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·综述·

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脂肪组织氧化应激与运动干预

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摘要 脂肪组织在调控代谢稳态和运动适应中扮演着重要的角色。肥胖引起的脂肪组织氧化应激是2型糖尿病与代谢综合征等的重要病理特征,是促进脂肪组织炎症和胰岛素抵抗的重要机制。氧化应激可以引起脂肪细胞趋化因子表达,募集炎症细胞浸润脂肪组织,炎症细胞分泌大量的炎症因子,并促进了局部和系统的胰岛素抵抗与慢性炎症。运动对肥胖相关的慢性代谢病的有效干预与运动的抗氧化效应相关。本文总结了氧化应激在脂肪组织炎症和胰岛素抵抗中的作用,以及运动对脂肪组织氧化应激的调控。

关键词 运动; 脂肪组织; 氧化应激; 胰岛素抵抗; 慢性炎症
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Oxidative Stress of Adipose Tissues and Exercise Intervention

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Abstract Adipose tissues play important roles in metabolic homeostasis and exercise adaptation. Obesity-induced oxidative stress in adipose tissues is one of the main pathological features of type 2 diabetes, metabolic syndrome, as well as an important risk factor inflammation and insulin resistance in adipose tissues. Oxidative stress induces the expression of chemokines, which recruit monocytes/macrophages into adipose tissues. Pro-inflammatory cytokines secreted by pro-inflammatory immune cells contribute a lot to local and systemic insulin resistance and inflammation. The positive effect of exercise on obesity-related chronic metabolic diseases is closely associated with anti-oxidative effects induced by exercise. This paper reviews recent studies about the role of oxidative stress in adipose tissue inflammation, insulin resistance as well as the regulation of oxidative stress in adipose tissues by exercise.

Key words exercise; adipose tissue; oxidative stress; insulin resistance; chronic inflammation

脂肪组织在调控运动生理适应中扮演重要的角色[1]。长期运动训练能引起脂肪组织产生积极显型适应,细胞水平体现在脂肪细胞体积减小、脂滴含脂量下降、细胞表型转化等;分子水平体现在线粒体数量增加、代谢酶活性提高等;组织水平体现在脂肪总量下降和重新分布等方面,这对机体代谢稳态调控有重要意义[2]。高热量饮食、长期久坐等生活方式的转变,大大提高了慢性代谢疾病的风险。脂肪沉积与胰岛素抵抗、2型糖尿病(type 2 diabetes mellitus, T2DM)、心血管疾病(cardiovascular disease, CVD)、代谢综合征(metabolic syndrom, MetS)等高度相关。随着脂肪细胞肥大,活性氧(reactive oxygen species, ROS)产生增加。清除活性氧能改善脂肪细胞胰岛素敏感性,提示氧化应激是引起胰岛素抵抗的重要因素[3]。氧化应激能激活炎症信号通路,体外棕榈酸和高糖环境下脂肪细胞肿瘤坏死因子(tumor necrosis factor α, TNF-α)、单核细胞趋化蛋白(monocyte chemoattractant protein 1, MCP-1)、白细胞介素6(interleukin 6, IL-6)等炎症

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因子表达提高,与活性氧激活转录因子核因子 κB(nuclear factor κB, NF-κB)有关,N-乙酰半胱氨酸(N-acetylcysteine, NAC)、过氧化氢酶(catalase, CAT)、超氧化物歧化酶(superoxide dismutase, SOD)等抗氧化物均能抑制 NF-κB,降低炎症因子表达[4]。规律性运动,尤其是耐力训练能改善脂肪组织氧化应激,在降低脂肪组织炎症因子表达中发挥重要作用,近几年发现高强度间歇训练同样能改善氧化应激,降低炎症[5]。本文总结了脂肪组织氧化应激与慢性炎症和胰岛素抵抗的关系,以及不同形式运动、热量限制对脂肪组织氧化应激的影响。

1　肥胖、氧化应激与胰岛素抵抗

1.1　肥胖与氧化应激

肥胖提高细胞应激水平,影响大量细胞信号通路,包括胰岛素信号通路。氧化应激需要特别关注,肥胖中系统氧化应激水平提高,其来源主要就是脂肪组织[6]。生理水平的活性氧对调控细胞、组织乃至整个机体的适应应答不可或缺,长期过量的活性氧会造成细胞生理功能紊乱。活性氧能攻击生物膜脂质双分子层的不饱和脂肪酸,引起脂质过氧化,产生 4-羟基壬烯醛(4-hydroxynoneal, 4-HNE)、4-氧壬烯醛(4-oxynonylaldehyde, 4-ONE)和丙二醛(malonaldehyde dehydrogenase, MDA)等产物。肥胖小鼠的内脏脂肪 4-HNE 能提高 5 ~ 11 倍[7]。4-HNE 能降低脂肪细胞胰岛素受体磷酸化和下游的磷脂酰肌醇(-3)激酶(phosphatidylinositol 3-kinase, PI3K)、蛋白激酶 B(protein kinase B, Akt)活性[8]。活性氧还引起蛋白质产生不可逆的羰基化,脂质过氧化引起的二次羰基化比直接羰基化存在更为普遍。肥胖小鼠脂肪组织蛋白质羰基化总体提高[9]。肥胖小鼠脂肪组织中 4-HNE 的主要降解酶脂肪醛脱氢酶(fatty aldehyde dehydrogenase, FALDH)、谷胱甘肽-S 转移酶 A4(glutathione S-transferase alpha4, GSTA4)表达显著下降[8,10]。活性氧还能引起 DNA 碱基修饰或断裂。肥胖动物内脏脂肪 DNA 损伤标志物 8-羟化脱氧鸟苷(8-hydroxy-2 deoxyguanosine, 8-OHdG)显著升高,DNA 损伤修复因子 p53 被激活,这是引起脂肪组织炎症和胰岛素抵抗的重要机制[11]。氧化应激还与人类代谢疾病相关。肥胖人群血浆脂质过氧化标志物 8-异前列腺素 F2(8-Iso-Prostaglandin F2, 8-Iso-PGF2)、硫代巴比妥酸反应物(thiobarbituric acid reactive substances, TBARS)或尿 8-Iso-PGF2 水平显著高于健康人群,这些氧化应激标志物同身体质量指数(body mass index, BMI)、内脏脂肪重量、腰围等高度相关[6, 12, 13]。肥胖和肥胖代谢综合征人群血浆氧化低密度脂蛋白(oxidized-low density lipoprotein, ox-LDL)水平同样显著高于健康人群,与内脏脂肪重量高度相关[14, 15]。2 型糖尿病人群血浆丙二醛、羰基化蛋白含量显著高于健康人群,SOD 活性显著下降,DNA 损伤提高[16]。虽然氧化应激主要来源是脂肪组织,但关于脂肪组织氧化应激的研究较少。有研究报道,肥胖人群内脏脂肪总体蛋白质羰基化显著高于健康人群[17]。但有研究发现,肥胖 2 型糖尿病组内脏脂肪丙二醛、DNA 损伤等要低于健康和肥胖组,这与脂肪组织抗氧化系统的激活有关[18]。肥胖中还存在一种代谢健康型肥胖,体成分上该人群体脂量、内脏脂肪和皮下脂肪等都低于不健康型肥胖人群,血压、血糖和氧化应激显著低于代谢综合征人群,因为,代谢健康型肥胖人群脂肪酸氧化率更高,呼吸商显著低于代谢综合征和 2 型糖尿病人群[19-21]。

1.2　氧化应激与胰岛素抵抗

氧化应激是触发胰岛素抵抗和慢性炎症的前导因素,动物研究发现高脂饮食干预下氧化应激总是在慢性炎症和胰岛素抵抗前提高[22],多种抗氧化剂均可改善肥胖鼠的胰岛素抵抗[23,24]。氧化应激能改变脂肪细胞脂肪因子表达,体外过氧化氢提高脂肪细胞 MCP-1、IL-6 等炎症因子表达,同时还降低胰岛素致敏因子脂联素的表达[6],这与激活细胞外蛋白调节激酶(extracellular regulated protein kinase, ERK)和 c-Jun 氨基末端激酶(c-Jun N-terminal kinase, JNK)有关[25]。蛋白激酶 ERK 和 JNK 通过磷酸化调控炎症信号通路关键酶和下游转录因子,从而调节炎症因子表达,炎症因子作用于胰岛素信号通路,影响胰岛素敏感性[26, 27]。抗氧化剂能改善慢性炎症,例如,N-乙酰半胱氨酸能提高脂肪细胞脂联素表达,降低 MCP-1、IL-6 等表达[6];维生素 C 能降低肥胖小鼠脂肪组织氧化应激,抑制巨噬细胞活性,提高胰岛素敏感性[28]。NADPH 氧化酶(nicotinamide adenine dinucleotide phosphate oxidase, NOX)为脂肪细胞活性氧主要来源。NOX 抑制剂夹竹桃麻素(apocynin)能显著抑制脂肪细胞活性氧产生,相反,抑制黄嘌呤氧化酶和线粒体呼吸链不影响活性氧的产生;Apocynin 还降低了肥胖小鼠脂肪组织 TNF-α、IL-6、MCP-1 表达,以及血浆 TNF-α 和 IL-6 的浓度[6,29]。脂肪细胞分泌的趋化因子募集单核

细胞浸润脂肪组织,巨噬细胞产生的炎症因子如 TNF-α、IL-1β 在胰岛素抵抗中扮演着关键角色[30]。炎症因子反过来又能作用于脂肪细胞,抑制胰岛素信号通路的同时提高脂肪细胞活性氧产生,形成恶性循环[31]。综上所述,脂肪组织氧化应激和慢性炎症的相互作用,对扩大胰岛素抵抗的发生发挥了重要作用。

2 不同肥胖时期的脂肪组织氧化应激

脂肪组织中不但存在脂肪细胞,基质血管部分(stromal vascular fraction, SVF)还存在着免疫细胞、前体脂肪细胞、成纤维细胞、血管内皮细胞等,在肥胖发生和发展的不同阶段,脂肪组织氧化应激的来源并不一样。在肥胖早期,富余的葡萄糖、游离脂肪酸转化为甘油三酯储存在脂肪细胞,脂肪组织摄取的大部分葡萄糖经磷酸戊糖途径氧化,产生 NADPH,脂肪细胞编导的 NADPH 氧化酶 4(NOX4)被激活,NOX4 转移 NADPH 电子到氧产生·O_2^-提高脂肪细胞氧化应激。敲除 *NOX*4 会降低脂肪细胞炎症因子表达[32]。肥胖时脂肪组织磷酸戊糖途径和 NOX4 活性显著提高,脂肪组织氧化应激水平提高,敲除脂肪细胞 *NOX*4,能延缓高脂饮食下小鼠胰岛素抵抗和脂肪组织炎症的发生[33]。在肥胖中期,脂肪组织免疫细胞的大量增加,成为维持胰岛素抵抗的主要原因[30]。这一阶段巨噬细胞表达的 NOX2 成为脂肪组织氧化应激的重要来源。全身敲除 *NOX*2,降低内脏脂肪重量和巨噬细胞数量,同时改善高脂饮食干预下小鼠的糖代谢[34]。在肥胖后期,胰岛素抵抗显著降低脂肪细胞的葡萄糖摄取,通过脂肪组织磷酸戊糖途径活性和 NADPH 含量下降即可看出。同时,脂肪细胞不再储存甘油三酯,相反开始利用甘油三酯水解产生的游离脂肪酸,脂肪酸氧化负荷远超脂肪细胞线粒体工作能力,产生的活性氧在维持脂肪组织炎症中起主要作用。

3 热量限制与氧化应激和慢性炎症

热量限制(caloric restriction, CR)是指在提供充分的营养成分如必需氨基酸、维生素等,保证生物体不会发生营养不良的情况下,限制摄取的总热量。热量限制是延缓衰老的有效方法,能推迟和降低多种老龄相关疾病的发生,能有效改善肥胖、2 型糖尿病等的代谢紊乱。体重和脂肪总量的下降是热量限制的一个重要标志,这个过程伴随着胰岛素敏感性提高以及氧化应激和炎症的下降。在普通动物实验中发现,与自由摄食组相比,8 ~ 16 周轻到中度的热量限制(15% ~60%)显著降低小鼠或大鼠体重、脂肪总量、肝脏甘油三酯及脂肪组织和肝脏炎症因子表达,提高血浆脂联素水平[35,36]。在肥胖动物研究发现,8 ~ 16 周轻到中度的热量限制(30% -60%)显著缓解高脂饮食诱导的代谢紊乱及脂肪组织炎症基因表达[37,38]。在人体实验中观察到类似的现象,轻度热量限制(25%)第 5 天,在肥胖人群(BMI 32 ± 5.8)体重和脂肪含量未见显著变化的情况下血浆脂质过氧化出现显著降低,随着热量限制时间延长这一趋势更加明显[39]。4 ~ 24 周的低到极低热量饮食($3.4 \times 10^5 \sim 4.2 \times 10^5$ J/d)在降低超重、肥胖及肥胖代谢综合征人群(BMI 25 ~ 64.4)体重的同时,显著降低血浆脂质过氧化和蛋白质羰基化水平,摄入热量越低氧化应激下降越明显[40-42]。2 ~ 6 周极低热量饮食($2.5 \sim 3.5 \times 10^5$ J/d)还能降低肥胖、T2DM 等人群血液中 CRP、IL-6 水平以及血中炎症细胞数量和脂肪组织部分炎症因子表达[43,44]。然而,热量限制调节脂肪组织氧化应激和炎症反应的分子机制尚不清楚。综上所述,适度热量限制能降低体重,减少脂肪的堆积,改善脂肪细胞内分泌功能,降低氧化应激和炎症,而不同热量限制方案对体重、氧化应激、代谢等的影响与热量限制的时间和比例有关。

4 体育运动与脂肪组织氧化应激和慢性炎症

体育运动是预防代谢疾病的有效干预措施。早在 1991 年对宾夕法尼亚大学约 6 000 名毕业生进行长达 14 年的跟踪调查,显示体育运动量每提高 2.1×10^3 J,糖尿病风险下降 6%[45]。对 21 271 名美国男性医生的随访显示,每周至少一次出汗的体育运动同样降低糖尿病风险[46]。体育运动通过增加能量消耗,不但能减少脂肪沉积,改变脂肪细胞体积与含脂量,改变脂肪因子表达模式,还能降低脂肪组织应激水平。氧化应激水平的下降是体育运动防治代谢疾病的主要机制(Fig. 1)。

4.1 体育运动对脂肪组织氧化应激的作用

体育运动能降低脂肪组织氧化应激,这是脂肪组织炎性脂肪因子表达下降的主要机制之一。对普通动物的研究发现,持续有氧运动训练显著降低大鼠附睾脂肪、腹膜后脂肪脂质过氧化水平和 TNF-α、MCP-1 等炎症因子的表达[5]。脂肪细胞肥大时,活性氧产生增加是激活 ERK 是上调单核细胞趋化蛋

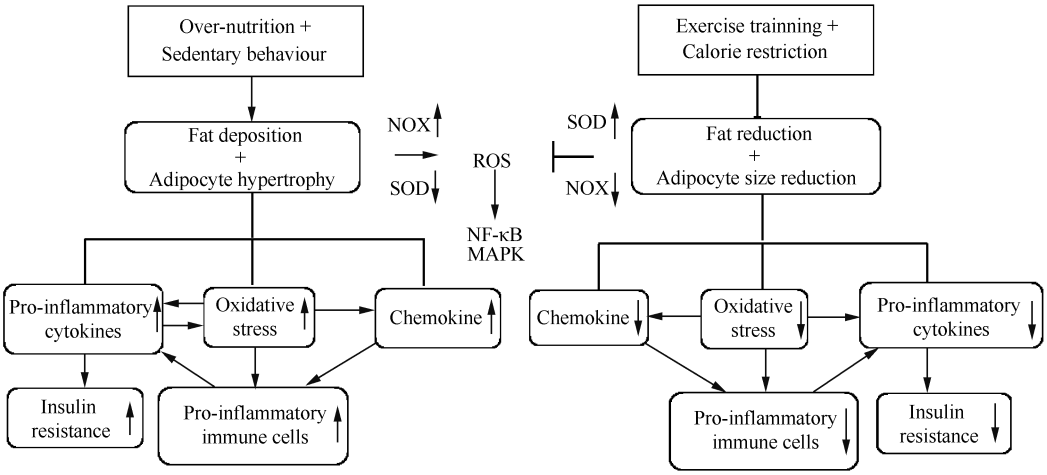

Fig. 1　Regulation of oxidative stress in adipose tissues and adipokines expression　Over-nutrition and sedentary lifestyle lead to fat accumulation, characterized by hypertrophy of adipocytes and increased production of ROS induced by increased expression and activation of NOX and reduced expression and activity of SOD. The activation of NF-κB initiates the expression of pro-inflammatory cytokines and chemokines, which play a crucial role in recruiting monocytes and macrophages into adipose tissues. TNF-α and IL-1β secreted by macrophages induce insulin resistance in local adipocytes and peripheral tissues. Exercise training and caloric restriction reduce the size of adipocytes and total fat mass by increasing energy consumption or restricting energy intake. Exercise reduces the expression of NOX and inactivates it, increases the expression and activity of SOD. Thus the oxidative stress and expression of pro-inflammatory cytokines and chemokines were alleviated. And the number of pro-inflammatory immune cells was reduced. Insulin resistance was largely improved. However, the mechanism of caloric restriction in improving inflammation and insulin resistance is still not clear

白(MCP-1)表达的关键[47],持续有氧运动对脂肪组织 ERK 磷酸化水平及 MCP-1 表达的下调与氧化应激下降有关[5]。对肥胖鼠的研究同样发现,持续有氧运动能降低肥胖动物的脂肪组织氧化应激,例如 8 周游泳运动训练降低肥胖小鼠脂肪组织脂质过氧化和蛋白质羰基化,氧化应激的下降有利于改善脂肪水解[48]。运动对脂肪组织氧化应激的影响与 NADPH 氧化酶(NOX)、SOD 和过氧化氢酶(CAT)等酶的含量及活性变化有关。运动训练降低健康大鼠脂肪组织脂质过氧化,这与 NOX2 表达下降以及 Mn-SOD 表达提高有关[5]。运动训练还降低肥胖鼠脂肪组织 NOX2 表达,提高 Mn-SOD 表达。8 周有氧跑台运动能提高脂肪组织 Mn-SOD 与 CAT 等抗氧化酶的活性[49]。还有研究探索了运动结合饮食控制对脂肪组织氧化应激的影响,4 周有氧运动结合热量限制,显著降低肥胖青少年体重与体脂量的同时,提高血中抗氧化酶 GPX 和 SOD 活性,蛋白质羰基含量显著下降[50]。8 周跑台训练干预尽管对高脂饮食小鼠脂肪组织 NOX2 与 Mn-SOD 表达无显著影响,但降低了 MCP-1 表达,虽然热量限制会提高 MCP-1 表达,但运动结合热量限制则显著降低 MCP-1 表达[51]。NOX4 在脂肪组织氧化应激中发挥重要作用,但目前有氧运动对脂肪组织 NOX4 表达及活性的影响尚不清楚。

高强度间歇训练(high intensity interval training, HIIT)是一种省时高效的训练方法,能在短时间内提高个体的代谢率,通过运动后过量氧耗,提高机体脂肪燃烧,能有效改善多种慢性病。动物实验发现,高强度间歇训练和中等强度持续有氧训练能显著改善肥胖小鼠代谢紊乱,体现在体重、体脂率下降,脂肪细胞体积减小,提高肝脏脂肪酸氧化,提高脂肪组织敏感性,且高强度间歇训练效果更好[52]。在代谢综合征大鼠中同样发现,高强度间歇训练对降低心血管风险和改善脂肪组织、骨骼肌、肝脏等代谢的效果强于持续耐力训练[53]。在人体研究中发现类似现象,不同时长的高强度间歇训练能有效改善肥胖、2 型糖尿病人群的体成分和糖代谢紊乱及胰岛素抵抗,且效果不比持续有氧训练差,高强度间歇训练还能提高心肺功能[54,55]。高强度间歇训练对氧化应激的影响研究极其有限。个别研究发现,急性高强度间歇训练提高游泳运动员外周血氧化应激,但同时提高了抗氧化物质含量[56]。有研究探索了短期高强度间歇训练对健康人群氧化应激及抗氧化系统的影响,结果发现外周血 TBARS、蛋白羰基含量较训练前下降,总抗氧化能力提高[57]。比较高强度间歇训练和持续训练对 2 型糖尿病人群氧化应激的影

响,结果显示,两种训练模式均能改善 2 型糖尿病人群的代谢,高强度间歇训练对降低氧化应激效果似乎更有效,主要与抗氧化系统的激活有关[58]。最近也有研究比较了高强度间歇训练和中等强度持续训练对肥胖大鼠肝脏和血液氧化应激的影响,结果发现,两种训练模式均降低了肝脏和血液脂质过氧化和蛋白羰基含量,提高了抗氧化酶含量[59]。然而,高强度间歇训练对动物或人体脂肪组织氧化应激的影响尚不清楚。

综上,体育运动是降低脂肪组织氧化应激的重要手段,运动结合饮食控制,对降低脂肪组织氧化应激的效果更明显,高强度间歇训练是改善代谢紊乱的有效运动方式,效果甚至比持续有氧运动好,能改善系统氧化应激,但对脂肪组织氧化应激的影响尚不清楚。

4.2 体育运动对脂肪组织脂肪因子表达的影响

体育运动能影响脂肪组织脂肪因子的表达。研究对象与运动干预方案的不同使获得的结果不一致[60]。在普通动物研究中发现,9 周跑台有氧训练降低健康大鼠脂肪组织 TNF-α 与 MCP-1 蛋白含量,且内脏脂肪比皮下脂肪下降明显[5]。在肥胖动物同样发现了运动能改变脂肪组织脂肪因子表达,12 周中等强度跑台有氧训练,显著抑制高脂饮食小鼠内脏脂肪 TNF-α、MCP-1 与 F4/80 的表达[61]。有研究探索了自主跑轮运动对肥胖动物脂肪组织脂肪因子表达的影响,6 周自主跑轮运动能抑制高脂饮食小鼠内脏脂肪 TNF-α、MCP-1 等表达,改善胰岛素敏感性和脂肪组织炎症[62]。本室早期的研究发现,4 周游泳训练显著降低 2 型糖尿病小鼠内脏脂肪 TNF-α、IL-6 与 F4/80 等表达[63]。另有不同的研究发现,12 周自主跑轮运动虽然降低高糖饮食小鼠肠系膜脂肪含量,但却提高肠系膜脂肪 TNF-α 表达[64],8 周中等强度跑台训练,尽管提高了高脂饮食小鼠肠系膜脂肪 IL-10 含量,但是 TNF-α 含量同样提高[65]。综上,不同的训练方案对肥胖、糖尿病动物脂肪因子表达带来不同的影响。同样,人体研究表明,体育运动能影响脂肪组织因子的表达及血液中脂肪因子的浓度。有研究发现,12 周单纯的有氧训练对肥胖人群皮下脂肪 TNF-α、MCP-1、IL-6、CD68 等表达未见显著影响,仅提高了脂联素表达,仅有血 MCP-1 水平降低,IL-6、IL-8 与 IL-15 等未见显著变化,相反,12 周热量限制或热量限制结合有氧训练显著降低血 MCP-1、IL-15、IL-18 水平,三种不同干预方式引起的体重下降与血液炎症因子变化是相关的[66]。同样有研究发现 15 周中等强度运动配合饮食控制,显著降低肥胖者皮下脂肪 TNF-α、IL-6 与 IL-8 表达,血浆 CRP、MCP-1、IL-6 与 IL-8 同样下降,脂联素表达和血浆脂联素提高[67]。另有针对肥胖女性的研究同样发现,12 周单纯的有氧训练对腹部皮下脂肪 IL-6 和 TNF-α 表达,及以血液 IL-6 与 TNF-α 浓度均无影响[68]。综上所述,大部分证据支持运动训练降低附睾脂肪和腹膜后脂肪 TNF-α 表达,提高肠系膜脂肪 TNF-α 表达,对皮下脂肪 TNF-α 影响小,这种差异的机制尚不清楚,运动结合热量限制对脂肪组织炎症因子表达和分泌效果强于单一干预。事实上,不同部位脂肪因子表达模式是不同的,肠系膜脂肪 TNF-α 表达远高于大网膜脂肪和皮下脂肪[69]。脂肪组织巨噬细胞数量同样影响 TNF-α 表达对运动的应答变化。肥胖时脂肪组织 TNF-α 主要来自巨噬细胞,健康和肥胖人群内脏脂肪 CD68 + 巨噬细胞数量都高于皮下脂肪[70]。有学者比较了高强度和中等强度间歇训练对肥胖女性血脂联素的影响,结果发现,两种训练模式均显著降低体重、体脂率,低密度脂蛋白-胆固醇(low density lipoprotein- cholesterol, LDL-c)显著下降,高密度脂蛋白-胆固醇(high density lipoprotein- cholesterol, HDL-c)、脂联素显著提高,胰岛素抵抗得到改善[71]。高强度间歇训练还能影响血网膜素浓度,网膜素是胰岛素致敏因子,肥胖人群血网膜素浓度下降,8 周高强度间歇训练能提高超重和肥胖人群血网膜素浓度[72]。高强度间歇训练还能降低代谢综合征人群血液中促炎症因子含量,提高抗炎因子含量[73]。动物实验还发现高强度间歇训练同持续有氧训练一样能降低肥胖小鼠脂肪组织炎症,提高脂肪组织胰岛素敏感性,且高强度间歇训练效果更明显[52]。不同的研究发现高强度间歇训练虽能改善肥胖人群体成分,降低心血管疾病(CVD)风险因子,但对脂肪因子表达无显著影响[74]。

综上所述,高强度间歇训练对改善肥胖、2 型糖尿病和代谢综合征人群代谢紊乱效果非常明显,还能改边脂肪因子的分泌,降低脂肪组织炎症,但对脂肪因子的影响受到受试者背景、运动方案等因素的影响结果并不一致,还需要进一步的研究。

5 问题和展望

脂肪组织是肥胖时氧化应激的主要来源。不同来源的活性氧在肥胖不同阶段的脂肪组织炎症、胰岛素抵抗中扮演着不同角色。抗氧化是改善胰岛素

抵抗和脂肪组织炎症的重要策略。运动对炎症因子表达的调控与氧化应激下降有关,运动提高脂肪组织抗氧化酶表达和抗氧化酶活性的同时降低了 NADPH 氧化酶 2 表达。然而,运动对脂肪组织 NADPH 氧化酶 4 的表达及活性的影响尚不清楚。同时,热量限制对降低系统氧化应激和炎症效果明显,但对脂肪组织氧化应激的影响还需要进一步研究。运动结合饮食控制,对改善氧化应激和炎症的效果要好于单一干预。高强度间歇训练作为一种省时高效的锻炼方式,在改善体成分和慢性病的干预中逐渐崭露头角。不过,高强度间歇训练对脂肪组织氧化应激的影响有待深入研究。

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