

肥胖与心血管疾病的临床研究进展

杨丽丽*, 孙 欣#

内蒙古民族大学第二临床医学院(内蒙古林业总医院), 内蒙古 牙克石

收稿日期: 2025年7月19日; 录用日期: 2025年8月12日; 发布日期: 2025年8月20日

摘 要

肥胖是心血管疾病(CVD)发展的既定危险因素。肥胖会增加患CVD的风险, 并导致其他心血管危险因素(如糖尿病、血脂异常和高血压)的发展。肥胖与代谢、激素和炎症有关, 可导致动脉粥样硬化并增加冠状动脉疾病的风险, 心肌重塑增加心力衰竭的风险。本文将研究肥胖对CVD风险的影响, 并探讨肥胖和CVD之间的关系。

关键词

肥胖, 心血管疾病, 临床研究进展

Clinical Research Advances in Obesity and Cardiovascular Disorders

Lili Yang*, Xin Sun#

Second Clinical Medical College of Inner Mongolia Minzu University (Root Forestry General Hospital), Yakeshi Inner Mongolia

Received: Jul. 19th, 2025; accepted: Aug. 12th, 2025; published: Aug. 20th, 2025

Abstract

Obesity is a well-established risk factor for the development of cardiovascular disease (CVD). It increases the risk of CVD and contributes to the development of other cardiovascular risk factors, such as diabetes, dyslipidemia, and hypertension. Obesity is associated with metabolic, hormonal, and inflammation changes that can lead to atherosclerosis and elevate the risk of coronary artery disease, while cardiac remodeling increases the risk of heart failure. This article examines the impact of obesity on CVD risk and explores the relationship between obesity and CVD.

*第一作者。

#通讯作者。

文章引用: 杨丽丽, 孙欣. 肥胖与心血管疾病的临床研究进展[J]. 临床医学进展, 2025, 15(8): 1196-1201.

DOI: 10.12677/acm.2025.1582352

Keywords

Obesity, CVD, Progress in Clinical Studies

Copyright © 2025 by author(s) and Hans Publishers Inc.

This work is licensed under the Creative Commons Attribution International License (CC BY 4.0).

<http://creativecommons.org/licenses/by/4.0/>



Open Access

1. 引言

肥胖及其相关疾病已成为全球主要的健康问题, 超重和肥胖是导致健康问题的主要生活方式疾病之一, 并导致许多慢性疾病, 如癌症、糖尿病、代谢综合征和心血管疾病[1]。肥胖与多种心血管疾病的患病率增加有关, 从冠心病到心房颤动甚至心力衰竭。

2. 肥胖和心血管疾病的概述

2.1. 肥胖的定义

肥胖是一种多因素疾病, 其发病机制复杂, 与生物社会心理[2]、社会经济[3]和环境因素[4]以及导致不良健康结果的途径和机制有关, 通常由体重指数(BMI)定义, 世界卫生组织将肥胖定义为 $BMI \geq 30 \text{ kg/m}^2$, 但由于世界人类种族不同, 将中国人肥胖定义为 $BMI \geq 28 \text{ kg/m}^2$ [5]。

2.2. 肥胖和冠状动脉疾病(CAD)

肥胖是 CAD 的独立危险因素, 包括胰岛素抵抗[6]、高血压和血脂异常。肥胖部分是通过炎症过程导致 CAD, 该过程会加速动脉粥样硬化斑块的形成。这种炎症状态会促进低密度脂蛋白的氧化, 来促进动脉粥样硬化生成[7]。炎症和氧化应激会导致内皮细胞功能障碍[8]。脂肪组织具有释放脂肪因子的能力, 脂肪因子是由激素、趋化因子和细胞因子组成的生物活性分子[9]。肥胖患者的脂肪组织功能障碍使促炎脂肪因子增殖, 进而使脂肪因子失衡, 并通过诱导胰岛素抵抗、内皮功能障碍、高凝状态和全身炎症反应来促进动脉粥样硬化[10]。

2.3. 肥胖和心力衰竭(HF)

肥胖通过影响心肌的血流动力学和代谢变化导致心力衰竭。肾素 - 血管紧张素 - 醛固酮系统和交感神经系统在肥胖患者中均上调[11], 导致心肌细胞肥大和凋亡以及心肌纤维化, 与肥胖相关的代谢、激素和炎症变化会导致心肌重塑, 炎症因子(TNF- α 、IL-1、IL-6、IL-8 等)在肥胖症中增加, 从而增加心力衰竭的风险。与肥胖相关的促炎环境会促进动脉粥样硬化, 频繁出现的胰岛素抵抗会降低心肌的收缩力[12], 这也可能导致最终收缩功能障碍和临床心力衰竭。心脏脂肪有不同类型, 脂肪可能在心脏周围积聚, 或以肌细胞脂肪形式在心肌本身积聚[13], 后者称为异位脂肪[14]。在肥胖患者中, 心脏内部和周围的脂肪沉积会促进代谢改变、心脏脂毒性和功能障碍[15]。心肌脂质积累和纤维化增强也可以在各种心律失常中起致病作用, 这也可能导致心力衰竭的发生和发展[16]。

2.4. 肥胖和心房颤动

肥胖患者中, 白细胞数量及各种炎性细胞因子(TNF- α 、IL-6 和 C 反应蛋白)增加, TNF- α 可能会增加肺静脉的局部心律失常脆弱性, 从而引起心房颤动[17]。肥胖也可能导致自主神经功能紊乱, 在超重患者

中可检测到交感神经活动过度 and 迷走神经张力下降, 致心律紊乱[18]。

2.5. 肥胖和心源性猝死

心源性猝死和肥胖之间存在密切联系[19], 肥胖被认为是室性快速性心律失常发展的独立危险因素。肥胖患者心室肌细胞重塑致左心室肥厚及收缩和舒张功能障碍。肥胖也可能与长时间和不均匀的心室复极化有关, 表现为 QT 间期延长, 增加心源性猝死的风险[20]。

2.6. 肥胖与高血压(HT)

肥胖是一种以体内脂肪过度堆积为特征的疾病, 可对健康产生不利影响[21]。随着肥胖患病率的上升, 内脏脂肪在 HT 中的作用已得到越来越多的认可。研究表明, 内脏脂肪的过度积累是导致 CVD 和心力衰竭的关键因素之一[22]。高血压是肥胖的结果, 也是心血管疾病、肾脏疾病和痴呆的全球主要危险因素[23]。尽管高血压在肥胖症中普遍存在, 但其具体发病机制尚不十分清楚, 目前有提出几个因素。

2.6.1. 瘦素因子

瘦素是脂肪细胞分泌一种在调节体重和肥胖相关高血压中起重要作用的激素。瘦素水平升高与食欲不振、饱腹感增加和体重增加减少有关[24]。瘦素可通过刺激下丘脑瘦素受体(MC4R)对血压产生影响, 在动物实验中, 缺乏 MC4R 的雄性小鼠虽然患有严重和高瘦素血症, 但不会发展为高血压, 这表明这种信号通路在瘦素介导的发展中至关重要[24]。

2.6.2. 交感神经系统(SNS)因素

SNS 过度活跃在肥胖相关高血压中起重要作用[25], 在肥胖患者中, 交感神经活动(SNA)通常会增加, 副交感神经活动减少, SNA 可通过外周血管收缩、外周阻力增加和肾小管钠重吸收增加导致长期高血压。肥胖患者血管中内皮素-1 的产生增加也会导致肥胖相关高血压[26]。

2.6.3. 肾素血管紧张素醛固酮系统因子

RAAS 已被证明在肥胖相关高血压的发病机制中起重要作用[27]。内脏脂肪增加的肥胖高血压患者血管紧张素原水平、血浆肾素水平、血管紧张素 II 和醛固酮水平升高[28]。除了肾素受体外, 还有盐皮质激素受体和醛固酮受体在肥胖相关高血压中起作用, 为治疗肥胖相关高血压和预防靶器官损伤提供了重要的治疗方向[29]。

2.6.4. 利钠肽因子

利钠肽因子由心房利钠肽(ANP)、脑利钠肽(BNP)和 C 型利钠肽(CNP)组成, 由大脑、肾脏和心脏产生, 在全身心血管和电解质稳态中重要作用[30]。它们具有利尿、利钠、舒张血管和分解脂肪作用[31]。另一方面, 它们可激活脂联素, 脂联素可诱导瘦素释放, 起防止体重增加和增加胰岛素分泌及敏感性的作用。

3. 肥胖悖论

虽然肥胖与许多已经确定的 CVD 危险因素有关, 但据临床观察, 在某些特定人群中, 与非肥胖个体相比, 已确诊 CVD 的超重或肥胖患者在重大 CVD 事件和介入手术或心脏手术后似乎比瘦的患者有更好的短期和中期预后[32], 这种肥胖悖论已在心力衰竭[20]、冠状动脉疾病和心房颤动患者中观察到[33]。对于肥胖悖论有几种可能的解释: 在心力衰竭分解代谢状态下有更大的代谢储备、细胞因子和脂肪因子的保护性改变、循环 B 型利钠肽水平低下导致的有利血流动力学特征、循环脂蛋白升高的抗炎作用、对肾素 - 血管紧张素 - 醛固酮系统激活的反应减弱、肥胖症状加重导致早期就诊, 以及耐受更高剂量心脏保护降压药的能力[34] [35]。

肥胖悖论在 CVD 中存在,但不能直接理解为肥胖有益,心脏健康和身体活动干预已被证明可以改善有 CVD 风险患者的心肺健康[36],健康状况的改善可能会减弱肥胖悖论。

4. 肥胖相关 CVD 的管理

4.1. 肥胖症 CAD 的诊断

肥胖症 CAD 的诊断可使用无创诊断方法和有创诊断方法。无创诊断方法有心电图、跑步机压力测试、单光子发射 CT、PET (铷)、负荷超声心动图、负荷心脏 MRI、CT 钙扫描以及冠状动脉 CTA。有创诊断方法主要为心脏冠状动脉造影。

4.2. 肥胖 CVD 患者的治疗挑战及策略

在确诊 CVD 的患者中,运动训练可改善预后、结局和提高整体生活质量[37],在多数成人中,各种类型的体力活动都会减轻 CVD 危险因素并改善 CAD 的总体预后[38]。肥胖的治疗仍然是一个复杂的问题,有多种因素会影响治疗结果。

目前缺乏关于肥胖对药物药代动力学影响的数据,但受影响最大的参数是体积分布和清除率。脂肪量的增加会产生更大的亲脂性药物分布容积。分布仅限于瘦组织的药物负荷剂量应该根据肥胖患者的理想体重。在存在非酒精性脂肪肝的情况下,血流量减少。CYP450 3A4 活性降低,CYP450 2E1 活性增加。

肥胖对药效学因素影响的数据相对缺乏,由于受体表达和亲和力的差异,每个人对药物的反应不同。过多的脂肪组织会增加内在的胰岛素裂解活性,导致胰岛素需求增加,肿瘤坏死因子 α 增加,胰岛素抵抗增加。

使用体表面积来索引肥胖患者的主动脉瓣面积时,在心脏瓣膜置换术或植入后,可能会发生患者假体不匹配,因为瓣膜面积更多地取决于身高而不是体重,导致选择不合适的小瓣膜。

代谢性减肥手术是长期以来对病态肥胖唯一有效的减肥手段,包括 Roux-en-Y 胃旁路手术和袖状胃切除术等减肥手术。Roux-en-Y 胃旁路手术后,血清炎症标志物和 C 反应蛋白水平降低,氧化应激指标包括总抗氧化能力、总氧化状态和丙二醛水平显著降低。在袖状胃切除术中,大约 80% 的胃被缩小并转化为管状胃,限制食物的摄入量,从而导致体重减轻,减重手术后 IL-6、CRP 水平降低,体重指数降低。脂肪组织在氧化应激和炎症因子的诱导中起重要作用,因此改善炎症和氧化应激是其改善 CVD 疾病可能的机制[39]。

新开发药物治疗如胰高血糖素样肽-1 (GLP-1)是一种肠道激素,可作为饱腹感信号、刺激胰岛素释放、抑制胰高血糖素分泌并调节为排空。GLP-1 对心血管疾病也有潜在有益作用,包括利钠、利尿、降低血压和改善炎症,利拉鲁肽和索马鲁肽这两种 GLP-1 为肥胖但没有糖尿病的 CVD 患者提供了一种可参考治疗方法[40]。但是代谢肥胖手术和新兴药物虽然具有一定的临床前景,但在当前我国人群中的应用有限,待疾病具体机制明确后,希望可以在临床中得到更多的应用。

5. 总结

肥胖被认为是一种异质性疾病,相似 BMI 的个体可能具有不同的 CVD 风险特征,是 CVD 的重要危险因素,并可导致其他心血管危险因素,如:糖尿病、血脂异常和高血压的发展。肥胖相关的血流动力学、神经激素信号和心肌代谢变化会增加 CAD 和 HF 的风险。未来应侧重于开发有效、安全的干预措施,以降低肥胖症的患病率,提升肥胖患者的整体生活质量。

参考文献

- [1] Safaei, M., Sundararajan, E.A., Driss, M., Boulila, W. and Shapi'i, A. (2021) A Systematic Literature Review on Obesity:

- Understanding the Causes & Consequences of Obesity and Reviewing Various Machine Learning Approaches Used to Predict Obesity. *Computers in Biology and Medicine*, **136**, Article ID: 104754. <https://doi.org/10.1016/j.compbiomed.2021.104754>
- [2] Gebreab, S.Z., Vandeleur, C.L., Rudaz, D., Strippoli, M.F., Gholam-Rezaee, M., Castela, E., *et al.* (2018) Psychosocial Stress over the Lifespan, Psychological Factors, and Cardiometabolic Risk in the Community. *Psychosomatic Medicine*, **80**, 628-639. <https://doi.org/10.1097/psy.0000000000000621>
 - [3] Sommer, I., Griebler, U., Mahlknecht, P., Thaler, K., Bouskill, K., Gartlehner, G., *et al.* (2015) Socioeconomic Inequalities in Non-Communicable Diseases and Their Risk Factors: An Overview of Systematic Reviews. *BMC Public Health*, **15**, Article No. 914. <https://doi.org/10.1186/s12889-015-2227-y>
 - [4] Sallis, J.F. and Glanz, K. (2009) Physical Activity and Food Environments: Solutions to the Obesity Epidemic. *The Milbank Quarterly*, **87**, 123-154. <https://doi.org/10.1111/j.1468-0009.2009.00550.x>
 - [5] (2000) Obesity: Preventing and Managing the Global Epidemic. Report of a WHO Consultation. World Health Organ. Tech. Rep. Ser., 894: i-xii, 1-253.
 - [6] Powell-Wiley, T.M., Poirier, P., Burke, L.E., Després, J., Gordon-Larsen, P., Lavie, C.J., *et al.* (2021) Obesity and Cardiovascular Disease: A Scientific Statement from the American Heart Association. *Circulation*, **143**, e984-e1010. <https://doi.org/10.1161/cir.0000000000000973>
 - [7] Mandviwala, T., Khalid, U. and Deswal, A. (2016) Obesity and Cardiovascular Disease: A Risk Factor or a Risk Marker? *Current Atherosclerosis Reports*, **18**, Article No. 21. <https://doi.org/10.1007/s11883-016-0575-4>
 - [8] Engin, A. (2017) Endothelial Dysfunction in Obesity. In: *Advances in Experimental Medicine and Biology*, Springer International Publishing, 345-379. https://doi.org/10.1007/978-3-319-48382-5_15
 - [9] Kershaw, E.E. and Flier, J.S. (2004) Adipose Tissue as an Endocrine Organ. *The Journal of Clinical Endocrinology & Metabolism*, **89**, 2548-2556. <https://doi.org/10.1210/jc.2004-0395>
 - [10] Nakamura, K., Fuster, J.J. and Walsh, K. (2014) Adipokines: A Link between Obesity and Cardiovascular Disease. *Journal of Cardiology*, **63**, 250-259. <https://doi.org/10.1016/j.jicc.2013.11.006>
 - [11] Ebong, I.A., Goff, D.C., Rodriguez, C.J., Chen, H. and Bertoni, A.G. (2014) Mechanisms of Heart Failure in Obesity. *Obesity Research & Clinical Practice*, **8**, e540-e548. <https://doi.org/10.1016/j.orcp.2013.12.005>
 - [12] Horwich, T.B. and Fonarow, G.C. (2010) Glucose, Obesity, Metabolic Syndrome, and Diabetes Relevance to Incidence of Heart Failure. *Journal of the American College of Cardiology*, **55**, 283-293. <https://doi.org/10.1016/j.jacc.2009.07.029>
 - [13] Thomas, S.Y., Harmancey, R. and Taegtmeyer, H. (2010) Fat around the Heart. *JACC: Cardiovascular Imaging*, **3**, 786-787. <https://doi.org/10.1016/j.jcmg.2010.05.001>
 - [14] Haidar, A. and Taegtmeyer, H. (2022) Strategies for Imaging Metabolic Remodeling of the Heart in Obesity and Heart Failure. *Current Cardiology Reports*, **24**, 327-335. <https://doi.org/10.1007/s11886-022-01650-3>
 - [15] Sharma, S., Adrogue, J.V., Golfman, L., Uray, I., Lemm, J., Youker, K., *et al.* (2004) Intramyocardial Lipid Accumulation in the Failing Human Heart Resembles the Lipotoxic Rat Heart. *The FASEB Journal*, **18**, 1692-1700. <https://doi.org/10.1096/fj.04-2263com>
 - [16] Asghar, O., Alam, U., A. Hayat, S., Aghamohammadzadeh, R., M. Heagerty, A. and A. Malik, R. (2012) Obesity, Diabetes and Atrial Fibrillation; Epidemiology, Mechanisms and Interventions. *Current Cardiology Reviews*, **8**, 253-264. <https://doi.org/10.2174/157340312803760749>
 - [17] Lee, S., Chen, Y., Chen, Y., Chang, S., Tai, C., Wongcharoen, W., *et al.* (2007) Tumor Necrosis Factor- α Alters Calcium Handling and Increases Arrhythmogenesis of Pulmonary Vein Cardiomyocytes. *Life Sciences*, **80**, 1806-1815. <https://doi.org/10.1016/j.lfs.2007.02.029>
 - [18] Karason, K., Mølgaard, H., Wikstrand, J. and Sjöström, L. (1999) Heart Rate Variability in Obesity and the Effect of Weight Loss. *The American Journal of Cardiology*, **83**, 1242-1247. [https://doi.org/10.1016/s0002-9149\(99\)00066-1](https://doi.org/10.1016/s0002-9149(99)00066-1)
 - [19] Jouven, X., Desnos, M., Guerot, C. and Ducimetière, P. (1999) Predicting Sudden Death in the Population: The Paris Prospective Study I. *Circulation*, **99**, 1978-1983. <https://doi.org/10.1161/01.cir.99.15.1978>
 - [20] Csige, I., Ujvárosy, D., Szabó, Z., Lőrincz, I., Paragh, G., Harangi, M., *et al.* (2018) The Impact of Obesity on the Cardiovascular System. *Journal of Diabetes Research*, **2018**, Article ID: 3407306. <https://doi.org/10.1155/2018/3407306>
 - [21] Haslam, D.W. and James, W.P.T. (2005) Obesity. *The Lancet*, **366**, 1197-1209. [https://doi.org/10.1016/s0140-6736\(05\)67483-1](https://doi.org/10.1016/s0140-6736(05)67483-1)
 - [22] Chen, Q., Zhang, Z., Luo, N. and Qi, Y. (2023) Elevated Visceral Adiposity Index Is Associated with Increased Stroke Prevalence and Earlier Age at First Stroke Onset: Based on a National Cross-Sectional Study. *Frontiers in Endocrinology*, **13**, Article ID: 1086936. <https://doi.org/10.3389/fendo.2022.1086936>
 - [23] NCD Risk Factor Collaboration (NCD-RisC) (2021) Worldwide Trends in Hypertension Prevalence and Progress in

- Treatment and Control from 1990 to 2019: A Pooled Analysis of 1201 Population-Representative Studies with 104 Million Participants. *The Lancet*, **398**, 957-980.
- [24] Hall, J.E., do Carmo, J.M., da Silva, A.A., Wang, Z. and Hall, M.E. (2015) Obesity-Induced Hypertension: Interaction of Neurohumoral and Renal Mechanisms. *Circulation Research*, **116**, 991-1006. <https://doi.org/10.1161/circresaha.116.305697>
- [25] Lambert, E.A., Straznicki, N.E., Dixon, J.B. and Lambert, G.W. (2015) Should the Sympathetic Nervous System Be a Target to Improve Cardiometabolic Risk in Obesity? *American Journal of Physiology-Heart and Circulatory Physiology*, **309**, H244-H258. <https://doi.org/10.1152/ajpheart.00096.2015>
- [26] Cardillo, C., Campia, U., Iantorno, M. and Panza, J.A. (2004) Enhanced Vascular Activity of Endogenous Endothelin-1 in Obese Hypertensive Patients. *Hypertension*, **43**, 36-40. <https://doi.org/10.1161/01.hyp.0000103868.45064.81>
- [27] Chrysant, S.G. (2019) Pathophysiology and Treatment of Obesity-Related Hypertension. *The Journal of Clinical Hypertension*, **21**, 555-559. <https://doi.org/10.1111/jch.13518>
- [28] DeMarco, V.G., Aroor, A.R. and Sowers, J.R. (2014) The Pathophysiology of Hypertension in Patients with Obesity. *Nature Reviews Endocrinology*, **10**, 364-376. <https://doi.org/10.1038/nrendo.2014.44>
- [29] Lewis, E.J., Hunsicker, L.G., Clarke, W.R., Berl, T., Pohl, M.A., Lewis, J.B., *et al.* (2001) Renoprotective Effect of the Angiotensin-Receptor Antagonist Irbesartan in Patients with Nephropathy Due to Type 2 Diabetes. *New England Journal of Medicine*, **345**, 851-860. <https://doi.org/10.1056/nejmoa011303>
- [30] Schlueter, N., de Sterke, A., Willmes, D.M., Spranger, J., Jordan, J. and Birkenfeld, A.L. (2014) Metabolic Actions of Natriuretic Peptides and Therapeutic Potential in the Metabolic Syndrome. *Pharmacology & Therapeutics*, **144**, 12-27. <https://doi.org/10.1016/j.pharmthera.2014.04.007>
- [31] Moro, C. (2013) Natriuretic Peptides and Fat Metabolism. *Current Opinion in Clinical Nutrition and Metabolic Care*, **16**, 645-649. <https://doi.org/10.1097/mco.0b013e32836510ed>
- [32] Lavie, C.J., Arena, R., Alpert, M.A., Milani, R.V. and Ventura, H.O. (2017) Management of Cardiovascular Diseases in Patients with Obesity. *Nature Reviews Cardiology*, **15**, 45-56. <https://doi.org/10.1038/nrcardio.2017.108>
- [33] Elagizi, A., Kachur, S., Lavie, C.J., Carbone, S., Pandey, A., Ortega, F.B., *et al.* (2018) An Overview and Update on Obesity and the Obesity Paradox in Cardiovascular Diseases. *Progress in Cardiovascular Diseases*, **61**, 142-150. <https://doi.org/10.1016/j.pcad.2018.07.003>
- [34] Koliaki, C., Liatis, S. and Kokkinos, A. (2019) Obesity and Cardiovascular Disease: Revisiting an Old Relationship. *Metabolism*, **92**, 98-107. <https://doi.org/10.1016/j.metabol.2018.10.011>
- [35] Horwich, T.B., Fonarow, G.C. and Clark, A.L. (2018) Obesity and the Obesity Paradox in Heart Failure. *Progress in Cardiovascular Diseases*, **61**, 151-156. <https://doi.org/10.1016/j.pcad.2018.05.005>
- [36] Haidar, A. and Horwich, T. (2023) Obesity, Cardiorespiratory Fitness, and Cardiovascular Disease. *Current Cardiology Reports*, **25**, 1565-1571. <https://doi.org/10.1007/s11886-023-01975-7>
- [37] Tutor, A., Lavie, C.J., Kachur, S., Dinshaw, H. and Milani, R.V. (2022) Impact of Cardiorespiratory Fitness on Outcomes in Cardiac Rehabilitation. *Progress in Cardiovascular Diseases*, **70**, 2-7. <https://doi.org/10.1016/j.pcad.2021.11.001>
- [38] DeFina, L.F., Haskell, W.L., Willis, B.L., Barlow, C.E., Finley, C.E., Levine, B.D., *et al.* (2015) Physical Activity versus Cardiorespiratory Fitness: Two (Partly) Distinct Components of Cardiovascular Health? *Progress in Cardiovascular Diseases*, **57**, 324-329. <https://doi.org/10.1016/j.pcad.2014.09.008>
- [39] Abulmeaty, M.M.A., Ghneim, H.K., Alkhathaami, A., Alnumair, K., Al Zaben, M., Razak, S., *et al.* (2023) Inflammatory Cytokines, Redox Status, and Cardiovascular Diseases Risk after Weight Loss via Bariatric Surgery and Lifestyle Intervention. *Medicina*, **59**, Article No. 751. <https://doi.org/10.3390/medicina59040751>
- [40] Ryan, D.H., Lingvay, I., Colhoun, H.M., Deanfield, J., Emerson, S.S., Kahn, S.E., *et al.* (2020) Semaglutide Effects on Cardiovascular Outcomes in People with Overweight or Obesity (SELECT) Rationale and Design. *American Heart Journal*, **229**, 61-69. <https://doi.org/10.1016/j.ahj.2020.07.008>