

睡眠剥夺对肥胖的影响

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摘要

近几年, 越来越多的研究表明睡眠剥夺与肥胖风险增加有关。睡眠剥夺通过食欲调节、能量摄入、食物选择、奖赏机制等促进肥胖的发生和发展, 并且, 与体重管理期间患者的减肥失败有关。改善睡眠有利于减轻体质量。本文综述睡眠剥夺引起肥胖的潜在机制, 旨在进一步丰富预防和治疗肥胖症的方法, 以及对相关公共卫生策略的制定提供理论依据。

关键词

肥胖症, 睡眠剥夺, 食欲, 激素, 减重

The Effects of Sleep Deprivation on Obesity

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Abstract

In recent years, a growing number of studies have linked sleep deprivation to an increased risk of obesity. Sleep deprivation promotes the occurrence and development of obesity through appetite regulation, energy intake, food selection, reward mechanisms, etc., and is associated with weight loss failure in patients during weight management. Better sleep can help reduce body mass. This paper reviews the potential mechanisms of obesity caused by sleep deprivation, aiming to further enrich the prevention and treatment methods of obesity, and provide theoretical basis for the formulation of related public health strategies.

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Keywords

Obesity, Sleep Deprivation, Appetite, Hormone, Weight Loss

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1. 引言

肥胖症的流行对代谢性疾病、心血管疾病和某些类型的癌症的发展构成了重大威胁。睡眠剥夺,是指睡眠时间、结构和/或质量不足的异常睡眠状况[1]。在过去的 20~30 年里,肥胖率急剧上升,现在已知遗传和环境因素多有助于肥胖的发生,致肥胖饮食和低体力活动被认为是两个主要的风险因素,然而,近年来,其他潜在的环境因素,如睡眠时间短,越来越受到关注[2]。从流行病学研究结果显示,睡眠不足与体重指数(Body mass index, BMI)升高有关[3]。Logistic 回归分析发现,每增加一小时的额外睡眠限制与肥胖几率增加 10%相关[4],这表明睡眠剥夺者的肥胖发生率较高,睡眠剥夺会增加肥胖的风险[5]-[7]。本文综述睡眠剥夺与肥胖症相关性的研究进展,旨在进一步丰富预防和治疗肥胖症的方法,以及对相关公共卫生策略的制定提供理论依据。

2. 睡眠剥夺对肥胖的影响

多项针对成人、儿童、非肥胖人群的研究均表明,睡眠障碍与肥胖的相关性,例如,两项对成人和儿童队列研究的荟萃分析都强调睡眠时间短是肥胖的诱因之一[8] [9]。对 33 项儿童和青少年的队列研究的荟萃分析显示,短睡眠时间的肥胖风险为 1.57 [10]。与睡眠 7~8 小时的人相比,睡眠 5 小时或更少的人的风险增加了 15%,睡眠 6 小时的人的风险增加了 6%。研究发现,睡眠减少 1 小时与身体质量指数(BMI)增加 0.35 有关[5]。在一项针对健康非肥胖个体的随机对照实验中,在睡眠限制(4 小时睡眠时间,为期 14 天)和对照条件下,体脂总量的变化没有差异,但在睡眠限制期间,腹部和内脏脂肪明显增加[11]。此外,有关整个生命周期中睡眠和肥胖风险的关系,Miller 等[12]最近的一项荟萃分析发现,随着时间的推移,睡眠时间短与婴儿期(相对危险度[RR] 1.40, 95%置信区间[CI] 1.19~1.65)、儿童早期(RR 1.57, 95% CI 1.40~1.76)、儿童中期(RR 2.23, 95% CI 2.18~2.27)和青春期(RR 1.30, 95% CI 1.11~1.53)发生超重或肥胖的风险增加有关。因此,在所研究的每个年龄组中,睡眠不足都容易导致体重过度增加,尽管关联程度不同,在儿童中期达到顶峰,此后略有下降。

3. 睡眠障碍引起肥胖的机制

睡眠时间短可能会影响食欲和能量消耗,调节食欲相关激素的活动水平和功能[13]-[15]。此外,睡眠时间短是体内的慢性压力源,直接影响下丘脑-垂体-肾上腺轴的功能,以及皮质醇的合成和分泌。下丘脑是大脑皮层下的高级中枢,调节内脏活动,将其与其他生理活动联系起来,并调节重要的生理功能,如生物节律、摄食、水平衡、体温和内分泌腺活动[16]。

睡眠剥夺时能量摄入与消耗 Patterson 等[17]的研究表明,与睡眠时间 ≤ 6 小时/天的人相比,睡眠时间 ≥ 9 小时的成年人每天消耗的热量减少了 178 千卡。然而, Zhu 等[18]的研究表明,睡眠限制对总能量消耗没有影响而与能量摄入增加有关,随机对照试验的荟萃分析表明,与习惯性睡眠相比,部分睡眠剥夺会增加总能量摄入[19][20],横断面研究也显示了睡眠不足和能量摄入增加之间的关联[21]。总体而言,

大多数肥胖病例是由于额外的能量摄入而导致的正能量平衡[22]。将睡眠时间短与能量摄入增加联系起来的机制较多,包括调节能量稳态的激素变化。

3.1. 睡眠剥夺对食欲影响

基于人群的实验研究表明,睡眠时间短或部分睡眠剥夺与饥饿感增加和食欲增加有关,并随着睡眠延长而逆转[18]。食欲的增加似乎与较高的血糖指数、血糖负荷和碳水化合物摄入量特别相关,也有其他研究显示,增加的食欲与脂肪摄入有关[18][23]。现有研究指出睡眠剥夺可引起食欲相关激素,包括瘦素、生长激素释放肽、食欲素和胰岛素的分泌变化,从而引起食欲变化和肥胖的发生[13]-[15]。

3.1.1. 瘦素

瘦素主要来源于白色脂肪组织,可以引起饱腹感,其主要作用是抑制下丘脑的食欲,增加能量消耗和抑制脂肪合成。在一些研究中,睡眠不足会引起瘦素的减少,一项对儿童和青少年的研究表明,睡眠剥夺会影响血清瘦素水平,影响食欲调节并导致体重增加[14]。与此不同的是,在最近一项荟萃分析的实验亚组中,相对于正常睡眠,短睡眠者的瘦素水平更高[24]。但也有研究表明,当瘦素达到其阈值时,血脑屏障会限制瘦素从外周血转运到中枢神经系统,从而抑制其活性[25]。这种情况解释了由于睡眠限制导致瘦素水平升高,但其有效性减弱的现象。

3.1.2. 生长激素释放肽

生长激素释放肽是从胃泌酸腺和下丘脑弓状核(ARC)的神经元释放的一种促进饥饿感的肠道肽,刺激生长激素释放和食物摄入。在睡眠剥夺期间,与正常睡眠相比,生长素释放肽在夜间开始时减少,在后半夜增加,最终在第二天早上升高生长素释放肽,增加饥饿感并促进食物摄入。Speath [26]等的研究证明,在两个晚上睡4个小时的情况下,循环中生长激素释放肽增加28%。并且,与睡眠时间正常的参与者相比,睡眠时间短的参与者的生长激素释放肽水平高出14% [24]。

3.1.3. 食欲素

食欲素是由下丘脑神经元分泌的神经肽,于1998年首次在大鼠脑组织提取物中被发现,有食欲素-A和食欲素-B两种,食欲素神经元参与控制多种稳态功能,包括进食和能量消耗。一项针对大鼠的研究证实,睡眠不足会增加食欲素神经元对兴奋性驱动[27]。有临床研究发现失眠症患者的食欲素-A水平明显高于正常睡眠者,且其升高水平与睡眠的病程和严重程度有关[28]。王忠等[29]的研究,同样证实了睡眠剥夺可以使食欲素-A水平升高。

3.1.4. 胰岛素及敏感性

胰岛素是机体内唯一降低血糖的激素,在维持正常能量代谢中起关键作用,其在中枢神经系统发挥抑制食欲、减轻体重等作用,但外周胰岛素的作用与中枢相反。在一项随机对照实验中,10名健康个体在睡眠实验中经历了4天正常睡眠和4天睡眠剥夺,每天清晨进行胰岛素测量,结果显示睡眠剥夺实验中胰岛素曲线下面积高于对照组[30],此外基于临床证据的叙述性综述报告称,睡眠剥夺或睡眠质量差与胰岛素敏感性降低相关[31]。通过恒河猴模型的动物研究同样表明,睡眠剥夺会导致胰岛素敏感性降低[32]。睡眠不足后胰岛素敏感性降低的推测机制包括生长激素分泌延长、皮质醇升高、交感神经系统的激活增加以及去甲肾上腺素的升高等[33]。

3.2. 睡眠剥夺对食物选择的影响

睡眠不足的人通常会寻找并食用美味的食物,这些食物大多是不健康的,即高能量、高脂肪和/或高糖含量的食物[34][35]。与此一致的是,一项对将延长睡眠作为改善心脏代谢健康策略的干预措施的系统

综述表明, 延长睡眠可能与总体食欲减少、对甜食和咸味食物的渴望以及每日从蛋白质中摄取能量的百分比增加有关[36]。据报道, 在中国睡眠不足的学龄儿童中, 水果和蔬菜的消费量较低[37]。在一组 2~6 岁的丹麦儿童中, 睡眠时间变化较大的儿童摄入的水果和蔬菜较少, 摄入的添加糖和含糖饮料较多[38]。此外, 在一项针对 118,462 名韩国青少年的横断面研究中, <6 小时的睡眠时间不足与软饮料和甜食的摄入量增加有关, 7~8 小时睡眠质量差与水果、蔬菜和牛奶的摄入量减少, 而苏打水、软饮料、快餐、方便面和糖果的摄入量增加有关[39]。除了食物的选择, 较晚的进食时间似乎也会促进成人和儿童的代谢失调, 从而导致体脂增加[40] [41]。大多数试验都认同睡眠不足对奖励系统激活和高能量食物选择的影响[42] [43]。

3.3. 大脑奖赏系统

睡眠限制与参与奖赏的大脑网络的激活有关[18]。奖励系统和主观食物评价活动的增加可能发生在没有饥饿和食欲相关激素改变的情况下[44]。在一些肥胖病例中, 大脑中动机结构的超敏化导致了过度的食物“欲望”[45]。Demos 等[46]人表明, 与充足的睡眠(9 小时/晚)相比, 短时间睡眠(6 小时/晚)会导致大脑奖励处理区域对食物图像的活跃这种效应主要见于肥胖个体。Jensen 等[47]人发现, 在睡眠限制中, 正常体重和超重/肥胖的青少年无论体重状况如何, 在奖励区域都表现出更高的活动。

另外, 内源性大麻素系统是一种神经调节系统, 与控制喂养和食欲有关, 特别是与对食物的享乐驱动有关, 导致正能量平衡和体重的增加[48]。2-花生四烯酰甘油(2-AG)是内源性大麻素 CB1 和 CB2 受体的内源性激动剂, Hanlon 等[49]发现, 睡眠剥夺后的 2-AG 峰值更高且持续时间更长, 这表明在睡眠剥夺之后, 下午早些时候的享乐饮食驱动力可能更强, 持续时间更长。

4. 睡眠剥夺对减重的影响

除了睡眠剥夺引起肥胖的数据外, 在减肥计划中, 睡眠障碍与减肥失败的风险增加有关[42]。在一项为期 24 周 600 千卡限制饮食的纵向研究中, 睡眠时间短或睡眠质量差的人减掉的脂肪量较少, 每增加 1 小时的睡眠, 脂肪减少会增加 0.77% [50]。此外一项对 14 名接受减肥手术患者的小型试点研究中, 强调了睡眠在术后 6 年和 9 年长期维持减重的作用[51]。

5. 改善睡眠对减重的作用

研究显示, 更好的睡眠质量和更长的睡眠时间都与更高的减重成功率有关[52]-[54]。睡眠时间的增加和睡眠障碍的纠正可能伴随着调节食欲激素的稳定, 从而使得葡萄糖耐量增强和皮质醇水平的降低[55]。针对睡眠干预的结果表明, 改善睡眠持续时间或睡眠质量可能有助于减少儿童体重增加[9]。此外, 在实验性睡眠不足的情况下, 改用充足的睡眠时间表减少了成年人的食物摄入量[56]。即使每周睡眠的增加也与能量限制期间脂肪量的更大减少有关[50] [57]。因此, 延长睡眠时间被作为一种降低肥胖风险的策略[58]。然而值得注意的是, 在工作周内睡眠不足时, 周末恢复睡眠并不能防止能量摄入和体重的增加[59]。

6. 结语

这篇综述强调了睡眠剥夺引起肥胖的证据, 睡眠剥夺已被证明会通过调节食欲相关激素的表达和功能导致机体的能量正平衡, 并且通过奖赏系统等进一步促进肥胖的发展。随着研究的深入, 改善睡眠对减重的作用日渐明确, 因此, 在对超重及肥胖患者的体重管理过程中应积极给予睡眠指导。进一步的研究应侧重于确定有效的睡眠模式, 可以为预防和治疗肥胖症提供理论支持及健康指导。

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