

• 科研论著 •

减重率与肥胖青少年静息代谢率变化量的剂量-效应关系研究

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摘要: **目的** 探究肥胖青少年减重率与静息代谢率变化量间的剂量-效应关系, 为肥胖青少年预防体重反弹提供科学精准的理论支撑。**方法** 2023 年 7—8 月, 招募 10~17 岁肥胖青少年 60 名, 进行 4 周减重干预。减重率 = (干预后体重 - 干预前体重) / 干预前体重 × 100%; 采用气体代谢分析仪收集氧气吸入量和二氧化碳呼出量, 代入 Weir 公式求得静息代谢率, 计算调整体重的 Δ 相对静息代谢率 = 干预后 - 干预前。采用配对样本 t 检验比较减重干预前后体重和相对静息代谢率差异, 线性回归方法分析减重率与 Δ 相对静息代谢率的剂量-效应关系, 限制性立方样条(RCS)分析减重率与 Δ 相对静息代谢率的阈值效应。**结果** 最终纳入 49 名肥胖青少年进行统计分析, 男性 26 例, 平均年龄 (13.15 ± 1.80) 岁, 体质指数(BMI)为 (31.20 ± 3.91) kg/m²。4 周减重后体重明显下降 ($t = 20.486, P < 0.05$), 干预前后相对静息代谢率差异无统计学意义 ($t = 0.222, P > 0.05$)。线性回归分析显示, 减重率每增加 1%, Δ 相对静息代谢率增加 0.874 kCal/(d · kg) (95% CI: 0.185 ~ 1.563, $P < 0.05$)。RCS 结果表明调整年龄、性别、基线体重后, 减重率与肥胖青少年 Δ 相对静息代谢率存在非线性的剂量-效应关系 ($P < 0.05$); 当减重率 ≤ 7.90%, 每增加 1% 的减重率, Δ 相对静息代谢率增加 2.245 kCal/(d · kg) (95% CI: 1.032 ~ 3.467, $P < 0.05$)。当减重率 > 7.90%, 减重率的增加并不能显著增加 Δ 相对静息代谢率 ($P > 0.05$)。**结论** 肥胖青少年 4 周减重率与静息代谢率变化量存在非线性的剂量-效应关系和阈值效应。当减重率不超过 7.90%, 其与静息代谢率变化量间存在量化关系, 增加减重率可逐渐提升静息代谢率变化量; 一旦超过 7.90%, 两者间关系并不显著。

关键词: 减重率; 静息代谢率; 剂量-效应关系; 肥胖; 青少年

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Dose-response relationship between weight loss rate and changes in resting metabolic rate of obese adolescents

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Abstract: **Objective** To investigate the dose-response relationship between weight loss rate and changes in resting metabolic rate (RMR) in adolescents with obesity, so as to provide scientific and accurate theoretical support for the prevention of weight rebound in adolescents with obesity. **Methods** From July to August 2023, sixty obese adolescents aged 10 - 17 were recruited and underwent a 4-week weight loss intervention. The weight loss rate was calculated as [(post-intervention weight after intervention - pre-intervention weight) / weight before intervention] × 100%. A gas metabolism analyzer was used to collect oxygen intake and carbon dioxide exhalation, which were then substituted into the Weir formula to determine RMR. The adjusted Δ relative resting metabolic rate (Δ RMR) per kilogram of body weight change was calculated as the difference between post-intervention and pre-intervention values. Paired-sample t -test was used to compare differences in weight and relative RMR before and after the weight loss intervention. Linear regression analysis was conducted to assess the dose-effect relationship between weight loss rate and Δ RMR, while restricted cubic spline (RCS) analysis was employed to examine threshold effects. **Results** Forty-nine obese adolescents were included in the statistical analysis, (including 26 males) with a mean age of (13.15 ± 1.80) years and a mean body mass index (BMI) of (31.20 ± 3.91) kg/m². Significant weight loss was observed after the 4-week intervention ($t = 20.486, P < 0.05$), while there was no significant change in relative RMR before and after the intervention ($t = 0.222, P > 0.05$). Linear regression

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analysis showed that for every 1% increase in weight loss rate, ΔRMR increased by $0.874\text{kCal}/(\text{d} \cdot \text{kg})$ ($95\% \text{CI}$: 0.185 to 1.563 , $P < 0.05$). RCS results indicated a nonlinear dose-effect relationship between weight loss rate and ΔRMR among obese adolescents after adjusting for age, sex, and baseline weight ($P\text{-Nonlinear} < 0.05$). Specifically, when the weight loss rate was $\leq 7.90\%$, each additional 1% of weight loss was associated with an increase in ΔRMR by $2.245\text{kCal}/(\text{d} \cdot \text{kg})$ ($95\% \text{CI}$: 1.032 to 3.467 , $P < 0.05$). However, when the weight loss rate exceeded 7.90% , further increases in the weight loss rate did not significantly elevate ΔRMR ($P > 0.05$). **Conclusions** There is a nonlinear dose-response relationship and threshold effect between 4-week weight loss and changes in RMR among obese adolescents. When the weight loss rate does not exceed 7.90% , there is a quantitative relationship with changes in RMR, such that increasing the weight loss rate gradually enhances changes in RMR. Once the weight loss rate exceeds 7.90% , the relationship between them is not significant.

Keywords: weight loss rate; resting metabolic rate; dose-response relationship; obesity; adolescents

青少年肥胖已成为一个严重的全球健康问题,其发病率在世界范围内呈上升趋势^[1-2]。研究表明,肥胖从青少年期至成年期可持续存在,是冠心病、2型糖尿病、高血压、血脂异常、非酒精性脂肪肝病和过早死亡的危险因素^[3-4]。肥胖发病率的上升很大程度上归因于能量代谢的失衡^[5]。而静息代谢率占每日总能量消耗的 $50\% \sim 70\%$,低静息代谢率被认为是肥胖和体重反弹的主要原因^[6]。此外,减重是改善肥胖及其相关疾病的直接方式^[7],但仅有 15% 的肥胖个体能够长期维持减重效果^[5]。因此,减重过程中保留静息代谢率愈发重要。既往研究聚焦于相同减重率、不同减重周期对静息代谢率的影响^[8-9]。但尚无研究考虑相同的减重周期内,不同减重率与静息代谢率变化量的关系。因此,目前缺乏足够证据指出肥胖青少年短期保留静息代谢率的最佳减重剂量。本研究旨在监测相同干预环境与周期下的肥胖青少年减重率及静息代谢率,深入探讨两者存在的剂量-效应关系及阈值效应,为肥胖青少年改善短期减重效果,预防体重反弹提供科学精准的理论支撑。

1 对象和方法

1.1 对象 2023 年 7—8 月,从华南地区封闭式减肥营地招募年龄为 $10 \sim 17$ 岁的肥胖青少年 60 名,纳入标准:1)符合国家卫计委发布的 WS/T 586-2018《学龄儿童青少年超重与肥胖筛查》^[10] 的肥胖标准;2)平时未进行规律的体育运动;3)通过运动风险筛查。排除标准:1)患有严重肥胖并发症^[11];2)服用药物治疗的肥胖青少年,受试对象符合以上一项排除标准,均不纳入研究。所有受试者及监护人均签署知情同意书。该研究已通过广州体育学院伦理委员会审批(编号:2023LCLL-36)。

1.2 方法

1.2.1 样本量估算 采用 G-power 3.1 软件计算样本量。本研究属于自身前后对照试验,选择双侧

检验,效应量为 0.50 , $\alpha = 0.05$,检验效应为 0.80 ,最少需要 34 名受试者。

1.2.2 体格测量 采用身高计和体重秤测量受试者身高和体重,身高以 cm 为单位,体重以 kg 为单位,计算体质量指数 (body mass index, BMI) = 体重/身高² (kg/m^2),精确到小数点后一位。

1.2.3 静息代谢测试 测试前要求受试者禁食 2h 且 24h 内禁止饮用咖啡、酒精类饮品^[12]。受试者到达测试房间(室温在 $25^\circ\text{C} \sim 27^\circ\text{C}$),静坐休息 20min 后由专业人员佩戴气体代谢分析仪 (Cortex Meta Max 3B, CORTEX, 德国)进行 15min 的静息代谢测试。测试过程前 10min 为适应期,截取平稳的后 5min 数据进行分析(平稳定义为 VCO_2 和 VO_2 的变化量小于 10%)。根据 Weir 公式^[13] 计算静息代谢率 (kCal/d) = $[3.941 \times \text{VO}_2 (\text{L}/\text{min}) + 1.11 \times \text{VCO}_2 (\text{L}/\text{min})] \times 1440$,调整体重后得到相对静息代谢率 [$\text{kCal}/(\text{d} \cdot \text{kg})$]。

上述体格测量和静息代谢测试由专业测试人员在受试者减重干预前后各执行 1 次。

1.2.4 减重方案 所有受试者在营期间接受中等强度有氧运动,强度区间为靶心率 = 安静心率 + $(20\% - 40\%) \times$ 心率储备(心率储备 = $220 -$ 年龄 - 安静心率)^[14]。使用心率仪 (Polar, OH1, 芬兰)实时监测受试者运动强度。运动方式有健身操、折返跑、慢跑等,运动时间为 $4\text{h}/\text{d}$ (上下午各 2h),运动频率为 $6\text{d}/\text{周}$,持续 4 周。受试者的饮食方案统一由专业营养师制定,总能量摄入根据静息代谢率确定。能量构成比为碳水化合物约占总能量的 $55\% \sim 65\%$,蛋白质约占 $10\% \sim 15\%$,脂肪约占 $20\% \sim 35\%$,三餐能量比为 $3:4:3$ 。餐食中包含膳食纤维、维生素、矿物质等营养物质,从而保证营养均衡与科学^[15]。

1.3 统计学方法 所有数据经正态性检验后表示为 $\bar{x} \pm s$ 。减重干预前后各指标比较采用配对样本 t 检验。减重率 = $(\text{干预后体重} - \text{干预前体重}) \times$

(-1)/干预前体重 $\times 100\%$, Δ 相对静息代谢率 = 干预后 - 干预前。采用线性回归分析减重率(%)与 Δ 相对静息代谢率间的剂量-效应关系;调整年龄、性别、基线体重后,采用 3 个节点的限制性立方样条(restricted cubic spline, RCS)建模来探讨减重率(%)与 Δ 相对静息代谢率间的非线性关系,若存在非线性关系,则采用分段线性回归模型探讨剂量-效应关系。数据分析与限制性立方样条绘制分别采用 SPSS 26.0 与 Rstudio 4.4.0 的 rms 和 ggplot 包。 $P < 0.05$ 为差异有统计学意义。

2 结果

2.1 受试者基本特征 因 11 人未完成 4 周干预,

故排除,排除样本与纳入样本基本特征间差异无统计学意义($P > 0.05$)。本研究最终纳入 49 名肥胖青少年,其中男生 26 名,女生 23 名,平均年龄为(13.15 $\pm$ 1.80)岁,身高为(161.50 $\pm$ 6.20)cm, BMI 为(31.20 $\pm$ 3.91)kg/m²。

2.2 受试者减重干预前后体重和静息代谢率变化情况 49 名肥胖青少年体重和静息代谢率较干预前均显著下降,差异有统计学意义($t = 20.486$, -3.041 , $P < 0.05$)。相对静息代谢率较干预前并无显著改善($P > 0.05$)。见表 1。

2.3 减重率与 Δ 相对静息代谢率的剂量-效应关系分析 线性回归分析显示,减重率每增加 1%, Δ 相对静息代谢率增加 0.874kCal/(d $\cdot$ kg)(95%CI:

表 1 受试者减重干预前后体重和静息代谢率变化情况

Tab. 1 Changes in body weight and resting metabolic rate before and after the weight-loss intervention

指标	干预前(n=49)	干预后(n=49)	变化量(Δ)	t 值	P 值
体重(kg)	81.67 $\pm$ 14.34	75.58 $\pm$ 13.19	-6.08 $\pm$ 2.07	20.486	<0.001
静息代谢率(kCal/d)	2 280.25 $\pm$ 447.82	2 116.48 $\pm$ 353.49	-163.78 $\pm$ 377.00	3.041	0.004
相对静息代谢率[kCal/(d $\cdot$ kg)]	28.19 $\pm$ 4.37	28.34 $\pm$ 4.28	0.15 $\pm$ 4.85	0.222	0.826

0.185~1.563, $P = 0.014$);调整年龄、性别、基线体重后,减重率每增加 1%, Δ 相对静息代谢率增加 0.913kCal/(d $\cdot$ kg)(95%CI: 0.192~1.634, $P = 0.014$)。

2.4 减重率与 Δ 相对静息代谢率的阈值效应分析

调整年龄、性别、基线体重后,减重率与肥胖青少年 Δ 相对静息代谢率存在非线性的剂量-效应关系($P < 0.05$)。见图 1。分段回归显示,当减重率 $\leq 7.90\%$,每增加 1%的减重率, Δ 相对静息代谢率增加 2.13kCal/(d $\cdot$ kg)(95%CI: 0.913~3.348, $P < 0.05$);调整年龄、性别、基线体重后,每增加 1%的减重率, Δ 相对静息代谢率增加 2.245kCal/(d $\cdot$ kg)(95%CI: 1.032~3.467, $P < 0.05$)。当减重率 $> 7.90\%$,减重率与 Δ 相对静息代谢率的剂量-效应关系并不显著($P > 0.05$)。见表 2。

3 讨论

本研究发现,在一定范围内(减重率 $\leq 7.90\%$),肥胖青少年减重率与 Δ 相对静息代谢率间存在显著的剂量-效应关系;即随着减重率的增加,肥胖青少年的相对静息代谢率降低越少,甚至出现干预后相较于干预前增加的情况。但当减重率 $> 7.90\%$,随着减重率的增加, Δ 相对静息代谢率转变为下降趋势,但两者关系并不显著。这说明减重率与肥胖青少年的 Δ 静息代谢率之间存在显著的非线性剂量-

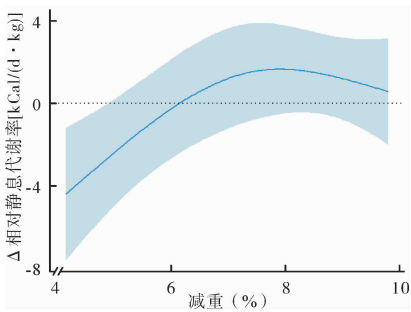


图 1 减重率与 Δ 相对静息代谢率阈值效应关系图

Fig. 1 Relationship between weight loss rate and Δ relative resting metabolic rate threshold

表 2 减重率与 Δ 相对静息代谢率的阈值效应分析

Tab. 2 Threshold effect analysis of weight loss and Δ relative resting metabolic rate

减重率 (%)	模型 I		模型 II	
	β 值(95%CI)	P 值	β 值(95%CI)	P 值
≤ 7.90	2.130(0.913, 3.348)	0.001	2.245(1.032, 3.467)	0.001
> 7.90	-0.409(-2.685, 1.867)	0.712	-1.077(-3.744, 1.590)	0.406

注:模型 I 表示未调整;模型 II 表示调整年龄、性别、基线体重。

效应关系和阈值效应。

3.1 减重引起的代谢适应是影响静息代谢率变化的重要因素 吴一凡等^[16]横断面研究指出体重、脂肪量、瘦体重与肥胖男青年静息代谢率呈正相关,而 Zhang 等^[17]指出体重、瘦体重与肥胖青少年相对静息代谢率呈负相关。其差异的可能原因考虑是静息

代谢率的单位不同所致。针对肥胖人群,已有研究建议应使用调整体重的相对静息代谢率^[18]。实验研究发现,减重率与静息代谢率变化量存在量化关系,Johannsen 等^[19]对严重肥胖受试者采用剧烈运动结合限制饮食减重方案,丢失 $(38 \pm 9)\%$ 的体重后 $[(17 \pm 8)\%$ 瘦体重和 $(83 \pm 8)\%$ 脂肪量],虽然尽可能保留了瘦体重,但静息代谢率仍不成比例下降,出现代谢适应;另外指出体重的丢失和静息代谢率之间不是线性关系,第 6 周下降 (244 ± 231) kCal/d,第 30 周下降 (504 ± 171) kCal/d。本研究在此基础上采用适合肥胖人群研究的相对静息代谢率指标发现,减重率与 Δ 相对静息代谢率呈倒“U”型曲线关系。当减重率 $\leq 7.90\%$,随着减重率增加,肥胖青少年的相对静息代谢率降低逐渐减少,这可能与运动引起的骨骼肌工作效率增加相关。一方面脂肪产热量的丢失被骨骼肌所代替,而肌肉组织在静息状态下的代谢率又高于脂肪组织^[18],故出现 Δ 相对静息代谢率为正的情况;另一方面,骨骼肌转化葡萄糖的能力被氧化脂肪所替代可逐渐增加产热降低体重^[20]。但当减重率 $> 7.90\%$,随着减重的增加, Δ 相对静息代谢率转变为下降趋势,这考虑机体出现新一阶段的代谢适应,启动机体自我保护机制,节省能量消耗,恢复能量平衡^[18]。Martin 等^[21]指出减重过程中代谢适应对静息代谢率的影响可达 40%。而 Koehler 等^[22]却发现在正常体重女性中,尽管去脂体重被完全保留,采取热量限制和运动结合减重方式结果还是出现了低于预期的体重下降;严重能量亏缺组(< 5 kg)中静息代谢率降低的参与者比静息代谢率未下降的参与者丢失了更多的体重。这说明减重伴随代谢适应,在一定范围内可根据代谢适应的严重程度判断减重效果。本研究与之相反,可能原因是受试对象年龄、性别、体重等因素不同所致。

3.2 减重伴随瘦体重、代谢紊乱和激素水平改善从而影响静息代谢率 既往研究集中于减重周期与静息代谢率的关系。一项囊括了 13 项减重研究(包含热量限制、运动和手术,0.2~3.2 kg/周)的综述发现减重周期长,静息代谢率下降少;反之,静息代谢率会因去脂体重丢失多和体脂肪丢失少而下降更多;但是这些差异在减重后 2~4 周就减弱,且不影响 9~33 个月体重恢复的速度^[7]。Coutinho 等^[23]发现当受试者处于能量平衡状态,快速(4 周)和渐进(8 周)具有相似的体重丢失($\approx 9\%$),但渐进减重具有减慢静息代谢率下降的优势。上述研究强调减重周期越长,代谢适应越轻,但现实条件易出现受试者难以坚

持的情况。而探讨相同的减重周期中减重率与 Δ 静息代谢率的关系,有助于在有限的时间内达到科学精确的减重效应最大化。有研究对肥胖受试者进行生活方式干预,4 年后随访研究发现减重率与最大脂肪氧化速率之间是 U 型抛物线关系,认为预防体重反弹与机体高摄氧量、高肌内脂肪氧化能力和较高的血浆脂肪酸有关^[24]。而静息代谢率的决定因素除了代谢适应,主要与运动减重的同时伴随瘦体重^[25]、代谢紊乱^[26]和激素水平^[27-28]的改善相关。Siervo 等^[8]采用限制饮食减重方式发现肥胖男性减重率与实测静息代谢率和预测静息代谢率差值呈负相关($r = -0.42, P = 0.01$),表明减重率越大,静息代谢率下降越多。本研究与之相反,考虑是减重方式不同所致。饮食、手术减重并不能保留瘦体重高产热的组织,机体易出现适应性产热^[29-31]。然而,运动减重对降低代谢适应、能量补偿是行之有效的。本研究采用运动减重方式,结果显示随着减重率的增加, Δ 相对静息代谢率不降反增甚至出现阈值现象。这一定程度与运动可通过增加瘦体重提升骨骼肌线粒体活性、增加肌肉生长和修复过程所需能量、调节激素分泌、改善代谢紊乱等途径改善静息代谢率有关^[25-26, 32-34]。

本研究存在以下优势和局限性。使用限制性立方样条分析肥胖青少年减重率和 Δ 静息代谢率间的非线性关系,避免了线性回归分析两者简单的线性关系。但本试验仅设计了 4 周短期减重,长期减重是否存在这一剂量-效应关系还有待证实;其次,未评估机体代谢健康水平,未来需要探究静息代谢率与代谢指标改善之间的关系,但是上述指标的改善都是源于减重,有研究证实仅运动未减重的受试者并没有提升代谢水平^[35];最后,样本量较少,但研究对象和测试指标的特殊性限制了样本量的进一步扩大。

综上,肥胖青少年 4 周减重率与静息代谢率变化量间存在非线性的剂量-效应关系和阈值效应。当减重率不超过 7.90%,与静息代谢率变化量间存在量化关系,增加减重率可逐渐提升静息代谢率变化量;一旦超过 7.90%,两者间关系并不显著。

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