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论文 ID：10

Application of External Counterpulsation Combined with Lower Limb Muscle Strength Training for Fall Prevention in Elderly Patients with Essential Hypertension

Xinwen Liu*[1], 郑玲燕 [1], Jin Chen[1], Liuyi Dai[2], xusheng yang[1], Rongfang Zhou[1]

1. Zhejiang Hospital

2. Hangzhou Dianzi University

Corresponding Author E-mail: lxw_76@163.com

Background： To evaluate the effects of combining external counterpulsation (ECP) with lower limb muscle training (LMT) on fall prevention in elderly patients with essential hypertension (EH), by comparing hemodynamic responses and functional outcomes to those achieved with LMT alone.

Methods： In this prospective cohort study, a total of 105 elderly patients with EH (NYHA Class I–III) were allocated to either a combined ECP and LMT group (n = 34) or LMT-alone group (n = 71). All participants underwent 8 weeks of intervention. Hemodynamic parameters including stroke volume(SV), cardiac output(CO), total peripheral resistance(TPR), global group index(GGI), brachial-ankle pulse wave velocity(baPWV), ankle-brachial index(ABI) and functional capacity (Short physical performance battery(SPPB), anterior/posterior reach tests) were assessed separately before and after intervention.

Results： Both groups demonstrated parallel improvements in functional outcomes (SPPB: ECP Δ+2.27 vs. non-ECP Δ+2.33, P=0.823; anterior reach: Δ+27.41 cm vs. Δ+26.22 cm, P=0.983). However,the ECP group exhibited superior hemodynamic efficiency, with a significantly smaller SV increase (+1.68 mL vs. +4.98 mL, P=0.032) stable cardiovascular load (unchanged GGI vs. increased GGI in non-ECP, P<0.001), and preserved vascular integrity (stable baPWV/ABI vs. non-ECP's paradoxical baPWV reduction with ABI decline, P<0.01) despite similar reductions in total peripheral resistance.

Conclusion： The combination of ECP and LMT achieves functional improvements comparable to LMT alone, but it reduces afterload without increasing cardiac strain or compromising peripheral perfusion through a physiologically optimized pathway, which may offer a sustainable strategy for fall prevention in elderly patients with EH.

Key words： external counterpulsation, lower limb muscle strength training, fall prevention, elderly patients, essential hypertension.

论文 ID：11

Dynamic Trajectories of a Novel Proteomic Signature Enhance Coronary Heart Disease Prediction

Li Yang*

Zhejiang Hospital

Corresponding Author E-mail: yangli84331@163.com

Background： Hypertension is a major Coronary Heart Disease (CHD) risk factor. Existing CHD risk models based on static measurements have performance limitations. We hypothesized that dynamic protein biomarker trajectories could significantly improve risk prediction, particularly in high-risk individuals with hypertension.

Methods： We conducted a nested case-control study (90 cases, 90 matched controls) from a 40,000-strong high-risk cohort (Zhejiang, China), characterized by high hypertension prevalence, to identify and validate novel biomarkers. We screened plasma-derived extracellular vesicle proteins (LC-MS/MS). We quantified a panel of selected biomarkers—sex hormone-binding globulin (SHBG), cholesteryl ester transfer protein (CETP), phospholipid transfer protein (PLTP), and serum amyloid A protein 1 (SAA1)—across three annual time points preceding CHD diagnosis using targeted proteomics. Three predictive models were developed: 1) traditional risk factors (age, sex, BMI, SBP, smoking, LDL-C); 2) traditional + baseline biomarkers; and 3) traditional + dynamic trajectories (baseline levels + individual slopes) of the four biomarkers.

Results： CHD cases exhibited significantly higher baseline SBP (153.3 ± 11.3 vs. 129.2 ± 8.8 mmHg, $p<0.001$) and hypertension prevalence (80.0% vs. 50.0%, $p<0.001$) compared to controls. In the CHD group, SHBG declined while CETP, PLTP, and SAA1 rose over three years pre-diagnosis (all p for trend < 0.001); controls showed no such trends. Model 3 (dynamic trajectories) achieved superior predictive performance (AUC: 0.921, 95% CI: 0.881-0.941), significantly outperforming Model 1 (AUC: 0.777; $p<0.001$) and Model 2 (AUC: 0.875; $p<0.05$). Model 3 also yielded superior clinical net benefit.

Conclusion： Dynamic trajectories of SHBG, CETP, PLTP, and SAA1 powerfully predict incident CHD. Integrating this dynamic proteomic signature with traditional risk factors significantly enhances risk stratification, particularly for individuals with hypertension. This offers a promising strategy for precision cardiovascular disease prevention.

Key words： Dynamic Trajectories; Proteomic; biomarkers; risk factors; CHD; Prediction

论文 ID：18

α-Ketoglutarate Alleviates Hypobaric Hypoxia-Induced Pulmonary Hypertension via Suppression of Pulmonary Artery Smooth Muscle Cell Proliferation and Induction of Programmed Cell Death

Haoran Li, Luxun Tang*, Yongjian Yang, Cong Lan

西部战区总医院

Corresponding Author E-mail: tangluxun@sina.com

Background： Pulmonary arterial hypertension (PAH) is a severe cardiovascular disease characterized by persistent constriction and remodeling of pulmonary vessels. The excessive proliferation and inhibited apoptosis of hypoxia-driven pulmonary arterial smooth muscle cells (PASMCs) serve as primary triggers of pulmonary vascular remodeling. As a key intermediate of the tricarboxylic acid cycle, α-ketoglutarate (α-KG) possesses multiple regulatory functions, including energy coupling and epigenetic reprogramming. Additionally, it has demonstrated anti-

proliferative and pro-apoptotic effects in tumors. However, its therapeutic potential and underlying mechanisms in hypoxic PAH remain to be elucidated.

Methods： Primary mouse PASMCs were cultured to establish an in vitro model of hypoxia. The cells were treated with the α-KG derivative DM-α-KG and subjected to oxoglutarate dehydrogenase (OGDH) siRNA transfection. The proliferation and migration capabilities of PASMCs were quantitatively assessed using the CCK-8 cell viability assay, Ki-67 immunofluorescence labeling, wound-healing assay, and qPCR detection of proliferation-related markers. Subsequently, RNA sequencing was performed on PASMCs with or without OGDH siRNA treatment to generate whole-genome transcriptome expression profiles and enrich key signaling pathways. The AAV1-SM22α-shOGDH vector was utilized to specifically knock down OGDH in PASMC, and a mouse model of PAH induced by chronic hypoxia was established. Right ventricular systolic pressure (RVSP) was recorded in real-time using right heart catheterization. High-frequency cardiac ultrasound was employed to assess right ventricular ejection fraction (RVEF), tricuspid annular plane systolic excursion (TAPSE), and pulmonary artery acceleration time (PAAT). H&E staining and α-SMA immunofluorescence staining were used to quantify the percentage of vascular wall thickness (WT%) and the degree of muscularization.

Results ： The specific OGDH knockdown in smooth muscle of mice significantly improved RVSP and RVEF after 4 weeks of hypobaric hypoxia treatment. Additionally, TAPSE increased, and PAAT was prolonged. Furthermore, the WT% and degree of muscularization were reduced in specific OGDH knockdown mice. Under chronic hypoxia, both DM-α-KG treatment and OGDH-siRNA silencing significantly inhibited the proliferation and migration of PASMCs. RNA sequencing analysis revealed that downregulated genes after OGDH siRNA treatment were significantly enriched in pathways related to vascular development, positive regulation of cell migration, and the MAPK cascade, while upregulated genes were notably enriched in the positive regulation of programmed cell death. Further investigations demonstrated that OGDH deficiency resulted in the accumulation of α-KG, which could activate prolyl hydroxylases (PHDs), leading to the ubiquitination and degradation of hypoxia-inducible factor alpha (HIF-α), thereby terminating the hypoxic adaptation program. Concurrently, the increased expression of DNA DNA fragmentation factor subunit alpha (DFFA) promoted programmed cell death in PASMCs, inhibited pulmonary vascular remodeling, and delayed the progression of PAH.

Conclusion： Our study demonstrated that both DM-α-KG pharmacological intervention and smooth muscle-specific OGDH gene knockdown effectively disrupted the "proliferation-promoting and anti-programmed cell death " vicious cycle, reduced pulmonary vascular remodeling, and alleviated hypobaric hypoxia-induced PAH. Therefore, targeting the OGDH/α-KG/PHD/HIF-α axis presents a highly promising therapeutic strategy for PAH.

Key words： Hypobaric Hypoxia-Induced Pulmonary Hypertension，α-Ketoglutarate，Programmed Cell Death，Pulmonary Arterial Smooth Muscle Cell Proliferation，oxoglutarate dehydrogenase

论文 ID：19

Mir-128 targeting PPAR-γ improves vascular remodeling in spontaneously hypertensive rats by regulating the TLR4/NFkb inflammatory pathway

Zhoufei Fang*

The First Affiliated Hospital of Fujian Medical University

Corresponding Author E-mail: 64967607@qq.com

Background： To investigate the regulatory effect and underlying mechanism of microRNA-128 (miR-128) on vascular remodeling in spontaneously hypertensive rats (SHRs), with a focus on its targeted regulation of peroxisome proliferator-activated receptor gamma (PPAR-γ) and the Toll-like receptor 4/nuclear factor kappa B (TLR4/NF-κB) inflammatory signaling pathway.

Methods： All surgical and experimental procedures were approveled by the Animal Care and Use Committee of Fujian Medical University. ten-week-old male SHR and age-matched Wistar-Kyoto (WKY) rats purchased by Vital River Laboratory Animal Technology Company (Beijing, China). SHR were randomly assigned to the RDN group (n=6) 、Sac/Val group (n=6) and Sham group (n=6). WKY was used as the control group with normal blood pressure (n=6）.

In vivo,Ten-week-old SHRs were subjected to renal denervation (RDN) surgery. Eight weeks post-operation, mesenteric arteries were collected for histological and functional examinations. Serum miR-128 levels were detected by inductively coupled plasma mass spectrometry (ICP-MS). The expression levels of key proteins in the vascular wall were assessed via immunofluorescence (IF), immunohistochemistry (IHC), and Western blotting (WB). Bioinformatics analysis and RNA sequencing (RNA-seq) were employed to identify core genes and signaling pathways associated with hypertension-induced pathological inflammation.

Results： In vivo, in the SHR sham-operated group, elevated blood pressure, severe vascular remodeling, and impaired vasodilatory function were observed, accompanied by downregulated miR-128 expression and upregulated TLR4/NF-κB signaling activity (all $P < 0.0001$).RDN postoperative，miR-128 expression was significantly restored, which in turn inhibited the TLR4/NF-κB pathway, reduced the production of pro-inflammatory cytokines (including IL-1β, IL-6, and TNF-α), and ameliorated vascular dilation dysfunction in SHRs (all $P < 0.0001$). Mechanistically, miR-128 negatively regulated the TLR4/NF-κB signaling pathway while upregulating the expression of PPAR-γ ($P < 0.05$).

Conclusion： RDN not only exerts a hypotensive effect but also improves hypertensive vascular remodeling. miR-128 inhibits excessive inflammation in vascular smooth muscle cells and alleviates vascular remodeling in SHRs via the PPAR-γ/TLR4/NF-κB axis. These findings identify miR-128 as a potential therapeutic target for RDN in the treatment of hypertension, providing a novel regulatory strategy for the precision management of cardiovascular diseases.

Key words： hypertension.renal denervation (RDN) ， PPAR-γ/TLR4/NF-κB axis.microRNA-128 (miR-128)

论文 ID：21

miR-128 Inhibits Angiotensin II-Induced Vascular Smooth Muscle Cell Migration via Regulation of the PPAR-γ/TLR4/NF-κB Inflammatory Pathway

Zhoufei Fang, Hai Cai*

The First Affiliated Hospital of Fujian Medical University

Corresponding Author E-mail: 153182524@qq.com

Background： Abnormal migration and phenotypic transformation of vascular smooth muscle cells (VSMCs) are the core cytological basis of hypertensive vascular remodeling. MicroRNA-128 (miR-128) plays a critical role in

maintaining cardiovascular homeostasis. Our previous in vivo studies have confirmed that miR-128 can improve vascular remodeling and vasodilation function in hypertensive states, but its specific role and molecular mechanism in angiotensin II (Ang-II)-induced VSMC dysfunction remain to be further elucidated.

Methods： An Ang-II-induced VSMC injury model was established, and the cells were treated with miR-128 mimic, miR-128 inhibitor (sh-miR-128), and the PPAR-γ agonist rosiglitazone (RSG), respectively. Transwell assay was used to detect cell migration ability. Dual-luciferase reporter gene assay was performed to verify the direct targeting relationship between miR-128 and PPAR-γ. Fluorescence resonance energy transfer (FRET) was applied to detect intracellular PPAR-γ activity. Laser confocal microscopy was used to observe the expression and co-localization of PPAR-γ and α-actin. qRT-PCR and Western blot were conducted to detect the expression levels of key proteins in the TLR4/NF-κB inflammatory pathway, the inflammatory factor IL-6, and molecules related to cell phenotypic transformation.

Results： Ang-II stimulation significantly upregulated the expression of miR-128 in VSMCs in a time- and concentration-dependent manner ($P<0.01$). Dual-luciferase reporter gene assay confirmed that miR-128 directly targets and negatively regulates PPAR-γ expression.Ang-II treatment significantly reduced PPAR-γ expression in VSMCs and caused abnormally elevated α-actin expression, indicating a phenotypic switch of VSMCs from a contractile to a synthetic phenotype.Compared with the Ang-II alone treatment group:

RSG intervention partially upregulated PPAR-γ expression and inhibited VSMC migration to a certain extent, but the improvement in α-actin expression was relatively limited, remaining at a high level. sh-miR-128 intervention significantly upregulated PPAR-γ expression and simultaneously downregulated α-actin expression to near-normal levels. This change was accompanied by a marked reduction in cell migration ability, indicating effective improvement of abnormal proliferation and migration of VSMCs.

Conclusion： miR-128 is highly expressed in Ang-II-induced VSMCs, and directly targets and inhibits PPAR-γ, thereby activating the downstream TLR4/NF-κB inflammatory pathway and promoting VSMC migration and phenotypic transformation. Functional rescue experiments demonstrated that inhibiting miR-128 exerts superior effects on reversing VSMC phenotype and suppressing migration compared with direct PPAR-γ activation by RSG intervention. Targeted inhibition of miR-128 or upregulation of PPAR-γ is expected to be a potential intervention strategy for reversing VSMC dysfunction in hypertensive states.

Key words： miR-128，Vascular Smooth Muscle Cell Migration，PPAR-γ/TLR4/NF-κB axis， Inflammatory Pathway

论文 ID：27

Sorafenib induces ferroptosis in cardiomyocytes leading to cardiotoxicity by promoting NFE2L2 expression.

Yanmin Xu*, ya ping kong

The second hospital of tianjin medical university tianjin china

Corresponding Author E-mail: xuyanminphdmd@163.com

Background： Sorafenib is a multi-target tyrosine kinase inhibitor (TKI) that exerts its antitumor effects primarily by suppressing signaling pathways such as those mediated by vascular endothelial growth factor receptor (VEGFR) and Raf kinase. Ferroptosis is a form of regulated cell death driven by iron-dependent lipid peroxidation. By exacerbating oxidative stress responses in cardiomyocytes, ferroptosis leads to cardiomyocyte injury and dysfunction. Sorafenib-induced cardiotoxicity exhibits dose-dependent and time-dependent characteristics, yet its underlying mechanisms remain incompletely elucidated.

Methods： Twenty-four rats aged 12 weeks were used for in vivo studies. After the suckling mice were decapitated, primary rat cardiomyocytes were isolated and cultured and used for subsequent experiments after continuing to be cultured for 12 hours. After treatment with sorafenib, ferroptosis inhibitor Ferrostatin-1 (Fer-1), and deferoxamine (DFO), the expression of NFE2L2 and ferritin in myocardial tissue and cardiomyocytes were measured. Western Blot (WB) was used to detect the expression levels of related proteins. Real-time quantitative polymerase chain reaction (RT-qPCR) was used to detect the transcription levels of genes. Statistical analysis used GraphPad Prism software for data analysis and chart drawing. All data are expressed as mean ± SD. Comparison between two groups: For data with a normal distribution, use the paired t test (Unpaired two-tailed Student's t-test); for data with a non-normal distribution, use the Mann-Whitney U test.

Results： 1. Sorafenib promotes the expression of NFE2L2 in cardiomyocytes

The gene expression of the redox-regulated transcription factor NFE2L2 was significantly up-regulated in cardiomyocytes in the sorafenib-treated group (Figure 1.1 A). The mRNA expression level of NFE2L2 in the myocardial tissue of mice in the sorafenib-treated group was significantly increased (Figure 1.1 B). The mRNA expression level of NFE2L2 was significantly increased in rat primary cardiomyocytes (Figure 1.1 C).

2. Sorafenib promotes the expression of NFE2L2 in cardiomyocytes by inducing ferroptosis.

Mice were treated with sorafenib in combination with the ferroptosis inhibitor Fer-1 or DFO. The results showed that both Fer-1 and DFO significantly inhibited the expression level of Nfe2l2 mRNA in the myocardial tissue of mice induced by sorafenib (Figure 2. A). Treatment with sorafenib combined with the ferroptosis inhibitor Fer-1 and DFO inhibited the induction of Nfe2l2 transcription in cardiomyocytes (Figure 2. B). In vivo experiments showed that Fer-1 or DFO inhibited sorafenib-induced NFE2L2 protein expression in mouse myocardial tissue (Figure 2. C-D). In primary cardiomyocytes, Fer-1 or DFO significantly inhibited sorafenib-induced upregulation of NFE2L2 protein levels

Conclusion： Sorafenib induces ferroptosis in cardiomyocytes leading to cardiotoxicity by promoting NFE2L2 expression.

Key words： Sorafenib; ferroptosis; cardiotoxicity; NFE2L2 expression.

论文 ID：47

Ferroptosis Inhibition via Nrf2/GPX4 Activation Underlies the Broad-Spectrum Cardioprotection by Human alpha-1-Antitrypsin

Yanxiao Li*

新疆医科大学第一附属医院

Corresponding Author E-mail: li62292628@163.com

Background： Ferroptosis is increasingly recognized as a common pathogenic mechanism in diverse cardiac injuries, including doxorubicin-induced cardiomyopathy and acute myocardial infarction. Human alpha-1-antitrypsin (hAAT) has established cytoprotective and antioxidant properties, but its role in cardiac ferroptosis and the underlying mechanism remain unclear. This study investigated whether endogenous hAAT protects the heart against toxic and ischemic injury by suppressing ferroptosis through the Nrf2/GPX4 pathway.

Methods： CRISPR/Cas9-mediated humanized knock-in mouse models expressing either functional wild-type SERPINA1 (SERPINA1WT) or a loss-of-function mutant SERPINA1Q129* were generated. Mice were subjected to doxorubicin-induced cardiomyopathy or permanent left anterior descending coronary artery ligation to establish an acute myocardial infarction model. Cardiac function, histopathological injury, fibrosis, infarct size, serum myocardial injury markers, oxidative stress, iron accumulation, mitochondrial ultrastructure, and ferroptosis-related signaling were evaluated. In vitro validation was performed in doxorubicin-treated H9c2 cardiomyoblasts and hypoxia-treated AC16 human cardiomyocytes, with the Nrf2 inhibitor ML385 used to test pathway dependency.

Results： Endogenous wild-type hAAT markedly preserved cardiac function, reduced myocardial injury and fibrosis, attenuated ventricular remodeling, and lowered cardiac injury biomarkers in both doxorubicin-induced cardiomyopathy and acute myocardial infarction models. These benefits were absent in SERPINA1Q129* mice, indicating strict dependence on functional hAAT integrity. Mechanistically, hAAT suppressed key features of ferroptosis, including lipid peroxidation, iron overload, mitochondrial shrinkage, and disruption of redox homeostasis. hAAT activated Nrf2 signaling and upregulated major anti-ferroptotic effectors, including GPX4, HO-1, and xCT, while restoring iron metabolism in a context-dependent manner. In H9c2 and AC16 cells, hAAT showed ferroptosis-inhibitory effects comparable to ferrostatin-1, and ML385 largely abolished hAAT-mediated protection, confirming the essential role of Nrf2 activation.

Conclusion： Functional hAAT is a novel broad-spectrum endogenous inhibitor of cardiac ferroptosis. Its cardioprotective effect is mediated primarily through activation of the Nrf2/GPX4 axis and regulation of iron metabolism, and requires an intact wild-type hAAT structure. These findings expand the biological function of hAAT and support hAAT augmentation or related strategies as potential therapeutic approaches for ferroptosis-driven cardiomyopathies.

Key words： Ferroptosis; human alpha-1-antitrypsin; Nrf2/GPX4; doxorubicin-induced cardiomyopathy; acute myocardial infarction; cardioprotection

论文 ID：53

Clinical Characteristics of Target Organ Damage in Patients with Obesity-Related Hypertension and Efficacy Evaluation of Adipokine-Based Intervention

Shumei Li*

Wenjiang District Hospital of Traditional Chinese Medicine, Chengdu City

Corresponding Author E-mail: 1067861536@qq.com

Background： To analyze the clinical characteristics of target organ damage (TOD) in patients with obesity-related hypertension (OH), and evaluate the intervention efficacy of adipokine regulator liraglutide on reversing TOD in OH population, so as to provide evidence for individualized management of OH patients.

Methods： This was a single-center, randomized, controlled trial. A total of 216 OH patients admitted to our hospital from January 2023 to January 2025 were enrolled, and 108 patients with essential hypertension (EH) without obesity during the same period were included as control group to compare the differences in TOD prevalence and severity between the two groups. OH patients were randomly divided into liraglutide group (n=108, given liraglutide 1.2mg subcutaneous injection once daily on the basis of conventional antihypertensive therapy) and conventional treatment group (n=108, given conventional antihypertensive therapy alone). The follow-up period was 12 months. The changes of cardiac, renal, vascular TOD indexes and adipokine levels (leptin, adiponectin, resistin) before and after treatment were compared between the two groups, and the correlation between adipokine level changes and TOD improvement was analyzed.

Results： The prevalence rates of left ventricular hypertrophy (LVH, 48.15% vs 29.63%), microalbuminuria (MAU, 42.13% vs 24.07%), and carotid intima-media thickness (cIMT) thickening (51.39% vs 33.33%) in OH group were significantly higher than those in EH group (all $P<0.05$). After 12 months of intervention, the liraglutide group showed greater improvements in LV mass index (LVMI, 112.3±12.5 g/m² vs 121.7±13.2 g/m²), urinary albumin/creatinine ratio (UACR, 28.6±8.3 mg/g vs 39.2±9.7 mg/g), and cIMT (0.92±0.11 mm vs 1.01±0.13 mm) compared with the conventional treatment group (all $P<0.001$). The adiponectin level in liraglutide group increased by 32.4% while leptin and resistin levels decreased by 27.8% and 21.5% respectively, and the changes of adipokine levels were significantly correlated with the improvement of TOD indexes ($r=0.412\sim0.537$, all $P<0.001$).

Conclusion： OH patients have higher prevalence and more severe TOD compared with non-obese EH patients. Adipokine regulator liraglutide can significantly improve adipokine disorder and effectively reverse multiple TOD in OH population on the basis of conventional blood pressure control, which is worthy of clinical promotion.

Key words： Obesity-related hypertension; Target organ damage; Adipokine; Liraglutide; Intervention efficacy

论文 ID：63

Predictive value of TyG combined with LDL-C for MACE in patients with hypertension and type 2 diabetes mellitus

Adila Wulamu[1], Hongjian Li*[1], Dilare Adi[2]

1. 新疆医科大学第五附属医院

2. 新疆医科大学第一附属医院

Corresponding Author E-mail: lhjdoctor_109@126.com

Background： To investigate the predictive value of the triglyceride-glucose index (TyG index) and low-density lipoprotein cholesterol (LDL-C) for major adverse cardiovascular events (MACE) in patients with hypertension complicated by type 2 diabetes

Methods： A retrospective study enrolled 190 patients with hypertension and type 2 diabetes, followed up for 2 years. Patients were divided into an MACE group (n=35) and a non-MACE group (n=155) based on whether MACE occurred. The TyG index, LDL-C, and other indicators were collected and compared between the two groups to analyze their associations with MACE. Kaplan-Meier curves, Cox regression, ROC curves, and subgroup analyses were used for evaluation

Results： Among the 190 patients, 35 cases were in the MACE group (18.4%). Compared with the non-MACE group, the TyG index of patients in the MACE group was significantly higher [9.38 (8.95, 9.87) vs. 8.61 (8.17, 9.07), $P < 0.001$], and LDL-C was also significantly higher [2.65 (2.25, 2.99) mmol/L vs. 2.01 (1.60, 2.66) mmol/L, $P < 0.001$]. Univariate Cox regression showed that the TyG index (HR = 3.50, 95% CI: 2.26–5.42, $P < 0.001$) and LDL-C (HR = 1.79, 95% CI: 1.26–2.53, $P = 0.001$) were significantly associated with the risk of MACE. After multivariate stepwise correction (Models 1-3: correcting for age, gender, BMI, and LVEF), the TyG index (HR = 3.79, 95% CI: 2.26–6.36, $P < 0.001$) was an independent risk factor for MACE, while LDL-C (HR = 1.35, 95% CI: 0.97–1.89, $P = 0.077$) did not reach statistical significance. The tertile analysis showed that the highest tertile risk was significant (TyG: HR = 8.15 - 13.92; LDL-C: HR = 4.93 - 5.54, $P < 0.01$), while the middle tertile was not significant. Non-linear test indicated that both were linearly related to MACE ($P > 0.05$). The ROC curve showed that the area under the curve (AUC) for predicting MACE by TyG index, LDL-C, and their combination was 0.770 (95% CI: 0.680–0.859), 0.695 (95% CI: 0.603–0.788), and 0.807 (95% CI: 0.733–0.881), respectively. The combined model was significantly better than LDL-C alone ($P = 0.042$). The KM curve showed that the cumulative incidence of MACE in the high LDL-C group was significantly higher than that in the low LDL-C group (log-rank $P = 0.00015$); the differences in cumulative incidence among the four groups in the combined grouping (low/high TyG × low/high LDL-C) were significant (log-rank $P < 0.0001$), with the high TyG + high LDL-C group having the highest risk. Subgroup analysis showed that the effect of the TyG index was consistent in different age, gender, smoking, and BMI groups (interaction $P > 0.05$)

Conclusion： The TyG index is a strong independent risk factor for major adverse cardiovascular events (MACE) in patients with hypertension and type 2 diabetes. Combining it with LDL-C can improve the predictive efficacy, and it can be clinically applied for the identification of high-risk individuals at an early stage.

Key words： Hypertension; Type 2 Diabetes Mellitus; Triglyceride-to-Glucose Product Index; Low-Density Lipoprotein Cholesterol; Major Adverse Cardiovascular Events

论文 ID：73

Enhancing the Clinical Decision-Making Capabilities of Large Language Models in Hypertension Management via Multi-Type Prompt Engineering: A Rigorous, Multi-Stage Validation Study

Jun Wang[1,2], Jinjun Liu*[1]

1. 蚌埠医科大学第一附属医院

2. The First Affiliated Hospital of Bengbu Medical University

Corresponding Author E-mail: byyfyliujinjun@163.com

Background： Large Language Models (LLMs) hold significant promise for improving clinical decision-making in hypertension management. However, their vulnerability to hallucinations and inconsistent outputs presents serious

safety challenges for practical clinical use. The effects of various prompt engineering strategies on LLM performance in hypertension decision-making are not yet fully understood.

Methods： We conducted a comprehensive two-stage validation study using 300 de-identified simulated hypertension cases based on real-world clinical scenarios. In Stage 1, we evaluated four state-of-the-art LLMs (ChatGPT-4.1, Claude-4, Gemini-2.5 Pro, and DeepSeek-V3) across 18 distinct prompt strategies. These strategies were categorized into zero-shot, few-shot, and guidance prompts, and were combined with five advanced reasoning techniques (Chain-of-Thought, Tree-of-Thought, Reflexion, ReAct, and Self-Consistency). We assessed performance using accuracy, precision, recall, and regimen appropriateness scores against expert-defined reference standards. In Stage 2, six physicians from different hospital levels made treatment decisions both independently and with support from the best- performing and worst-performing LLM-prompt configurations, allowing for direct evaluation of the clinical impact.

Results： Guidance prompts significantly outperformed few-shot and zero-shot prompts. ChatGPT-4.1 with Guidance-Self-Consistency achieved optimal performance (91.3% accuracy, AUC 0.936), nearing expert-level competency, while zero-shot prompting yielded suboptimal results (62.7% with DeepSeek-V3). Optimal LLM assistance consistently enhanced physician average of accuracy across all levels (community hospital: 73.4% to 82.5%; county hospital: 84.0% to 87.9%; teach hospital: 91.5% to 92.0%) and reduced inappropriate regimen rates. Conversely, suboptimal LLM configurations decreased physician performance below baseline, increasing inappropriate regimen rates from 26.6% to 35.2% across all levels.

Conclusion： Effectively designed prompt strategies enable LLMs to provide reliable hypertension treatment recommendations, thereby supporting physicians' clinical decisions. However, suboptimal LLM-prompt combinations can compromise decision quality, underscoring the necessity for rigorous validation prior to the clinical deployment of LLM-based decision support tools.

Key words： Hypertension; Large Language Models; Clinical decision-making; Multi-Type Prompt Engineering .

论文 ID：95

Clinical Features of Multiple Endocrine Neoplasia-Related Secondary Hypertension and Independent Risk Factors for Refractory Hypertension Development

Shumei Li*

成都市温江区中医医院

Corresponding Author E-mail: 1067861536@qq.com

Background： To analyze the clinical characteristics of secondary hypertension caused by multiple endocrine neoplasia (MEN), and identify independent risk factors for the development of refractory hypertension (RH) in this population.

Methods： A retrospective cohort study was conducted on 98 patients with MEN-related secondary hypertension admitted to our hospital from January 2018 to December 2024. Clinical data including demographic characteristics, MEN subtype, endocrine hormone levels, blood pressure parameters, target organ damage status, and treatment

outcomes were collected. Patients were divided into RH group (n=42) and non-RH group (n=56) according to whether they met the diagnostic criteria of RH. Univariate and multivariate logistic regression analyses were performed to identify independent risk factors for RH development, and a predictive nomogram was constructed based on the regression results.

Results： Among 98 included patients, 72 (73.47%) were MEN type 1 and 26 (26.53%) were MEN type 2. The most common pathogenic causes of hypertension were primary hyperparathyroidism (47.96%) and pheochromocytoma (31.63%). The overall incidence of RH in this population was 42.86%. Multivariate logistic regression analysis showed that MEN type 2 (OR=3.24, 95%CI: 1.12-9.39, P=0.030), duration of hypertension before diagnosis ≥5 years (OR=4.71, 95%CI: 1.76-12.60, P=0.002), coexistence of ≥2 endocrine pathogenic lesions (OR=3.87, 95%CI: 1.43-10.49, P=0.008), and baseline left ventricular mass index ≥120g/m² (OR=2.96, 95%CI: 1.09-8.05, P=0.034) were independent risk factors for RH development. The nomogram constructed based on the above factors had good predictive efficacy, with an area under the receiver operating characteristic curve of 0.872 (95%CI: 0.795-0.928).

Conclusion： Patients with MEN-related secondary hypertension have a high incidence of RH. Early identification of high-risk populations based on the above risk factors and timely etiological treatment may reduce the risk of RH and improve long-term prognosis.

Key words： Multiple endocrine neoplasia; Secondary hypertension; Refractory hypertension; Risk factor; Nomogram

论文 ID：103

Targeting ROCK2 and the Downstream Effector PARP1 Attenuates Tivozanib-Induced Hypertensive Cardiac Injury in a Tumor-Bearing Mouse Model

Caie Li*, Liping Qiao, Heng Yu, Jing Yu

Lanzhou University Second Hospital

Corresponding Author E-mail: caie_li@126.com

Background： Cardiovascular toxicity limits the clinical use of vascular endothelial growth factor receptor tyrosine kinase inhibitors (VEGFR-TKIs), yet the mechanisms linking these agents to myocardial remodeling remain incompletely understood, particularly in the tumor-bearing setting. Tivozanib is a potent and selective VEGFR inhibitor used in advanced renal cell carcinoma, but whether its cardiac effects are attributable solely to hypertension or also involve intrinsic profibrotic myocardial signaling remains unclear. We investigated the mechanisms of tivozanib-induced cardiac remodeling in a tumor-bearing mouse model, focusing on upstream ROCK2 signaling and downstream PARP1-related injury.

Methods： A 786-0 clear cell renal cell carcinoma xenograft model was established in BALB/c nude mice. Tivozanib-associated hypertension was confirmed before initiation of mechanism-directed interventions. Mice were assigned to vehicle control, tivozanib alone, tivozanib plus chlorthalidone, tivozanib plus olaparib, or tivozanib plus belumosudil. Blood pressure was monitored longitudinally. Cardiac remodeling was assessed by echocardiography, cardiac magnetic resonance imaging, histological analysis, immunofluorescence, immunoblotting for collagen I, ROCK2, and α-smooth muscle actin, and transcriptomic profiling of cardiac tissue.

Results：Tivozanib induced sustained hypertension and promoted adverse cardiac remodeling in tumor-bearing mice, as evidenced by structural and functional abnormalities on echocardiography and cardiac magnetic resonance imaging, increased myocardial fibrosis on histological examination, and upregulation of profibrotic markers including collagen I, ROCK2, and α-smooth muscle actin. Transcriptomic profiling identified a ROCK2-centered profibrotic remodeling program in tivozanib-treated hearts. Although chlorthalidone improved blood pressure control, it provided incomplete protection against myocardial structural and molecular remodeling, indicating that the cardiac phenotype was not fully explained by hypertension alone. In contrast, ROCK2 inhibition with belumosudil substantially attenuated tivozanib-induced cardiac remodeling and suppressed profibrotic signaling. PARP1 inhibition with olaparib also mitigated myocardial injury and fibrosis, supporting a contributory role for downstream PARP1-associated injury signaling in this process.

Conclusion：Tivozanib induces cardiac remodeling in tumor-bearing mice through mechanisms that extend beyond blood pressure elevation alone and involve a ROCK2-centered profibrotic program with downstream PARP1-associated injury signaling. Targeting ROCK2 and the downstream effector PARP1 confers greater cardioprotection than conventional antihypertensive therapy alone and may represent a promising mechanism-based strategy to limit VEGFR-TKI-associated cardiac injury.

Key words：Onco-hypertension，tivozanib，ROCK2，PARP1

论文 ID：111

The miR-192-5p/DHFR/eNOS Axis Regulates Oxidative Stress and Vascular Dysfunction in Hypertension

Bo Zhou, Changqing Yu*

重庆市璧山区人民医院

Corresponding Author E-mail: 2269194173@qq.com

Background：Hypertension is characterized by endothelial dysfunction, oxidative stress, and vascular remodeling. Dihydrofolate reductase (DHFR) plays a critical role in maintaining endothelial nitric oxide synthase (eNOS) coupling and nitric oxide (NO) bioavailability. However, the involvement of miR-192-5p in regulating DHFR signaling during hypertension remains unclear.

Methods：Hypertension was induced in wild-type mice by angiotensin II (Ang II) infusion. Mice received tail-vein injections of a miR-192-5p inhibitor or negative control. Blood pressure was monitored throughout the study. Vascular function was assessed using wire myography. Reactive oxygen species (ROS) and nitric oxide (NO) production were evaluated by dihydroethidium (DHE) and DAF-FM fluorescence imaging, respectively. Expression of miR-192-5p was measured by quantitative PCR, while DHFR and eNOS expression were analyzed by immunohistochemistry.

Results：Ang II infusion significantly elevated blood pressure and impaired endothelium-dependent vasorelaxation. Inhibition of miR-192-5p attenuated the hypertensive response and improved vascular relaxation. Ang II markedly increased vascular ROS production and reduced NO bioavailability, whereas miR-192-5p inhibition reversed these alterations. Furthermore, DHFR and eNOS expression were substantially reduced in Ang II-treated mice but

restored following miR-192-5p inhibition. Histological analysis demonstrated that inhibition of miR-192-5p attenuated Ang II-induced vascular hypertrophy and remodeling.

Conclusion： miR-192-5p contributes to the development of hypertension and vascular dysfunction. Pharmacological inhibition of miR-192-5p restores DHFR/eNOS signaling, reduces oxidative stress, improves endothelial function, and attenuates vascular remodeling. These findings identify miR-192-5p as a potential therapeutic target for hypertension.

Key words： Hypertension, ROS, DHFR, miR192-5

论文 ID：112

Anthocyanin attenuates pulmonary arterial hypertension and associated heart failure via improving mitochondrial function through Nrf2-dependent mechanism

Bo Zhou[1,2], Liu Xiao[1], Changqing Yu*[1]

1. 重庆医科大学附属璧山医院

2. People's Hospital of Bishan District of Chongqing

Corresponding Author E-mail: hangqingyu-2@163.com

Background： Mitochondrial dysfunction of pulmonary vascular endothelial cells is one of the important pathogenesis of pulmonary arterial hypertension (PAH). Anthocyanin can protect mitochondrial function, but whether anthocyanin can prevent PAH and its related mechanism has not been reported.

Methods： Using rodent PAH models induced by chronic hypoxia for 21 days, we investigated the changes of hemodynamics, histopathology and vascular endothelial function after anthocyanin treatment for 21 days.

Results： We found that anthocyanin treatment improved pulmonary endothelial function, vascular remodeling and associated right heart failure. Mechanistically, we found that anthocyanin treatment promoted the nucleus translocation of Nrf2 and improved the impaired mitochondrial function. The beneficial effects of anthocyanin on regulation of mitochondrial function were abolished by inhibition of Nrf2. Finally, in Nrf2 deficient mice, the protective effects of anthocyanin on PAH were largely vanished.

Conclusion： Collectively, we showed that anthocyanin attenuated PAH and associated right heart failure via improving mitochondrial function through Nrf2-dependent mechanism, suggesting that anthocyanin may represent a novel therapeutic potential for PAH.

Key words： Anthocyanin; pulmonary arterial hypertension; right heart failure; mitochondrial function; Nrf2

论文 ID：114

Inhibition of DYRK1A by Harmine Ameliorates Ferroptosis and Vascular Smooth Muscle Cell Osteogenic differentiation in VitD3-overloaded induced Vascular Calcification

Qi Wang, Ruiqiang Qi, Xiaoyan Yang, Jiuchang Zhong*

首都医科大学附属北京朝阳医院

Corresponding Author E-mail: jczhong@sina.com

Background： Vascular calcification (VC) critically exacerbates arterial stiffness, a key pathophysiological driver of isolated systolic hypertension and increased pulse pressure, yet effective treatments remain limited. Harmine, a natural product-derived DYRK1A inhibitor, holds promise for cardiovascular protection, but its specific role in VC pathogenesis remains elusive.

Methods： Wild-type male C57BL/6 mice were injected subcutaneously with Vitamin D3 (VitD3,5.5*10^5 IU/kg/day) to induce VC, with a subset receiving Harmine. Concurrently, primary rat vascular smooth muscle cells (VSMCs) were exposed to calcification medium (CM; β-GP+ CaCl2) to model osteogenic-differentiation under Harmine treatment or siRNA-mediated DYRK1A knockdown.

Results： Aortic tissue analysis from VitD3-treated mice showed an upregulation of DYRK1A expression alongside a decreased level of the contractile marker α-SMA. Consistently, CM stimulation increased both the protein and mRNA levels of DYRK1A in primary VSMCs. Pharmacological inhibition of DYRK1A by Harmine in-vivo strikingly reduced VC, as evidenced by decreased Von Kossa and Alizarin Red S positive areas, lower aortic calcium content, and the downregulation of Runx2 and BMP2. In-vitro, disrupting DYRK1A via Harmine or siRNA inhibited VSMCs osteogenic differentiation, diminishing Alizarin Red S/ALP staining and calcium accumulation while restoring α-SMA expression. Mechanistically, CM stimulation induced ferroptosis in VSMCs, whereas Harmine suppressed this effect, lowering ROS, iron, and MDA levels while maintaining GPx activity. Furthermore, Erastin-induced ferroptosis activation accelerated matrix calcification and osteogenic marker expression; however, simultaneous Harmine treatment blocked this acceleration, reducing calcium deposition despite Erastin presence.

Conclusion： This study demonstrates for the first time that the DYRK1A inhibitor Harmine alleviates VC and VSMCs osteogenic differentiation by suppressing ferroptosis. These findings highlight Harmine and the DYRK1A-ferroptosis axis as promising therapeutic targets for VC.

Key words： Harmine; Vascular calcification; Ferroptosis; DYRK1A; Vascular smooth muscle cells.

论文 ID：115

Prognostic value of four insulin resistance indices in predicting new-onset hypertension: a retrospective cohort study

Xinyue Yang, Qingwei He, Yukun Cao, Haitao Zhang*

中国人民解放军空军特色医学中心

Corresponding Author E-mail: kjzht@sina.com

Background： Primary prevention of hypertension depends on early detection of high-risk individuals. Several risk-stratification tools are provided by lipid-glucose-insulin from standard blood testing. We compared the predictive performance of four indices for incident hypertension: Atherogenic Index of Plasma (AIP), Triglyceride-Glucose (TyG) index, Cholesterol-HDL-Glucose (CHG) index, and Metabolic Score for Insulin Resistance (METS-IR).

Methods： A total of 1079 adults with initially normal blood pressure were enrolled between January 2016 and January 2019 and followed up until January 2024. Multivariable Cox proportional hazards models were used to assess the associations between the four insulin resistance indices and the incidence of hypertension, adjusting for a comprehensive set of clinical and biochemical confounders. Time-dependent receiver operating characteristic (ROC) curve analysis was used to assess predictive accuracy at 12, 36, and 60 months.

Results： Among the participants, 297 developed hypertension during follow-up. In the fully adjusted model, only the CHG index maintained a statistically independent association with hypertension risk. Each unit increase in the CHG index was associated with a more than twofold higher risk of hypertension (Hazard Ratio [HR] 2.186, 95% Confidence Interval [CI] 1.269-3.766). However, the AIP, TyG index, and METS-IR index showed no statistical significance after final adjustment. According to time-dependent ROC analysis, both the CHG index and METS-IR showed excellent and consistent discriminative capacity across initial, medium, and long-term follow-up (AUCs consistently >0.71). The TyG index showed intermediate predictive performance, whereas AIP consistently exhibited the lowest predictive power at all intervals.

Conclusion： The CHG index demonstrated the most independent association with incident hypertension, better than the AIP, TyG, and METS-IR indices in predictive accuracy.

Key words： hypertension, insulin resistance, risk prediction, metabolic indices, triglyceride-glucose index, cholesterol, high-density lipoprotein, glucose index (CHG index)

论文 ID：131

Prognostic Value of the Kings Score for Long-Term Survival in Patients With Coronary Artery Disease

Ying Pan[1], Ting-Ting Wu[2], Chang-Jiang Deng[1], Ying-Ying Zheng[1], Xiang Xie*[1]

1. 新疆医科大学第一附属医院

2. 新疆医科大学

Corresponding Author E-mail: xiangxie999@sina.com

Background： The prognostic significance of Kings score (KS) in patients with coronary artery disease (CAD) has not been investigated. This study aimed to evaluate the relationship between KS and adverse clinical outcomes in CAD patients.

Methods： We prospectively analyzed 12686 CAD patients and devided them into two groups [KS: age × aspartate aminotransferase (AST) × international normalized ratio (INR) / platelet count (PC)], low KS: <6.6 (n = 7434),

high KS: ≥6.6 (n = 5252)]. Multivariate Cox regression models were constructed to assess the association between KS and adverse outcomes in patients with CAD.

Results： During a median follow-up period of 24 months, a total of 376 ACMs, 278 CMs, 967 MACEs and 1062 MACCEs were recorded. The incidences of these adverse outcomes were significant different between two groups (all Ps < 0.001). Meanwhile, RCS functions demonstrated that there was a liner relationship between KS and adverse outcomes. Compared to those in low KS group, the risk of ACM and CM in high KS group increased 69.5% [adjusted HR: 1.695, 95% CI: 1.265-2.271, P <0.001] and 48.7% [adjusted HR: 1.487, 95% CI: 1.068-2.069, P <0.019] , respectively.

Conclusion： Elevated KS was associated with long-term mortality and could serve as an independent and excellent index for assessing long-term mortality within the CAD population.

Key words： Kings score, mortility, predictor, coronary artery disease

论文 ID：132

Serum Butyrylcholinesterase as an Independent Predictor of All-Cause Mortality in Heart Failure: A 10-Year Real-World Cohort Study

Ying Pan[1], Ting-Ting Wu[2], Chang-Jiang Deng[1], Ying-Ying Zheng[1], Xiang Xie*[1]

1. 新疆医科大学第一附属医院

2. 新疆医科大学

Corresponding Author E-mail: xiangxie999@sina.com

Background： Serum butyrylcholinesterase (BChE) is an important biomarker in liver cirrhosis and can reflect the nutritional status in daily practice. However, the relationship between BChE and mortality in heart failure (HF) patients remaineds unclear. This study aims to evaluate the predictive value of BChE on mortality in HF patients.

Methods： From January 2012 to January 2022, a total of 2331 HF patients with available BChE data were retrospectively enrolled and followed up in this observational study. They were classified into two groups according to BChE at admission: low-BChE (< 4658 U/L, n = 796) group and high-BChE (≥ 4658 U/L, n = 1535) group. And we conducted further studies in HF patients of different ages. The longest follow-up time was 10 years. The endpoint of this study was all-cause mortality (ACM).

Results： In the total cohort, there were 1116 ACMs were recorded. The low-BChE group had higher incidence of ACM (71.1% vs 35.8%, P<0.001) compared with the high-BChE group. Multivariate Cox regression analysis showed that the risk of ACM in low-BChE group increased 87.1% (hazard risk [HR] = 1.871, 95% confidence interval [CI]: 1.520-2.303, p <0.001), compared with that in high-BChE group. A similar relation was observed in patients aged <60 years and patients aged ≥60 years.

Conclusion： BChE was an independent and powerful predictor for ACM in HF patients, regardless of age.

Key words： Butyrylcholinesterase, Heart failure, All-cause mortality, Predictor

论文 ID：133

An elevated uric acid to high-density cholesterol ratio (UHR) is associated with mortality in coronary artery disease patients: Evidence from a large prospective cohort

Ying Pan[1], Ting-Ting Wu[2], Chang-Jiang Deng[1], Ying-Ying Zheng[1], Xiang Xie*[1]

1. 新疆医科大学第一附属医院

2. 新疆医科大学

Corresponding Author E-mail: xiangxie999@sina.com

Background： To investigate the relationship between uric acid to high-density lipoprotein cholesterol ratio (UHR) and long-term clinical outcomes of patients with coronary artery disease (CAD).

Methods： A prospective study used data from the PRACTICE study constructed from December 2016 to October 2021. The primary exposure was UHR value, calculated by dividing UA (mg/dL) by HDL (mg/dL) and multiplying by 100. All participants were divided into four groups according to UHR value: Q1(UHR ≤ 6.5, n =3561), Q2(6.5 < UHR ≤ 8.7, n = 3542), Q3(8.7 < UHR ≤ 11.6, n = 3642), Q4(UHR > 11.6, n = 3530).The primary outcome was mortality, including all-cause mortality (ACM) and cardiac mortality (CM). The longest follow-up period was 5 years. Multivariable COX regression was performed to assess the relationship between the UHR and adverse events in CAD paitients.

Results： Overall, in 14 275 participants, 674 patients died during a median follow-up period of 24 months, of which 389 (57.7%) were attributed to ACM. The lowest-Q1 group had a significantly higher incidence of ACM ($p < 0.001$), CM ($p < 0.001$), major adverse cardiac events (MACEs) ($p < 0.001$), and major adverse cardiac and cerebrovascular events (MACCEs) ($p = 0.006$). Kaplan–Meier analysis revealed that the higher UHR was positively associated with adverse outcomes in CAD patients. After adjusting for a number of conventional factors, multivariate Cox regression analysis demonstrated that excess elevated UHR was linked with the increasing risk of ACM and CM. Namely, compared with Q1 group, the hazard ratios (HRs) for Q4 group were 4.164 [95%CI (1.847-9.386), p=0.001] and 2.693 [95%CI (1.092-6.639), p=0.031] for ACM and CM, respectively; Subgroup analysis showed that association of UHR with ACM was remarkable among individuals with ACS [HR: 4.011, 95%CI (1.394-11.537)].

Conclusion： This study revealed that an elevated UHR could predicted increased risk of long-term mortality in CAD patients.This finding suggested that the UHR might be a promising indicator for evaluating adverse outcomes of CAD patients.

Key words： uric acid to high-density cholesterol ratio (UHR), coronary artery disease, mortality, acute coronary syndrome

论文 ID：134

Both Low and High Hematocrit Predict Increased Long-term Mortality in CAD Patients: A U-shaped Relationship from the PRACTICE Study

Ting-Ting Wu[1], Ying Pan[2], Chang-Jiang Deng[2], Ying-Ying Zheng[2], Xiang Xie*[2]

1. 新疆医科大学

2. 新疆医科大学第一附属医院

Corresponding Author E-mail: xiangxie999@sina.com

Background： Hematocrit (HCT) was one of the major determinants of blood viscosity, which could indicate susceptibility to thrombosis. However, evidence is scarce on the long-term prognostic value of HCT in coronary artery disease (CAD) patients. This study aims to clarify this issue.

Methods： We conducted a large prospective cohort study at Xinjiang Medical University Affiliated First Hospital from December 2016 to October 2021. 15,237 CAD patients, including 11,271 males and 3,966 females, were divided into four groups based on HCT quartile and were regularly followed up.The maximum duration was60 months. We evaluated the incidence of all-cause mortality (ACM), cardiac mortality (CM) as the primary endpoints.

Results： A U-shaped relationship was observed between HCT levels and long-term adverse outcomes, with the risk of poor long-term outcomes being lowest when HCT values ranged from 45% to 50% in males and 35% to 40% in females. After adjusting for a number of conventional confounders, including hypertension and diabetes mellitus, age, and smoking, multivariate Cox regression analysis showed that excess elevated or low levels of HCT were associated with an increased risk of ACM and CM.After additional adjustment for various critical risk factors, the nonlinear relationship still persisted in males.Namely, compared with the reference group, when one with HCT < 40%, the hazard ratio(HR) of ACM was 1.772, 95%CI (1.162-2.703), P=0.008; the HR of CM was 2.041,95%CI (1.212-3.437), P=0.007; when one with HCT ≥ 50%, the HR of ACM was 2.241(1.304-3.852), P=0.003] and HR of CM was 3.025(1.627-5.624), P<0.001].

Conclusion： This study identified a U-shaped relationship between HCT values and long-term adverse outcomes.. HCT may be a powerful predictor of long-term mortality in people with CAD.

Key words： Hematocrit, Coronary artery disease, Mortality, Male

论文 ID：135

Multi-Omics-Based Mendelian Randomization: Effects and Mechanisms of Insomnia-Related Genes on Myocardial Infarction

Ting-Ting Wu*[1], Ying Pan[2], Yun-Lei Wang[1], Hai-Xi Zhu[1], Xiang Ling[1], Jiang-Tao-Chen Fu[1]

1. 新疆医科大学

2. 新疆医科大学第一附属医院

Corresponding Author E-mail: 1255723526@qq.com

Background： Insomnia has been recognized as an independent risk factor for myocardial infarction (MI), but the underlying molecular mechanisms remain unclear. This study aimed to identify key biomarkers that are dysregulated in insomnia and persistently associated with MI risk, and to characterize their regulatory networks and immune landscape.

Methods： We integrated insomnia-related genes from bulk transcriptomic data (GSE66360 and GSE60993), and GWAS summary data for MI (ebi-a-GCST90018877). Differentially expressed genes (DEGs) were identified, followed by functional enrichment analysis. Two-sample Mendelian randomization (MR) was performed to evaluate causal effects of candidate genes on MI risk. Machine learning algorithms (Stepglm[both]+GBM) were applied to refine key biomarkers, which were validated in external cohorts. Immune infiltration was assessed by ssGSEA, and single-cell RNA-seq (GSE269269) was used to explore cellular heterogeneity and cell-cell communication.

Results： A total of 2,584 DEGs were identified, of which 401 overlapped with insomnia-related genes. MR analysis revealed nine genes with significant causal associations with MI, including protective factors (CYB561, RORC, VEGFA, NCAPD2, ARG1) and risk factors (CD177, WDR59, LYSMD2, IFI44L). Machine learning integration identified CD177 (AUC = 0.778) and RORC (AUC = 0.787) as definitive diagnostic biomarkers. A nomogram incorporating these two biomarkers achieved robust predictive performance (AUC = 0.826). Immune infiltration analysis showed CD177 negatively correlated with Macrophages, while RORC positively correlated with Central memory CD4 T cells. Single-cell analysis revealed CD177 predominantly expressed in Monocytes and RORC enriched in T cells and CD34 cells. Virtual knockout of CD177 implicated regulation of Th1/Th17 differentiation pathways.

Conclusion： This study identifies CD177 and RORC as robust diagnostic biomarkers linking insomnia to MI, providing insights into inflammatory mechanisms and offering potential targets for risk stratification and therapeutic intervention.

Key words： Insomnia; Myocardial infarction; Mendelian randomization; Multi-omics; Immune infiltration

论文 ID：136

Intervention-Modified Associations between Cumulative Blood Pressure Burden and Long-Term Functional Outcomes Among People With Stroke in Rural China

Zian Zheng[1,2], Bolu Yang[1,4], Xiaoyan Yan[3], Fei Wu[5,9], Xiong Ding[6], Xingxing Chen[1,4], Brian Oldenburg[7,10], Haizhu Song[1], Yuxuan Zhou[1], Hanting Zhang[1], Bifeng Yuan[4], Lijing Yan[1,4,11], Enying Gong*[8,12]

1. Global Health Research Center, Duke Kunshan University

2. Duke Kunshan University

3. Clinical Research Institute, Institute of Advanced Clinical Medicine Peking University

4. School of Public Health, Wuhan University

5. Saw Swee Hock School of Public Health, National University of Singapore

6. Center for Endemic Disease Control, Chinese Center for Disease Control and Prevention, Harbin Medical University

7. Baker Heart and Diabetes Institute

8. School of Population Medicine and Public Health, Chinese Academy of Medical Sciences & Peking Union Medical College

9. Global Health and Population, Harvard T.H. Chan School of Public Health

10. La Trobe University

11. Duke Global Health Institute, Duke University

12. State Key Laboratory of Respiratory Health and Multimorbidity, Chinese Academy of Medical Sciences & Peking Union Medical College

Corresponding Author E-mail: gongenying@cams.cn

Background： The one-year SINEMA trial demonstrated improved blood pressure (BP) control and reduced mortality up to 72 months post-intervention. This study assessed between-arm differences in mean annual cumulative BP and whether cumulative BP–biofunctional outcome associations differed by trial arm.

Methods： We conducted a post-hoc secondary analysis of the SINEMA cluster-randomized trial, recruiting 1299 adults with stroke from 50 rural villages in Hebei, China (2017-2018). The 12-month intervention was followed by observations at 72 and 84 months.

BP was measured by blinded assessors at baseline, 12, 72, and 84 months. Mean annual cumulative systolic BP (SBP), diastolic BP (DBP), mean arterial pressure (MAP), and pulse pressure (PP) were calculated. Biofunctional outcomes included health-related quality of life, modified Rankin Scale, activities of daily living, physical function, and cognition.

Results： Among 897 participants (mean age 65.0 years; 46.0% female) with complete data, the intervention arm demonstrated significantly lower mean annual cumulative SBP (−2.2 mm Hg; 95% CI, −3.9 to −0.6), DBP (−1.6 mm Hg; 95% CI, −2.4 to −0.7), and MAP (−1.8 mm Hg; 95% CI, −2.8 to −0.8), than usual care. Higher cumulative BP was significantly associated with poorer biofunctional outcomes in the control but not intervention arm. Arm-by-cumulative-BP interactions were significant for several outcomes, particularly SBP.

Conclusion： SINEMA intervention was linked to lower cumulative BP over 72-84 months but did not enhance biofunctional outcomes. The BP–biofunctional outcome relationship varied by trial arm, suggesting cumulative BP exposure may indicate long-term risk and that the intervention may alter BP–outcome relationships.

Key words： Stroke; Cumulative Blood Pressure; Biofunctional Outcomes; Digital Health Intervention; Secondary Prevention

论文 ID：149

Obese ADSC-Derived Exosomal SNHG8 Promotes Obesity-Related Hypertension Through HuR-Mediated Stabilization of COX-2 mRNA

Xiaoyong Hu[1], Hongjian Li*[2]

1. Department of Cardiology, The Second Affiliated Hospital of Henan University of Science and Technology

2. Department of Hypertension, The Fifth Affiliated Hospital of Xinjiang Medical University,

Corresponding Author E-mail: lhjdoctor_109@126.com

Background： Obesity-related hypertension is a major cardiometabolic disorder characterized by endothelial dysfunction, vascular remodeling, and imbalance between vasodilatory and vasoconstrictive mediators. Although adipose tissue is increasingly recognized as an active endocrine and paracrine organ, the molecular communication between obese adipose-derived stem cells (ADSCs) and vascular endothelial cells remains incompletely understood. Exosomes derived from obese ADSCs may serve as pathogenic carriers that transmit obesity-associated non-coding RNA signals to the vasculature. This study aimed to determine whether obese ADSC-derived exosomes promote obesity-related hypertension by delivering long non-coding RNA SNHG8 to endothelial cells and to elucidate the underlying HuR/COX-2-dependent mechanism.

Methods： A male Sprague-Dawley rat model of obesity-related hypertension was established by high-fat diet feeding. ADSCs were isolated from epididymal adipose tissue of normal and obese rats, and ADSC-derived exosomes were extracted, purified, and characterized. Normal ADSC-derived exosomes (N-Exo), obese ADSC-derived exosomes (H-Exo), and exosomes with SNHG8 knockdown or related molecular interventions were administered via tail vein injection. Blood pressure, metabolic parameters, and aortic pathological alterations were evaluated. In vitro, rat aortic endothelial cells were treated with different ADSC-derived exosomes and subjected to lentivirus-mediated SNHG8 knockdown or overexpression. RT-qPCR, Western blotting, enzyme-linked immunosorbent assay, RNA immunoprecipitation, and mRNA stability assays were performed to investigate the regulatory effects of SNHG8 on COX-2 expression and downstream vasoactive mediators, including prostacyclin, angiotensin II, and thromboxane A_2.

Results： Compared with N-Exo, H-Exo markedly increased blood pressure, aggravated metabolic abnormalities, and promoted aortic wall thickening in obese hypertensive rats. Mechanistically, H-Exo carried abundant SNHG8 and efficiently transferred SNHG8 into vascular endothelial cells. Elevated SNHG8 disrupted endothelial vasoactive homeostasis, characterized by reduced prostacyclin production and increased levels of angiotensin II and thromboxane A_2, thereby favoring vasoconstriction and vascular injury. SNHG8 knockdown in H-Exo significantly attenuated blood pressure elevation, improved metabolic disturbance, and alleviated aortic pathological remodeling. In endothelial cells, SNHG8 upregulated COX-2 expression at the post-transcriptional level rather than merely acting as a passive exosomal biomarker. RNA immunoprecipitation and mRNA stability assays demonstrated that SNHG8 recruited the RNA-binding protein HuR to COX-2 mRNA, enhanced COX-2 mRNA stability, and consequently increased COX-2 protein abundance. Functionally, knockdown of either SNHG8 or COX-2 significantly reversed H-Exo-induced endothelial dysfunction and restored the imbalance between vasodilatory and vasoconstrictive factors.

Conclusion： This study identifies obese ADSC-derived exosomal SNHG8 as a previously unrecognized pathogenic signal linking adipose dysfunction to vascular endothelial injury in obesity-related hypertension. H-Exo delivers SNHG8 into endothelial cells, where SNHG8 recruits HuR to stabilize COX-2 mRNA, leading to aberrant COX-2 upregulation, vasoactive factor imbalance, endothelial dysfunction, vascular remodeling, and blood pressure elevation. These findings reveal a novel exosome-mediated adipose–vascular communication mechanism and suggest that targeting the SNHG8/HuR/COX-2 axis may represent a promising therapeutic strategy for obesity-related hypertension.

Key words： Obesity-related hypertension; Adipose-derived stem cell exosomes; Long non-coding RNA SNHG8; Cyclooxygenase-2

论文 ID：152

Real-World Data and Drug-Target Mendelian Randomization Analysis of Adverse Reactions to Dihydropyridine Calcium Channel Blockers

Hanshuo Li*

郑州大学

Corresponding Author E-mail: lhshuoo@163.com

Background： Dihydropyridine calcium channel blockers (DHP-CCBs) are widely used in the treatment of hypertension and related cardiovascular diseases. Although this class of drugs is generally considered safe, long-term and widespread use may still lead to various adverse drug reactions (ADRs). This study aimed to identify adverse event signals associated with DHP-CCBs using real-world data and to evaluate potential causal associations, providing evidence to support safer clinical use.

Methods： Adverse event reports related to DHP-CCBs were collected from the Henan ADR Monitoring Database and the U.S. Food and Drug Administration Adverse Event Reporting System (FAERS) from January 1, 2010, to December 31, 2023. Signal detection was performed using the reporting odds ratio (ROR) method and proportional reporting ratio (PRR) method. In addition, drug-target Mendelian randomization (MR) was applied to assess potential causal relationships.

Results： A total of 30,189 and 26,066 reports related to DHP-CCBs were identified from the Henan ADR database and the FAERS database, respectively. We detected 52 and 838 positive signals at the preferred term (PT) level in the two databases. 32 overlapping positive signals were identified across both databases, including non-infectious gingivitis, gingival hypertrophy, periodontal disease, oedema peripheral, and nocturia. MR analysis suggested that DHP-CCBs may increase the risk of polyuria through their action on antihypertensive drug targets.

Conclusion： Multiple adverse event signals associated with DHP-CCBs were identified, including gingival hypertrophy, oedema peripheral, and nocturia. Polyuria may represent a potential adverse reaction worthy of further investigation. In clinical practice, the benefits and risks of DHP-CCBs should be carefully evaluated.

Key words： dihydropyridine calcium channel blockers; adverse drug reaction; drug safety monitoring; disproportionality analysis; Mendelian randomization

论文 ID：154

Association between the Serum uric acid-to-creatinine ratio index and the Risk of Preeclampsia in Elderly pregnant women: A Retrospective Cohort Study

Sulan Huang*

The first people’s hospital of Changde city

Corresponding Author E-mail: huangsulanjiayou3@126.com

Background： The serum uric acid-to-creatinine ratio (SUA/sCr) has been proposed as an indicator of systemic oxidative stress. However, evidence regarding its association with preeclampsia remains scarce. This study investigated the relationship between SUA/sCr levels and the risk of preeclampsia among women of advanced maternal age.

Methods： A total of 2,296 pregnant women aged≥35 years were included. Multivariable logistic regression models were applied to examine the association between SUA/sCr and preeclampsia, generating odds ratios (ORs) and 95% confidence intervals (CIs). Restricted cubic spline analysis was performed to assess the dose-response pattern, and subgroup analyses were conducted to explore potential effect modification.

Results： After adjustment for age, gravidity, parity, and family history of hypertension, each standard deviation increase in SUA/sCr was associated with a 29% higher risk of preeclampsia (OR = 1.29; 95% CI: 1.13–1.47; P = 0.0001). A threshold value of 8.18 (95% CI: 7.60–8.55) was identified. Below this threshold, the risk of preeclampsia increased significantly (OR = 1.23; 95% CI: 1.11–1.36; P < 0.0001), whereas no significant association was observed above it (OR = 1.02; 95% CI: 0.89–1.18; P = 0.7561). The calculated E-value of 1.73 suggests that substantial unmeasured confounding would be required to fully explain the observed association. Subgroup analyses demonstrated a significant interaction with urinary protein status (interaction P = 0.0146).

Conclusion： Higher SUA/sCr levels are independently associated with an increased risk of preeclampsia in women aged 35 years and older. As a simple and accessible biomarker, SUA/sCr may aid in early identification of high-risk individuals within this population.

Key words： Serum uric acid-to-creatinine ratio, Preeclampsia, Oxidative stress, Advanced maternal age, Pregnant women

论文 ID：155

Elevated Red Cell Distribution Width-to-Albumin Ratio as a Risk Factor for Preeclampsia in Advanced Maternal Age: Implications for Clinical Risk Assessment

Sulan Huang*

The First People's Hospital of Changde City

Corresponding Author E-mail: huangsulanjiayou3@126.com

Background： Preeclampsia is a leading cause of maternal and perinatal morbidity, particularly among women of advanced maternal age (AMA). Simple, inexpensive biomarkers for early risk stratification in this population are lacking. The red cell distribution width-to-albumin ratio (RAR), an integrated index of erythrocyte anisocytosis and hypoalbuminemia, has emerged as a marker of systemic inflammation and vascular stress in cardiovascular and critical care settings. Its relevance to preeclampsia is unknown.

Methods： We conducted a retrospective cohort study of 2,296 women aged ≥35 years with singleton pregnancies delivering at a tertiary hospital between 2013 and 2023. RAR was calculated as red cell distribution width (RDW, %) divided by serum albumin (g/L) and analyzed as RAR multiplied by 10 (RAR×10) and by tertiles. Preeclampsia was diagnosed after 20 gestational weeks using contemporary criteria. Multivariable logistic regression, restricted cubic splines, and prespecified subgroup analyses were performed to evaluate associations between RAR and preeclampsia.

Results： The mean age was 37.8 ± 2.7 years; 328 women (14.3%) developed preeclampsia. Preeclampsia prevalence increased across RAR tertiles (9.9%, 14.1%, 18.8%; p<0.001). Each one-unit increase in RAR×10 was associated with higher odds of preeclampsia (adjusted OR 1.15, 95% CI 1.02–1.28). Compared with the lowest tertile, adjusted ORs were 1.58 (95% CI 1.11–2.24) and 2.03 (95% CI 1.45–2.86) for tertiles 2 and 3, respectively (p-trend<0.001). Restricted cubic splines revealed a J-shaped association, with risk rising steeply above RAR×10≈3.95. The association was markedly stronger in women with BMI ≥28 kg/m² (p-interaction<0.001) and was broadly consistent across other clinical subgroups.

Conclusion： Among AMA pregnancies, elevated RAR is independently and nonlinearly associated with the risk of preeclampsia, with particularly pronounced effects in obese women. Given its ubiquity, low cost, and analytic simplicity, RAR×10 may serve as a practical and inexpensive biomarker to aid early risk stratification in routine prenatal care.

Key words： red cell distribution width-to-albumin ratio; RAR; preeclampsia; advanced maternal age; inflammatory biomarker; pregnancy

论文 ID：194

Higher Triglycerides to High-Density Lipoprotein Cholesterol (TG/HDL-C) Ratio increases of risk of chronic kidney disease in hypertensive and abnormal glucose metabolism patients:a cohort study

Xiufang Li, qing Zhu, nanfang li*

新疆维吾尔自治区人民医院

Corresponding Author E-mail: 987947586@qq.com

Background： The triglyceride/high-density lipoprotein cholesterol (TG/HDL) index is a recognized surrogate marker of insulin resistance. Our previous studies using this cohort have demonstrated that other insulin resistance-related indices, such as the triglyceride-glucose (TyG) index, as well as plasma aldosterone and renin levels, are independently associated with chronic kidney disease (CKD) risk in hypertensive patients with abnormal glucose metabolism (AGM). However, the specific relationship between the TG/HDL index and incident CKD in this high-risk population remains unexplored. This study aimed to investigate whether the TG/HDL index is associated with new-onset CKD in the same cohort.

Methods： This retrospective cohort study included 2,033 hypertensive patients with AGM from a previously described prospective cohort (detailed inclusion/exclusion criteria reported elsewhere). Patients were stratified into tertiles according to baseline TG/HDL index: Tertile 1 (≤1.40, n=675), Tertile 2 (1.41–2.31, n=681), and Tertile 3 (≥2.32, n=677). The primary outcome was incident CKD, defined as estimated glomerular filtration rate <60 mL/min/1.73 m². Multivariate Cox proportional hazards regression models, adjusted for potential confounders were used to assess the independent association between the TG/HDL index and CKD risk. Restricted cubic spline analysis was performed to explore non-linear dose-response relationships.

Results： During a median follow-up of 30.8 months, 302 participants developed CKD. The incidence rates per 1,000 person-years increased significantly across tertiles: 41.84 (Tertile 1), 55.56 (Tertile 2), and 78.38 (Tertile 3). Per 1-unit increase in the TG/HDL index was independently associated with an elevated CKD risk (adjusted hazard ratio

[HR]: 1.05, 95% confidence interval [CI]: 1.01–1.10, P=0.019). Compared with Tertile 1, patients in Tertile 3 had a 58% higher risk of CKD (HR: 1.58, 95% CI: 1.17–2.15, P=0.003). Restricted cubic spline analysis revealed a significant non-linear association (P for non-linearity=0.015), with CKD risk rising rapidly as the TG/HDL index increased. Sensitivity analyses, excluding patients with follow-up <12 months or extreme triglyceride levels (>3 mmol/L or >7 mmol/L), confirmed the robustness of these findings.

Conclusion： An elevated TG/HDL index is independently and non-linearly associated with an increased risk of incident CKD in hypertensive patients with AGM. The TG/HDL index, a simple and low-cost biomarker, may complement existing risk stratification tools in this high-risk population. Further studies are warranted to determine whether interventions targeting the TG/HDL index can reduce CKD incidence.

Key words： Triglyceride/high-density lipoprotein cholesterol index, Chronic kidney disease, Hypertension, abnormal glucose metabolism

论文 ID：196

Cumulative TyG-related indices in relation to incident arterial stiffness and carotid intima-media thickening: a 6-year follow-up within the 36-year Hanzhong Adolescent Hypertension Cohort

Teng Zhang[1,2], Jian-Jun Mu*[1]

1. 西安交通大学第一附属医院

2. The First Affiliated Hospital of Xi'an Jiaotong University

Corresponding Author E-mail: mujjun@163.com

Background： Insulin resistance promotes both arterial stiffening and carotid atherosclerosis, but the comparative value of TyG-related indices for these phenotypes is unclear. Using a cohort with childhood anthropometry measured in 1987, we compared two vascular phenotypes, functional arterial stiffness (AS) and structural carotid intima–media thickening (cIMT), in relation to a common metabolic exposure, tested independence from early-life adiposity, and examined how the choice of exposure design (baseline, cumulative, or change) shaped these associations.

Methods： Within the Hanzhong Adolescent Hypertension Cohort (established 1987), we analysed 1,294 (incident AS) and 1,408 (incident cIMT) adults free of the respective outcome in 2017 and re-examined in 2023. Eight indices (TyG, TyG-BMI, TyG-WC, TyG-WHtR, TyG-WWI, TyG-BRI, TyG-CVAI, METS-IR) were modelled as baseline, cumulative, and 6-year change exposures. Incident AS was defined as brachial–ankle pulse wave velocity ≥1,400 cm/s, and incident cIMT thickening as mean cIMT ≥0.9 mm. Logistic regression, restricted cubic splines, and discrimination/reclassification metrics were applied, with additional adjustment for childhood (1987) BMI Z-score.

Results： Over 6 years, 296 (22.9%) and 464 (33.0%) participants developed AS and cIMT thickening. All eight cumulative indices were positively associated with both outcomes (all $P < 0.05$), with stronger associations for AS. TyG-WC showed the strongest AS association (OR per 1-SD 1.83, 95% CI 1.55–2.17; $P < 0.001$), whereas cIMT associations were modest (OR 1.17–1.24; all $P < 0.05$). The 6-year change exposure was largely not associated with incident cIMT thickening. Adding the indices to a conventional model improved discrimination for AS (ΔAUC 0.079–

0.101; all DeLong P < 0.001) but only marginally for cIMT (ΔAUC 0.006–0.010). Estimates were unchanged after childhood-BMI adjustment and in sensitivity analyses.

Conclusion：Cumulative TyG-related indices, led by TyG-WC, were strongly related to incident arterial stiffness but only weakly to carotid intima–media thickening, independent of early-life adiposity. Long-term cumulative metabolic burden, rather than a single measurement or short-term change, better characterised vascular risk related to insulin resistance.

Key words：triglyceride–glucose index; insulin resistance; arterial stiffness; carotid intima–media thickness; cumulative exposure; prospective cohort

论文 ID：203

Childhood Isolated Systolic Hypertension Is Not Benign: Adult Hypertension and Cardiovascular Remodeling

Minjun Li[1], Hong Cheng[2], Jingfan Xiong[3], Pei Xiao[1], Yan Li[3], Yinkun Yan[1], Hongbo Dong[1], Jie Mi*[1]

1. Beijing Children's Hospital, Capital Medical University

2. Department of Epidemiology, Capital Institute of Pediatrics

3. Child and Adolescent Chronic Disease Prevention and Control Department, Shenzhen Center for Chronic Disease Control

Corresponding Author E-mail: jiemi12@vip.sina.com

Background：The long-term consequences of childhood hypertension (HTN) subtypes remain unclear. We examined whether childhood isolated systolic hypertension (ISH), isolated diastolic hypertension (IDH), and systolic-diastolic hypertension (SDH) are differentially associated with adult HTN and subclinical target organ damage (TOD).

Methods：This study included 1,228 participants from the Beijing Blood Pressure Cohort Study with a mean follow-up of 22.9±0.6 years. Childhood blood pressure subtypes were classified as non-hypertensive, ISH, IDH, or SDH. Adult outcomes included HTN, cardiac remodeling, arterial stiffness, kidney damage, and overall TOD, defined as the presence of any of these abnormalities.

Results：ISH was the predominant childhood hypertensive subtype (78.4%). Compared with non-hypertensive children, those with childhood ISH had a higher risk of adult HTN and cardiac remodeling and showed adverse arterial stiffness phenotypes, including higher carotid-femoral and brachial-ankle pulse wave velocity and greater risks of aortic and peripheral arterial stiffness. Childhood IDH and SDH showed distinct patterns of association, mainly involving aortic stiffness and left ventricular hypertrophy, respectively; these subgroup-specific estimates should be interpreted cautiously.

Conclusion：Childhood hypertensive subtypes were associated with differing long-term cardiovascular phenotypes, with the most consistent evidence observed for ISH. These findings support closer long-term surveillance of childhood HTN, particularly ISH.

Key words：Childhood Hypertension, Hypertensive Subtypes, Isolated Systolic Hypertension, Cardiovascular Remodeling, Cohort Study

论文 ID：213

Higher Primary Aldosteronism Prevalence in Hypertensive Patients with Abdominal Aortic Calcification

Bire_saiding Sai, qin Luo*, nanfang Li

Hypertension Center of People's Hospital of Xinjiang Uygur Autonomous Region, Urumqi, Xinjiang, People's Republic of China;

Corresponding Author E-mail: 2325386339@qq.com

Background： Abdominal aortic calcification (AAC) is more prevalent in patients with primary aldosteronism (PA) than in those with essential hypertension.We aimed to determine the prevalence of PA in hypertensive patients with AAC. Since PA promotes vascular calcification, this investigation was designed to offer practical clues for improving clinical PA screening.

Methods： This cross-sectional study recruited 1871 consecutive hypertensive patients from the Hypertension Center of People's Hospital of Xinjiang Uygur Autonomous Region from January to March 2023. AAC was assessed via adrenal thin-slice CT, and Abdominal Aortic Calcification Index (ACI）was used to quantify calcification severity. Patients were divided into AAC and non-AAC groups based on ACI measurements. All participants received standardized PA screening. Propensity score matching (PSM), subgroup analyses and multivariate logistic regression were performed to analyze the association between AAC and PA detection.

Results： Among all participants, 785 were enrolled in the AAC group and 1086 in the non-AAC group. The AAC group exhibited a significantly higher PA detection rate than the non-AAC group (21.7% vs. 13.8%, $P < 0.001$), and the difference remained significant after PSM (25.9% vs. 13.2%, $P < 0.001$). Subgroup analyses showed higher PA prevalence in AAC patients aged under 45 years, males and those with hypertension duration less than five years (all $P < 0.001$). Multivariate regression confirmed AAC as an independent risk factor for PA (OR=2.416, 95% CI: 1.786–3.267, P=0.018).

Conclusion： In conclusion, PA is prevalent among hypertensive patients with AAC. These findings underscore the importance of screening for PA in hypertensive patients exhibiting AAC, especially in young individuals or individuals who have short history of hypertension.

Key words： Primary aldosteronism, Abdominal aortic calcification, hypertension

论文 ID：215

Zinc finger protein 36 like 2 protects vasculogenesis through alleviating endothelial ferroptosis

Yang-Xi Hu*[1,2], Huihui Yuan[3], Yue Wu[3], Hongmin You[5], Meirong Bai[6], Haoran Ding[4], Hui Chen[3], Jian Zhao[3], Zhiqing He[3], Yihong Chen[3], Qing Jing[4], Chun Liang[3]

1. Daping Hospital, Army Medical University

2. 中国人民解放军陆军特色医学中心

3. Second Affiliated Hospital of Naval Medical University

4. Shanghai Institute of Nutrition and Health, University of Chinese Academy of Sciences, Chinese Academy of Sciences

5. Shanghai General Hospital, School of Medicine, Shanghai Jiao Tong University

6. Xinhua Hospital, School of Medicine, Shanghai Jiao Tong University

Corresponding Author E-mail: yxhu@tmmu.edu.cn

Background： Angiogenesis is a key process for establishing functional vasculature during embryogenesis and involves different signaling mechanisms. The RNA binding protein Zfp36l2 was reported to be involved in various diseases in different species, including cardiovascular diseases. However, whether Zfp36l2 works like mammalian Zfp36l2 and if their molecular mechanisms are different remain unclear.

Methods： We used transparent transgenic and wild-type zebrafish larvae to dynamically investigate the early stage of angiogenesis with confocal in vivo, after the knockout of Zfp36l2 by CRISPR/Cas9. In situ hybridization and fluorescence-activated cell sorting were performed to detect Zfp36l2 expression. RNA sequencing and luciferase reporter experiments were performed to further explore the role of Zfp36l2 in angiogenesis. Virtual screening of over 17800 small molecule compounds and in vivo/vitro verification were conducted to find potential drugs targeting ZFP36L2 against angiogenesis-associated diseases.

Results： We found that knockout of Zfp36l2 led to defected angiogenesis in dorsal arteries (DAs) and posterior cardinal veins (PCVs). Moreover, knockout of Zfp36l2 upregulated she expression, while knockout of she resulted in partly relief of angiogenesis defects induced by Zfp36l2 knockout. Besides, simultaneous knockout of Zfp36l2 and she alleviated the ferroptosis of endothelial cells in DAs and PCVs caused by Zfp36l2 knockout. Among the over 17800 compounds tested, SCD1 inhibitor-3 showed efficient suppression on the regulation of SHE expression of ZFP36L2 with high in vivo and in vitro safety. We also demonstrated evidence for the high binding affinity of SCD1 inhibitor-3 to ZFP36L2.

Conclusion： Our results show that Zfp36l2 is responsible for maintaining normal vessel diameter during embryogenesis partly through suppressing she expression and she-mediated ferroptosis of endothelial cells. In addition, SCD1 inhibitor-3 might be a potential drug targeting ZFP36L2 for angiogenetic diseases.

Key words： RNA binding protein; Zfp36l2; Endothelial cell; Ferroptosis; Angiogenesis; Zebrafish

论文 ID：216

Residual Metabolic Abnormality Burden and Incident Cardiovascular Disease Among Chinese Adults With Controlled Hypertension: A Landmark Cohort Study

Lerui Wang*, Boda Zhou

Beijing Tsinghua Changgung Hospital，Tsinghua University

Corresponding Author E-mail: docwlr666@163.com

Background： Blood pressure control does not necessarily indicate comprehensive cardiovascular risk control. Whether residual metabolic abnormalities identify individuals at increased cardiovascular risk after achieving guideline-recommended blood pressure targets remains insufficiently characterized.

Methods： This landmark cohort study used data from the China Health and Retirement Longitudinal Study. Participants were aged 45 years or older and had hypertension at the 2011 examination, defined by a mean systolic blood pressure of at least 140 mm Hg, a mean diastolic blood pressure of at least 90 mm Hg, a physician diagnosis of hypertension, or antihypertensive treatment. Participants with heart disease or stroke at baseline or with incident cardiovascular disease before the 2015 landmark were excluded. The primary analysis was restricted to participants with blood pressure controlled to less than 140/90 mm Hg in 2015, complete metabolic measurements, and direct cardiovascular follow-up. Residual metabolic abnormality burden was defined as the number of abnormal domains present in 2015—glycemic abnormality, elevated triglycerides, reduced high-density lipoprotein cholesterol, and central obesity—yielding a prespecified score ranging from 0 to 4. The primary outcome was first incident cardiovascular disease, defined as newly reported heart disease or stroke, from 2015 through 2020. Person-period data were constructed for the 2015–2018 and 2018–2020 intervals. Associations were estimated using discrete-time complementary log-log models with participant-clustered robust standard errors. The primary model adjusted for age, sex, educational attainment, rural or urban residence, marital status, smoking, alcohol consumption, and 2011 systolic and diastolic blood pressure. Model-standardized cumulative risks were estimated using 1000 participant-level bootstrap resamples.

Results： The cohort included 827 participants, among whom 162 incident cardiovascular disease events occurred. The primary adjusted analysis included 824 participants and 160 events. Each additional residual metabolic abnormality domain was associated with a higher hazard of incident cardiovascular disease (hazard ratio [HR], 1.192; 95% CI, 1.035–1.372; P=.015). Compared with participants with 0 or 1 abnormal domain, the HR was 1.367 (95% CI, 0.925–2.022) for those with 2 domains and 1.613 (95% CI, 1.092–2.383) for those with 3 or more domains. There was evidence of a graded association across categories (P for trend=.013), with no clear evidence of departure from linearity (P for nonlinearity=.455). Model-standardized predicted 5-year cumulative cardiovascular disease risks were 17.0%, 22.4%, and 25.7% for participants with 0 or 1, 2, and 3 or more abnormal domains, respectively. The direction of the association was generally consistent across prespecified sensitivity analyses using alternative metabolic definitions, further adjustment for 2015 blood pressure and antihypertensive treatment, and alternative cohort definitions.

Conclusion： Among Chinese middle-aged and older adults with hypertension and controlled blood pressure, a greater residual metabolic abnormality burden was associated with a higher subsequent risk of cardiovascular disease. These findings suggest that assessment of glycemic status, lipid abnormalities, and central obesity may provide clinically relevant risk information beyond whether blood pressure targets have been achieved.

Key words： hypertension; blood pressure control; metabolic abnormalities; cardiovascular disease; landmark analysis; CHARLS

论文 ID：217

Modulation of hepatic lipid homeostasis by ANP is dependent on palmitoleic acid in a mouse model of MASLD

Minghui Yang, wei song*, Delin Li

A FIRST AFFILIATED HOSPITAL OF DALIAN MEDICAL UNIVERSITY

Corresponding Author E-mail: songwei8124@163.com

Background： Metabolic dysfunction–associated steatotic liver disease (MASLD) is the leading cause of chronic liver disease, with insulin resistance as its central driver. Atrial natriuretic peptide (ANP) has emerged as a pleiotropic regulator with favorable metabolic effects, yet its role in hepatic free fatty acid (FFA) metabolism in MASLD remains poorly defined.

Methods： We conducted a case-control study enrolling 92 hypertensive patients with MASLD and 178 hypertensive controls. Plasma FFA profiles were measured, and binary logistic regression was performed to identify risk factors. In parallel, high-fat diet (HFD)-fed wild-type (WT) and liver-specific NPRA-knockout (KO) mice were treated with ANP or saline via osmotic mini-pumps. Hepatic targeted FFA metabolomics, histopathology, oxidative stress markers, inflammatory cytokines, and expression of PPARα, CPT1, and SCD1 were assessed.

Results： Clinically, BMI, fasting glucose, systolic blood pressure, TG, AST, and myristic acid were independent risk factors for MASLD, whereas palmitoleic acid and nervonic acid were protective. In mice, ANP significantly reduced body weight, liver index, serum TC, TG, ALT, and AST, attenuated hepatic steatosis and inflammation, and alleviated oxidative stress in HFD-fed WT mice. These effects were largely abolished in NPRA-KO mice. Metabolomics revealed that ANP upregulated palmitoleic acid and enriched the unsaturated fatty acid biosynthesis pathway. Mechanistically, ANP reversed HFD-induced suppression of PPARα, CPT1, and SCD1 expression at both protein and mRNA levels in WT mice, but not in KO mice.

Conclusion： ANP exerts protective effects against MASLD-associated metabolic disturbances, hepatic steatosis, inflammation, and oxidative stress through hepatic NPRA-dependent activation of the PPARα/CPT1/SCD1 signaling axis. These findings highlight ANP/NPRA signaling as a potential therapeutic target linking cardiac function to hepatic lipid metabolism in MASLD.

论文 ID：218

OX1R/BACH1-Driven Ferroptosis as a Therapeutic Target: Renal Denervation Mitigates Hypertensive Myocardial Fibrosis

Delin Li, wei song*

大连医科大学附属第一医院

Corresponding Author E-mail: songwei8124@163.com

Background： Hypertensive heart disease is characterized by cardiac fibroblast activation and excessive extracellular matrix deposition, ultimately leading to myocardial fibrosis and diastolic dysfunction. High salt intake activates orexin receptor 1 (OX1R) in the hypothalamic paraventricular nucleus (PVN), enhancing sympathetic outflow and driving peripheral sympathetic overactivity, which is a critical initiator of hypertensive target organ damage. Renal denervation (RDN) reduces systemic sympathetic activity by ablating renal sympathetic nerve fibers, but whether RDN exerts direct anti fibrotic effects beyond blood pressure lowering remains unclear, and the underlying molecular mechanisms are poorly defined. Ferroptosis, a novel iron dependent programmed cell death, has recently been

implicated as a key promoter of myocardial fibrosis. Our preliminary data revealed that OX1R and the transcriptional regulator BACH1 are upregulated in fibrotic myocardial tissues and that they exhibit a hierarchical regulatory relationship. We therefore propose that high salt intake activates the PVN OX1R/sympathetic axis, which in turn upregulates cardiac OX1R, subsequently activates ferroptosis via BACH1, and drives hypertensive myocardial fibrosis; RDN counteracts this process by suppressing this signaling axis.

Methods：(1) Animal experiments: Six week old male Dahl/SS rats were fed a high salt diet (8% NaCl) for 12 weeks. At week 6, rats were subjected to RDN, treated with the OX1R antagonist SB-408124, or received sham/control interventions. Blood pressure and echocardiographic parameters were monitored. Myocardial collagen deposition was assessed by Masson's trichrome staining. Protein and mRNA levels of fibrotic markers (α-SMA, Fibronectin, Collagen I/III), ferroptosis markers (GPX4, SLC7A11, ACSL4, etc.), and OX1R/BACH1 were measured by Western blot and RT-qPCR. (2) Cell experiments: Primary cardiac fibroblasts were isolated from Dahl/SS rats and stimulated with TGF-β1 to establish a fibrotic cell model. OX1R or BACH1 was knocked down or overexpressed via plasmid transfection, and the same panel of markers was evaluated.

Results：(1) RDN significantly reduced high salt induced blood pressure elevation, improved ejection fraction (EF%) and fractional shortening (FS%), attenuated myocardial interstitial collagen deposition, and downregulated fibrotic marker expression. (2) RDN upregulated GPX4 and SLC7A11 while downregulating ACSL4 in myocardial tissues, indicating suppressed ferroptosis; concomitantly, OX1R and BACH1 expression were markedly decreased. (3) TGF-β1 stimulation upregulated both OX1R and BACH1 in cardiac fibroblasts. Knockdown of OX1R reduced BACH1 expression, whereas OX1R overexpression increased BACH1, confirming that BACH1 acts downstream of OX1R. (4) Knockdown of OX1R or BACH1 inhibited TGF-β1 induced upregulation of α-SMA and Fibronectin, and reversed ferroptosis marker changes (increased GPX4/SLC7A11 and decreased ACSL4); overexpression produced the opposite effects.

Conclusion：RDN effectively reverses hypertensive myocardial fibrosis by downregulating the OX1R/BACH1 axis and suppressing ferroptosis in cardiac fibroblasts. This study is the first to link high salt induced PVN-OX1R/sympathetic activation with peripheral myocardial OX1R/BACH1/ferroptosis signaling, providing a novel central to peripheral mechanistic framework for the cardioprotective effects of RDN and identifying OX1R as a potential therapeutic target for anti-fibrotic drug development.

论文 ID：219

Targeting OX1R/BACH1-Driven Ferroptosis: Renal Denervation Counteracts Hypertensive Renal Interstitial Fibrosis

Delin Li, wei song*

大连医科大学附属第一医院

Corresponding Author E-mail: songwei8124@163.com

Background：Hypertension is a pivotal risk factor for the development and progression of chronic kidney disease (CKD). Renal tubular epithelial–mesenchymal transition (EMT) and interstitial fibroblast activation jointly drive renal interstitial fibrosis and progressive loss of renal function. Renal denervation (RDN) offers a novel

therapeutic option for resistant hypertension; observational studies have reported reduced proteinuria after RDN, yet the blood-pressure-independent renoprotective mechanisms remain poorly understood. Ferroptosis has been demonstrated to induce tubular epithelial cell injury and create a pro-fibrotic microenvironment in kidney disease. Our preliminary data revealed that OX1R and BACH1 are upregulated in fibrotic renal tissues and exhibit a hierarchical regulatory relationship, prompting the hypothesis that OX1R/BACH1 promotes renal fibrosis through ferroptosis activation, and that RDN may confer renal protection via suppression of this pathway.

Methods：(1) Animal experiments: Six-week-old male Dahl/SS rats were fed a high-salt diet (8% NaCl) for 12 weeks. At week 6, rats underwent RDN, received the OX1R antagonist SB-408124, or were subjected to control/sham interventions. Blood pressure and 24-hour urinary protein excretion were monitored. Renal interstitial collagen deposition was assessed by Masson's trichrome staining. Protein and mRNA levels of fibrotic markers (α-SMA, Fibronectin, TGF-β1, Collagen I/III), ferroptosis markers (GPX4, SLC7A11, SLC40A1, ACSL4, TFRC), and OX1R/BACH1 were measured by Western blot and RT-qPCR. (2) Cell experiments: Primary renal tubular epithelial cells were isolated from Dahl/SS rats and stimulated with TGF-β1 to establish an in vitro fibrotic model. OX1R or BACH1 was knocked down or overexpressed via plasmid transfection, and the same panel of markers was evaluated.

Results：(1) RDN significantly lowered blood pressure, reduced 24-hour urinary protein excretion, attenuated renal interstitial collagen deposition, and downregulated the expression of Fibronectin, Collagen I/III, and TGF-β1. (2) RDN upregulated GPX4, SLC7A11, and SLC40A1 while downregulating ACSL4 and TFRC in renal tissues, indicating suppression of ferroptosis; concomitantly, OX1R and BACH1 expression were markedly reduced at both protein and mRNA levels. (3) TGF-β1 stimulation upregulated OX1R and BACH1 in renal tubular epithelial cells; knockdown of OX1R decreased BACH1, whereas overexpression of OX1R increased BACH1, confirming that BACH1 functions downstream of OX1R. (4) Knockdown of OX1R or BACH1 effectively inhibited TGF-β1-induced upregulation of α-SMA and Fibronectin, accompanied by elevated GPX4/SLC7A11 and reduced ACSL4, indicating decreased ferroptosis; overexpression of OX1R or BACH1 reversed these effects, confirming that OX1R/BACH1 drives tubular EMT and fibrotic progression through ferroptosis activation.

Conclusion：RDN effectively alleviates salt-sensitive hypertensive renal interstitial fibrosis and proteinuria by downregulating the OX1R/BACH1 axis and suppressing ferroptosis in renal tubular epithelial cells. This study is the first to elucidate the pathogenic role of the OX1R/BACH1/ferroptosis pathway in hypertensive renal fibrosis, providing a novel molecular framework for the renoprotective effects of RDN.

论文 ID：229

Elevated visit-to-visit blood pressure variability is associated with increased risk of sudden cardiac death across two population-based cohorts

Junle Tong, Yaoquan Cao, Hong Yuan, Yao Lu, Liyong Zhu*

The Third Xiangya Hospital of Central South University

Corresponding Author E-mail: zly8128@csu.edu.cn

Background：Sudden cardiac death (SCD) constitutes a substantial global health burden, yet over one-third of cases occur without structural heart disease, eluding current risk stratification. Although autonomic dysfunction is

recognised as a key contributor to SCD, whether visit-to-visit blood pressure variability (BPV), a readily quantifiable marker of autonomic instability, contributes to SCD risk remains unknown.

Methods： Two population-based cohorts were used with distinct analytical roles. A large European cohort was used as the discovery cohort to assess the long-term association between visit-to-visit BPV and SCD. An independent Chinese cohort was used as the validation cohort to examine the reproducibility of these associations. BPV was derived from repeated blood pressure measurements across clinical visits. SCD was identified using hospital admission and death registry data. Multivariable Cox regression models were used to estimate SCD risk, with additional adjustment for heart rate.

Results： In the European discovery cohort, over 13.6 years of median follow-up, higher systolic and diastolic BPV were associated with increased SCD risk (HR 1.55, 95% CI 1.40–1.71; HR 1.52, 95% CI 1.37–1.68, respectively). In the Chinese validation cohort, over 6 years of median follow-up, associations were directionally consistent and stronger (HR 2.64, 95% CI 1.10–6.33; HR 2.23, 95% CI 1.02–4.87, respectively). Associations persisted after heart rate adjustment and were more pronounced in older adults and high-risk cardiometabolic subgroups.

Conclusion： Elevated long-term visit-to-visit BPV is independently associated with a higher risk of SCD, suggesting BPV as an accessible marker for SCD risk stratification beyond conventional blood pressure levels and heart rate.

Key words： Visit-to-visit blood pressure variability; Sudden cardiac death; Risk factors

论文 ID：240

Association of Red Cell Distribution Width/Albumin Ratio and Early Renal Impairment in Patients with Hypertension

Suining Xu[1], Haijian Xing[2], Gang Tian*[1]

1. The First Affiliated Hospital of Xi’an Jiaotong University

2. Xi 'an Children's Hospital

Corresponding Author E-mail: tiangang@xjtu.edu.cn

Background： Hypertension is a major risk factor for chronic kidney disease, accelerating renal injury by inflammatory indicators. The red cell distribution width- to-albumin ratio (RAR), a composite biomarker integrating inflammatory, is associated with CKD progression, but its relationship with early renal impairment in hypertensive patients remains unclear. This study aimed to investigate the association between RAR and urinary albumin-to-creatinine ratio (UACR) in patients with hypertension.

Methods： A total of 440 hypertensive patients were retrospectively enrolled from the Department of Cardiology of First Affiliated Hospital of Xi’an Jiaotong University between January 2017 to January 2024. Participants were categorized into early renal impairment group (UACR ≥30 mg/g, n = 176) and normal group (UACR <30 mg/g, n = 264). Binary logistic regression analysis was performed to determine the association between RAR and UACR. Receiver operating characteristic (ROC) curve analysis was used to evaluate the discriminative ability of RAR and other inflammatory indicators.

Results： Patients with early renal impairment exhibited significantly higher levels of RAR, monocyte-to-lymphocyte ratio (MLR), neutrophil-tolymphocyte ratio (NLR), and platelet-to-lymphocyte ratio (PLR) compared to the normal group (all $P < 0.001$). Multivariate logistic regression analysis revealed that RAR was significantly associated with early renal impairment (OR = 1.32, 95% CI: 1.15–1.53), and the highest RAR quartile (RAR > 3.03) was associated with a 4.79-fold increased risk of early renal impairment (OR = 4.79, 95%CI: 2.84–8.10) after adjusting for potential confounding factors. ROC analysis showed that RAR had the highest AUC at 0.708 for identifying early renal impairment, outperforming MLR, NLR, and PLR.

Conclusion： Elevated RAR is associated with early renal impairment in patients with hypertension, suggesting its potential use in early risk stratification.

Key words： hypertension, red blood cell distribution width, albumin, urinary albumin-to-creatinine ratio

论文 ID：241

Crotonylation of AOC3 promotes vascular remodeling by enhancing fatty acid synthesis in Ang II-induced hypertensive mice

Qiuwen Wu[1,2,3], Shuo Zhang*[1]

1. Department of Cardiology, the 2nd Affiliated Hospital of Harbin Medical University, Harbin 150001, China

2. National Key Laboratory of Frigid Zone Cardiovascular Diseases (NKLFZCD); The Key Laboratory of Myocardial Ischemia, Chinese Ministry of Education, Harbin 150001, China

3. the 2nd Affiliated Hospital of Harbin Medical University

Corresponding Author E-mail: zhangshuo@hrbmu.edu.cn

Background： Vascular smooth muscle cells (VSMCs) phenotypic transformation and its associated vascular remodeling serve as a pivotal pathological basis underlying the development of hypertensive target organ damage. Lysine crotonylation (Kcr) is a recently discovered posttranslational modification whose role in hypertensive vascular remodeling remains largely unknown.

Methods： The quantitative proteomics analysis was performed to identify the crotonylated proteins in the aortic tissues of hypertensive mice induced by Ang II infusion. We generated adeno-associated virus 9 vector carrying VSMC-specific AOC3 3KR (lysine to arginine mutation at sites K202, K527, K596) or 3KQ (lysine to glutamine mutation at sites K202, K527, K596) mutant mice to assess the functional role of AOC3 Kcr in pathological vascular remodeling. Moreover, we combined coimmunoprecipitation, mass spectrometry, and high throughput transcriptome sequencing analysis that was followed by multiple molecular biological methodologies to identify the crotonyltransferase P300/CBP-associated factor (PCAF), the target protein fatty acid synthase (FASN) and corresponding molecular events contributing to the function of AOC3 Kcr.

Results： Herein we report a quantitative proteomics analysis of global crotonylome in the aortic tissues of hypertensive mice induced by Ang II infusion, and demonstrate 985 Kcr site alterations across 773 proteins. Specifically, we observe a significant increase in K202cr, K527cr and K596cr of Amine oxidase copper containing 3 (AOC3) in Ang II-induced VSMCs in vitro and in vivo. PCAF is upregulated and interacts with AOC3, leading to the

upregulation of AOC3 Kcr under AngII treatment. The recombinant adenoviral vector carrying VSMC-specific AOC3 3KR mutant attenuated, while the 3KQ mutant aggravated Ang II-induced vascular remodeling. Abolishment of AOC3 Kcr promotes Ang II-mediated VSMCs phenotypic transformation by regulating de novo FAS. Mechanistically, we identified fatty acid synthase (FASN), a crucial enzyme in fatty acid synthesis, as a direct target of AOC3.

Conclusion： Our findings show that AOC3 Kcr is a signal in coordinating de novo FAS and phenotypic transformation in VSMCs, which may serve as a a therapeutic target for the treatment hypertensive vascular remodeling.

Key words： Hypertension; Vascular remodeling; AOC3; Crotonylation; FASN

论文 ID：246

Association of Intensive Blood Pressure Target with Risk of Atherosclerotic Cardiovascular Disease in Patients with Early-Onset Hypertension

Chi Wang*[1], Shouling Wu[2], Hao Xue[1]

1. Chinese PLA General Hospital

2. Kailuan General Hospital

Corresponding Author E-mail: wangchidr@163.com

Background： Early-onset hypertension carries a higher lifetime cardiovascular risk compared with late-onset phenotypes. This study aimed to investigate whether intensive blood pressure (BP) target (<120/80 mmHg) was associated with lower risk of atherosclerotic cardiovascular disease (ASCVD) events among patients with early-onset hypertension.

Methods： The current study included 8,902 patients with early-onset hypertension (age of onset <55 y) who had achieved BP control <140/90 mmHg, and 8,902 age- and sex- matched normotensive controls from the Kailuan cohort study. Patients with early-onset hypertension were stratified into three categories: BP <120/80 mmHg, isolated attainment of systolic BP <120 mmHg or diastolic BP <80 mmHg, and systolic BP ≥120 mmHg and diastolic BP ≥80 mmHg. ASCVD events comprised myocardial infarction and ischemic stroke. Multivariable Cox proportional hazard regression was used to estimate hazard ratios (HRs) and 95% confidence intervals (CIs) for the association between BP target groups and ASCVD risk.

Results： During a median follow-up period of 9.60 years, a total of 494 cases of ASCVD were documented: 33/1,328 in the <120/80 mmHg group, 65/2,137 in the isolated attainment of systolic BP <120 mmHg or diastolic BP <80 mmHg group, 211/5,437 in the systolic BP ≥120 mmHg and diastolic BP ≥80 mmHg group, 185/8,902 in the control group. Compared with normotensive controls, patients with systolic BP ≥120 mmHg and diastolic BP ≥80 mmHg had a 71% higher risk of ASCVD (adjusted HR and 95% CI: 1.71 [1.39–2.08]), those with either systolic BP <120 mmHg or diastolic BP <80 mmHg had a 53% higher risk (adjusted HR and 95% CI: 1.53 [1.15–2.03]), whereas patients with BP <120/80 mmHg showed a comparable risk to controls (adjusted HR and 95% CI: 1.17 [0.81–1.70]). Similar findings were observed for myocardial infarction and ischemic stroke, respectively.

Conclusion：An intensive BP target of <120/80 mmHg was associated with lower risk of ASCVD in patients with early-onset hypertension. These findings provide evidence on the clinical benefits of an intensive BP management in this high-risk population.

Key words：early-onset hypertension, intensive BP target, ASCVD

论文 ID：249

Perirenal fat thickness is associated with increased long-term blood pressure variability in high-normal blood pressure patients: aldosterone is a potential mediating factor

Pengjie Gao*[1], Suining Xu[1], Danni Wang[1], Haijian Xing[2], Gang Tian[1]

1. First Affiliated Hospital of Xi'an Jiaotong University

2. Affiliated Children's Hospital of Xi'an Jiaotong University

Corresponding Author E-mail: 1285420504@qq.com

Background： Patients with high-normal blood pressure (HNBP) are more susceptible to developing hypertension and cardiovascular diseases (CVD), particularly those with increased blood pressure variability. Long-term blood pressure variability (BPV) derived from home blood pressure telemonitoring (HBPT) is considered to better reflect real blood pressure fluctuations. Perirenal fat thickness (PRFT) is related to the incidence and prognosis of hypertension. However, the exact pathophysiological mechanism by which perirenal adipose tissue accumulation increases the CVD risk remains controversial, and whether it is associated with elevated long-term BPV, a well-established risk factor for CVD, remains unclear. We designed an observational cohort study aiming to investigate whether PRFT was associated with long-term BPV in HNBP patients, as well as potential mediating effects, in order to provide clues for studying the pathophysiological mechanisms underlying PRAT accumulation.

Methods：This study consecutively enrolled 202 HNBP patients (defined as office systolic blood pressure [SBP] between 120-139 mmHg or diastolic blood pressure [DBP] between 70-89 mmHg) from May 2023 to May 2024. Morning fasting blood samples were collected from patients at baseline for laboratory test. Patients were instructed to collect 24-hour urine and record urine volume. Plasma was collected from participants in the upright position for aldosterone concentration measurement. PRFT at the lower pole of the left kidney was measured using ultrasound test. The cohort was followed up for 24 months. During the follow-up period, patients were instructed to perform HBPT twice a month according to standard measurement procedures. BP data was collected via a smartphone application linked to the BP monitor by docters, and the average of two readings was taken as the monthly BP value. Long-term BPV was assessed using the standard deviation (SD), coefficient of variation (CV) and average real variability (ARV) of SBP and DBP, with both parameters measured monthly over the 24-month follow-up period (24 measurements in total). BPV ratio (BPVR) was calculated based on the ratio of the SD of SBP to that of DBP. Based on previous studies, elevated long-term BPV was defined as SBP-SD >12.8 mmHg or SBP-CV >9.8%. Linear regression, logistic regression, firth regression were employed to investigate the relationship between PRFT and long-term BPV. Additionally, we employed the third tertile and fourth quartile of SBP-SD and SBP-CV to define elevated long-term BPV in sensitivity analyses. Finally, based on exploring the correlations among PRFT, plasma and urine aldosterone, and long-term BPV

parameters, mediation analysis was used to explore the mediating effect of plasma and 24-hour urine aldosterone on the relationship between PRFT and long-term BPV parameters.

Results： After excluding patients with missing PRFT data and those with a follow-up duration of less than 24 months, 188 HNBP patients were finally included.The participants had a mean age of 49.78 ± 9.43 years and a mean PRFT of 30.27 ± 10.74 mm. Compared to participants in the first tertile of PRFT, those in the highest tertile had a higher proportion of males, greater weight and waist circumference, and higher levels of plasma fasting glucose, serum uric acid, and total cholesterol. Their SD, CV, and ARV of BP were also significantly higher. Linear regression analysis and generalized additive model revealed that PRFT was positively correlated with SBP/DBP-SD, SBP/DBP-CV and SBP/DBP-ARV after adjusting for confounding factors (e.g., SBP-SD: β=0.102, 95% confidence interval [95% CI]=0.052-0.152; SBP-CV: β=0.077, 95%CI=0.039-0.114). Logistic regression analysis showed that PRFT was associated with elevated long-term BPV defined by two criteria independently (SBP-SD: Odds Ratio [OR]=1.082, 95%CI=1.010-1.160; SBP-CV: OR=1.096, 95%CI=1.023-1.174). The results were similar in sensitivity analysis. Subgroup analysis suggested that gender, obesity status and hypertension incidence did not affect this association. Mediation analysis suggested that aldosterone levels partially mediated the association between PRFT and SBP-SD and SBP-CV. The mediated proportions of plasma and 24-hour urine aldosterone on the association between PRFT and SBP-SD were 14.83% and 26.41%, respectively.

Conclusion： Increased PRFT was independently associated with elevated long-term BPV during follow-up in HNBP patients, and aldosterone (especially 24-hour urine aldosterone) mediated the association. Assessing PRFT in newly diagnosed HNBP patients may help identify high-risk patients for closer follow-up and monitoring, as well as facilitate individualized treatment.

Key words： High-normal blood pressure, perirenal fat thickness, long-term blood pressure variability, aldosterone, standard deviation.

论文 ID：250

Associations of Insulin Resistance Surrogates with Incident Hypertension in Chinese Middle-aged and Older Adults with Dysglycaemia: A Prospective Study from CHARLS

Yunfeng Li, Deyu Fu*, Bo Lu

Yueyang Hospital of Integrated Traditional Chinese and Western Medicine, Shanghai University of Traditional Chinese Medicine

Corresponding Author E-mail: fdy65@126.com

Background： Insulin resistance (IR) is a pivotal contributor to hypertension development in individuals with dysglycaemia. However, the relative predictive value of various IR surrogate indices remains poorly defined. This study compared the associations of nine IR surrogates with incident hypertension among middle-aged and older Chinese adults with dysglycaemia.

Methods： This prospective cohort study included 2,674 Chinese adults aged ≥ 45 years with prediabetes or diabetes and without baseline hypertension, sourced from the China Health and Retirement Longitudinal Study (2011–2020). Nine IR surrogates derived from routine anthropometric and biochemical measurements were evaluated.

Multivariable logistic regression was employed to assess associations with incident hypertension, complemented by dose–response and subgroup analyses.

Results： Over a 9-year follow-up period, 1,197 participants (44.76%) developed hypertension. The Chinese visceral adiposity index (CVAI) exhibited the strongest association with incident hypertension (fully adjusted OR = 1.414, 95% CI: 1.301–1.537). The modified cardiometabolic index and triglyceride–glucose–based indices maintained independent associations, whereas the metabolic score for IR yielded non-significant results after full adjustment. Associations were predominantly linear. Subgroup analyses demonstrated a more pronounced CVAI–hypertension association in women (OR = 1.734, 95% CI: 1.512–1.988) than in men (OR = 1.227, 95% CI: 1.103–1.365); notably, multiple indices showed stronger independent associations among individuals without central obesity.

Conclusion： In middle-aged and older Chinese adults with dysglycaemia, IR surrogates are robustly associated with incident hypertension. CVAI serves as a practical adjunctive marker for early risk stratification, particularly in women. Furthermore, these indices hold potential for identifying "metabolically unhealthy non-obese" individuals who might otherwise be overlooked by obesity-centered screening.

Key words： Insulin resistance surrogate indices; Dysglycaemia; Hypertension; Prospective cohort; CHARLS

论文 ID：254

Proteomic profiling reveals novel insights into hypercortisolism adrenal adenomas

Xuyang Shi[1,2,3], Yi Yang[1], Qifu Li[1], Linqiang Ma*[1]

1. 重庆医科大学附属第一医院

2. the First Afffliated Hospital of Chongqing Medical University

3. The First Affiliated Hospital of Chongqing Medical University

Corresponding Author E-mail: tom_linqiang@163.com

Background： Adrenal cortisol-producing adenomas (CPAs) are the most common functional adrenal incidentalomas, yet the molecular drivers of CPAs remain unclear.

Methods： DIA proteomics of 9 Cushing's syndrome (CS), 10 subclinical CS (SCS), and 10 non-functioning adenomas (NFAs) was integrated with lipidomic and cell validation.

Results： CPAs showed stepwise cholesterol metabolic reprogramming from NFA to SCS and CS. LDLR, HMGCS1, LIPE, and SYP emerged as candidate tissue markers. Calcium binding protein 39 like (CAB39L) loss enhanced cortisol secretion by suppressing AMPK and activating SREBP2, whereas restoration reduced cholesterol accumulation and steroidogenesis.

Conclusion： Cholesterol metabolic reprogramming is a hallmark of CPAs; CAB39L may enable diagnosis and targeted therapy.

Key words： Adrenal cortisol-producing adenomas Cholesterol metabolic reprogramming Proteomics Calcium binding protein 39 like (CAB39L)

论文 ID：263

Predictive Factor Analysis and Nomogram Development for CT-Based Subtyping in Primary Aldosteronism: Optimizing Surgical Decision-Making

Xiaoxue Guo[1,2], Wei Song*[1]

1. 大连医科大学附属第一医院

2. Dalian Medical University First Affiliated Hospital

Corresponding Author E-mail: songwei8124@163.com

Background： Accurate subtype classification is crucial for primary aldosteronism (PA) management. Although adrenal CT is widely used for subtype assessment, its accuracy remains limited. This study aimed to identify predictors of CT-based subtyping accuracy and develop a predictive model for patients with CT-detected unilateral adrenal lesions to optimize AVS utilization and surgical decision-making.

Methods： A total of 551 patients with PA undergoing AVS for subtype classification were retrospectively included. CT-based subtype classification accuracy was assessed by concordance with AVS results. Logistic regression analyses identified factors associated with CT accuracy, and a nomogram was developed in patients with CT-detected unilateral adrenal lesions.

Results： Using AVS-based subtype classification as the reference standard, the overall accuracy of CT-based subtype classification was 47.5% (262/551). Subgroup analyses demonstrated higher CT classification accuracy in patients aged <35 years (65.9%), those with CT-detected unilateral adrenal lesions (56.0%), hypokalemia (51.7%), and ARR ≥64.3 (53.5%). Among patients with unilateral CT lesions (n=302), age, serum potassium, ARR, and CT-detected adenoma were independent predictors of CT classification accuracy. A nomogram incorporating these predictors showed good discrimination (AUC=0.857; 95% CI, 0.815–0.899), calibration, and clinical utility.

Conclusion： CT-based subtyping accuracy is influenced by age, serum potassium, ARR, and adrenal CT findings. A nomogram incorporating these factors may identify patients with unilateral aldosterone excess and support surgical decision-making in PA.

Key words： primary aldosteronism, adrenal venous sampling, computed tomography, aldosterone, hypokalemia

论文 ID：265

High Salt Intake and Its Dual Impact on Obesity and Cardiovascular Mortality in Individuals Over 50: A Paradoxical Relationship

Qiang Li*[1], Xiaona Bu[1], Yushuang Luo[2], Zhiming Zhu[1]

1. Army Medical Center of PLA

2. Southwest University

Corresponding Author E-mail: liqianglife@126.com

Background： There is still debatable of the optimal standards for obesity and salt intake, particularly their potential impacts on mortality among older population. This study aimed to characterized the complex relationships among high-salt diet, obesity, and the cardiovascular disease (CVD) and all-cause mortality in US adults, with a focus on individuals aged 50 years or older.

Methods： This is a retrospective cohort analysis of data from the National Health and Nutrition Examination Survey (2005-2020, n=11,977). Data on individual body weight status and dietary sodium intake were extracted. Daily salt intake above 9g is considered a high salt consumption. Adjusted weighted multivariable logistic regression models were was used for the analysis of the association among salt intake, obesity, and CVD outcomes. Cox proportional hazards model to estimate the association between salt intake and mortality. Mediation analysis was performed to investigate whether insulin levels mediate the relationship between salt intake and obesity.

Results： Individuals with high-salt intake were more likely to exhibit both overall (OR: 1.33; 95% CI: 1.16-1.53) and central obesity (OR: 1.29; 95% CI: 1.13-1.48) after adjusting for potential confounders. Among individuals aged 50 years or older, the risks of overall obesity and central obesity were higher in those following a high-salt diet. No such relationships were observed in individuals younger than 50 years of age. High salt intake was associated with lower risks of both all-cause and CVD mortality compared to low salt intake in individuals over 50 years. In the older population with either overall (HR: 0.39, 95% CI: 0.17-0.89, P = 0.0254) or central obesity (HR: 0.40, 95% CI: 0.22-0.72, P = 0.0025), high salt intake was linked to a lower risk of CVD mortality compared to low salt intake. In individuals aged younger than 50 years and without obesity, high salt intake was associated with increased risk of CVD mortality.

Conclusion： This study found that higher salt intake was associated with an increased risk of obesity, whereas had an inverse association with all-cause and CVD mortality in adults aged over 50 years. Caution is warranted in interpreting these findings due to potential selection bias. Further longitudinal studies incorporating objective evaluation of salt intake are necessary to establish evidence-based recommendations for healthy lifestyle practices aimed at promoting healthy aging.

Key words： High salt intake, Obesity, NHANES, All-cause mortality, Cardiovascular mortality

论文 ID：270

High salt intake exacerbates aldosterone-related target organ damage in patients with primary aldosteronism

Xiaona Bu, Fang Sun, Qiang Li, Hongbo He, Zhiming Zhu*

Army Medical Center of PLA

Corresponding Author E-mail: hbpcms@sina.com

Background： Primary aldosteronism (PA) is susceptible to cardiovascular and renal damage. However, the role of dietary salt intake in aldosterone-related target organ damage (TOD) in PA remains elusive. This study aimed to investigate whether high salt intake enhances the effect of aldosterone on TOD in PA patients.

Methods： A total of 361 patients with PA and 102 patients with essential hypertension (EH) were enrolled in this study. All participants underwent tests for the aldosterone/renin ratio, 24-hour urinary sodium, biochemical examination, and TOD assessment via echocardiography, carotid ultrasound, and urine microalbumin measurements. Patients were categorized on the basis of salt intake and aldosterone levels.

Results： Compared with th control group, PA patients with high salt intake presented an increased urinary albumin-to-creatinine ratio (UACR), a more significant effect of aldosterone on blood pressure, and remarkable cardiac hypertrophy and renal damage ($p < 0.05$). Quintile-based regression analysis revealed that the odds ratio between the highest quintile of the plasma aldosterone level and the UACR was twice that of the lowest quintile. In the adjustment models, the relationships between aldosterone and the left ventricular mass index (LVMI) were not statistically significant. Linear correlation analysis further revealed that high salt intake could reinforce the correlation between aldosterone and creatinine, log-UACR and LVMI in PA patients, but this effect was not observed in EH patients.

Conclusion： This study suggests that high salt intake can exacerbate aldosterone-related renal impairment, while it is crucial in aldosterone-related cardiac damage in PA patients

Key words： Primary aldosteronism, salt intake, urinary albumin excretion, left ventricular hypertrophy

论文 ID：280

The correlation between early renal injury and vascular stiffness and ARR ratio in patients with newly diagnosed primary aldosteronism

Shuojie Chen, Xiaofang Zhang, Shuli Wang, Mingze Li, Fu Meng, Yubin Mi, Lianjie Song, Dan Zhang, Xinyue Zhang, Qiongying Wang*

Lanzhou University Second Hospital

Corresponding Author E-mail: wqyconnie@163.com

Background： The purpose of this study was to compare the differences in general clinical characteristics between the two subtypes of idiopathic hyperaldosteronism (IHA) and aldosterone-producing adenoma (APA) in newly diagnosed primary aldosteronism (PA) and essential hypertension (EH). To explore the possible clinical causes of early renal injury and vascular stiffness in PA patients.

Methods： A total of 151 patients (108 patients with IHA and 43 patients with APA) who were hospitalized and diagnosed with PA for the first time between January 2024 and December 2024 were included, and 46 patients with simple essential hypertension who were hospitalized during the same period were matched as controls. The general characteristics, biochemical indicators, ambulatory blood pressure, echocardiography, and vascular stiffness parameters of all patients were collected. The correlation between microalbuminuria (m-ALB) and brachial-ankle pulse wave velocity (baPWV) was analyzed by partial correlation analysis after confounding factors were controlled. The difference of baPWV between groups was compared after m-ALB was stratified.

Results： The ratio of aldosterone to renin activity (ARR) in APA group, IHA group and EH group was 119.60(73.03,357.50)，67.44(41.96,171.15)，11.08(5.68,26.96), respectively. Compared with the EH group, the ARR values of the IHA group ($P < 0.001$) and the APA ($P < 0.001$) group were significantly increased. Compared with the

IHA group, the ARR value of the APA group was higher (P = 0.012). Compared with EH group, serum potassium was significantly lower in APA group (P < 0.001) and IHA group (P < 0.001). Compared with patients in the IHA group, patients in the APA group also showed lower serum potassium levels (P = 0.003). Compared with patients in IHA group. The 24-hour mean diastolic blood pressure (89.07 ± 8.80 vs 84.74 ± 9.95 mmHg, P = 0.014), daytime mean diastolic blood pressure (90.42 ± 8.73 vs 86.11 ± 10.02 mmHg, P = 0.015), morning diastolic blood pressure peak (92.35 ± 8.97 vs 87.90 ± 12.35, P = 0.041), 24-hour diastolic blood pressure load [(73.68 ± 23.41) % vs (60.25 ± 26.98) %, P = 0.004], daytime diastolic blood pressure load [(69.11 ± 25.64) % vs (54.40 ± 29.89) %, P = 0.004], and nighttime diastolic blood pressure load [(86.47 ± 22.27) % vs (76.81 ± 27.54) %, P = 0.038). The mean baPWV in APA group was significantly higher than that in EH group (P < 0.001) and IHA group (P = 0.024).Compared with the IHA group, the APA group had higher left ventricular mass index (LVMI) (110.85±25.70 vs 94.51±22.84 g/m2, P < 0.001) and higher m-ALB level [28.80(19.10，59.85) vs 14.95(7.00，30.40) mg/L, P <0.001]. Partial correlation analysis showed that m-ALB was positively correlated with baPWV in EH group (r = 0.485, P = 0.014). After stratification of m-ALB, the baPWV value of APA group was higher than that of IHA group and EH group in the early stage of renal injury.

Conclusion： In newly diagnosed PA patients, APA subtype had more severe target organ damage than IHA subtype, lower serum potassium level, higher 24-hour mean diastolic blood pressure, daytime mean diastolic blood pressure, 24-hour diastolic blood pressure load, daytime diastolic blood pressure load and nighttime diastolic blood pressure load, and higher LVMI and baPWV values. Elevated ARR is closely related to early renal injury and increased vascular stiffness, suggesting that early identification of PA subtypes and evaluation of target organ damage are of great significance for improving the prognosis of patients.

Key words： primary aldosteronism, idiopathic hyperaldosteronism, aldosterone-producing adenoma, brachial ankle pulse wave velocity, microalbuminuria

论文 ID：285

Postprandial blood pressure trajectories and their predictors in older adults with hypertension in long-term care facilities

Lei Huang, Fengying Zhang*, Xinjun Zhang, Linfang Xu

四川大学华西医院

Corresponding Author E-mail: zhangfengying@scu.edu.cn

Background： In modern long-term care facilities (LTCFs), older adults with hypertension spend a substantial portion of their day in a postprandial state because they consume several meals every 24 hours. Individuals with low blood pressure (BP) are significantly more prone to falls, cardiovascular events, and even death, which poses a considerable threat to their well-being. Preventing postprandial BP excursions in older adults with hypertension in LTCFs is clinically important. However, targeted strategies remain scarce due to inadequate understanding of postprandial BP trajectories and their predictors in this population.

This study aimed to examine (1) postprandial systolic BP (SBP) and diastolic BP (DBP) trajectories, and (2) the predictors of these trajectories in older adults with hypertension in LTCFs.

Methods: The study enrolled older adults with hypertension from 25 LTCFs in different Chengdu districts between June 2022 and August 2024. These facilities provide care for residents with a broad spectrum of functional dependency, ranging from complete independence to severe disability. Eligible participants were those aged ≥ 65 years who were diagnosed with hypertension (defined as SBP ≥ 140 mmHg, DBP ≥ 90 mmHg, or current use of antihypertensive medication), exhibited a stable clinical condition, were able to maintain a sitting position, had a comprehensive and clear medical history along with daily medication records, and lived in an LTCF for at least 3 days. Participants were excluded if they failed to complete the BP measurements.

The seated BP and pulse rate (PR) of the participants were assessed at baseline (immediately before breakfast) and at 0, 30, 60, 90, and 120 min after breakfast. Preprandial BP and PR were measured at least twice and averaged. Postprandial BP and PR refer to multiple measurements taken within 2 h after meal consumption. Additionally, participants fasted overnight before the BP and PR measurements and refrained from consuming caffeine, smoking, or alcohol until the completion of the measurements. Routine medications were maintained to preserve real-world treatment conditions and meet ethical requirements. Furthermore, measurements were performed using validated automatic BP monitors (OMRON HEM-7121) with appropriately sized cuffs under standardized conditions, with participants resting quietly for approximately 5 min before each measurement and refraining from vigorous activity during the measurement duration.

Data regarding patient demographics, medical history, and routine medication use were recorded using standardized case report forms. Polypharmacy was defined as the use of five or more prescription or non-prescription medications. Furthermore, comprehensive assessments were conducted by researchers using the following validated instruments: the Chinese version of the FRAIL-NH scale (C-FRAIL-NH) for frailty status determination; the education-adjusted Chinese Mini-Mental State Examination (C-MMSE) for cognitive function assessment; and the Chinese version of the modified Barthel index (C-MBI) for evaluating basic activities of daily living. Detailed information on these instruments has been reported previously. Additionally, the weight (g) of the breakfast consumed on the day of BP measurements was carefully documented.

The primary statistical analytical methods included group-based trajectory modeling (GBTM) and logistic regression models. All statistical analyses were conducted using IBM SPSS Statistics (version 26.0) and R (version 4.4.1). Statistical significance was defined as a two-sided *P*-value of < 0.05.

Results: The statistical analysis included data from 608 participants who had a median age of 86.0 (IQR 8.0) years, and 66.9% were women. The most frequently prescribed antihypertensive medications were CCBs (65.6%), ARBs (43.1%), β-adrenoceptor antagonists (28.8%), and diuretics (15.5%). Additionally, 16.3% of participants were taking α-glucosidase inhibitors, and 13.2% were taking metformin. The mean preprandial BP was 140.1 (SD 0.7) /78.5 (SD 0.4) mmHg. Following ingestion of a meal with a median weight of 472.0 (IQR 208.0) g, the lowest recorded postprandial BP was 116.0 (IQR 20.0)/66.0 (IQR 12.0) mmHg, representing a maximum decrease of 21.0 (IQR 18.0)/12.0 (IQR 9.4) mmHg.

GBTM classified postprandial systolic and diastolic BPs into low, medium, and high groups, accounting for 24.3%, 53.6%, and 22.0% of systolic trajectories and 37.5%, 46.7%, and 15.8% of diastolic trajectories, respectively. Multivariable logistic regression identified several novel findings: low preprandial BP (odds ratio [OR]: 0.794–0.885) was associated with low postprandial BP trajectories, whereas high preprandial BP (OR: 1.126–1.200) and a history of cerebrovascular disease (OR: 2.173–2.439) were associated with high postprandial BP trajectories. Moreover, β-adrenoceptor antagonists (OR: 1.890, 95% confidence interval [CI]: 1.024–3.486) and statins (OR: 0.506, 95% CI: 0.275–0.931) respectively reduced low and high postprandial systolic BP trajectory risks. A high pulse rate trajectory

(OR: 0.345; 95% CI: 0.176–0.675) and diabetes mellitus (OR: 0.450; 95% CI: 0.223–0.907) lowered the odds of a low postprandial diastolic BP trajectory. Results were unchanged in sensitivity analyses.

Conclusion：Postprandial BP fluctuations follow distinct low, medium, and high trajectories in older adults with hypertension in LTCFs, which are influenced by multiple clinical factors. Further studies are required to validate and expand these findings.

Key words：Long-term care facilities, Older adults with hypertension, Post-breakfast blood pressure trajectories, Predictors

论文 ID：288

COL1A1-Enhanced CD44/SLC7A11 Interaction and Cystine Uptake Result in CD34+ Foam-Like Macrophage Accumulation in Transplant Arteriosclerosis

Junru Wu*[1,3], Hong Yuan[1], Qingbo Xu[2], Jingjing Cai[1,3]

1. Xiangya Third Hospital of Central South University

2. The First Affiliated Hospital of Zhejiang University

3. First Affiliated Hospital of Gannan Medical University

Corresponding Author E-mail: junru@csu.edu.cn

Background：Chronic transplant arteriosclerosis is the primary cause of long-term graft failure. Selectively targeting specific inflammatory macrophage subpopulations is essential for inhibiting the primary triggers of inflammatory and immune responses. Therefore, elucidating the origins and regulatory mechanisms of these macrophages in allograft arteriosclerosis is critical for the development of targeted therapies.

Methods：We performed scRNA-seq and spatial transcriptomics or integrated transcriptomic data from human chronic allograft vasculopathy specimens and mouse vascular allograft models. Flow cytometry and immunofluorescence staining were employed to characterize macrophage subpopulations within remodeled allograft arteries. To determine cellular origins, CD34+ lineage-tracing and depletion strategies were used. The interactions between COL1A1, CD44, and SLC7A11 were analyzed using proximity ligation assays and co-immunoprecipitation. Furthermore, metabolic profiles were investigated via ultra-performance liquid chromatography coupled with high-resolution mass spectrometry. To validate the role of cystine transport in macrophage differentiation, we employed pharmacological inhibitors and a genetic approach using myeloid-specific SLC7A11 knockout mice (Lysm-Slc7a11-KO). The mechanisms identified in vivo were further corroborated through in vitro experiments.

Results：We identified a novel proinflammatory foam-like macrophage phenotype in allograft arterial adventitia. These macrophages primarily originated from bone marrow-derived $CD34^+$ lineage cells and exhibit heightened de novo lipogenesis and proinflammatory activity. Their lipogenesis is driven by increased cystine uptake, facilitated by enhanced membrane expression of the CD44-SLC7A11 complex, which activates mTORC1-HIF-1α signaling. We also revealed that fibroblast-secreted COL1A1 is essential for anchoring the complex to the cell membrane via its direct interaction with CD44. Either blocking COL1A1, CD44, or SLC7A11 effectively attenuated mTORC1-HIF-1α

signaling, inflammation, and lipogenesis in macrophages as well as accumulation of foam-like cells and intimal hyperplasia in allograft arteries.

Conclusion： This study has revealed previously uncharacterized foam-like macrophages in transplant arteriosclerosis, with COL1A1-enhanced amino acid metabolism modulating lipogenesis and foamy macrophage formation. This study offers potential therapeutic targets to modulate immune response and enhance transplant outcomes.

Key words： Transplant arteriosclerosis; Foam-like macrophage; Cystine; SLC7A11; COL1A1

论文 ID：289

Association between relative fat mass and cardiometabolic multimorbidity in middle-aged and older adults and those with sarcopenia in two prospective cohorts

Yingxuan Gong*[1,2], Xiexiong Zhao[1], Tianrui Shi[1], Xuewei Huang[1], Zhenxin Li[1], Qiaoyu Zhou[1], Wenjuan Wang[1], Jingjing Cai[1,3], Liping Peng[1]

1. 中南大学湘雅三医院

2. Clinical Research Center, Department of Cardiology, The Third Xiangya Hospital, Central South University, Changsha, China.

3. 赣南医科大学第一附属医院

Corresponding Author E-mail: 1029414190@qq.com

Background： Obesity is a well-established risk factor for cardiometabolic diseases. While the Relative Fat Mass (RFM) index has been proposed as an alternative adiposity measure, its value for predicting cardiometabolic multimorbidity (CMM) in Chinese and American populations remains insufficiently evidenced. Sarcopenia poses a significant health burden to the aging Chinese population, but the predictive utility of RFM for CMM risk specifically in middle-aged and older adults with sarcopenia is currently unexplored. This study aimed to evaluate whether RFM is a superior predictor of CMM compared to Body Mass Index (BMI) and Waist-to-Height Ratio (WHtR), and to assess its specific predictive advantage over BMI within the Chinese sarcopenic population.

Methods： This study utilized data from two prospective cohorts: China Health and Retirement Longitudinal Study (CHARLS) and Health and Retirement Study (HRS). RFM was calculated using height, waist circumference, and sex. CMM was defined as the co-occurrence of two or more conditions among diabetes, heart disease, and stroke. Associations between RFM and CMM were assessed using logistic regression for prevalence and Cox proportional hazards models for incidence. Model predictive performance was compared using Harrell's C-index, NRI, and IDI.

Results： A total of 13,102 participants from CHARLS (male: 47.5%, median age: 59 years [IQR: 52.0-66.0]) and 8,470 from HRS (male: 42.1%, median age: 64 years [IQR: 56.0-74.0]) were included. The median follow-up periods was 9 years in the CHARLS and 14 years in the HRS. RFM (CHARLS, OR 1.68, 95% CI 1.19-2.40; HRS, OR 1.85, 95% CI 1.28-2.68), BMI (CHARLS, OR 1.17, 95% CI 1.01-1.34; HRS, OR 1.23, 95% CI 1.01-1.47), and WHtR (CHARLS, OR 1.25, 95% CI 1.06-1.48; HRS, OR 1.29, 95% CI 1.07-1.52) were all associated with CMM prevalence. A linear association was observed between RFM and CMM risk (CHARLS: p for nonlinearity = 0.770; HRS: p for nonlinearity

= 0.744), whereas associations for BMI (BMI: CHARLS, p for nonlinearity < 0.001; HRS, p for nonlinearity = 0.003) and WHtR (CHARLS, p for nonlinearity = 0.014; HRS, p for nonlinearity = 0.001) were nonlinear. Regarding incidence, higher RFM (CHARLS, HR 2.22, 95% CI 1.84-2.69; HRS, HR 1.83, 95% CI 1.46-2.28), BMI (CHARLS, HR 1.25, 95% CI 1.17-1.32; HRS, HR 1.35, 95% CI 1.20-1.52), and WHtR (CHARLS, HR 1.43, 95% CI 1.31-1.56; HRS, HR 1.35, 95% CI 1.21-1.51) were associated with increased CMM risk. Adding RFM, BMI, or WHtR to a basic model improved its predictive performance (Harrell's C-index and NRI). Notably, the IDI improvement was greatest for models incorporating RFM (CHARLS: IDI = 0.011, 95% CI 0.004-0.020; HRS: IDI = 0.013, 95% CI 0.002-0.028) compared to those adding BMI (CHARLS: IDI = 0.005，95% CI 0.001-0.012; HRS: IDI = 0.006，95% CI 0.001-0.011) or WHtR (CHARLS: IDI = 0.010，95% CI 0.003-0.021; HRS: IDI = 0.009，95% CI 0.001-0.016). Crucially, within the Chinese middle-aged and older adults with sarcopenia, RFM (HR 1.84, 95% CI 1.23-2.74) was significantly associated with CMM incidence, while BMI (HR 1.15, 95% CI 0.98-1.35) was not.

Conclusion：RFM is a better predictor of CMM risk than BMI and WHtR in middle-aged and older Chinese and American adults. It demonstrates a particular advantage over BMI for CMM risk assessment in the Chinese population with sarcopenia.

Key words：relative fat mass; cardiometabolic multimorbidity; middle-aged and older adults; sarcopenia

论文 ID：308

Association between stress hyperglycemia ratio and hypertension in children and adolescents based on machine learning algorithms

Chen Zhang[3], chen zhang*[1,2]

1. Henan Provincial People's Hospital

2. Henan Provincial People's Hospital

3. Henan Provincial People's Hospital

Corresponding Author E-mail: zchen18037763215@163.com

Background：From childhood and adolescence to adulthood, the presence of hypertension has consistently been a risk factor for the development of cardiovascular diseases. The impact of elevated blood pressure in childhood on early atherosclerosis can be largely reversed if blood pressure normalizes in adulthood. Children and adolescents with hypertension are, to a certain extent, in a state of chronic stress, characterized by nonspecific physiological and metabolic changes. Previous studies have shown that the stress hyperglycemia ratio (SHR) can serve as a reliable predictor of adverse outcomes in various cardiovascular and cerebrovascular diseases. However, there is a lack of research on the association between SHR and hypertension in children and adolescents. Therefore, this study aims to investigate the relationship between SHR and hypertension in children and adolescents.

Methods：This study collected clinical data from 28,844 normotensive and hypertensive children and adolescents in Henan Province. Hypertension was identified according to the diagnostic criteria of Chinese and European hypertension guidelines, respectively. Based on the quartiles of SHR, logistic regression analysis and mediation effect algorithms were used to explore the relationship between SHR and outcomes. The least absolute shrinkage and selection

operator (LASSO) regression was applied to screen relevant factors, and 11 machine learning algorithms were used to construct predictive models.

Results： This study ultimately analyzed the clinical data of 27,912 children and adolescents. Using the first quartile of SHR as a reference, higher SHR quartiles were significantly associated with the occurrence of hypertension (OR > 1, P < 0.05). Mediation analysis showed that SHR positively mediated the occurrence of hypertension through the A-body shape index and conicity index, with mediation proportions of 4.6%–13.6%, respectively. The area under the receiver operating characteristic curve (AUC) of predictive models established by 11 machine learning algorithms was as follows: Lightgbm (0.7686), Xgboost (0.7671), Logistic (0.7602), Ridge (0.7569), ENet (0.7537), MLP (0.7433), RF (0.7254), KNN (0.7011), DT (0.6891), and SVM (0.6125). The results in subgroups of heart rate >73 beats/min and gender were consistent with the main analysis.

Conclusion： Higher SHR was significantly associated with the occurrence of hypertension in children and adolescents, particularly among participants with a heart rate >73 beats/min.

Key words： Hypertension, Children and Adolescents, Stress hyperglycemia ratio, Machine Learning Algorithms, Risk Prediction Models

论文 ID：311

Sex Differences Between Ambulatory Blood Pressure Load and Cardiac Remodeling and Diastolic Function in Newly Diagnosed Primary Aldosteronism

Mingze Li, Xiaofang Zhang, Miaomiao Qi, Heng Yu, Runmin Sun, Wei Liang, Shuojie Chen, Shuli Wang, Qiongying Wang*

Lanzhou University Second Hospital

Corresponding Author E-mail: wqyconnie@163.com

Background： Primary aldosteronism (PA) is closely associated with hypertension-mediated target-organ damage. However, whether ambulatory blood pressure load, cardiac remodeling, and diastolic function differ by sex among patients with newly diagnosed PA, and whether differences in cardiac phenotypes can be explained by ambulatory blood pressure load, remain unclear.

Methods： This single-center, retrospective, cross-sectional study included 411 patients with newly diagnosed PA, comprising 206 men and 205 women. Ambulatory blood pressure monitoring (ABPM) was used to assess mean systolic blood pressure (SBP) and diastolic blood pressure (DBP) over 24 hours and during daytime and nighttime periods, as well as the corresponding SBP and DBP loads. Left ventricular hypertrophy (LVH) was defined using sex-specific left ventricular mass index (LVMI) thresholds and classified as mild, moderate, or severe. Diastolic function was assessed using the average E/e′ ratio. Multivariable logistic regression models were sequentially adjusted for age, body mass index, hypertension duration, serum potassium, and log-transformed plasma aldosterone concentration (PAC) or aldosterone-to-renin ratio (ARR). Among patients with complete ABPM data, 24-hour DBP load was additionally included to determine whether the associations of female sex with LVH and elevated average E/e′ could be explained by differences in ambulatory blood pressure load.

Results：Compared with women, men had higher 24-hour mean SBP (144.22±15.89 vs 138.93±15.67 mmHg; P=0.005), 24-hour mean DBP (88.63±10.16 vs 83.92±9.08 mmHg; P<0.001), daytime mean SBP (146.13±15.50 vs 140.61±16.01 mmHg; P=0.003), daytime mean DBP (89.86±10.00 vs 85.12±9.18 mmHg; P<0.001), and nighttime mean DBP (84.99±12.22 vs 80.60±10.71 mmHg; P=0.001). The 24-hour and daytime SBP and DBP loads were also higher in men (all P≤0.005), whereas nighttime SBP and DBP loads did not differ significantly between the sexes. Despite lower ambulatory blood pressure levels, women had higher proportions of moderate and severe LVH than men when classified using sex-specific LVMI thresholds (5.7% vs 1.5% and 6.7% vs 2.1%, respectively), and the overall distribution of LVMI severity differed significantly by sex (P=0.017). Women also had a higher average E/e′ ratio than men (12.84±3.66 vs 11.56±3.01; P<0.001). Among 281 patients with complete ABPM data, female sex remained independently associated with elevated average E/e′ after additional adjustment for 24-hour DBP load (Model 3-PAC: odds ratio [OR], 2.86; 95% confidence interval [CI], 1.56–5.24; Model 3-ARR: OR, 2.85; 95% CI, 1.56–5.21; both P<0.001).

Conclusion：Among patients with newly diagnosed PA, men exhibited greater ambulatory blood pressure exposure, whereas women, despite having lower ambulatory blood pressure levels, showed a greater burden of moderate-to-severe LVH and more impaired diastolic function. These findings suggest that sex differences in cardiac structure and function in PA cannot be fully explained by ambulatory blood pressure load and may be related to PA-associated hormonal abnormalities, including suppressed renin activity and elevated aldosterone levels.

Key words：primary aldosteronism; sex differences; ambulatory blood pressure monitoring; blood pressure load; left ventricular hypertrophy; left ventricular filling pressure

论文 ID：317

Epicardial Adipose Tissue Volume and Density in Heart Failure Complicated by Pulmonary Hypertension: Prognostic Value and Incremental Model Reclassification

Mengxuan Wang, Yan Liu*

The First Affiliated Hospital of Dalian Medical University

Corresponding Author E-mail: liuyanjulie@outlook.com

Background：Epicardial adipose tissue volume (EATV) and density (EATD) are key imaging biomarkers for cardiovascular risk. However, their independent prognostic utility and joint stratification capacity in heart failure (HF) with pulmonary hypertension (PH) remain unclear. This study aimed to evaluate the multivariable associations of EATV and EATD with a long-term cardiovascular endpoint and to quantify their incremental predictive value beyond traditional risk factors.

Methods：We retrospectively analyzed 265 HF patients with concomitant PH (defined as pulmonary artery systolic pressure (PASP) ≥ 35mmHg on echocardiography) who underwent cardiac enhanced CT at the Cardiac Department of the First Affiliated Hospital of Dalian Medical University between July 2015 and December 2025. EATV and EATD were quantified using Siemens Healthcare GmbH Client (threshold: -190 to -30 HU). The primary endpoint was a composite of all-cause mortality and HF hospitalization. Kaplan-Meier curves and log-rank tests compared event-free survival across groups; Cox proportional hazards models were used to assess the associations of

continuous and discretized EAT metrics with the outcome, with hierarchical adjustments (Model 1: age, sex, BMI, CAD; Model 2: Model 1 + eGFR, total cholesterol, log-BNP; Model 3: Model 2 + spironolactone, beta-blockers, SGLT2i; Model 4: Model 3 + LVEF, PASP). To assess the incremental prognostic value of EATV beyond conventional clinical risk factors, two nested multivariable Cox proportional hazards prediction models were established: a baseline model comprising 12 established clinical covariates (Model 4) and an advanced model integrating EATV dichotomized at its optimal cut-point (determined via maximally selected rank statistics) into the clinical baseline model. The predictive efficacy was assessed using the likelihood ratio test (χ^2), C-index, time-dependent AUC, Brier score, continuous Net Reclassification Improvement (NRI, including Event NRI+ and Non-event NRI- components), Integrated Discrimination Improvement (IDI), and decision curve analysis (DCA). All patients were divided into four joint subtypes based on the median values of EATV and EATD to establish a joint stratification model, and a linear trend test across the four subgroups was conducted using Cox proportional hazards regression with assigned equidistant ordinal scores.

Results： Over a median follow-up of 64 months (IQR 35–93), 136 primary endpoint events (51.3%) occurred. Patients who experienced events had significantly larger EATV [median (IQR): 73.30 (57.64, 87.86) vs. 64.98 (51.91, 83.49) cm^3, $P < 0.05$] and higher EATD (−72.50 ± 6.42 vs. −74.42 ± 5.82 HU, $P < 0.05$). In the fully adjusted Model 4, continuous EATV independently predicted the primary endpoint (HR 1.009, 95% CI [1.001–1.018]; $P = 0.023$), whereas continuous EATD lost significance after multivariable adjustment ($P = 0.858$). The MaxStat-dichotomized EATV (> 57.31 cm^3) demonstrated strong prognostic separation, with an adjusted HR of 2.126 (95% CI [1.386–3.262]; $P < 0.001$) in Model 4. Incorporating this dichotomized variable into the 12-factor baseline model significantly improved global fit ($\Delta\chi^2 = 12.901$, $P < 0.001$), enhanced model calibration, and reduced the 5-year Brier score from 0.2014 to 0.1858. Time-dependent discrimination analysis revealed a significant increase in the 5-year AUC from 0.762 to 0.806 (ΔAUC = 0.044, $P = 0.025$). Crucially, EATV significantly improved long-term risk reclassification, with continuous NRI increasing at 3 years (0.346, $P = 0.011$) and 5 years (0.373, $P = 0.006$). This improvement was primarily driven by event-side reclassification (3-year Event NRI = 0.338, $P < 0.001$; 5-year Event NRI = 0.412, $P < 0.001$), highlighting the clinical value of EATV for accurately identifying high-risk patients. The joint classification revealed a striking stepwise risk gradient across groups: the High EATV/Low EATD (HR 2.123, 95% CI [1.130–3.989]; $P = 0.019$) and High EATV/High EATD (HR 2.592, 95% CI [1.299–5.171]; $P = 0.007$) groups exhibited significantly elevated risks compared with the Low EATV/Low EATD reference group (P for trend < 0.001).

Conclusion： In patients with heart failure (HF) complicated by pulmonary hypertension (PH), epicardial adipose tissue volume (EATV) is a strong independent predictor of adverse long-term clinical outcomes. Combining EATV with epicardial adipose tissue density (EATD) metrics delivers superior clinical risk stratification. Furthermore, integrating EATV into conventional clinical models significantly improves risk reclassification and increases net clinical decision benefit.

Key words： Epicardial adipose tissue , Heart failure , Pulmonary hypertension , Prognosis , Risk reclassification

论文 ID：318

Comparison of Lateralization Outcomes Between Simultaneous and Sequential Adrenal Venous Sampling Without ACTH Stimulation in Primary Aldosteronism

Hongliang Xiong[1], Yijun Wu[2], Li Du[3], Jiao Yang[1], Weitong Hu[1], Jiabing Huang[1], Yunde Li[1], Xi Chen[1], Yifei Dong*[1,4]

1. Department of Cardiovascular Medicine,The Second Affiliated Hospital of Nanchang University,NanChang,JiangXi,China,330006

2. Department of General Medicine, The Third Hospital of Jingdezhen City.

3. Department of Medical Imaging Center, The 2nd Affiliated Hospital, Jiangxi Medical College, Nanchang University, Jiangxi, China; Nanchang of Jiangxi, 330006, China.

4. Molecular Medicine of Jiangxi Key Laboratory, The Second Affiliated Hospital of Nanchang University, Nanchang of Jiangxi, 330006, China.

Corresponding Author E-mail: yf_dong66@126.com

Background： Adrenal venous sampling (AVS) is the gold standard for subtype classification in primary aldosteronism (PA). However, whether sequential AVS yields comparable results to simultaneous AVS remains controversial, and the optimal time interval between bilateral samplings has not been established.

Methods： We prospectively enrolled 73 PA patients who underwent AVS between April 2023 and December 2023. Simultaneous AVS (t=0) was compared with sequential AVS simulated by pairing the right AVS at t=0 min with the left AVS obtained at 5 or 15 minutes. Hormonal dynamics, lateralization concordance (ka

Results： During sequential sampling, left-sided cortisol and selectivity index (SI) decreased progressively over 15 minutes (both $P < 0.001$), while the aldosterone/cortisol (A/C) ratio rose from 1.2 to 2.6; aldosterone remained unchanged (both $P > 0.05$). Compared with simultaneous AVS, concordance was 71.2% (κ=0.570) at 5 minutes and declined to 56.2% (κ=0.359) at 15 minutes. Complete contralateral reversal occurred in 5.5% at 15 minutes (none at 5 minutes). Hypokalemia (OR= 4.96–5.58) and adrenal adenoma (OR= 3.83–7.00) independently predicted discordant lateralization.

Conclusion： Sequential AVS performed within 5 minutes yields acceptable concordance with simultaneous AVS, whereas delays beyond 5 minutes significantly increase discordance and lateralization reversal risk. Hypokalemia and adenoma are associated with more stable lateralization, as they independently predict lower discordance. We recommend limiting sequential AVS to ≤5 minutes when simultaneous sampling is not feasible.

Key words： primary aldosteronism; adrenal venous sampling; simultaneous AVS; sequential AVS; lateralization; concordance.

论文 ID：321

The Diagnostic Value of Six Obesity Indices Combined with Pulse Wave Velocity for the Prevalence of Left Ventricular Hypertrophy: Mediation and Interaction Analyses

Adila Wulamu[1,2], Hongjian Li*[2]

1. Xinjiang Medical University

2. The Fifth Affiliated Hospital of Xinjiang Medical University

Corresponding Author E-mail: lhjdoctor_109@126.com

Background： Hypertension is the primary cause of left ventricular hypertrophy (LVH), but blood pressure levels alone do not fully explain susceptibility to it.Obesity is a key driver of cardiovascular disease, and central obesity contributes to cardiac remodeling by increasing arterial stiffness. Body Mass Index (BMI) has limitations in capturing fat distribution and associated cardiovascular risks. Although new body fat indices are superior to BMI, they cannot replace the information on vascular function provided by the estimated pulse wave velocity (ePWV). The interplay and mediating pathway between obesity indices and ePWV in LVH identification remain unclear.

Methods： This study included 2,448 patients with hypertension, and WHtR, BRI, ABSI, WWI , CI ,and CVAI were calculated. We used multivariate logistic regression to examine the association between each indicator and LVH. We categorized each indicator and ePWV into four groups based on the median (both low, indicator alone, ePWV alone, both high) to evaluate combined effects. We used receiver operating characteristic (ROC) curves to evaluate combined diagnostic performance and restricted cubic spline(RCS) curves to explore dose-response relationships.We conduct a mediation analysis with ePWV as the mediator variable, and analyze the interaction effects of obesity stratified by BMI.

Results： The LVH group showed significant elevations in all six anthropometric indices (all $P < 0.001$). BMI, however, was comparable between groups ($P = 0.532$). After adjustment, all six indices were linked to LVH as continuous variables (odds ratio [OR]: 1.169–1.339, all $P < 0.05$). The top quartile of each index raised LVH risk (ORs: 1.552–2.354, all $P < 0.05$). WHtR and BRI showed higher risk from Q3, while WWI had the strongest effect in Q4.The RCS curves show that all indicators have a significant nonlinear association with LVH (all $P < 0.001$). WHtR combined with ePWV achieved an the area under the curve (AUC) of 0.701 (95% confidence interval [CI]: 0.664–0.738), outperforming each indicator alone. oint elevation of both markers produced the highest LVH risk (OR=4.32, P<0.001) with a clear dose-response pattern (P for trend<0.001). ABSI and WWI had substantial mediating effects through ePWV. The proportions were 57.4% and 49.1%, respectively (both P<0.001). Subgroup analysis revealed significant interactions between WWI and a history of diabetes (P=0.006), and between WHtR/BRI and cerebrovascular disease (both P=0.029). BMI stratification showed that BRI had the strongest effect in normal-weight individuals (OR=2.068). The effect declined as BMI increased (overweight: OR=1.438; obese: OR=1.295). Obesity significantly modifies the association between body fat indices and LVH, making these indices especially useful for detecting hidden visceral obesity in normal-weight individuals.

Conclusion： Body fat distribution indices outperform BMI in diagnosing LVH in hypertensive patients. WHtR combined with ePWV offers the best diagnostic performance. Arterial stiffness is a key pathway linking abnormal body fat to LVH (57.4% and 49.1% of the effects of ABSI and WWI, respectively, are mediated by ePWV). Obesity significantly modifies the associations. BRI, WHtR, and CVAI showed the strongest effects in normal-weight individuals. Their effects decreased as BMI increased, highlighting their value in detecting hidden visceral obesity in normal-weight people.

Key words： Primary hypertension;Left ventricular hypertrophy ;Anthropometric measures; arterial stiffness; mediation analysis; interaction

论文 ID：327

Cumulative Exposure to Red Blood Cell Traits Across Diabetes Progression and Risk of Cardiovascular Disease

Fan Yang, Yalan Li, Hong Yuan, Yao Lu*

湘雅三医院

Corresponding Author E-mail: luyao0719@163.com

Background： Red blood cell (RBC) traits have been associated with cardiovascular risk. However, their longitudinal patterns and cumulative effects in individuals with diabetes remain poorly understood. The aim of this study was to investigate the associations between cumulative exposure to RBC traits across different time windows and the risk of incident cardiovascular disease (CVD) among individuals with diabetes, and to explore the role of supplements use.

Methods： This ongoing longitudinal cohort study used data from the Health Management Center of the Third Xiangya Hospital (2005-2021). We included 5,668 participants (≥ 18 years) who had at least one physical examination before or after the diagnosis of diabetes. Repeated measurements of RBC traits were integrated within a predefined disease-stage framework to quantify cumulative exposure as the area under the curve (AUC) across prediagnosis and postdiagnosis windows. Stage-specific associations between cumulative exposure to RBC traits and incident CVD were evaluated using Cox proportional hazards models, and potential effect modification by supplement use was further assessed.

Results： Over an average follow-up period of 4.74 years, 895 participants patients developed cardiovascular disease. Cumulative RBC traits exhibited distinct temporal patterns of cardiovascular risk during diabetes progression. Higher cumulative hemoglobin (HGB) exposure was consistently associated with a lower risk of CVD throughout the diabetes course, with the most pronounced risk reduction observed across the entire diabetes course (HR = 0.68; 95% CI: [0.47, 0.99]). RBC count showed a stage-dependent pattern, with its inverse relationship with CVD risk becoming most evident during the intermediate post-diabetes period (HR = 0.64; 95% CI: [0.44, 0.93]). In contrast, elevated cumulative RBC distribution width (RDW) identified a higher-risk phenotype, particularly during the same intermediate post-diabetes period (HR = 1.25; 95% CI: [1.11, 1.41]). Moreover, the protective pattern associated with cumulative HGB exposure was amplified among supplement users, suggesting a potential modifiable pathway.

Conclusion： Cumulative exposure to RBC traits was associated with CVD risk among individuals with diabetes, with distinct patterns across disease stages. These findings highlight the potential value of longitudinal RBC traits monitoring in diabetes and warrant further investigation of modifiable factors, including supplement use.

论文 ID：328

Atherogenic Index of Plasma and Cardiovascular High-Risk Status in the ChinaHEART Luohe Cohort: Multivariable Association Modeling with Nonlinear

Haoran Wang, Zhiwei Huang, Li Wu, Qiaotao Xie*

Luohe Central Hospital

Corresponding Author E-mail: 18639998899@126.com

Background： Community-based cardiovascular disease (CVD) screening programs increasingly adopt the World Health Organization (WHO) CVD risk charts to identify individuals at high predicted 10-year risk (>=20%) who may benefit most from intensified preventive interventions. In resource-constrained primary care settings, risk stratification

frequently relies on scalable,standardized tools, and pragmatic biomarkers calculable from routinely collected data are particularly attractive. The atherogenic index of plasma (AIP), calculated as log10(triglycerides [TG] / high-density lipoprotein cholesterol [HDL-C]), is a simple composite marker that captures the combined atherogenic implication of elevated TG and reduced HDL-C--a dyslipidemic pattern closely linked to insulin resistance, central adiposity, and systemic low-grade inflammation. Because TG and HDL-C are routinely measured in community lipid panels, AIP is attractive as a zero-additional-cost, easily interpretable adjunct marker. However, whether AIP is associated with WHO chart-defined CVD high-risk status in community-dwelling adults, whether the dose-response relationship is linear across the full AIP distribution, and how AIP ranks in importance relative to other modifiable predictors remain clinically relevant questions that have not been fully addressed.

Methods： We conducted a cross-sectional analysis of baseline data from the China Health Evaluation And risk Reduction through nationwide Teamwork (ChinaHEART) community screening program in Luohe, Henan Province, China. Among 6,860 screened participants aged 35-75 years, 6,702 with complete data for AIP computation, WHO risk classification, and prespecified covariates were included (median age 58 years; 38% men). AIP was calculated from fasting TG and HDL-C. The primary outcome was WHO CVD risk chart-defined predicted 10-year CVD high-risk status (≥20%). Logistic regression was performed under three stepwise models: Model 1 (crude), Model 2 (+ alcohol use, marital status), and Model 3 (+ waist circumference, education, family history of stroke, antidiabetic/antihypertensive drugs, statin use). Restricted cubic splines (RCS) with 3 knots tested nonlinearity. ROC analyses with bootstrap confidence intervals evaluated discrimination; the Youden index derived the optimal AIP cutoff (0.231, 95% CI: 0.220-0.244). A pragmatic rounded cutoff of 0.2 was used for descriptive and categorical analyses to facilitate clinical interpretability. Subgroup and interaction analyses explored effect heterogeneity across prespecified strata. Finally, an explainable machine learning pipeline (LASSO logistic regression with 10-fold cross-validation for feature selection, forcing AIP into the model; random forest classifier with 500 trees; and SHAP-based interpretation via Monte Carlo approximation) was implemented to quantify AIP's multivariable predictive importance.

Results： Of 6,702 participants, 1,440 (21%) met the WHO CVD high-risk criterion. High-AIP participants (>=0.2, n=1,477, 22%) were slightly younger (median 57 vs. 59 years, P=0.024) and had a lower proportion of men (32% vs. 39%, P<0.001), yet exhibited more adverse cardiometabolic profiles: higher waist circumference (0.90 vs. 0.85 m), higher systolic/diastolic blood pressure (144/87 vs. 136/82 mmHg), higher fasting glucose (5.70 vs. 5.40 mmol/L), markedly higher TG (2.78 vs. 1.37 mmol/L), and lower HDL-C (1.21 vs. 1.48 mmol/L) (all P<0.001). CVD high-risk prevalence was 31% in the high-AIP group versus 19% in the low-AIP group (P<0.001). In logistic regression, AIP was positively associated with CVD high-risk status: each 1-unit increase in AIP was associated with higher odds in the crude model (OR: 2.415, 95% CI: 1.888-3.090, P<0.001) and remained significant after full adjustment (Model 3 OR: 1.717, 95% CI: 1.324-2.227, P<0.001). Using the categorical cutoff, high AIP conferred 55.3% higher odds (OR: 1.553, 95% CI: 1.356-1.780, P<0.001). RCS analyses supported a nonlinear association (P<0.05 for nonlinearity). AIP alone showed modest discrimination (AUC: 0.557, 95% CI: 0.541-0.576); discrimination improved with covariate adjustment (Model 3 AUC: 0.650, 95% CI: 0.634-0.667). In the LASSO-random forest pipeline, the model achieved AUC 0.792 on the test set. SHAP analyses ranked LDL-C and hypertension history as the strongest contributors, with AIP retained among the top predictive features, confirming its complementary value even after accounting for multiple conventional risk factors in a multivariable framework.

Conclusion： Higher AIP was nonlinearly and independently associated with WHO CVD risk chart-defined predicted 10-year CVD high-risk category in this large community-based ChinaHEART population. Although AIP alone had limited stand-alone discrimination, it was consistently retained in machine learning-based feature selection and contributed complementary information beyond traditional risk factors. The observed nonlinear dose-response pattern suggests that the risk gradient may steepen at higher AIP levels, which has implications for threshold-based

screening approaches. These findings support AIP as a simple, cost-free adjunct marker that may help triage individuals toward more formal multivariable CVD risk assessment in primary care and community screening settings. Prospective longitudinal studies with adjudicated incident CVD events are warranted to validate its clinical utility and to determine whether AIP-guided screening strategies can improve cardiovascular outcomes.

Key words： Atherogenic dyslipidemia; atherogenic index of plasma; cardiovascular disease risk; ChinaHEART; community screening; WHO CVD risk charts; machine learning; SHAP

论文 ID：330

Osteoporosis and Cardiac Remodeling in Middle-Aged and Older Adults: A Cross-Sectional Study

Qiaotao Xie*, Haoran Wang, Liying Shi

Luohe Central Hospital

Corresponding Author E-mail: 18639998899@126.com

Background： Osteoporosis, characterized by low bone mineral density (BMD) and microarchitectural deterioration of bone tissue, affects over 20% of the elderly population globally and represents a leading cause of fragility fractures, disability, and excess mortality. An accumulating body of evidence suggests a bidirectional pathophysiological relationship between osteoporosis and cardiovascular disease (CVD), with shared mechanisms including chronic low-grade systemic inflammation, dysregulated calcium and phosphate homeostasis, oxidative stress, hormonal changes (particularly estrogen deficiency), and altered signaling via the RANK/RANKL/OPG pathway--all of which may simultaneously drive bone resorption and adverse cardiac remodeling. However, while prior epidemiological studies have predominantly focused on the association between osteoporosis and clinical cardiovascular events such as myocardial infarction or stroke, the relationship between bone mineral density and subclinical cardiac structural and functional parameters--including left atrial size, ventricular wall thickness, and diastolic function--remains incompletely characterized. Understanding this relationship is important because subclinical cardiac remodeling often precedes overt cardiovascular events and may represent an earlier, potentially modifiable stage of disease.

Methods： This cross-sectional study analyzed baseline data from 1,233 participants (median age 60 years; 59% female) enrolled in the Longitudinal Investigation of Osteoarthritis and Cardiovascular Health Status cohort in Luohe, China (November 2023 to November 2024). Among 1,762 individuals in the parent cohort, 317 were excluded for missing BMD measurements and 212 for missing echocardiographic data. BMD was measured via tibial quantitative ultrasound (OsteoKJ7000+, Nanjing, China) with high intra-observer (kappa = 0.935) and inter-observer (kappa = 0.902) agreement. Participants were categorized as normal BMD (T-score >= -1.0, n=364), osteopenia (-2.5 < T-score < -1.0, n=404), or osteoporosis (T-score <= -2.5, n=465). Comprehensive transthoracic echocardiography assessed left atrial volume index (LAVI) via area-length method, left ventricular mass index (LVMI) via the Devereux formula, relative wall thickness (RWT) as (IVS+PWT)/LVIDd, left ventricular ejection fraction (LVEF) via Teichholz formula, and diastolic function metrics including E/e' ratio and tissue Doppler septal and lateral e' velocities. Multivariable linear regression models adjusted sequentially: Model 1 (unadjusted), Model 2 (age, sex), Model 3 (age, sex, BMI, SBP, total cholesterol, LDL-C, HDL-C, TG, smoking, alcohol, hypertension, diabetes, CHD, stroke). Collinearity diagnostics

(variance inflation factor) ensured model stability. Subgroup analyses were stratified by sex and age (<50, 50-60, >60 years), with formal interaction tests.

Results： Baseline characteristics differed significantly across BMD groups. Osteoporosis participants were older (median 64 vs. 58 years), predominantly female (74% vs. 40%), had lower smoking prevalence (14% vs. 32%), and higher triglyceride levels (1.73 vs. 1.48 mmol/L) (all P<0.05). Significant differences emerged in cardiac structural parameters: osteoporosis was associated with higher LAVI (median: 29 vs. 26 mL/m2, P<0.001) and higher RWT (0.38 vs. 0.37, P=0.047) compared to normal BMD. Kruskal-Wallis testing confirmed overall group differences for both LAVI (P<0.001) and RWT (P=0.019). After full multivariable adjustment (Model 3), T-score was independently and inversely associated with LAVI (beta = -0.358, P=0.043) and RWT (beta = -0.003, P=0.013), indicating that each 1-unit decrement in T-score corresponded to a 0.358 mL/m2 increase in LAVI and a 0.003 increase in RWT. Subgroup analyses revealed stronger associations in women (LAVI beta = -0.737, P=0.001 vs. men: beta = -0.116, P=0.696) and in participants younger than 50 years (LAVI beta = -0.909, P=0.022 vs. >60 years: beta = -0.468). A significant age interaction was observed for RWT (P for interaction = 0.006). No significant associations were found for LVMI, LVEF, or diastolic function metrics, suggesting that the bone-cardiac relationship selectively manifests through left atrial enlargement and concentric remodeling rather than through systolic dysfunction or overt diastolic impairment.

Conclusion： Lower BMD, measured by tibial quantitative ultrasound, is independently associated with left atrial enlargement and increased ventricular wall thickness in middle-aged and older adults, with notably stronger associations observed in women and younger individuals. These findings provide novel evidence supporting shared pathophysiological mechanisms between bone loss and subclinical cardiac structural remodeling, particularly in the left atrium and ventricular wall. The stronger associations in younger age groups and in women suggest potential windows of opportunity for early, sex-specific interventions targeting both skeletal and cardiovascular health. Integration of echocardiographic assessment into osteoporosis management pathways may help identify individuals at elevated risk for adverse cardiac remodeling.

Key words： Osteoporosis; bone mineral density; cardiac structure; left atrial volume; relative wall thickness; echocardiography; cross-sectional study; quantitative ultrasound

论文 ID：331

Association between Body Roundness Index and Diastolic Function in Middle-Aged and Older Adults: A Cross-Sectional Study

Qiaotao Xie*, Haoran Wang, Liying Shi

Luohe Central Hospital

Corresponding Author E-mail: 18639998899@126.com

Background： Diastolic dysfunction (DD) represents an early and potentially reversible indicator of cardiac impairment that is strongly linked to adverse cardiovascular outcomes including incident heart failure with preserved ejection fraction, atrial fibrillation, and all-cause mortality. Identification of modifiable risk factors for DD is therefore an important public health and clinical priority. While body mass index (BMI) has long been the standard anthropometric measure of adiposity, it does not differentiate fat from lean mass and fails to capture body shape and

central fat distribution--both of which are critical determinants of cardiometabolic risk. Central adiposity, in particular, is associated with pericardial fat accumulation, myocardial lipotoxicity, and pro-inflammatory cytokine release, all of which may contribute to impaired myocardial relaxation. The Body Roundness Index (BRI), a novel geometric anthropometric metric derived solely from waist circumference (WC) and height using the formula BRI = 364.2 - 365.5 x sqrt[1 - (WC/(2 x pi x Height/2))^2], was developed to approximate body roundness and central adiposity more accurately than BMI. However, the specific association between BRI and echocardiographically assessed diastolic function in community-dwelling middle-aged and older adults has not been comprehensively characterized.

Methods： This cross-sectional analysis included 1,466 participants from the Longitudinal Investigation of Osteoarthritis and Cardiovascular Health Status cohort in Luohe, China (median age ~60 years). After excluding 292 participants without sufficient echocardiographic data and 4 with missing anthropometric measurements, 1,466 individuals were retained. BRI was calculated from standardized measurements of WC (at the umbilical level) and height (via stadiometer). Comprehensive transthoracic echocardiography was performed by trained sonographers using a Philips Epic-7 system in accordance with 2025 American Society of Echocardiography guidelines. Key parameters included tissue Doppler-derived septal and lateral mitral annular e' velocities, E/e' ratio (transmitral early diastolic velocity divided by average e'), left atrial volume index (LAVI), left ventricular mass index (LVMI), and left ventricular ejection fraction (LVEF). DD was defined using age-dependent e' velocity cutoffs and E/e' thresholds. The optimal BRI cutoff was derived via 1,000-iteration bootstrap ROC analysis using the Youden index. Multivariable logistic regression examined the association between BRI (continuous and categorical) and DD, adjusting for age, sex, systolic blood pressure, total cholesterol, LDL-C, HDL-C, triglycerides, current smoking, alcohol use, hypertension, diabetes, coronary heart disease, and stroke history. Robust linear regression quantified associations between BRI and continuous echocardiographic parameters. Restricted cubic spline (RCS) models with 3 knots explored dose-response patterns, and prespecified subgroup and interaction analyses assessed potential effect modification.

Results： Higher BRI was significantly and independently associated with increased odds of DD. In fully adjusted logistic regression, each 1-unit increase in BRI was associated with 14.4% higher odds of DD (OR = 1.144, 95% CI: 1.046-1.251, P=0.003). Using the bootstrap-derived optimal cutoff (BRI >= 4.2), high BRI conferred 41.7% higher odds of DD compared with low BRI (OR = 1.417, 95% CI: 1.114-1.804, P=0.004). Robust linear regression further quantified the relationship with continuous echocardiographic parameters: BRI was inversely associated with septal e' velocity (beta = -0.164, P = 8.10 x 10^-4), inversely associated with lateral e' velocity (beta = -0.167, P = 5.59 x 10^-3), and showed a borderline positive association with E/e' ratio (beta = 0.121, P = 0.053). These findings indicate that higher BRI corresponds to lower tissue Doppler relaxation velocities and a trend toward elevated left ventricular filling pressures. RCS models demonstrated a significant nonlinear association between BRI and DD probability (P<0.001 for overall association; P=0.006 for nonlinearity), suggesting that the risk of DD may accelerate beyond certain BRI thresholds. Interaction analyses indicated that BRI's effect on DD was not significantly modulated by blood pressure levels or lipid profiles, pointing to a relatively robust and consistent relationship across metabolic strata.

Conclusion： BRI is nonlinearly and independently associated with impaired diastolic function--including significantly reduced tissue Doppler e' velocities and a trend toward elevated filling pressures--in middle-aged and older community-dwelling adults. As a simple, non-invasive anthropometric measure requiring only a measuring tape and stadiometer, without any laboratory testing or specialized imaging, BRI may serve as a practical and low-cost screening tool to identify individuals at elevated risk for subclinical diastolic dysfunction in primary care, community health screening programs, and resource-limited settings. These findings provide novel evidence linking body shape metrics to early cardiac functional impairment and support further prospective investigation of BRI as a predictor of incident diastolic dysfunction.

Key words： Adiposity; Body Roundness Index; cardiovascular disease; diastolic dysfunction; echocardiography; tissue Doppler imaging; central obesity

论文 ID：332

Adiposity Measured by Body Roundness Index Is Significantly Associated with Increased Stroke Prevalence: A Population-Based Cross-Sectional Study

Qiaotao Xie*, Haoran Wang, Zhiwei Huang, Liying Shi

Luohe Central Hospital

Corresponding Author E-mail: 18639998899@126.com

Background： Stroke remains a leading cause of disability and mortality worldwide, with a particularly heavy and growing burden in China driven by rapid population aging and the rising prevalence of modifiable risk factors including hypertension, diabetes, and obesity. Central adiposity is metabolically active and mechanistically linked to several pathways relevant to cerebrovascular disease: chronic low-grade inflammation, insulin resistance, endothelial dysfunction, oxidative stress, and pro-thrombotic states. Traditional anthropometric measures such as body mass index (BMI) do not distinguish fat distribution and may substantially underestimate the cerebrovascular impact of central adiposity. The Body Roundness Index (BRI), derived from waist circumference (WC) and height using a validated geometric formula, was specifically designed to capture body shape and central adiposity more effectively than BMI. While BRI has been associated with various cardiometabolic outcomes, evidence from large-scale community-based cardiovascular screening programs regarding its association with prevalent stroke--and the incremental discriminative value it provides beyond conventional risk factors--remains limited.

Methods： We performed a cross-sectional analysis using data from 6,858 adults aged 35-75 years from the Luohe branch of the ChinaHEART community cardiovascular screening program in central China. BRI was calculated from standardized anthropometric measurements using the validated formula: BRI = 364.2 - 365.5 x sqrt[1 - (WC/(2 x pi x Height/2))^2], where WC and height are expressed in meters. Prevalent stroke was ascertained by self-reported physician diagnosis. Bootstrap ROC analysis (1,000 iterations) with Youden index optimization determined the operational BRI cutoff (median threshold: 4.597, 95% CI: 4.149-4.798), and a rounded value of 4.6 was used for categorical analyses. Three nested logistic regression models were constructed: Model 1 (BRI only), Model 2 (+ age, sex), and Model 3 (+ marital status, smoking, alcohol use, hypertension, diabetes, systolic and diastolic blood pressure, fasting glucose, total cholesterol, HDL-C, LDL-C, triglycerides). Incremental discrimination was quantified as delta-AUC (AUC with BRI minus AUC without BRI) with 1,000-iteration stratified bootstrap 95% CIs. Restricted cubic spline (RCS) analysis with 3 knots evaluated dose-response linearity. Prespecified subgroup analyses examined strata of sex, age (<60 vs. >=60 years), smoking, alcohol use, hypertension, and diabetes, with formal interaction tests via likelihood ratio tests. A sensitivity analysis additionally adjusted for socioeconomic indicators (education, occupation, household income).

Results： Among 6,858 participants, 192 (2.8%) reported prior physician-diagnosed stroke. Participants with high BRI (>=4.6, n=2,261) had a stroke prevalence of 4.2% (96/2,261) compared with 2.1% (96/4,597) in the low-BRI group (absolute difference: 2.1 percentage points). High-BRI participants were older (median 60 vs. 57 years), had higher blood pressure and worse lipid profiles, and had higher prevalence of hypertension and diabetes (all P<0.001). BRI

alone showed limited discrimination (Model 1 AUC: 0.584, 95% CI: 0.541-0.628). Discrimination improved progressively with covariate addition: Model 2 AUC was 0.683 (95% CI: 0.648-0.719), and fully adjusted Model 3 AUC reached 0.740 (95% CI: 0.704-0.775). The incremental discriminative contribution of BRI was significant in Model 2 (delta-AUC: 0.012, 95% CI: 0.001-0.035) but modest in Model 3 (delta-AUC: 0.004, 95% CI: -0.000-0.017). Sensitivity analysis with additional socioeconomic adjustment yielded a delta-AUC of 0.005 (95% CI: 0.000-0.017). In the fully adjusted logistic model, high BRI was associated with significantly elevated odds of prevalent stroke (OR: 1.766, 95% CI: 1.279-2.438). RCS analysis showed no significant evidence of nonlinearity (P for nonlinearity = 0.915), supporting a linear dose-response relationship across the BRI distribution. Subgroup analyses demonstrated broadly consistent associations across sex, age, smoking, and comorbidity strata.

Conclusion： BRI is independently and linearly associated with prevalent stroke in a large community-based Chinese cardiovascular screening population. As a simple, non-invasive anthropometric measure requiring only a measuring tape and stadiometer, BRI may offer complementary discriminatory information for community stroke screening and risk triage beyond conventional demographic and clinical factors. The linear dose-response relationship suggests that even modest reductions in central adiposity, as reflected by incrementally lower BRI values, may correspond to incrementally lower stroke prevalence. Prospective cohort studies with adjudicated incident stroke outcomes are warranted to evaluate BRI's longitudinal predictive value and to determine whether BRI-guided screening strategies can improve cerebrovascular risk stratification in community settings. BRI is independently and linearly associated with prevalent stroke in a large community-based Chinese cardiovascular screening population. As a simple, non-invasive anthropometric measure requiring only a measuring tape and stadiometer, BRI may offer complementary discriminatory information for community stroke screening and risk triage beyond conventional demographic and clinical factors. The linear dose-response relationship suggests that even modest reductions in central adiposity, as reflected by incrementally lower BRI values, may correspond to incrementally lower stroke prevalence. Prospective cohort studies with adjudicated incident stroke outcomes are warranted to evaluate BRI's longitudinal predictive value and to determine whether BRI-guided screening strategies can improve cerebrovascular risk stratification in community settings.

Key words： Anthropometry; Body Roundness Index; cross-sectional study; population study; screening triage; stroke prevalence; central adiposity; ChinaHEART

论文 ID：333

Triangulating Evidence for Cardiometabolic Index: ROC Cutoff, Spline Nonlinearity, and Explainable Machine Learning for CVD High Risk

li wu*, Haoran Wang, Zhiwei Huang, Qiaotao Xie

Luohe Central Hospital

Corresponding Author E-mail: 13781735727@163.com

Background： Adiposity-related metabolic dysfunction and lipid abnormalities frequently co-occur and jointly accelerate cardiometabolic deterioration. Conventional single-domain markers--such as waist circumference or triglycerides alone--may not fully capture the combined burden of central adiposity and atherogenic dyslipidemia. The cardiometabolic index (CMI), calculated as (TG/HDL-C) x waist-to-height ratio (WHtR), was designed to

simultaneously integrate two complementary domains: an anthropometric component reflecting central adiposity (WHtR) and a biochemical component reflecting the balance between atherogenic and protective lipid fractions (TG/HDL-C). This dual-domain architecture may provide more comprehensive cardiometabolic risk information than isolated indicators. While accumulating evidence links composite metabolic indices to hypertension, diabetes, and other cardiometabolic outcomes, less is known about whether CMI aligns with guideline-relevant cardiovascular risk categories--specifically, WHO chart-defined CVD high-risk status--in community-dwelling Chinese adults. Moreover, the dose-response pattern may be nonlinear, the relative importance of CMI among conventional predictors has not been characterized across complementary analytical frameworks, and the association may differ across clinically relevant subgroups.

Methods： We analyzed cross-sectional screening data from 6,701 participants (median age 58 years; 38% men) in the ChinaHEART Luohe cohort, conducted between March 2021 and February 2022. WHO chart-defined CVD high risk was the primary outcome (10-year predicted risk >=20%). CMI was calculated as (TG/HDL-C) x WHtR, where WHtR = waist circumference/height. CMI was evaluated per 1-unit increase and dichotomized using a rounded pragmatic analytic threshold of 0.7, derived from the crude ROC Youden optimal threshold of 0.723 (95% CI: 0.683-0.795). Analytical evidence was triangulated across multiple complementary frameworks: raincloud plots for distributional comparison between risk groups; bootstrap ROC analyses (AUC with 95% CI) under stepwise models; restricted cubic splines (RCS) for characterizing dose-response nonlinearity; stepwise logistic regression under three nested models (Model 1: crude; Model 2: + alcohol, marital status; Model 3: + education, family history of stroke, antidiabetic/antihypertensive drugs, statin use); exploratory subgroup forest plots with formal likelihood-ratio interaction tests; and an explainable machine learning (ML) pipeline incorporating LASSO logistic regression (10-fold cross-validation, lambda.1se) for feature selection, random forest classifier (500 trees, 70/30 split) for discrimination, and SHAP values (fastshap) for model interpretability and feature importance ranking.

Results： Among 6,701 participants, 1,439 (21%) were classified as CVD high risk. Using the pragmatic cutoff (CMI >= 0.7), 2,326 (35%) were categorized as high CMI. The high-CMI group exhibited significantly worse cardiometabolic profiles: higher systolic/diastolic blood pressure (143/86 vs. 135/82 mmHg), higher fasting glucose (5.70 vs. 5.40 mmol/L), higher triglycerides (2.33 vs. 1.27 mmol/L), lower HDL-C (1.26 vs. 1.52 mmol/L), and higher prevalence of hypertension (32% vs. 18%) and diabetes (9.0% vs. 4.8%) (all P<0.001). CVD high-risk prevalence was 29% in the high-CMI group versus 18% in the low-CMI group (P<0.001). In ROC analysis, CMI alone showed modest discrimination (AUC: 0.571, 95% CI: 0.555-0.586), while multivariable models incorporating CMI achieved improved discrimination (Model 3 AUC: 0.642, 95% CI: 0.626-0.659). Raincloud plots confirmed significantly higher CMI distributions in the high-risk group (P<0.001). RCS analysis revealed a significant nonlinear dose-response relationship (P overall < 0.001; P for nonlinearity = 0.020). In fully adjusted Model 3, CMI was independently associated with CVD high risk both continuously (OR: 1.31 per 1-unit, 95% CI: 1.16-1.48, P<0.001) and categorically (OR for >=0.7 vs. <0.7: 1.50, 95% CI: 1.32-1.71, P<0.001). Significant interactions were observed for age (P=0.008), antihypertensive drug use (P<0.001), and statin use (P<0.001). In the ML pipeline, random forest achieved AUC 0.814 on the test set. SHAP analysis ranked CMI as the 3rd most important predictor among 7 LASSO-selected features, following age and systolic blood pressure, confirming robust and consistent importance across analytical paradigms.

Conclusion： By triangulating evidence from conventional logistic regression, nonlinear spline modeling, and explainable machine learning, we demonstrate that higher CMI is consistently and nonlinearly associated with WHO-defined CVD high-risk status. CMI was ranked among the top three predictive features in SHAP analysis, reinforcing its potential value as a pragmatic, dual-domain screening adjunct. The integration of central adiposity (WHtR) and atherogenic dyslipidemia (TG/HDL-C) into a single index may capture complementary risk information beyond that

provided by single-domain markers. The observed effect modification by medication use underscores the importance of considering treatment status when interpreting CMI in clinical screening contexts.

Key words： Cardiometabolic index; cardiovascular disease; interaction; machine learning; restricted cubic spline; risk stratification; SHAP; subgroup analysis; waist-to-height ratio

论文 ID：334

Association between the Ratio of Total Cholesterol to High-Density Lipoprotein Cholesterol and High Cardiovascular Risk: A Cross-Sectional Study

Qiaotao Xie*, Haoran Wang, Shuaifang Yuan, Liying Shi

Luohe Central Hospital

Corresponding Author E-mail: 18639998899@126.com

Background： Cardiovascular disease (CVD) remains the leading cause of global mortality and disability. Early identification of individuals at high CVD risk and timely implementation of preventive interventions can substantially reduce the incidence of major adverse cardiovascular events. While lowering low-density lipoprotein cholesterol (LDL-C) is the cornerstone of primary CVD prevention, substantial residual risk persists even after adequate LDL-C control, highlighting the need for more comprehensive lipid-based risk assessment parameters that extend beyond single lipid measures. The total cholesterol to high-density lipoprotein cholesterol (TC/HDL-C) ratio is a composite lipid index that simultaneously captures three complementary lipid-related risk dimensions: the protective effects of HDL-C-mediated reverse cholesterol transport, the atherogenic burden of LDL-C, and the contribution of triglyceride-rich remnant lipoproteins and lipoprotein(a). This integrative property makes the TC/HDL-C ratio potentially more informative than any single lipid parameter (TC, LDL-C, or HDL-C alone). Moreover, it is immediately calculable from any standard fasting or non-fasting lipid panel without additional cost or testing. Despite growing evidence of its prognostic role, large-scale verification of its ability to identify WHO-defined high CVD risk individuals in general community populations--using internationally recognized risk assessment frameworks--has been limited.

Methods： This cross-sectional study used community screening data from the ChinaHEART program in Luohe, Henan Province, China (2021-2022). Among 6,860 adults aged 35-75 years initially screened, 274 participants (4.0%) were excluded due to irreversible missing lipid profile data (invalid fingertip blood samples), leaving 6,586 participants for final analysis. The TC/HDL-C ratio was calculated as total cholesterol (mmol/L) divided by HDL-C (mmol/L). The primary outcome was WHO chart-defined high 10-year CVD risk (>=20%) using the laboratory-based WHO/ISH Risk Prediction Charts calibrated for Chinese populations. Restricted cubic spline (RCS) analysis with 5 knots placed at the 10th, 25th, 50th, 75th, and 90th percentiles explored dose-response patterns. ROC analysis with 1,000-iteration bootstrap correction assessed discriminatory performance. Three nested logistic regression models were constructed: Model 1 (unadjusted), Model 2 (+ age, sex), and Model 3 (+ marital status, BMI, waist circumference, fasting glucose, smoking history, alcohol use, diabetes, and hypertension history). Quartile-based analysis compared the highest versus lowest TC/HDL-C quartiles. Sample size calculation confirmed adequate statistical power: the minimum required total sample was 3,624 (906 per quartile), substantially exceeded by the actual analytic sample.

Results：Among 6,586 participants (median age 58 years; 38% male), 1,414 (21%) were classified as CVD high risk. A clear gradient was observed across TC/ HDL-C quartiles: the prevalence of CVD high-risk status increased from 16% in Q1 (0.35-2.75) to 15% in Q2 (2.75-3.28), 18% in Q3 (3.28-3.94), and 38% in Q4 (3.94-14.29) (P<0.001). Participants in Q4 exhibited significantly worse cardiometabolic profiles than those in Q1: higher systolic BP (142 vs. 135 mmHg, P<0.001), higher BMI (26.0 vs. 24.1 kg/m2, P<0.001), larger waist circumference (89 vs. 83 cm, P<0.001), higher fasting glucose (5.70 vs. 5.40 mmol/L, P<0.001), higher LDL-C (3.35 vs. 1.96 mmol/L), and higher triglycerides (1.94 vs. 1.21 mmol/L), with correspondingly higher hypertension (28% vs. 20%) and diabetes prevalence (8.9% vs. 4.4%). RCS analysis demonstrated a positive, monotonic association between TC/HDL-C and high CVD risk. TC/HDL-C alone showed AUC of 0.613 (95% CI: 0.596-0.630). After incorporating all covariates in Model 3, AUC increased substantially to 0.847 (95% CI: 0.835-0.859). In fully adjusted logistic regression, each 1-unit increase in TC/HDL-C was associated with a 97% higher odds of CVD high risk (OR: 1.97, 95% CI: 1.83-2.13, P<0.001). Compared with Q1, the highest quartile (Q4) had an adjusted OR of 2.95 (95% CI: 2.42-3.60, P<0.001).

Conclusion：The TC/HDL-C ratio is independently, strongly, and positively associated with WHO-defined high CVD risk in a large community-dwelling Chinese adult population, demonstrating excellent combined discriminatory performance (AUC 0.847) in fully adjusted models. As a readily available, cost-free lipid-derived parameter that can be immediately calculated from any standard lipid panel, the TC/HDL-C ratio represents a valuable practical tool for enhancing CVD risk assessment in primary prevention, community screening, and public health settings. Its strong, graded association--with nearly three-fold higher odds in the highest versus lowest quartile--supports its integration into risk stratification workflows to complement existing assessment frameworks.

Key words：Cardiovascular disease; ChinaHEART; high cardiovascular risk; lipid ratio; TC/HDL-C ratio; WHO CVD risk chart; primary prevention; risk stratification

论文 ID：335

The Relationship between Triglyceride to High-Density Lipoprotein Cholesterol Ratio and Cardiovascular High Risk: A Cross-Sectional Investigation

Haoran Wang*, Li Wu, Qiaotao Xie

Luohe Central Hospital

Corresponding Author E-mail: washingtonhr@163.com

Background：Cardiovascular diseases (CVDs) remain the leading cause of morbidity and mortality worldwide, posing a substantial global health and economic burden. Early identification of individuals at high risk of developing CVD is crucial for implementing targeted prevention and management strategies. While traditional risk factors such as hypertension, dyslipidemia, smoking, and diabetes are well-established, there is growing interest in exploring novel, readily available biomarkers that may enhance risk stratification in the general population. The triglyceride to high-density lipoprotein cholesterol (TG/HDL-C) ratio has emerged as a promising candidate marker for cardiovascular risk assessment. This ratio reflects the physiological balance between atherogenic triglyceride-rich lipoproteins (including VLDL, IDL, and chylomicron remnants) and anti-atherogenic HDL-C-mediated reverse cholesterol transport. By simultaneously capturing two opposing lipid forces, the TG/HDL-C ratio provides more comprehensive lipid-related risk information than either parameter alone. The ratio has been associated with insulin resistance, small dense LDL

particle phenotype, cardiometabolic syndrome, and cardiovascular morbidity and mortality in diverse populations. However, its precise utility in identifying WHO-defined CVD high-risk groups within the general community population--using internationally recognized, standardized risk assessment frameworks--remains underexplored and requires large-scale community-based validation.

Methods： This cross-sectional investigation was a branch of the ChinaHEART cohort in middle China, involving 6,593 community-dwelling adults aged 35-75 years recruited from two township hospitals in Luohe City between November 2, 2021 and February 20, 2022. After excluding 267 participants (3.9%) with irreversible missing lipid profile data, 6,593 participants were retained. The TG/HDL-C ratio was calculated from fasting lipid profiles. CVD high risk was defined using WHO/ISH Risk Prediction Charts for the Western Pacific Region B (10-year risk >=20%). Participants were stratified into quartiles of TG/HDL-C for descriptive analysis. Restricted cubic spline (RCS) analysis with 5 knots at the 10th, 25th, 50th, 75th, and 90th percentiles evaluated dose-response patterns. ROC analysis with 1,000-iteration bootstrap correction assessed discriminatory performance, and the Youden index determined the optimal cutoff. Logistic regression was performed with Model 1 (unadjusted) and Model 2 (fully adjusted for age, sex, marital status, BMI, waist circumference, fasting glucose, smoking, alcohol use, diabetes, systolic blood pressure, and diastolic blood pressure). Net reclassification improvement (NRI) quantified the incremental discriminative value of adding TG/HDL-C to the base model.

Results： Among 6,593 participants (median age 58 years; 38% male), 1,417 (21.5%) were classified as CVD high risk. Baseline characteristics across TG/HDL-C quartiles revealed a pronounced cardiometabolic gradient: comparing Q4 versus Q1, participants had higher BMI (26.0 vs. 24.1 kg/m2), larger waist circumference (89 vs. 83 cm), higher systolic BP (141.5 vs. 135 mmHg), higher diastolic BP (85 vs. 82.5 mmHg), higher fasting glucose (5.70 vs. 5.40 mmol/L), higher TC (5.62 vs. 4.08 mmol/L), higher LDL-C (3.35 vs. 1.96 mmol/L), and elevated TG (1.94 vs. 1.21 mmol/L) with lower HDL-C (1.21 vs. 1.71 mmol/L) (all P<0.001). The prevalence of hypertension increased from 20% (Q1) to 28% (Q4), and diabetes from 4% to 9% (P<0.001). The distribution of CVD high-risk individuals across quartiles was: 16% (Q1), 15% (Q2), 18% (Q3), and 38% (Q4). RCS analysis demonstrated a positive and broadly linear association. In ROC analysis, TG/HDL-C alone showed AUC of 0.558 (95% CI: 0.541-0.575), which increased substantially to 0.820 (95% CI: 0.806-0.833) in the fully adjusted Model 2. The NRI index was 0.107 +/- 0.030 (P=0.016), confirming statistically significant incremental discriminative improvement. The Youden-derived optimal TG/HDL-C cutoff was 1.392 (95% CI: 1.313-1.551). In fully adjusted logistic regression, each 1-unit increase in TG/HDL-C was associated with 21% higher odds of CVD high risk (OR: 1.21, 95% CI: 1.17-1.25, P<0.001).

Conclusion： The TG/HDL-C ratio demonstrates significant promise as a practical, readily available biomarker for CVD risk assessment in community settings, exhibiting excellent combined discriminatory power (AUC 0.820) in fully adjusted models and providing statistically significant reclassification improvement (NRI 0.107, P=0.016). As a simple, cost-free marker routinely derivable from standard fasting lipid panels, the TG/HDL-C ratio can be immediately incorporated into existing cardiovascular risk assessment workflows to provide supplementary information that may help healthcare professionals refine risk stratification and guide preventive intervention strategies in primary care and population-level screening programs.

Key words： TG/HDL-C ratio; cardiovascular high risk; cross-sectional study; public health; lipid fractions; risk stratification; ChinaHEART; net reclassification improvement

论文 ID：336

TyHGB and CVD High-Risk Stratification: Nonlinear Association and Discrimination in the ChinaHEART Luohe Study

Zhiwei Huang, Haoran Wang, Li Wu, Qiaotao Xie*

Luohe Central Hospital

Corresponding Author E-mail: 18639998899@126.com

Background： Cardiovascular disease (CVD) remains a leading cause of morbidity and mortality worldwide, and identifying individuals at high risk is central to efficient prevention strategies. Conventional risk prediction tools, including the WHO CVD risk charts, enable population-level screening and prioritization of preventive interventions. However, traditional risk factors do not fully capture residual risk, motivating interest in pragmatic composite metabolic indices derived from routinely available measurements. The triglyceride-glucose (TyG) index has emerged as a convenient surrogate for insulin resistance and has been associated with cardiometabolic outcomes in diverse populations. Nonetheless, TyG alone--calculated solely from fasting triglycerides and glucose--may not adequately reflect the combined metabolic burden contributed by adiposity and protective lipid fractions. TyHGB is a novel composite index that extends the conceptual framework of TyG by integrating four routinely measured parameters: TyHGB = (TG/HDL-C) + 0.7 x fasting plasma glucose (FPG) + 0.1 x BMI. This formula simultaneously captures atherogenic dyslipidemia (via TG/HDL-C ratio), glycemic status (via FPG), and overall adiposity (via BMI), potentially providing a more comprehensive and multidimensional reflection of cardiometabolic risk. However, evidence regarding TyHGB's association with guideline-defined CVD high-risk categories, its comparative discrimination against established indices such as TyG, and its incremental value beyond conventional risk factors remains limited and has not been evaluated in large Chinese community cohorts.

Methods： This cross-sectional study analyzed 6,755 adults (median age 58 years) from the community-based ChinaHEART Luohe screening cohort (2021-2022), after excluding 105 participants with missing TyHGB data. CVD high risk was defined as an estimated 10-year WHO CVD risk >=20%. TyHGB was calculated using the published formula (TG/HDL-C + 0.7 x FPG + 0.1 x BMI) with TG, HDL-C, and FPG in mmol/L and BMI in kg/m2. TyHGB was examined per 1-unit increase and as high versus low using an ROC-derived Youden cutoff (~7.6). The TyG index, calculated as ln[TG(mg/dL) x FPG(mg/dL)/2] after unit conversion, was analyzed in parallel for direct comparison. Three nested logistic regression models were constructed: Model 1 (crude), Model 2 (+ age, sex), and Model 3 (+ current smoking, alcohol consumption, waist circumference, systolic blood pressure, family history of stroke, heart rate). Restricted cubic splines (RCS) explored nonlinear dose-response patterns. ROC analyses with bootstrap AUC estimates evaluated discrimination, and DeLong's test formally compared AUCs between TyHGB and TyG. Incremental value was assessed using integrated discrimination improvement (IDI) and categorical net reclassification improvement (NRI). An exploratory machine learning pipeline (LASSO logistic regression, 10-fold CV, lambda.1se -> random forest, 500 trees, 70/30 split -> SHAP via fastshap) provided complementary feature importance ranking. Sensitivity analyses employed multiple imputation by chained equations (MICE, 20 imputations, 20 iterations) under a missing-at-random assumption.

Results： CVD high-risk prevalence was 22% in the analytic sample. In unadjusted ROC analyses, TyHGB demonstrated significantly higher discrimination than TyG (AUC: 0.624 vs. 0.585; DeLong P<0.001). TyHGB was independently associated with CVD high risk across all models: the fully adjusted (Model 3) OR per 1-unit TyHGB

increase was 1.131 (95% CI: 1.096-1.167, P<0.001). High TyHGB (>=7.6) was associated with 64.2% higher odds of CVD high risk compared with low TyHGB (OR: 1.642, 95% CI: 1.446-1.864, P<0.001). RCS analysis confirmed a significant nonlinear dose-response relationship (P overall <0.001; P for nonlinearity <0.05). Adding TyHGB to a base risk-factor model produced modest but statistically significant incremental improvements: IDI = 0.003 (P<0.001) and categorical NRI = 0.216 (P<0.001), with the NRI primarily driven by improved classification of non-events. In the exploratory ML pipeline, TyHGB was consistently retained by LASSO feature selection and ranked among the top predictive features in SHAP analysis alongside age, LDL-C, and non-HDL-C. Sensitivity analyses using MICE with 20 imputations yielded pooled estimates consistent with the primary complete-case analysis, confirming robustness to missing data patterns.

Conclusion： TyHGB is positively, independently, and nonlinearly associated with WHO-defined CVD high-risk status in community-dwelling Chinese adults. The composite index demonstrated significantly higher unadjusted discrimination than the established TyG index (AUC 0.624 vs. 0.585, P<0.001) and provided modest but statistically significant incremental reclassification improvement beyond conventional risk factors (NRI 0.216). By simultaneously integrating glyco-lipid perturbation, glycemic status, and adiposity into a single practical score, TyHGB may serve as a useful composite screening tool for community-based cardiovascular risk stratification. These cross-sectional findings relate to estimated risk status rather than adjudicated incident CVD events, and prospective validation with hard cardiovascular endpoints is warranted.

Key words： Cardiovascular disease high-risk; ChinaHEART cohort; risk stratification; triglyceride-glucose index (TyG); TyHGB; WHO CVD risk charts; composite metabolic index; net reclassification improvement

论文 ID：337

Integrating Epidemiologic Modeling and Explainable Machine Learning to Evaluate Body Roundness Index for WHO-Defined High Cardiovascular Risk: Evidence from the ChinaHEART-Luohe Screening Cohort

li wu*, Qiaotao Xie, Zhiwei Huang, Haoran Wang

Luohe Central Hospital

Corresponding Author E-mail: 13781735727@163.com

Background： Efficient identification of individuals at high cardiovascular disease (CVD) risk is essential for prevention in middle-aged and older adults, particularly in primary-care and community screening programs where resources and time are limited. While body mass index (BMI) is widely used to characterize adiposity, it does not capture body shape or central fat distribution patterns that may better reflect metabolic dysfunction. The Body Roundness Index (BRI), a geometric anthropometric index derived solely from waist circumference and height using the formula BRI = 364.2 - 365.5 x sqrt[1 - (WC/(2 x pi x Height/2))^2], approximates body shape and central adiposity without requiring specialized equipment. If BRI is independently associated with validated, guideline-based CVD risk classification, it could serve as a pragmatic, low-cost adjunct marker to prioritize individuals for more comprehensive evaluation. However, evidence integrating conventional epidemiologic approaches with explainable machine learning to evaluate BRI for WHO-defined CVD high-risk status remains limited.

Methods： This cross-sectional study used baseline data from 6,858 adults aged 35-75 years in the Luohe branch of the ChinaHEART community screening program (March 2021 to February 2022). WHO-defined CVD high-risk status was determined using WHO CVD risk charts (10-year risk >=20%). BRI was analyzed as a continuous variable (per 1-unit increase), quartiles, and a binary variable using an ROC-derived pragmatic threshold (BRI >= 4, derived from the Youden-index optimal cutoff of 4.151). Three stepwise logistic regression models were constructed: Model 1 (crude), Model 2 (+ alcohol consumption, triglycerides), and Model 3 (+ education, marital status, diastolic blood pressure, heart rate). Covariates were intentionally restricted to non-chart variables to reduce circularity with the WHO risk-chart outcome. Restricted cubic splines (RCS) characterized dose-response nonlinearity. ROC analyses with bootstrap confidence intervals evaluated discrimination and cutoff determination. Prespecified subgroup and interaction analyses examined heterogeneity across age, sex, education, marital status, alcohol use, diastolic blood pressure, heart rate, and triglyceride strata, with Benjamini-Hochberg false discovery rate (FDR) correction. An explainable machine learning pipeline (LASSO logistic regression with 10-fold cross-validation, random forest classifier with 500 trees and 70/30 split, and SHAP interpretation) provided complementary feature importance evidence. Supplementary analyses compared BRI with BMI and waist circumference using delta-AUC, continuous net reclassification improvement (NRI), and decision-curve analysis.

Results： Among 6,858 participants (median age 58 years; 38% men), 1,489 (22%) were classified as WHO-defined CVD high risk. High-BRI participants (>=4, n=3,546) were older, predominantly female (65% vs. 60%), and exhibited significantly worse cardiometabolic profiles: higher systolic BP (143 vs. 133 mmHg), diastolic BP (86 vs. 81 mmHg), BMI (27 vs. 23 kg/m2), waist circumference (92 vs. 80 cm), triglycerides (1.66 vs. 1.41 mmol/L), and fasting glucose (5.60 vs. 5.30 mmol/L), with higher hypertension (32% vs. 13%) and diabetes prevalence (8.4% vs. 3.9%) (all P<0.001). CVD high-risk prevalence was 26% in the high-BRI group versus 17% in the low-BRI group (P<0.001). In logistic regression, BRI was independently associated with CVD high risk: the fully adjusted (Model 3) OR per 1-unit BRI increase was 1.078 (95% CI: 1.025-1.134, P=0.004); high BRI (>=4) conferred 24.7% higher odds (OR: 1.247, 95% CI: 1.093-1.423, P=0.001); and Q4 versus Q1 showed OR of 1.291 (95% CI: 1.072-1.555, P=0.007). RCS analysis revealed a significant nonlinear dose-response relationship (P overall <0.001; P for nonlinearity = 0.004), with a steeper risk gradient above BRI ~4. BRI alone showed modest discrimination (AUC: 0.590, 95% CI: 0.572-0.607), improving to 0.738 (95% CI: 0.721-0.753) in Model 3. In supplementary analyses, BRI demonstrated the largest incremental gain over the Model 2 base (delta-AUC 0.041, 95% CI: 0.028-0.055; continuous NRI 0.260, 95% CI: 0.203-0.317), outperforming BMI and waist circumference. Subgroup analyses revealed significant effect modification by sex (P for interaction = 0.001; stronger in men), diastolic BP quartiles (P<0.001; attenuated in Q4), and triglyceride quartiles (P<0.001; attenuated in Q4), with sex and cardiometabolic interactions remaining significant after FDR correction. In the ML pipeline, LASSO selected four predictors (diastolic BP, age, BRI, triglycerides); random forest achieved AUC 0.784; and SHAP ranked BRI 3rd of 4 selected features by mean absolute SHAP value.

Conclusion： Higher BRI was consistently and nonlinearly associated with WHO-defined CVD high-risk status across conventional regression, spline modeling, and explainable machine learning frameworks. The nonlinear dose-response pattern--with a steeper risk gradient above BRI ~4--suggests that the adverse association of central adiposity with cardiovascular risk classification intensifies at higher body roundness levels. BRI demonstrated the greatest incremental discriminative value among anthropometric indices in simpler screening models, though its added value in more clinically comprehensive models was modest. Significant effect modification by sex and cardiometabolic strata indicates that BRI-based screening strategies may benefit from sex-specific and risk-factor-specific interpretation. As a simple, equipment-free anthropometric index, BRI may serve as a pragmatic, low-cost adjunct to prioritize individuals for formal CVD risk assessment in community screening settings, particularly where laboratory and clinical resources are constrained.

Key words： Body roundness index; cardiovascular risk; community screening; logistic regression; machine learning; obesity phenotype; SHAP; WHO CVD risk charts; restricted cubic spline

论文 ID：339

Diagnostic Efficacy of ^{18}F-Pentixather-PET/CT for Subtyping and Localization of Primary Aldosteronism: Based on Adrenal Venous Sampling and CYP11B2 Immunohistochemical Staining

Yang Yu[1,2], Wei Song*[1]

1. The First Affiliated Hospital of Dalian Medical University

2. Tne First Affiliated Hospital of Dalian Medical University

Corresponding Author E-mail: songwei8124@163.com

Background： Primary aldosteronism (PA) is a common cause of secondary hypertension. Diagnostic classification and localization are key steps in the management of PA, as they determine the appropriate therapeutic approach and influence the patient's prognosis. Positron emission tomography/computed tomography (PET/CT) targeting C-X-C chemokine receptor 4 (CXCR4) has shown value in the diagnostic classification and localization of PA; however, the radionuclides used and the cut-off values established vary between centers. The purpose of this study is to evaluate the value of ^{18}F-Pentixather-PET/CT in distinguishing patients with unilateral PA (UPA) and in identifying classic pathological manifestations (aldosterone-producing adenoma and aldosterone-producing nodule, APA/APN), using adrenal venous sampling (AVS) and CYP11B2 immunohistochemistry as reference standards, respectively.

Methods： The study included 74 patients with a diagnosis of PA who underwent both ^{18}F-Pentixather-PET/CT and AVS, and 45 patients with a diagnosis of PA or suspected PA who underwent both ^{18}F-Pentixather-PET/CT and adrenalectomy. All surgical patients underwent CYP11B2 immunohistochemical staining and postoperative follow-up. Image analysis used standardized uptake value (SUVmax), the lateralization index of SUVmax (LI-SUVmax) and lesion-to-liver ratio (LLR). Analysis of staining results according to the consensus on the histopathology of PA.

Results： Of the 74 patients, 51 had UPA and 23 had bilateral PA (BPA) determined by AVS. SUVmax was positively correlated with the ipsilateral suppression index (defined as [aldosterone-cortisol ratio of adrenal vein] / [aldosterone-cortisol ratio of inferior vena cava]). When the optimal cut-off value of LI-SUVmax = 1.45, the sensitivity and specificity of ^{18}F-Pentixather-PET/CT for distinguishing between UPA and BPA were 66.7% and 91.3%, respectively (AUC = 0.824, 95%CI 0.728-0.919). The consistency rate between ^{18}F-Pentixather-PET/CT and AVS was 70.3% (Kappa = 0.558, $P < 0.001$), which was superior to that between computed tomography (CT) and AVS (47.3%, Kappa = 0.230, $P = 0.003$). Among patients with unilateral or bilateral adenomas on CT, the consistency rate between ^{18}F-Pentixather-PET/CT and AVS increased further to 75.0%. Based on CYP11B2 immunohistochemical staining and postoperative follow-up results, a total of 60 adrenal lesions were identified in 45 patients. Of these, 40 were classic PA lesions (APA/APN), and 20 were non-APA/APN lesions. When the optimal cut-off value of SUVmax = 6.40, ^{18}F-Pentixather-PET/CT had a sensitivity of 87.5% and a specificity of 70.0% for distinguishing between APA/APN and non-APA/APN lesions (AUC = 0.864, 95%CI 0.762-0.967). When the optimal cut-off value of LLR = 5.04, ^{18}F-Pentixather-PET/CT had a sensitivity of 77.5% and a specificity of 90.0% for distinguishing between APA/APN lesions

and non-APA/APN lesions (AUC = 0.894, 95%CI 0.810-0.979). The consistency rate between ^{18}F-Pentixather-PET/CT and CYP11B2 was 82.7% (Kappa = 0.620, P < 0.001).

Conclusion： ^{18}F-Pentixather-PET/CT targeting CXCR4 can provide an effective supplementary approach to AVS. When AVS cannot be performed or fails to provide an accurate classification, ^{18}F-Pentixather-PET/CT is a valuable alternative diagnostic tool for classification and localization of PA, which contributes to guiding personalized treatment decisions.

Key words： primary aldosteronism, 18F-Pentixather-PET/CT, adrenal venous sampling, CYP11B2, subtyping

论文 ID：351

Effects of renal denervation on glycemic outcomes in patients with hypertension: a systematic review and meta-analysis

Shuyi Wu, Tianjiao Lv, Rongxue Xiao, Zheyi Ni, Fangyu Zhou, Chuang Guo, Zongjun Liu, Junqing Gao*

上海市普陀区中心医院

Corresponding Author E-mail: kevingjq@sina.com

Background： The glycemic effects of renal denervation (RDN) remain uncertain.

Methods： Three databases were searched through January 2026, prioritizing controlled comparisons.

Results： Twenty-four studies (2,565 participants) were included. Versus control, RDN reduced SBP and DBP (MD, -8.43 and -3.38 mmHg), but not fasting glucose (MD, -0.30 mmol/L; 95% CI, -0.73 to 0.14), HbA1c, fasting insulin, or HOMA-IR. Exploratory analyses suggested larger fasting-glucose reductions with greater baseline metabolic burden.

Conclusion： RDN lowered blood pressure, but controlled evidence does not support a universal glucose-lowering effect. Any metabolic benefit may be selective and more apparent in higher-burden populations, while lower-burden populations appear to have a neutral response.

Key words： renal denervation; hypertension; glycemic outcomes; meta-analysis

论文 ID：353

Remnant Cholesterol Inflammatory Index, its 18-year Transition and Subclinical Target Organ Damage in Midlife: Insights from Hanzhong Adolescent Hypertension Cohort Study

Ziyue Man, Mu Jianjun*, Hu Guilin, Zhang Teng, Chang Mingke

First Affiliated Hospital of Xi'an Jiaotong University,

Corresponding Author E-mail: mujjun@163.com

Background： The novel remnant cholesterol inflammation index (RCII) integrates remnant cholesterol (RC) and high-sensitivity C-reactive protein (hs-CRP) to capture the synergistic burden of dyslipidemia and chronic inflammation. However, the impact of RCII on cardiovascular subclinical target organ damage (TOD) remains undefined.

Methods： The study population comprised 2061 participants from the 2023 survey of the Hanzhong Adolescent Hypertension Cohort, with a longitudinal sub-cohort of 160 individuals tracked from 2005 to 2023. Participants were categorized into four groups based on the quartile transition of RCII from early adulthood to midlife: control, incident, resolution and persistent. RCII was calculated as RC [mg/dL] × hs-CRP [mg/L] / 10. Subclinical TOD includes arterial stiffness (AS), left ventricular diastolic dysfunction (LVDD) and albuminuria.

Results： In the cross-sectional analysis, participants with the highest RCII quartile (Q4) vs. the lowest (Q1, reference) had elevated subclinical TOD risks: AS (odds ratio [OR] = 2.30, 95% confidence interval [CI]: 1.67 - 3.20), albuminuria (OR = 1.59, 95% CI: 1.06 - 2.41) and LVDD (OR = 1.68, 95% CI: 1.18 - 2.41) in fully adjusted models. Longitudinal analysis revealed heterogeneous associations between transition of RCII patterns and incident subclinical TOD. Compared with the control group, the incident and resolution groups were associated with a high risk of incident albuminuria. However, no significant associations with AS or LVDD were observed after full adjustment.

Conclusion： RCII was consistently associated with subclinical TOD in cross-sectional analyses, while longitudinal transition of RCII patterns exhibited heterogeneous associations with incident organ damage.

Key words： Remnant cholesterol inflammatory index; Subclinical organ damage; Arterial stiffness; Left ventricular diastolic dysfunction; Albuminuria.

论文 ID：363

Environmental Exposures and Risk of Incident Heart Failure in Patients With Atrial Fibrillation

Wenjie Yang, Jie Wang, Yalan Li, Chenxi Ouyang, Yanqi Zhu, Hong Yuan, Yao Lu*

the third Xiangya hospital of Central South University

Corresponding Author E-mail: luyao0719@163.com

Background： Atrial fibrillation (AF) is closely linked to the development of heart failure (HF). Emerging evidence from environmental cardiology suggest that environmental exposures constitute critical in cardiovascular disease progression beyond traditional factors. Nevertheless, whether environmental exposures contribute to HF development among patients with established AF and the underlying biological mechanisms remain unclear.

Methods： In this study, we included AF patients from UK Biobank from 2006 to 2010. The exposure include air pollution, noise, drinking water mineral and physicochemical indicators, and green space. Outcome was defined the incidence of heart failure or death caused by heart failure. To identify the important environmental exposures, Cox proportional hazards models were performed to examine the associations between each exposure and incident HF with false discovery rate correction for multiple testing. Exposures with an adjusted $P < 0.05$ and $HR > 1.05$ were retained for downstream analysis. In proteomics and metabolomics data, a two-step screening strategy was used to explore the potential mediations. Proteins and metabolites associated with core environmental exposures were first screened via

covariate-adjusted linear regression, then tested their associations with HF using covariate-adjusted Cox models. Overlapping proteins and metabolites were defined as potential key mediators linking environmental exposures to HF. Functional enrichment analyses were then conducted to uncover relevant biological pathways.

Results： A total of 5380 participants with AF included, with a median follow-up of 13.08 years. During follow-up, 1129 incident HF events occurred. Among 26 environmental exposures, we found higher exposure to PM2.5 (HR=1.08, 95% CI=1.02-1.14, adjusted P=0.043) and PM10 (HR=1.06, 95% CI=1.02-1.09, adjusted P=0.016) was significantly increase incident HF risk in AF patients. We identified 54 and 89 candidate mediating proteins for PM10 and PM2.5, respectively, alongside 34 overlapping candidate metabolites shared by both exposures. Enrichment analyses revealed that mediating metabolites were significantly enriched in pathways related to aromatic amino acid metabolism. Functional enrichment analysis of candidate mediator proteins further revealed that PM2.5 and PM10 were each characterized by a distinct core regulatory module: immune inflammatory signaling and extracellular matrix remodeling, respectively.

Conclusion： For patients with AF, PM2.5 and PM10 was associated with HF or death caused by HF. These findings support the integration of air pollution exposure assessment into cardiovascular risk management. Our findings suggest that PM-induced disruption of aromatic amino acid homeostasis may serve as an upstream metabolic driver of cardiac inflammation and ventricular remodeling, warranting further mechanistic investigation.

论文 ID：364

The gut microbiota-metabolite-muscle axis mediates muscle quality decline in obstructive sleep apnea: A Translational Mediation Analysis

Yanyan Deng, Dasheng Lu*

The Second Affiliated Hospital of Wannan Medical University

Corresponding Author E-mail: ldslds@126.com

Background： Sarcopenia is closely associated with obstructive sleep apnea (OSA), yet the link between OSA severity and erector spinae muscle (ESM) quality, as well as its underlying mechanisms involving gut microbiota and circulating metabolites, remains unclear in hypertensive patients. This study aimed to explore the association between OSA and ESM quality, and to investigate the potential mediating role of gut microbiota and plasma metabolites in this relationship.

Methods： A total of 44 hypertensive patients were enrolled, including 34 OSA patients and 10 controls. ESM parameters (fat infiltration rate, FIR; muscle density, mdensity) were quantified by chest CT. Gut microbiota was analyzed by 16S rRNA sequencing, and non-target metabolomic analysis was performed on fasting plasma. Correlation analyses, mediation analysis with Bootstrap validation, and dose-response analysis were conducted.

Results： OSA patients had higher FIR than controls (8.18 ± 4.49% vs. 4.47 ± 2.50%, P=0.0023). Three differential gut bacteria were identified: Roseburia (downregulated), Fusobacteria (downregulated), and Megamonas (upregulated). Correlation analysis revealed significant associations between differential bacteria, metabolites, and muscle parameters. Mediation analysis identified three significant indirect pathways: Roseburia → M733T273 → mdensity (Bootstrap 95% CI: 15.18-107.48, P=0.006), Roseburia → M709T276 → mdensity (95% CI: 8.58-84.91,

P=0.006), and Roseburia → M568T279 → mdensity (95% CI: 6.13-90.16, P=0.011). Dose-response analysis showed that FIR increased significantly with AHI severity (P_trend=0.014).

Conclusion：OSA is associated with ESM fat infiltration and gut microbiota dysbiosis in hypertensive patients. The gut microbiota-metabolite-muscle axis may play an important role in OSA-related muscle quality impairment, providing novel insights into the mechanisms and potential intervention targets for OSA-related sarcopenia.

Key words：Obstructive sleep apnea, Sarcopenia, Gut microbiota, Metabolomics, Mediation analysis, CT scan

论文 ID：372

Office and Ambulatory Blood Pressure Control and Antihypertensive Treatment Patterns in a Multicentre Real-World Hypertension Cohort in China

Zhanyang Zhou, Zheng Yin, Xue Li, Yunxi Han, Huanhuan Miao, Yuqing Zhang*

Beijing Fuwai hospital

Corresponding Author E-mail: yqzhang9988@163.com

Background：Suboptimal blood pressure (BP) control remains a major challenge in hypertension management. We aimed to describe office BP burden, antihypertensive treatment patterns, ambulatory BP control, and concordance between office and ambulatory BP control in a large multicentre real-world hypertension cohort in China.

Methods：This cross-sectional study analysed baseline data from a prospective, multicentre, real-world hypertension cohort. Controlled office BP was defined as systolic BP <140 mmHg and diastolic BP <90 mmHg. Grade 1, 2, and 3 hypertension were defined as systolic BP of 140–159, 160–179, and ≥180 mmHg, respectively, or diastolic BP of 90–99, 100–109, and ≥110 mmHg, with the higher category used when systolic and diastolic grades differed. Controlled 24-hour, daytime, and nighttime ambulatory BP were defined as <130/80, <135/85, and <120/70 mmHg, respectively.

Results：Among 89950 participants, 86999 (96.7%) had complete office BP measurements. Overall, 27575 (31.7%) had controlled office BP and 59424 (68.3%) had uncontrolled office BP. Grade 1, 2, and 3 hypertension accounted for 31.4%, 21.4%, and 15.4% of participants, respectively. Among 31380 participants with available medication-class information, 67.4% received one, 26.6% received two, and 5.6% received three or more antihypertensive medication classes. Calcium channel blockers (63.8%) and angiotensin receptor blockers (40.0%) were the most frequently recorded classes, whereas diuretics and mineralocorticoid receptor antagonists were recorded in 6.2% and 0.7%, respectively. Among 13487 participants with complete 24-hour ABPM data, 24-hour, daytime, and nighttime BP control rates were 35.6%, 43.1%, and 24.3%, respectively. Among 13423 participants with both office and ambulatory BP data, 53.1% had uncontrolled BP by both methods, 21.1% had uncontrolled office but controlled ambulatory BP, and 11.3% had controlled office but uncontrolled ambulatory BP.

Conclusion：This multicentre real-world cohort was characterized by a high burden of uncontrolled office BP, predominantly low-to-moderate treatment intensity among participants with recorded medication data, and particularly poor nighttime BP control. Approximately one-third of participants had discordant office and ambulatory BP control

status, highlighting the complementary role of ABPM in identifying clinically relevant 24-hour and nighttime BP burden.

Key words： Ambulatory blood pressure monitoring ; Blood pressure control; Real-world research

论文 ID：375

Correlation of the CHG Index and Its Derivative indicators with Short-Term Prognosis in Female Patients with HFpEF Complicated by Hypertension

yanwen Gao, Jing Yu*

The Second Hospital of Lanzhou University

Corresponding Author E-mail: ery_jyu@lzu.edu.cn

Background： Background: Insulin resistance (IR) is a component of metabolic syndrome and is closely associated with the occurrence and progression of cardiovascular diseases. In the general adult population, the prevalence of IR ranges from approximately 15% to 46%; this prevalence is even higher in patients with heart failure, particularly within the HFpEF subgroup. Among HFpEF patients, the incidence rate is higher in females than in males, and hypertension is the most common and significant risk factor for heart failure.The cholesterol, high-density lipoprotein, and glucose (CHG) index is a novel composite biomarker, and its association with the prognosis of HFpEF patients remains unclear. This study aims to investigate the correlation between the CHG index and its derived indicator (CHG-BMI) and the short-term prognosis of female HFpEF patients with comorbid hypertension, and to compare these parameters with traditional surrogate markers for IR.

Methods： This retrospective study enrolled 301 female patients diagnosed with HFpEF complicated by hypertension at the Second Hospital of Lanzhou University between January 2020 and December 2024. Patients were divided into a major adverse cardiovascular event (MACE) group (n = 105) and a non-MACE group (n = 196) based on whether MACE occurred during the 12-month follow-up. The CHG index, TyG index, TG/HDL-C ratio, METS-IR, and CHG-BMI were categorized into low, medium, and high groups according to tertiles. The endpoint event was MACE, which included all-cause mortality, rehospitalization for worsening heart failure, non-fatal myocardial infarction, non-fatal stroke, and malignant arrhythmias. Survival curves were plotted using the Kaplan–Meier method and the Log-rank test; the Cox proportional hazards regression model was employed to analyze the correlations between each indicator and the risk of MACE; and receiver operating characteristic (ROC) curves were constructed to evaluate the predictive value of these indicators.

Results： During the follow-up period, 105 patients experienced MACE (34.9%). Compared with the non-MACE group, patients in the MACE group exhibited significantly higher levels of serum creatinine, left atrial anterior-posterior diameter, glucose, triglycerides, prevalence of coronary heart disease and diabetes mellitus, SGLT2 inhibitor usage rate, CHG, CHG-BMI, TyG, TG/HDL-C, and METS-IR, as well as a significantly lower eGFR (all $P < 0.05$). Kaplan–Meier analysis demonstrated that the incidence of MACE was higher in both the high-CHG group and the high-TyG group (Q3) compared with the low-level groups (Q1) (all $P < 0.001$), with the greatest intergroup difference observed for the CHG index ($\chi^2 = 25.879$); however, no statistically significant differences were found among groups for the TG/HDL-C ratio, METS-IR, or CHG-BMI (all $P > 0.05$). Multivariate Cox regression analysis revealed that, using the low-CHG

group as the reference, both the medium-and high-CHG groups showed independent associations with MACE (HR = 2.890,95% CI: 1.613–5.176; HR = 2.935,95% CI: 1.603–5.374; all P <0.001), whereas only the high levels of TyG, METS-IR, and CHG-BMI demonstrated significant associations with MACE; the TG/HDL-C ratio did not exhibit significant prognostic stratification capability. The ROC curve analysis demonstrated that the univariate AUC for the CHG index was 0.698 (95% CI: 0.638–0.759); the adjusted ROC curve showed that the AUC of the baseline clinical model was 0.679, which improved to 0.729 upon inclusion of the CHG index—both values outperforming those of traditional IR surrogate markers. The derived indicator CHG-BMI did not exhibit superior prognostic discrimination efficacy compared to the CHG index.

Conclusion：An elevated CHG index is an independent risk factor for short-term MACE in female patients with HFpEF and hypertension, and this effect is superior to that of traditional IR surrogate markers.

Key words：HFpEF; hypertension; female; Cholesterol, high-density lipoprotein, and glucose index; Insulin resistance; prognosis

论文 ID：376

Fat–Muscle Imbalance, Visceral Adiposity, and Electrocardiographic ST–T Abnormalities in a Chinese Community Population: A Sex-Specific Cross-Sectional Study

Teng Zhang[1,2], Jianjun Mu*[1]

1. The First Affiliated Hospital of Xi'an Jiaotong University

2. The First Affiliated Hospital of Xi'an Jiaotong University

Corresponding Author E-mail: mujjun@163.com

Background：Body mass index (BMI) does not distinguish adiposity from skeletal muscle or capture fat distribution. This limitation is relevant to cardiovascular prevention in Asian populations, in whom cardiometabolic risk may emerge at lower BMI and scalable body-composition assessment may complement community health examinations. We examined whether visceral adiposity and fat–muscle imbalance, assessed by bioelectrical impedance analysis (BIA), were associated with electrocardiographic ST–T abnormalities and whether associations differed by sex.

Methods：This cross-sectional analysis included 8,805 adults with an interpretable resting 12-lead electrocardiogram. Body composition was assessed by BIA. Fat–muscle imbalance was summarised by the fat-to-muscle ratio (FMR) and by joint body-fat/muscle phenotypes. ST–T abnormalities were coded using the Minnesota Code. Multivariable logistic regression was supplemented by mutual-adjustment, quartile, dose–response, additive-interaction, sex-stratified, metabolic-attenuation, and sensitivity analyses.

Results：ST–T abnormalities were present in 1,159 participants (13.2%). Per 1-SD increment, visceral fat grade (adjusted odds ratio [OR] 1.20, 95% confidence interval [CI] 1.13–1.28), BMI (OR 1.15, 95% CI 1.08–1.23), and FMR (OR 1.27, 95% CI 1.19–1.37) were associated with ST–T abnormalities. After mutual adjustment, visceral fat grade remained associated (OR 1.22, 95% CI 1.08–1.39), whereas BMI did not (OR 0.97, 95% CI 0.86–1.10). Excess odds for FMR were concentrated in the highest quartile (OR 1.49, 95% CI 1.22–1.82). The high-fat/low-muscle phenotype

carried the highest odds (OR 1.47, 95% CI 1.23–1.77), without significant additive interaction. Associations were stronger in women (FMR per 1-SD increment: OR 1.33 in women vs 1.17 in men; P for interaction=0.037).

Conclusion： Fat–muscle imbalance and visceral adiposity were associated with electrocardiographic ST–T abnormalities beyond BMI, particularly among women. These findings support further evaluation of accessible body-composition phenotyping in community cardiovascular prevention, especially in Asian populations, but do not establish causality or individual predictive utility.

Key words： Body composition; visceral adiposity; fat-to-muscle ratio; electrocardiography; cardiovascular prevention; sex differences

论文 ID：377

Uric Acid-to-HDL Cholesterol Ratio-Based Composite Indices and Incident Metabolic Multimorbidity: A Six-Year Prospective Cohort Study

Teng Zhang, Jianjun Mu*

The First Affiliated Hospital of Xi'an Jiaotong University

Corresponding Author E-mail: mujjun@163.com

Background： Metabolic multimorbidity, defined as the co-occurrence of two or more metabolic disorders, is an increasing public health challenge. The uric acid-to-high-density lipoprotein cholesterol ratio (UHR) reflects the balance between urate-related metabolic stress and HDL-related protection. However, whether UHR-based composite indices are associated with the development of metabolic multimorbidity remains unclear.

Methods： We conducted a six-year prospective analysis using data from the Hanzhong Adolescent Hypertension Cohort. A total of 2,359 adults free of metabolic multimorbidity in 2017 were followed until 2023. Metabolic multimorbidity was defined as the presence of two or more of hypertension, diabetes, dyslipidaemia, hepatic steatosis, and kidney damage. UHR was calculated as serum uric acid divided by HDL cholesterol. Seven adiposity-weighted UHR composite indices were constructed by multiplying UHR by body mass index, waist circumference, waist-to-height ratio, weight-adjusted waist index, body roundness index, a body shape index, and waist-to-hip ratio. Multivariable logistic regression, dose-response analyses, discrimination assessment, subgroup analyses, and sensitivity analyses were performed.

Results： During follow-up, 395 participants developed incident metabolic multimorbidity. In fully adjusted models, baseline UHR was associated with higher odds of incident metabolic multimorbidity, with an odds ratio of 1.37 per 1-SD increase. Adiposity-weighted UHR indices showed stronger associations, particularly UHR × body roundness index, which showed the strongest baseline association. Cumulative UHR-based indices demonstrated stronger associations than baseline indices, with cumulative UHR × body roundness index showing the largest effect estimate. Participants in the highest quartile of cumulative UHR × body roundness index had markedly higher odds of metabolic multimorbidity than those in the lowest quartile. Adding cumulative UHR × body roundness index to the base model improved discrimination, reclassification, and model fit. Associations were generally consistent across major subgroups and remained robust in sensitivity analyses.

Conclusion： UHR-based composite indices were independently associated with incident metabolic multimorbidity in this six-year prospective cohort. Among the evaluated indices, UHR × body roundness index showed the strongest and most consistent performance. These findings suggest that UHR-based adiposity-weighted indices may serve as accessible markers for identifying individuals at increased risk of metabolic disease accumulation.

Key words： uric acid-to-HDL cholesterol ratio; metabolic multimorbidity; body roundness index; cumulative exposure; prospective cohort; risk prediction.

论文 ID：388

Estradiol protects telomeres and delays the development of PCAD through regulating the SIRT1/eNOS pathway

Fei Li*, Liming Peng

XiangYa hospital of Central South University

Corresponding Author E-mail: lifeixn@126.com

Background： Premature coronary artery disease (PCAD) is prevailing. Telomere shortening and cell senescence are associated with PCAD. Here, effect of estradiol on telomere and cell senescence in PCAD was evaluated.

Methods： Serum samples were collected from PCAD patients. Endothelial progenitor cells (EPCs) were isolated from peripheral blood mononuclear cells (PBMCs). EPCs were treated with different concentrations of estradiol (E2). The mRNA level of TRF1, TRF2, POT1 and SIRT1 was determined by RT-qPCR. Telomere length of EPCs was determined by qPCR. Protein expression was detected by Western blot. Identification of EPCs was conducted by immunofluorescence staining. Serum levels of E2, VEGF and CK-MB were measured by Enzyme-linked immunosorbent assay (ELISA). SA-β-gal staining was used to detect EPCs senescence. Flow cytometry tested EPCs markers CD45 and CD34. CAD mouse model was established by feeding a high-heat diet for 8 weeks. HE staining evaluated myocardial injury. TUNEL staining detected apoptosis in myocardial tissue.

Results： In PCAD patients, there was a reduction in E2 levels, and its concentration exhibited a negative correlation with patients' severity. E2 increased telomere length and suppressed EPCs senescence in PCAD. SIRT1 expression decreased and showed a positive association with E2 levels. E2 increased eNOS activity by triggering SIRT1-mediated deacetylation. Inhibiting SIRT1 or eNOS reversed the impact of E2 on telomeres and EPCs senescence. E2 mitigated PCAD progression by regulating SIRT1/eNOS signaling in mice.

Conclusion： Estradiol safeguards telomeres and improves the progression of PCAD by modulating the SIRT1/eNOS, which may be a new reagent for PCAD treatment.

论文 ID：396

Childhood-to-Adulthood Overweight Status Transitions and Midlife Insulin Resistance: A 36-Year Prospective Cohort Study

Guilin Hu, Mingke Chang, Dan Wang, Ziyue Man, Jingruo Zhang, Xue Shi, Yu Yan, Tongshuai Guo, Wenling Zheng, Chao Chu, Teng Zhang, Mingfei Du, Yang Wang, Jianjun Mu*

First Affiliated Hospital of Xi'an Jiaotong University

Corresponding Author E-mail: mujjun@163.com

Background： Childhood overweight is associated with adverse metabolic consequences later in life; however, whether changes in overweight status from childhood to adulthood influence midlife insulin resistance remains unclear. We aimed to examine the associations between childhood-to-adulthood overweight transitions and midlife insulin resistance in a prospective cohort study.

Methods： This 36-year prospective cohort included 1,781 participants. Based on BMI status during childhood and adulthood, participants were categorized into four groups: control (normal both), incident (normal childhood to overweight adulthood), resolution (overweight childhood to normal adulthood), and persistent (overweight both). Childhood overweight was defined as BMI z-score >1, whereas adult overweight was defined as BMI ≥24 kg/m^2 according to Chinese criteria. Insulin resistance (IR) was assessed using the homeostasis model assessment of insulin resistance (HOMA-IR) and the triglyceride-glucose (TyG) index. Elevated HOMA-IR and TyG index were defined as values at or above the 75th percentile of their respective distributions in the study population. In addition, combined IR was defined as the presence of either elevated HOMA-IR or elevated TyG index in sensitivity analysis. Multivariable regression models adjusted for demographic characteristics, lifestyle factors, and cardiometabolic factors.

Results： Compared with the congrol group, the incident and persistent groups were associated with significantly higher odds of insulin resistance across all definitions (all P < 0.001). ORs for HOMA-IR-defined IR were 4.29 (95% confidence interval [CI] 2.98–6.30) for incident and 3.48 (95% CI 2.32–5.30) for persistent. ORs for TyG-defined IR were 2.97 (95% CI 2.12–4.22) and 2.54 (95% CI 1.74–3.75). For the combined definition (either index above the 75th percentile), ORs were 3.53 (95% CI 2.62–4.79) and 2.75 (95% CI 1.97–3.88). Participants in resolution group had odds comparable to the reference group. Continuous analyses demonstrated graded associations between BMI changes and insulin resistance. Subgroup analysis showed pronounced associations in non-diabetic participants but not in those with diabetes. Sensitivity analyses using alternative cutoffs were robust.

Conclusion： Adult overweight, whether new-onset or persistent from childhood, is associated with increased risk of midlife IR, normalization of overweight status by adulthood may attenuate the elevated risk associated with childhood overweight, highlighting the importance of early weight management.

Key words： Life-course epidemiology; overweight status transition; insulin resistance; HOMA-IR; triglyceride-glucose index.

论文 ID：397

Osteoglycin Protects Against Abdominal Aortic Aneurysm by Antagonizing IGF1R-Mediated VSMC Ferroptosis

Mengyao Li*, Xiaoxi Pan, Qirong Zhang, Yuan Liu, Jiguang Wang, Xiaodong Li, Pingjin Gao

Department of Cardiovascular Medicine, Department of Hypertension, State Key Laboratory of Medical Genomics, Shanghai Key Laboratory of Hypertension, Shanghai Institute of Hypertension, Ruijin Hospital, Shanghai Jiao Tong University School of Medicine, China

Corresponding Author E-mail: lmyb5108@163.com

Background： Abdominal aortic aneurysm (AAA) is a life-threatening condition lacking effective pharmacotherapy. Osteoglycin (OGN), a small leucine-rich proteoglycan, with known cardioprotective properties, but its role in the pathogenesis of AAA remains unclear.

Methods： Single-cell RNA sequencing combined with public RNA sequencing datasets identifies OGN as a top candidate for AAA.The role of OGN was investigated using VSMC-specific Ogn knockout and overexpression mice across multiple AAA models. Mass spectrometry, co-immunoprecipitation and proximity ligation assays were performed to identify the receptor that interacts with OGN. Gain- and loss-of-function experiments were performed to investigate the role of OGN in vascular smooth muscle cell (VSMC) ferroptosis.

Results： OGN expression was reduced in human AAA tissues and AngII-infused mouse abdominal aortas. Plasma OGN levels in patients were inversely correlated with maximum abdominal aortic diameter. VSMC-specific Ogn deficiency exacerbated AAA development, whereas Ogn overexpression ameliorated it. OGN protected against VSMC ferroptosis and associated mitochondrial dysfunction. Mechanistically, OGN directly and competitively bound insulin-like growth factor 1 receptor (IGF1R), reducing IGF1 engagement and attenuating Akt signaling, thereby promoting AMPK activation and GPX4 expression. Pharmacological inhibition of IGF1R attenuated AAA progression in Ogn-deficient mice. Chromatin immunoprecipitation results revealed that interferon regulatory factor 1 (IRF1) repressed OGN transcription by recruiting HDAC1 to the OGN promoter and reducing H3K27 acetylation. Elevated IRF1 levels accordingly correlated with reduced OGN expression in human AAA samples.

Conclusion： Our study demonstrates that OGN serves as an endogenous suppressor of IGF1R signaling, thereby protecting VSMCs from ferroptosis in AAA. OGN may be a new therapeutic target for the prevention and treatment of AAA.

Key words： abdominal aortic aneurysm; angiotensin II; ferroptosis; mitochondria; vascular smooth muscle cell.

论文 ID：405

Cumulative Exposure and Dynamic Changes of C-Reactive Protein-Triglyceride-Glucose Index in Relation to Subclinical Cardiovascular and Renal Damage in Middle-Aged Adults: A Prospective Cohort Study

Guilin Hu, Ziyue Man, Mingke Chang, Tongshuai Guo, Dan Wang, Wenling Zheng, Yu Yan, Chao Chu, Xue Shi, Mingfei Du, Jianjun Mu*

First Affiliated Hospital of Xi'an Jiaotong University

Corresponding Author E-mail: mujjun@163.com

Background： Metabolic dysfunction and chronic low-grade inflammation are key contributors to cardiovascular-kidney-metabolic (CKM) syndrome. The C-reactive protein–triglyceride–glucose index (CTI), integrating insulin resistance and systemic inflammation, has emerged as a potential marker of metabolic-inflammatory burden. However, whether cumulative exposure and longitudinal changes in CTI are associated with subclinical cardiovascular and renal damage remains unclear. This study aimed to investigate the associations of CTI cumulative burden and dynamic trajectories with subclinical cardiovascular-kidney damage in middle-aged adults.

Methods： Using data from the Hanzhong Adolescent Hypertension Study, we conducted a 6-year prospective cohort study involving 1,906 participants. CTI was calculated based on the triglyceride-glucose (TyG) index and high-sensitivity C-reactive protein (hs-CRP). Long-term metabolic-inflammatory burden was assessed using cumulative CTI exposure (cumCTI), whereas short-term changes were evaluated by ΔCTI. K-means clustering was applied to identify distinct longitudinal CTI trajectories. Multivariable logistic regression models and restricted cubic spline analyses were performed to evaluate associations between CTI-related indicators and subclinical cardiovascular and renal damage. Receiver operating characteristic (ROC) curves were used to assess predictive performance.

Results： A total of 1906 participants were included in this study. Among them, 520 individuals (27.3%) had arterial stiffness (AS), 426 individuals (22.4%) had left ventricular diastolic dysfunction (LVDD), and 426 individuals (22.4%) had albuminuria. As cumCTI levels increased, the odds of subclinical cardiovascular-renal damage also increased. Participants in the highest quartile (Q4) exhibited significantly higher odds of AS (OR 2.96, 95% CI 2.06 - 4.29), LVDD (OR 1.90, 95% CI 1.33 - 2.73), and albuminuria (OR 2.18, 95% CI 1.42 - 3.40) compared with those in the lowest quartile (Q1). The K-means clustering analysis identified three distinct change trajectories of CTI. Persistent high-risk group were significantly associated with an increased odds of albuminuria (OR 2.95 [1.96–4.44]), as well as with AS (OR 2.84 [2.03-3.98]) and LVDD (OR 1.90 [1.33–2.73]). In contrast, ΔCTI did not show an independent predictive effect on the risk of albuminuria.

Conclusion： Our study shows that cumCTI is significantly associated with dynamic changes of CTI and with an increased risk of subclinical cardiovascular-renal damage in middle age. In contrast, short-term changes in CTI (ΔCTI) did not show independent predictive value in albuminuria.

Key words： C-reactive protein-triglycerides-to-glucose index, Subclinical organ damage, Dynamic change, Cumulative exposure, K-means clustering

论文 ID：406

long-term BMI burden, its variability from childhood to adulthood and Cardiovascular-Kidney-Metabolic syndrome in midlife: a 36-year cohort study

Guilin Hu, Tongshuai Guo, Dan Wang, Ziyue Man, Jingruo Zhang, Mingke Chang, Shenghao Zuo, Chao Chu, Yu Yan, Jianjun Mu*

First Affiliated Hospital of Xi'an Jiaotong University

Corresponding Author E-mail: mujjun@163.com

Background： Limited data exist on the longitudinal associations of body mass index (BMI) burden and variability from childhood to adulthood with the development and severity of Cardiovascular-Kidney-Metabolic (CKM) syndrome in midlife. This study assessed the relationship between long-term BMI burden, visit-to-visit BMI variability, and midlife CKM stages and its components.

Methods： Utilizing data from the Hanzhong Adolescent Hypertension Study (initiated 1987; follow-up 2023), we classified 1948 participants into CKM stages 0-4. For analysis, stages were grouped as: Early Stage (0 & 1), Intermediate Stage 2, and Advanced Stage (3 & 4). Mean BMI and cumulative BMI burden (calculated as the area under the BMI-time curve [AUC-BMI] using trapezoidal integration) were derived from BMI measurements collected in 1987, 1989, 1992, 1995, 2005, 2013, 2017, and 2023. BMI variability was quantified using standard deviation (SD), coefficient of variation (CV), average real variability (ARV), and variability independent of the mean (VIM). Associations with CKM outcomes were evaluated using multivariable logistic regression.

Results： For Intermediate Stage CKM, adjusted odds ratios (95% CI) per 1-SD increase were: mean BMI: 1.24 (1.16-1.33); cumulative BMI burden: 1.68 (1.45-1.95); BMI SD: 1.55 (1.39-1.74); BMI CV: 1.08 (1.06-1.11); BMI ARV: 1.80 (1.48-2.22); BMI VIM: 1.40 (1.26-1.55). For Advanced Stage CKM, corresponding ORs were: mean BMI: 1.42 (1.31-1.55); cumulative BMI burden: 2.18 (1.82-2.63); BMI SD: 1.57 (1.38-1.78); BMI CV: 1.07 (1.05-1.10); BMI ARV: 1.90 (1.52-2.40); BMI VIM: 1.33 (1.18-1.51). Associations for all variability indices (SD, CV, ARV, VIM) with CKM stages remained significant after adjustment for mean BMI and cumulative burden. Higher cumulative burden and variability were also associated with increased risks of left ventricular diastolic dysfunction (LVDD), subclinical renal damage, (SRD) albuminuria, and metabolic abnormalities.

Conclusion： Elevated long-term cumulative BMI burden and greater visit-to-visit BMI variability from childhood to midlife are associated with increased severity of CKM syndrome and its key components by midlife.

Key words： Cardiovascular-Kidney-Metabolic syndrome; Body mass index; Cumulative burden; BMI variability; Childhood; Midlife

论文 ID：410

A 7-Year Comparative Study of Personalized versus Conventional Blood Pressure Assessment: An Analysis Based on 1005 Cases

Lianyuan Feng*[1,2], Yong Feng[1], Xiaohua Wen[1], Wenxiu Liu[1]

1. Bethun international peace hosptal

2. Dept of neurology ,Bethun international peace hosptal

Corresponding Author E-mail: fengly2012@163.com

Background： Chinese hypertension guidelines recommend unified population BP cut-offs for diagnosis and stratification, which are suitable for population screening but ignore individual long-term baseline heterogeneity.

Identical BP readings carry distinct clinical meanings for patients with chronically low or elevated baseline BP. Borderline populations (high-normal BP, grade 1 hypertension) are prone to misjudgment, highlighting the necessity of individualized evaluation to improve clinical accuracy.

Methods： 1005 participants with consecutive 7-year physical examination BP records from 2012 to 2018 were recruited. Conventional assessment adopted guideline fixed thresholds; individualized BP values were calculated via PRIP correction formula. Statistical analyses including paired t-test, weighted Kappa test and annual variation analysis were performed with SPSS 26.0 to quantify differences in BP levels, classification consistency, long-term fluctuation and intervention modification rate.

Results： Corrected systolic and diastolic BP values were significantly lower than conventional measurements ($P < 0.001$), and gender BP gap was eliminated after individual correction. Overall grading consistency of two methods was less than 80%, with the highest disagreement in high-normal diastolic BP group. Conventional assessment presented larger annual BP swings, while individualized evaluation yielded milder long-term fluctuations free from single-measurement interference. More than 20% participants required revised intervention plans; individualized evaluation effectively identified masked hypertension and reduced unnecessary antihypertensive treatment among borderline patients.

Conclusion： Traditional population-referenced BP assessment has prominent individual bias affecting nearly one-fifth of patients. Individualized BP evaluation based on multi-year personal baseline minimizes measurement fluctuation interference and improves diagnostic precision for borderline hypertension, balancing missed diagnosis and overtreatment. Combined application of individualized BP assessment is recommended in primary chronic disease management and long-term follow-up populations to advance precise hypertension care.

Key words： Hypertension; individualized blood pressure assessment; borderline hypertension; annual blood pressure variability; clinical diagnosis and treatment

论文 ID：414

Online Prediction Model for Primary Aldosteronism in Chinese Hypertensive Patients: A Single-Center Clinical Study

Jinghan Hai, Yun Ti, Peili Bu*

State Key Laboratory for Innovation and Transformation of Luobing Theory; Key Laboratory of Cardiovascular Remodeling and Function Research of MOE, NHC, CAMS and Shandong Province; Department of Cardiology, Qilu Hospital of Shandong University, Jinan, China, 250012.

Corresponding Author E-mail: bupeili@outlook.com

Background： Primary aldosteronism (PA) is an endocrine syndrome in which the adrenal glands autonomously overproduce aldosterone, leading to renin suppression, sodium and water retention, and potassium excretion. It is characterized by hypertension with or without hypokalemia. As a common curable cause of secondary hypertension, PA is vastly underdiagnosed. Approximately 5%–10% of individuals with hypertension have PA, yet the actual diagnosis rate is below 1%. More than half of patients have normal blood potassium levels and are easily misdiagnosed with essential hypertension. Prolonged underdiagnosis significantly increases the risk of heart failure, stroke, and renal

failure. Raising screening awareness is urgently needed. This study aims to develop and validate a simple and effective online prediction model to improve the screening rate and diagnostic efficiency of PA in hypertensive patients.

Methods： This study is a single-center retrospective study that collected clinical characteristics and laboratory data of 1380 hypertensive patients from Qilu Hospital of Shandong University between January 2018 and January 2023. The patients were randomly divided into a training cohort (965 cases, 70%) and an internal validation cohort (415 cases, 30%), with an additional 259 patients serving as an external validation set. Clinical characteristics and laboratory results between the two cohorts were compared using the Wilcoxon test. Univariate COX regression analysis was used in the training cohort for feature selection to identify variables associated with PA. The association of variables was represented by HR values, and a p-value of < 0.05 was considered statistically significant. A backward multivariate COX regression analysis was conducted on these variables to identify independent risk factors for PA prediction ($p < 0.05$). The variance inflation factor (VIF) was used to assess multicollinearity among all predictive variables, and the model's performance was evaluated through sensitivity, specificity, and accuracy.

Results： Among the 1380 hypertensive patients, 314 were diagnosed with PA. Using machine learning algorithms, independent risk factors for predicting PA were identified, including age, sex, TyG index, BMI, FT3, TSH, NLR, serum sodium-to-potassium ratio, and hypokalemia. The prediction model's area under the curve (AUC) values in the training set, internal validation set, and external validation set were 0.85 (95% CI: 0.82–0.88), 0.846 (95% CI: 0.81–0.88), and 0.847 (95% CI: 0.765–0.928), respectively. The results demonstrated that the prediction model performed well in both the training and validation sets. The calibration curves showed good consistency between the predicted risk and the actual risk, further validating the model's reliability.

Conclusion： The online prediction model developed in this study uses routine clinical characteristics and laboratory data to conveniently and reliably predict the risk of PA in hypertensive patients. This model can significantly enhance the screening rate and diagnostic efficiency for PA.

Key words： Primary Aldosteronism；Prediction Model

论文 ID：421

Investigating the cut-off values for primary aldosteronism using the novel LC-MS method: a retrospective cohort study

Chen Chen, Dongsheng Sun, Li Zhang, Lin Wang, Shaokun Xu, Yi Cheng, Yanan Zhang, Ning Zhou, Jianhong Xie, Li Li, Huiling Huang, Dibo Lao, Liqun Tang, Qinyang Jin*

Zhejiang Provincial People's Hospital

Corresponding Author E-mail: jqy@163.com

Background： Liquid chromatography-mass spectrometry (LC-MS) provides a more accurate measurement of aldosterone. However, its method-specific cut-off values for the diagnosis of primary aldosteronism (PA) have not been validated. This study aims to establish aldosterone thresholds for PA diagnosis during the saline infusion test (SIT) and captopril challenge test (CCT) using LC-MS/MS.

Methods： We conducted a retrospective cohort study of 343 samples collected from 2018 to 2026, including 125 patients with primary aldosteronism and 218 patients with essential hypertension. The area under the curve (AUC) and calibration curves were used to evaluate predictive accuracy and discrimination. Decision curve analysis (DCA) was further employed to assess clinical utility.

Results： In this study, we established and validated specific cut-off values for the screening and confirmation of Primary Aldosteronism based on LC-MS/MS in a Chinese cohort. Our data suggests that an ARR cut-off of 11 (ng/dl)/[ng/(ml·h)] provides a high sensitivity of 90.4%, making it a robust screening tool. Furthermore, we determined optimized cut-off values for confirmatory testing: a post-saline infusion aldosterone level of 5 ng/dL (Sensitivity 67.9%, Specificity 90.1%) and a post-captopril challenge aldosterone level of 7.3 ng/dL (Sensitivity 65.4%, Specificity 91.7%). These findings highlight the necessity of method-specific reference intervals, as conventional immunoassay cut-offs may not be directly transferable to mass spectrometry-based workflows.

Conclusion： This study provides LC-MS/MS-specific aldosterone cut-offs (5 ng/dL for SIT and 7.3 ng/dL for CCT) for PA confirmation, which demonstrate acceptable diagnostic accuracy and clinical net benefit.

论文 ID：424

Uncoupling of Cardiac Remodeling and Micro-inflammation: A Hemodynamic Phenomapping Study

Rong Cao, Liangdi Xie*

The First Affiliated Hospital of Fujian Medical University

Corresponding Author E-mail: ldxield@163.com

Background： Background: Hypertension-mediated organ damage (HMOD) is traditionally viewed as a uniform consequence of pressure overload. However, this homogenous perspective fails to explain the marked clinical heterogeneity where cardiac remodeling and systemic inflammation often manifest independently. It remains unexplored whether distinct hemodynamic drivers—specifically steady-state resistance versus pulsatile stiffness—independently dictate these divergent pathological pathways. This study aimed to identify distinct hemodynamic phenotypes via machine-learning phenomapping and investigate their specific associations with cardiac and inflammatory damage.

Methods： Unsupervised K-means clustering was applied to six hemodynamic parameters—central mean arterial pressure (cMAP), carotid-femoral pulse wave velocity (cf-PWV), augmentation index (AIx), Max dP/dt, subendocardial viability ratio (SEVR), and heart rate—in 3,914 participants from the Fuzhou Study. Multivariable regression, propensity score matching, and causal mediation analyses were used to test the pathological uncoupling hypothesis. Micro-inflammation analysis included 3,010 individuals with available high-sensitivity C-reactive protein (hs-CRP) data (complete-case analysis).

Results： Three phenotypes were identified: Preserved Hemodynamics, High-Resistance, and High-Stiffness. We observed a striking pathological uncoupling: High-Resistance was independently associated with cardiac remodeling (elevated LVMI; $P < 0.001$) but not micro-inflammation ($P = 0.6$). Conversely, High-Stiffness was selectively associated with systemic micro-inflammation ($\beta = 1.7$, $P < 0.001$) without corresponding LVMI changes ($P = 0.8$). After 1:1 propensity score matching (892 pairs), this association remained significant ($P < 0.001$), confirming the

robustness of the hemodynamic-inflammatory coupling. Sensitivity analysis confirmed that age-related missingness did not bias these associations. Mediation analysis established that micro-inflammation is a terminal event of arterial stiffness rather than an intermediary pathway to cardiac remodeling (indirect effect P = 0.392).

Conclusion： s: Hemodynamic phenomapping reveals a distinct uncoupling of target organ damage. Resistance primarily dictates macroscopic cardiac remodeling, while stiffness selectively triggers microvascular inflammation. These findings support transitioning from "blood pressure-centric" to phenotype-guided precision cardiovascular prevention.

Key words： Keywords Machine learning; Phenomapping; Hemodynamics; Arterial stiffness; Hypertension-mediated organ damage; Micro-inflammation; Cardiac remodeling; Precision medicine.

论文 ID：434

Relationship between cumulative exposure of lactate dehydrogenase concentrations and major adverse cardiac and cerebrovascular events in hypertensive patients: a retrospective cohort study

Hui Wang[1,2,3,4], Nanfang Li*[1,2,3]

1. Center for Hypertension of The People's Hospital Of Xinjiang Uygur Autonomous Region China

2. NHC Key Laboratory of Hypertension Clinical Research

3. Hypertension Institute of Xinjiang

4. Institute for Post Graduate, Xinjiang Medical University

Corresponding Author E-mail: 2414616844@QQ.COM

Background： As of 2023, cardiovascular disease (CVD) affects 626 million people globally, imposing a heavy burden on both society and families. Despite advances in antihypertensive therapy, residual risk persists, underscoring the need for potential biomarkers to optimize risk stratification. Lactate dehydrogenase (LDH), traditionally a marker of tissue injury, may reflect metabolic or inflammatory stress associated with vascular damage. This study investigates the association between annual cumulative LDH exposure and incident major adverse cardiac and cerebrovascular events (MACCE) in hypertensive patients, providing insights for prevention strategies.

Methods： In this retrospective cohort study, we included hypertensive patients treated at a large tertiary hospital in Urumqi, China, from January 2011 to April 2025. Kaplan-Meier curves visualized survival differences, and Cox proportional hazards models assessed the association between annual LDH_AUC and MACCE risk. Stratified and sensitivity analyses evaluated robustness, while C-statistics compared the predictive performance of annual LDH_AUC versus single-timepoint LDH.

Results： During a median follow-up period of 47 months, a total of 879 new MACCE were recorded. The research results showed that whether the annual LDH_AUC was used as a continuous variable or a categorical variable, an increase in annual LDH_AUC would increase the risk of MACCE (HR=1.012, 95% CI, 1.007-1.017; HR=1.431, 95% CI, 1.185-1.727). Moreover, the predictive value of annual LDH_AUC for MACCE is superior to that of a single LDH measurement.

Conclusion： In hypertensive patients, elevated annual LDH_AUC levels is associated with an elevated risk MACCE.

Key words： hypertension, lactate dehydrogenase, major adverse cardiac and cerebrovascular events

论文 ID：439

Effects of group education on cigarette smoking: a feasibility study

Jiabo Zhu[1], Wenyuanyue Wang[2], Yuanyuan Kang[1], Jiguang Wang*[1]

1. The Shanghai Institute of Hypertension, National Research Centre for Translational Medicine at Shanghai, Ruijin Hospital, Shanghai Jiao Tong University School of Medicine

2. School of Public Health, Shanghai Jiao Tong University School of Medicine

Corresponding Author E-mail: kylezhu99@163.com

Background： We investigated the effects of group-based smoking cessation education on blood pressure reduction compared with routine smoking cessation management and whether changes in smoking behavior were associated with changes in blood pressure among older smokers with hypertension.

Methods： Method: The study participants were older male current smokers with diagnosed hypertension from 7 community health centers in Shanghai. Participants were cluster-randomized to either group-based smoking cessation education or routine smoking cessation management based on their affiliated community healthcare centers and followed up for 6 months. The primary outcome was the reduction of office blood pressure between baseline and follow-up. Secondary outcomes included changes in home blood pressure, smoking-related characteristics, and the associations between changes in cigarette consumption and blood pressure. Adjusted linear regression analyses were performed to identify factors associated with changes in blood pressure.

Results： Result: 90 participants with mean (±SD) age of 72.5 ± 3.7years completed follow-up were included. Both office and home systolic/diastolic blood pressure significantly decreased after the intervention (-9.0 ± 17.2/-4.3 ± 8.1 mmHg and -9.6 ± 13.2/-3.9 ± 6.2 mmHg, respectively, $P<0.001$). However, no significant differences in blood pressure reduction were observed between two groups ($P\geq0.18$). A greater proportion of participants in the education group achieved smoking cessation or reduced cigarette consumption compared with the routine management group (39.0% vs. 24.5%), although the difference was not statistically significant ($P = 0.44$). After adjustment for potential confounders, reductions in daily cigarette consumption were independently associated with improvements in morning home blood pressure ($P \leq 0.045$).

Conclusion： s: Both group-based smoking cessation education and routine smoking cessation management were associated with significant reductions in blood pressure among older male smokers with hypertension. Although group-based education did not provide additional blood pressure-lowering benefits compared with routine management, reductions in cigarette consumption were associated with improvements in morning home blood pressure, suggesting that smoking reduction may represent an important behavioral target for optimizing blood pressure control.

Key words： Smoking cessation; Group-based intervention; Hypertension; Blood pressure; Older adults; Community-based intervention

论文 ID：446

Age-Adapted Renal Function Assessment Predicts Mortality in Hypertension: A Prospective Cohort Study

Meng Ning, Yingwu Liu, Chenguang Zheng*

Tianjin Third Central Hospital

Corresponding Author E-mail: lemon2104@126.com

Background： Hypertension persists as the preeminent global risk factor for morbidity and mortality, emphasizing the pressing necessity for accurate risk stratification to direct therapeutic strategies and optimize resource allocation. While conventional risk factors like age and diabetes are firmly established, mounting evidence emphasizes the prognostic significance of subclinical organ damage, particularly renal impairment. This is true even when the decreases in estimated glomerular filtration rate (eGFR) are above the conventional chronic kidney disease (CKD) thresholds. Nevertheless, the implementation of uniform eGFR cut - offs across all age groups may conceal clinically significant risk variations, especially among older adults, owing to the physiological decline in renal function associated with aging. Age - adjusted eGFR thresholds have been put forward to more effectively detect early, asymptomatic renal dysfunction (referred to as subclinical impaired renal function, SIRF). However, their independent prognostic value in hypertension and their additional utility beyond traditional models are still insufficiently defined.

Methods： To investigate whether age - adapted assessment of renal function can predict long - term all - cause mortality in hypertensive patients and enhance risk discrimination when compared to traditional uniform estimated glomerular filtration rate (eGFR) thresholds. Design: A prospective cohort study. Setting: Secondary care; a single tertiary hospital in Tianjin, China. Participants: A total of 573 consecutively enrolled adults with confirmed hypertension were followed up for a median of 7.6 years. After excluding 31 (5.4%) patients who were lost to follow - up, 542 patients were included in the analysis. Inclusion criteria: hypertension (systolic blood pressure ≥140 mmHg and/or diastolic blood pressure ≥90 mmHg, or under treatment). Exclusion criteria: end - stage renal disease (eGFR <15), severe heart failure (New York Heart Association (NYHA) class IV), or life - limiting non - cardiovascular diseases. Primary outcome measure: All - cause mortality. Renal function was classified into age - adapted groups: normal renal function (NRF), subclinical impairment of renal function (SIRF), and impaired renal function (IRF) based on pre - specified age - decile eGFR thresholds.

Results： SIRF was identified as a robust and independent predictor of mortality (adjusted hazard ratios [HR] = 2.98, $p < 0.001$), while the conventionally defined impaired renal function was not. The NLPM demonstrated superior discriminative performance (AUC: 0.827 vs. 0.798, $p = 0.011$). In the subgroup analysis, NRF was identified as a protective factor against all - cause death in patients without a history of diabetes (HR = 0.528, 95% CI: 0.295 - 0.945). SIRF (HR = 1.827, 95% CI: 1.092 - 3.058) and IRF (HR = 3.690, 95% CI: 1.059 - 12.854) were determined to be independent risk factors for all - cause death in patients without a history of cerebrovascular disease. Among patients with a history of CHD, NRF was found to be a significant protective factor (HR = 0.573, 95% CI: 0.341 - 0.963), while IRF was recognized as an independent risk factor for all - cause death.

Conclusion： Age-adapted eGFR assessment for identifying SIRF serves as a robust and independent predictor of mortality in hypertension and significantly enhances risk stratification. Further multi-centre validation is necessary.

Key words： Age-adapted eGFR, hypertension, all - cause mortality, traditional linear prediction model (TLPM)

论文 ID：457

Association between average systolic blood pressure during the acute phase and mortality in very elderly (≥80 years) patients with acute myocardial infarction

Yuanyuan Fan*

Peking University Third Hospital

Corresponding Author E-mail: fanyy2014@163.com

Background： Optimal blood pressure (BP) targets during acute myocardial infarction (AMI) in patients aged ≥80 years remain undefined, as this population is systematically under-represented in cardiovascular trials. We aimed to assess the association between average acute-phase BP and short-and long-term mortality in this vulnerable cohort.

Methods： This ambidirectional cohort study consecutively enrolled patients aged ≥80 years admitted for AMI to Peking University Third Hospital between 2013 and 2023. Patients with cardiogenic shock or cardiac arrest at admission were excluded. Average in-hospital systolic BP (aSBP) and diastolic BP (aDBP) were derived from serial measurements during the first week of hospitalization. The primary outcome was 1-year all-cause mortality.

Results： Among 872 analyzed patients (mean age 84.2 years; 59.3% male), 208 (23.9%) died within 1 year. Restricted cubic spline analysis revealed an inverse J-shaped association between aSBP and mortality, with risk decreasing steeply up to approximately 120 mmHg and plateauing thereafter. Compared with aSBP <120 mmHg, aSBP ≥130 mmHg was independently associated with a 40% lower risk of 1-year mortality (OR, 0.60; 95% CI, 0.35–0.93; P=0.041), while the 120-130 mmHg group showed a non-significant trend toward lower risk (OR, 0.92; 95% CI, 0.63-1.62; P=0.598). A U-shaped relationship was observed between aDBP and mortality, with the lowest risk at 60-70 mmHg. Using aDBP <60 mmHg as the reference, patients with aDBP of 60-70 mmHg had a lower risk of 1-year mortality that did not reach statistical significance (OR, 0.61; 95% CI, 0.38-1.37; P=0.068).

Conclusion： In very elderly AMI patients, higher average in-hospital SBP (≥130 mmHg) was significantly associated with lower 1-year mortality. These findings highlight the importance of cautious acute phase BP monitoring. Given the observational design, results are hypothesis-generating and warrant validation in prospective interventional trials specifically targeting this under-represented population.

Key words： acute myocardial infarction • blood pressure • all-cause mortality • elderly patients

论文 ID：458

Association of Hypertension with Prevalent and Incident of Osteoporosis: Combined Cross-Sectional and Prospective Cohort Analysis Based on a Large Health Check-Up Population

Jingna Cheng*

301hospital

Corresponding Author E-mail: 1228780361@qq.com

Background： Based on a large-scale health examination population, this study employed a dual-design approach integrating cross-sectional analysis with a retrospective cohort to elucidate the association between hypertension and osteopenia and to assess the predictive value of hypertension for incident osteopenia. The findings aim to inform the prevention of chronic comorbidities and guide early bone health interventions.

Methods： In this observational cross-sectional study, 151,671 individuals aged ≥18 years who underwent bone mineral density (BMD) assessments between April 2013 and February 2025 were enrolled. Based on BMD screening results, participants were classified into an osteopenia group and a normal bone mass control group. Hypertension status was defined according to the Chinese Guidelines for the Management of Hypertension. A retrospective cohort of 32,172 participants with no baseline osteopenia and at least two follow-up visits was established. Logistic regression, Kaplan–Meier survival analysis, and Cox proportional hazards models were utilized to examine associations.

Results： Multiple clinical parameters differed significantly between the osteopenia group and the control group. Using a Poisson generalized linear model with a log link, hypertension was significantly associated with osteopenia prevalence. Compared with normotensive individuals, the crude prevalence ratio (PR) of osteopenia among hypertensive participants was 1.516 (95% CI: 1.477–1.556, P<0.001). After adjusting for sex, smoking status, alcohol consumption, age, and body mass index (BMI), the PR remained significant at 1.211 (95% CI: 1.177–1.244, P<0.001), indicating that hypertension is an independent risk factor for osteopenia. In the retrospective cohort, 2,329 new cases of osteopenia occurred during follow-up. The incidence rate of incident osteopenia among hypertensive participants was 29.33 per 1,000 person-years, significantly higher than 21.03 per 1,000 person-years in the normotensive group (P<0.001). Kaplan–Meier analysis revealed that hypertensive participants had a significantly higher cumulative risk of osteopenia (Log-rank χ^2 = 55.583, P<0.001). The osteopenia-free survival curves diverged progressively over time, indicating lower survival probabilities in the hypertensive group. Multivariable Cox regression analysis further confirmed that hypertension independently predicted incident osteopenia (adjusted hazard ratio [HR] = 1.317, 95% CI: 1.181–1.469, P<0.001), corresponding to a 31.7% increased risk compared with normotensive individuals.

Conclusion： Hypertension is strongly associated with prevalent osteopenia and serves as an independent risk factor for its incidence. In clinical practice and health management, routine BMD screening and longitudinal monitoring should be prioritized for hypertensive populations to enable early bone health interventions and mitigate the future risk of osteoporosis and fragility fractures.

Key words： health check-up; bone mineral density; osteopenia; hypertension; fracture

论文 ID：465

Association between autonomic dysfunction and arterial stiffness in hypertensive patients

Jijing Wang, jinyi xu*

Henan Provincial People's Hospital

Corresponding Author E-mail: 1004222047@qq.com

Background： The purpose of this study was to investigate whether heart rate variability (HRV), a predictor of autonomic function, is associated with arterial stiffness in hypertensive patients.

Methods： A total of 1,132 essential hypertension patients were included in this retrospective study. The standard deviation of normal-to-normal intervals (SDNN), an indicator of HRV, was selected to assess autonomic function. Arterial stiffness was evaluated by measuring the brachial-ankle pulse wave velocity (baPWV). Patients were categorized into tertiles based on their SDNN values.

Results： Participants in the lowest tertile of SDNN were older and exhibited higher levels of triglycerides and fasting blood glucose compared to those in the highest tertile of SDNN. Multivariate linear regression analyses indicated that SDNN had an independent negative correlation with baPWV (β=−3.60; 95% confidence interval [CI] =−4.78~−2.41) after adjusting for all covariates. Consistently, multiple logistic regression analyses revealed a negative relationship between SDNN and the elevated baPWV (>75th percentile) (odds ratio [OR]=0.97; 95% CI=0.95~0.99). Evaluations utilizing restricted cubic splines confirmed that the relationships between SDNN and baPWV displayed an L-shaped curve (non-linear, P<0.001). Subgroup analyses indicated that more pronounced associations between SDNN and baPWV were observed in younger individuals (under 65 years) (P for interaction< 0.05).

Conclusion： This study demonstrated that the SDNN is independently and negatively associated with baPWV in hypertensive patients, particularly in those under 65 years of age. These findings suggest a potential relationship between arterial stiffness and autonomic nervous system function.

Key words： Heart rate variability, Brachial-ankle pulse wave velocity, Resting heart rate, Arterial stiffness, Hypertension

论文 ID：468

Heart rate deceleration and acceleration capacities associated with circadian rhythm of blood pressure in essential hypertension

Jijing Wang*

Henan Provincial People's Hospital

Corresponding Author E-mail: wangjijing1988@163.com

Background： This study aimed to investigate the potential association between the circadian rhythm of blood pressure and deceleration capacity (DC)/acceleration capacity (AC) in patients with essential hypertension.

Methods： This study included 318 patients with essential hypertension, whether or not they were being treated with anti-hypertensive drugs, who underwent 24-hour ambulatory blood pressure monitoring (ABPM). Patients were categorized into three groups based on the percentage of nocturnal systolic blood pressure (SBP) dipping: the dipper, non-dipper and reverse dipper groups. Baseline demographic characteristics, ambulatory blood pressure monitoring parameters, Holter recordings (including DC and AC), and echocardiographic parameters were collected.

Results：In this study, the lowest DC values were observed in the reverse dipper group, followed by the non-dipper and dipper groups (6.46±2.06 vs. 6.65±1.95 vs. 8.07±1.79 ms, P<.001). Additionally, the AC gradually decreased (-6.32±2.02 vs. -6.55±1.95 vs. -7.80±1.73 ms, P<.001). There was a significant association between DC (r=.307, P<.001), AC (r=-.303, P<.001) and nocturnal SBP decline. Furthermore, DC (β=0.785, P=.001) was positively associated with nocturnal SBP decline, whereas AC was negatively associated with nocturnal SBP (β = -0.753, P=.002). By multivariate logistic regression analysis, deceleration capacity [OR (95% CI): 0.705 (0.594–0.836), p<.001], and acceleration capacity [OR (95% CI): 1.357 (1.141–1.614), p=.001] were identified as independent risk factors for blood pressure nondipper status. The analysis of ROC curves revealed that the area under the curve for DC/AC in predicting the circadian rhythm of blood pressure was 0.711/0.697, with a sensitivity of 73.4%/65.1% and specificity of 66.7%/71.2%.

Conclusion：Abnormal DC and AC density were correlated with a blunted decline in nighttime SBP, suggesting a potential association between the circadian rhythm of blood pressure in essential hypertension patients and autonomic nervous dysfunction.

Key words： Deceleration capacity, Heart rate variability, Circadian rhythm of blood pressure, Essential hypertension.

论文 ID：470

Association of Birth Weight with Childhood Hypertension and the Potential Role of Gut Microbiota: a Retrospective Cohort Study

Xiaorui Ruan[1], Kebin Chen[1], Manjun Luo[1], Xinxin Bao[1], Abudurexiti Xirennayi[1], Chenglu Zhou[1], Zhanwen Li[1], Wan Zhao[1], Yue Yuan[1], Tingting Wang[1], Yaqing Tan[2], Jiabi Qin*[3]

1. Central South University

2. Hunan Provincial Center for Disease Control and Prevention

3. School of Public Health, Kunming Medical University

Corresponding Author E-mail: qinjiabi123@163.com

Background：Childhood hypertension represents a critical public health concern. Although birth weight has been associated with blood pressure variations, its potential nonlinear relationship with childhood hypertension and the role of gut microbiome in this association remain unclear.

Methods：A retrospective cohort study of 2,389 children aged 8-14 years and their parents was conducted in China between December 2023 and April 2024. Log-binomial regression and restricted cubic spline analysis were used to examine associations between birth weight and childhood hypertension, adjusting for potential confounders. A nested case-control study within the cohort included 80 children with hypertension and 560 normotensive controls, from whom fecal samples were collected for shotgun metagenomic sequencing. Microbial diversity, taxonomic composition, and mediation analyses were performed to assess the potential role of gut microbiota.

Results： Among 2,389 children (10.53 ± 0.91 years; 47.30% girls), 363 (15.19%) were identified with hypertension. Birth weight showed a U-shaped association with childhood hypertension (P-nonlinear < 0.05), with an

inflection point at 3,100 (95%CI: 2,755-3,445) g. Below this threshold, children with each 500-g birth weight growth had a lower risk of hypertension (RR = 0.76, 95% CI: 0.62-0.93); while this risk increased when birth weight exceeds 3,100 g (RR = 1.17, 1.02-1.35). This association was consistent in subgroup analyses. In the metagenomic subset, children with hypertension exhibited significantly decreased microbial diversity and altered composition compared with normotensive controls (α diversity: Shannon index, P = 0.021; β diversity: PERMANOVA, P = 0.002). We identified 51 hypertension-associated species, with enrichment of Streptococcus, Ruminococcus, and Blautia (within the phylum Bacillota) in children with hypertension, and Bacteroides, Phocaeicola, and Parabacteroides (within the phylum Bacteroidota) in normotensive controls. Among children with birth weight of 3,100 g or greater, Tyzzerella nexilis and Streptococcus suis showed significant negative indirect effects on hypertension risk.

Conclusion： Birth weight was nonlinearly associated with childhood hypertension. Gut microbiome dysbiosis were associated with hypertension and may partially mediate the relationship between higher birth weight and childhood hypertension. Our findings identify a potential microbial pathway linking early-life growth with later blood pressure regulation, suggesting microbial targets for childhood cardiovascular prevention.

Key words： Birth weight; Childhood hypertension; Gut microbiota; Metagenomic sequencing; Nonlinear relationship; Mediation analysis

论文 ID：510

Comparison of Novel Systemic Inflammatory Indices for Identification of Left Ventricular Hypertrophy in Hypertensive Patients: A Large-Scale Cross-Sectional Study in China

Ning Yang[1,2], Nanfang Li*[1]

1. Hypertension Center of People's Hospital of Xinjiang Uygur Autonomous Region

2. People's Hospital of Xinjiang Uygur Autonomous Region

Corresponding Author E-mail: lnanfang2016@sina.com

Background： Inflammation plays a key role in the onset of hypertension and left ventricular hypertrophy (LVH), large-scale studies on inflammatory markers in hypertension-related LVH are lacking. This study aims to investigate the relationship between inflammatory markers and LVH in hypertensive patients and evaluate their clinical diagnostic value.

Methods： The present cross-sectional study included 21486 patients diagnosed with hypertension from a China Hospital. Six inflammatory indices are calculated based on complete blood counts, including the Aggregate Index of Systemic Inflammation (AISI), Systemic Inflammation Response Index (SIRI), Systemic Immune Inflammation Index (SII), Neutrophil-to-Lymphocyte Ratio (NLR), Neutrophil-to-Platelet Ratio (NPR) and the Platelet-to-Lymphocyte Ratio (PLR). Multivariate logistic regression models, receiver operating characteristic (ROC) curves, the area under the curve (AUC), the net reclassification improvement (NRI), the improved discriminant index (IDI), decision curve analysis (DCA), sensitivity analysis and subgroup analysis were performed.

Results： Levels of all inflammatory indices were significantly higher in hypertensive patients with LVH compared to those without LVH. Following multivariate adjustment, the odds of prevalent LVH increased by 76%, 48%

and 39% for each one-standard-deviation increase in SII, NLR and AISI, respectively. After incorporating SII into the traditional risk factor model, the AUC of the combined diagnostic model increased significantly to 0.750, with an NRI of 0.437 and an IDI of 0.047. The AUC values for the combined models incorporating NLR, AISI, SIRI, PLR and NPR were 0.736, 0.728, 0.726, 0.720 and 0.719, respectively.

Conclusion： In hypertensive patients, inflammatory indices, particularly SII, demonstrate a significant association with LVH and may provide incremental value for risk stratification beyond traditional risk factors. Further studies are needed to evaluate the clinical efficacy of these indices in management LVH.

Key words： left ventricular hypertrophy; inflammatory indices; hypertension; diagnostic accuracy

论文 ID：816

Association Between HDL-C and Intensive Bloodpressure Control in Patients with Hypertension: A post-hoc analysis of SPRINT

Wenli Cheng*, Rufei Liu

Beijing Anzhen Hospital, Capital Medical University

Corresponding Author E-mail: chengwenli2000@163.com

Background： Previous studies in patients with hypertension have demonstrated that there is a U- shaped association between HDL-C (high-density lipoprotein cholesterol) and the risk of cardiovascular events in male patients with hypertension. However, to the best of our knowledge, the relationship between HDL-C and intensive blood pressure control in specific cardiovascular events has never been investigated.

Methods： To fill this knowledge gap, the authors analyzed the relationship between HDL-C levels and cardiovascular events in hypertensive patients within the Systolic Blood Pressure Intervention Trial (SPRINT). The SPRINT evaluated the impact of intensive blood pressure control (systolic blood pressure < 120 mm Hg) versus standard blood pressure control (sys- tolic blood pressure < 140 mm Hg). The Cox proportional risk regression was used to investigate the association between different HDL-C status and clinical outcomes. Additional stratified analyzes were performed to evaluate the robustness of sex difference.

Results： A total of 9323 participants (6016 [64.53%] males and 3307 [35.47%] females) with hypertension from the SPRINT research were included in the analysis. The median follow-up period was 3.26 years. Our population was divided into five groups based on the HDL-C plasma levels: HDL-C < 30 mg/dL, HDL-C between 30 and 40 mg/dL, HDL- C between 40and 60mg/dL, HDL-C between 60and 80mg/dLand HDL-C >80mg/dL. Sensitivity analyzes showed that in the SPRINT, women in the HDL-C high population hada higher risk of mortality from all causes than men.

Conclusion： In this cohort study, results suggest that patients with HDL-C levels higher than 80 mg/dL had lower risk of SPRINT primary outcome, cardiovascular death, and stroke, but this study tested association, not causation. HDL-C levels were associated with composite cardiovascular outcomes in male but not female patients. Our results demonstrated that in patients with hyper- tension, the association between HDL-C and risk of cardiovascular events is L-shaped.

Key words： HDL-C Intensive Bloodpressure Control Hypertension