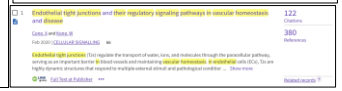
序号	学系	第一作者	责任作者	论文题目（英文）	期刊	论文完整信息	期刊影响因子	论文第一单位	论文简介
1	生理学与病理生理学系	翁琳	郑铭	TGF-β1/SMAD3 regulates programmed cell death 5 that suppresses cardiac fibrosis post-myocardial infarction by inhibiting HDAC3	CIRCULATION RESEARCH	Weng L, Ye J, Yang F, Jia S, Leng M, Jia B, Xu C, Zhao Y, Liu R, Xiong Y, Zhou Y, Zhao J, Zheng M*. TGF-β1/SMAD3 Regulates Programmed Cell Death 5 That Suppresses Cardiac Fibrosis Post-Myocardial Infarction by Inhibiting HDAC3. Circ Res. 2023 Jul 21; 133:237-251	20.1	Department of Physiology and Pathophysiology, Health Science Center, Peking University	该研究发现PDCD5是经典纤维化信号TGF-β/SMAD3的新靶点，在心梗后心脏纤维化患者和纤维化小鼠心脏中上调，并通过抑制HDAC3负反馈性下预纤维化进展。提示PDCD5是心脏纤维化的内源性稳态调节因子，在干预纤维化重塑相关的心血管疾病中具有重要的转化前景。
2	生理学与病理生理学系	刘佳钰、王谨	周菁	Liquid-liquid phase separation of DDR1 counteracts the Hippo pathway to orchestrate arterial stiffening.	CIRCULATION RESEARCH	Liu J#, Wang J#, Liu Y, Xie S, Zhang J, Zhao C, Zhou Y, Pang W, Yao W, Peng Q, Wang X*, Zhou J*. Liquid-liquid phase separation of DDR1 counteracts the Hippo pathway to orchestrate arterial stiffening. Circ Res. Jan 6;132(1):87-105. 2023.	20.1	Department of Physiology and Pathophysiology, School of Basic Medical Sciences, Peking University, Beijing 100191, China.	该研究揭示在理、化刺激（ECM刚度升高或胶原结合）下的VSMC DDR1发生了“液-液”相分离（LLPS），活化的DDR1与Hippo通路的核心激酶LATS1形成共凝聚物，从而抑制了LATS1对YAP的磷酸化，导致YAP的入核和下游靶基因的激活，加重VSMC细胞表型转换与钙化和血管硬化。
3	生理学与病理生理学系	刘佳钰、赵川榕	周菁	Endothelial discoidin domain receptor 1 senses flow to modulate YAP activation.	NATURE COMMUNICATIONS	Liu J#, Zhao C#, Xiao X, Li A, Liu Y, Zhao J, Fan L, Liang Z, Pang W, Yao W, Li W*, Zhou J*. Endothelial discoidin domain receptor 1 senses flow to modulate YAP activation. Nat Commun. 14(1):6457. 2023.	16.6	Department of Physiology and Pathophysiology, School of Basic Medical Sciences, Peking University, Beijing 100191, China.	该研究首次发现并证明了盘状蛋白结构域受体DDR1可以充当内皮细胞中直接的力学感受器，并通过单分子磁镊技术解析了DDR1在力学刺激后发生构象改变的关键结构域。
4	生理学与病理生理学系	赵佳楠	周菁	DNMT1 mediates the disturbed flow-induced endothelial to mesenchymal transition through disrupting b-alanine and carnosine homeostasis.	THERANOSTICS	Zhao J, Zhao C, Yang F, Jiang Z, Zhu J, Yao W, Pang W, Zhou J*. DNMT1 mediates the disturbed flow-induced endothelial to mesenchymal transition through disrupting b-alanine and carnosine homeostasis. Theranostics. 13(13):4392-4411. 2023.	12.4	Department of Physiology and Pathophysiology, School of Basic Medical Sciences, Peking University, Beijing 100191, China.	该研究发现血流扰动上调DNA甲基转移酶1（DNMT1）的表达，DNMT1增加ALDH2、ALDH3A1和ALDH6A1启动子区域的甲基化而抑制其表达，而ALDH2、ALDH3A1和ALDH6A1表达降低导致β丙氨酸代谢通路的产物肌肽和乙酰精酶A合成降低，继而促进Smad2/3的磷酸化而激活TGF-β信号通路，从而诱导内皮向间质转化（EndoMT）的发生。
5	生理学与病理生理学系	王佳乐（学生）、丁金辉（学生）	孙金鹏	Functional screening and rational design of compounds targeting GPR132 to treat diabetes	NATURE METABOLISM	Wang JL, Dou XD, Cheng J, Gao MX, Xu GF, Ding W, Ding JH, Li y, Wang SH, Ji ZW, Zhao XY, Huo TY, Zhang CF, Liu YM, Sha XY, Gao JR, Zhang WH, Hao Y, Zhang C, Sun JP#, Jiao N# and Yu X#. Functional screening and rational design of compounds targeting GPR132 to treat diabetes. Nat Metab. 2023 Oct;5(10):1726-1746.	20.1	Dept. of Physiology and Pathophysiology,School of Basic Medical Sciences, PKU	我们研究发现氧化脂肪酸脂代谢产物激活GPR132对胰岛驻留型巨噬细胞重编程发挥重要的调控作用，是促发胰岛炎症的重要原因之一。进一步，联合研究团队通过阐明GPR132识别内源性配体9(S)-HODE，NPGLY并激活的分子机制，结合计算机辅助药物设计、合成药物化学以及药理学检测评估手段开发了靶向GPR132的高亲和力及选择性拮抗剂候选小分子NOX-6-18，能够显著改善糖尿病小鼠的胰岛炎症，并提高糖代谢稳态，为治疗糖尿病提供了新的思路。
6	生理学与病理生理学系	熊梦瑶（学生）、蔡菜（学生）、贾英丽（教工）	孙金鹏、杨宝学	Single hormone or synthetic agonist induces Gs/Gi coupling selectivity of EP receptors via distinct binding modes and propagating paths	PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES OF AMERICA	Huang SM, Xiong MY, Liu L, Mu J, Wang MW, Jia YL, Cai K, Tie L, Zhang C, Cao S, Wen X, Wang JL, Guo SC, Li Y, Qu CX, He QT, Cai BY, Xue C, Gan S, Xie Y, Cong X, Yang Z, Kong W, Li S, Li Z, Xiao P, Yang F, Yu X, Guan YF, Zhang XF, Liu ZJ#, Yang BX#, Du Y#, Sun JP#. Single hormone or synthetic agonist induces Gs/Gi coupling selectivity of EP receptors via distinct binding modes and propagating paths. Proc Natl	11.1	Department of Physiology and Pathophysiology, School of Basic Medical Sciences, State Key Laboratory of Vascular Homeostasis and Remodeling, Peking University, Beijing 100191, China.	我们以前列腺素E2受体（Prostaglandin E2 receptors，EP）家族为研究模型，揭示了单个内源性配体引起同一GPCR多样性信号转导的分子机制，为理解脂肪酸配体调控GPCR选择性激活不同下游信号通路的结构机制做了重要补充，为精准靶向EP受体家族治疗肾脏疾病，维持肾脏功能稳态提供了新思路和结构理论基础。
7	生理学与病理生理学系	马子涵（博士生）、毛晨峰（博士生）	孔炜、付毅	Peptide Vaccine Against ADAMTS-7 Ameliorates Atherosclerosis and Postinjury Neointima Hyperplasia	CIRCULATION	Zihan Ma#, Chenfeng Mao#, Xiao Chen, Shiyu Yang, Zhihua Qiu, Baoqi Yu, Yiting Jia, Chao Wu, Yiyi Wang, Yuhui Wang, Rui Gu, Fang Yu, Yanhui Yin, Xian Wang, Qingbo Xu, Chuanjiu Liu, Yuhua Liao*, Jingang Zheng*, Yi Fu*, Wei Kong*. Peptide Vaccine Against ADAMTS-7 Ameliorates Atherosclerosis and Postinjury Neointima Hyperplasia. Circulation. 2023 Feb 28;147(9):728-742.	37.8	Department of Physiology and Pathophysiology, School of Basic Medical Sciences, Peking University Health Science Center, 38 Xueyuan Rd, Haidian Distrct, Beijing	慢病疫苗是近年来新兴的一种非常具有临床转化前景的针对非感染性疾病的干预策略。现有针对动脉粥样硬化的疫苗研发主要集中于降脂的靶点。我们研发了一种以血管本身的重构病变为靶点的ATS7vac疫苗，是现有临床上降脂为主的抗动脉粥样硬化治疗策略的有效补充，都尼盟工业大学Thorsten Kessler教授等在Eur Heart J发表文章，认为我们的ADAMTS-7是高其非常令人振奋的抗动脉粥样硬化效果：Circulation同期发表述评，认为我们发现的疫苗在抗炎及动脉粥样硬化预防治疗中具有广阔前景。
8	生理学与病理生理学系	马子涵（博士生）、毛晨峰（博士生）	付毅、孔炜	ADAMTS7-Mediated Complement Factor H Degradation Potentiates Complement Activation to Contributing to Renal Injuries.	JOURNAL OF THE AMERICAN SOCIETY OF NEPHROLOGY	Ma, Zihan; Mao, Chenfeng; Jia, Yiting; Yu, Fang; Xu, Ping; Tan, Yingz; Zou, Qing-Hua; Zhou, Xu-Jie; Kong, Wei; Fu, Yi*. ADAMTS7-Mediated Complement Factor H Degradation Potentiates Complement Activation to Contributing to Renal Injuries. J Am Soc Nephrol. 2023 Feb 1;34(2):291-308	13.6	Department of Physiology and Pathophysiology, School of Basic Medical Sciences, Peking University, Key Laboratory of Molecular Cardiovascular Science, Ministry of Education, Beijing, China	我们的工作阐明了血管内皮紧密连接表达和功能异常是胸主动脉瘤/夹层发生发展的早期事件，以检测内皮屏障功能为基础合成的新型探针可作为胸主动脉瘤/夹层早期预警指标，而使用紧密连接封闭剂可显著降低疾病发生率。这一发现为胸主动脉瘤/夹层这一内险的心血管疾病的早期诊断与治疗提供新的思路及靶点。安特卫普大学血管疾病研究专家Bart Loeys教授在同期发表述评认为我们发现的“内皮屏障破损探针可能在临床胸主动脉夹层的诊断中发挥重要应用”。
9	生理学与病理生理学系	杨雪媛（博士生）	孔炜、丛馨	Targeting endothelial tight junctions to predict and protect thoracic aortic aneurysm and dissection	EUROPEAN HEART JOURNAL	Xueyuan Yang#,Chen Xu#, Fang Yao, Qianhui Ding, Hao Liu, Congcong Luo, Daidai Wang, Jiaqi Huang, Zhiqing Li, Yicong Shen, Weijie Yang, Zhuofan Li, Fang Yu, Yi Fu, Li Wang, Qingbian Ma, Junming Zhu, Fujian Xu*, Xin Cong*, Wei Kong*. Eur Heart J. 2023 Apr 7;44(14):1248-1261.	39.3	Department of Physiology and Pathophysiology, School of Basic Medical Sciences, Peking University, Key Laboratory of Molecular Cardiovascular Science, Ministry of Education, Beijing 100191, China	我们的工作阐明了血管内皮紧密连接表达和功能异常是胸主动脉瘤/夹层发生发展的早期事件，以检测内皮屏障功能为基础合成的新型探针可作为胸主动脉瘤/夹层早期预警指标，而使用紧密连接封闭剂可显著降低疾病发生率。这一发现为胸主动脉瘤/夹层这一内险的心血管疾病的早期诊断与治疗提供新的思路及靶点。安特卫普大学血管疾病研究专家Bart Loeys教授在同期发表述评认为我们发现的“内皮屏障破损探针可能在临床胸主动脉夹层的诊断中发挥重要应用”。
10	生理学与病理生理学系	沈一聪（博士生）、董志刚（博士生）	付毅、孔炜	Targeting cytokine-like protein FAM3D lowers blood pressure in hypertension	CELL REPORTS MEDICINE	Yicong Shen#, Zhigang Dong#, Fangfang Fan, Kaiyin Li, Shirong Zhu, Rongbo Dai, Jiaqi Huang, Nan Xie, Li He, Ze Gong, Xueyuan Yang, Jiaia Tan, Limei Liu, Fang Yu, Yida Tang, Zhen You, Jianzhong Xi, Ying Wang, Wei Kong*, Yan Zhang*, Yi Fu*. Targeting cytokine-like protein FAM3D lowers blood pressure in hypertension. Cell Rep Med. 2023 Jun 20;4(6):101072.	14.3	Department of Physiology and Pathophysiology, School of Basic Medical Sciences, Peking University, Beijing 100191, China	我们通过病例对照研究和小鼠模型发现，序列相似家族3成员D（FAM3D）在高血压患者中升高，与高血压发病率呈正相关，FAM3D缺乏可显著改善小鼠血管紧张素II（AngII）诱导的高血压。机制研究结果显示，FAM3D直接导致内皮型一氧化氮合酶（eNOS）解偶联并损害内皮依赖性血管舒张，甲酰基肽受体1 和2（FPR1和FPR2）的拮抗或氧化应激的抑制会减弱FAM3D诱导的eNOS解偶联。通过腺相关病毒载体特异性敲除内皮细胞中的FAM3D或给予FAM3D中和抗体，可显著逆转内皮功能障碍，改善高血压。我们的研究发现FAM3D是高血压的潜在治疗靶点，为高血压的治疗提供新思路。
11	生理学与病理生理学系	张煦（博士生）	孔炜、付毅、姜长涛	Alkaline ceramidase 1–mediated platelet ceramide catabolism mitigates vascular inflammation and abdominal aortic aneurysm formation	NATURE CARDIOVASCULAR RESEARCH	Xu Zhang, Ze Gong, Yicong Shen, Zeyu Cai, Liu Yang, Tao Zhang, Weihao Li, Yang Zhao, Shirong Zhu, Cihang Liu, Jin Wang, Xian Wang, Ruomei Qi, Junling Liu, Xiaoguang Lei, Weiqiang Wang, Changtao Jiang*, Yi Fu*, Wei Kong*. Alkaline ceramidase 1–mediated platelet ceramide catabolism mitigates vascular inflammation and abdominal aortic	Nature新子刊，未出影响因子	Department of Physiology and Pathophysiology, School of Basic Medical Sciences, State Key Laboratory of Vascular Homeostasis and Remodeling, Peking University, Beijing, China.	该研究通过使用靶向脂质分析发现，在AAA小鼠和患者中，血小板押经酰胺酶均显著升高；进一步针对押经酰胺代谢通路中的关键酶进行筛选，发现仅有水解神经酰胺的碱性神经酰胺酶 1（Acer1）在AAA的血小板中显著下调且酶活性下降，而其他神经酰胺合成通路的关键酶并无显著变化。利用构建的血小板Acer1条件敲除小鼠，发现Acer1缺病引起血小板神经酰胺蓄积，加重血管紧张素II诱导AAA的进展。机制上，Acer1缺病引起的血小板神经酰胺蓄积显著促进血小板促炎细胞因子的分泌和P-选择素介导的血小板-白细胞聚集形成，加重血管炎症，该作用依赖于血小板p38 MAPK信号的活化。有趣的是，Acer1介导的血小板神经酰胺水解并不影响血小板参与生理性止血或血栓形成的功能。综上，血小板神经酰胺在促进动脉粥样硬化小鼠模型血管炎症中具有潜在靶点价值。这有力地证明了血小板应激和
12	生理学与病理生理学系	汪锴、张志威	姜长涛	Microbial-host-isozyme analyses reveal microbial DPP4 as a potential antidiabetic target	SCIENCE	Wang K#, Zhang Z#, Hang J#, Liu J#, Guo F#, Ding Y, Li M, Nie Q, Lin J, Zhuo Y, Sun L, Luo X, Zhong Q, Ye C, Yun C, Zhang Y, Wang J, Bao R, Pang Y, Wang G*, Gonzalez FJ*, Lei X*, Qiao J*, Jiang CT*. Microbial-host-isozyme analyses reveal microbial DPP4 as a potential antidiabetic target. Science. 2023 Aug 4;381(6657).	56.9	Dept. of Physiology and Pathophysiology,School of Basic Medical Sciences, PKU	这论文通过菌源宿主同工酶挖掘体系，发现多种菌源宿主同工酶的广泛分布，其中菌源二肽基肽酶4（DPP4）能够降解宿主胰高血糖素样肽-1（GLP-1），加重代谢性疾病进程，影响肠道DPP4抑制剂西格列汀疗效，并开发了靶向菌源DPP4的抑制剂Dau-d4，能够通过特异性抑制菌源DPP4改善2型糖尿病。该成果获得Nature Reviews Drug Discovery, Cell Metabolism, Nature Chemistry Biology等杂志亮点介绍与述评，认为肠道菌源宿主同工酶是全新的代谢性疾病治疗靶点。
13	生理学与病理生理学系	杜幸	冯娟 王宪	B cell-derived anti-beta 2 glycoprotein I antibody mediates hyperhomocysteinemia-aggravated hypertensive glomerular lesions by triggering	Signal Transduct Target Ther.	2. Du X, Ma X, Tan Y, Shao F, Li C, Zhao Y, Miao Y, Han L, Dang G, Song Y, Yang D, Deng Z, Wang Y, Jiang C, Kong W, Feng J*, Wang X*. B cell-derived anti-beta 2 glycoprotein I antibody mediates hyperhomocysteinemia-aggravated hypertensive glomerular lesions by triggering ferroptosis. Signal Transduct Target Ther. 2023 Mar 13; 8:103.	39.3	Department of Physiology and Pathophysiology, School of Basic Medical Sciences, Key Laboratory of Molecular Cardiovascular Science, Ministry of Education, Peking University, 100191, Beijing, P. R. China.	该文在临床和小鼠模型样本中，证实脂质过氧化介导的铁死亡在高同型半胱氨酸加速肾脏损伤过程的作用。从脂质和铁代谢的角度提出高血压和高同型半胱氨酸的协同作用机制，为血管损伤性疾病的预警和诊疗提供新策略。

优秀论文引用（2020.1.1以后发表、发表后三年内他引次数>30次论文）

序号	学系	第一作者	责任作者	论文题目（英文）	期刊	论文完整信息	影响因子	论文第一单位 （即论文所有单位中排第一的单位，非作者第一单位，可直接copy原文中的第一单位）	引用情况(仅他引次数)
1	生理学与病理生理学系	季昀	张炜真	Anti-Inflammatory and Anti-Oxidative Activity of Indole-3-Acetic Acid Involves Induction of HO-1 and Neutralization of Free Radicals in RAW264.7 Cells	INTERNATIONAL JOURNAL OF MOLECULAR SCIENCES	Ji Y#, Yin W, Liang Y, Sun L, Yin Y*, Zhang W. Anti-Inflammatory and Anti-Oxidative Activity of Indole-3-Acetic Acid Involves Induction of HO-1 and Neutralization of Free Radicals in RAW264.7 Cells. Int J Mol Sci. 2020 Feb 25;21(5):1579.	5.923	Peking Univ, Hlth Sci Ctr, Dept Physiol & Pathophysiol,	34次
2	生理学与病理生理学系	季昀	张炜真	The Molecular and Mechanistic Insights Based on Gut-Liver Axis: Nutritional Target for Non-Alcoholic Fatty Liver Disease (NAFLD) Improvement.	INTERNATIONAL JOURNAL OF MOLECULAR SCIENCES	Ji Y, Yin Y, Sun L, Zhang W*. The Molecular and Mechanistic Insights Based on Gut-Liver Axis: Nutritional Target for Non-Alcoholic Fatty Liver Disease (NAFLD) Improvement. Int J Mol Sci. 2020 Apr 26;21(9):3066.	5.923	Peking Univ, Hlth Sci Ctr, Key Lab Mol Cardiovasc Sci, Minist Educ,Dept Physiol & Pathophysiol	62次
3	生理学与病理生理学系	汪锴	姜长涛	Intestinal hypoxia-inducible factor 2a regulates lactate levels to shape the gut microbiome and alter thermogenesis	CELL METABOLISM	Wu Q#, Liang X#, Wang K#, Lin J, Wang X, Wang P, Zhang Y, Nie Q, Liu H, Zhang Z, Liu J, Pang Y, Jiang C*. Intestinal hypoxia-inducible factor 2a regulates lactate levels to shape the gut microbiome and alter thermogenesis. Cell Metab. 2021 Oct 5;33(10):1988-2003.e7.	31.4	Dept. of Physiology and Pathophysiology,School of Basic Medical Sciences, PKU	56次
4	生理学与病理生理学系	丛馨	孔炜	Endothelial tight junctions and their regulatory signaling pathways in vascular homeostasis and disease	CELLULAR SIGNALLING	Cong X, Kong W. Endothelial tight junctions and their regulatory signaling pathways in vascular homeostasis and disease. Cell Signal. 2020 Feb;66:109485. doi: 10.1016/j.cellsig.2019.109485. Epub 2019 Nov 23. PMID: 31770579.	4.315	Department of Physiology and Pathophysiology, Peking University School of Basic Medical Sciences, Key Laboratory of Molecular Cardiovascular Sciences, Ministry of Education, Beijing Key Laboratory of Cardiovascular Receptors Research, Beijing 100191, China.	122次