# 李香君 教授

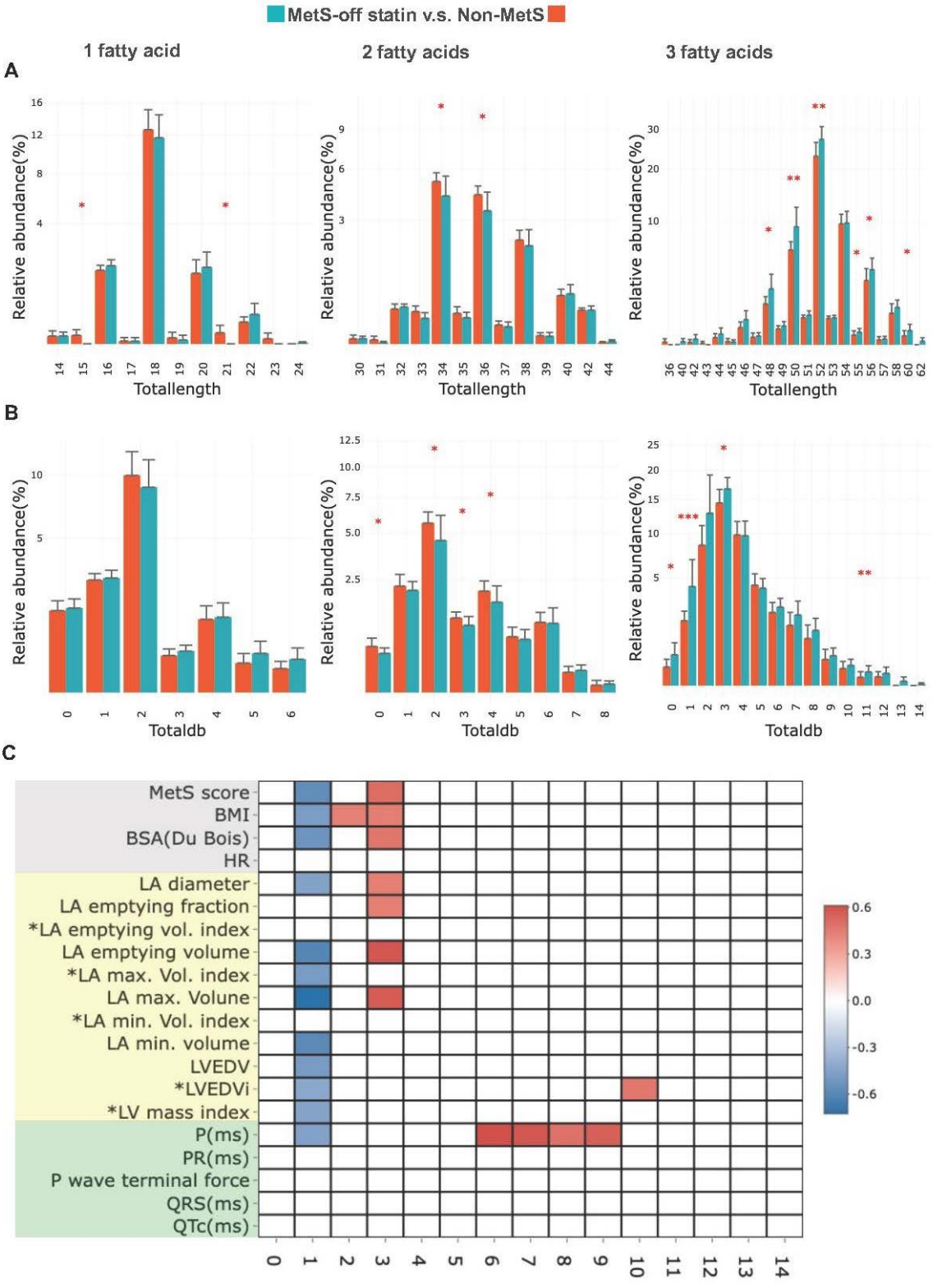
## 醫學院/醫學系

- 研究關於脂質異常如何導致各種器官與組織損害、功能異常、致病機轉中扮演角色、加速老化等，同時提升研究與教學的量能。

李香君醫師/教授的團隊自 2014 年起研究脂質，尤其是極低密度脂蛋白，在代謝症候群中藉由引發心臟內脂質積累所導致的心肌病變，以細胞實驗、動物模式和臨床研究，應用多種研究方法，逐漸建立一項新的學說，即餐後血脂、藉由極低密度脂蛋白會運送到心臟組織內做利用，由於代謝症候群造成了極低密度脂蛋白的質變（包括陰電性增加與攜帶的毒性脂質增加）而促進心肌病變形成，此為目前最常見的心律不整，心房顫動的早期病理變化。

近一年內發表的重要文章（Scientific Reports, 2023），為臨床合併基礎研究。其中代謝症候群病人隨機分為兩組，一組請病人停止服用史他汀（Statin 臨床上最廣為使用的降血脂口服藥）兩個星期，並對照組（沒有任何代謝症候群表徵的健康志願參與者），一共三組，將其血液分別收集，以超高速離心機分出極低密度脂蛋白並純化後，用質譜儀來分析脂質種類與含量。每一位參與研究者都接受完整的心臟超音波和心電圖檢查。資料齊全後，研究人員將脂質種類做生物資訊分析，並以統計方法來找出與心臟的心房病變表徵，如心房內徑、心電圖 P 波變化等參數具有顯著相關的脂質種類。結果顯示，代謝症候群病人的極低密度脂蛋白包含的脂質，在停止服用降血脂藥史他汀之後，具有促進心房心肌病變的毒性脂質顯著增加，且富含雙鍵的不飽和脂肪酸與心房擴大和 P 波變化有顯著相關性。此研究更近一步證實我們提出的學說，即極低密度脂蛋白藉由引發心臟內脂質造成心肌病變，此為在代謝症候群中好發心房顫動的重要病理機制之一。

研究論文：Lee HC*, Cheng WC, Ma WL, Lin YH, Shin SJ, Lin YH. Association of lipid composition and unsaturated fatty acids of VLDL with atrial remodeling in metabolic syndrome. Scientific Reports. 2023. 13:6575.



**【具體成果】**

**重要學術成就事項:**

1. 2024/3. Invited Foreign Lecturer, the English for Medical Professionals for senior medical students, at the University of Miyazaki, Japan.
2. 2024/7. Class Attendee and elected as the Class Leader, OXCEP Academic Medicine. St Edmund Hall in the University of Oxford.
3. 2024/8. Moderated Poster presentor. VLDL induces EZH2/PRC2 complex-mediated cellular senescence in metabolic syndrome. ESC Congress 2024, London, UK. 2024/8/31 (Moderated ePoster).
4. 2024/11 指導後醫系四年級學生前往 100th AHA Annual Conference 發表論文。VLDL induces neuronal growth with increased tyrosine hydroxylase expression through cardiomyocyte-secreted exosomes in metabolic syndrome. 100th AHA 2024, Chicago, IL, USA (Moderated ePoster).

**高被引論文:**

Lee HC, Shiou YL, Jhuo SJ, Chang CY, Liu PL, Jhuang WJ, Dai ZK, Chen WY, Chen YF, Lee AS. The sodium-glucose co-transporter 2 inhibitor empagliflozin attenuates cardiac fibrosis and improves ventricular hemodynamics in hypertensive heart failure rats. Cardiovascular Diabetology 2019 Apr. 1;18(1):45. (189 citations)

**國際合作:**

1. 日本群馬大學：Masami Murakami, MD, PhD. President, Japanese Promotion Council for Laboratory Testing (JPCLT); Distinguished Professor, Department of Clinical Laboratory Medicine, Gunma University Graduate School of Medicine.（國科會計畫國際合作案）
2. 美國約翰霍普金斯大學：Dr. Dao Fu Dai 校友戴道福醫師（共同發表論文 10.3390/antiox12051001）
3. 英國牛津大學：Prof. Charalambos Antoniades/ Deputy Head, Division of Cardiovascular Medicine 牛津大學附設醫院心血管中心主任（參與國際臨床研究 ORFAN study）

**【研究團隊】**

**團隊成員：**脂質科學暨老化研究中心 （https://lsarc.kmu.edu.tw/index.php/zh-tw/overview）

**團隊簡介：**我們致力於研究關於脂質異常如何導致各種器官與組織損害、功能異常、致病機轉中扮演角色、加速老化等，同時提升研究與教學的量能。本中心之任務如下：

一、研究脂質體學與脂質代謝在細胞分子層面之影響與相關機轉。

二、研究脂質體學與脂質代謝在老化之影響與相關機轉。

三、建構與維護研究平台、創新應用科技發明與治療標的之研發。

四、脂質代謝相關之轉譯醫學研究。

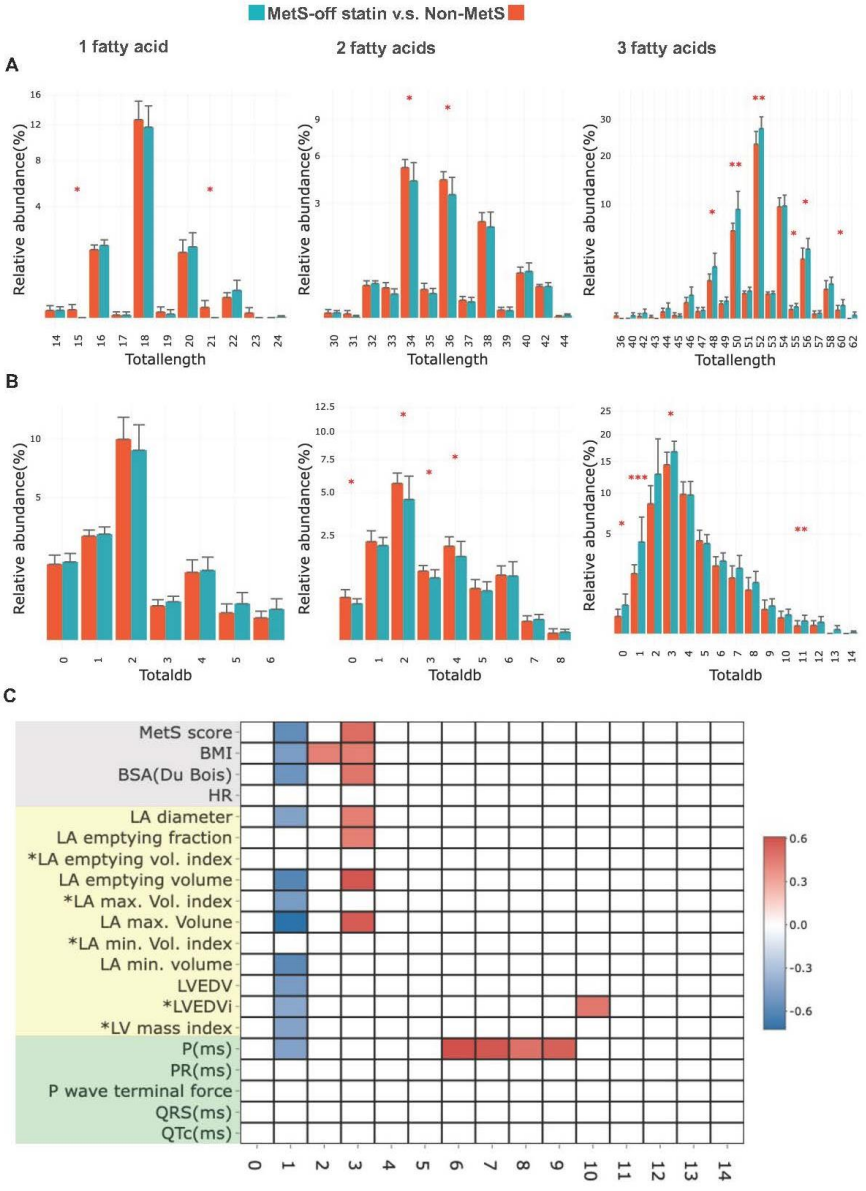
五、老化相關之轉譯醫學研究。

**研究聯繫 Email：**lsarc@kmu.edu.tw

Since 2014, Prof. Dr. Hsiang-Chun Lee's team has been studying the effects of lipids on cardiometabolic disorders, especially the effects of very low-density lipoprotein (VLDL), by inducing excess lipid accumulation in the heart during metabolic syndrome. A new perspective has been articulated through a series of translational and clinical studies. We proposed that the qualitative changes in VLDL in metabolic syndrome (including the increase in negative electricity and the increase in toxic lipids carried), particularly prominent in the postprandial phase, promotes the development of atrial myopathy, which is an early pathological change for the most common arrhythmia, atrial fibrillation.

An article published in recent years (Scientific Reports, 2023) showed the important results of our combined clinical and basic research. Patients with metabolic syndrome were randomly divided into two groups, and one group was asked to temporarily stop taking statins (the most widely used lipid-lowering drug) for two weeks. The other group of patients with metabolic syndrome received statins as usual. The third group was a control group (healthy volunteer participants without any signs of metabolic syndrome). All participants in the three groups underwent isolation and purification of VLDL from venous blood for subsequent lipidomic analysis using mass spectrometry.

Each participant underwent a complete echocardiography and electrocardiography. After the data were complete, the researchers performed bioinformatics analysis of lipid species and used statistical methods to identify the lipid species that were significantly related to the characterization of atrial lesions of the heart, such as atrial diameter, electrocardiogram P-wave changes, and other parameters. The results showed that the VLDL contained in patients with metabolic syndrome after two weeks of statin-off had a significant increase in toxic lipids that promoted atrial myopathy. In addition, unsaturated fatty acids rich in double bonds were significantly associated with atrial enlargement and P-wave changes. This study further supports our theory that VLDL causes atrial myopathy by triggering lipotoxicity in the heart, which is an important pathological mechanism of atrial fibrillation in metabolic syndrome.



**Publication**:Lee HC*, Cheng WC, Ma WL, Lin YH, Shin SJ, Lin YH. Association of lipid composition and unsaturated fatty acids of VLDL with atrial remodeling in metabolic syndrome. Scientific Reports. 2023. 13:6575.

**Concrete Results:**

**Important Academic Achievements**

1. 2024/3. Invited Foreign Lecturer, the English for Medical Professionals for senior medical students, at the University of Miyazaki, Japan.
2. 2024/7. Class Attendee and elected as the Class Leader, OXCEP Academic Medicine. St Edmund Hall in the University of Oxford.
3. 2024/8. Moderated Poster. VLDL induces EZH2/PRC2 complex-mediated cellular senescence in metabolic syndrome. ESC Congress 2024, London, UK. 2024/8/31 (moderated ePoster).
4. 2024/11 Guide and teach the postbaccalaureate medicine student Alexander Lan to participate and present an abstract at the 100$^{th}$ AHA Annual Conference in Chicago, USA. Title: VLDL induces neuronal growth with increased tyrosine hydroxylase expression through cardiomyocyte-secreted exosomes in metabolic syndrome. 100$^{th}$ AHA 2024, Chicago, IL, USA (moderated ePoster).

**Highly cited papers:**

Lee HC, Shiou YL, Jhuo SJ, Chang CY, Liu PL, Jhuang WJ, Dai ZK, Chen WY, Chen YF, Lee AS. The sodium-glucose co-transporter 2 inhibitor empagliflozin attenuates cardiac fibrosis and improves ventricular hemodynamics in hypertensive heart failure rats. Cardiovascular Diabetology 2019 Apr. 1;18(1):45. (189 citations)

**International collaboration**

1. Masami Murakami, MD, PhD. President, Japanese Promotion Council for Laboratory Testing (JPCLT); Distinguished Professor, Department of Clinical Laboratory Medicine, Gunma University Graduate School of Medicine. (The National Science Council Project, international collaboration)

2. Dr. Dao Fu Dai, Johns Hopkins University, USA（Collaboration publication: 10.3390/antiox12051001）

3. Prof. Charalambos Antoniades/ Deputy Head, Division of Cardiovascular Medicine, Director of the Cardiovascular Centre at the University of Oxford (ORFAN study)

【Research Team】

**Team Members:** Lipid Science and Aging Research Center (LSARC), Kaohsiung Medical University （https://lsarc.kmu.edu.tw/index.php/zh-tw/overview ）
**Research Team Introduction:** We are committed to studying how lipids can lead to damage to various organs and tissues, functional abnormalities, roles in pathogenesis, accelerated aging, etc., while improving the capacity of research and teaching. The mission of the LSARC is as follows:

1. To study the effects of lipoproteins and lipid metabolism at the cellular and molecular levels and related mechanisms.
2. To study the influence of lipoproteins and lipid metabolism on aging and related mechanisms.
3. Construct and maintain research platforms, innovative applications of scientific and technological inventions, and research and development of therapeutic targets.
4. Translational medical research related to lipid metabolism.
5. Translational medical research related to aging.

**Research Contacts Email:** lsarc@kmu.edu.tw