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心外膜脂肪组织与心血管疾病的研究进展 *

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摘要 心外膜脂肪组织(epicardial adipose tissue，EAT)是一种特殊的具有局部和全身效应的多功能脂肪组织，其解剖位置特殊，代谢和组织学特征明显区别于其他脂肪组织．在生理条件下，EAT 具有产热和保护心脏的作用；而在病理状态下，EAT 通过分泌多种促炎细胞因子 / 脂肪因子，参与心血管疾病(cardiovascular disease，CVD)的发生发展．EAT 的厚度 / 体积及其引发的慢性炎症反应与 CVD 的严重程度呈显著正相关，运动、减轻体重和药物等均可恢复 EAT 对心血管的保护作用，提示其有望成为 CVD 诊断、治疗和预后评价的指标．本文通过对 EAT 的特征、功能、调节机制以及在血管损伤后重构、动脉粥样硬化、高血压病、心律失常、心功能不全等 CVD 中的作用做一综述，以期为 CVD 的防治提供新靶点．

关键词 心外膜脂肪组织，心血管疾病，血管外膜，炎症，巨噬细胞
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肥胖引发的多种慢性疾病严重威胁人类生命健康，流行病学调查显示，中国 35～64 岁人群肥胖率已达 20.2%，而与肥胖相关的心血管疾病(cardiovascular disease，CVD)的致残率和致死率也急剧上升[1]．心外膜脂肪组织(epicardial adipose tissue，EAT)是心脏的内脏脂肪组织，由于其特殊的解剖位置以及特有的代谢特性和临床可观测性，目前已成为肥胖相关 CVD 风险评价和防治研究的热点．本文通过对 EAT 的特征、功能、调节机制以及在 CVD，如血管损伤后重构、动脉粥样硬化、高血压病、心律失常、心功能不全等中的作用做一综述，为 CVD 的防治提供新的思路．

1 EAT 结构及功能

EAT 是指来源于脏层中胚层并沉积在心脏外膜的内脏脂肪组织，主要位于心脏房室沟和室间沟区域，可从心外膜表面扩张至心肌，与冠脉分支共享血供[2]．EAT 主要由脂肪细胞构成，但同时也存在间质血管细胞、炎症 / 免疫细胞、神经节等[3]．在生理条件下，EAT 约占心脏质量的 20%，覆盖 80%的心脏表面．EAT 和冠脉 / 心肌之间不存在解剖屏障(无筋膜)，因此其分泌的生物活性物质可通过旁分泌 / 自分泌途径直接作用于冠脉 / 心肌[3]．EAT 通常被认为属于白色脂肪组织，然而最新研究显示，EAT 脂肪细胞主要为线粒体棕色脂肪解偶联蛋白 1(uncoupling protein 1，UCP1)阳性的小单房脂肪细胞，UCP1 是唯一在棕色脂肪组织中表达并通过参与产热和调节能量代谢维持机体能量稳态的解偶联蛋白质，表明 EAT 具有棕色或浅棕色脂肪组织特征，这与皮下脂肪组织和大网膜脂肪组织存在明显不同[4](表 1)．

生理条件下，EAT 具有产热、代谢和保护冠脉 / 心肌的作用[3]．研究证实，EAT 中 UCP1 阳性小单房脂肪细胞在寒冷 / 体温降低等应激条件下可产生大量的热量，维持心脏体温及其功能稳态[4]．当体内游离脂肪酸(free fatty acids，FFAs)水平过高

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时，EAT 可通过其 FFAs 摄取能力调节心肌脂肪酸代谢平衡，保护冠脉 / 心肌[5]. 作为内分泌器官，EAT 还可分泌多种保护性脂肪 / 细胞因子，如脂联素(adiponectin, APN)、肾上腺髓质素(adrenomedullin, ADM)、外膜源性舒张因子及白介素 10 (interleukin-10，IL-10)等，从而舒张血管、减轻炎症反应、调节冠脉 / 心肌功能[6-7]. 然而，病理状态下 EAT 表型及功能发生明显改变，UCP1 显著减少，脂肪细胞体积增大并伴有炎症反应的增加，EAT 由棕色脂肪组织逐渐转变为白色脂肪组织，导致 EAT 功能失衡，并且，在此转化过程中线粒体含量明显减少，提示代谢水平的改变可能是引发 EAT 表型转化的重要环节[8]. 5′单磷酸腺苷活化蛋白激酶(AMP-activated protein kinase，AMPK)是维持细胞代谢平衡的关键蛋白酶，研究发现，肥胖状态下 EAT 的 AMPK 活性受到显著抑制，脂肪酸氧化(fatty acid oxidation，FAO)水平明显下降，有氧糖酵解(aerobic glycolysis，AG)活性显著增加，组织内饱和脂肪酸大量蓄积，提示调控 AMPK 活性可能是 EAT 功能调节的一条重要途径[9-11]. 在 EAT 结构 / 功能改变过程中，保护性脂肪因子的产生和分泌显著下调，损伤性脂肪因子如瘦素、内脂素等的表达显著上调，促炎因子如肿瘤坏死因子 α (tumor necrosis factor-α，TNF-α)、单核细胞趋化因子 1(monocyte chemotactic protein-1，MCP-1)、白介素 1β (interleukin-1β，IL-1β) 和白介素 6 (interleukin-6，IL-6)的表达也明显增加，并通过自分泌 / 旁分泌途径进入冠脉 / 心肌，参与其组织损伤[3, 12](表 2).

Table 1　Characteristics of different adipose tissue

表 1　不同脂肪组织特征

区别	心外膜脂肪组织	皮下脂肪组织	大网膜脂肪组织	参考文献
来源	中胚层	神经脊、中胚层	神经脊、中胚层	[3, 13-15]
含量	约心脏质量的 20%	约体内脂肪总量的 85%	-	[16-17]
血液供应	冠状动脉分支血供	表浅静脉	胃网膜左右动脉	[18-20]
解剖位置	心肌 / 冠状动脉外周	真皮层以下，筋膜层以上	胃 / 横结肠周围	[21-22]
组织类型	棕色脂肪组织	白色脂肪组织	白色脂肪组织	[3-4, 16]
细胞形态	小而紧密	较大而疏松	较大而疏松	[23-24]
代谢特点	脂肪酸代谢显著高于其他脂肪组织	脂肪酸代谢明显低于 EAT	脂肪酸代谢高于皮下脂肪组织	[2, 21, 24]
分泌功能	分泌大量的 APN、ADM	瘦素、脂联素的主要来源	抵抗素	[7, 24-25]
生理功能	机械性保护心脏 / 冠脉	贮量、填充、维持体温	缓冲	[3, 16]
炎症反应	巨噬细胞大量浸润、促炎因子显著增加，炎症反应较为明显	巨噬细胞浸润及促炎因子分泌较少，炎症反应显著低于 EAT	含有大量免疫 / 炎症细胞，与皮下脂肪相比可分泌大量的促炎因子	[6, 21, 24]
相关疾病	心血管疾病	脂肪营养不良疾病	肥胖、消化道肿瘤、2 型糖尿病	[26-30]

Table 2　The function of EAT under different condition

表 2　不同状态下 EAT 的功能变化

功能	生理状态	病理状态	参考文献
产热功能	含有大量 UCP1，类似于棕色脂肪组织，提供热量，维持能量稳态	UCP1 减少，棕色脂肪组织向白色脂肪组织转变，产热量降低，能量稳态失衡	[4, 8]
代谢功能	具有较高的 FFAs 释放和摄取能力，保护冠脉 / 心肌	脂肪酸大量蓄积，细胞 / 组织病理性增大、沉积，损伤冠脉 / 心肌	[5, 9-11]
分泌功能	抗炎脂肪因子 APN、ADM 分泌增加	促炎脂肪因子瘦素、内脂素分泌增加	[3, 6-7, 12]
免疫调节功能	免疫细胞产生大量的抗炎因子如 IL-10、IL-4	单核 / 巨噬细胞大量浸润，促炎因子 TNF-α、IL-1β、IL-6 等增加	[18, 31]

2　EAT 病理功能的调节

巨噬细胞是 EAT 的主要免疫 / 炎症细胞，在 EAT 功能调节中起着关键作用. 对于局部刺激，心外膜脂肪组织巨噬细胞(epicardial adipose tissue macrophage，EATM)呈现出不同极化表型并执行不

同的调节功能，主要分为两大类亚群：促炎 M1 型和抗炎 M2 型[11]. 促炎 M1 型产生大量促炎因子 TNF-α、IL-1β、MCP-1 等引发机体炎症反应；抗炎 M2 型产生 IL-10 等抗炎因子参与炎症消除和组织修复. 研究者发现，肥胖患者 EAT 中巨噬细胞大量浸润、M1 型巨噬细胞增加、M2 型巨噬细胞减少[11]，其 M1/M2 比例增加与 EAT 功能紊乱密切相关[32]，该效应可能与 M1 型巨噬细胞 Toll 样受体 2/4(Toll-like receptor 2/4，TLR-2/4)介导分泌的促炎细胞因子，如 TNF-α、IL-1β 等，参与 EAT 脂肪细胞肥大 / 变性有关[33](图 1).

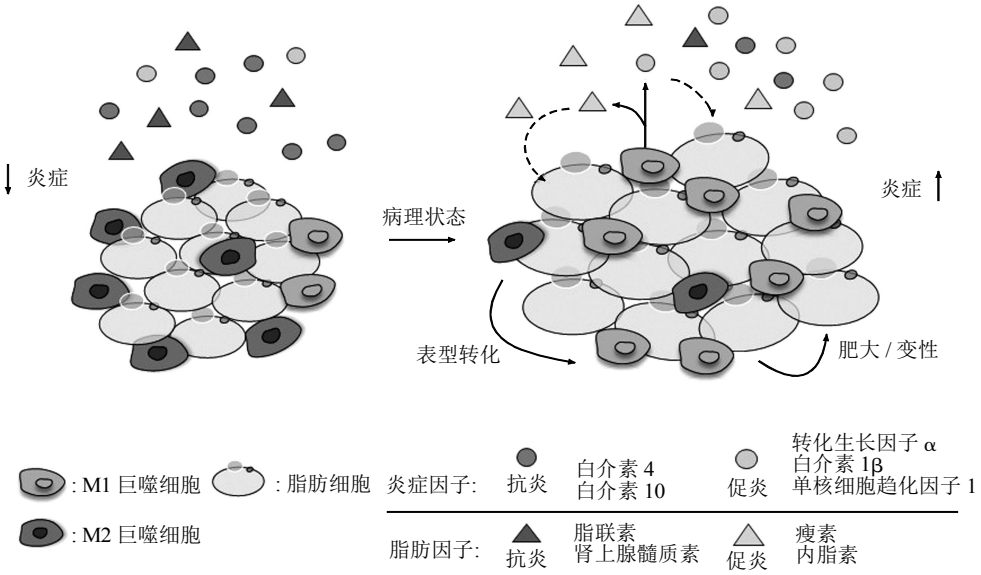

Fig. 1　The regulation role of macrophage to the EAT

图 1　巨噬细胞对 EAT 的功能调节

EAT 功能调节具有可逆性，运动、减轻体重和药物治疗等手段均可显著降低 EAT 的厚度 / 体积及其功能紊乱[34-36]. 研究显示，运动 / 减轻体重可有效减轻肥胖患者房室间沟 EAT 厚度，并改善 EAT 促炎功能，缓解体内胰岛素抵抗状态[34-36]. Park 等[37]发现他汀类药物可显著降低 EAT 的炎症反应及厚度，并抑制经皮冠状动脉介入(percutaneous coronary intervention，PCI)术后再狭窄. 噻唑烷二酮[38]、血管紧张素Ⅱ1 型受体拮抗剂[39]、胰高血糖素样肽 1 受体激动剂、二肽基肽酶 4 抑制剂等药物均被发现能够调节 EAT 厚度及其炎症反应，改善动脉粥样硬化(atherosclerosis，As)、糖尿病等疾病的发生发展. 近来的大量研究证实 EAT 表型转化在其功能调节过程中发挥着重要的作用，但上述调节因素在改善 EAT 功能过程中是否促进 EAT 棕色化仍有待进一步研究. 本课题组前期发现高密度脂蛋白(high-density lipoprotein，HDL)及其主要成分载脂蛋白 A1(apolipoprotein A1，apoA-1)能够显著抑制巨噬细胞炎症反应[40]，Umemoto 等[41]发现 HDL/apoA-1 能够显著抑制脂肪组织炎症反应，提示 HDL/apoA-1 可能通过调控 EATM 炎症参与对 EAT 功能的调节，从而抑制心血管疾病的发生发展[42]. 近来研究发现，维生素 D (vitamin D, VD)对 EAT 炎症具有重要的调节作用. Agrawal 等[43]在高胆固醇血症小型猪模型中发现，VD 缺乏可明显促进 EAT 炎症，从而导致心肌肥厚. 在冠心病患者，Dozio 等[44]也发现 VD 水平与 EAT 炎症程度和冠心病严重程度呈显著负相关，提示 VD 是 EAT 功能重要的调控因素. 本课题组前期发现 VD 能够显著抑制 As 斑块中巨噬细胞促炎 M1 极化[45]，提示 VD 具有调控 EATM 促炎极化的效应，其机制还有待进一步明确.

3　EAT 与心血管疾病

3.1　EAT 与血管损伤后重构

血管损伤后重构是指血管在受到各种内外部因素(损伤、疾病、老化等)刺激后，血管壁组织结构 / 功能发生病理性改变的过程. PCI 术后再狭窄是血管损伤后重构的重要类型，其病理过程具有组织损伤后修复的特征[46]. 尽管药物支架的使用一定程

度降低了支架内再狭窄的发生，但药物支架再狭窄率仍然达到 8%～10%，尤其在肥胖患者中其再狭窄率明显升高[46-47]，提示支架内再狭窄的机制还有待进一步明确．近来研究发现血管外膜在血管损伤后重构中起着关键的作用[48-49]．在颈动脉球囊损伤、主动脉移植动物模型中，研究者均发现在血管损伤早期外膜成纤维细胞增殖 / 分化先于内皮的功能异常，并伴有大量的巨噬细胞浸润及炎症因子表达[50-51]．由于 EAT 与血管外膜不存在任何解剖屏障，研究认为肥胖状态下 EAT 的功能紊乱可能参与了血管外膜炎症“微环境”的变化，从而参与 PCI 术后再狭窄的发生发展．Park 等[52]发现肥胖患者 EAT 厚度明显增加，并与 PCI 术后再狭窄显著正相关．Fukamachi 等[53]证实急性心肌梗死(acute myocardial infarction，AMI)患者接受 PCI 治疗两周后，多支血管病变患者 EAT 厚度明显高于单支血管病变患者，并且，EAT 厚度可作为多支血管病变的唯一独立预测因素．Zencirci 等[54]发现 PCI 术后心肌组织无灌注或灌注不良现象与 EAT 厚度呈正相关，提示降低 EAT 厚度 / 体积有助于 PCI 术后冠脉复流，并抑制血管再狭窄．EAT 调控 PCI 术后血管重构的机制还有待进一步明确，研究证实肥胖状态下 EAT 中 M1 型巨噬细胞显著增多，炎症因子的表达明显升高[33, 55]．上述结果表明，利用 EAT 功能调节的可逆性，经药物及基因治疗等一系列治疗手段靶向干预 EAT，从而改善血管外膜炎症状态 / 功能紊乱可能是临床防治 PCI 术后再狭窄的新途径[42]．

3.2 EAT 与动脉粥样硬化

动脉粥样硬化(atherosclerosis，As)是好发于大、中动脉的慢性炎症病变，以病变区域脂质蓄积和免疫细胞大量浸润为主要特点．Harada 等[32]发现在急性冠脉综合症患者中，EAT 体积可作为预测富含脂质斑块的独立风险因素，并与富含脂质斑块所占斑块比值呈显著正相关．Sag 等[56]发现 EAT 体积与颈动脉内膜 - 中层厚度(carotid intima- media thickness，CIMT)呈显著正相关，并独立于其他危险因素．在小型猪 As 模型，研究者发现切除冠状动脉左前降支部位 EAT，可明显减少 As 斑块形成[57]．由于 EAT 能够被常规的影像手段观测，上述结果提示 EAT 厚度可作为冠心病(coronary atherosclerotic heart disease，CAD)诊断和危险分层的新指标．EAT 致 As 的机制主要与其炎症及代谢状态有关，研究者对 CAD 患者 EAT 进行检测，发现 EAT 中巨噬细胞 M1 促炎表型增多，并与 CAD 严重程度显著正相关[58-60]．研究还发现，病理状态下 EAT 可通过分泌基质金属蛋白酶(matrix metalloproteinases，MMPs)进入血管外膜、中膜、内膜，促进 As 斑块基质降解，增加 As 斑块易损风险[5, 22, 61]．研究证实，高糖及炎症反应可有效降低脂肪形成诱导的 EAT 基质细胞脂联素的分泌，但这一过程在成熟的脂肪细胞中显著减少，提示靶向作用于 EAT 脂肪形成期的炎症反应将为 As 防治提供新靶点[62]．Vacca 等[63]发现 CAD 患者 EAT 脂代谢水平、氧化磷酸化、线粒体功能、核受体转录因子(过氧化物酶体增殖物激活受体(peroxisome proliferator activated receptors，PPARs)、肝 X 受体(liver X receptors，LXRs)、胆固醇调节元件结合蛋白 (sterol-regulatory element binding proteins 1，SREBP-1)等)活性明显受到抑制，但抗原递呈，趋化信号及炎症因子(单核细胞趋化因子 1(monocyte chemotactic protein-1， MCP-1)、白介素 6 (interleukin-6， IL-6)、血管内皮生长因子 C (vascular endothelial growth factor C，VEGF-C)等)的表达水平显著升高，提示 EAT 炎症反应与代谢途径可能存在某种潜在调节机制并进一步作用于 CAD 的发生发展．As 作为 CVD 主要的病理基础，其形成包括多种病理过程(如内皮功能异常、泡沫化细胞形成、平滑肌细胞增殖等)，研究已证实 EAT 病理 / 功能改变与 As 发展进程相关(图 2)．传统的观念认为血管炎症始于内膜，近来越来越多证据表明血管外膜炎症反应先于内膜病变并在 As 中发挥重要作用[64]，然而 EAT 通过作用于血管外膜影响 As 病理形成过程的具体机制仍有待进一步研究．

3.3 EAT 与高血压病

高血压病(hypertension，HTN)作为临床常见的慢性病，以动脉收缩压和 / 或舒张压升高为主要病理表现，血压升高可造成多种慢性靶器官(脑、血管、心脏、肾脏等)损害．近来大量研究证实，EAT 可作为 HTN 的独立预测指标，调节 HTN 及其并发症的发生发展[65]．Ruan 等[66]在 DOCA-salt 诱导的 HTN 小鼠模型中发现，随着血管外周脂肪组织(perivascular adipose tissue，PVAT)中 M1 型巨噬细胞比例的升高，HTN 引起的血管重构显著增加，提示 EAT 作为一种特殊的 PVAT，EATM 促炎表型的增加将进一步促进 HTN 的发生发展．临床研究发现，肥胖 HTN 患者血压昼夜节律变化明显高

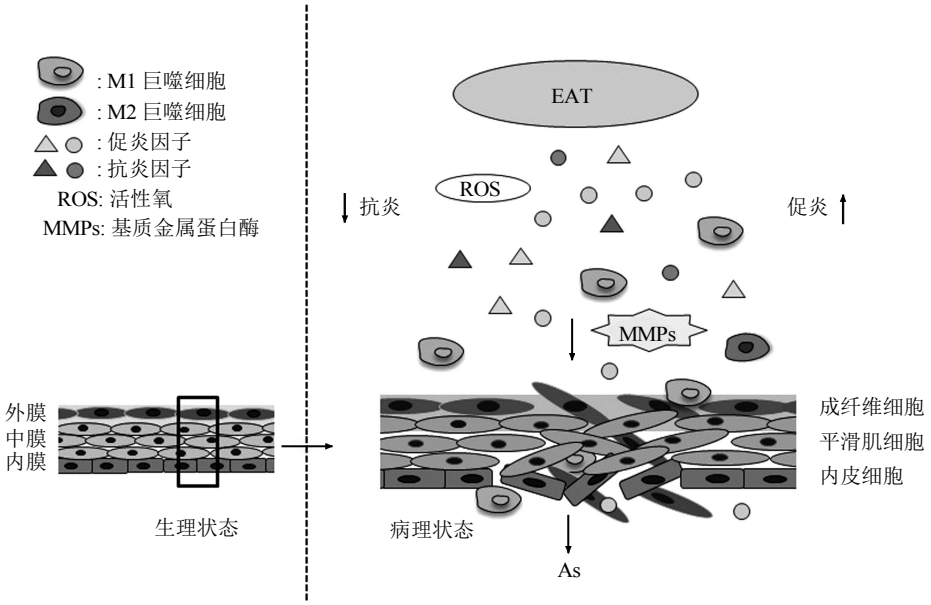

Fig. 2　The role of EAT to regulate As

图 2　EAT 对 As 的调节作用

于普通 HTN 患者，并与 EAT 厚度相关[67-69]．同时，最近研究发现 EAT 体积可作为原发性高血压患者血压昼夜节律的独立预测因子[70]．Erdogan 等[71]发现，顽固性高血压(resistant hypertension，RHTN)患者 EAT 厚度与血压水平呈显著正相关，其中 EAT 厚度达到 3.42 cm 及以上可作为判定 RHTN 的最佳指标(82%敏感性、77%特异性)．Ertas 等还发现，EAT 体积与勺型及非勺型血压曲线显著相关，并独立于其他危险因素，EAT 体积大于或等于 7 mm 可作为判定非勺型 HTN 的独立性指标(74%敏感性，71%特异性)[67-69]．EAT 促 HTN 机制可能主要与 EAT 厚度 / 体积增加导致动脉顺应力(arterial compliance，AC)降低，动脉硬化指标心 - 踝血管指数(cardio-ankle vascular index，CAVI)升高，致使升主动脉扩张，主动脉弹性功能受损有关[72-74]．HTN 由于其诱因的多样性，血压控制达标率一直较低，并且对于顽固性高血压的治疗仍存在一定难度，对 EAT 调控 HTN 机制的深入研究将为 HTN 的防治提供新思路．最近，国内高平进课题组发现 PVAT 巨噬细胞促炎 M1 极化明显促进 HTN 诱导的肾血管重构，然而 PVAT 和 EAT 是否参与 HTN 诱导的心血管重构还有待进一步研究[66].

3.4　EAT 与心律失常

心律失常(cardiac arrhythmia，CA)，尤其是室性心动过速、心房 / 室颤动等恶性心律失常不仅加重原有心脏疾病，还可诱发心源性猝死．近来研究发现，EAT 可作为独立危险因素，参与心房颤动(atrial fibrillation，AF)等 CA 的发生发展[75]．Mazurek 等[76]通过 FDG-PET / 计算机断层扫描发现，AF 患者左心房、房室沟及左主动脉 EAT 含量显著增加，并且上述部位的 EAT 炎性反应水平也明显高于其他皮下 / 内脏组织．Hatem 等[77]发现，病理状态下 EAT 可分泌大量的活化素 A(转化生长因子 β 家族成员)与 MMPs，两者表达的增加均可进一步促进心肌纤维化，加速 AF 进程．Akyel 等[78]发现 EAT 厚度与 AF 患者心房电机械延迟也呈显著正相关，进一步促进 AF 的发生发展．Iacobellis 等[79]发现，持续性 AF 患者 EAT 厚度明显高于阵发性 AF 患者，减轻肥胖患者 EAT 体积可有效维持窦性心律稳态，改善 AF 进程[80]．Nakatani 等[81]对非阵发性 AF 患者行环肺静脉电隔离术后，利用高主频(high dominant frequency，HDF)及持续性碎裂(complex fractionated activation electrogram，CFAE)电位对 AF 患者标测，发现 AF 组左心房 EAT 处 HDF 电位的分布明显低于窦性心律组，提示 EAT 导致左心房 HDF 电位紊乱可能是促进 AF 发生发展的重要因素．Kocyigit 等[82]证实心房周 EAT 厚度也可作为肺静脉隔离术后房颤复发的独立预测因子．同时，研究发现，左心房 EAT 厚度可独立预测 AF 消融术后早期复发，但对晚期复发并无显著

影响，提示针对消融术后早期阶段对 EAT 的治疗可有效作用于心律失常[83]. 综上所述，EAT 促 AF 机制可能是由于病理状态下 EAT 产生大量炎症因子和损伤性脂肪因子，导致心房肌层纤维化，引起心电极化 / 传导紊乱，从而诱导 AF 的发生发展.

3.5 EAT 与心功能不全

心功能不全(heart failure，HF)是各种 CVD 发展的终末阶段，以心肌收缩力下降不能满足机体代谢需要为主要表现，具有不可逆性. 近来研究发现，除了心肌损伤、心脏前后负荷增加等因素外，炎症反应、内分泌紊乱等也参与了 HF 的发生发展. Liu 等[84]研究发现，当心脏受到损伤刺激时，心外膜细胞又可进一步转变为脂肪细胞，从而促进损伤部位 EAT 的形成，参与 HF 的发生发展. 研究发现，左心室舒张功能不全患者 EAT 明显增厚，其体积与舒张功能不全的等级呈线性正相关，并独立于其他危险因素，提示 EAT 可作为预测左心室舒张功能不全的独立性指标[85-86]. Iacobellis 等[87]发现，肥胖患者 EAT 厚度与右心室舒张末期内径均显著增加，并且随着 EAT 厚度的增加右心室出现明显的肥厚. Agra 等[7]发现，HF 患者 EAT 厚度及其炎症因子的分泌显著增加，而保护性脂肪因子 APN 明显减少. 临床研究发现，肥胖人群 EAT 厚度与其血清 FFAs 水平明显相关，EAT 厚度增加引起的心脏局部脂质蓄积可能是导致左心功能不全的主要因素[88]. Fosshaug 等[89]发现，HF 患者 EAT 的 IL-6、肾上腺髓质素、游离脂肪酸结合蛋白 3 以及单不饱和脂肪酸与棕榈油酸等的表达明显升高，EAT 的棕榈油酸水平与心肌重构参数左室舒张末期内径和 N 末端 B 型利钠肽前体的增加呈正相关，促进 HF 患者心肌重构. Burgeiro 等[90]发现，HF 患者 EAT 葡萄糖摄取及脂质分解能力明显降低皮下脂肪组织，其脂质分解能力与脂质分解 / 蓄积因子、炎症因子的表达显著相关，并且提示 EAT 糖脂代谢紊乱可能是 HF 患者重要的调控靶点. 同时，Parisi 等[91]提出，在收缩性 HF 患者中，EAT 厚度明显增加并进一步促进儿茶酚胺水平及儿茶酚胺合成酶的表达，从而导致心肌肾上腺素水平的增加，提示 EAT 有可能通过调控心肌交感神经兴奋作用于 HF 的发生发展. 目前，对于 HF 治疗仍以对症治疗为主，不能有效减缓其进行性恶化，以 EAT 为切入点，在炎症、代谢、神经等方面对 HF 的发病机制进一步探索，为 HF 的治疗提供新机制.

4 问题与展望

EAT 作为一种独特的具有局部和全身效应的多功能内脏脂肪组织，直接或间接地参与多种 CVD 的发生发展. 由于 EAT 具有独特的解剖位置，采用常规影像学方法，如超声心动图、CT 及磁共振等可对其厚度 / 体积进行评估，这为临床 CVD 的诊断、预后及危险分层提供了新的途径. 运动、减轻体重及药物治疗等可有效降低 EAT 厚度 / 体积，改善 EAT 炎症状态和代谢紊乱，恢复其对冠脉 / 心肌的保护作用. 但目前对于 EAT 的组织学特点、功能的调控机制以及 EAT 参与冠脉 / 心肌病理生理过程的具体作用机制尚不十分清楚，对其机制的深入探讨将为 CVD 的防治提供新的思路和靶点.

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Research Advances of Epicardial Adipose Tissue and Cardiovascular Disease*

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Abstract Epicardial adipose tissue (EAT) is a unique fat depot which has multifaceted effect on local and systemic. EAT has a special anatomical location. The metabolic and histological features are distinguished from other fat depots. Under normal physiological conditions, EAT has thermogenic and mechanical characteristics, which is beneficial for the heart. Under pathological conditions, however, EAT secrete a variety of proinflammatory cytokinesis and adipokines involved in the pathological process of cardiovascular diseases. Thickness/volume and chronic inflammation of EAT was significantly positively correlated with the severity of cardiovascular disease. Exercise, weight loss and some drugs can restore the protective functions of EAT for cardiovascular system, suggesting that EAT maybe serve as a novel indicator for diagnosis, treatment and prognostic evaluation of cardiovascular disease. In this review, we summary the anatomy, function and regulation of EAT, and then discuss the potential roles of the EAT in cardiovascular diseases including vascular remodeling after injury, atherosclerosis, hypertension, arrhythmias and heart failure, which may provide new targets for the prevention and treatment of cardiovascular diseases.

Key words epicardial adipose tissue, cardiovascular disease, adventitia, inflammation, macrophages

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