of Medicine, Seoul, Republic of Korea

Objectives: To examine long-term trends and regional disparities in 30-day case fatality rates (CFRs) after myocardial infarction (MI) in Korea from 2003 to 2023, and to assess whether geographic inequalities in short-term mortality have narrowed over time.
Methods: Using nationwide claims data from the National Health Insurance Service linked with mortality data from Statistics Korea, we identified MI hospitalizations between 2003 and 2023. Annual 30-day CFRs were calculated for each region. Age-standardized CFRs (ASFRs) were estimated using the 2023 MI case age distribution as the reference. Regional disparities were quantified annually using the absolute gap (maximum-minimum) and standard deviation across regions. To reduce annual fluctuations, three-year grouped analyses were performed. Trends were additionally compared between metropolitan (Seoul, Gyeonggi, Incheon) and non-metropolitan regions.
Results: Nationally, the crude 30-day CFR showed little overall change over the study period, whereas the age-standardized CFR declined substantially from 11.30% in 2003 to 8.93% in 2023 (Figure 1). Considerable regional variation persisted throughout the study period (Figure 2). The absolute inter-regional gap in ASFR decreased from 7.0 percentage points in 2003 to 2.9 percentage points in 2023, although intermittent widening occurred in the mid-2010s and early 2020s. Three-year grouped analyses showed that the absolute regional disparity declined from 9.1 percentage points in 2003-2005 to 5.9 percentage points in 2012-2014, followed by renewed widening to 7.8 percentage points in 2021-2023 (Figure 3). Regional disparities narrowed from the early 2000s to the mid-2010s but subsequently fluctuated rather than continuing to decline. Both metropolitan and non-metropolitan regions showed decreasing ASFRs over time, but metropolitan regions generally had higher rates and the gap persisted through 2023.
Conclusions: Although 30-day mortality after MI has declined nationally over the past two decades, regional disparities persist. Despite some convergence, geographic inequalities in short-term mortality were not fully eliminated, highlighting the need for sustained regional health system strengthening.
Keyword: Myocardial infarction, Case fatality

OP2-3 *4. Clinical Vascular Disease & Nutrition*

Lean metabolic dysfunction-associated steatotic liver disease as an independent predictor of subclinical coronary artery calcification

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

[1]울산의대 서울아산병원 내분비내과, [2]울산의대 서울아산병원 건강의학과

Objectives: While metabolic dysfunction-associated steatotic liver disease (MASLD) is traditionally associated with obesity, the "lean MASLD" phenotype has recently emerged as a critical clinical entity. However, its cardiovascular risk profile remains under-recognized. We aimed to evaluate whether lean MASLD is independently associated with the burden of subclinical coronary atherosclerosis in an asymptomatic population.
Methods: This cross-sectional study analyzed 4,610 asymptomatic adults undergoing routine health screenings at Asan Medical Center, Seoul, Korea. Participants were stratified into four distinct phenotypes based on body mass index (BMI; lean < 23 kg/m^2) and the presence of MASLD (diagnosed via abdominal ultrasonography): (1) lean without MASLD, (2) lean MASLD, (3) non-lean without MASLD, and (4) non-lean MASLD. Subclinical coronary atherosclerosis was quantified using the coronary artery calcium score (CACS) derived from multi-detector computed tomography.
Results: The study population comprised the lean without MASLD (n=1,376, 29.8%), lean MASLD (n=199, 4.3%), non-lean without MASLD (n=1,533, 33.3%), and non-lean MASLD (n=1,502, 32.6%) groups. Notably, the lean MASLD group exhibited the highest mean CACS among all groups, surpassing even the non-lean MASLD group. Multivariable logistic regression analysis, adjusted for age, sex, smoking, physical activity, uric acid, and renal function, revealed that lean MASLD was a robust and independent predictor of coronary artery calcification (CAC > 0). Compared to the lean without MASLD reference, the lean MASLD group demonstrated a substantially higher risk of CAC (Odds Ratio [OR] 1.500; 95% Confidence Interval [CI] 1.074-2.096; p=0.017), while the ORs for non-lean without MASLD and non-lean MASLD were 1.203 (95% CI 1.009-1.435) and 1.380 (95% CI 1.155-1.649), respectively.
Conclusions: Our findings underscore that non-obese individuals with MASLD face a significantly higher burden of subclinical coronary atherosclerosis than their lean counterparts. These results highlight the need for proactive cardiovascular screening and aggressive risk factor management in the lean MASLD population, regardless of their BMI.
Keyword: Metabolic dysfunction-associated steatotic liver disease, Coronary artery calcium score, Subclinical atherosclerosis

OP2-4 *4. Clinical Vascular Disease & Nutrition*

Soluble LDL receptor dynamics in the cholesterol and pharmacogenetics study: simvastatin effects and links to lipid metabolism

Shieon Kim[1*], Ronald M. Krauss[2], Min-Jeong Shin[1]

[1]고려대학교 대학원, [2]University of California, San Francisco

Objectives: sLDLR, a soluble form of low-density lipoprotein receptor (LDLR) produced by proteolytic cleavage, has been linked to triglycerides (TG) and LDL cholesterol (LDL-C). However, most studies were cross-sectional, and longitudinal associations between changes in sLDLR and changes in lipids and lipoproteins, particularly across lipoprotein particle size subclasses, remain unclear. Therefore, we measured plasma sLDLR, examined whether changes in sLDLR were associated with lipid profiles, and evaluated the impact of statins on plasma sLDLR levels.
Methods: This study (Cholesterol and Pharmacogenetics (CAP) Study, NCT00451828) included 184 white, non-smoking adults (92 male, 92 female) aged ≥30 years who received simvastatin (40 mg/day) for 6 weeks. Plasma was collected at baseline and 6 weeks. sLDLR was measured by ELISA, lipids by standard assays, and lipoprotein subfractions by ion mobility. LDL-C was calculated using the Friedewald equation. Changes were defined as the difference between average pretreatment and on-treatment values, and associations with lipid and lipoprotein particle measures were examined using regression models.
Results: Simvastatin significantly reduced plasma sLDLR levels (p=0.003). At baseline, sLDLR positively correlated with TG (Beta 0.75; 95% CI [0.65, 0.84]), LDL-C (0.35 [0.22, 0.48]), large very-low-density lipoprotein (VLDL) (0.57 [0.45, 0.70]), small LDL (0.54 [0.41, 0.68]), and very small LDL (0.59 [0.47, 0.71]), and negatively correlated with high-density lipoprotein cholesterol (HDL-C) (-0.41 [-0.54, -0.28]). Changes in sLDLR were positively associated with TG (0.45 [0.34, 0.60]) and large VLDL (0.27 [0.12, 0.42]) and negatively associated with HDL-C (-0.21 [-0.35, -0.07]).
Conclusions: This study shows that changes in plasma sLDLR were strongly associated with key atherogenic lipoprotein phenotype (ALP) features, including TG, large VLDL, and small LDL, suggesting that sLDLR may be a component of ALP. Plasma sLDLR also decreased significantly with statin therapy, which is associated with the improvements in this ALP. Therefore, targeting sLDLR shedding to lower plasma levels may improve atherogenic dyslipidemia.
Keyword: Soluble LDL receptor (sLDLR), Atherogenic lipoprotein phenotype (ALP), Statin therapy

OP2-5 *4. Clinical Vascular Disease & Nutrition*

Smoking cessation or reduction after myocardial infarction and subsequent risk of cardiovascular events

Sojung Shin[1,2,3*], Jaewon Khil[1,2], Dae Young Cheon[4], Jaeyong Lee[1], Kyoung Hwa Ha[1,2], Hyeok-Hee Lee[5], Hyeon Chang Kim[1,2], Hokyou Lee[1,2]

[1]Department of Preventive Medicine, Yonsei University College of Medicine, [2]Yonsei Institute for Digital Health, Yonsei University, [3]Department of Public Health, Yonsei University Graduate School, [4]Division of Cardiology, Department of Internal Medicine, Hallym University Dongtan Sacred Heart Hospital, Republic of Korea, [5]Richard A. and Susan F. Smith Center for Outcomes Research in Cardiology, Beth Israel Deaconess Medical Center, Harvard Medical School, USA

Objectives: While smoking is a major risk factor for cardiovascular disease, data are scarce regarding outcomes of smoking cessation and reduction after myocardial infarction (MI). We hypothesized that smoking cessation but not reduction was associated with a lower risk of major adverse cardiovascular events (MACE) after a first MI.
Methods: Using the Korean National Health Insurance Service database, we identified adults aged ≥19 years with a first MI between 2011 and 2020 who underwent health examinations before MI and during the 2-year landmark period after MI. Smoking status and amount (cigarettes/day) were self-reported. Based on changes between pre- and post-MI examinations, participants were categorized as sustained non-smokers, initiators/relapsers, quitters, or continuing smokers. Among pre-MI smokers, participants were further categorized by post-MI smoking amount as quitters, reducers (≥50% decrease in cigarettes/day), or non-reducers. The primary outcome was first MACE (composite of cardiovascular death, stroke, or recurrent MI). Secondary outcomes included all-cause death, first occurrence of each component of MACE, and total MACE (i.e., recurrent event analysis).
Results: Among 45,316 adults (mean age 61.5±11.0 years; 16.2% women), 56.4% of pre-MI smokers quit and 10.5% reduced smoking by ≥50%, whereas 2.0% of pre-MI non-smokers initiated or relapsed. During a median follow-up of 5.8 years (IQR, 3.4-7.8 years), 3,842 primary events occurred. Compared with sustained non-smokers, multivariable-adjusted HRs (95% CIs) for MACE were 1.56 (1.27-1.91) for initiators/relapsers, 1.01 (0.93-1.11) for quitters, and 1.53 (1.40-1.67) for continuing smokers. Among pre-MI smokers, quitting was associated with a lower MACE risk (HR, 0.65; 95% CI, 0.58-0.73), whereas reducing was not (HR, 0.92; 95% CI, 0.78-1.09) compared with non-reduction; cumulative incidence curves were consistent with these findings. Results were consistent across secondary and recurrent outcomes.
Conclusions: Smoking cessation after MI was associated with a substantially lower risk of subsequent cardiovascular events, whereas smoking reduction without cessation did not confer a lower risk.
Keyword: Myocardial infarction, Smoking behavior change, Secondary prevention, Recurrent cardiovascular events, Mortality

OP3-1 *1. Basic Science of Lipids & Lipoproteins*

Adiponectin regulates brain endothelial cell function under hyperammonia condition

송동준*, 정설원, 조단비, 송주현
전남대학교 의과대학 해부학교실

Objectives: Hyperammonemia is a key driver of neuroinflammation, oxidative stress, and blood-brain barrier (BBB) dysfunction during liver failure. Brain endothelial cells are particularly vulnerable to ammonia-induced mitochondrial injury and tight junction disruption. Adiponectin, an adipokine with anti-inflammatory and cytoprotective properties, may regulate endothelial resilience, but its role under hyperammonemic conditions remains unclear.
Methods: Here, we investigated adiponectin effects in a bile duct ligation (BDL) mouse model and in ammonia-challenged brain endothelial cells (bEnd.3). Male C57BL/6J mice received adiponectin (5 μg/kg/day, intraperitoneal) for 6 days starting 5 days after BDL; cortical tissues were collected on day 11.
Results: In the cortex, adiponectin tended to increase PSD95 and claudin-5 protein levels and attenuated stress-related transcriptional changes, including reduced Cyp2e1 and Caspase-9 expression trends. In vitro, adiponectin pretreatment suppressed ammonia-induced inflammatory responses, preserved tight junction proteins, restored mitochondrial membrane potential, and reduced reactive oxygen species. Mechanistically, adiponectin increased AdipoR1/AdipoR2 protein expression and counteracted ammonia-driven loss of occludin in bEnd.3 cells, while most ROS/antioxidant gene transcripts showed minimal changes.
Conclusions: Together, these data indicate that adiponectin enhances endothelial stress resistance and supports BBB-associated molecular integrity under hyperammonemic stress, highlighting adiponectin signaling as a candidate target for protecting the neurovascular unit in hepatic encephalopathy-relevant conditions.
Keyword: Blood brain barrier, Adiponec tin, Hyperammonia, Brain endothelial cells

OP3-2 *1. Basic Science of Lipids & Lipoproteins*

TRPC1-mediated calcium dysregulation in cardiac dysfunction and metabolic impairment

Maria Victoria Faith Garcia[1*], Jubert Marquez[1,3], Nammi Park[1], Jessa Flores[1,2], Hyoung Kyu Kim[1,2], Jin Han[1,2]

[1]순환기/심장내과, Inje University, Cardiovascular and Metabolic Disease Center, Smart Marine Therapeutics Center, Department of Physiology, [2]순환기/심장내과, Inje University, College of Medicine, Department of Physiology, Republic of Korea, [3]De La Salle University, College of Science, Department of Biology, Manila, Philippines

Objectives: Transient receptor potential canonical 1 (TRPC1) is a Ca^{2+}-permeable ion channel widely expressed in cardiac tissue and implicated in calcium homeostasis. Given the importance of regulated Ca^{2+} influx in cardiomyocyte function and metabolic signaling, this study aimed to elucidate the role of TRPC1 in modulating cardiac calcium entry under stress conditions relevant to ischemia-reperfusion injury and its potential contribution to cardiometabolic disease mechanisms.
Methods: Experiments were conducted using cellular and murine models to evaluate TRPC1 expression and functional activity. HEK293 cells with manipulated TRPC1 expression were employed to quantify TRPC1-mediated currents and calcium transients. Ex vivo ischemia-reperfusion models in mice were used to assess cardiac TRPC1 expression changes under pathological conditions. Functional assessments of Ca^{2+} influx were performed using electrophysiological recordings, and protein interaction studies were used to investigate mechanisms regulating TRPC1 levels and activity.
Results: Upregulation of TRPC1 expression in engineered cellular systems led to increased current density and enhanced calcium transients, indicating robust TRPC1-dependent Ca^{2+} entry pathways. In murine hearts subjected to ischemia-reperfusion stress, TRPC1 expression was significantly elevated compared with control conditions, suggesting a dynamic regulation of TRPC1 under cardiac stress. Mechanistic studies revealed specific domain interactions that influence TRPC1 channel stability and functional expression in cardiomyocytes. These findings collectively highlight TRPC1 as a significant contributor to cardiac Ca^{2+} influx, with implications for cellular responses to metabolic and ischemic challenge.
Conclusions: TRPC1 plays a central role in regulating cardiac calcium entry under both physiological and pathological conditions. Enhanced TRPC1 expression and activity correlate with increased Ca^{2+} influx, suggesting that TRPC1 contributes to cardiomyocyte calcium handling during stress. These results underscore the importance of TRPC1-mediated calcium dynamics in cardiac function and support the potential of TRPC1-focused research for understanding and potentially targeting cardiometabolic and ischemia-related pathophysiology. Further investigation into TRPC1 regulation may yield new insights into therapeutic strategies for cardiometabolic disease.
Keyword: Calcium, Ischemia/Reperfusion

OP3-3 2. Basic Science of Atherosclerosis

GLP-1 receptor agonist suppresses PDGF-induced VSMC remodeling

윤동현[1*], 유하미[1], Lira Kim[2], 허경선[1]
[1]충남대학교 약학과, [2]NeuraGene Inc, Republic of Korea

Objectives: The phenotypic transition of vascular smooth muscle cells (VSMCs) toward a synthetic state is characterized by enhanced proliferation and migration and is associated with vascular diseases such as restenosis and atherosclerosis. Glucagon-like peptide-1 (GLP-1) receptor agonists such as liraglutide (Lira) not only improve glycemic control in type 2 diabetes but also demonstrate significant reductions in cardiovascular disease risk. However, their direct effects on VSMC proliferation remain unclear. This study aimed to investigate the mitochondria-reactive oxygen species (ROS)-mediated apoptotic signaling mechanisms by which liraglutide regulates VSMC proliferation induced by platelet-derived growth factor-BB (PDGF-BB).
Methods: Rat aortic smooth muscle cells were pretreated with 100 nM Lira for 1 hour and then stimulated with 20 ng/ml PDGF-BB. Wound-healing assay and MTT assay were performed to validate cell proliferation and migration. The underlying molecular mechanisms were explored through western blotting, immunofluorescence staining, DCF-DA, and mitochondria-ROS analysis.
Results: MTT assay and wound-healing assay showed that PDGF-BB-induced cell proliferation and migration were significantly attenuated by Lira treatment. The inhibitory effect of Lira on cell proliferation was confirmed by PCNA expression. Interestingly, Lira significantly reduced PDGF-BB-induced total intracellular ROS but not mitochondria ROS, as indicated by DCF-DA assay and Mito-SOX staining, respectively. Notably, Lira increased the expression of cleaved-caspase 3, suggesting that Lira may involves in cell apoptosis. Consistently, treatment with either caspase-3 inhibitor (Z-VAD-FMK) or mitochondrial ROS inhibitor (Mito-Tempo) abolished inhibitory effect of Lira on PDGF-BB induced cell migration.
Conclusions: These results suggest that Lira inhibits VSMC proliferation stimulated by PDGF-BB by activating cleaved-caspase 3 through the mitochondrial stress-induced pathway.
Keyword: Liraglutide, Mitochondria ROS

OP3-4 2. Basic Science of Atherosclerosis

Loss of thyroid hormone receptor beta in myeloid cells promotes atherosclerosis

김채영*, 박규성, 박신희, 박상은, 고유진, 장수빈, 최재훈
한양대학교 기초과학

Objectives: Macrophage-derived foam cell formation is a key feature of atherosclerotic lesion development. Identifying transcriptional regulators of foamy macrophages may provide insight into disease progression. This study aimed to identify key transcription factors associated with foamy macrophages and to define the role of thyroid hormone receptor β (TRβ) in myeloid cells during atherosclerosis.
Methods: Foamy and non-foamy macrophages were isolated from atherosclerotic lesions and analyzed by ATAC-seq, integrated with bulk RNA-seq to identify candidate transcription factors. For in vivo studies, bone marrow from myeloid-specific Thrb-deficient mice (Lyz2-Cre; $Thrb^{fl/fl}$) was transplanted into $Ldlr^{-/-}$ recipients. After recovery, mice were fed a high-cholesterol diet for 12 weeks. Lesions were assessed by Oil Red O staining of whole aortas and aortic sinus sections, and macrophage accumulation was evaluated by CD68 immunohistochemistry. In vitro, BMDMs were treated with oxLDL and foam cell-related gene expression was analyzed by qPCR.
Results: Integrated analyses identified TRβ as a key transcription factor associated with the foamy macrophage phenotype. After 12 weeks of high-cholesterol diet, the proportion of Oil Red O-positive lesions in the whole aorta was significantly increased in the aortic arch of ThrbΔmyeloid → $Ldlr^{-/-}$ mice. In addition, $CD68^{+}$ macrophage accumulation was significantly elevated in the aortic sinus. In oxLDL-treated BMDMs, ABCA1 expression was reduced in the TRβ-deficient group. As ABCA1 mediates cholesterol efflux, its downregulation suggests impaired cholesterol handling that may contribute to foam cell formation and macrophage retention within lesions.
Conclusions: Myeloid TRβ appears to play a protective role in atherosclerosis. Its deficiency was associated with increased lesion burden and macrophage accumulation. Reduced ABCA1 expression in TRβ-deficient macrophages suggests a potential impairment in cholesterol handling capacity. These findings indicate that TRβ in macrophage may contribute to lipid regulation and lesion progression. Further studies are needed to clarify the mechanisms linking TRβ signaling to cholesterol metabolism and foam cell formation.
Keyword: Atherosclerosis, TRβ

OP3-5 *2. Basic Science of Atherosclerosis*

Implication of dapagliflozin on endothelial dysfunction by targeting ERK1/2/p90RSK signaling pathway

Phuc Nguyen Tran-Duc*, Kyung-Sun Heo

College of Pharmacy, Chungnam National University, Daejeon, Republic of Korea

Objectives: Dapagliflozin is the potent sodium-glucose cotransporter-2 inhibitor widely used for antidiabetic treatment. Moreover, this drug has been shown to improve cardiovascular outcomes independent of blood glucose levels. However, its underlying molecular mechanisms remain elusive. This study aims to investigate the role of dapagliflozin in endothelial dysfunction, a pathological state that increases the risk of cardiovascular disease.
Methods: EA.hy926 cells were pretreated with dapagliflozin followed by stimulant treatment. Western blotting was performed to evaluate modulation of signaling pathway. In silico docking analysis and cellular thermal shift assay (CETSA) were utilized to confirm direct targets of dapagliflozin.
Results: Upon ERK1/2/p90RSK signaling activation, dapagliflozin was found to inhibit this pathway. This inhibitory effect of dapagliflozin can be explained by its favorable binding affinity toward p90RSK, as indicated by low binding Gibbs free energy values and key residues involved in the interactions. Consistently, CETSA revealed that, upon dapagliflozin treatment, p90RSK exhibited increased thermal stability, indicating stabilizing effects resulting from ligand-protein interaction.
Conclusions: Dapagliflozin inhibits the ERK1/2/p90RSK signaling pathway through direct interaction with p90RSK. This finding suggests that dapagliflozin may serve as an endothelial protecting agent.
Keyword: Dapagliflozin, Endothelial dysfunction

OP3-6 *5. Others*

Role of ginsenoside Rg2 on angiotensin II-induced phenotypic conversion of vascular smooth muscle cells

Nhi Thi Thao Le*, Minji Kim, Kyung-Sun Heo

College of Pharmacy, Chungnam National University, Daejeon, Republic of Korea

Objectives: The abnormal phenotypic transformation of vascular smooth muscle cells (VSMCs), characterized by increased proliferation and migration, is a key feature of several cardiovascular diseases (CVDs) such as aortic aneurysms and atherosclerosis. We investigate the effect of ginsenoside Rg2 on Ang II-induced phenotypic conversion in VSMCs.
Methods: Rat aortic smooth muscle cells were pretreated with ginsenoside Rg2 or specific inhibitors, followed by Ang II stimulation. Cell proliferation and migration were assessed via Ki-67 staining, wound healing assays, and cell cycle analysis. Western blotting was conducted to elucidate the underlying signaling mechanisms.
Results: Rg2 pretreatment markedly attenuated Ang II-induced proliferation, migration, and phenotypic switching in VSMCs by suppressing the upregulation of PCNA, Cyclin D1, and CDK6 via ERK1/2 and Akt/mTOR signaling pathways. Furthermore, Rg2 restored the expression of the contractile marker α-smooth muscle actin while reducing the levels of synthetic phenotype-associated proteins, including osteopontin and vimentin. Molecular docking analysis revealed that Rg2's protective effect is based on its interaction with the angiotensin II type 1 receptor (AT1R), blocking Ang II-induced activation and downstream signaling pathways.
Conclusions: This study highlights the therapeutic potential of ginsenoside Rg2 in preventing vascular remodeling and the progression of cardiovascular disease.
Keyword: Angiotensin II, Ginsenoside Rg2, Vascular smooth muscle cells, Phenotypic switching

SoLA 2026

한국지질·동맥경화학회 춘계학술대회

2026 Spring Congress on
Lipid and Atherosclerosis of KSoLA

Mini-Oral Presentations

Mini-Oral Presentation 1

☞ **일시: 2026년 4월 3일(금) 17:00-17:50**
☞ **장소: 포이어(로비) 내 발표구역 A-D**

- **Mini-Oral Presentation 1-1 (발표구역 A)**
  - 좌장 : 박용현(부산의대 순환기내과)
  - 발표 : MOP1-1-1 ~ MOP1-1-4
- **Mini-Oral Presentation 1-2 (발표구역 B)**
  - 좌장 : 정재훈(동국의대 심장내과)
  - 발표 : MOP1-2-1 ~ MOP1-2-4
- **Mini-Oral Presentation 1-3 (발표구역 C)**
  - 좌장 : 김정민(서울의대 신경과)
  - 발표 : MOP1-3-1 ~ MOP1-3-4
- **Mini-Oral Presentation 1-4 (발표구역 D)**
  - 좌장 : 김원진(차의대 내분비내과)
  - 발표 : MOP1-4-1 ~ MOP1-4-4

Mini-Oral Presentation 2

☞ **일시: 2026년 4월 4일(토) 10:50-12:00**
☞ **장소: 포이어(로비) 내 발표구역 A-D**

- **Mini-Oral Presentation 2-1 (발표구역 A)**
  - 좌장 : 양여리(가톨릭의대 내분비내과)
  - 발표 : MOP2-1-1 ~ MOP2-1-5
- **Mini-Oral Presentation 2-2 (발표구역 B)**
  - 좌장 : 우종신(경희의대 심장내과)
  - 발표 : MOP2-2-1 ~ MOP2-2-6
- **Mini-Oral Presentation 2-3 (발표구역 C)**
  - 좌장 : 정미향(가톨릭의대 순환기내과)
  - 발표 : MOP2-3-1 ~ MOP2-3-6
- **Mini-Oral Presentation 2-4 (발표구역 D)**
  - 좌장 : 전재한(경북의대 내분비대사내과)
  - 발표 : MOP2-4-1 ~ MOP2-4-5

SoLA 2026

한국지질·동맥경화학회 춘계학술대회

2026 Spring Congress on
Lipid and Atherosclerosis of KSoLA

MOP1-1-1 *4. Clinical Vascular Disease & Nutrition*

Cardioprotective effects of the neopetroside A in murine ischemia/reperfusion injury GSK-3β inhibition

Prycelline Abedejos*, Hyoung Kyu Kim, Jubert Marquez, Jin Han

Physiology, Inje University, Republic of Korea

Objectives: This study investigated the cardioprotective potential of neopetroside A (NPS A), a natural pyridine nucleoside, against myocardial ischemia/reperfusion (I/R) injury and myocardial infarction (MI). Researchers specifically explored how NPS A regulates mitochondrial metabolism and heart function while identifying its direct molecular targets.
Methods: The researchers employed a multi-faceted approach using in vitro rat cardiomyoblast (H9c2) cells, ex vivo Langendorff-perfused rat hearts, and in vivo C57BL/6 mouse models. Methods included kinase screening assays, molecular docking simulations, and surface plasmon resonance (SPR) to identify binding targets. They also utilized siRNA knockdown of Nrf2 and Nqo1 to delineate the underlying signaling pathways.
Results: Results identified NPS A as a direct inhibitor of GSK-3β, binding to its active site in a phosphorylation-independent manner. In vitro, NPS A enhanced glycolysis, oxidative phosphorylation, and ATP production. Ex vivo experiments demonstrated that NPS A preserved hemodynamic status and significantly reduced infarct size and reactive oxygen species generation. In vivo, NPS A treatment improved survival rates (80% vs. 53% in controls) and reduced cardiac fibrosis and infarct areas in mice following MI. Mechanistically, this protection was mediated by activating the Nrf2/Nqo1 axis, which increased the NAD+/NADH ratio and preserved mitochondrial function.
Conclusions: The study concludes that NPS A is an effective cardioprotective agent that mitigates I/R injury and fibrosis by targeting the GSK-3β/Nrf2/Nqo1 signaling pathway. This unique phosphorylation-independent inhibition of GSK-3β provides a novel therapeutic foundation for treating cardiovascular diseases.
Keyword: GSK-3b inhibition, Ischemia/reperfusion injury, Mitochondria, Neopetroside A

MOP1-1-2 *5. Others*

PTP4A1 alleviates angiotensin II-induced aortic aneurysmal lesions by regulating immature mural neovascularisation

Min Ji Cho[2*], Jong-Gil Park[1,3]

[1]Biotherapeutics Translational Research Centre, Korea Research Institute of Bioscience and Biotechnology (KRIBB), Daejeon, [2]Bio-Design & Editing Research Center, Korea Research Institute of Bioscience & Biotechnology (KRIBB), Daejeon, [3]Department of Bioscience, KRIBB School of Bioscience, Korea University of Science and Technology (UST), Daejeon, Republic of Korea

Objectives: Abdominal aortic aneurysm (AAA) progression is driven by inflammation, extracellular matrix degradation, and pathological mural neovascularisation, particularly immature and leaky microvessels that weaken the vessel wall. Protein tyrosine phosphatase type 4A1 (PTP4A1) regulates endothelial inflammation, but its role in AAA remains unclear. We investigated whether PTP4A1 protects against angiotensin II (Ang II)-induced AAA and explored its mechanistic link to mural neovessel maturation.
Methods: Public single-cell RNA-seq data (GSE164678) were analysed to evaluate Ptp4a1 expression in normal and aneurysmal aortas. Human endothelial cells (huECs) overexpressing PTP4A1 underwent RNA sequencing and gene ontology analysis. Endothelial-targeted Tie2-Ptp4a1 transgenic mice were crossed with $ApoE^{-/-}$ mice and infused with Ang II for 4 weeks. Aneurysm incidence, rupture-related mortality, maximal aortic diameter, and lesion severity were assessed. Inflammatory markers, matrix metalloproteinases, and neovessel maturation markers were analysed by immunoblotting. Vascular leakage was evaluated using FITC-dextran extravasation. Plasma TGF-β levels were measured, and the functional role of TGF-β signalling was tested using the TGF-β receptor inhibitor LY364947.
Results: Single-cell analysis showed reduced Ptp4a1 expression in endothelial cells within AAA lesions and altered associations with vascular maturation genes. PTP4A1 overexpression in huECs enriched angiogenesis- and TGF-β-related gene programs. In vivo, PTP4A1 overexpression significantly reduced Ang II-induced aneurysm rupture-related mortality, aneurysm incidence, maximal aortic diameter, and lesion severity in $ApoE^{-/-}$ mice. These protective effects were accompanied by decreased inflammatory and proteolytic markers (ICAM-1, VCAM-1, MMP2, and MMP9), features of more mature neovessels (reduced VEGFR2 and increased PDGFRβ), and diminished vascular leakage and apoptosis. Plasma TGF-β levels were elevated in PTP4A1 transgenic mice, and inhibition of TGF-β signalling abrogated the protective effects of PTP4A1.
Conclusions: PTP4A1 protects against Ang II-induced AAA by suppressing inflammation and promoting mural neovessel maturation via a TGF-β-dependent mechanism, identifying the PTP4A1-TGF-β axis as a potential therapeutic target for AAA.
Keyword: AAA, Neovascularisation, Vascular maturation, Inflammation

MOP1-1-3 *5. Others*

Role of CHIP E3 ligase on Ang II-induced VSMC phenotypic switching via regulating ERK1/2-p90RSK axis

Thuy Le Lam Nguyen*, Diem Thi Ngoc Huynh, Yujin Jin, Kyung-Sun Heo

College of Pharmacy, Chungnam National University, Daejeon, Republic of Korea

Objectives: Mitochondria dysfunction contributes to the dysfunction of vascular smooth muscle cells (VSMCs), leading to vascular diseases. The carboxyl terminus of Hsc70 interacting protein (CHIP), a U-box-type chaperone-associated E3 ligase, plays an important role in regulating protein stability; however, its function in VSMCs remains unclear. This study aimed to investigate the role of CHIP in VSMC dysfunction, focusing on its regulation of the 90 kDa ribosomal S6 kinase (p90RSK) pathway, activated by angiotensin II (Ang II).
Methods: In vitro, VMSCs were isolated from the aortas of wild-type (WT) and CHIP heterozygous (CHIP+/-) mice. Western blotting, qRT-PCR analysis, MitoTracker staining, JC-1 staining, MitoSOX staining, and a luciferase assay were employed to elucidate the underlying molecular mechanisms.
Results: We found that Ang II induced ERK1/2/p90RSK/KLF4 mediated cell proliferation and migration, whereas CHIP deficiency suppressed this effect of Ang II. In addition, DRP1 positively regulated Ang II-induced KLF4 expression and promoter activity. Interestingly, CHIP silencing abolished mitochondrial ROS production, DRP1 (Ser616) phosphorylation and mitochondria fragmentation induced by Ang II. Notably, inhibition of ERK1/2/p90RSK signaling attenuated DRP1 phosphorylation, subsequently suppressing KLF4-mediated synthetic switching markers vimentin.
Conclusions: Mitochondria fission is involved in Ang II-induced ERK1/2/p90RSK/KLF4-mediated VSMC proliferation and migration, and CHIP modulates mitochondria fission through DRP1 regulation, suggesting that CHIP is a promising therapeutic target for vascular diseases.
Keyword: Mitochondrial fission, VSMC dysfunction, CHIP, p90RSK, ERK1/2

MOP1-1-4 *5. Others*

Downregulation of TRPA1 by CRBN decreases fibrotic markers in TGFβ1-induced cardiac fibroblast

Jessa Flores[1,2*], Nammi Park[2], Jubert Marquez[2], Maria Victoria Faith Garcia[2], Hyoung Kyu Kim[1,2], Jin Han[1,2]

[1]Department of Physiology, College of Medicine, Inje University, Busan,
[2]Cardiovascular and Metabolic Core Research Support Center, College of Medicine, Inje University, Busan, Republic of Korea

Objectives: Cereblon (CRBN) is the substrate recognition component of E3 ubiquitin ligase and is responsible for degradation of various protein targets. Meanwhile, transient receptor potential ankyrin 1 (TRPA1) is a non-selective cation channel which plays a role in nociception. In this study, we aimed to evaluate if CRBN interacts with TRPA1 to facilitate its degradation; and to determine the role of TRPA1-CRBN in the physiology of cardiac fibrosis in vitro.
Methods: Western blot was performed to check the TRPA1 protein levels in vivo and in vitro CRBN KO model and in vitro fibrotic model. Immunoprecipitation (IP) was done to confirm the direct binding of CRBN and TRPA1. RT-PCR analysis was used to measure gene expression and immunocytochemistry was used to visualize the protein expression in cells. rhTGFβ1 treatment was used to induce fibrosis while siRNA transfection was used to knockdown TRPA1. Adenovirus with CRBN-Flag is used to overexpress CRBN in MCF under fibrotic conditions.
Results: TRPA1 protein levels were found to be inversely correlated with CRBN expression. On the other hand, presence or absence of CRBN did not change TRPA1 gene expression, implying a post-translational regulation. In addition, TRPA1 and CRBN were found to be directly binding with each other through IP. CRBN also decreases the level of functional TRPA1 according to cell surface assay and Ca2+ influx measurement results. Meanwhile, TRPA1 levels were found to increase under fibrotic conditions and knockdown of TRPA1 decreases the levels of fibrotic markers. Lastly, the overexpression of CRBN in fibrotic model decreased the phosphorylation of SMAD2 and ERK1/2 proteins which led to decreased fibrotic markers.
Conclusions: The findings of this study showed that CRBN binds with TRPA1; modulating its degradation. This study also suggests that TRPA1 inhibition by CRBN potentially exerts a protective role against TGF-β1-induced cardiac fibrosis by downregulating the fibrotic ERK1/2 signaling pathway.
Keyword: CRBN, TRPA1, Fibrosis

MOP1-2-1 *1. Basic Science of Lipids & Lipoproteins*

Vasculoprotective effects of adiponectin under hyperammonic stress in human endothelial cells

정설원*, 송동준, 조단비, 송주현
전남대학교 의과대학 해부학교실

Objectives: Hepatic encephalopathy (HE) is a serious complication of liver failure in which systemic hyperammonemia serves as a key pathological signal. In addition to neurotoxicity, elevated ammonia can directly disrupt vascular endothelial homeostasis by increasing oxidative stress, triggering inflammatory responses, and impairing mitochondrial function, thereby contributing to systemic vascular dysfunction. Adiponectin is an adipokine with recognized anti-inflammatory and vasculoprotective actions, but its ability to protect endothelial cells under hyperammonic stress relevant to HE remains incompletely understood.
Methods: Here, we examined whether adiponectin pretreatment mitigates hyperammonia-induced injury in human umbilical vein endothelial cells (HUVECs), focusing on reactive oxygen species (ROS), inflammatory cytokines, apoptosis-related signaling, and mitochondrial integrity. HUVECs were pretreated with adiponectin and subsequently exposed to ammonia to model hyperammonic stress.
Results: Endothelial responses were assessed by measuring adiponectin receptor expression, pro-inflammatory cytokine transcripts (including IL-1β and TNF-α), ROS production and ROS-associated enzymes (e.g., CYP2E1 and CYP4A1), apoptosis-associated markers (e.g., caspase-9), and mitochondrial status. Mitochondrial function was evaluated using mitochondrial membrane potential assays (JC-1 staining) and mitochondrial maintenance/biogenesis-related gene expression (TFAM). Adiponectin pretreatment attenuated hyperammonia-driven endothelial stress. Compared with hyperammonia alone, adiponectin reduced intracellular ROS accumulation and downregulated ROS-related enzyme expression, indicating suppression of oxidative stress pathways. Adiponectin also decreased induction of inflammatory cytokines, supporting an anti-inflammatory effect under hyperammonic conditions. In parallel, adiponectin improved mitochondrial integrity, reflected by preservation of mitochondrial membrane potential and increased TFAM expression, and it reduced apoptosis-associated signaling, suggesting enhanced endothelial resilience.
Conclusions: These results indicate that adiponectin confers protective effects against hyperammonia-induced vascular endothelial dysfunction by limiting oxidative stress, dampening inflammatory activation, and alleviating mitochondrial impairment. Our findings support adiponectin as a potential systemic vasculoprotective strategy in hyperammonemia-associated conditions such as HE, and they provide a mechanistic rationale for further in vivo validation and translational exploration.
Keyword: Hyperammonia, Hepatic encephalopathy, Adiponectin, HUVEC cells

MOP1-2-2 *1. Basic Science of Lipids & Lipoproteins*

Anti-obesity effects of Panax ginseng-derived exosomes via AMPK-mediated inhibition of adipocyte differentiation and lipogenesis

Jong Hyun Oh[1*], Yoon A Lee[2], Do Hyeon Jin[2], Su Jeong Hwang[2], Sun Hye Lee[1,2], Jin Woo Kim[1,2]
[1]선문대학교 응용생물과학과, [2]선문대학교 식품공학영양학부

Objectives: Obesity involves excessive adipocyte differentiation, lipid accumulation, and disrupted energy metabolism. This study investigated the anti-obesity effects of Panax ginseng-derived exosomes (PGE) by evaluating their physicochemical characteristics and regulatory effects on adipogenesis, lipogenesis, and AMPK-mediated energy signaling in 3T3-L1 preadipocytes.
Methods: Exosomes were purified using tangential flow filtration and characterized by particle size and marker expression. Adipogenesis was assessed by Oil Red O staining and analysis of adipogenic and lipogenic markers (PPAR-γ, C/EBP-α, C/EBP-β, FABP4, SREBP-1c, ACC, FAS). AMPK expression and mitochondrial and cytoskeletal changes were evaluated using gene/protein analysis and immunofluorescence staining.
Results: Purified exosomes with a mean diameter of 159.5 nm and a concentration of 3.9×10^{12} particles/mL showed enriched expression of the exosomal markers CD9, CD63, and TET-8, confirming successful vesicle isolation and enrichment. Functionally, PGE markedly suppressed lipid accumulation in differentiated 3T3-L1 cells by 72.1%, accompanied by coordinated downregulation of adipogenic transcription factors, including PPAR-γ, C/EBP-α, C/EBP-β, and FABP4, as well as lipogenic regulators such as SREBP-1c, ACC, and FAS. These reductions ranged from 23.6 to 41.0% at the gene level and from 22.8 to 35.2% at the protein level. In contrast, AMPK expression increased by up to 53.8% at the transcriptional level and 47.9% at the protein level, indicating activation of cellular energy-sensing pathways. Consistent with these molecular changes, immunofluorescence analysis demonstrated a decrease in mitochondrial signal intensity of 16.6 to 45.7% and a concomitant increase in F-actin organization of 52.9 to 129.4%, reflecting metabolic and structural remodeling of adipocytes.
Conclusions: Panax ginseng-derived exosomes suppressed adipogenic and lipogenic signaling while enhancing AMPK activation, supporting their potential as plant-derived modulators of adipocyte differentiation and lipid metabolism.
Keyword: Anti-obesity, Lipid metabolism, AMPK signaling, Panax ginseng, Exosome

MOP1-2-3 *3. Clinical Lipidology & Genetics*

Ginseng-derived exosomes restore CCK-mediated satiety and remodel gut microbiota in high-fat diet-induced obesity

정기하[1*], 김진우[1], 채현우[1], 정은지[2], 채서연[2], 이선혜[1]

[1]선문대학교 식품공학·영양학부, [2]선문대학교 응용생물과학과

Objectives: Obesity is associated with adipose tissue dysfunction, impaired satiety signaling, gut microbiota dysbiosis, and chronic low-grade inflammation, which contribute to metabolic and cardiovascular risk. Ginseng-derived exosomes (GDE) have emerged as a novel bioactive component distinct from conventional ginsenosides; however, their metabolic effects under obesogenic conditions remain unclear. This study aimed to investigate whether oral administration of GDE attenuates obesity-related metabolic dysfunction by modulating adipose remodeling, cholecystokinin (CCK)-mediated satiety responsiveness, gut microbiota composition, and intestinal inflammation in a high-fat diet (HFD)-induced obesity model.
Methods: Male C57BL/6J mice were fed a low-fat diet (LF), HFD, or HFD supplemented with orally administered GDE for 9 weeks. Body weight, energy intake, adipose tissue histology, and leptin mRNA expression were evaluated. CCK-mediated satiety was assessed using CCK-8 injection followed by short-term food intake measurement. Gut microbiota composition was analyzed by 16S rRNA sequencing. Intestinal morphology, crypt depth, mucus staining, and inflammatory gene expression were examined using histological analysis and qRT-PCR.
Results: GDE supplementation significantly reduced adipocyte diameter and normalized epididymal leptin mRNA expression despite comparable energy intake between HFD groups. HFD-fed mice showed blunted anorexigenic responses to CCK, whereas GDE-treated mice exhibited restored CCK-induced suppression of food intake comparable to LF controls. Microbiota analysis revealed decreased Blautia, Colidextribacter, and Alistipes and increased Anaerotruncus and Parabacteroides. In addition, GDE significantly attenuated ileal TNF-α expression and increased colonic crypt depth without significant changes in tight junction gene expression or mucus coverage.
Conclusions: Oral administration of ginseng-derived exosomes attenuates adipocyte hypertrophy, restores CCK-mediated satiety responsiveness, remodels gut microbiota, and reduces intestinal inflammation independently of energy intake reduction. These findings suggest that GDE exerts multifactorial metabolic benefits relevant to obesity-associated metabolic and inflammatory disorders.
Keyword: Ginseng-derived exosomes, Obesity

MOP1-2-4 *4. Clinical Vascular Disease & Nutrition*

Nutrient-driven immunometabolic reprogramming of skeletal muscle by taurine and leucine in diabetic sarcopenia

Amara Zulfiqar[2*], Jung-Eun Yim[1,2]

[1]Department of Food and Nutrition, [2]Interdisciplinary Program in Senior Human Ecology, Changwon National University, Republic of Korea

Objectives: Diet-induced diabetic sarcopenia is driven by insulin resistance and chronic low-grade inflammation that programs skeletal muscle toward catabolism and reduced oxidative capacity. We tested whether taurine and/or leucine supplementation counteract these pathways in a high-fat/high-sucrose (HFHS) model.
Methods: Male C57BL/6J mice (6 weeks old; n=10/group) received a standard diet (normal) or HFHS for 24 weeks. HFHS-fed mice received metformin (150mg/kg; positive control) or taurine (1.5% w/v), leucine (1.5% w/v), or taurine+leucine (each 1.5% w/v; total 3% w/v). Glucose tolerance was assessed by OGTT and AUC. Tibialis muscle was analyzed by RT-qPCR for inflammatory mediators (CCL2, IL-6, NF-κB subunits), metabolic/oxidative regulators (AMPK, Pgc-1α), and myogenic/catabolic regulators (MEF2C, KLF15).
Results: HFHS feeding increased OGTT AUC versus normal ($p<0.0001$). Among HFHS groups, leucine reduced AUC compared with HFHS control ($p<0.01$), while taurine and taurine+leucine showed non-significant reductions. In muscle, HFHS-induced chemokine/cytokine activation (CCL2, IL-6) and increased NF-κB related transcripts. Taurine+-Leucine most robustly suppressed CCl2 and lowered IL-6 toward baseline, indicating attenuation of inflammatory signaling, HFHF-induced KLF15 upregulation was normalized by metformin and partially reduced by taurine. Taurine (alone and combined) increased PGC-1α and shifted AMPK expression toward the normal profile. mTOR and RAGE transcripts were also assessed but are not shown because no between-group differences were observed.
Conclusions: Taurine-based supplementation—particularly when combined with taurine+leucine—reprogrammed skeletal muscle toward a less inflammatory and more oxidative phenotype, while leucine produced the clearest improvement in systemic glucose tolerance. Together, these complementary immunometabolic effects support taurine and leucine as practical nutritional candidates for mitigating diabetic sarcopenia.
Keyword: High fat/high-sucrose model, Insulin resistance, Inflammation, Taurine, Leucine

MOP1-3-1 *4. Clinical Vascular Disease & Nutrition*

Dietary protein quality and dyslipidemia in Korean adults: analysis of KNHANES 2016-2024

함현지*, 하경호

제주대학교 식품영양학과

Objectives: Dietary protein plays a significant role in cardiometabolic health, and emerging evidence suggests that beyond mere quantity, the quality of protein intake is also a critical factor influencing lipid metabolism. However, epidemiological evidence on protein quality—particularly indicators reflecting protein sources and amino acid composition—and dyslipidemia remains limited. This study aimed to examine the associations between dietary protein quality indicators and dyslipidemia among Korean adults.
Methods: This cross-sectional study included 21,651 adults from the Korea National Health and Nutrition Examination Survey (KNHANES) 2016-2024. Protein quality indicators included the Healthy Plant and Protein Quality Index (HPPQI), plant-to-animal protein ratio (P:A ratio), and essential amino acid-to-total protein ratio (EAA:total protein ratio). Each indicator was categorized into quintiles. Multivariable logistic regression models were used to estimate odds ratios (ORs) and 95% confidence intervals (CIs) for dyslipidemia and its components, adjusting for sociodemographic and lifestyle factors.
Results: Higher HPPQI was associated with lower odds of hypertriglyceridemia (OR 0.76, 95% CI 0.63-0.91, p for trend=0.0016), low HDL-cholesterolemia (OR 0.76, 95% CI 0.63-0.92, p for trend=0.0022), and dyslipidemia (OR 0.83, 95% CI 0.73-0.94, p for trend=0.0026) In contrast, a higher P:A ratio was associated with higher odds of hypertriglyceridemia (OR 1.45, 95% CI 1.23-1.71, p for trend<0.0001), low HDL-cholesterolemia (OR 1.28, 95% CI 1.09-1.51, p for trend=0.0009), and dyslipidemia (OR 1.15, 95% CI 1.02-1.29, p for trend=0.0344). The EAA:total protein ratio showed no significant association.
Conclusions: Dietary protein quality indices, such as HPPQI and P:A ratio, were significantly associated with dyslipidemia in Korean adults. These findings suggest that qualitative composition of dietary protein, rather than the simple proportion of protein sources, should be considered in relation to lipid metabolism. [This study was supported by the National Research Foundation of Korea (NRF) grant funded by the Korean government (MSIT) (No. 2021R1G1A1008495 & RS-2025-25429813).]
Keyword: Dietary protein quality, Protein quality indicators, Dyslipidemia, Korean adults

MOP1-3-2 *4. Clinical Vascular Disease & Nutrition*

Skin carotenoids as biomarkers of fruit and vegetable intake are associated with lipid profiles and dyslipidemia in Korean adults

윤예진[1*], 최종민[2], 박진영[2], 문현석[2], 정명훈[2], 엄근선[2], 정효지[1]

[1]서울대학교 보건대학원 보건학과, [3]Mobile eXperience (MX) Business, Device eXperience (DX) Division, 삼성전자(주)

Objectives: Skin carotenoids have been proposed as objective biomarkers of fruit and vegetable (FV) intake. This research aimed to investigate the associations between skin carotenoid level and blood lipid profiles, as well as the risk of dyslipidemia in Korean adults.
Methods: Theses associations were examined using a cross-sectional study comprising 540 Korean adult (male: 244, female: 296, mean age: 39.43±10.82). Skin carotenoid level was measured using Samsung Galaxy Watch (Raman spectroscopy device), and fasting blood samples were collected to assess low-density lipoprotein (LDL) cholesterol, high-density lipoprotein (HDL) cholesterol, and triglycerides. Dyslipidemia was defined as LDL ≥160 mg/dL, HDL <40 mg/dL or triglycerides ≥200 mg/dL. Linear regression assessed associations with lipid markers, and logistic regression estimated odds ratios (ORs) and 95% confidence intervals (CIs) for dyslipidemia.
Results: Skin carotenoid levels were positively associated with HDL cholesterol (β=0.18; 95% CI: 0.12 to 0.25; p<.0001) and inversely associated with triglyceride (β=-0.06; 95% CI: -0.09 to -0.03; p=0.0003), whereas no significant association was observed with LDL cholesterol. In logistic regression analyses, greater skin carotenoid levels were associated with reduced odds of dyslipidemia (OR=0.97; 95% CI: 0.96 to 0.99; p=0.009).
Conclusions: Higher skin carotenoid level, reflecting greater FV intake, were associated with more favorable lipid profiles and a lower likelihood of dyslipidemia. Skin carotenoids may serve as a useful biomarker for identifying individuals with a healthier cardiometabolic profile. *This research was funded by Samsung Electronics Co., Ltd.
Keyword: Fruit and vegetable, Skin carotenoids, Dyslipidemia

MOP1-3-3 *4. Clinical Vascular Disease & Nutrition*

이상지질혈증 노인의 다량영양소 섭취 양상 분석: 2022-2024년 국민건강영양조사 자료를 이용하여

이경빈*, 함현지, 하경호

제주대학교 식품영양학과

Objectives: 이상지질혈증은 노인 인구에서 심혈관질환의 주요 위험요인이며 다량영양소의 섭취 구성은 혈중 지질 농도와 밀접하게 관련되어 있으나, 국내 노인을 대상으로 이상지질혈증 세부 유형별 다량영양소 섭취 특성을 비교한 연구는 제한적이다. 이에 본 연구는 노인의 이상지질혈증 유형별 다량영양소 섭취 특성을 비교하고자 하였다.
Methods: 2022-2024년 국민건강영양조사에 참여한 65세 이상 노인(n=4,623)중 이상지질혈증을 갖고 있는 2,623명을 대상으로 하였으며, 이상지질혈증은 한국 지질동맥경화학회의 기준에 따라 고중성지방혈증, 고콜레스테롤혈증, 저HDL-콜레스테롤혈증, 고LDL-콜레스테롤혈증, 이상지질혈증으로 정의하였다. 1일 24시간 회상법을 이용해 다량영양소 섭취량을 산출하였고, 이상지질혈증 유무에 따라 섭취량 및 에너지 적정 비율(AMDR) 충족률의 차이를 비교하였다.
Results: 이상지질혈증 유형별 다량영양소 섭취 수준을 분석한 결과 고LDL-콜레스테롤혈증군과 고콜레스테롤혈증군에서 탄수화물 섭취량(g/day) 및 에너지 기여율이 정상군에 비해 유의하게 낮았으나($p<0.05$), 단백질과 지방의 에너지 기여율은 유의하게 높았다($p<0.05$). 반면, 저HDL-콜레스테롤혈증군과 고중성지방혈증군은 탄수화물 섭취량 및 에너지 기여율이 유의하게 높았으며($p<0.001$), 지방 섭취량과 에너지 기여율은 유의하게 낮았다($p<0.05$). AMDR을 기준으로 분석한 결과, 탄수화물을 총 에너지의 65%를 초과하여 섭취하는 비율은 저HDL-콜레스테롤혈증군과 고중성지방혈증군에서 정상군에 비해 유의하게 높았으며, 반대로 고LDL-콜레스테롤혈증군과 고콜레스테롤혈증군에서는 지방을 30% 초과하여 섭취하는 비율이 높았다($P<0.05$). 특히 고콜레스테롤혈증군은 단백질 20% 초과 및 포화지방 7% 초과 섭취 비율이 높았으며, 고LDL콜레스테롤군도 단백질 20% 초과 섭취 비율이 높은 경향을 보였다. 반면, 세부 유형을 통합한 전체 이상지질혈증 유병 여부에 따른 유의한 차이는 관찰되지 않았다.
Conclusions: 65세 이상 노인에서 이상지질혈증의 세부 유형에 따라 다량영양소 섭취 구성 및 AMDR 대비 분포의 유의한 차이가 확인됐다. 고중성지방혈증과 저HDL-콜레스테롤혈증군은 탄수화물 기여율이 높은 식사 패턴을 보인 반면, 고LDL-콜레스테롤혈증군에서는 지방, 특히 포화지방산의 섭취 비율이 상대적으로 높은 경향을 보였다. 따라서 향후 노인 이상지질혈증 관리 및 중재시 세부 유형을 고려한 맞춤형 영양 중재 전략 수립이 필요할 것이다.
Keyword: 이상지질혈증, 다량영양소, 노인, 탄수화물

MOP1-3-4 *4. Clinical Vascular Disease & Nutrition*

Sex-specific dietary pattern associations with abdominal adiposity and pulmonary function trajectories in a population-based cohort

박지현[1,2*], 임수빈[2], 김민지[2], 박소연[2], 이다빈[2], 김정선[1], 김오연[2,3]

[1]Cancer AI and Digital Health, Graduate School of Cancer Science and Policy, National Cancer Center, Republic of Korea, [2]동아대학교 대학원 건강과학과 (임상영양전공) 식품영양학과, [3]동아대학교 식품영양학과

Objectives: Pulmonary function decline is a major determinant of chronic respiratory morbidity and mortality. Although smoking is a well-established risk factor, dietary patterns may also influence pulmonary health through metabolic mechanisms, including abdominal obesity. However, sex-specific longitudinal associations remain insufficiently explored. Therefore, we examined the associations between dietary patterns, changes in waist circumference, and pulmonary function decline according to sex in a Korean population-based cohort.
Methods: We analyzed 1,749 adults with complete follow-up from baseline to the seventh examination cycle in the Ansan-Ansung cohort of the Korean Genome and Epidemiology Study. Dietary patterns were identified using factor analysis of a validated semi-quantitative food frequency questionnaire. Participants were categorized into sex-specific tertiles of dietary pattern scores. Analyses focused on the balanced dietary pattern in men and the unhealthy dietary pattern in women. Longitudinal changes in waist circumference and percent predicted forced expiratory volume in one second (FEV1p) and forced vital capacity (FVCp) were compared across tertiles.
Results: In men, lower adherence to the balanced dietary pattern was associated with greater increases in waist circumference and a higher tendency toward chronic obstructive pulmonary disease (COPD) prevalence. Despite higher smoking rates, men in the highest tertile exhibited smaller increases in abdominal obesity and a slower decline in pulmonary function than those in the lowest tertile. In women, higher adherence to the unhealthy dietary pattern was associated with greater declines in FEV1p and FVCp. Women with both high unhealthy diet scores and baseline abdominal obesity experienced the most pronounced FEV1p decline, whereas those with low scores and normal waist circumference showed the least decline.
Conclusions: Dietary patterns are longitudinally associated with abdominal obesity and pulmonary function decline in a sex-specific manner, supporting tailored dietary strategies to preserve respiratory health.
Keyword: Pulmonary function, Waist circumference, Dietary pattern, Gender-difference

MOP1-4-1 *3. Clinical Lipidology & Genetics*

Anti-obesity effects of Rice Bran-derived exosomes through AMPK-mediated inhibition of adipocyte differentiation and fatty acid synthesis

Do Hyeon Jin[1*], Ju Hwan Lee[2], Jong Hyun Oh[2], Jin Hee Park[1], Su Jeong Hwang[1], Ye Eun Choi[1], Sun Hye Lee[1,2], Jin Woo Kim[1,2]

[1]선문대학교 식품공학영양학부, [2]선문대학교 응용생물과학과

Objectives: Obesity is characterized by excessive lipid accumulation driven by activation of adipogenic transcription factors and lipogenic enzymes. AMP-activated protein kinase (AMPK) functions as a central metabolic regulator that suppresses adipogenesis and lipid synthesis. This study aimed to investigate the anti-obesity effects of rice bran-derived exosomes (RBDE) and their involvement in AMPK-mediated metabolic regulation during adipocyte differentiation.
Methods: RBDE were isolated by sequential membrane filtration followed by tangential flow filtration (TFF). Particle size was measured by nanoparticle tracking analysis, and exosomal identity was confirmed by TET-8 enrichment. Anti-adipogenic effects were evaluated in 3T3-L1 preadipocytes within a non-cytotoxic range ($\leq 8.3 \times 10^{11}$ particles/mL). Neutral lipid accumulation was quantified by Oil Red O staining. AMPK-α, adipogenic markers (PPAR-γ, C/EBP-α, FABP4), and lipogenic enzymes (ACL, ACC, FAS) were analyzed by RT-PCR and Western blotting. Single-cell AMPK-α expression was quantified by flow cytometry in RBDE-treated 3T3-L1 adipocytes.
Results: TFF-purified RBDE exhibited a mean diameter of 105.7 nm and a 2.6-fold enrichment of TET-8 relative to membrane-filtered fractions. RBDE reduced neutral lipid accumulation during adipocyte differentiation, with a maximal decrease of 84.7% versus the differentiation-induced control. AMPK-α expression, suppressed under differentiation, increased by 23.3-41.1% following RBDE treatment. PPAR-γ and C/EBP-α decreased by 18.9-30.3% and 2.6-14.1%, respectively, while FABP4 decreased by 27.2-30.2% at the mRNA level. ACL, ACC, and FAS were reduced by 22.4-26.8%, 11.2-23.4%, and 18.8-28.3%, respectively, with protein levels decreased by 18-40.8%. RBDE suppressed adipogenic progression (76.2-77.7%) and restored AMPK-α expression (94.0-98.6%), supporting AMPK-mediated metabolic regulation.
Conclusion: RBDE suppress adipocyte differentiation and lipid biosynthesis through AMPK-centered metabolic regulation, accompanied by attenuation of structural remodeling and oxidative stress during adipogenesis. These findings support RBDE as a promising plant-derived nanobiomaterial for anti-obesity applications.
Keyword: Anti-obesity mechanism, Rice bran-derived exosomes, AMPK, Adipocyte differentiation, Lipid metabolism regulation

MOP1-4-2 *3. Clinical Lipidology & Genetics*

Multi-evidence prioritization of lipid-trait genes in Asian cohorts integrating fine-mapping, colocalization, SMR, and MAGMA

지용호[1*], 신종원[3], Wes Spiller[4], 송태진[2]

[1]이화여자대학교 서울병원 첨단의생명연구원, [2]이화여자대학교 의과대학 신경과, [3]서울아산병원 진단검사의학과, [4]연세대학교 보건대학원

Objectives: To systematically prioritize lipid-trait genes in East Asian populations by integrating multi-layer genomic evidence and to refine drug-target candidates beyond single-method inference.
Methods: We meta-analyzed GWAS summary statistics for LDL-cholesterol (LDL-C), HDL-cholesterol (HDL-C), and triglycerides (TG) from three East Asian biobanks (KCPS-II, KoGES, TWB; total ≈400,000 participants). Genome-wide significant loci were refined using conditional fine-mapping (FM). Gene-level functional support was evaluated using colocalization with GTEx v10 eQTLs (PP4 $\geq$ 0.90) and SMR-HEIDI (P_SMR $< 5 \times 10^{-8}$, P_HEIDI > 0.01). Cross-method consensus (FM∩COLOC, FM∩SMR, triple overlap) and cross-trait replication were quantified. Pathway-level support was incorporated using MAGMA to derive a composite prioritization score.
Results: LDL-C fine-mapping recapitulated canonical loci including PCSK9, HMGCR, APOB, and SORT1, validating the analytical framework. Gene-level overlap analysis showed 31 FM∩COLOC genes for LDL-C and one triple-overlap gene for HDL-C (PLTP). Liver-specific colocalization highlighted biologically coherent candidates across lipid traits, including PLTP, APOC4, LIPG (HDL axis), and ATP4A, BCAM, and C20orf173 (LDL axis). Notably, several established targets (e.g., LDLR, NPC1L1) showed limited colocalization support, suggesting East Asian-specific LD architecture and tissue-dependent regulatory mechanisms. Overall, 44 novel candidates were identified, with the top 15 demonstrating multi-method consensus and cross-trait reinforcement.
Conclusions: An integrative FM-COLOC-SMR-MAGMA framework in East Asian cohorts enables robust prioritization of lipid-trait genes. By leveraging orthogonal evidence and tissue context, this approach refines translational target selection and highlights population-specific genetic architecture relevant for drug development.
Keyword: Fine-mapping, Lipid traits, Multi-omics integration

MOP1-4-3 *4. Clinical Vascular Disease & Nutrition*

Gene-diet interaction using polygenic risk score and dietary inflammatory index on the incidence of hypertension: a prospective cohort study

Daeun Jang[1*], Dahyun Park[2], Garam Jo[2], Min-Jeong Shin[3]

[1]Interdisciplinary Program in Precision Public Health, Korea University, [2]Institute for BioMaterials, Korea University, [3]School of Biosystems and Biomedical Sciences, Korea University, Republic of Korea

Objectives: The Empirical Dietary Inflammatory Pattern (EDIP), an inflammatory biomarker-based dietary index, is particularly relevant to hypertension given the central role of inflammation in vascular dysfunction. This study aimed to evaluate whether adherence to an anti-inflammatory diet is associated with incident hypertension in the context of genetic susceptibility calculated by polygenic risk score (PRS) in a Korean population.
Methods: A genome-wide association study for hypertension was conducted in the KoGES Ansan-Ansung cohort, and 16 genome-wide significant independent SNPs ($P<5\times10^{-8}$) were used to construct the polygenic risk score. Participants were classified into quartiles according to the distribution of the reverse-coded EDIP (rEDIP), in which higher scores indicate greater adherence to an anti-inflammatory dietary pattern. A total of 5886 individuals were included in hazard analysis after excluding those with baseline hypertension or implausible energy intake. Cox proportional hazard models were used to estimate hazard ratios (HR).
Results: During 10.01 mean person-years of follow-up, 2354 participants developed hypertension. Higher genetic risk was significantly associated with an increased risk of hypertension (HR=1.71 in 3rd tertiles). In contrast, higher rEDIP scores showed inverse associations with incident hypertension in the 3rd and 4th quartiles (HR=0.90 and 0.84, respectively). A significant gene-diet interaction was observed (P for interaction = 0.016), with individuals in the highest PRS tertile and the third quartile of rEDIP showing a significantly increased risk compared with the reference group.
Conclusions: Genetic predisposition significantly increases hypertension risk, while adherence to a less inflammatory diet may attenuate the risk, suggesting that dietary modification may help attenuate hypertension risk among those who are genetically highly susceptible.
Keyword: Hypertension, Polygenic risk score, Gene-diet interaction, Empirical dietary inflammatory pattern

MOP1-4-4 *4. Clinical Vascular Disease & Nutrition*

Genotype-specific variability in BMI response to short-term lifestyle modification: roles of FTO and AdipoQ polymorphisms in Korean women

임수빈[1*], 박지현[1], 김민지[1], 박소연[1], 이다빈[1], 김채린[1], 김예진[1], 최미옥[2,3], 안원석[4], 김오연[1,2]

[1]동아대학교 대학원 건강과학과 (임상영양전공) 식품영양학과, [2]동아대학교 식품영양학과, [3]동아대학교병원 영양과, [4]동아대학교병원 신장내과

Objectives: Inter-individual variability in weight-loss response to lifestyle interventions may partly reflect genetic susceptibility. Polymorphisms in the fat mass and obesity-associated gene (FTO) and adiponectin gene (AdipoQ) have been linked to obesity and metabolic traits; however, their influence on short-term dietary intervention responsiveness in Asian populations remains unclear. This study aimed to examine whether FTO rs9939609 and AdipoQ polymorphisms (+45T>G, +276G>T, -11377C>G) are associated with changes in obesity-related parameters following a 4-week lifestyle modification program in overweight or obese Korean women with metabolic syndrome risk factors.
Methods: Sixty women participated in a structured 4-week dietary education intervention. Anthropometric measures, blood pressure, and nutrient intake were assessed before and after the intervention. Genotyping was conducted using standard molecular methods. Genotype-stratified and combined-genotype analyses were performed with age adjustment.
Results: After 4 weeks, BMI, systolic blood pressure, fat mass, and body fat percentage significantly decreased (all $P<0.05$). Total energy intake (P=0.045), fat intake percentage (P=0.048), and carbohydrate intake (P=0.018) were also reduced. Genotype-stratified analyses demonstrated differential BMI responsiveness. A higher proportion of BMI reduction was observed among FTO wild-type carriers (82.1%) and AdipoQ+45 minor-allele carriers (82.4%), whereas individuals carrying both FTO minor alleles and AdipoQ minor alleles (+276 or -11377) showed a greater frequency of BMI increase. Combined genotype analyses suggested additive effects on BMI response patterns.
Conclusions: FTO and AdipoQ polymorphisms may contribute to genotype-specific variability in short-term BMI response to lifestyle modification. These exploratory findings support further investigation in larger, well-powered studies to clarify the role of common obesity-related variants in precision nutrition strategies.
Keyword: FTOrs9939609, AdipoQ single nucleotide polymorphism, Overweight, Obesity, Body mass index, Metabolic syndrome, Lifestyle modification

MOP2-1-1 *2. Basic Science of Atherosclerosis*

AMPK activation by pioglitazone attenuates cholesterol-induced VSMC phenotypic modulation and atherosclerosis via inhibition of STAT3-mediated inflammation and senescence

김슬기[1,2*], 최푸름[1,2], 최형철[1,2]

[1]영남대학교 의과대학 약리학교실, [2]영남대학교 세노테라피 기반 대사질환 제어 연구센터

Objectives: Atherosclerosis is considered a chronic inflammatory disease, and senescent cells secrete the inflammatory cytokines and chemokines, leading to acceleration of atherosclerosis. VSMCs are the predominant cell type in all stages of atherosclerotic plaques. We investigated the underlying mechanism in VSMC phenotypic modulation and relationship between senescence and atherosclerosis.
Methods: We treated with cholesterol (20 μg/ml) for 24 h to induction of VSMC phenotypic modulation. The verification of VSMC phenotypic modulation was investigated by Oil Red O staining and qPCR. Moreover, the effects of pioglitazone were examined by western blot analysis, Oil Red O staining, β-gal staining, qPCR, immunofluorescence analysis, and ATP level analysis in cholesterol-treated VSMCs. For in vivo study, ApoE KO mice were fed a chow diet or high-fat / high-cholesterol diet (HFHC) for 12 weeks. HFHC-fed mice were treated with pioglitazone (20 mg/kg/day) by oral administration for 12 weeks. After sacrifice, whole aortas were obtained and analyzed by β-gal staining, Oil Red O staining, and western blot analysis.
Results: We found that cholesterol caused VSMC phenotypic modulation to macrophage-like cells and simultaneously induced lipid accumulation and cellular in rat and human VSMCs. Pioglitazone treatment reduced the ATP levels, leading to AMPK activation in PPARγ-independent manner. Pioglitazone decreased cholesterol-induced cellular senescence and lipid accumulation, and inflammatory cytokines through AMPK-STAT3 signaling pathway in rat and human VSMCs. Furthermore, in vivo results showed that pioglitazone reduced not only senescence but also atherosclerotic plaque, and induced AMPK-STAT3 signaling pathway in aortas of HFHC-fed ApoE KO mice.
Conclusions: Our results indicate that anti-diabetic drug pioglitazone could activate AMPK as another signaling pathway. Pioglitazone-activated AMPK inhibits inflammation and cellular senescence leading to alleviation of VSMC phenotypic modulation and atherosclerosis. These findings suggest that AMPK-STAT3 signaling pathway might be a potential therapeutic or preventive target for atherosclerosis.
Keyword: VSMC, Senescence, Pioglitazone, STAT3, AMPK

MOP2-1-2 *2. Basic Science of Atherosclerosis*

Preventive mechanism of SGLT-2 inhibitor in each organ for diabetic rabbit model

Seul-Gee Lee[1*], Yong-Jun Lee[2], Oh-Hyun Lee[3], Choong-Ki Kim[4], Jung-Sun Kim[2]

[1]Integrative Research Center for Cerebrovascular and Cardiovascular Diseases, Yonsei University, [2]순환기/심장내과, Severance Cardiovascular Hospital, Yonsei University College of Medicine, [3]순환기/심장내과, Yongin Severance Hospital, Yonsei University College of Medicine, [4]순환기/심장내과, EwhaWomans University Seoul Hospital, Republic of Korea

Objectives: The Sodium Glucose Cotransporter-2 (SGLT-2) inhibitor, a new class of diabetic medication, reduces inflammatory responses. However, mechanism for the protective effect has not been clearly identified in each organ. Therefore, we sought to investigate each organ of anti-inflammatory mechanism of SGLT-2 inhibitor in rabbit model.
Methods: Rabbit divided into two groups (each group/n=5); diabetic group, diabetic dapagliflozin group. Dapagliflozin was given for a total of 8 weeks. Rabbits with fasting glucose levels more than 200mg/dl after 48 hours of ALX injection were considered as diabetic and used for further study.
Results: At 8 week follow up, mesangial matrix of kidney accumulation was detected in the diabetic group. However, mesangial matrix accumulation was less than those in the diabetic dapagliflozin group compared with that in the diabetic group. Also, interstitial fibrosis was suppressed in the diabetic dapagliflozin group. The liver of lipid droplet area and size in dapagliflozin treated group was smaller and fewer than in diabetic group. The adipose tissue of crown-like structures was found in diabetic group. Also, fat cells size was significantly decrease than in diabetic group. The SGLT-2 mRNA level of kidney was significantly down-regulated in the dapagliflozin treated group compared to the diabetic group and RAGE and fibronectin level were reduced tendency. The adipose tissue of several inflammatory markers such as IL-18 and IL-6 mRNA level were significantly decreased in dapagliflozin group compared to diabetic group, and a reducing tendency of NLRP3 and IL-1β level.
Conclusions: These results demonstrate that administration of dapagliflozin ameliorates interstitial fibrosis in kidney also improve lipid droplets area and size of liver tissue in diabetic model. In addition, improved adipocyte size and inflammation levels of adipose tissue. Our results may suggest potential clinical implication for dapagliflozin for the treatment of organ damage in diabetes patients.
Keyword: SGLT-2 inhibitor, Dapagliflozin, Diabetic model, Inflammation, Fibrosis

MOP2-1-3 *3. Clinical Lipidology & Genetics*

Protective effects of lactobacillus delbrueckii on metabolic dysfunction and hepatic steatosis in high-fat diet-fed obese mice

두미애[1*], 이지수[2], 김연우[2], 조민서[2], 하정헌[2]

[1]국립군산대학교 식품영양학과, [2]단국대학교 식품영양학과

Objectives: Lactobacillus delbrueckii has emerged as a potential probiotic candidate for improving metabolic disorders, including metabolic dysfunction-associated steatotic liver disease (MASLD); however, in vivo evidence remains limited. This study evaluated the effects of L. delbrueckii supplementation on insulin resistance, lipid metabolism, and hepatic steatosis in a high-fat diet (HFD)-induced obese mouse model.
Methods: Male C57BL/6J mice were assigned to four groups (n=12/group): Normal chow diet, HFD, HFD with L. delbrueckii, or HFD with resmetirom(thyroid receptor β agonist) and treated for 12 weeks following obesity induction.
Results: L. delbrueckii supplementation significantly reduced body weight, serum insulin levels, and HOMA-IR, indicating improved insulin sensitivity, and decreased circulating triglyceride levels without altering total cholesterol. Hepatic triglyceride and cholesterol accumulation, liver weight, and histopathological features of MASLD, including NAFLD activity scores and fibrosis, were markedly attenuated. Liver function was improved, accompanied by enhanced phosphorylation of adenosine monophosphate-activated protein kinase (AMPK), although the expression of lipid metabolism-related genes remained largely unchanged. In epididymal adipose tissue, leptin levels were reduced, while adiponectin levels were unaffected, and adipocyte hypertrophy was observed.
Conclusions: Overall, L. delbrueckii supplementation ameliorated insulin resistance and hepatic lipid deposition and improved liver pathology in HFD-induced obese mice, supporting its potential as a probiotic-based therapeutic approach for obesity-related metabolic dysfunction and MASLD.
Keyword: Metabolic dysfunction-associated steatotic liver disease, High-fat diet-induced obesity, Probiotics, Triglycerides

MOP2-1-4 *5. Others*

Liraglutide alters gut microbiota and improves endothelium-dependent relaxation in db/db mice

최수경[1*], 오은이[1], 서수환[2], 변선희[1], 이주용[1], 이영호[1]

[1]연세의대 기초과학, [2]건양대 기초과학

Objectives: Endothelial dysfunction is a hallmark of type 2 diabetes mellitus (T2DM) and a major contributor to cardiovascular complications. Although glucagon-like peptide-1 receptor agonists (GLP-1RAs) improve glycemic control and cardiovascular outcomes, the mechanisms linking GLP-1RA therapy, gut microbiome modulation, and endothelial function remain incompletely understood. In this study, we investigated whether the GLP-1RA liraglutide improves endothelial dysfunction in T2DM through microbiome-associated mechanisms that support vascular homeostasis.
Methods: Male db/db mice and non-diabetic controls were treated with liraglutide (300 μg/kg/day, intraperitoneally) or saline for two weeks. Vascular function was assessed in mesenteric resistance arteries using wire myography. Human umbilical vein endothelial cells (HUVECs) were exposed to high glucose with or without liraglutide or the short chain fatty acid (SCFA), butyrate. Endothelial nitric oxide (NO) signaling was evaluated by eNOS (at Ser1177) phosphorylation and nitrite production.
Results: Gut microbiota composition was analyzed by 16S rRNA gene sequencing. Liraglutide significantly improved endothelium-dependent relaxation in db/db mice and restored high glucose-induced impairment of eNOS phosphorylation and NO production in HUVECs. In vivo, diabetes was associated with marked gut dysbiosis characterized by reduced alpha diversity and depletion of SCFA-producing taxa. Liraglutide treatment substantially restored microbial diversity and enriched beneficial genera, including Lachnospiraceae and Lactobacillus. Consistently, low-dose butyrate modestly enhanced NO production in endothelial cells.
Conclusions: These findings support the concept of a GLP-1RA-microbiome-vascular axis, in which liraglutide-associated remodeling of the gut microbiota may contribute to improved endothelial NO signaling and vascular function in diabetes.
Keyword: Liraglutide, Microbiome, Artery

MOP2-1-5 *5. Others*

Empagliflozin binds JNK3 and suppresses c-Jun/c-Fos signaling to protect against diabetic cardiomyopathy

Trong Kha Pham[1,2*], Hyoung Kyu Kim[1], Hoai T.T. Nguyen[1], Maria Victoria Faith Garcia[1], Changshin Yoon[1], Mário Albino Sozinho Indarua[1], Thu Thi Vu[2], Jin Han[1]

[1]Cardiovascular and Metabolic Disease Center, Smart Marine Therapeutics Center, Department of Physiology, Inje University, Republic of Korea, [2]Department of Physiology and Human Biology, VNU University of Science, Vietnam National University-Hanoi, Vietnam

Objectives: Identify the direct molecular target and mechanistic pathways underlying the cardioprotective effects of EMPA in diabetic cardiomyopathy.
Methods: Kinase activity assay was used to screen 107 kinases for potential EMPA targets, confirming binding through protein-ligand docking, and surface plasmon resonance (SPR) between EMPA and c-Jun N-terminal kinase 3 (JNK3). Cardioprotection was assessed in male db/db mice receiving EMPA (10 mg/kg/day) for 10 weeks and AC16 human cardiomyocytes under diabetic conditions (high glucose and palmitic acid) were treated with EMPA (500 nM). The central role of JNK3 was further validated in vitro using a selective inhibitor SR-3576, overexpression and siRNA-mediated knockdown.
Results: In db/db mice, EMPA reduced cardiac fibrosis, attenuated mitochondrial injury, and improved systolic and diastolic function, correlating with c-Jun/c-Fos signaling suppression. EMPA selectively inhibited JNK3 kinase activity; docking and SPR demonstrated direct EMPA-JNK3 binding. In AC16 cells, EMPA or JNK3 inhibition decreased c-Jun phosphorylation and c-Fos expression. In addition, JNK3 overexpression increased c-Jun/c-Fos signaling and impaired mitochondrial function, which EMPA treatment reversed. Conversely, siRNA knockdown of JNK3 improved mitochondrial function independent of EMPA, underscoring the central role of JNK3.
Conclusions: EMPA protects against DCM by directly binding to and selectively inhibiting JNK3, thereby suppressing c-Jun/c-Fos signaling, alleviating mitochondrial dysfunction and limiting cardiac fibrosis. These findings identify JNK3 as a novel, direct EMPA target and reveal EMPA's profound cardioprotective effect via inhibition JNK3/c-Jun/c-Fos axis.
Keyword: Diabetic cardiomyopathy, SGLT2 inhibitor, Empagliflozin

MOP2-2-1 *2. Basic Science of Atherosclerosis*

Macrophage-specific CAR deletion modifies macrophage polarity and induces atherosclerotic plaque formation

김홍기*, 임병관

중원대학교 기초과학

Objectives: The Coxsackievirus and Adenovirus Receptor (CAR) is a cell surface protein that mediates CVB3 and Type 5 adenovirus infections and is a critical component of epithelial tight junctions and cardiac intercalated discs. While macrophages are essential mediators of early-stage inflammatory conditions, the specific role of CAR within macrophage populations remains unexplored in metabolic disease.
Methods: To investigate this, we generated a macrophage-specific conditional CAR knockout (CAR-KO; F/F Cre) mouse model using lysozyme-Cre recombinase. Following 12 weeks of a high-fat diet (HFD). Revised histological and Immunological findings by using liver lipid stain and flow cytometry (FACS) of peritoneal cavity cells.
Results: CAR-KO mice exhibited significant increases in body weight, abdominal white adipose tissue, and liver weight compared to wild-type (WT) controls. These metabolic changes were accompanied by elevated serum cholesterol and liver triglyceride levels, which subsequently induced atherosclerotic plaque formation in the aortic arch. Histological analysis of CAR-KO mice revealed increased inflammatory cell infiltration in the liver, lipid droplet accumulation, and enlarged adipocytes. Furthermore, F4/80 and Ly6G-positive cells were dramatically increased in the peritoneal cavity of CAR-KO mice.
Conclusions: Our results demonstrate that macrophage-specific CAR deletion exacerbates macrophage activity, leading to dysregulated blood lipid levels and hepatic lipid accumulation. These findings suggest that CAR in macrophages plays a pivotal role in regulating chronic inflammation and the progression of obesity.
Keyword: Coxsackievirus and adenovirus receptor, Atherogenesis, Fatty liver, Obesity

MOP2-2-2 *2. Basic Science of Atherosclerosis*

ANGPTL4 preserves endothelial homeostasis and suppresses EndMT in atherosclerosis: extension to a human iPSC-derived atheroid model

조동임[1*], 김용숙[2], 강보경[1], 조미영[1], 유진[1], 김찬율[1], 안영근[1,2]
[1]전남대학교병원 순환기/심장내과, [2]전남대학교 순환기/심장내과

Objectives: Atherosclerosis progresses through endothelial dysfunction, vascular inflammation, endothelial-to-mesenchymal transition (EndMT), and plaque instability. We previously identified angiopoietin-like 4 (ANGPTL4) as a critical endothelial-protective factor that suppresses inflammatory and TGF-β-driven EndMT by restoring KLF2 signaling, thereby preserving vascular integrity in vitro, in vivo, and in human atherosclerotic lesions. However, human-relevant platforms that integrate immune-vascular interactions remain limited.
Methods: In our prior work, the effects of ANGPTL4 were investigated using murine atherosclerosis models, primary human endothelial cells, iPSC-derived endothelial cells, and patient-derived clinical data. To extend these findings, we are developing a human iPSC-derived atheroid model by integrating vascular spheroids with iPSC-derived monocytes exposed to TNF-α and oxidized LDL, enabling controlled formation of an inflammatory lesion-like core within a 3D human vascular context.
Results: ANGPTL4 suppressed endothelial inflammation, preserved barrier integrity, and inhibited TGF-β-Smad2-mediated EndMT via KLF2 restoration, with EndMT markers correlating with plaque complexity in human atherosclerotic samples. In parallel, preliminary data from the iPSC-derived atheroid system demonstrate early endothelial activation, selective recruitment of inflammatory monocytes, lipid-laden macrophage-like features, smooth muscle phenotypic modulation, and extracellular matrix remodeling, recapitulating key features of inflammatory vascular pathology without overt cytotoxicity.
Conclusions: Together, our established findings identify ANGPTL4 as a pivotal regulator of endothelial homeostasis in atherosclerosis, while ongoing work suggests that a human iPSC-derived atheroid model may provide a complementary platform to study immune-vascular interactions and to functionally evaluate endothelial-protective mechanisms in a human-relevant setting.
Keyword: ANGPTL4, Atherosclerosis, EndMT, Endothelial dysfunction

MOP2-2-3 *2. Basic Science of Atherosclerosis*

Smooth muscle cell-specific deletion of TXNIP ameliorates medial vascular calcification

Ae-Rang Hwang*, Changhoon Woo
영남대학교 의과대학 약리학과

Objectives: Vascular calcification is a significant pathological characteristic of cardiovascular diseases, often exacerbated by metabolic disorders such as chronic kidney disease and diabetes mellitus. Recent studies have highlighted oxidative stress and inflammation as central mechanisms in the development of vascular calcification. While TXNIP is known to positively regulate reactive oxygen species (ROS) generation and inflammasome activation, the specific contributions of TXNIP to vascular calcification have not been thoroughly elucidated. This study aimed to elucidate the role of TXNIP in the pathogenesis of vascular calcification through combined in vitro and in vivo approaches.
Methods: Medial vascular calcification was evaluated in vivo using 5/6 nephrectomy-induced chronic kidney disease and vitamin D_3-induced nephropathy mouse models. In vitro, primary vascular smooth muscle cells (VSMCs) isolated from TXNIP wild-type (WT) and knockout mice were cultured under calcifying conditions. Calcium deposition was assessed by Alizarin Red and Von Kossa staining. Mitochondrial dysfunction was evaluated using Seahorse extracellular flux analysis and JC-1 staining. In addition, RNA sequencing was performed to identify differentially expressed cell cycle-related genes associated with TXNIP overexpression.
Results: In vivo analyses showed that TXNIP expression was markedly increased in vascular smooth muscle cells and was associated with enhanced medial calcification. This was evidenced by increased calcium deposition, upregulation of osteogenic markers, and elevated mitochondrial-derived ROS production. In contrast, smooth muscle cell-specific TXNIP deficiency significantly attenuated vascular calcification and reduced osteogenic marker expression. TXNIP suppression also inhibited inflammasome activation and improved mitochondrial function. These findings suggest a novel mechanistic link between TXNIP activity and osteogenic differentiation pathways in medial vascular calcification.
Conclusions: These findings demonstrate that TXNIP plays a pivotal role in medial vascular calcification by promoting oxidative stress, inflammasome activation, and mitochondrial dysfunction in vascular smooth muscle cells. Targeting TXNIP may represent a promising therapeutic strategy for preventing medial calcification in cardiovascular diseases associated with metabolic disorders.
Keyword: Mitotic cell cycle, Mitochondrial dysfunction, TXNIP, Vascular calcification

MOP2-2-4 *2. Basic Science of Atherosclerosis*

Identification of circular RNAs in ischemic heart disease

Dahee Jeong[1,2,3*], Yeongseo Ryu[1,2,3], Young-Kook Kim[1,2,3]

[1]Department of Biochemistry, Chonnam National University Medical School, [2]BioMedical Sciences Graduate Program (BMSGP), Chonnam National University, [3]Medical Research Center for Innovative Control of Cardiovascular Remodeling Diseases, Hwasun, Jeollanam-do, Republic of Korea

Objectives: To identify circRNAs that are dysregulated in ischemic heart disease (IHD) and to investigate their roles in oxidative stress and cell survival pathways.
Methods: Publicly available RNA sequencing data from patients with IHD were analyzed to identify differentially expressed circRNAs between invasive coronary angiography (ICA)-positive and ICA-negative groups. A candidate circRNA was selected based on differential expression analysis. Subcellular localization was determined, and functional studies were performed in AC16 human cardiomyocyte cells using circRNA knockdown. Reactive oxygen species (ROS) levels and gene expression changes were assessed, and transcriptome analysis was conducted to identify affected pathways. Potential miRNA interactors and downstream target mRNAs were predicted to explore the regulatory mechanism.
Results: CircIHD was identified as a candidate circRNA significantly upregulated in ICA-positive patients compared to ICA-negative patients. CircIHD consists of five exons derived from its host gene and predominantly localizes to the cytoplasm. Knockdown of circIHD in AC16 cells significantly altered the expression of genes associated with antioxidant defense and cell death and reduced intracellular ROS levels. Transcriptome analysis revealed that circIHD depletion primarily influenced pathways related to cell survival and structural remodeling.
Conclusions: CircIHD modulates oxidative stress responses and cell survival pathways in cardiomyocytes, potentially through a circRNA/miRNA/mRNA regulatory axis. These findings suggest that circIHD may serve as a novel molecular regulator and potential therapeutic target in ischemic heart disease.
Keyword: Circular RNA, MiRNA, Ischemic heart disease

MOP2-2-5 *2. Basic Science of Atherosclerosis*

CircAFF3 modulation of p53-ID2 signaling in the retinal pigment epithelium links inflammation with cell death in dry age-related macular degeneration

Yeongseo Ryu[1,2*], Dahee Jeong[1,2], Young-Kook Kim[1,2]

[1]Department of Biochemistry, [2]BioMedical Sciences Graduate Program (BMSGP), Chonnam National University Medical School, Hwasun, Jeollanam-do, Republic of Korea

Objectives: Age-related macular degeneration (AMD) represents a multifactorial disease that is influenced by age, genetic, and environmental factors. AMD is characterized by dysfunction of the retinal pigment epithelium (RPE) resulting from oxidative stress, inflammation, and complement activation. As the disease progresses, the loss of the RPE and photoreceptors leads to geographic atrophy, which is a hallmark of dry AMD. Although research is ongoing, there is currently no established effective treatment for dry AMD. Notably, circular RNAs (circRNAs) have been studied in various diseases; however, the role of circRNAs in eye diseases remains poorly understood. To fill this gap, this study aimed to investigate circRNAs as potential therapeutic targets for dry AMD.
Methods: To identify circRNAs potentially involved in dry AMD pathogenesis, we used a laser-induced choroidal neovascularization (CNV) model. Analyzing circRNA expression in the early stages of this model identified circAFF3 as a candidate. Subsequently, to investigate the cytoplasmic function of circAFF3, we performed knockdown experiments using siRNA targeting the back-splicing junction of circAFF3.
Results: This study demonstrated that circAFF3 expression is consistently downregulated in models of dry AMD pathogenesis, including RPE at day 3 post-laser injury and NaIO3-treated ARPE-19 cells. Functional analysis of circAFF3 revealed that silencing circAFF3 triggers inflammatory responses through p65 activation in ARPE-19 cells. Additionally, we demonstrated that circAFF3, which is predominantly cytoplasmic, directly binds p53 in ARPE-19 cells. Accordingly, depletion of circAFF3 facilitated the translocation of p53 between the nucleus and cytoplasm, leading to the nuclear accumulation of p53. Subsequently, elevated nuclear p53 repressed ID2 expression, promoting oxidative stress and inducing regulated cell death pathways, such as apoptosis and ferroptosis.
Conclusions: Collectively, our study reveals that circAFF3 plays a crucial role in RPE dysfunction by modulating a circAFF3/p53/ID2 pathway, suggesting that circAFF3 could serve as a promising therapeutic target for dry AMD.
Keyword: Age-related macular degeneration, circAFF3, Retinal pigment epithelium

MOP2-2-6 *2. Basic Science of Atherosclerosis*

Transcriptome analysis of aortic arch in T2DM diabetes-induced atherosclerotic mice

Jeong-Hyun Kim*, A Yeon Hwang, Nari Kim
인제대학교 심혈관대사질환센터

Objectives: Since an understanding of the early progression of type 2 diabetes mellitus (T2DM)-induced atherosclerotic biomolecular changes is nuclear due to the complex and synergistical pathways interaction in the pathogenesis, the aim of this study is to investigate the underlying mechanism.
Methods: This study employed a preclinical atherosclerotic and streptozotocin (STZ)-induced T2DM diabetic mouse model with ApoE-/- fed a high-fat diet (HFD) and performed RNA sequencing of the atherosclerotic aortic arch regions from the mouse model.
Results: STZ-treated and HFD-fed ApoE-/- mice exhibited typical of T2DM conditions of hyperglycemia, dyslipidemia, and insulin resistance, together with significantly increased plaque area and necrotic core percentage compared to controls. RNA-seq analysis showed that differentially expressed genes (DEGs) in the early aggravated progression of the STZ-induced T2DM diabetic mice were enriched in the multiple pathways including PLC/PKC, inflammation, notch, extracellular matrix (ECM) responses. In additional network interaction analysis of 381 STZ-specific DEGs included in the enriched pathways, DEGs in the collagen/ECM cluster appeared to play an important role in the progression of diabetes-aggravated atherosclerosis.
Conclusions: Despite needs of further replication and functional studies, the results of the present study may provide underlying pathophysiological mechanisms of the progression of diabetes-aggravated atherosclerosis and will help to establish preventive and therapeutic strategies for the related diseases.
Keyword: T2DM, Atherosclerosis, Transcriptome, Streptozotocin, Collagen

MOP2-3-1 *3. Clinical Lipidology & Genetics*

Combined effects of lipoprotein(a) and HDL cholesterol on cardiovascular mortality: a longitudinal study

김병진*
성균관의대 강북삼성병원 순환기/심장내과

Objectives: Lipoprotein(a) [LP(a)] is a well-established cardiovascular risk factor, and its association with cardiovascular (CV) mortality has been previously demonstrated. However, the role of HDL cholesterol (HDL-C) in modulating the relationship between LP(a) and CV outcomes remains unclear, necessitating further investigation.
Methods: This longitudinal cohort study included 268,221 participants (mean age 38 years, men 51%) from the Kangbuk Samsung Health Study, who were enrolled between 2003 and 2016. Participants were categorized into four groups based on HDL-C levels (cutoff: 40 mg/dL) and LP(a) levels (cutoff: 50 mg/dL): high HDL-C with low LP(a), high HDL-C with high LP(a), low HDL-C with low LP(a), and low HDL-C with high LP(a). The median follow-up duration was 6.6 years.
Results: The median HDL-C and Lp(a) levels were 57.0 mg/dL and 18.4 mg/dL, respectively. High Lp(a) and low HDL-C were each independently associated with a significantly increased risk for both cardiovascular (1.84[1.27, 2.65] and 1.55[1.09, 2.21]) and all-cause(1.23[1.05, 1.44] and 1.16[1.00, 1.34]) mortality. Compared with participants with high HDL-C and low Lp(a), the multivariable-adjusted hazard ratios for CV mortality were 1.64[1.06, 2.52] in the high HDL-C and high Lp(a) group, 1.24[0.84, 1.85] in the low HDL-C and low Lp(a) group, and 3.77[1.82, 7.80] in the low HDL-C and high Lp(a) group. The results of all-cause mortality among the four groups were similar to those of CV mortality. In HDL-C stratified analysis, the increased risk for CV mortality associated with high Lp(a) was more pronounced and significant in the low HDL-C group (2.39[1.19, 4.83] vs. 1.67[1.08, 2.58].
Conclusions: Both Lp(a) and HDL-C are independently and jointly associated with increased risks of cardiovascular and all-cause mortality. Individuals with both high Lp(a) and low HDL-C are at particularly high risk. These findings suggest that combined assessment of these two lipid markers may improve risk stratification for CV outcomes, beyond their individual effects.
Keyword: Lipoprotein(a), HDL cholesterol

MOP2-3-2 *3. Clinical Lipidology & Genetics*

Discordance between apolipoprotein B and low-density lipoprotein cholesterol refines cardiovascular risk stratification in metabolically healthy obesity

김지아*, 박민승, 조은혜, 우희연, 박효순, 권민정

강북삼성병원 진단검사의학과

Objectives: Metabolically healthy obesity (MHO) is an intermediate-risk phenotype with heterogeneous prognosis. Since low-density lipoprotein cholesterol (LDL-C) often fails to reflect the true atherogenic burden, we investigated the association between apoB-LDL-C discordance and coronary artery calcium (CAC) progression across metabolic phenotypes.
Methods: We analyzed 15,738 Korean adults who underwent serial computed tomography for CAC assessment (median age 37 years; 86.3% men). MHO was defined as body mass index ≥25 kg/m^2 in the absence of any metabolic syndrome components. Discordance was quantified using residuals from a linear regression of apoB on LDL-C and categorized as discordant-high (>75th percentile), concordant (25th-75th percentiles), or discordant-low (<25th percentile).
Results: During a median follow-up of six (interquartile range, 4-9) years, 3,072 participants had CAC progression. MHO was associated with higher CAC progression risk than metabolically healthy lean (MHL) individuals (odds ratio [OR], 1.26; 95% Confidence Interval [CI], 1.03-1.53). Stratified analysis demonstrated that discordance significantly discriminated risk within metabolically healthy groups. MHO with high discordance had significantly increased risk (OR, 1.74; 95% CI, 1.13-2.66), comparable to metabolically unhealthy phenotypes. Conversely, MHO with low discordance showed no excess risk (OR, 1.03; 95% CI, 0.71-1.48) compared with the MHL concordant reference group. High discordance also identified elevated risk in MHL (OR, 1.73; 95% CI, 1.02-2.92) and transition to metabolically unhealthiness in MHO participants (OR, 1.62; 95% CI, 1.12-2.35).
Conclusions: ApoB-LDL-C discordance refines cardiovascular risk stratification within metabolically healthy phenotypes. High discordance identifies MHO individuals with residual atherosclerosis risk and increased metabolic deterioration likelihood, highlighting the need for earlier preventive strategies in these ostensibly healthy groups.
Keyword: Obesity, Metabolically benign, Apolipoproteins B, Cholesterol, LDL, Vascular calcification

MOP2-3-3 *3. Clinical Lipidology & Genetics*

Long-term trends and widening regional variation in mortality from diabetes in Korea, 1998-2023: potential impact of the COVID-19 pandemic

조민성[1*], 하경화[2,3], 이호규[2,3], 김현창[2,3]

[1]연세대학교 일반대학원 보건학과, [2]연세대학교 의과대학 예방의학과, [3]연세대학교 디지털헬스연구원

Objectives: Although diabetes mortality has declined nationally in Korea, it remains unclear whether these improvements were equitably distributed across regions during the COVID-19 period. Therefore, this study examined long-term temporal trends and regional variation in mortality from diabetes in Korea with specific attention to changes during this period.
Methods: Using nationwide cause-of-death microdata from Statistics Korea spanning 1998-2023, we identified deaths with diabetes as those with an underlying cause coded as E10-E14. Deaths with missing age information were excluded from age standardization. Annual crude and age- and sex-standardized death rates (ASDRs) were calculated at the national and municipal levels using the 2005 Korean population as the standard. Regional variation was assessed using municipal-level distributions of ASDRs, focusing on absolute variation (standard deviation, SD) and relative variation (coefficient of variation, CV).
Results: ASDRs declined from 27.2 to 8.0 per 100,000 populations between 1998 and 2019 but rebounded to 9.4 in 2022 and 9.0 in 2023. Despite this overall decline in mortality, relative regional variation increased markedly over the same period, with the CV rising from 0.26 to 0.43, even as the municipal mean and SD decreased. This elevated relative variation persisted during the COVID-19 pandemic, with the CV remaining at 0.41 through 2023. Over the entire study period, the CV showed an overall upward trend and remained at an elevated level in recent years, suggesting that the widened regional disparities may have become entrenched.
Conclusions: ASDR from diabetes declined substantially over time but partly rebounded during the COVID-19 period, accompanied by persistently widened regional variation. Strengthening diabetes care and expanding targeted support for high-burden municipalities are needed to prevent further increases in regional variation in mortality from diabetes.
Keyword: COVID-19 pandemic, Diabetes mellitus, Geographic variation, Korea

MOP2-3-4 *3. Clinical Lipidology & Genetics*

Placental response to gestational diabetes mellitus suggests compartment-specific fibrinolytic regulation

Yosub Kim[1*], Hyeonji Mun[1], Jiwoo Jung[1], Jueun Park[1], Jiyoon Park[1], Joo Young Kweon[1], Myeong Ryeol Choi[1], Young Joo Lee[2], Yong Joo Ahn[1]

[1]POSTECH, [2]경희의료원 산부인과

Objectives: Gestational diabetes mellitus (GDM), a common complication of pregnancy, frequently accompanies metabolic alterations that are associated with structural and vascular abnormalities in the placenta. Previous studies have reported increased prevalence of delayed villous maturation, syncytial knot formation, and fibrin deposition in GDM placentas, suggesting a state of vascular malperfusion that could further compromise maternal-fetal exchange. In this study, we examined HIF-1α expression as an indicator of oxygen-responsive adaptation and PAI-1 as a key regulator of fibrinolytic balance. In addition, we assessed whether these responses differ between the amnion and placental bed, reflecting compartment-specific adaptation.
Methods: Placental tissues were obtained from pregnancies with GDM and normoglycemic controls at Kyung-Hee Medical Center. Tissue segments were collected from the fetal membrane region and the surface on the opposite side, corresponding to the amnion and placental bed, respectively. mRNA and protein levels were assessed by qPCR and Western blot, respectively.
Results: HIF-1α expression did not differ between GDM and normoglycemic placentas at either the mRNA or protein level. However, PAI-1 protein expression was significantly decreased in the amnion of GDM placentas, while no significant difference was observed in the placental bed. qPCR analysis revealed compartment-specific transcriptional trends across inflammatory and vascular-related genes; however, these changes did not reach statistical significance.
Conclusions: HIF-1α expression remained unaltered in GDM placentas, indicating preserved hypoxia-responsive signaling at term. In contrast, PAI-1 protein expression was significantly reduced in the amnion but not in the placental bed, revealing compartment-specific regulation. Notably, PAI-1 transcript levels in the amnion exhibited an upward trend despite reduced protein abundance, implying potential post-transcriptional modulation of fibrinolytic pathways at the fetal interface. Together, these findings point toward spatially distinct homeostatic regulation within the placenta in response to metabolic stress.
Keyword: Gestational diabetes mellitus, Placenta, Fibrin

MOP2-3-5 *3. Clinical Lipidology & Genetics*

Cumulative blood pressure burden above optimal level and risk of cardiovascular disease in patients with diabetes

Dasom Son[1,2,3*], Kyoung Hwa Ha[1,2], Hyeok-Hee Lee[4], Hyeon Chang Kim[1,2], Minyoung Lee[5], Hokyou Lee[1,2]

[1]Department of Preventive Medicine, Yonsei University College of Medicine, Seoul, [2]Yonsei Institute for Digital Health, Yonsei University, Seoul, [3]Department of Public Health, Yonsei University Graduate School, Seoul, Republic of Korea, [4]Richard A. and Susan F. Smith Center for Outcomes Research in Cardiology, Beth Israel Deaconess Medical Center, Harvard Medical School, Boston, MA, USA, [5]Department of Internal Medicine, Yonsei University College of Medicine, Seoul, Republic of Korea

Objectives: The 2025 AHA/ACC BP Guidelines recommend BP targets <130/80 mm Hg, with encouragement to <120 mm Hg SBP, for adults with diabetes and hypertension. We examined whether greater cumulative BP exposure above optimal levels is associated with higher CVD risk.
Methods: Among adults aged ≥20 years who participated in the 2009-2013 Korean National Health Insurance general health screening, we included those with diabetes for ≥5 years, ≥3 BP-measuring visits, and no prior CVD. Cumulative BP burden was calculated as the area under the interpolated BP curve above optimal level (SBP ≥120 mm Hg; DBP ≥80 mm Hg), annualized by dividing by the total exposure years. Outcomes were incident total CVD (composite of atherosclerotic CVD [ASCVD] or heart failure [HF]), ASCVD (myocardial infarction, fatal coronary heart disease, or fatal/nonfatal stroke), and HF.
Results: Among the 499,562 participants, the mean±SD age was 60.8±10.2 years, and 40.0% were women. Over a median follow-up of 12.6 years, 92,505 CVD events, 53,779 ASCVD events, and 53,746 HF events occurred. Cumulative incidence and multivariable-adjusted HRs of all outcomes increased monotonically toward higher cumulative SBP and DBP burden groups. Each 10 mm Hg higher SBP above 120 mm Hg and 5 mm Hg higher DBP above 80 mm Hg was associated with 11% (HR, 1.11; 95% CI, 1.10-1.11) and 10% (HR, 1.10; 95% CI, 1.09-1.11) higher hazard of total CVD; 14% (HR, 1.14; 95% CI, 1.13-1.15) and 15% (HR, 1.15; 95% CI, 1.13-1.16) higher hazard of ASCVD; and 8% (HR, 1.08; 95% CI, 1.07-1.10) and 6% (HR, 1.05; 95% CI, 1.04-1.07) higher hazard of HF in a dose-dependent manner.
Conclusions: In patients with diabetes, a higher cumulative BP burden was associated with an increased risk of CVD, highlighting the importance of sustained maintenance of optimal blood pressure below 120/80 mm Hg for the primary prevention of CVD in diabetes.
Keyword: Diabetes, Cardiovascular disease, BP burden, Primary prevention

MOP2-3-6 *3. Clinical Lipidology & Genetics*

Optimal threshold for lipoprotein(a) for cardiovascular event prevention in Korean patients

임수빈[1*], 주형준[2]

[1]이대서울병원 순환기/심장내과, [2]고대안암병원 순환기/심장내과

Objectives: Lipoprotein(a), or Lp(a), is a well-known predictor of adverse cardiovascular outcome, with sixfold higher atherogenicity compared with low-density lipoprotein cholesterol. While international guidelines suggest >30 or 50 mg/dL as high-risk, the evidence has been primarily based on Western studies, and the optimal cutoff in Asian populations is uncertain.
Methods: A total of 24,892 patients with Lp(a) measurements between January 2019 and July 2025 at tertiary centers in South Korea were included in the study. For derivation of optimal Lp(a) cutoff, receiver-operator curve (ROC) analysis and categorization into quartiles and deciles were used. The primary outcome was the incidence of major adverse cardiovascular events (MACE), a composite of cardiovascular death, new-onset MI, stroke, or coronary revascularization.
Results: The ROC analysis yielded 15mg/dL as the optimal cutoff for Lp(a) in predicting MACE (AUC 0.55, 95% CI 0.53-0.57). Patients with higher Lp(a) had overall higher prevalent comorbidities such as hypertension, dyslipidemia and chronic kidney disease. High Lp(a) showed a higher incidence of MACE compared with low Lp(a) (4.5% vs. 3.1%; HR 1.44; 95% CI 1.236-1.63, p
Conclusions: Using a cutoff of 15mg/dL for Lp(a) better predicted adverse cardiovascular outcomes compared with previously suggested 30 or 50 mg/dL. A differential approach for selection of patients at high risk of cardiovascular events in Asian population may be clinically useful, with consolidation from future studies.
Keyword: Lipoprotein(a), MACE, Electronic health records

MOP2-4-1 *4. Clinical Vascular Disease & Nutrition*

Serum phosphate and the progression of arterial stiffness in CKD: from the KNOW-CKD study

김유정[1*], 이주영[1], 허다슬[2], 양지현[1], 이규백[1], 오국환[3], 현영율[1]

[1]성균관의대 강북삼성병원 신장내과, [2]한림의대 강동성심병원 신장내과, [3]서울의대 서울대병원 신장내과

Objectives: Serum phosphate is a well-known risk factor for cardiovascular events and high mortality in the general population and in patients with CKD (chronic kidney disease). Both hyperphosphatemia and increased arterial stiffness are prevalent in CKD. Arterial stiffness is one of the suggested mechanisms linking phosphate and poor prognosis. Previous studies have shown that high serum phosphate is associated with increased arterial stiffness. However, little is known about the relationship between serum phosphate and arterial stiffness progression. The purpose of this study is to examine the relationship between serum phosphate and the progression of arterial stiffness in predialysis CKD patients.
Methods: This study analyzed 1,036 participants from the KNOW-CKD cohort. The arterial stiffness was assessed by measuring brachial-ankle pulse wave velocity (baPWV). We measured baPWV at baseline and 4 years after enrolment and the mean of the right and left baPWV (mPWV) was used as a marker of arterial stiffness. The progression of arterial stiffness was defined as 10% or more increase of mPWV at 4 years compared with baseline.
Results: The arterial stiffness progression was more common in the higher serum phosphate quartile groups (18.1%, 26.5%, 22.9%, and 32.5% for the 1st to 4th quartiles of serum phosphate group, respectively, P for trend=0.008). In multivariate logistic regression (Model 3), compared with the 1st quartile group, the adjusted odds ratios (95% confidence interval) for arterial stiffness progression were 1.59 (1.07-2.36), 1.67 (1.10-2.54) and 1.75 (1.16-2.64) for the 2nd to 4th quartile of serum phosphate group.
Conclusions: High serum phosphate is independently associated with the progression of arterial stiffness in CKD.
Keyword: Phosphate, Arterial stiffness, Chronic kidney disease

MOP2-4-2 *4. Clinical Vascular Disease & Nutrition*

Long-term temporal patterns and regional persistence of district-level obesity, hypertension, and diabetes in Korea, 2008-2024

Jeonga Park[1*], Kyoung Hwa Ha[2,3], Hansol Choi[2,3], Hokyou Lee[2,3], Hyeon Chang Kim[2,3]

[1]Department of Public Health, Yonsei University Graduate School, [2]Department of Preventive Medicine, Yonsei University College of Medicine, [3]Yonsei Institute of Digital Health, Yonsei University, Republic of Korea

Objectives: This study aimed to assess 17-year temporal patterns and trajectories in district-level prevalence of obesity, hypertension, and diabetes in Korea and to determine whether these burdens persist over time in specific districts.
Methods: Using the Korea Community Health Survey (2008-2024), we estimated district-level prevalence of obesity, hypertension, and diabetes across 250 districts. For each indicator, districts were categorized into 3 prevalence groups (top 25%, middle 50%, and bottom 25%). Long-term regional persistence was assessed using intraclass correlation coefficients (ICCs) from mixed-effects models and weighted kappa statistics for agreement in district group rankings over time. Group transitions were evaluated using 3-year mean prevalence values for 2008-2010 (time 1), 2015-2017 (time 2), and 2022-2024 (time 3). Pairwise district-level correlations among obesity, hypertension, and diabetes were also assessed to evaluate co-occurrence of these burdens across districts.
Results: Over the study period, ICCs were highest for hypertension (0.4), followed by diabetes (0.2) and obesity (0.1), indicating the greatest regional persistence for hypertension and low-to-moderate persistence overall. Weighted kappa statistics showed a broadly similar pattern for hypertension and diabetes, whereas weighted kappa for obesity increased from 0.3 to 0.5, suggesting increasing persistence of regional disparities in recent years. Group transition analyses showed that Yeoncheon-gun, Pocheon-si, and Dongducheon-si (Gyeonggi Province) and Jeongseon-gun (Gangwon Province) consistently remained in the top 25% group, while Songpa-gu (Seoul) and Bundang-gu (Seongnam-si, Gyeonggi Province) consistently remained in the bottom 25% group. In 2024, all pairwise correlations among obesity, hypertension, and diabetes were 0.5.
Conclusions: Long-term regional persistence was low to moderate overall, with the strongest persistence for hypertension and increasing recent persistence for obesity. These findings support region-specific strategies for chronic disease prevention and management.
Keyword: Diabetes, Hypertension, Obesity, Regional health inequality

MOP2-4-3 *4. Clinical Vascular Disease & Nutrition*

Socioeconomic disparities in stroke incidence and case fatality in Korea, 2010-2023

이재용[*], 김현창, 이호규

연세대학교 의과대학 예방의학과

Objectives: While overall stroke outcomes have improved, socioeconomic inequalities remain a critical public health challenge. This study aimed to investigate trends in stroke incidence and 1-year case fatality rates across different socioeconomic subgroups in South Korea.
Methods: Using the National Health Insurance Service database, we identified incident stroke cases and calculated sex- and age-standardized incidence rates and 1-year case fatality rates from 2010 to 2023. Socioeconomic status was categorized by income quartiles and residential urbanization (metropolitan, urban, and rural). Sex- and age-standardized rates were calculated through direct standardization using the 2023 insured population as the reference.
Results: Across all socioeconomic subgroups, the standardized stroke incidence rates showed a continuous decline from 2010 to 2023, while the 1-year case fatality rates decreased until 2019, rose between 2020 and 2022, and declined again in 2023. Across all income groups, lower-income individuals consistently exhibited higher incidence and case fatality rates, with the gap between groups persisting over time (Figure A and B). Regarding urbanization, rural areas showed a higher incidence compared to metropolitan and urban areas (Figure C). However, 1-year case fatality rates did not differ significantly by urbanization and followed a similar temporal trend (Figure D).
Conclusions: Significant disparities in stroke incidence and fatality persist across income levels, with regional differences primarily observed in incidence rates. Future efforts should focus on addressing these socioeconomic barriers to bridge the gap in stroke incidence and fatality.
Keyword: Incidence, Mortality, Socioeconomic disparities in health, Stroke

MOP2-4-4 *4. Clinical Vascular Disease & Nutrition*

Single-pill combination vs separate antihypertensive and statin medications and cardiovascular outcomes in young adults

Kyoung Hwa Ha[1,2*], Dae Young Cheon[3], Dasom Son[1], Hansol Choi[1,2], Hyeon Chang Kim[1,2], Hokyou Lee[1,2]

[1]Department of Preventive Medicine, Yonsei University College of Medicine,
[2]Yonsei Institute for Digital Health, Yonsei University,
[3]Division of Cardiology, Department of Internal Medicine, Hallym University Dongtan Sacred Heart Hospital, Republic of Korea

Objectives: Despite established clinical benefits, blood pressure- and lipid-lowering medications remain underutilized in young adults with high blood pressure and cholesterol. Single-pill combination (SPC) therapy may reduce this treatment gap by simplifying regimen and improving adherence. We evaluated the comparative effectiveness of an SPC vs separate-pill antihypertensive and statin therapy for long-term cardiovascular disease (CVD) outcomes in young adults.
Methods: Using the Korean National Health Insurance Service database, we conducted a retrospective cohort study with a new-user, active-comparator design among young adults aged 20-44 years without prior CVD. Participants who initiated angiotensin receptor blocker (ARB)/statin combination therapy between 2013-2018 as either an SPC or two separate pills were included and followed through 2023. Baseline covariates were balanced using inverse probability of treatment weighting. The primary outcome was incident CVD, defined as a composite of hospitalization for myocardial infarction, stroke, or heart failure, or cardiovascular death.
Results: Among 65,404 participants initiating ARB/statin combination therapy (median age, 41 years; 19.8% women), 11,665 initiated SPC and 53,739 initiated two separate pills. Over a median follow-up of 7.9 years, 3,333 CVD events occurred. All events occurred before 65 years of age and therefore would be considered premature. SPC initiation was associated with a 26% lower risk of incident CVD compared with initiation as separate pills therapy (hazard ratio [HR], 0.74; 95% CI, 0.71-0.78). For the individual components of the primary outcome, SPC initiation was associated with a lower risk of myocardial infarction (HR, 0.74; 95% CI, 0.66-0.83), stroke (HR, 0.67; 95% CI, 0.62-0.74), and heart failure (HR, 0.80; 95% CI, 0.74-0.86), but not cardiovascular death (HR, 1.07; 95% CI, 0.90-1.28).
Conclusions: Among young adults, initiation of SPC vs separate-pill antihypertensive and statin therapy was associated with lower risks of premature cardiovascular events.
Keyword: Angiotensin receptor blockers, Antihypertensive agents, Cardiovascular diseases, Single-pill combination, Statins

MOP2-4-5 *5. Others*

Socioeconomic disparities in adherence to 24-hour movement guideline and PREVENT-predicted cardiovascular risk: a national survey analysis

김별*, 김현정, 최태구, 김선정, 송현수, 제세영

서울시립대학교 스포츠과학과

Objectives: The 24-hour movement guidelines integrate physical activity, sedentary behavior, and sleep as a unified behavioral framework for cardiovascular prevention. However, the relationships of socioeconomic status (SES) with guideline adherence and with contemporary predicted cardiovascular risk have not been well characterized. We examined the associations of household income and adherence to the 24-hour movement behavior guidelines with ten-year predicted risks of total cardiovascular disease (CVD) and atherosclerotic CVD (ASCVD) among adults.
Methods: We analyzed 23,410 adults aged 19 years or older using the 2018 - 2023 Korea National Health and Nutrition Examination Survey. Adherence to the 24-hour movement guidelines was quantified by the number of components met: leisure-time moderate to vigorous physical activity of at least 600 MET-minutes per week, sedentary time of 8 hours per day or less, and sleep duration of 7 to 9 hours per day. SES was assessed using household income quartiles. Ten-year predicted risks of total cardiovascular disease (CVD) and atherosclerotic CVD (ASCVD) were estimated using the 2023 American Heart Association PREVENT equations, after excluding participants with a prior history of CVD.
Results: Only 5.7% of participants met all three guideline components. Adults in the lowest income quartile were substantially less likely to meet all components than those in the highest quartile (2.9% vs 7.1%, $p<0.001$). Predicted 10-year risks of both total CVD and ASCVD were higher in lower-income groups ($p<0.001$). Across income strata, greater adherence to the movement guidelines was associated with lower predicted cardiovascular risk ($p<0.001$), with borderline evidence of effect modification for total CVD risk (interaction $p=0.052$).
Conclusions: Lower SES was associated with poorer adherence to integrated 24-hour movement behaviors and higher predicted cardiovascular risk. Greater adherence was associated with a more favorable predicted risk profile across all SES groups, with a possible tendency toward stronger associations in lower-income adults.
Keyword: 24-hour movement guidelines, Socioeconomic status, Physical activity, Sedentary behavior, Sleep

SoLA 2026

한국지질·동맥경화학회 춘계학술대회

2026 Spring Congress on
Lipid and Atherosclerosis of KSoLA

SoLA 2026

한국지질·동맥경화학회 춘계학술대회

2026 Spring Congress on
Lipid and Atherosclerosis of KSoLA

E-Posters

E-Posters

☞ 일시: 2026년 4월 3일(금) 12:00-4일(토) 17:00

☞ 장소: 포이어(로비) 내 발표구역 A-D

SoLA 2026

한국지질·동맥경화학회 춘계학술대회

2026 Spring Congress on
Lipid and Atherosclerosis of KSoLA

PE01

1. Basic Science of Lipids & Lipoproteins

SGLT-2 inhibition improves hepatic steatosis and modulates lipid metabolism in diet-induced obese mice

오다희*, 전지은, 황유철, 안규정, 정호연, 정인경

강동경희대학교병원 내분비내과

Objectives: Metabolic dysfunction-associated steatotic liver disease (MASLD) is characterized by excessive hepatic lipid accumulation driven by obesity and dysregulated lipid metabolism. Beyond glucose lowering, SGLT-2 inhibitors exert systemic metabolic effects, yet their impact on hepatic lipid handling under high-fat diet conditions remains incompletely understood. This study investigated whether SGLT-2 inhibition improves hepatic steatosis and modulates lipid metabolic pathways in diet-induced obese (DIO) mice.
Methods: Male C57BL/6J mice were fed a 60% high-fat diet for 8 weeks to induce obesity and metabolic dysfunction, followed by 8 weeks of treatment with vehicle or enavogliflozin (SGLT-2 inhibitor). Normal diet-fed mice served as controls. Body weight and casual blood glucose levels were monitored. Plasma triglyceride (TG), total cholesterol (TC), aspartate aminotransferase (AST), and alanine aminotransferase (ALT) levels were measured. Hepatic steatosis was evaluated using hematoxylin and eosin (H&E) staining, and disease activity was assessed by NAFLD activity score (NAS). Hepatic mRNA expression of genes involved in lipid synthesis (SREBP-1c, FAS, ACC) and fatty acid oxidation (PPARα, CPT1α) was analyzed by real-time PCR.
Results: SGLT-2 inhibitor treatment significantly reduced body weight and casual glucose levels compared with vehicle-treated DIO mice. Plasma TG and TC levels were markedly decreased, accompanied by significant reductions in AST and ALT. Histological analysis demonstrated substantial attenuation of hepatic lipid accumulation, with a significant reduction in NAS. At the molecular level, lipogenic gene expression was downregulated, whereas genes associated with fatty acid oxidation were upregulated, indicating a shift toward enhanced hepatic lipid utilization.
Conclusions: SGLT-2 inhibition improves systemic lipid profiles and attenuates hepatic steatosis in diet-induced obesity. These effects are associated with suppression of hepatic lipid synthesis and enhancement of fatty acid oxidation, supporting a beneficial role of SGLT-2 inhibitors in regulating lipid metabolism under metabolic dysfunction conditions.
Keyword: MASLD, SGLT-2 inhibitor

PE02

2. Basic Science of Atherosclerosis

Patient-specific hemodynamic prediction of coronary risks post-PCI using computational fluid dynamics

Ayeon Hwang[1*], Jeong-Hyun Kim[2], Nari Kim[1]

[1]인제대학교 생리학, [2]인제대학교 심혈관대사질환센터

Objectives: Percutaneous coronary intervention (PCI) is a cornerstone in treating coronary artery disease; however, post-procedural complications such as restenosis and intimal hyperplasia remain significant challenges. These adverse outcomes are intimately linked to hemodynamic disturbances induced by stent implantation. This study aims to evaluate patient-specific coronary hemodynamics before and after PCI using computational fluid dynamics (CFD) and to identify hemodynamic risk factors associated with stent-induced vascular remodeling to improve prognostic accuracy in a non-invasive manner.
Methods: Three-dimensional coronary artery geometries, including the implanted stent, were reconstructed from patient-specific computed tomography (CT) images. To ensure high fidelity, the actual stent configuration and its apposition status within the stenosed region were precisely modeled to reflect the real-world post-procedural vascular environment. Transient CFD simulations were conducted under physiological pulsatile flow conditions using a non-Newtonian blood viscosity model. Hemodynamic parameters, including time-averaged wall shear stress (TAWSS), oscillatory shear index (OSI), and relative residence time (RRT), were calculated to quantify disturbed flow patterns at the strut level.
Results: Compared with the pre-PCI model, the post-PCI model demonstrated significant changes in local hemodynamic parameters near the stented region. Areas exposed to low TAWSS and high OSI increased downstream of the stent struts, indicating an elevated risk of plaque progression and restenosis. In addition, RRT values markedly increased in areas with flow recirculation, suggesting prolonged residence of pro-inflammatory elements. These findings demonstrate that the mechanical microenvironment altered by actual stent geometry significantly contributes to adverse vascular remodeling.
Conclusions: Patient-specific CFD analysis incorporating actual stent geometry provides a robust, non-invasive framework for evaluating post-PCI hemodynamic shifts. This approach may assist in optimizing treatment strategies and individualized procedural planning by enabling high-resolution quantitative assessment of vascular physiology and long-term risk stratification.
Keyword: Computational fluid dynamics (CFD), Coronary artery disease, Stent, Wall shear stress

PE03 *2. Basic Science of Atherosclerosis*

Cardioprotective mechanism of tomatidine in pathological cardiac hypertrophy

최승학[1,2*], 한진[1,2], 김형규[1,2], 김재호[3], 윤창신[2], Pham Trong Kha[2], Garcia Maria Victoria Faith[2], Jessa Flores[1], 설보민[1], 염재범[1]

[1]인제대학교 의과대학 생리학교실, [2]인제대학교 의과대학 심혈관대사질환센터, [3]부산대학교 의과대학 생리학

Objectives: Pathological cardiac hypertrophy is a maladaptive response to sustained β-adrenergic stimulation and is characterized by oxidative stress, mitochondrial dysfunction, calcium mishandling, and electrical remodeling, ultimately leading to heart failure. This study aimed to evaluate the cardioprotective effects of tomatidine in an isoproterenol (ISO)-induced cardiac hypertrophy model and to elucidate its underlying cellular and electrophysiological mechanisms.
Methods: Cardiac hypertrophy was induced in C57BL/6 mice by chronic ISO administration, followed by tomatidine treatment at multiple doses. Cardiac structure and function were assessed by echocardiography, heart weight indices, histological analyses, and electrophysiological recordings. Complementary in vitro experiments were performed in human AC16 cardiomyocytes to evaluate cell viability, reactive oxygen species (ROS) production, mitochondrial function, and intracellular calcium handling.
Results: ISO administration induced pronounced cardiac hypertrophy, myocardial fibrosis, impaired systolic and diastolic function, prolonged action potential duration, and increased L-type Ca^{2+} current density. Tomatidine significantly attenuated these pathological changes in a dose-dependent manner, restoring cardiac performance and normalizing electrophysiological remodeling without affecting basal channel activity. In AC16 cardiomyocytes, tomatidine suppressed ISO-induced ROS accumulation, preserved mitochondrial integrity and ATP production, stabilized calcium homeostasis, and reduced hypertrophic marker expression.
Conclusions: Tomatidine exerts cardioprotective effects against ISO-induced cardiac hypertrophy by mitigating oxidative stress, preserving mitochondrial function, and normalizing calcium and electrical remodeling. These findings identify tomatidine as a potential therapeutic agent for preventing pathological cardiac remodeling and progression to heart failure.
Keyword: Cardiac hypertrophy, Tomatidine, Reactive oxygen species, Mitochondria

PE04 *3. Clinical Lipidology & Genetics*

Improving prediction of suboptimal medication adherence in dyslipidemia using generative data augmentation: a KNHANES-based machine learning study

JiHeon Kim[1*], Jihye Shin[2], Chaeyeong Yi[2], Sangyong Jo[3], Hyeon Ji Kim[1], Bongjo Kim[2], Minkook Son[2]

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

Objectives: Dyslipidemia is a major modifiable risk factor for atherosclerotic cardiovascular disease. Despite the availability of highly effective lipid-lowering therapies, suboptimal medication adherence remains a critical barrier to achieving target lipid levels. This study aimed to develop a predictive model using demographic and clinical data to identify individuals at high risk of suboptimal adherence.
Methods: Data was obtained from the Korean National Health and Nutrition Examination Survey (KNHANES) from 2010 to 2021 (N=6,741). Medication adherence was assessed using a self-reported questionnaire, defining daily use as adherent and all other frequencies as suboptimally adherent. The dataset was split into training and test sets (8:2). Five machine learning algorithms—Decision Tree, Support Vector Machine, Extreme Gradient Boosting, Random Forest, and Logistic Regression—were trained using 28 features. To address class imbalance, Synthetic Minority Over-sampling Technique (SMOTE) and a hybrid generative framework combining Generative Pre-trained Transformer 5.2 (GPT-5.2), DistilGPT-2, and Conditional Tabular Generative Adversarial Network (CTGAN) were applied. Oversampling ratios from 1- to 10-fold were evaluated. Model performance was assessed on an independent test set (N=1,349) using the area under the receiver operating characteristic curve (AUROC), sensitivity, specificity, and positive predictive value (PPV).
Results: The dataset included 6,356 adherent (94.3%) and 385 suboptimally adherent individuals (5.7%). In the original dataset, the Random Forest model achieved the best performance (AUROC=0.7554). But SMOTE did not improve model performance. The highest performance was achieved using Random Forest with GPT-5.2-based 10-fold augmentation, yielding an AUROC of 0.7667 (Figure 1). This model demonstrated high sensitivity (0.9351), moderate specificity (0.4851), and low PPV (0.0990).
Conclusions: High-ratio generative augmentation using a large language model (GPT-5.2) improved predictive performance in highly imbalanced clinical data compared to conventional methods. Improved model performance may facilitate more accurate identification of patients with suboptimal adherence, ultimately leading to better patient outcomes through targeted interventions.
Keyword: Dyslipidemia, Adherence, Prediction, KNHANES, Hybrid synthetic data generation

PE05

3. Clinical Lipidology & Genetics

Dose-dependent association between excess apolipoprotein A and increased risk of myocardial infarction

배한준*, 라모니, 이재용

대구가톨릭대학교 순환기/심장내과

Objectives: Low-density lipoprotein cholesterol (LDL-C) and apolipoprotein A (apoA) are highly correlated markers of atherogenic lipoproteins. This study aimed to evaluate whether apoA—defined as the difference between observed apoA levels and those predicted by LDL-C—is associated with an increased risk of myocardial infarction (MI).
Methods: Data of population-based cohort were obtained from the Korea National Health institute of health during January 2001 to December 2020. Among 10,030 participants were enrolled. We analyzed 5,096 individuals with apoA and LDL-C levels. LDL-C levels were calculated using the Sampson equation. Cox proportional hazards models were used to estimate the risk of MI (63 events) over the follow-up period.
Results: During about 10-year follow up period, A total of 120 MI events were recorded. We observed a significant, dose-dependent association between apoA and MI risk. Individuals with high levels of measured apoA relative to their LDL-C levels showed a markedly higher hazard ratio for MI, even after adjusting for traditional cardiovascular risk factors.
Conclusions: During the follow-up period, apoA provided significant incremental predictive value for MI beyond LDL-C alone. These findings suggest that incorporating apoA into clinical practice may offer a more precise assessment of myocardial infarction risk across the entire LDL-C spectrum.
Keyword: Low-density lipoprotein cholesterol, Apolipoprotein A

PE06

3. Clinical Lipidology & Genetics

Panax ginseng callus-derived extracellular vesicles attenuate TNF-α-induced barrier disruption and inflammatory responses in Caco-2 epithelial monolayers

채현우[1]*, 정기하[1], 정은지[2], 채서연[2], 김진우[1], 이선혜[1]

[1]선문대학교 식품공학·영양학부, [2]선문대학교 응용생물과학과

Objectives: Plant derived extracellular vesicle like nanostructures may modulate epithelial barrier responses during inflammatory challenge. This study tested whether extracellular vesicles isolated from Panax ginseng callus cultures (GCEVs) attenuate TNF-α induced barrier disruption and inflammatory signaling in differentiated Caco-2 epithelial monolayers in an in vitro model.
Methods: Differentiated Caco-2 monolayers were stimulated with TNF-α (10 ng/mL) and co treated with GCEVs at 6.9×10^7, 13.8×10^7, or 27.5×10^7 particles/mL. Barrier integrity was assessed by transepithelial electrical resistance (TEER) and phenol red paracellular permeability. Tight junction related mRNA expression was measured for CLDN1, OCLN, and TJP1 (ZO-1). Inflammatory readouts included IL6, IL1B, TNF, NF-κB (p65) and COX-2 at mRNA level and NF-κB (p65) and COX-2 protein abundance by western blot. ZO-1 junctional continuity was evaluated by immunofluorescence. Cell viability was assessed by MTT. Statistics were performed in GraphPad Prism using ANOVA with significance indicated by P values.
Results: TNF-α reduced TEER and increased paracellular permeability with decreased tight junction markers and increased inflammatory markers. GCEV co treatment attenuated TEER loss and reduced permeability while restoring tight junction related expression and improving ZO-1 junctional continuity. GCEVs suppressed TNF-α induced cytokine mRNA expression and reduced NF-κB (p65) and COX-2 at mRNA and protein levels. No cytotoxicity was observed under the tested conditions.
Conclusions: GCEVs mitigated TNF-α induced epithelial barrier dysfunction and inflammatory responses in Caco-2 monolayers. These findings support biological plausibility of ginseng callus derived vesicle interactions with intestinal epithelial cells in a controlled in vitro system without inference of physiological or clinical outcomes.
Keyword: Panax ginseng callus, Extracellular vesicle, Caco-2 epithelial model, Tight junction protein, Tumor necrosis factor alpha, Nuclear factor kappa B signaling

PE07

3. Clinical Lipidology & Genetics

Triglycerides and hypertension in a Korean population: an individual-level mendelian randomization analysis

김민주*, Ximei Huang

한남대학교 식품영양학과

Objectives: Elevated triglyceride (TG) levels are consistently associated with hypertension in observational studies; however, whether this association reflects a causal relationship remains uncertain, particularly in East Asian populations. We aimed to evaluate the potential causal effect of TG levels on hypertension in a Korean population using an individual-level Mendelian randomization (MR) framework and to contrast genetically informed estimates with conventional observational associations within the same cohort.
Methods: We analyzed 2159 Korean adults (20-86 years) whose individual-level genetic and phenotypic data were obtained from a cross-sectional health check cohort. Candidate TG-associated genetic variants were identified using genome-wide association analysis and evaluated as instrumental variables (IVs). An individual-level, two-stage IV Mendelian randomization (MR) framework was applied to assess the potential effect of TGs on hypertension, alongside conventional observational analyses using logistic regression.
Results: Three candidate TG-associated single-nucleotide polymorphisms (SNPs)—rs78115082 (TRPC7), rs117867615 (TTLL1), and rs34463296 (LINC03019)—were identified and combined to construct a weighted genetic risk score (GRS). Although all the instruments met the conventional strength criteria (F statistics > 10), they explained only a modest proportion of the variance in TG levels (partial R2, 0.008-0.020). Observational analyses showed a strong positive association between TG levels and hypertension (crude odds ratio [OR]=2.12; 95% confidence interval [CI]: 1.76-2.54; adjusted OR=1.43; 95% CI: 1.16-1.75). In contrast, MR estimates based on individual SNPs and the GRS were directionally positive but statistically nonsignificant, with wide CIs crossing the null, indicating limited precision.
Conclusions: In this Korean cohort, observational analyses demonstrated a robust association between TG levels and hypertension, whereas individual-level MR provided inconclusive genetic evidence for a causal effect under the available instruments. The difference between the observational and genetic estimates is compatible with the finding that TG levels reflect broader cardiometabolic dysregulation rather than acting as an isolated causal determinant of hypertension. These findings underscore the need for larger studies with stronger, externally derived instruments to refine the causal inference in East Asian populations.
Keyword: Hypertension, Triglycerides, Mendelian randomization, Genetic risk score, Korean population

PE08

4. Clinical Vascular Disease & Nutrition

Atherosclerotic cardiovascular disease risk among cancer survivors in Korea: a population-based study using multiple risk prediction models

하창희[1*], 정다운[2], 장정윤[3]

[1]건국대학교병원 진단검사의학과, [2]서울대학교 의과대학 예방의학과, [3]건국대학교병원 방사선종양학과

Objectives: Cancer survivors are at increased risk of atherosclerotic cardiovascular disease (ASCVD) due to shared risk factors and treatment-related vascular injury. We evaluated the 10-year ASCVD risk among Korean cancer survivors compared with the general population, using a nationally representative sample.
Methods: We analyzed Korea National Health and Nutrition Examination Survey data between 2007 and 2021. Participants aged 40-79 years without prior cardiovascular disease were included. Cancer survivorship was defined by self-reported physician diagnosis. The 10-year ASCVD risk was estimated using the Pooled Cohort Equations (PCE), Korean Risk Prediction Model (KRPM) and Korean ASCVD Risk Prediction (K-CVD) models. Multivariable logistic regression estimated odds ratios (ORs) for high ASCVD risk.
Results: A total of 42,251 participants were included in the final analysis, comprising 2,207 cancer survivors and 40,044 cancer-free individuals. Except for age, with cancer survivors being older, baseline characteristics were well balanced between the two groups. Cancer survivors had significantly higher ASCVD risk estimates across all three models (all P<0.001). Median predicted risks were higher in cancer survivors across all models, at 5.33% versus 4.22% for the PCE, 5.98% versus 3.96% for the KRPM, and 1.48% versus 1.11% for the K-CVD model. After adjustment, cancer survivorship remained independently associated with higher odds of high-risk 10-year ASCVD. The adjusted ORs were 1.43 (95% confidence interval [CI] 1.28-1.60), 1.71 (95% CI 1.51-1.92), and 1.46 (95% CI 1.30-1.65) for the PCE, KRPM and K-CVD models, respectively (all P<0.001). Risk varied by cancer type, with prostate, liver and genitourinary cancer survivors exhibited higher risk compared to other cancer types.
Conclusions: Cancer survivors in Korea have a higher 10-year ASCVD risk compared to the general population, consistent across multiple risk prediction models. Our study highlights the importance of ASCVD risk screening and surveillance for cancer survivors in the Korean population.
Keyword: Cancer survivor, ASCVD risk, Population-based study, Nationwide survey, Risk prediction models

PE09 *4. Clinical Vascular Disease & Nutrition*

Trends in the prevalence of comorbidities according to hypertension status: The Korea National Health and Nutrition Examination Survey 2005-2024

Hyoeun An[1*], Hokyou Lee[2,3], Hansol Choi[2,3], Kyoung Hwa Ha[2,3], Hyeon Chang Kim[2,3]

[1]Department of Public Health, Yonsei University Graduate School, [2]Department of Preventive Medicine, Yonsei University College of Medicine, [3]Yonsei Insititute for Digital Health, Yonsei University, Republic of Korea

Objectives: As hypertension prevalence rises, there is a growing concern regarding the comorbidity burden in South Korea. While the association between hypertension and metabolic disorders is well-known, long-term secular trends in the accumulation of these conditions remain understudied. This study aimed to evaluate 20-year trends in the prevalence of metabolic comorbidities across different hypertension status groups and to analyze the shifting composition of hypertension status according to the total comorbidity burden.
Methods: We analyzed data from 79,182 participants in the Korea National Health and Nutrition Examination Survey, 3-9 (2005-2024 years). Hypertension status was categorized into hypertension, prehypertension, and normal based on the Korean Society of Hypertension guidelines. Five metabolic comorbidities were included: obesity, diabetes mellitus, dyslipidemia, chronic kidney disease, and non-alcoholic fatty liver disease. We examined prevalence trends across survey cycles and analyzed the composition of hypertension categories according to the number of comorbidities.
Results: The mean age of participants was 50.5 years, and 55.8% were female. The overall population consisted of 29.8% with hypertension, 25.9% with prehypertension, and 44.3% with normal status. From the 3rd to the 9th cycle, hypertension prevalence increased. Across all five comorbidities, the prevalence was significantly higher in the hypertension group compared to the normal group. Notably, diabetes mellitus and dyslipidemia exhibited a steeper upward trend from the 3rd to the 9th cycle within the hypertension group. Furthermore, as the number of comorbidities increased, the proportion of individuals with hypertension expanded stepwise, while the proportion of normal individuals significantly decreased.
Conclusions: Over the past two decades, the burden of metabolic comorbidities has increased alongside the rising prevalence of hypertension in Korea. These findings underscore the necessity of a paradigm shift in clinical management toward comprehensive intervention for interlinked metabolic conditions, which should be initiated at the prehypertension stage rather than focusing on blood pressure control.
Keyword: Hypertension, Secular trends, Comorbidity, Metabolic disorders

PE10 *4. Clinical Vascular Disease & Nutrition*

Changes in hypertension management among Korean young adults by employment status after expansion of national health screening: estimates from the Korea National Health and Nutrition Examination Survey, 2014-2023

Sang Gyeong Lee[1*], Jaeyong Lee[2], Dasom Son[1,2,3], Hansol Choi[3], Hyeon Chang Kim[2,3], Hokyou Lee[2,3]

[1]Department of Public Health, Yonsei University Graduate School, [2]Department of Preventive Medicine, Yonsei University College of Medicine, [3]Yonsei Institute for Digital Health, Yonsei University, Republic of Korea

Objectives: The expansion of national health screening to include younger unemployed adults in 2019 may have contributed to improvements of hypertension management among Korean adults aged 20-39 years. This study compares changes in awareness, treatment, and control rates according to prior access to health screening before and after 2019.
Methods: Nationally representative data from the 2014-2023 Korea National Health and Nutrition Examination Survey were analyzed, with participants stratified by employment status: year-round employees (≥1-year contract) and temporary or non-employees (<1-year contract, self-employed, or unemployed). Hypertension was defined as systolic blood pressure (BP) ≥140 mm Hg or diastolic BP ≥90 mm Hg, or self-reported use of antihypertensive medication. Awareness, treatment, and control were defined as the proportion, among those with hypertension, of those with a self-reported physician diagnosis among those with hypertension, those taking antihypertensive medication for ≥20 days per month, and those with BP <140/90 mm Hg, respectively. We compared rates of these indicators between the pre-period (2014-2018) and the post-period (2019-2023) using the Rao-Scott chi-squared test and quantified differences as absolute percentage-point changes.
Results: A total of 888 participants aged 20-39 years with hypertension were included in the analysis. Among temporary or non-employees, awareness increased from 16.8% in 2014-2018 to 30.7% in 2019-2023 (p=0.004; Figure), with difference of +13.9%p (95% CI: 4.4-23.4). Similarly, treatment rose by +12.7%p (95% CI: 3.8-21.5; 23.6% in 2019-2023 vs. 10.9% in 2014-2018; p=0.004) and control rose by +11.5%p (95% CI: 3.0-20.0; 20.7% in 2019-2023 vs. 9.2% in 2014-2018; p=0.006). Conversely, year-round employees showed smaller and non-significant changes, with awareness increasing by +2.5%p (95% CI: -6.3-11.4), treatment by +2.6%p (95% CI: -5.6-10.8), and control by +4.4%p (95% CI: -2.8-11.6).
Conclusions: Awareness, treatment, and control of hypertension has increased more pronounced among temporary or unemployed young adults after eligibility expansion.
Keyword: Hypertension, Management, Young adult, Health screening

PE11

4. Clinical Vascular Disease & Nutrition

Imaging biomarkers from Whole-Body CT as independent predictors of high ASCVD risk

Jahae Kim[1*], Soo Jin Lee[2], Ji Young Kim[2], Young Seo Kim[3], Kang-Ho Choi[4]

[1]전남대학교병원 핵의학과, [2]한양대학교병원 핵의학과, [3]한양대학교병원 신경과, [4]전남대학교병원 신경과

Objectives: Accurately identifying asymptomatic individuals at high risk for atherosclerotic cardiovascular disease (ASCVD) is crucial for primary prevention. However, traditional risk assessment models relying solely on clinical factors may fail to capture subclinical atherosclerosis, leading to suboptimal risk stratification. Whole-body CT, increasingly utilized for routine health screening, offers a unique opportunity to simultaneously evaluate metabolic and vascular health. This study aimed to investigate the clinical utility of
Methods: We retrospectively analyzed 298 whole-body CT scans obtained from three university hospitals. Body composition factors (visceral fat area, subcutaneous fat area, skeletal muscle area) and coronary artery calcification factors (Agatston score, number of lesions, plaque area) were quantified using automated image analysis software. The 10-year ASCVD risk was estimated for all participants based on the 2019 ACC/AHA guidelines. Multivariate logistic regression analysis was performed to identify independent imaging predictors of elevated ASCVD risk, adjusting for confounding variables.
Results: Among the body composition factors, visceral fat area (101.0±66.8 cm^2 in low-risk vs. 153.8±78.2 cm^2 in high-risk), subcutaneous fat area (163.9±84.4 cm^2 vs. 143.7±63.0 cm^2), and skeletal muscle area (156.9±45.9 cm^2 vs. 171.5±41.9 cm^2) were significantly different between the two groups. Agatston score (4.2±30.0 vs. 128.8±432.6), the number of calcified lesions (0.4±1.7 vs. 4.2±8.3), and plaque area (1.8±12.1 mm^2 vs. 41.8±117.8 mm^2) also differed significantly (all $p<0.05$). In the multivariate model, both log-transformed visceral fat area (OR 2.78, p=0.005) and log-transformed Agatston score (OR 2.23, p=0.009) were identified as significant independent predictors of high ASCVD risk.
Conclusions: Increased visceral adiposity and coronary calcification burden detectable on whole-body CT images are strongly associated with elevated estimated ASCVD risk in asymptomatic adults. These findings suggest that standardized quantification of these structural metrics could serve as valuable biomarkers, enhancing cardiovascular risk stratification beyond traditional clinical factors in routine clinical practice.
Keyword: Imaging, Biomarkers, CT, ASCVD

PE12

4. Clinical Vascular Disease & Nutrition

The role of early ezetimibe combination with atorvastatin in patients with atherosclerotic cardiovascular disease (Better Trial)

권주성[1*], 강시혁[1], 권성욱[2], 이종영[3], 서석민[4], 남창욱[5], 박경민[6], 홍영준[7], 이원영[8], 장정은[8], 채인호[1]

[1]분당서울대학교병원 순환기/심장내과, [2]인제대학교일산백병원 순환기/심장내과, [3]한림대학교성심병원 순환기/심장내과,
[4]가톨릭대학교 은평성모병원 순환기/심장내과, [5]계명대학교 동산병원 순환기/심장내과,
[6]울산대학교병원 순환기/심장내과, [7]전남대학교병원 순환기/심장내과, [8]한국오가논

Objectives: This study evaluated the efficacy and safety of early addition of ezetimibe (EZ) with atorvastatin (AS), before reaching the maximally tolerated statin dose, in very high-risk patients.
Methods: This phase 4 (NCT05761444), multicenter, randomized, open-label, active-controlled study in Korea enrolled patients at very high-risk who failed to achieve LDL-C <70 mg/dL. Patients ≥30 years were eligible if on low/moderate intensity statin monotherapy, statin-naïve, or not on a stable statin regimen for 4 weeks prior to enrollment. Patients were randomized 1:1 to EZ/AS (10/40 mg) or AS (40 mg) for 12 weeks. The primary endpoint was the percentage change in LDL-C from baseline to week 6. Secondary endpoints included the percentage change in LDL-C from baseline to week 12 and proportion of patients achieving LDL-C goal (<55) after 6 and 12 weeks. The LDL-C changes from baseline were compared using ANCOVA. Between group differences were tested using Fisher's exact test (α=0.05).
Results: Patients (N=137) received EZ/AS (n=67) or AS (n=70) once a day. Mean (SD) age was 65.0 (10.35) years, and 54.5% were aged ≥65 years. Statin history of low/moderate intensity statin monotherapy was 65.2%. Lipid-lowering efficacy was significantly greater with EZ/AS compared with AS monotherapy at week 6 (Least squares mean difference [LSMD]: -21.22; 95% CI: -29.26, -13.19; P<0.0001) and week 12 (LSMD: -15.96; 95% CI: -23.56, -8.36; P<0.0001). Significantly higher proportions of patients achieved target LDL-C levels <55 mg/dL in EZ/AS group compared with AS group at week 6 (46.2% vs. 9.0%; P<0.0001) and week 12 (55.0% vs. 15.4%; P<0.0001). Safety profiles were comparable between groups, with no new safety concerns.
Conclusions: Early combination of EZ with AS, rather than a stepwise approach, significantly improved LDL-C reduction and target achievement compared to AS monotherapy, without new safety issues.
Keyword: Ezetimibe, Atorvastatin, Early combination therapy, LDL-C reduction

PE13

4. Clinical Vascular Disease & Nutrition

Association between revised macronutrient intake ratios and metabolic syndrome

곽정현*

인제대학교 식품영양학과

Objectives: Under the 2025 revised Korean Nutrient Intake Standards, the recommended energy intake range for carbohydrates was lowered to 50-65%, the protein range was adjusted to 10-20%, and fat remained at 15-30%. This study examined the association between macronutrient energy intake ratios and metabolic syndrome using 2022-2024 KNHANES data.
Methods: Of 20,191 participants, 14,768 adults aged ≥19 were included after excluding individuals with implausible energy intake and those with missing confounder data. The association between macronutrient intake ratios (carbohydrates, fat, and protein) and the prevalence of metabolic syndrome was examined using logistic regression.
Results: In the fully adjusted model controlling for demographic, socioeconomic, and lifestyle variables, consuming more than 65% of energy from carbohydrates was associated with higher odds of metabolic syndrome (OR 1.14, 95% CI 1.10-1.30) compared with those consuming 50-65%. In contrast, fat intake above 30% of energy (OR 0.83, 95% CI 0.71-0.92) and protein intake above 20% of energy (OR 0.78, 95% CI 0.67-0.92) were associated with lower odds compared with their respective recommended energy intake ranges. These associations were more pronounced in women. In age-stratified analyses, among adults aged 19-39 years, carbohydrate intake >65% (OR 1.63, 95% CI 1.08-2.43) and fat intake 20% was associated with lower odds of metabolic syndrome (OR 0.67, 95% CI 0.49-0.92) compared with the recommended energy intake range.
Conclusions: Higher carbohydrate intake was linked to greater odds of metabolic syndrome, whereas higher fat and protein intake were linked to lower odds, with stronger associations in women and variations by age.
Keyword: KNHANSE, Metabolic syndrome, Revised macronutrient intake ratios

PE14

4. Clinical Vascular Disease & Nutrition

Circulating CHI3L1 as a potential mediator of the sarcopenia-ischemic heart disease association with sex-specific mediation patterns

Hee Ju Jun[1*], Shieon Kim[1], Dahyun Park[2], Garam Jo[2], Min-Jeong Shin[1,3]

[1]Interdisciplinary Program in Precision Public Health, Graduate School of Korea University, Seoul, [2]Institute for BioMaterials, Korea University, Seoul, [3]School of Biosystems and Biomedical Sciences, College of Health Science, Korea University, Seoul, Republic of Korea

Objectives: Sarcopenia, characterized by loss of skeletal muscle mass and strength, is increasingly prevalent worldwide. This condition is associated with chronic low-grade systemic inflammation, which may contribute to endothelial dysfunction and accelerate atherosclerotic processes, ultimately increasing the risk of ischemic heart disease (IHD). However, the biological mechanisms underlying the link between sarcopenia and IHD remain unclear. Chitinase-3-like protein 1 (CHI3L1), an inflammatory biomarker associated with cardiovascular outcomes, may represent a potential biological mediator between sarcopenia and IHD. We therefore examined the association between CHI3L1 and incident IHD among individuals with sarcopenia and evaluated whether CHI3L1 mediates this relationship, with attention to sex-specific differences.
Methods: We used data from 44,531 UK Biobanks participants who were free of major disease at baseline and had measurements of plasma CHI3L1, lean mass and grip strength. Sarcopenia was defined according to established criteria based on lean mass and grip strength. For mediation analysis, we applied a 4-way decomposition analysis. IHD was defined using ICD-10 code (I20-I25). Cox proportional hazards regression models were used to estimate hazard ratios (HRs) and 95% confidence intervals (CIs).
Results: Among individuals with sarcopenia, higher CHI3L1 levels were associated with increased IHD (HR: 1.31, 95% CI: 1.16-1.47), with stronger associations observed in women (HR: 1.40, 95% CI: 1.18-1.66) than in men (HR: 1.23, 95% CI: 1.04-1.47). In 4-way mediation analysis, CHI3L1 partially mediated the association between sarcopenia and incident IHD. Both significant pure indirect and mediated interaction effects were observed. In sex-specific analyses, a pure indirect effect was observed in both men and women, whereas mediated interaction effects were evident only in women (β =0.0369, p=0.045), suggesting a differential pattern by sex.
Conclusions: Higher CHI3L1 levels were associated with increased IHD risk among individuals with sarcopenia, and CHI3L1 partially mediated the association between sarcopenia and IHD, with sex-specific differences in mediation patterns.
Keyword: Chitinase-3-like protein 1, Sarcopenia, Ischemic heart disease, Mediation analysis, Sex-specific difference

PE15

4. Clinical Vascular Disease & Nutrition

ALDH2 rs671 is associated with lower risk of dyslipidemia in Korean ever-drinkers: 16-Year KoGES Follow-up

Jinyoung Kim*, Shieon Kim, Hee Ju Jun, Dahyun Park, Min-Jeong Shin
고려대학교 대학원

Objectives: The aldehyde dehydrogenase 2 (ALDH2) rs671 variant is a key genetic factor in alcohol metabolism. Previous studies have suggsested that rs671 is associated with various lipid traits such as triglycerides (TG), low-density lipoprotein (LDL), and high-density lipoprotein (HDL). However, its longitudinal association with incident dyslipidemia remains unclear. Therefore, we investigated the association between the rs671 variant and incident dyslipidemia during 16- years of follow-up among individuals with a history of alcohol consumption.
Methods: Using data from the Korean Genome and Epidemiology Study (KoGES) Ansan-Ansung (community-based) and CAVAS (rural-based) cohorts, we analyzed 2,471 with a history of alcohol consumption who were free of dyslipidemia at baseline. Dyslipidemia was defined as TG ≥150 mg/dL, HDL cholesterol
Results: At baseline, rs671 variant carriers (GA+AA) reported lower alcohol intake than GG carriers and also exhibited significantly lower TG levels ($p < 0.05$). Those carriers had a 20% reduced risk of dyslipidemia compared to GG carriers (HR: 0.80 [95% CI: 0.69-0.93], p=0.003). This protective effect was observed in both sexes and was stronger in females (0.77 [0.60-0.99], p=0.049) than in males (0.82 [0.68-0.98], p=0.031). Furthermore, BMI-stratified analysis revealed significant protective effects in individuals with BMI <25 and 25-30 kg/m^2, but not in those with BMI ≥30 kg/m^2.
Conclusions: The ALDH2 rs671 variant was associated with a reduced risk of dyslipidemia in alcohol drinkers, with a stronger association in females than in males. Reduced alcohol consumption among carriers, particularly in females, might partly explain these results. Moreover, the association was not observed in severe obesity, possibly due to altered aldehyde detoxification and redox regulation related to ALDH2 that might affect lipid metabolism.
Keyword: rs671, Alcohol consumption, Dyslipidemia, Gene-environment interaction

PE16

4. Clinical Vascular Disease & Nutrition

Short-term dietary quality improvement is linked to coordinated changes in metabolic health, pulmonary function, and gut-oral microbiome diversity

박지현[1*], 임수빈[1], 김민지[1], 박소연[1], 이다빈[1], 김효진[1], 황수민[1], 오수민[1], 최미옥[2,3], 안원석[4], 김오연[1,2]
[1]동아대학교 대학원 건강과학과 (임상영양전공) 식품영양학과, [2]동아대학교 식품영양학과, [3]동아대학교병원 영양과, [4]동아대학교병원 신장내과

Objectives: Obesity and metabolic syndrome are strongly influenced by dietary behaviors and are increasingly linked to impaired pulmonary function. Emerging evidence suggests that microbiome dynamics may mediate diet-related metabolic and respiratory outcomes. This study aimed to evaluate the short-term effects of a structured nutrition intervention on cardio-metabolic parameters, pulmonary function, and gut and salivary microbiome diversity.
Methods: In this 4-week intervention study, 37 participants who completed follow-up were analyzed. Anthropometric measurements, pulmonary function tests, dietary intake assessments, and fecal and salivary microbiome profiling were conducted before and after the intervention. Changes in metabolic parameters, Nutrition Quotient (NQ), and microbial diversity indices were examined, and correlation analyses were performed to explore diet-metabolism-lung interactions.
Results: The intervention significantly improved obesity-related parameters and overall diet quality, reflected by increased NQ scores. Dietary modifications included altered fat composition and increased fiber intake. Fecal microbiome composition demonstrated significant shifts in alpha diversity, including Shannon and Simpson indices, while salivary microbiome analysis revealed significant increases in species richness and diversity. Participants with BMI reduction exhibited broad metabolic improvements. Greater increases in NQ were associated with reductions in waist circumference and increases in percent predicted forced vital capacity (FVCp). Notably, metabolic improvements were more pronounced among individuals with improved diet quality, even when BMI changes were comparable. Waist circumference was positively correlated with total energy and fat intake. Animal-based fat intake was inversely associated with percent predicted FEV1, whereas plant-based fat intake was positively associated with FEV1/FVC ratio.
Conclusions: Short-term dietary intervention induces integrated improvements in metabolic health, pulmonary function, and gut-oral microbiome diversity. These findings underscore the importance of personalized nutrition strategies targeting diet quality and nutrient composition to optimize cardio-metabolic and respiratory health.
Keyword: Obesity, Metabolic syndrome, Pulmonary function, Dietary intervention, Nutrient intake, Microbiome

PE17 *4. Clinical Vascular Disease & Nutrition*

Association between psoas muscle metabolic activity and adverse clinical outcomes in early stage lung cancer

하지연[1*], 팽진철[2], 김영서[3], 이수진[4], 김지영[4], 최윤영[4], 홍일기[5], 김정민[1]

[1]서울대학교병원 신경과, [2]서울대학교병원 핵의학과, [3]한양대학교병원 신경과, [4]한양대학교병원 핵의학과, [5]경희대학교병원 핵의학과

Objectives: Metabolic activity of the psoas muscle measured by 18F-FDG PET has been previously associated with several physiological processes. However, its potential as a prognostic marker for cardiovascular events or adverse outcomes in patients with malignancy remains unknown.
Methods: This multicenter retrospective cohort study included 154 patients with stage I lung cancer who underwent surgical treatment in 2019 across three hospitals in the Seoul Metropolitan area. Patients were followed up based on available clinical records from the time of the PET scan until April 2025. Clinical characteristics and metabolic activities of various organs were compared between patients who experienced cardiovascular events or all-cause death during the follow up period and those who did not. To identify predictors of adverse clinical outcomes, we performed mixed effects and penalized logistic regression analyses adjusting for clinical covariates and hospital clustering.
Results: During a median follow up of 4.26 years, there were eight cases of cardiovascular disease or all-cause death. Patients who experienced these events demonstrated significantly higher metabolic activity of the psoas muscle. A logistic regression model adjusting for age and sex demonstrated that higher metabolic activity of the psoas muscle (OR=23.6, 95% CI 1.64 to 337.9) was associated with cardiovascular disease or all-cause death. Correlation analysis revealed that the metabolic activity of the psoas muscle correlated with hs-CRP levels ($r=0.383$, $p<0.001$).
Conclusions: Increased metabolic activity of the psoas muscle in patients with early-stage lung cancer may be associated with future adverse events. Psoas muscle metabolic activity may be linked to metabolic disorder or systemic inflammation. Further studies are needed to explore the role of psoas muscle activity as a predictive biomarker for future adverse events and its underlying mechanisms in the context of malignancy.
Keyword: Metabolic activity, Cardiovascular disease

PE18 *5. Others*

Safety of incretin-based therapies in overweight and obese adults without diabetes mellitus: a meta-analysis of randomized controlled trials

최혜덕[*], 김지혜

영남대학교 약학과

Objectives: Incretin-based therapies—glucagon-like peptide-1 receptor agonists and tirzepatide—have emerged as highly effective anti-obesity agents; however, their safety profile in individuals without diabetes remains debated. We, therefore, performed a meta-analysis to evaluate the safety of incretin-based therapies in overweight and obese adults without diabetes mellitus.
Methods: Randomized controlled trials that compared liraglutide, semaglutide, or tirzepatide with placebo were retrieved. Pooled risk ratios (RRs) for overall, serious, and prespecified adverse events were calculated using fixed- or random-effects models according to heterogeneity. Leave-one-out sensitivity analyses were conducted to determine the influence of individual studies. The protocol for this meta-analysis was registered in PROSPERO (CRD420251000593).
Results: Compared with placebo, incretin-based therapies modestly increased the risk of any adverse event (RR=1.06, 95% CI 1.04–1.07) but did not raise the risk of serious adverse events (RR=0.93, 95% CI 0.82–1.05). They increased the incidence of hypoglycemia and gallbladder disease, yet reduced cardiovascular, gastrointestinal, and hepatic events. No differences were observed for pancreatitis, psychiatric disorders, or renal events.
Conclusions: Liraglutide, semaglutide and tirzepatide appear generally safe for weight management in adults without diabetes, with no excess of serious adverse events and potential cardiovascular benefits. Nonetheless, clinicians should monitor for hypoglycemia and gallbladder disease.
Keyword: GLP-1 receptor agonist, Tirzepatide, Obesity, Safety

PE19 5. Others

Interplay with p90RSK-KLF4 in angiotensin II-induced abdominal aortic aneurysm

Thuy Le Lam Nguyen*, Nhi Thi Thao Le, Kyung-Sun Heo

College of Pharmacy, Chungnam National University, Daejeon, Republic of Korea

Objectives: The aberrant phenotypic transformation of vascular smooth muscle cells (VSMCs) is a critical contributor to aortic aneurysm (AA) formation. Angiotensin II (Ang II) plays a central role in driving abnormal VSMC function. This study aims to investigate the role of the 90 kDa ribosomal S6 kinase (p90RSK) in Ang II-stimulated cell proliferation, migration, and osteogenic switch of VSMCs.
Methods: In vitro, rat and human aortic SMCs were pretreated with FMK, a specific p90RSK kinase activation inhibitor, followed by Ang II stimulation. Molecular mechanisms were explored through western blotting, immunofluorescence staining, and qRT-PCR. In vivo, effects of FMK on AAA were evaluated in C57BL/6 mice challenged with β-aminopropionitrile and Ang II infusion (1000 ng/kg/min) for 28 days. The aortas were subjected to histological analysis, immunohistochemistry staining and Elastin Verhoeff-van Gieson (EVVG) staining.
Results: Ang II induced cell proliferation by activating ERK1/2/p90RSK/KLF4 signaling pathway, which is crucial for cell cycle progression. FMK treatment effectively downregulated Ang II-induced cell migration by inhibiting NF-κB-mediated MMP2/9 and VCAM-1 expression. In vivo, effects of FMK on AAA were evaluated in C57BL/6 mice challenged with β-aminopropionitrile and Ang II infusion for 28 days. Inhibition of p90RSK abolished Ang II-induced AA incidence. EVVG staining confirmed that FMK prevented Ang II-induced elastin degradation. Additionally, FMK reduced osteogenic transformation and inflammation, as indicated by decreased osteopontin and CD45 expression in abdominal aortic sections.
Conclusions: These findings demonstrate that modulating p90RSK-involved signaling pathways effectively inhibits Ang II-induced vascular remodeling and inflammation, emphasizing the therapeutic potential of targeting p90RSK for the treatment of AA.
Keyword: p90RSK, Aortic aneurysm, KLF4

PE20 5. Others

Divergent sex-specific associations of abdominal obesity and metabolic syndrome transition with incident COPD and pulmonary function decline in a Korean population-based cohort

박지현[1*], 임수빈[1], 김민지[1], 박소연[1], 이다빈[1], 김오연[1,2]

[1]동아대학교 대학원 건강과학과 (임상영양전공) 식품영양학과, [2]동아대학교 식품영양학과

Objectives: Abdominal obesity, a central component of metabolic syndrome (MetS), has been implicated in respiratory dysfunction. However, sex-specific longitudinal associations between waist circumference (WC) changes and pulmonary function decline remain unclear. Therefore, we investigated the sex-specific associations between changes in WC, MetS status, and pulmonary function decline in a Korean population-based cohort.
Methods: We analyzed 1,749 participants with complete follow-up from baseline to the seventh examination cycle in the Korean Genome and Epidemiology Study. Participants were categorized according to changes in MetS status over time. Incident chronic obstructive pulmonary disease (COPD) risk was evaluated using logistic regression and multivariable Cox proportional hazards models with time-dependent covariates. Correlations between longitudinal WC changes and pulmonary function indices (FEV1p and FVCp) were examined across follow-up intervals.
Results: Transition from normal to MetS status was associated with an elevated COPD risk among both former and current smokers at baseline. In men, increased WC was strongly associated with higher COPD incidence (HR 2.078; 95% CI 1.227-3.518) and was negatively correlated with all pulmonary function indices across long-term follow-up. In contrast, women exhibited a lower COPD incidence with increasing WC (HR 0.462; 95% CI 0.218-0.979), and associations between WC and pulmonary function decline varied according to baseline WC status. Significant negative correlations in women were observed only for early follow-up intervals.
Conclusions: Increased abdominal obesity, particularly in men, is associated with elevated COPD risk and accelerated pulmonary function decline. In women, the relationship appears non-linear and influenced by baseline adiposity status. These findings highlight the importance of sex-specific interpretation of metabolic risk factors in respiratory health.
Keyword: Pulmonary function, Metabolic syndrome, Waist circumference, Gender-difference

PE21

5. Others

Impact of sleep quantity and quality on chronic kidney disease

최한솔[1*], 전주은[1], 안효은[2], 하경화[1], 이호규[1,3], 김현창[1,3]

[1]연세대학교 디지털헬스연구원, [2]연세대학교 대학원 보건학과, [3]연세대학교 의과대학 예방의학과

Objectives: Both short and long sleep durations, as well as poor sleep quality, are associated with adverse health outcomes. Sleep disorders and kidney disease have a bidirectional relationship. This study aimed to investigate the impact of sleep quantity and quality on chronic kidney disease (CKD) in the Korean population.
Methods: A total of 11,244 participants (mean age 53.6 years; 60% female) were included in this cross-sectional study. Sleep behaviors were assessed using interviewer-assisted questionnaires. Sleep duration was categorized into three groups: <7 hours/day (short sleep), 7-<9 hours/day (reference) and ≥9 hours/day (long sleep). Sleep quality was classified based on the presence of obstructive sleep apnea (OSA) using the Berlin Questionnaire. CKD was defined as either a physician diagnosis or an estimated glomerular filtration rate <60 mL/min/1.73 m^2 according to the Kidney Disease Improving Global Outcomes guidelines. Multiple logistic regression models were used to examine the independent and combined associations of sleep patterns with CKD.
Results: Compared with the reference group, participants with long sleep duration and those with OSA had a 1.89-fold and 1.79-fold higher risk of CKD respectively, whereas short sleep duration showed a 0.80-fold lower risk. Furthermore, participants who had both conditions showed a markedly increased risk of CKD (adjusted odds ratio: 3.94; 95% confidence interval: 2.81-5.53).
Conclusions: These findings suggest that long sleep duration and poor sleep quality are associated with an increased risk of CKD, and this risk escalates when both factors are present. Sleep quantity and quality are easily measurable and modifiable lifestyle factors; thus, their assessment and improvement may help reduce the kidney dysfunction.
Keyword: Sleep health, Chronic kidney disease, Obstructive sleep apnea

PE22

5. Others

Mechanotransduction-driven organelle remodeling in phenotypic switching of VSMCs

김주연*, 성현, 김치대

부산대학교 의과대학 약리학교실

Objectives: Vascular smooth muscle cells (VSMCs) undergo phenotypic switching from a contractile to a synthetic state in response to mechanical stress, a process that plays a central role in vascular remodeling in hypertension and atherosclerosis. However, the precise mechanisms by which mechanical forces induce organelle remodeling and subsequent phenotypic changes remain unclear. This study aims to investigate how mechanical stretch (MS) and oxidative stress regulate mitochondrial and endoplasmic reticulum (ER) dynamics to drive phenotypic switching in VSMCs.
Methods: A10 VSMCs were subjected to cyclic mechanical stretch (3-15%) or exogenous H2O2 (30 μM, 3 h) to simulate mechanical and oxidative stress. Phenotypic markers (α-SMA, Calponin) and organelle-related proteins (MFN2, DRP1, and VDAC1) were analyzed via Western blotting. Mitochondrial morphology was quantified using MitoTracker staining followed by skeletonization analysis to assess aspect ratio and circularity. ER structures were visualized using ER-Tracker. To inhibit PDGFR-β signaling, the PDGFR-β antagonist AG1295 was used.
Results: Mechanical stretch induced a strain- and time-dependent increase in PDGFR-β expression, coupled with a significant decrease in contractile markers (α-SMA, Calponin). MS (10%, 12 h) decreased the cellular aspect ratio and increased mitochondrial circularity, indicating pronounced mitochondrial fragmentation and cellular remodeling, along with elevated cytosolic and mitochondrial ROS production. Similarly, H2O2 treatment recapitulated these effects, inducing PDGFR-β upregulation and organelle remodeling, inducing mitochondrial fission and ER disruption. Notably, inhibition of PDGFR-β with AG1295 significantly attenuated MS-induced phosphorylation of ERK1/2 and DRP1 (Ser616), and restored contractile marker expression.
Conclusions: Our findings identify ROS-driven activation of the PDGFR-β/DRP1 signaling axis as a central mechanism linking mechanical stress to organelle remodeling and VSMC phenotypic switching Therapeutic strategies aimed at limiting ROS accumulation or disrupting this signaling pathway may therefore offer novel approaches for the treatment of mechanostress-associated vascular diseases.
Keyword: Mechanotransduction, VSMCs, Organelle remodeling, PDGFR-β, ROS

PE23 5. Others

Decreased PAK4 expression in cardiomyocytes exacerbates heart failure phenotype in high-fat diet and STZ-induced diabetic mice

Nguyen Thi To Hoai[1*], Trong Kha Pham[1,2], Hyoung Kyu Kim[1], Mário Albino Sozinho Indarua[1], Luu Thi Thu Phuong[2], Han Jin[1]

[1]Department of Physiology, Inje University, Cardiovascular and Metabolic Disease Center, Smart Marine Therapeutics Center, Republic of Korea, [2]Department of Physiology and Human Biology, VNU University of Science, Vietnam National University-Hanoi, Vietnam

Objectives: To define the mechanistic and functional role of cardiomyocyte PAK4 in diabetic heart failure by determining how PAK4 deficiency drives cardiac dysfunction and remodeling, and by identifying downstream signaling pathways that couple PAK4 loss to metabolic and mitochondrial impairment in HFD+STZ diabetic mice.
Methods: Cardiomyocyte-specific PAK4 knockout mice (PAK4cKO) were generated by crossing Pak4fl/fl mice with α MHC/Myh6-Cre transgenic mice (Tg(Myh6-cre)1Jmk/J). Pak4fl/fl littermates lacking Cre were used as controls. At 8 weeks of age, mice were fed a high-fat diet (HFD) and administered streptozotocin (STZ) to induce diabetic cardiomyopathy. Cardiac structure and function were assessed by transthoracic echocardiography. Myocardial remodeling, including interstitial fibrosis, was evaluated by histological staining and morphometric analysis. Alterations in metabolic and stress-response signaling were quantified by immunoblotting, and mitochondrial bioenergetics were examined using mitochondrial functional assays.
Results: Loss of cardiomyocyte PAK4 markedly worsened the diabetic cardiomyopathy phenotype. Compared with diabetic Pak4fl/fl controls, diabetic PAK4cKO mice exhibited more severe glucose intolerance, accelerated left ventricular dilation, and pronounced myocardial fibrosis, indicating exacerbated adverse remodeling. Consistent with these structural and functional changes, PAK4-deficient hearts showed profound mitochondrial bioenergetic impairment accompanied by disrupted metabolic signaling. Importantly, PAK4 loss significantly increased mortality in the HFD+STZ diabetes model, underscoring an essential role for PAK4 in survival under diabetic stress. In parallel, markers of cytoskeletal remodeling were robustly upregulated in PAK4cKO hearts, suggesting that PAK4 functions as a key molecular rheostat that coordinates metabolic adaptation and cytoskeletal/structural remodeling to preserve cardiac integrity during diabetic injury.
Conclusions: Cardiomyocyte PAK4 is required to maintain metabolic integrity and limit adverse remodeling in diabetic cardiomyopathy. Its loss worsens cardiac dysfunction and fibrosis and increases mortality, highlighting the PAK4 axis as a potential therapeutic target for diabetic heart failure.
Keyword: PAK4, Diabetic cardiomyopathy

PE24 5. Others

The effect of using a small rice bowl for meal planning on blood sugar and weight control in type 2 diabetes patients

서주형*, 박지영, 이다빈

대동병원 급식영양팀

Objectives: This case study aimed to evaluate the effectiveness of portion size control using a small (200 mL) rice bowl on weight reduction and glycemic improvement in a patient with type 2 diabetes and severe obesity. The study examined whether a simple environmental modification combined with individualized nutrition education could improve metabolic outcomes.
Methods: A 70-year-old male patient with type 2 diabetes and severe obesity (171 cm, 88.05 kg, BMI 30.11 kg/m^2; FBS 150 mg/dL; HbA1c 6.5%) participated in individualized nutrition counseling. Baseline assessment revealed habitual overeating due to large rice portions served in a soup bowl and preference for carbohydrate-rich meals.The intervention included:placing the usual bowl with a standardized 200 mL rice bowl, education on the food exchange system, application of the "reverse eating order" method (vegetables → protein → rice), and behavioral coaching to slow eating speed and enhance satiety. Anthropometric measurements and glycemic indices were evaluated before and after the intervention.
Results: Following the intervention, body weight decreased from 88.05 kg to 85.0 kg. Fasting blood glucose improved from 150 mg/dL to 102 mg/dL, and HbA1c decreased from 6.5% to 5.8%. The patient reported improved satiety and reduced portion size without significant dietary restriction stress.
Conclusions: Using a small rice bowl as a portion control strategy is a simple, low-cost, and sustainable behavioral intervention for patients with type 2 diabetes and obesity. Environmental modification combined with structured nutrition education can effectively improve body weight and glycemic outcomes. This approach may serve as a practical clinical tool for diabetes management.
Keyword: Portion size effect, Environmental modification

SoLA 2026

한국지질·동맥경화학회 춘계학술대회

2026 Spring Congress on
Lipid and Atherosclerosis of KSoLA

Author Index

SoLA 2026

한국지질·동맥경화학회 춘계학술대회

2026 Spring Congress on
Lipid and Atherosclerosis of KSoLA

발행일 2026년 3월 30일
인쇄일 2026년 4월 3일

발행처 한국지질 · 동맥경화학회
[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 1108호
전 화 02-6953-0582
이메일 info@planbear.co.kr

ICoLA 2026

The 15th International Congress on Lipid & Atherosclerosis

https://icola.org/

September
10 (Thu)-12 (Sat), 2026
Conrad Seoul, Republic of Korea

ICoLA by KSoLA International Congress on Lipid & Atherosclerosis

KSoLA The Korean Society of Lipid and Atherosclerosis