

老年人人体成分与衰弱相关性的研究进展

程圣钧^{1,2}, 石佳萌^{1,2}, 王敏娟^{3*}

¹西安医学院第一附属医院全科医学科, 陕西 西安

²西安医学院研工部, 陕西 西安

³西安医学院第三附属医院全科医学科, 陕西 西安

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摘要

随着人口老龄化加剧, 衰弱已成为影响老年人群健康预后与生活质量的關鍵公共卫生问题, 其发生与人体成分的动态变化密切相关。本文旨在整合现有研究证据, 重点阐述了体成分关键维度(骨骼肌质量、脂肪质量、身体水分、矿物质)与衰弱的关联, 为老年人衰弱的早期识别、机制研究及精准干预提供理论参考, 同时指出未来需进一步探索水分代谢调控等新兴干预靶点, 完善体成分导向的衰弱防控策略。

关键词

衰弱, 体成分, 老年人

Research Progress on the Correlation between Body Composition and Frailty in the Elderly

Shengjun Cheng^{1,2}, Jiameng Shi^{1,2}, Minjuan Wang^{3*}

¹Department of General Medicine, The First Affiliated Hospital of Xi'an Medical College, Xi'an Shaanxi

²Department of Research, Xi'an Medical College, Xi'an Shaanxi

³Department of General Medicine, The Third Affiliated Hospital of Xi'an Medical College, Xi'an Shaanxi

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Abstract

With the intensification of population aging, frailty has become a critical public health issue that affects the health outcomes and quality of life of the elderly population. Its occurrence is closely

*通讯作者。

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associated with dynamic changes in body composition. This article aims to integrate existing research evidence, focusing on elaborating the associations between key dimensions of body composition (skeletal muscle mass, fat mass, body water, and minerals) and frailty. It provides a theoretical reference for the early identification, mechanism research, and precise intervention of frailty in the elderly. Meanwhile, it points out that future research needs to further explore emerging intervention targets such as water metabolism regulation to improve the body - composition - oriented frailty prevention and control strategies.

Keywords

Frailty, Body Composition, Elderly

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1. 引言

随着全球人口老龄化进程的加速, 衰弱已成为老年医学领域亟待攻克的重大公共卫生挑战, 其不仅是老年人多系统生理功能衰退的综合表现, 更与跌倒、心血管疾病、住院率及死亡率等不良健康结局密切相关, 严重影响老年人群的生活质量与生存预后[1]。作为一种动态可逆的生理状态, 衰弱的病因被认为是多因素的, 其中体成分的变化似乎是衰弱的核心[2]。因此, 本文重点阐述人体成分关键指标与衰弱的相关性及其病理生理机制, 并总结相关干预策略的研究进展, 旨在为老年衰弱的早期识别、精准防控及临床干预提供全面的理论参考与实践依据。

2. 衰弱

2.1. 衰弱的概念

衰弱是一种多见于老年人的生理状态, 其特征为多个生理系统的功能或储备能力逐渐且持续衰退, 导致机体易损性增加, 在遭遇发热、感染、手术、跌倒及药物治疗等不良事件引发的内环境改变后, 难以维持或恢复内环境稳态[3] [4]。因此, 衰弱可被视为一种生物学状态, 因多个系统和器官普遍衰退, 表现为对应激源的抵抗力低下、应激反应能力减弱。因此, 衰弱可能会增加日常生活活动能力下降、心血管疾病、癌症、跌倒、活动受限的风险, 并提高住院率与死亡率[5] [6]。

2.2. 衰弱的评估工具

目前, 已有近 70 种用于评估衰弱的工具, 其中最为广泛的包括 Fried 衰弱表型、衰弱指数、衰弱筛查量表和临床衰弱量表。

2.2.1. Fried 衰弱表型(Fried Frailty Phenotype, FFP)

现在普遍应用的衰弱表型是在 2001 年 Fried 等[7]从躯体衰弱的概念出发, 认为老年人的衰弱可通过以下标准中至少 3 项的存在来识别: 非自愿体重减轻、疲劳或疲惫、体力活动减少、行走缓慢和肌肉力量下降, 只要有超过 3 项指标符合就证明患者已处于衰弱状态, 若有 1~2 项符合则划分为衰弱前期。

2.2.2. 衰弱指数(Frailty Index, FI)

衰弱指数也被称为 Rockwood 指数或累积缺陷指数, 由 Rockwood 和 Mitnitski 于 2001 年提出[8], 该

量表包括认知能力、慢性疾病、环境危险因素、社会心理危险因素、老年综合征(例如跌倒、谵妄和尿失禁)和与年龄相关的功能残疾,提供了对衰弱的全面评估。FI 的计算方式是存在缺陷数量与所考虑总缺陷数量的比值,这些缺陷可以是症状、体征、疾病、残疾、实验室检验、X 线或心电图异常以及社会特征。这种累积缺陷方法被证明可用于预测老年人的死亡率和和其他不良健康结局[1] [9]。

2.2.3. 衰弱筛查量表(The Frail Scale)

衰弱筛查量表是由国际营养健康和老龄化协会推出的简单易行的量表[10] [11]。这个工具已经被证实能够预测死亡率和残疾率,且量表的信效度[12]和反应速度都非常优秀。该衰弱评估量表包括五项内容:(1) 疲劳:您上周多数时间感到疲劳吗?(2) 阻力:您能上一层楼梯吗?(3) 行动力:您能行走一个街区(500 米)的距离吗?(4) 多种慢性病共存:您患有五种及以上的疾病吗(如下疾病:心脏病、高血压、卒中、帕金森、糖尿病、慢性肺病、哮喘、关节炎、骨质疏松、消化道溃疡、白内障、骨折、肿瘤、其他)?(5) 最近一年体重下降情况:您最近 1 年内体重下降超过 5%了吗?以上每个部分的回答分为是和否,分别记 1 分或 0 分,最终得分在 0~5 分之间,其中 0 分表示无衰弱,1 至 2 分代表衰弱前期,3 分及以上就表明处于衰弱期。

2.2.4. 临床衰弱量表(Clinical Frailty Scale, CFS)

CFS 是一种临床医生对患者整体健康状况的主观评价工具,评估包括多个生活领域,如活动能力、日常生活能力、认知能力、用药方案以及整体健康状况的近期变化,可以得出一个总体的衰弱评分,共分为 9 个等级,从非常健康到接近死亡,通常 ≥ 5 级定义为衰弱。CFS 量表是一种简单实用的衰弱评估工具,是医院环境下评估衰弱状况的理想选择,因为它实施起来耗时极少,且有相关指导可供参考。且除了临床实践外,CFS 目前还被纳入临床登记系统,并在研究中用作衰弱的标志物[13]。

3. 体成分与衰弱

3.1. 骨骼肌质量

骨骼肌质量是维系躯体功能与代谢稳态的核心组织,也是衰弱表型发生与加重的“发动机”。Fried 物理表型中,步速下降、握力降低及体力耗竭均直接源于肌肉数量与功能的同步丢失。相关文献表明,在 70 岁之后,男性和女性的肌肉质量每十年分别减少 4.7%和 3.7% [14]。结合欧洲老年人肌少症工作组(EWGSOP2)、亚洲肌少症工作组(AWGS)及中国相关专家共识,目前肌少症诊断标准核心围绕肌肉力量、肌肉质量、躯体功能三大维度来评价,其中肌肉质量维度的评估指标即四肢骨骼肌质量(ASM)、骨骼肌质量指数(SMI) [15]。刘金炜等[16]通过随机森林算法筛选出了 10 个与衰弱相关的重要特征变量,其中骨骼肌的减少与衰弱的发生有着密切关系。KURIYAN R [17]、KOŁODZIEJ M [18]等人的研究发现,衰弱前期人群的四肢肌肉质量及功能明显低于非衰弱人群。同时,一项纳入 115 名在加拿大癌症中心接受化疗的老年人的回顾性队列研究显示,基于 CT 的 L3 水平肌肉脂肪变性可提高衰弱预测老年癌症患者全因死亡率的性能[19]。由此可见,骨骼肌质量不仅与衰弱诊断高度重叠,更是预测不良结局的敏感指标。

骨骼肌质量减少是由于蛋白质合成与分解失衡所引发的状态,临床表现为肌力减退、易疲劳和运动障碍,与多种因素相关[20]。而控制这种合成和降解的途径主要是两条信号通路:胰岛素样生长因子 1-磷脂酰肌醇-3-激酶-Akt/蛋白激酶 B-雷帕霉素靶标(IGF1-PI3K-Akt/PKB-mTOR)通路和肌生长抑制素-SMAD3 (myostatin-SMAD3)信号通路,前者是肌肉生长的正调控因子,而后者是负调节因子[21]。其中胰岛素样生长因子-1 (IGF-1)是调节骨骼肌合成代谢和分解代谢途径的关键生长因子,可以通过正向通路促进骨骼肌蛋白质合成,同时可以抑制负向途径从而抑制肌肉萎缩[22]。已有研究报道 IGF-1 在各种慢性疾病中呈现减少的现象,包括癌症、充血性心力衰竭、慢性肾脏疾病和慢性阻塞性肺病以及衰老[23]。骨骼

肌衰减至衰弱并非单纯数量变化,而是多系统交互作用的结果。慢性炎症被认为是首要驱动因素,在慢性炎症或病理状态下,骨骼肌中的众多细胞分泌脂肪细胞因子,如肿瘤坏死因子- α (TNF- α)、白细胞介素-6 (IL-6)、抵抗素、瘦素及脂联素等,其持续过量会加速炎症反应,同时启动泛素-蛋白酶体途径和自噬-溶酶体途径加速蛋白质降解[24]。此外,肌肉能力往往与肌肉线粒体功能密切相关,随着年龄的增长,老化肌肉线粒体 DNA 缺失与活性氧累积导致能量短缺及氧化损伤,进而损害钙调节,并影响肌丝结构和功能,进一步削弱肌纤维再生能力,最终降低肌肉力量的产生[25]-[27]。

研究证明,衰弱是一个动态和可逆的过程,随着时间的推移,衰弱的中老年人可以经历衰弱状态的改善或恶化,肌少症也是如此[28]。一项着眼于以肌肉质量和功能干预研究的系统综述显示,运动干预可以改善肌肉力量和身体表现[29]。多数研究发现,锻炼能提升肌肉质量、力量和功能,因此通过增加肌肉质量、力量和活动能力,可能对肌少症具有保护性和有益的作用,而相对不活跃的人患肌少症的风险增加或加重其严重程度[30]-[32]。除运动锻炼之外,营养摄入不足也会增加肌少症的可能性,尽管老年人的营养需求通常更高,但他们摄入的能量和蛋白质却低于年轻人[33]。目前对于成年期饮食与老年肌少症风险的证据有限且零散,但饮食模式研究的结果已经就成年早期更高质量的饮食对后期身体机能的益处提供了一致的信息,不过迫切需要全膳食干预试验来了解深层相关性[34]。体力活动和营养支持,以改善物理功能,防止肌少症、衰弱和功能下降在文献中被广泛推荐[35]。综上,骨骼肌质量不仅是衰弱发生的核心介质,更是干预与监测的关键靶点。

3.2. 脂肪质量

40 岁以后,骨骼肌质量开始下降,特别是久坐不动的人,肌肉质量的减少通常被脂肪的增加所取代,这反映在体重指数(BMI)中[36]。而 BMI 作为常用肥胖评估指标,却难以准确反映脂肪分布,BMI 正常但体脂过高的个体,或面临更高的代谢疾病患病风险[37]。JAYANAMA [38]、SÃO ROMÃO [39]等的研究发现,体重过低、超重、肥胖及较高的体脂百分比与较高的衰弱程度有关。同样 XUL 等[40]的研究观察了住院患者老年人衰弱与体成分的关系,结果显示中国老年住院患者的衰弱表现为体重偏低、腰围偏高、骨骼肌质量偏低及体脂肪偏高。因此,体重不足、腹部肥胖和肌少性肥胖可能是干预衰弱的目标。同时近十年研究一致表明,脂肪分布比总量更重要。一项纳入了 1153 人甘肃省靖远县 60 岁及以上的老年人的横断面研究显示,中国内脏脂肪指数(CVAI)与衰弱风险呈正相关,且对衰弱风险存在预测价值[41]。同样地,一篇数据来源于 NHANES 的研究[42]显示,内脏脂肪指数(VAI)和脂质积累产物(LAP)与美国成年人的衰弱之间存在显著关联,其中 VAI 与衰弱线性相关。

脂肪组织是脂肪储存的主要场所,并参与器官间对话,以协调细胞对外源性脂质信号的反应[43] [44]。例如,脂肪细胞自身或脂肪组织内的浸润巨噬细胞会响应过量的游离脂肪酸,产生 IL-6 和 TNF- α 等促炎细胞因子,从而上调全身炎症反应[45]。正因如此,在肌少性肥胖的男性和女性中,IL-6 和 TNF- α 与肌肉质量和力量呈负相关[46] [47]。此外,IL-6 介导的 STAT3 激活也会通过刺激骨骼肌中与萎缩相关的信号通路,独立引发肌肉消耗[48]。并且,肌肉减少与特定的 II 型肌肉纤维萎缩和卫星细胞池减少有关,在肥胖的背景下,脂肪组织分泌体显著损害肌肉生成,同时减少卫星细胞的数量和增殖能力,降低肌肉质量和肌肉质量[49]-[51]。这种卫星细胞含量和增殖功能的减少可能与线粒体动力学受损有关,从而导致骨骼肌质量和功能的丧失[52]。其他促炎因子如瘦素(Leptin),也与肥胖密切相关。瘦素主要由白色脂肪组织(WAT)中的脂肪细胞分泌,其分泌水平与脂肪储存量成正比。当脂肪细胞增加时,瘦素水平会相应上升,然后与大脑中的瘦素受体(LEP-R)结合,这些受体会发送信号以抑制食物摄入并增加能量消耗,从而维持能量平衡[53]。瘦素还在肝脏和骨骼肌等外周组织发挥代谢作用,通过改善肌细胞代谢增加葡萄糖摄取量和降低肌细胞内脂质沉积,在骨骼肌萎缩中发挥关键作用。而肥胖的人群大多有瘦素抵抗,高水平

瘦素无法正常发挥效能[53][54]。相对地,某些抗炎脂肪细胞因子如脂联素,具有促进骨骼肌肥大、改善肌萎缩以及提高葡萄糖摄取和脂肪酸氧化的作用[55]。脂联素可以通过激活 AMPK 和 AKT 信号通路,抑制肌肉蛋白分解,在调节骨骼肌代谢和抗炎方面具有显著潜力[56]。然而不同研究表明,在老年人群中,脂联素与肌肉质量指数可能呈现“脂联素悖论”,即较高的脂联素水平与较低的肌肉质量指数相关[57][58]。这表明脂联素在不同病理状态下的作用机制较为复杂,未来仍需要进一步研究以明确其作为临床治疗靶点的可行性和安全性。

有两个重要生活方式因素在肌少症性肥胖等疾病的发生中起着重要作用:运动和营养。一项针对社区老年人的研究[59]显示,肌少症型肥胖的老年人在进行抗阻训练、有氧运动和联合训练干预后,实验组表现为肌肉量增加,同时总脂肪量和内脏脂肪面积减少。此外,弹性阻力训练也可以有效改善老年肥胖症患者的身体功能,同时减少脂肪质量[60][61]。由于内脏脂肪库会产生促炎脂肪因子影响肌肉的分解代谢,因此肥胖的受试者在能量限制下更容易肌肉萎缩[62]。此外,随着年龄的增长,脂肪渗入肌肉,将瘦肌肉转变为脂肪肌肉,这些内部变化再次导致肌肉功能的变化[63]。VILLAREAL [64]等同样得出相似结论,他们强调减重、饮食与运动相结合在改善身体机能方面比单独采用其中任何一种干预措施效果更好。这意味着在营养方面,亦需同时采取增加肌肉质量及减少脂肪质量,如肌少症肥胖老年人需每日摄入足够的蛋白质及必需氨基酸、亮氨酸、维生素 D 等[65][66]。因此,任何干预措施对肌少症性肥胖的影响不能仅通过体重的变化来衡量,而必须关注身体成分和功能参数的变化。

3.3. 身体水分

水是人体的主要成分,成年男性约占体重的 60%,成年女性约占 50%至 55%。瘦体重和脂肪组织中的水分含量分别约为 73%和 10%。总身体水分(TBW)在体内以细胞内水(ICW)和细胞外水(ECW)的形式分布,ICW 主要存在于骨骼肌,ECW 参与构成体内细胞生活的内环境,而 ICW 是评估肌少症的有效指标[67]。TIAN M 等[67]、ZHOU C 等[68]证实,在慢性血液透析患者中,较低的 ICW 与肌少症及肌少性肥胖的风险增加有关。而肌肉中 ECW 相对于 ICW 的相对增加是肌肉质量下降的一个因素,也是肌肉老化的生物标志物[69]。类似地,SERRA-PRAT M 等[70]的一项对 75 岁及以上社区居民进行的观察性横断面研究显示,在肌肉质量相近的老年人中,ICW 较高的人有更好的功能表现和较低的衰弱风险,这表明在独立于肌肉质量情况下,水分具有一定的保护作用。KEHAYIAS JJ 等[71]通过试验提出,ECW/TBW 可以通过模拟身体细胞团的代谢作用,作为替代方法用于临床的衰弱评估。尽管 TBW、ICW、ECW 及其相关比值与肌少症、衰弱的关联已得到多项研究证实,但目前针对水分分布调控以改善衰弱状态的干预措施仍较为缺乏,未来需进一步聚焦水分代谢对衰弱的影响机制及相关干预策略,为老年人群衰弱防控提供新的思路。

3.4. 矿物质

矿物质主要包括骨矿物质,也包含少量分布在血液、内脏、肌肉中的无机矿物质(如钙、磷、镁等)。钙磷代谢与骨代谢密切相关,钙磷为骨形成的原料,而骨组织是钙磷的重要储存库。生长过程中,骨矿物质含量与肌肉质量密切相关,多项证据表明骨质疏松症和肌少症存在共同的病理生理因素,并显示低骨密度与肌少症在男女中存在相关性[72]。由于老年人骨矿含量随年龄的增长存在不同程度的变化,致使老年人存在骨质疏松和骨折的危险。例如,随着年龄增长,钙信号通路紊乱与肌肉减少呈恶性循环:钙稳态失衡会损害肌肉收缩、再生功能,加速肌少症;而肌肉组织衰老又会进一步破坏钙调节机制。此外,铁、锌等矿物质的异常积累会通过钙的相互作用,放大这一病理过程:老年肌肉中锌浓度升高,会竞争性抑制钙通道(如 RyR2),阻碍钙释放,同时影响钙结合蛋白功能;老年后肌肉铁过多积累,通过 Fenton

反应产生过量活性氧(ROS),破坏钙通道稳定性,还会与钙竞争结合位点,加剧钙稳态失衡[73]。目前很少有试验直接研究衰弱与骨矿物质含量之间的相关性,但现有研究如 LIU J 等[74]通过一项双样本孟德尔随机化研究得出,骨-肌减少症和衰弱之间存在显著的因果关系。骨-肌减少症是指若个体同时满足肌少症和骨量减少或骨质疏松症的任一诊断标准,则符合诊断。研究表明大多数骨-肌减少症的老年人都会有衰弱,且骨-肌减少症是衰弱的一个非常强烈的风险因素[75]。因此,及早发现、干预和治疗这些肌肉骨骼疾病,可以有效减少衰弱的发生并延缓其进展。

4. 总结

综上,衰弱作为老龄化背景下影响老年人群健康的核心公共卫生问题,其发生发展与体成分的动态变化密切相关,体成分的评估监测及干预或将作为如何改善衰弱的新兴领域。目前,运动干预与营养支持已被证实可通过改善体成分延缓衰弱进程,但针对水分分布调控等新兴方向的干预研究仍较为匮乏。未来研究需进一步聚焦以下方向:一是深入探索水分代谢失衡影响衰弱的分子机制,开发针对性调控策略;二是优化体成分评估工具,构建更精准的衰弱风险预测模型;三是开展大样本、长期随访的干预试验,验证多维度联合干预的临床效果;四是关注特殊老年人群(如住院患者、慢性病患者)的体成分特征,制定个体化衰弱防控方案。通过上述研究,有望完善体成分导向的衰弱防控体系,为提升老年人群健康水平与生活质量提供更坚实的理论与实践支撑。

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