中華民國血脂及動脈硬化學會

2025 TSLA 春季會

上醫治未病

你的血管健康有管理了嗎？

Preventive Vascular Medicine

大會手冊

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理事長的話

親愛的貴賓、講者及與會夥伴們：

歡迎大家來到美麗的台南，參加 2025年中華民國血脂及動脈硬化學會（TSLA）春季會！今年的會議將於3月22日至23日在成功大學醫學院舉行，這座擁有悠久歷史與文化底蘊的城市，不僅是臺灣的學術重鎮，更以美食與熱情著稱，期待大家能在專業交流之餘，也能感受台南的獨特魅力。

本次會議涵蓋血脂管理、動脈硬化防治及心腎代謝綜合症等關鍵議題，並規劃多場專題演講、聯合研討會及臨床實務討論，希望促進學術交流，深化跨領域合作。
此外，特別安排了戒菸競賽頒獎、武林大會及創新研究分享，讓與會者能夠在專業學習之餘，也一同見證學術新秀的成長與突破。

感謝所有講者、與會者及籌備團隊的辛勤付出，讓這場學術盛會得以圓滿舉行。
誠摯邀請大家把握這次機會，深入交流臨床經驗與最新研究，也別忘了在會後漫步台南的古街巷弄，品嚐道地小吃，享受這座城市的溫度與人情味。

期待與大家共度這場充滿知識、合作與歡樂的學術之旅！

劉秉彥 醫師

中華民國血脂及動脈硬化學會 理事長

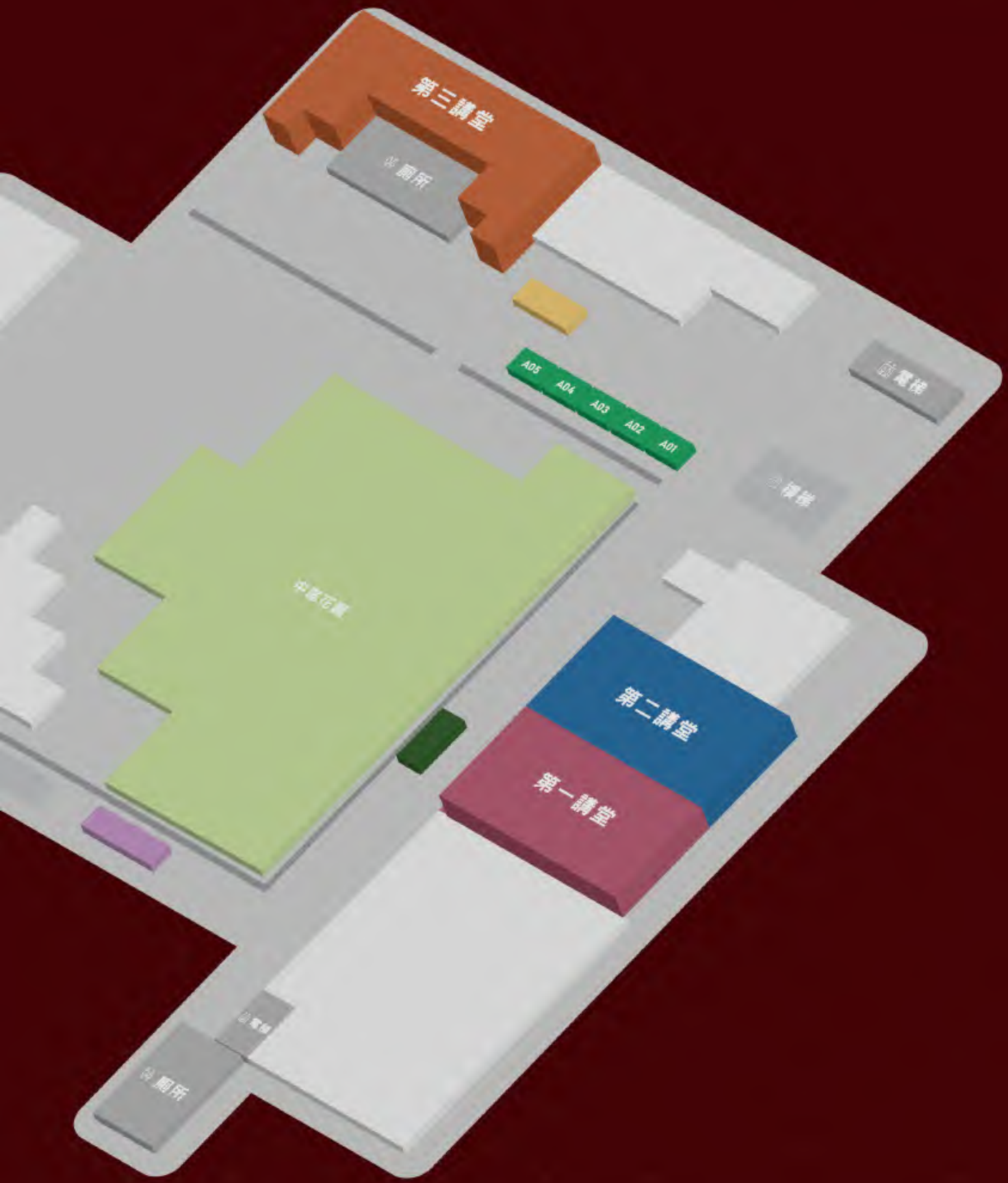
中華民國血脂及動脈硬化學會｜民國114年03月

會場配置圖

報到處　　議程總表

攤位展示區　　茶點區

A01｜賽諾菲
A02｜諾華
A03｜第一三共
A04｜安進
A05｜輝瑞


第三講堂
廁所
A05
A04
A03
A02
A01
電梯
樓梯
中庭花園
第二講堂
第一講堂
廁所

議程總表

3.22 SAT.

	第一講堂	第二講堂	第三講堂
13:30-14:50	UACR 333	心肌梗塞學會 Joint Symposium 急性心肌梗塞後的血脂管理： 從實證到實務	Cardiovascular- Kidney- Metabolic Syndrome
14:50-15:10	Coffee Break		
15:10-16:30	Clinical Application of Exsomes for Cardiovascular Health	台灣腦中風學會 Joint Symposium Innovations in Integrated Cerebrovascular Disease Management	戒菸頒獎典禮
16:30-17:30	Dinner Symposium – 1 （賽諾菲）	Dinner Symposium – 2 （安進）	Dinner Symposium –3 （輝瑞）

3.23 SUN.

	第一講堂
09:00-10:30	AI 與888
10:30-11:00	Coffee Break
10:30-11:30	2024 ASCVD 高風險患者優先競賽頒獎
11:30-12:20	Lunch Symposium – 1（台田）

	第二講堂
09:00-09:50	第三屆基礎醫學武林大會
10:30-11:00	Coffee Break
10:30-11:30	Moderate Drinking to Cardiovascular Health – A Friend? or Foe?
11:30-12:20	Lunch Symposium – 2（第一三共）

	第三講堂
09:00-11:30	預防心血管疾病運動與營養
10:30-11:00	Coffee Break

會議議程 3.22

第一講堂

UACR

TIME	TOPIC	SPEAKER	MODERATOR
13:30-13:40	Opening		
13:40-14:10	How Does UACR Matter CV and Renal Treatment and Prognosis?	徐千彝 副秘書長 北醫附醫｜心臟血管內科	吳懿哲 秘書長 馬偕醫院｜心臟血管內科
14:10-14:20	Panel Discussion	All	
14:20-14:40	DKD 高風險患者 心腎照護品質優化競賽說明	葉志凡 副秘書長 臺大醫院｜心臟內科	劉嚴文 理事 成大醫院｜心臟內科
14:40-15:10	Discussion & Closing		

Clinical Application of Exosomes for Cardiovascular Health

15:10-15:15	Opening		
15:15-15:35	Biological Functions of Exosomes And Their Impact on Atherosclerosis	張瑋婷 醫師 奇美醫院｜心臟血管內科	陳肇文 教授 北醫附醫｜心臟內科
15:35-15:55	Multifaceted Role of The Exosomes Secreted from Cardiac Lineage Cells	陳文彬 教授 臺大醫學院 實驗動物中心主任	楊鎧鍵 教授 臺大醫院｜心臟內科
15:55-16:15	It May The Right Time to Consider The Mesenchymal Stem Cell-derived Exosomes for Clinical Application for Different Disease Entities	葉漢根 教授 高雄長庚｜心臟內科	王朝永 理事 林口長庚｜心臟內科
16:15-16:30	Discussion & Closing		劉嚴文 理事 成大醫院｜心臟內科

SAT.

第二講堂

TSLA-TAMIS Joint Symposium 急性心肌梗塞後的血脂管理：從實證到實務

TIME	TOPIC	SPEAKER	MODERATOR
13:30-13:35	Opening		黃柏勳 名譽理事 臺北榮總｜心臟血管內科
13:35-13:55	心肌梗塞後血脂控制的實證醫學：最新指引與臨床研究 Evidence-Based Lipid Management After Myocardial Infarction: Latest Guidelines and Clinical Trials	黃逸群 副秘書長 林口長庚｜心臟內科	謝宜璋 常務理事 林口長庚｜心臟內科
13:55-14:15	心肌梗塞後血脂控制的臨床實務：治療策略與挑戰 Clinical Practice in Post-MI Lipid Management : Treatment Strategies and Challenges	林姝含 醫師 新光醫院｜心臟內科	王宇澄 副教授 亞大醫院｜心臟內科
14:15-14:35	血脂控制的整合性照護：跨專科團隊合作模式 Integrated Care in Lipid Management :Multi-disciplinary Team Approach	陳志維 醫師 北醫附設醫院｜心臟內科	黃偉春 副院長 屏東基督教醫院｜心臟內科
14:35-14:50	Discussion & Closing		黃群耀 理事長 北醫附設醫院｜心臟內科

TSLA & 台灣腦中風學會 Joint Symposium Innovations in Integrated Cerebrovascular Disease Management

15:10-15:15	Opening		劉秉彥 理事長 成大醫院｜心臟內科
15:15-15:35	Advancements in Precision Medicine for Stroke Management	湯頌君 教授 臺大醫院｜神經科	黃欽威 主任 成大醫院｜神經科
15:35-15:55	Cerebral Microbleeds in Cardiovascular Diseases : Pathophysiology and Personalized Therapies	蔡欣熹 醫師 臺大醫院｜神經科	湯頌君 教授 臺大醫院｜神經科
15:55-16:15	Cognitive Impairment in Cardiovascular Diseases : Screening and Management Approache	宋碧姍 醫師 成大醫院｜神經科	林維文 教授 臺中榮總｜心臟內科
16:15-16:30	Discussion & Closing		鄭建興 教授 臺大醫院｜神經科

會議議程 3.22

第三講堂

Cardiovascular-Kidney-Metabolic syndrome

TIME	TOPIC	SPEAKER	MODERATOR
13:30-13:35	Opening		劉秉彥 理事長 成大醫院｜心臟血管內科
13:35-13:55	The Definitions And Pathophysiology of Ckm Syndrome	黃金洲 副秘書長 臺北榮總｜心臟內科	林宗憲 常務理事 高醫附設中和醫院 ｜心臟血管內科
13:55-14:15	The Staging And Clinical Impacts of Ckm Syndrome	林威宏 教授 成大醫院｜一般內科	呂信邦 理事 臺北榮總｜心臟內科
14:15-14:35	The Optimal Strategies For Prevention And Management of Ckm Syndrom	王俊興 主任 臺中榮總｜新陳代謝科	許惠恒 常務監事 國衛院 副院長 ｜新陳代謝科
14:35-14:50	Discussion & Closing		林幸榮 名譽理事 臺北榮總｜心臟內科

SAT.

戒菸頒獎典禮

TIME	TOPIC	SPEAKER	MODERATOR
14:50-15:10	報到		
15:10-15:25	Effect of Smoking Cessation Treatment Service Contest on The Motivation of Physicians To Help High Cardiovascular Risk Smokers Quitting Smoking (戒菸治療服務競賽對醫師幫助心血管高風險吸菸者戒菸動機的影響)	李俊偉 醫師 心臟血管內科	葉宏一 名譽理事 馬偕醫院｜心臟血管內科
	「113年度獎勵提供心血管疾病病人戒菸服務暨獎勵基層醫療院所提供病人戒菸服務競賽」、「戒菸衛教師創意競賽」及「優良戒菸衛教師」頒獎典禮		
15:25-15:30	主辦單位致詞	葉宏一 名譽理事 馬偕醫院｜心臟血管內科	
15:30-15:35	長官致詞	國健署長官 衛生福利部國民健康署	
15:35-15:45	合辦單位致詞	各合辦學會代表 魏芳君 理事長 社團法人台灣菸害防制暨戒菸衛教學會	
15:45-16:10	「113年度獎勵提供心血管疾病病人戒菸服務暨獎勵基層醫療院所提供病人戒菸服務競賽」頒獎		
16:10-16:15	「戒菸衛教我最行-最有效的戒菸衛教創意競賽」頒獎		
	得獎作品播放		
16:15-16:18	金獎作品 得獎者：黃美嘉 戒菸個管師｜臺北市立萬芳醫院		
16:18-16:21	銀獎作品① 得獎者：鄧麗文 個管師、洪湘雯 個管師｜聯新國際醫院		
16:21-16:24	銀獎作品② 得獎者：吳玫諭 護理師、施銘峰 醫師、游碧真 護理師｜臺大醫院雲林分院		
16:24-16:30	「優良戒菸衛教師」頒獎		
	頒獎典禮結束		

主辦單位｜中華民國血脂及動脈硬化學會　　**指導單位**｜衛生福利部國民健康署

合辦單位｜社團法人台灣菸害防制暨戒菸衛教學會、中華民國心臟學會、台灣腦中風學會、社團法人中華民國糖尿病衛教學會、社團法人中華民國糖尿病學會、台灣腎臟醫學會、臺灣介入性心臟血管醫學會、社團法人台灣老人急重症醫學會

會議議程 3.23

第一講堂

AI與888

TIME	TOPIC	SPEAKER	MODERATOR
09:00-09:05	Opening		劉秉彥 理事長 成大醫院｜心臟血管內科
09:05-09:25	三高一腎888防治計畫的實踐	李貽恒 教授 成大醫院｜心臟血管內科	葉宏一 名譽理事 馬偕醫院｜心臟血管內科
09:25-09:35	Discussion		
09:35-09:55	Integrating Cardiology and AI : Advancing Solutions for Coronary Artery Disease Treatment	張詩聖 教授 中國附醫｜心臟血管內科	詹世鴻 主任 成大醫院｜心臟血管內科
09:55-10:05	Discussion		
10:05-10:25	Vascular Complications in Primary Aldosteronism	林彥宏 教授 臺北榮總｜心臟血管內科	黃柏勳 名譽理事 臺北榮總｜心臟血管內科
10:25-10:30	Discussion & Closing		蔡惟全 主任 成大醫院｜心臟血管內科

ASCVD 高風險患者優化競賽頒獎

TIME	TOPIC	SPEAKER	MODERATOR
10:30-10:35	Opening		劉秉彥 理事長 成大醫院｜心臟血管內科
10:35-10:55	2024 ASCVD 高風險患者優化競賽 頒獎		江福田 教授 輔大附醫｜心臟血管內科 劉秉彥 理事長 成大醫院｜心臟血管內科
10:55-11:00	大合照		
11:00-11:05 11:05-11:10 11:10-11:15	優化競賽分享		江福田 教授 輔大附醫｜心臟血管內科
11:15-11:20	QA		
11:20-11:30	Discussion & Closing (3-to-goal 2.0分享)		林肇鋒 理事 馬偕醫院｜心臟血管內科

SUN.

第二講堂

血脂動脈硬化基礎醫學武林大會

TIME	TOPIC	SPEAKER	MODERATOR
09:00-09:05	Opening		王朝永 教授 林口長庚｜心臟血管內科
09:05-09:15	Explore The Role of Hsa-mir-409-3p in Diabetes-induced Epc Senescence and Clinical Implication 李欣怡		李任光 醫師 臺大醫院｜心臟血管內科
09:15-09:25	Sodium Nitroprusside Improves Uremic Toxin-induced Vascular Endothelial Cell Dysfunction 陳 璟		
09:25-09:35	Porphyromonas Gingivalis Groel Accelerates Abdominal Aortic Aneurysm Formation by Induction of M1 Polarization in Macrophages 陳佳怡		
09:35-09:50	頒獎 & 領獎 & 合照 Closing Remarks		吳造中 教授 臺大醫院｜心臟血管內科

Lunch Symposium

Moderate Drinking to Cardiovascular Health – A Friend ? or Foe ?

TIME	TOPIC	SPEAKER	MODERATOR
10:00-10:05	Opening		劉秉彥 理事長 成大醫院｜心臟血管內科
10:05-10:25	Moderate Drinking And Cancer Disease	陳哲宏 醫師 Stanford University	徐國基 理事 新光醫院｜心臟血管內科
10:25-10:45	Moderate Drinking And Heart Failure	劉彥佑 醫師 馬偕醫院｜心臟血管內科	褚柏顯 教授 林口長庚｜心臟血管內科
10:45-11:05	Moderate Drinking And Atherosclerosis	吳懿哲 秘書長 馬偕醫院｜心臟血管內科	吳彥雯 理事 亞東醫院｜心臟血管內科
11:05-11:30	Discussion & Closing		洪傳岳 名譽理事 萬芳醫院｜心臟血管內科

會議議程 3.23 SUN.

第三講堂

營養學：預防心血管疾病運動與營養

TIME	TOPIC	SPEAKER	MODERATOR
09:00-09:10	Opening		
09:10-09:40	談心血管疾病的運動處方	謝佩君 醫師 成大醫院復健部｜復健科	潘文涵 理事 陽明交通大學｜公共衛生研究所
09:40-09:50	Discussion		
09:50-10:20	不同運動方式及強度對改善高血脂的效果	張振崗 副校長 國立台灣體育運動大學	蔡一賢 理事 馬偕醫院
10:20-10:30	Discussion		
10:30-11:00	遊山玩水之心血管健康促進與食慾反應	錢桂玉 教授 國立體育大學 運動科學研究所	郭素娥 理事 成大醫院
11:00-11:30	Discussion & Closing		

晚餐議程 3.22 SAT.

Dinner Symposium – 1賽諾菲 第一講堂

TIME	TOPIC	SPEAKER	MODERATOR
16:30-16:50	The Impact of Early LDL-C Lowering on CV Outcomes in ACS	徐千彝 醫師 臺北醫學大學附設醫院	黃柏勳 醫師 臺北榮民總醫院
16:50-17:10	Early PCSK9 mAb in ACS: Translating Real-World Evidence into Clinical Practice	李俊偉 醫師 馬偕紀念醫院	方志元 醫師 高雄長庚紀念醫院
17:10-17:30	Discussion & Closing	All	

Dinner Symposium - 2安進 第二講堂

TIME	TOPIC	SPEAKER	MODERATOR
16:30-16:35	Opening	吳懿哲 醫師 馬偕醫院	
16:35-17:15	Short-Term Efficacy of Advanced Lipid Treatment in Recent MI Patients	林姝含 醫師 新光醫院	吳懿哲 醫師 馬偕醫院
17:15-17:25	Panel Discussion	All	
17:25-17:30	Discussion & Closing	吳懿哲 醫師 馬偕醫院	

晚餐議程 3.22 SAT.

Dinner Symposium - 3輝瑞 第三講堂

TIME	TOPIC	SPEAKER	MODERATOR
16:30-16:40	Opening	葉宏一 名譽理事 馬偕醫院｜心臟血管內科	
16:40-17:10	Optimizing Success of Smoking Cessation for Patients with cardiovascular risks	丁革新 部長 雲林基督教醫院	葉宏一 名譽理事 馬偕醫院｜心臟血管內科
17:10-17:25	Panel Discussions	葉宏一 名譽理事 馬偕醫院｜心臟血管內科	
17:25-17:30	Closing	葉宏一 名譽理事 馬偕醫院｜心臟血管內科	

午餐會議 3.23 SUN.

Lunch Symposium – 1 台田 第一講堂

TIME	TOPIC	SPEAKER	MODERATOR
11:30-11:35	Opening Remarks	劉秉彥 理事長 成大醫院｜心臟血管科	
11:35-12:25	Beyond the Numbers : Why Sustained LDL-C Reduction Matters in Cardiovascular Risk Management	黃金洲 醫師 台北榮民總醫院｜心臟內科	劉秉彥 理事長 成大醫院｜心臟血管科
12:25-12:30	Panel Discussion & Closing Remarks	劉秉彥 理事長 成大醫院｜心臟血管科	

Lunch Symposium – 2 第一三共 第二講堂

TIME	TOPIC	SPEAKER	MODERATOR
11:30-11:35	Opening Remarks	黃柏勳 醫師 臺北榮民總醫院	
11:35-12:15	CLEAR NOW! Achieve Ldl-c Goal With Firstin Class Oral Lipid Lowering Therapy	林肇鋒 醫師 台北馬偕醫院	黃柏勳 醫師 臺北榮民總醫院
12:15-12:25	Discussion : • Real-World Practices in Lipid-Lowering Therapies • Unmet Clinical Needs and Challenges • Any Question	黃柏勳 醫師 臺北榮總 林肇鋒 醫師 臺北馬偕	
12:25-12:30	Panel Discussion & Closing Remarks	黃柏勳 醫師 臺北榮民總醫院	

3/22 SAT. | 13:40-14:10 第一講堂

How Does UACR Matter CV and Renal Treatment and Prognosis?

徐千彝 副秘書長 北醫附醫 | 心臟血管內科

現職

臺北醫學大學附設醫院心臟內科專任主治醫師
臺北醫學大學附設醫院研究部副主任
臺北醫學大學附設醫院心臟內科心臟衰竭組主任
臺北醫學大學專任副教授
教育部部定副教授 (副字第151134號)
台灣高血壓學會THS理事(第八屆、第九屆)，教育委員會委員(第七屆、第八屆、第九屆)
台灣動脈硬化暨血管病醫學會TSAVD理事(第十屆)
中華民國血脂及動脈硬化學會TSLA 副秘書長(第十一屆)
台灣心肌梗塞學會TAMIS 副秘書長(第二屆)，國際委員會主任委員(第二屆)
臺灣介入性心臟血管醫學會TSCI 研究暨登錄委員會委員(第十屆)，公共醫療政策委員會委員 (第十屆)
內科專科醫師甄審委員會資格審查小組委員 (2021, 2022, 2023, 2024)
歐洲心臟學會會士 (FESC, Fellow of European Society of Cardiology)
亞太心臟學會會士 (FAPSC, Fellow of Asian Pacific Society of Cardiology)

學歷

國立陽明大學醫學士
國立陽明大學臨床醫學研究所博士

3/22 SAT. | 14:20-14:40 第一講堂

DKD 高風險患者 心腎照護品質優化競賽說明

葉志凡 副秘書長 臺大醫院 | 心臟內科

經歷

2005/07-2005/08 | Exchange student, University of Houston, Texas
2006/06-2007/06 | Intern, National Taiwan University Hospital, Taipei
2008/07-2013/06 | Resident, Department of Internal Medicine, National Taiwan University Hospital, Taipei
2011/07-2013/06 | Clinical fellow, Division of Cardiology, Department of Internal Medicine, National Taiwan University Hospital, Taipei
2018/01-2018/10 | Attending physician, Division of Cardiology, Department of Internal
2021/01-2022/02 | Medicine, National Taiwan University Biomedical Park Hospital Chu-Tung Campus
2018/11-2020/11 | Visiting scholar, The University of Chicago
2013/07-Present | Attending physician, Division of Cardiology, Department of Internal Medicine, National Taiwan University Hospital, Taipei
2015/09-2023/01 | Clinical lecturer, College of Medicine, National Taiwan University
2023/02-Present | Clinical Assistant Professor, College of Medicine, National Taiwan University

學歷

2000/07-2007/06 | MD, Fu-Jen Catholic University, New Taipei City
2015/09-2022/01 | PhD, Department and Graduate institute of Pharmacology, College of Medicine, National Taiwan University, Taipei

3/22 SAT. | 15:15-15:35 第一講堂

Biological Functions of Exosomes And Their Impact on Atherosclerosis

張瑋婷 醫師 奇美醫院 | 心臟血管內科

經歷

2014/08 | Chi-Mei Medical Center, Tainan, Taiwan Attending physician, Department of Cardiology
2020/08 | Southern Taiwan University of Science and Technology, Taiwan Associate Professor, Department of Biotechnology
2023/08 | National Sun Yat-sen University, Taiwan Associate Professor, Department of Clinical Medicine

學歷

2000/09 – 2007/06 | Department of Medicine, National Cheng Kung University, Tainan, Taiwan Doctor of Medicine, 2007
2013/09 –2014/08 | Brigham and Women's Hospital, Harvard University, MA, USA Research Fellow, Cardiac Muscle Research Laboratory
2019/08 – 2023/06 | Graduate Institute of Clinical Medicine, National Cheng Kung University, Taiwan, PhD
2023/07 | Visiting Scholar with certificate of Precisional Medicine in St. Edmund Hall, Oxford (OXCEP)

摘要

Exosomes, small extracellular vesicles (30–150 nm) secreted by various cell types, play a crucial role in intercellular communication and have emerged as key regulators in atherosclerosis progression and resolution. Exosomes derived from endothelial cells, smooth muscle cells, macrophages, and platelets contain bioactive cargo, including microRNAs (miRNAs), proteins, and lipids, that influence atherosclerotic processes. Endothelial cell-derived exosomes can mediate vascular homeostasis or dysfunction.Macrophage-derived exosomes modulate immune responses, with pro-inflammatory exosomes accelerating plaque development by promoting foam cell formation and cytokine release, whereas anti-inflammatory exosomes facilitate plaque stability. Also, exosomes play a role in lipid metabolism and thrombosis. Smooth muscle cell-derived exosomes can either inhibit or promote plaque calcification, affecting plaque vulnerability. Platelet-derived exosomes contribute to coagulation and thrombosis, increasing the risk of atherosclerotic plaque rupture. For the therapeutic application, engineered exosomes hold promise for targeted drug delivery, reducing vascular inflammation and stabilizing plaques. Harnessing exosomes' natural ability to transfer functional molecules presents a novel strategy for treating atherosclerosis. In this talk, I will summarize the updated information regarding exosomes as critical mediators in atherosclerosis, influencing endothelial function, inflammation, lipid metabolism, and thrombosis.

3/22 SAT. | 15:35-15:55 第一講堂

Multifaceted Role of The Exosomes Secreted from Cardiac Lineage Cells

陳文彬 教授 臺大醫學院 實驗動物中心主任

經歷

國立臺灣大學藥物研究中心＆基因體中心，博士後 (2002-2008)
Cardiovascular Research Center, Massachusetts General Hospital, Boston, MA, USA. Postdoc (2009-2011)
國立臺灣大學醫學院藥理學科暨研究所 助理教授 (2011)
國立臺灣大學醫學院藥理學科暨研究所 副教授 (2016~now)
國立臺灣大學醫學院實驗動物中心 主任 (2020~now)

學歷

國立臺灣大學醫學院藥學系 學士 (1993)
國立臺灣大學醫學院藥理學科暨研究所 碩士 (1995)
國立臺灣大學醫學院藥理學科暨研究所 博士 (2001)

摘要

Background:
Exosomes are nano-sized extracellular vesicles secreted by cells for intercellular communication via transferring various biomolecules. A distinct profile of exosomal composition, including proteins, lipids, and nucleic acids, also reflect the characteristics of their parent cells. Therefore, it has been noticed the potential applications of the exosomes for disease diagnosis, drug delivery,and the regulation of signaling pathways.
Aims:
Our previous study aimed to characterize the molecular profile of the exosomes secreted from cardiac lineage cells at different development stages for the elucidation of their potential applications and the underlying mechanisms.
Methods:
The exosomes secreted from mouse postnatal Nkx2.5+ cardiac cells (Exo-mNkx2.5), cardiac fibroblast (Exo-Cfb), and cardiac precursor cells derived from human induced pluripotent stem cells (Exo-hCPC) were purified by ExoQuick. The nanoparticle property was examined by NanoSight. The exosomal miRs and proteins were characterized by RNAseq and LC-MS/MS, respectively. The transcriptional profile of cardiac cells treated with the exosomes was characterized by RNAseq. Cell survival was assessed by calcein-AM (live cells), propidium iodide (PI, dead cells) and Hoechst 33342 (total cells). Mitochondrial function was assessed by seahorse.
Results:
Postnatal mouse cardiac Nkx2.5+ cell is a unique cell population distinguished from cardiac fibroblast with significantly different transcriptomic profiling between them. It was also reflected in the prominently different miR and protein profiling in the exosomes secreted from

these two different types of cardiac cells. A cluster of miRs in Exo-mNkx2.5 mediated a prominent prosurvival effect in adult mouse cardiomyocytes in vitro, which was absent in Exo-Cfb. ExomNkx2.5 can produce cardioprotective action against isoprenaline-induced injury in mouse hearts in vivo as well as can protect human iPSC-derived cardiomyocyte (hCM) from H2O2-induced cell death through regulating oxidative stress and metabolic pathways. Further characterizing miR profile in Exo-hCPC reveals the potential target genes associated with myogenic differentiation and metabolic pathways functionally annotated by GOBP and KEGG. Functional validation demonstrated that Exo-hCPC can boost the differentiation of hiPSC to cardiomyocytes. Besides that, a cluster of metabolism-regulatory miRs was identified to improve mitochondrial function of hCM derived from hiPSC of the patient with Barth syndrome.
Conclusion:
Exosomes secreted from different cardiac lineage cells can exert versatile functions including prosurvival effect, boosting myogenic differentiation, and metabolic modulation. Understanding the underlying mechanism can facilitate the potential applications for metabolic rejuvenation.

3/22 SAT. | 15:55-16:15 第一講堂

It May The Right Time to Consider The Mesenchymal Stem Cell-derived Exosomes for Clinical Application for Different Disease Entities

葉漢根 教授 高雄長庚｜心臟內科

現職

中山醫學大學醫學系兼任教授
高雄長庚醫院心臟內科教授級主治醫師
長庚大學醫學系兼任教授

學歷

高雄醫學院醫學系

摘要

Abundant data, including experimental studies and clinical trials, have shown that mesenchymal stem cells (MSCs) therapy could improve ischemia-related, and inflammation induced organ dysfunction and prognostic outcomes. Basic research has proved that these improvements could be due to the fact that MSCs have capacities of anti-inflammation, immunomodulation and tissue regenerations. Experimental studies have shown that majorities of the cells, including MSCs, could secrete exosomes in response to physiological or pathological stimulation. Growing data disclose that MSCs-derived exosomes have properties of MSCs, i.e., containing capacities of anti-inflammation, attenuation of oxidative stress, immunomodulation and tissue regeneration. Accordingly, exosomes have experimentally utilized for treatment of various disease entities in animal models with fair outcomes. Plentiful experimental studies have supported that exosomes therapy may have greater advantage than in MSCs in with respect to gene/mRNA manipulations, large amount expansion by electrical or physical stimulation in ready for application. Of importance is that the safety and effectiveness of exosomes therapy is not inferior to MSCs. Accordingly, it is time to consider the clinical application of exosomes for those of diseases which are refractory to conventional medications.

Here, we will share the results of exosome therapy in the preclinical studies with audiences in this symposium.

3/22 SAT. | 13:35-13:55 第二講堂

心肌梗塞後血脂控制的實證醫學：最新指引與臨床研究
Evidence-Based Lipid Management After Myocardial Infarction: Latest Guidelines and Clinical Trials

黃逸群 副秘書長 林口長庚｜心臟內科

經歷

Attending Physician
Division of Cardiology, Department of Internal Medicine,
Chang Gung Memorial Hospital, Linkou Branch, Taoyuan, Taiwan

學歷

China Medical University, Taichung, Taiwan

摘要

Lipid management following acute coronary syndrome (ACS), is paramount for preventing recurrent cardiovascular events. Low-density lipoprotein cholesterol (LDL-C) plays a pivotal role in atherosclerotic plaque formation, making aggressive LDL-C lowering a primary therapeutic target. Recent trials have reinforced the significance of LDL-C in secondary prevention, leading to stricter LDL-C target recommendations in international guidelines. This presentation will review the latest evidence-based strategies for lipid management post-MI, incorporating recent advancements in ACS lipid control. In ACS, early initiation of intensive lipid-lowering therapy is emphasized to stabilize plaques and reduce event recurrence. Contemporary guidelines advocate for personalized lipid management based on risk stratification. Statins remain the cornerstone of LDL-C reduction, but challenges such as statin intolerance and failure to achieve target LDL-C persist. Ezetimibe, a cholesterol absorption inhibitor, has demonstrated efficacy in reducing cardiovascular events when combined with statins in ACS patients. PCSK9 inhibitors, inclisiran (siRNA), and bempedoic acid offer alternative or adjunctive therapies for patients with statin intolerance or inadequate LDL-C lowering. The CLEAR outcomes trial showed that bempedoic acid can reduce cardiovascular risk in patients with statin intolerance. This presentation will synthesize the latest guidelines and clinical trials on lipid management post-MI, emphasizing individualized strategies to minimize cardiovascular risk. Recent studies underscore the importance of early and intensive lipid control in ACS. This presentation aims to provide clinicians with a comprehensive overview of current evidence, enabling optimized lipid management in ACS patients.

3/22 SAT. | 13:55-14:15 第二講堂

心肌梗塞後血脂控制的臨床實務：治療策略與挑戰
Clinical Practice in Post-MI Lipid Management : Treatment Strategies and Challenges

林姝含 醫師 新光醫院 | 心臟內科

現職

2023 迄今 | 新光吳火獅紀念醫院心臟內科主治醫師
2023 迄今 | 教育部部定講師台灣周邊血管學會(TSPI)學術教育委員會委員
2023 迄今 | 台灣周邊血管學會(TSPI)學術教育委員會委員
2023 迄今 | 台灣周邊血管學會(TSPI)編輯暨研究委員會副主委
2024 迄今 | 台灣心肌梗塞學會(TAMIS)國際委員會委員
2024 迄今 | 臺灣介入性心臟血管醫學會(TSCI)青年委員會委員
2024 迄今 | 中華民國心臟學會(TSOC)青年醫師工作小組副主委
2024 迄今 | 中華民國心臟學會(TSOC)學術委員會委員

學歷

2008 - 2015 | 國立陽明交通大學醫學系學士

摘要

Lipid management is a cornerstone of secondary prevention following myocardial infarction (MI), with intensive LDL-C lowering strategies demonstrating significant benefits in reducing recurrent cardiovascular events. Early and aggressive lipid-lowering therapy is strongly recommended, yet real-world challenges, including clinical inertia, adherence issues, and therapeutic optimization, persist.

Recent clinical trials such as PACMAN-AMI, EVOLVE-MI, and HUYGENS provide compelling evidence for in-hospital initiation of PCSK9 inhibitors in high-risk patients. Very early lipid-lowering interventions have been shown to stabilize plaques, increase fibrous cap thickness, and reduce inflammation, offering a promising strategy to mitigate post-MI risk. While high-intensity statins remain the foundation of therapy, additional agents such as ezetimibe, PCSK9 inhibitors, and bempedoic acid offer alternative options, particularly for patients with statin intolerance.

Ongoing debates include the feasibility of achieving ultra-low LDL-C levels, the role of combination therapy, and the implications of systemic inflammation in residual cardiovascular risk. By integrating emerging evidence with clinical practice, optimizing lipid management in post-MI patients remains essential for improving long-term outcomes.

3/22 SAT. | 14:15-14:35 第二講堂

血脂控制的整合性照護：跨專科團隊合作模式
Integrated Care in Lipid Management :Multi-disciplinary Team Approach

陳志維 醫師 北醫附設醫院 | 心臟內科

經歷

臺北醫學大學附設醫院內科部住院醫師
臺北醫學大學附設醫院內科部總醫師
臺北醫學大學附設醫院心臟內科研究醫師

學歷

中山學醫學大學醫學士

摘要

在急性心肌梗塞後的患者中，血脂控制的成功率是心血管二次預防的重要指標，然而在實務中，患者低密度脂蛋白膽固醇（LDL-C）達標率仍然不理想。本次演講將探討「血脂控制的整合性照護：跨專科團隊合作模式」如何透過團隊協作提升患者的血脂控制成效。基於台北醫學大學附設醫院（TMUH）導入的急性冠心症（ACS）患者血脂管理經驗，將分享一套全面且整合的臨床策略，結合不同專科醫師、個案管理師及藥師的跨專科合作模式，從住院階段到出院後的長期追蹤，確保患者能夠持續改善。

這套模式包含多層面的干預措施，從high intensity statin起始治療、依據患者特質增強藥物組合（如ezetimibe）到動態調整治療方案。此外，個案管理師透過通訊軟體追蹤與即時提醒系統，提供患者教育與用藥依從性輔導，確保患者在回診期間能持續優化其LDL-C水準。

團隊的血脂控制流程中亦融入決策輔助系統，協助臨床醫師依據最新檢測數據快速決定是否需要調整用藥，並納入個別患者的共病條件，如糖尿病及周邊動脈疾病，從而個性化每位患者的治療方案。這種流程不僅顯著提升了LDL-C達標率，也改善了患者對治療的理解與參與。

在跨專科模式中，如何克服現實中的挑戰，例如門診醫師面臨的繁重病患量、患者服藥的依從性不足等問題。透過實際案例，參與者將能了解如何應用此模式於不同醫療機構中，推動血脂管理效能的提升，期望最終降低心血管事件的再發風險。

3/22 SAT. | 15:15-15:35 第二講堂

Advancements in Precision Medicine for Stroke Management

湯頌君 教授 臺大醫院 | 神經科

現職

2009/08 迄今 | 台大醫院 | 神經部 | 主治醫師
2024/02 迄今 | 台大醫學院 | 神經科 | 教授

學歷

2008/09-2013/07 | 國立台灣大學 | 臨床醫學研究所 | 博士
1992/09-1999/06 | 國立台灣大學 | 醫學系 | 學士

摘要

Acute stroke therapy is inherently complex, requiring swift and precise approaches to optimize patient outcomes. The integration of precision medicine into stroke management offers the potential for significant advancements, though its practical implementation remains challenging. This presentation will explore current advancements of precision medicine in stroke management, emphasizing its potential applications and limitations. Recent studies have highlighted the impact of genetic variants, such as CYP2C19, which contributes to clopidogrel resistance, and RNF213, a key marker for large vessel disease. These genetic insights can guide more personalized therapies. Additionally, measuring direct oral anticoagulants (DOACs) in acute stroke settings can help inform treatment choices and improve patient safety. Advanced imaging techniques play a pivotal role in distinguishing stroke types and guiding therapeutic interventions, ensuring that treatment is tailored to the patient's specific needs. However, while the promise of precision medicine in acute stroke management is evident, bridging the gap between cutting-edge research and routine clinical application remains a critical challenge.

3/22 SAT. | 15:35-15:55 第二講堂

Cerebral Microbleeds in Cardiovascular Diseases : Pathophysiology and Personalized Therapies

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現職

2022 迄今 | 台灣大學醫學院神經科臨床助理教授
2023 迄今 | 台大醫院神經部主治醫師

學歷

2021/06 | 台灣大學臨床醫學研究所 博士
2012/06 | 台灣大學醫學系 學士

摘要

Cerebral microbleeds (CMBs) is a radiological feature on blood-sensitive brain magnetic resonance imaging sequences, and are usually a sign of an underlying cerebral small vessel disease such as sporadic cerebral amyloid angiopathy or sporadic nonamyloid small vessel pathology (eg, arteriolosclerosis).

Epidemiological studies have identified associations between cardiovascular risk factors and CMBs, suggesting a crucial role of managing cardiovascular disease in the context of CMBs. For instance, atrial fibrillation and subsequent antithrombotic use are both associated with cerebral microhemorrhages; CMBs are also commonly reported in patients with coronary heart disease and are related to worse outcome.

From a neurological perspective, CMBs are strongly associated with risks of stroke, in particular intracerebral hemorrhage. Thus, phenotyping the burden and distribution and CMBs in patients with cardiovascular disease may not only implicate the underlying pathology in the brain, but also help stratify patient for risks of future stroke event. The personalized approach further help tailor antithrombotics use in managing cardiovascular diseases.

3/22 SAT. | 15:55-16:15 第二講堂

Cognitive Impairment in Cardiovascular Diseases : Screening and Management Approache

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學歷

2013/09-2023/02 | 成功大學 | 臨床醫學研究所 | 博士
1999/09-2006/07 | 成功大學 | 醫學系 | 學士

摘要

Cognitive impairment is increasingly recognized as a consequence of cardiovascular disease (CVD), with conditions such as hypertension, cardiac disease, atrial fibrillation, diabetes mellitus, and stroke playing a significant role in its development. These risk factors contribute to cognitive decline through mechanisms including chronic cerebral hypoperfusion, endothelial dysfunction, systemic inflammation, and metabolic dysregulation. Hypertension and cardiac disease are associated with reduced brain perfusion and white matter damage, while atrial fibrillation increases the risk of silent infarcts and stroke-related cognitive impairment. Diabetes mellitus exacerbates neurodegeneration through insulin resistance, oxidative stress, and microvascular complications. Stroke remains one of the strongest predictors of cognitive decline, with both ischemic and hemorrhagic events leading to long-term cognitive dysfunction. However, lipid control, despite its widespread use in cardiovascular disease management, has not been shown to induce cognitive impairment. Understanding the intricate relationship between cardiovascular health and cognition is essential for early detection, prevention, and management strategies. Addressing cardiovascular risk factors through targeted interventions may offer opportunities to reduce the burden of cognitive impairment and improve long-term neurological outcomes.

3/22 SAT. | 13:35-13:55 第三講堂

The Definitions And Pathophysiology of Ckm Syndrome

黃金洲 副秘書長 臺北榮總 | 心臟內科

現職

國立陽明交通大學內科學科教授
國立陽明交通大學藥理學科教授
臺北榮民總醫院內科部心臟內科主治醫師
中華民國血脂及動脈硬化學會副秘書長
台灣血脂衛教協會秘書長
台灣高血壓學會理事
中華民國心臟學會副秘書長
台灣醫學教育學會副秘書長
財團法人心臟醫學研究發展基金會副秘書長
高級心臟救命術指導員
中華民國心臟學會專科指導醫師
中華民國重症醫學會專科指導醫師
Fellow of the ESC (FESC)
Fellow of the AHA (FAHA)

學歷

國立陽明大學醫學系醫學士
國立陽明大學藥理研究所博士

摘要

In an effort to explore the bidirectional interplay between heart and kidney, the National Heart, Lung and Blood Institute established a 'Working Group on Cardiorenal Connections' and the American Heart Association (AHA) a 'Council on the Kidney in Cardiovascular Disease.' In 2004, the combination of the disorders was termed the 'cardiorenal syndrome' (CRS), who defined it as a 'moderate or greater renal dysfunction that exists or develops in patients with HF. In 2023, the AHA expanded cardiovascular and kidney risk factors into a new group of syndromes, the cardiovascular-kidney-metabolic (CKM) syndromes. The definitions and pathophysiology of CKD syndromes will be reviewed.

3/22 SAT. | 13:55-14:15 第三講堂

The Staging And Clinical Impacts of Ckm Syndrome

林威宏 教授 成大醫院 | 一般內科

現職

2006 迄今 | 國立成功大學醫學院附設醫院 內科部主治醫師
2018 迄今 | 成大醫院內科部PGY計畫 總導師
2019 迄今 | 國立成功大學醫學院附設醫院 教學中心執行長
2019 迄今 | 中華民國成杏會 理事
2020 迄今 | 臺灣醫療品質協會 病歷暨資訊品質委員會(委員)
2021 迄今 | 國立成功大學醫學院附設醫院 內科部臨床副教授
2022 迄今 | 全國OSCE考試 成大醫學院考場主任
2022 迄今 | 醫學臨床技能測驗試務委員會 委員
2023 迄今 | 台灣腎臟醫學會 雜誌編輯出版委員會副主任委員

學歷

1994-2001 | 國立成功大學醫學院 醫學系畢業
2007-2009 | 國立成功大學醫學院 臨床醫學研究所碩士
2009-2015 | 國立成功大學醫學院 臨床醫學研究所博士畢業

摘要

Cardiovascular-Kidney-Metabolic (CKM) syndrome represents an interrelated continuum of metabolic dysfunction, chronic kidney disease (CKD), and cardiovascular disease (CVD), which significantly increases morbidity and mortality. The recent classification of CKM syndrome into distinct stages provides a structured approach to understanding disease progression, facilitating risk stratification, and guiding clinical management. In this talk, we will discuss the pathophysiological interplay among metabolic disorders, renal dysfunction, and cardiovascular complications, emphasizing the bidirectional relationships that drive disease progression. We will also explore the clinical implications of CKM staging in patient management, highlighting strategies for early intervention, risk reduction, and personalized treatment approaches. By adopting a proactive and integrative perspective, nephrologists play a crucial role in mitigating the burden of CKM syndrome and improving patient outcomes.

3/22 SAT. | 13:35-13:55 第三講堂

The Optimal Strategies For Prevention And Management of Ckm Syndrome

王俊興 主任 臺中榮總 | 新陳代謝科

經歷

2023/02 迄今 | 臺中榮總內分泌新陳代謝科主任
2009/05 迄今 | 臺中榮總內分泌新陳代謝科主治醫師
2006/09-2009/04 | 臺中榮總內分泌新陳代謝科總醫師
2003/05-2006/08 | 臺中榮總內科部住院醫師

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摘要

Cardiovascular-Kidney-Metabolic (CKM) Syndrome is a complex, interrelated condition characterized by the coexistence of cardiovascular disease (CVD), chronic kidney disease (CKD), and metabolic disorders such as type 2 diabetes, obesity, and hypertension. These conditions share common risk factors and pathophysiological mechanisms, leading to a cycle of progressive organ dysfunction and increased morbidity and mortality. Patients with CKM Syndrome are at significantly higher risk for 1. heart failure and myocardial infarction, 2. stroke and peripheral artery disease, 3. CKD progressing to end-stage renal disease (ESRD), and 4. reduced life expectancy and poorer quality of life. Given its progressive nature, early identification and intervention in CKM Syndrome are crucial.

Prevention strategies of CKM syndrome include lifestyle modifications and risk factor control. The former include dietary interventions, such as DASH (Dietary Approaches to Stop Hypertension) and Mediterranean diets, Reduction in sodium, refined carbs, and processed foods, and increased intake of fiber, healthy fats, and lean protein; maintain physical activity, such as at least 150 minutes/week of moderate-intensity exercise, and resistance training for metabolic and cardiovascular benefits; and weight management, such as targeted weight loss strategies, especially for patients with obesity, and bariatric surgery in severe cases. Risk factor control includes blood pressure management, glucose control in diabetes, and lipid management (statins as first-line therapy, with possible PCSK9 inhibitors or ezetimibe).

3/22 SAT. | 15:10-15:25 第三講堂

Effect of Smoking Cessation Treatment Service Contest on The Motivation of Physicians To Help High Cardiovascular Risk Smokers Quitting Smoking (戒菸治療服務競賽對醫師幫助心血管高風險吸菸者戒菸動機的影響)

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摘要

Patients with high cardiovascular risk, including those under the care of cardiologists, neurologists, diabetologists, and nephrologists, are significantly impacted by smoking. Historically, these specialists in Taiwan seldom directly provided Smoking Cessation Treatment Services (SCTS). To address this, a medical society-initiated SCTS contest was conducted annually from 2018 to incentivize physician involvement. An analysis of data from the Health Promotion Administration covering 2018 to 2021 revealed that 859 physicians registered for the contest, enrolling 4,105 smokers. While annual case numbers rose from 2018 to 2020, they declined in 2021, likely due to the COVID-19 outbreak. Despite this, abstinence success rates at 3 and 6 months showed an overall upward trend over the four years, although a slight dip was observed in 2021. Medical centers generally demonstrated higher success rates than regional hospitals, though the differences were not statistically significant. Key factors contributing to successful abstinence included hospitalization, older age, varenicline use, and longer medication duration. The contest effectively motivated physicians to assist high CV risk patients in quitting smoking, and while COVID-19 reduced participation, it did not significantly hinder the overall progress. This model offers valuable insights for enhancing smoking cessation strategies in the future.

3/23 SUN. | 09:05-09:25 第一講堂

三高一腎888防治計畫的實踐

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摘要

心腦血管疾病是造成台灣民眾死亡的主要疾病。在台灣這些疾病有年輕化的趨勢；同時也導致後續慢性照顧經濟的重大負擔。心腦血管疾病可以透過主要風險因子三高，包括高血壓、糖尿病、高膽固醇，的控制來預防其發生。因此我們倡議應該由源頭做起，經由有效的管控三高來扭轉台灣心腦血管疾病的發展趨勢。

一、進行主要風險因子三高篩檢，包括高血壓、糖尿病及高膽固醇，以落實臨床準則所建議的初級預防策略。
二、建立以病人為中心之三高整合照護網系統，協助基層醫療及醫院，對高血壓、糖尿病及高膽固醇進行有效的管控。
三、與三高有關的專業醫學會合作，研發高血壓、糖尿病及高膽固醇三高的臨床照護指引；同時開發相關的衛教資源，提供民眾使用。
四、協調健保署依照台灣專業醫學會所制訂的三高最新臨床照護指引，提供有關三高藥物及諮詢的健保給付，以強化三高治療的持續性及有效性。

3/23 SUN. | 09:35-09:55 第一講堂

Integrating Cardiology and AI : Advancing Solutions for Coronary Artery Disease Treatment

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摘要

本演講探討人工智慧在冠狀動脈疾病診斷與治療中的應用，特別是提升導管室的診斷效率與準確性。傳統冠狀動脈造影雖是診斷的核心，但影像判讀耗時，僅提供解剖資訊，無法直接評估心肌缺血或心功能。人工智慧技術，如目標檢測與血管分割，可輔助病灶標註、改善手術規劃及支架長度計算。FFRangio 透過 3D 重建與血流阻力分析提供即時功能性指引，優化經皮冠狀動脈介入治療決策，降低風險與成本。此外，人工智慧應用於非閘控 CT 影像的冠狀動脈鈣化偵測，有助於心血管風險分層並實現早期介入。未來，人工智慧在疾病管理的發展將整合解剖與生理數據，提升診斷與治療品質，並透過大規模臨床試驗驗證其臨床效益。

3/23 SUN. | 10:05-10:25 第一講堂

Vascular Complications in Primary Aldosteronism

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摘要

Primary aldosteronism (PA) affects 5-13% of patients with hypertension, and is characterized by an inappropriate production of aldosterone. Increasing evidence indicates that PA is much more prevalent than previously believed, making this disease the most frequent cause of secondary hypertension. PA can be mainly divided into two subtypes: aldosterone-producing adenoma (APA) and idiopathic adrenal hyperplasia (IAH). In recent studies, the incidence of PA is getting higher and higher. In AHA 2017 guideline for hypertension prevention, the incidence of PA is 8 % in general hypertension and 20% in resistant hypertension. Excessive aldosterone influences multiple systems in human body and cause numerous diseases including cardiovascular, renal, immune, metabolic and psychological diseases. These make PA is a very important issue in hypertensive society.

In clinical aspect, PA patients had more cardiovascular complications, including coronary artery disease, myocardial infarction, stroke, transient ischemic attack, atrial fibrillation and heart failure than in patients with essential hypertension (EH). Moreover, in structure of cardiovascular system, PA patients have more prominent left ventricular mass, cardiac fibrosis, impaired endothelial dysfunction and increased arterial stiffness than EH patients. The altered structure can be reversible partially after treatment.

Due to the important role of PA, our research team (TAIPAI) was assembled in 2005 to organize multi-discipline researches. There are more than 2000 cases in our data registry. About 75-100 PA patients are new-diagnosed or referred to our team each year. We had published more than 100 papers and more than half of them were in cardiovascular field. The cardiovascular research in TAIPAI study group includes large clinical cohort study, basic researches, and population science. We focus on cardiac and vascular structure / functional change of PA patients and the reversibility after treatment.

This speech will cover new insights of vascular complications of PA, including our recent works related to somatic mutation and cortisol co-secretion on cardiovascular system in PA patients.

3/23 SUN. | 09:05-09:15 第二講堂

Explore The Role of Hsa-mir-409-3p in Diabetes-induced Epc Senescence and Clinical Implication

李欣怡

摘要

Adult endothelial progenitor cells (EPCs) play a crucial role in vascular repair and neovascularization. However, various factors affect the functionality of EPCs, such as aging, cardiovascular diseases, and diabetes. Previous studies have shown that hsa-miR-409-3p expression increases in senescent EPCs and inhibits angiogenesis through the PP2A-P38 signaling pathway. Literature indicates that diabetes accelerate EPC senescence in which hyperglycemic environment and advanced glycation end products (AGEs) are closely linked to the pathogenesis. Understanding diabetes-induced EPC senescence and the role of microRNAs in regulating EPCs activities would have potential clinical implications. In vitro experiments showed that human endothelial colony-forming cells (ECFCs) treated with each of advanced glycation end products of bovine serum albumin (AGE-BSA) and high glucose conditions led to up-regulation of hsa-miR-409-3p. Additionally, ECFCs exposed to high glucose exhibited decreased proliferation and increased β-galactosidase activity, suggesting that the diabetic environment accelerating ECFC senescence was in association with hsa-miR-409-3p. Furthermore, ECFCs treated with high glucose and transfected with a hsa-miR-409-3p antagonist down-regulated senescence marker p21. Meanwhile, analysis of the supernatants from ECFCs treated with AGE-BSA and high glucose revealed down-regulated fibroblast growth factors 19 (FGF19) levels and increased a list of inflammatory cytokines. Moreover, data from human peripheral blood mononuclear cells (PBMCs) demonstrated that hsa-miR-409-3p expression was significantly higher in diabetic patients compared to non-diabetic individuals. PBMC hsa-miR-409-3p level positively correlated with fasting glucose, HbA1c status, and Framingham Risk Score, with notably higher expression in groups with HbA1c levels exceeding 6.5% compared to controls. These findings suggest that hsa-miR-409-3p plays a critical role in diabetes-induced EPC senescence and may contribute to impaired vascular repair and increased risk of atherosclerosis in diabetic patients. Our study provides invaluable insights potential therapeutic targets by elucidating the molecular mechanisms through which hsa-miR-409-3p regulates EPC function, particularly its involvement in FGF19 signaling. Targeting hsa-miR-409-3p could offer a novel strategy to mitigate EPC senescence, enhance vascular regeneration, and improve outcome in patients with diabetes.

3/23 SUN. | 09:15-09:25 第二講堂

Sodium Nitroprusside Improves Uremic Toxin-induced Vascular Endothelial Cell Dysfunction

陳 璟

摘要

The incidence of cardiovascular diseases is higher in patients with chronic kidney disease, resulting in higher morbidity and mortality. Uremic toxins have been shown to increase oxidative stress and reduce nitric oxide bioavailability and production, ultimately leading to endothelial cell damage. Nitric oxide is crucial for the regulation of vascular physiology. The aim of this study was to investigate the potential of sodium nitroprusside (SNP), a nitric oxide donor, to improve endothelial cell dysfunction in uremic toxin-stimulated human aortic endothelial cells (HAECs). Uremic toxins such as indoxyl sulfate and asymmetric dimethylarginine were used. SNP increased cell proliferation in uremic toxin-stimulated HAECs. SNP also showed antioxidant and anti-inflammatory effects with reduced interleukin-1β/interleukin-6/ tumor necrosis factor-α protein expressions in uremic toxin-stimulated HAECs. Furthermore, SNP repaired endothelial cell functions with enhanced phospho-Akt/ endothelial nitric oxide synthase/vascular endothelial growth factor/stromal cell derived factor-1 protein expressions in uremic toxin-stimulated HAECs. In conclusion, SNP could improve uremic toxin-induced vascular endothelial cell dysfunction by its antioxidative, anti-inflammation, and pro-angiogenic abilities. This study may provide a theoretical basis for the potential application of SNP treatment in chronic kidney disease patients with vascular endothelial cell damage.
Keywords: Chronic kidney disease; Cardiovascular diseases; Uremic toxins; Vascular endothelial cells; Sodium nitroprusside

3/23 SUN. | 09:25-09:35 第二講堂

Porphyromonas Gingivalis Groel Accelerates Abdominal Aortic Aneurysm Formation by Induction of M1 Polarization in Macrophages

陳佳怡

摘要

Periodontal pathogens such as Porphyromonas gingivalis (P. gingivalis) can invade remote organs through the circulation and exacerbate cardiovascular diseases, respiratory diseases, neurodegenerative diseases, and cancer. GroEL, a virulence factor of P. gingivalis, can stimulate the host monocytes, gingival epithelial cells, and gingival fibroblasts to produce pro-inflammatory cytokines such as interleukin (IL)-6, IL-8, tumor necrosis factor-α, and IL-1β, which are key factors in inducing abdominal aortic aneurysm (AAA) formation. In fact, recent studies have indicated a positive correlation between periodontal disease and the occurrence of AAA. However, although a high proportion of P. gingivalis has been observed in clinical AAA specimens, the complete pathological mechanisms have not yet been identified. Our results from a previous study implied that the GroEL protein of P. gingivalis is highly correlated with smooth muscle cell activation in AAA progression; however, whether GroEL is highly correlated with macrophage inflammation in AAA progression remains unknown. In the present study, through animal experiments, we confirmed that GroEL promotes inflammatory cytokine production and macrophage differentiation, thereby accelerating and aggravating aortic remodeling and AAA formation. Moreover, we used human monocytic THP-1 cells to investigate the underlying mechanisms in vitro. The results demonstrated that GroEL promoted M1 macrophage polarization and inhibited IL-4/IL-13-induced M2 macrophage polarization. Additionally, GroEL induced M1 macrophage polarization via thrombomodulin and interferon regulatory factor 5 (IRF5) expression. Therefore, our findings suggest that P. gingivalis GroEL may contribute to AAA formation by affecting M1 macrophage polarization.

3/23 SUN. | 10:05-10:25 第二講堂

Moderate Drinking And Cancer Disease

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3/23 SUN. | 10:25-10:45 第二講堂

Moderate Drinking And Heart Failure

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摘要

Moderate alcohol consumption is often served as relatively harmless or even beneficial for cardiovascular health. However, it is crucial to understand that even moderate drinking can have significant implications for the heart, particularly in terms of developing heart failure. Cardiomyopathy, cardiac fibrosis, and cardiac arrhythmia are central to this condition.
Cardiomyopathy, a direct consequence of alcohol intake, involves the weakening and enlargement of the heart muscle. Even moderate alcohol consumption can induce toxic effects on cardiomyocytes, leading to mitochondrial damage and impaired calcium handling. These changes diminish the heart's contractile function, contributing to the development of heart failure.
Cardiac fibrosis is another critical factor in alcohol-related heart failure. Moderate alcohol intake can stimulate the activation of fibroblasts, leading to the excessive deposition of extracellular matrix proteins, such as collagen, within the myocardial tissue. This fibrotic remodeling stiffens the heart muscle and impairs its ability to contract effectively. The structural changes caused by fibrosis also disrupt the normal electrical conduction pathways, increasing the risk of arrhythmias.
Cardiac arrhythmias are a significant concern even with moderate alcohol consumption. Alcohol can provoke irregular heart rhythms, particularly atrial fibrillation. The mechanisms include direct toxic effects on cardiac cells, alterations in ion channel function, and increased sympathetic nervous system activity. Oxidative stress and inflammation further destabilize the heart's electrical activity, increasing the risk of arrhythmias and their complications.
In summary, moderate alcohol consumption can have detrimental effects on the heart,

leading to cardiomyopathy, cardiac fibrosis, and arrhythmias, all of which contribute to the development of heart failure. Understanding these mechanisms highlights the importance of cautious alcohol consumption to protect cardiovascular health.

3/23 SUN. | 10:45-11:05 第二講堂

Moderate Drinking And Atherosclerosis

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摘要

The relationship between moderate alcohol consumption and atherosclerosis remains a topic of significant debate. While observational studies have suggested potential cardioprotective effects of moderate alcohol intake, recent genetic and large-scale epidemiological analyses challenge this notion. Findings from Mendelian randomization studies, particularly in East Asian populations with ALDH2 polymorphisms, indicate a direct association between alcohol consumption and increased coronary artery calcification, hypertension, and stroke risk. Furthermore, global assessments, including data from the Global Burden of Disease Study, demonstrate that any level of alcohol consumption is associated with elevated cardiovascular and all-cause mortality risks, negating previous beliefs in a "safe threshold". Additionally, moderate alcohol consumption does not confer cardiovascular benefits in older adults and may contribute to increased cancer mortality. Given these insights, public health recommendations may require a reevaluation, shifting from moderation towards minimization of alcohol intake to reduce atherosclerotic disease burden.

3/23 SUN. | 09:00-09:40 第三講堂

談心血管疾病的運動處方

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摘要

1. 心臟復健常見的適應症: 冠狀動脈疾病，心衰竭，心律不整，繞道手術，瓣膜置換術，主動脈剝離術後，換心手術等等。各種心血管疾病患者的患者容易出現運動耐受度不佳，呼吸肌效能不佳，若合併心衰竭或是術後患者更易出現deconditioning with muscle atrophy，functional decline，起臥功能或是日常生活自理功能受限，或是上肢功能不佳的患者(上肢運動對於心臟疾病的患者負荷較大，尤其是合併心衰竭患者甚至連洗頭洗碗都會喘，撕不開即溶咖啡包等等)，此外還有一些患者是基本日常生活功能正常，進行以復工為目標導向的復健。
2. 以冠狀動脈疾病經心導管術後的患者為例說明復健之介入時機，流程及運動處方，人員場地設備，禁忌症及注意事項，實證效果。

A. 流程及運動處方:

I. 住院期:會診復健科醫師，評估是否有運動復健禁忌症，並進行風險評估，開立相應之運動處方
- 有氧運動(散步或固定式腳踏車或跑步機)每周至少3天，20~60分鐘或量力而為，運動心跳率為休息心跳+20 (上限<120) bpm，不累不喘的範圍。
- 低強度阻力訓練: 每週3天，可做3回合/天，8-12下/回合，強度15-20RM，量力而為。
- 功能訓練: reconditioning如bed mobility, 移位及平衡訓練, 躺坐站踏走(各動作之間需緩衝10~30秒，站起來先踏步10~20下若無暈眩再走），依照病患情況客製化

II. 出院後: 於復健科醫師門診評估是否有運動復健禁忌症，並進行風險評估，安排運動心肺功能檢查，依結果開立相應之運動處方
- 有氧運動(散步或固定式腳踏車或跑步機或橢圓機或登階機)每周至少3~5天，20~60分鐘/天；合併心衰竭患者則每日運動上限為60分鐘；運動心跳率則建議在心導管術後四周內運動心跳率上限120bpm，不累不喘不頭暈的強度；滿四周後可提升到中強度(40~80% Heart rate reserve method，或是低於年齡預估最大心跳率(220-年紀)的85%)，有點累有點喘但是不至於講不出話來的程度，能夠一邊運動一邊聊天的程度。
- 阻力訓練: 合併心衰竭患者建議有氧運動至少4周後才能提升到中強度阻力訓練，無心衰竭患者則可逕行每週三天，可做3~5回合/天，8-12下/回合，上肢強度15~20RM，下肢強度12~15RM，或是50~70% 1RM量力而為。
- 功能訓練:依照病患情況及期望目標(例如是否復工，依照工作的體能需求及負重需求量身訂做運動處方；或是特定目標例如爬山健行或是高爾夫球)客製化。

B. 人員場地設備: 美國運動醫學會的指南中有詳述心臟復健的setup流程以及場地需求，檢查同意書格式建議等等，細節小至例如溫溼度燈光空間大小等等都有特定的規定。在台灣的疾病認證規定則是復健科醫師及治療師需具有效的急救證照，醫師及治療師均須具備心臟復健受訓紀錄以及每年合理服務量的累積及相關在職教育時數。場地的部分須備有心臟復健病患專用的急救設備(電擊器急救車)，每日校正檢查清點，校正清點的人必須受過專門的急救設備校正之訓練教育且備有訓練紀錄。心臟復健病患中需要連續心電圖監控(可用有線的或是無線的)，血壓及血氧監控，需有急救流程人力及定期演練。。

C. 禁忌症及注意事項:

I. 開心術後患者因胸骨癒合需要三個月，所以三個月內雙手負重限制為<3公斤，雙肩的活動度建議術後兩周內不大於90度，術後第3~4周可逐漸增加至不引發疼痛的範圍，術後三個月內皆須避免擴胸的動作，術後三個月不宜開車或騎摩托車。

II. 術後三個月，從床上起來移位時，若手要撐床，請把腋下夾住，手臂靠著身體不要外展

III. 針對心衰竭的部分，由於上肢運動或出力對於心肌負荷較明顯，會比較容易喘或心跳快，因此上肢負重運動需晚一點恢復，生活上宜避免用力搓洗擦地或擦窗，或是晾很重的吸了水的衣物棉被，且要避免晾衣服到高處的曬衣桿，避免拔草挖土鏟地，避免抱小孩或小動物寵物，休閒娛樂的部分宜避免舉重，吊單槓、伏地挺身、釣魚拔河等等運動

IV. 所有心血管疾病或術後的患者，皆應避免等長運動(isometric exercise，例如棒式運動)，避免Valsava maneuver (憋氣出力，例如便祕用力的方式)

V. 心衰竭患者運動時宜避免骨盆底肌肉運動，例如凱格爾運動，或是抬臀撐住的運動，例如瑜珈的臀橋式運動(Bridging exercise)，若確實有需要從事此類運動，強烈建議為病患量身訂做處方且需嚴密監控。

D. 實證: 目前科學研究顯示運動訓練可減少心血管疾病再發率以及死亡率(針對冠狀動脈疾病及心衰竭患者可降低兩成)，改善血管內皮細胞功能，使血管擴張，改善四肢冰冷情況；改善自律神經功能，減少血壓的變動，提升體適能改善生活品質。

3. 風險評估: 運動高風險指標包含危險型心律不整，嚴重心衰竭(例如左心室射出率<40%)，曾被急救過(cardiopulmonary resuscitation (CPR)或電擊)，心血管處置中曾出現危急生命之併發症者。

4. 危急狀況之前兆:

A. 症狀: 有即將休克的前兆症狀例如暈眩噁心想吐或是冒冷汗，心悸或胸悶等等胸口不舒服，或是突然異常的喘

B. 心電圖變化: 複雜型心律不整(例如ventricular premature complex (VPC) quadrigeminy, trigeminy, bigeminy 或non-sustained ventricular tachycardia (NSVT)，越來越密集出現的VPCs，因為可能會演變成VT；或是atrial fibrillation with rapid ventricular response (AfRVR), 或是high degree AV block，Left bundle branch block (LBBB)等等)，ST波段上升超過1mm，或是下降超過2mm

C. 血壓: 運動中運動強度增加但血壓不增加或是反而下降(Flat BP response or exertional hypotension，表示心臟即將decompensate，很可能會休克)

D. 血氧:一般來說若沒有合併肺部疾病的心臟病患，血氧通常不會有異常（正常來說＞94%可不必給病人吸氧氣），一旦出現血氧下降，通常是很嚴重的問題了，例如急性心衰竭合併肺水腫就會血氧下降

5. 緊急狀況之處理: 先判斷是否啟動Advanced Cardiac Life Support (ACLS)流程(建議執行心臟復健的治療師應取得並具有效期內之ACLS證照)，若病患意識不清醒，或是生命徵象不穩定，要立刻停止運動，並採取緊急處理（包含呼救及ACLS處置），然後應盡快送急診。完整心臟復健亦須包含合格急救設備，急救人力以及緊急狀況SOP之設置。

縮寫:RM= repetition maximum

3/23 SUN. | 09:50-10:20 第三講堂

不同運動方式及強度對改善高血脂的效果

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摘要

許多研究已顯示運動訓練可改善高血脂，系統性回顧與統合分析顯示，有氧運動、阻力運動、或兩者結合均可顯著降低總膽固醇、低密度脂蛋白膽固醇、三酸甘油酯、極低密度脂蛋白膽固醇，並提升高密度脂蛋白膽固醇；而結合有氧運動與阻力運動對於血脂調控的效果最佳。在生理機制方面，運動透過多種機轉影響脂質代謝，例如運動增加肌肉對脂肪酸的攝取與氧化，進而降低血中三酸甘油酯濃度；而有氧運動能夠促進脂蛋白脂解酶活性，進而降低血中三酸甘油酯與極低密度脂蛋白膽固醇濃度；運動亦可增加肝臟低密度脂蛋白受體的數量，提升肝臟對低密度脂蛋白的清除效率等。整體而言，運動訓練雖對血脂的改善幅度屬於中等程度，但其長期效果可減少心血管疾病風險，並在特定族群中降低藥物使用需求。因此，針對高血脂患者，制定適當的運動處方，是改善高血脂的有效方法。

3/23 SUN. | 10:30-11:00 第三講堂

遊山玩水之心血管健康促進與食慾反應

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摘要

最佳運動營養策略有賴於對運動的掌握與對飲食行為的了解。安全有效是運動計畫基本條件，目前心肺以及阻力訓練對心血管健康促進效益相當明確，然而適合中高齡族群從事的水中運動，以及受民眾喜愛的登山健行運動的安全有效運動know how仍有待發展。本次內容第一部份與各位分享水中與登山健行之運動定量與心血管健康促進之研究成果。食慾是飲食行為之誘發因素，而食物選擇是飲食影響健康促進成效最直接且具個人特色的要素，第二部份與各位分享登山健行運動對食慾以及食物選擇之影響，期待找出個人登山健行運動營養教育與飲食指導缺口，進而發展適合國人登山健行之運動營養策略。

3/22 SAT. | 16:30-16:50 第一講堂

The Impact of Early LDL-C Lowering on CV Outcomes in ACS

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摘要

Overview evidence on early intensive LDL-C reduction, both post-ACS and earlier in life, highlighting its long-term CV benefits and plaque stabilization.

3/22 SAT. | 16:50-17:10 第一講堂

Early PCSK9 mAb in ACS: Translating Real-World Evidence into Clinical Practice

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摘要

Overview of real-world data and clinical cases supporting early PCSK9 mAb use in ACS, reinforcing guideline recommendations for intensified lipid lowering during ACS hospitalization and its impact on improving CV outcomes.

3/22 SAT. | 16:35-17:15 第二講堂

Short-Term Efficacy of Advanced Lipid Treatment in Recent MI Patients

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摘要

PCSK9抑制劑（PCSK9i）在recent MI病患中的應用，已經顯示出顯著的療效。這些藥物能夠快速且有效地降低低密度脂蛋白膽固醇（LDL-C）水平，並在短期內幫助穩定動脈斑塊，從而改善患者的預後。
PCSK9i屬於強效型降血脂藥物，臨床試驗顯示，PCSK9i對於降低LDL-C具有迅速且卓越的效果。對於ACS患者，特別是recent MI病患，快速和有效的膽固醇控制對於改善長期預後至關重要。多國的實際證據也顯示了PCSK9i對於患者的積極影響，這些效果使得PCSK9i在降血脂的治療中顯示出巨大的潛力。
其次，PCSK9i在使用三個月後即可幫助穩定斑塊。研究表明，使用PCSK9i三個月後，斑塊穩定性顯著提高，並且這種效果在九個月的觀察期內持續穩定斑塊。這一發現對於recent MI病患尤為重要，穩定的斑塊可以減少心血管事件的風險，從而改善患者的長期健康結果。
相關研究也支持了PCSK9i在ACS患者中的應用。研究顯示，PCSK9i能夠顯著降低LDL-C水平，並減少主要不良心血管事件（MACE）的發生率。這些研究結果進一步證實了PCSK9i在急性期和長期管理中的重要性。
總而言之，PCSK9i在ACS患者，特別是recent MI病患中的應用，展示了其快速下降血脂和穩定斑塊的優勢。臨床試驗和實際證據均支持其在改善患者預後方面的潛力。這些發現為醫療專業人員提供了新的治療選擇，幫助患者實現更好的長期健康結果。

3/22 SAT. | 16:40-17:10 第三講堂

Optimizing Success of Smoking Cessation for Patients with cardiovascular risks

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摘要

在全球公共衛生領域中，戒菸一直是降低吸菸相關疾病與死亡率的重要議題。近年來，隨著戒煙藥物的發展，吸菸者有了更有效的工具來幫助戒除菸癮。其中Varenicline已被多項國際研究證實能夠顯著提高戒煙成功率。

本演講將探討國際指引對戒菸藥物的建議，包括各國對於 Champix 在臨床應用中的角色。我們將分析其機制如何幫助減少吸菸渴望及戒斷症狀，並比較其與其他戒煙療法在戒煙成功率上的差異。

此外，隨著電子煙的興起，許多吸菸者將其視為替代品，但研究顯示電子煙不僅仍然具有尼古丁成癮性，還可能帶來肺部疾病、心血管風險，甚至促使年輕人養成吸菸習慣。因此，如何透過有效的戒煙藥物 來減少電子煙的使用，成為當前公共衛生政策的重要課題。

最後，我們將展望戒菸藥物的未來發展，包括新型尼古丁受體調節劑的研究以及如何利用數位健康科技來提升戒菸成效。在吸菸者所能採取減少心血管風險以及其他吸菸相關疾病的行動中，戒菸可說是最重要的。戒菸輔助藥物促進心血管健康的效益，勝於任何因治療引起心血管損傷的風險。本演講旨在提供醫療專業人士與公共衛生決策者更全面的戒煙策略，幫助更多吸菸者擺脫尼古丁依賴，邁向更健康的生活。

3/23 SUN. | 第一講堂

Beyond the Numbers : Why Sustained LDL-C Reduction Matters in Cardiovascular Risk Management

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摘要

This symposium explores the paradigm "lower for longer is better" in LDL-C management, demonstrating how sustained LDL-C reduction provides superior cardiovascular protection compared to intermittent or short-term approaches. Multiple clinical trials consistently show that prolonged, stable LDL-C reduction through continuous statin therapy significantly decreases major adverse cardiovascular events (MACE) over time, with benefits that accumulate and strengthen with extended treatment duration.

Among available statins, pitavastatin delivers exceptional long-term LDL-C control while offering a comprehensive approach to lipid management. Its pharmacological profile enables sustained efficacy at low doses with minimal drug-drug interactions, addressing critical barriers to therapy persistence. Importantly, pitavastatin demonstrates remarkable durability in LDL-C reduction while simultaneously improving HDL-C levels, providing holistic lipid profile optimization that translates to better cardiovascular outcomes.

The symposium addresses safety concerns regarding extended LDL-C lowering, examining alleged associations with cancer and cognitive decline. Evidence indicates that persistent LDL-C reduction with pitavastatin remains well-tolerated even during prolonged therapy, with particularly favorable outcomes in patients with diabetes and minimal impact on glycemic control.

This evidence-based review emphasizes that the cardiovascular benefits of LDL-C reduction are maximized not merely by achieving target levels, but by maintaining them consistently over years. When implementing "lower for longer" strategies in clinical practice, pitavastatin represents a preferred therapeutic option for primary prevention due to its efficacy, tolerability, and ability to support long-term treatment adherence.

3/23 SUN. | 11:35-12:15 第二講堂

CLEAR NOW! Achieve Ldl-c Goal With Firstin Class Oral Lipid Lowering Therapy

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