

Physical Exercise, Energy Expenditure and Weight Loss: An Assumption not Always Observed in Practice

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The increased prevalence of overweight and obesity observed in recent decades has been attributed mainly to behavioral changes, such as excessive food consumption and reduction in physical activity level over time, leading to a positive energy balance. Due to the technological advances in recent years, less physical effort is required to perform daily tasks. Moreover, the lifestyle has become more sedentary, with an increasing amount of time spent using digital gadgets, such as computers, televisions and mobile phones. However, Westerterp & Speakman¹ did not find a decrease in physical activity energy expenditure during the last 30 years preceding their study, despite an increase in the prevalence of obesity.¹ Other studies have also reported that the total energy expenditure of rural populations was similar to that of populations living in developed countries, despite the clear difference in behavioral patterns, particularly in relation to daily physical activities.²⁻⁴ This study aimed at presenting the “compensatory effect” as a possible explanation for these findings. When there is an increase in physical activity at a given time, behavioral and metabolic changes take place to maintain an energy setpoint, as presented below.

Physical exercise has been recommended as an important component for the prevention and treatment of obesity. For substantial adults’ health benefits, at least 150 minutes of moderate-intensity aerobic exercise, or

75 minutes of vigorous-intensity aerobic exercise; in addition, 2-3 times per week of resistance training is recommended.⁵ However, for a clinically significant reduction in body weight, Donnelly et al.,⁶ recommended that individuals should gradually increase the amount of physical activity and achieve a weekly volume of moderate physical exercise of over 250 minutes.⁶ The authors also reported a dose-response effect between physical activity and weight loss.

It is believed that the greater amount of physical exercise (duration and intensity), the higher the total energy expenditure, and consequently, greater loss of body weight. However, energy expenditure through exercise does not explain the variation in body weight observed in the long term. Several studies have shown a weight loss below the expected levels,^{7,8} even in controlled studies with a high rate of patient compliance.^{9,10} These results do not support the classic additive relationship between physical exercise and energy expenditure, thereby raising questions about the real impact of this strategy on weight control.^{8,11}

Some theories have tried to explain the reason for lower-than-expected weight loss and the difficulty in maintaining it over time with increased physical activity. In 1998, Rowland proposed the “activitystat” theory and defined it as a homeostatic mechanism, wherein a biological control center would be responsible for controlling the physical activity (or energy expenditure), similarly to other biologically controlled variables such as body temperature.¹² Whenever an imbalance occurs, the regulatory mechanisms are activated to restore a particular setpoint. According to this theory, an increase

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in the amount of physical activity at one time would be compensated with less physical activity at another time, to reestablish the balance of the system. This compensatory effect on subsequent spontaneous physical activities has been observed in several studies in children, elderly individuals, and young adults;¹³⁻¹⁵ however, other studies do not corroborate these findings.¹⁶

Aligned with this theory and in contrast to the classic “additive model,” Pontzer et al. (2016) proposed the “restricted model” to explain the relationship between energy expenditure and physical activity.¹⁷ According to this model, an individual’s metabolism adapts in response to the increased physical activity, and above a specific “critical point,” the increase in physical activity volume does not cause a concomitant increase in energy expenditure. According to the authors, this compensatory effect can be explained by behavioral (longer periods of sitting than standing or less fidgeting throughout the day compared to a period with no exercise) or metabolic changes (decreased resting metabolic rate, increased muscle efficiency for the same activity demand, or even hormonal changes, such as a decrease in estrogen and testosterone production, and a decrease in the activities of the immune system).¹⁷

This compensatory phenomenon in relation to physical activity is also observed in some species of birds and mammals. The “energy budget” of these animals is also limited, and any increase in energy expenditure to maintain the basal metabolism would leave a less amount of energy available for other functions, such as flying, fighting, or hunting.¹⁸

The effect of physical exercise in reducing body weight is further impaired due to compensatory responses on the other side of the energy balance. Martin et al.¹⁶ observed that overweight individuals submitted to high volumes of physical exercise (1760 Kcal/week) for a period greater than six months showed increased appetite and caloric intake compared to the moderate exercise group (700 Kcal/week) and the group without exercise. These results were corroborated by Myers et al.,¹⁹ who also observed increased hunger and food intake after 12 weeks of daily physical training (5 x 500 Kcal/week).¹⁹

Although these compensatory responses are frequently reported in clinical and epidemiological studies, there is significant interindividual variability in weight loss.²⁰ In a study conducted by McNeil et al. (2017) to evaluate the compensatory effect of exercise in 530 postmenopausal women, the authors observed that 9% of women lost

more weight than expected after 12 months of moderate exercise (150, 225, or 300 minutes per week); 20% compensated between 0 and 50%, indicating weight loss lower than expected; around 44% compensated between 50% and 100%, and 27% of women presented compensations above 100%, gaining weight at the end of the intervention.²¹ The mechanisms by which individuals respond in such a diverse way to the same stimulus are still a matter of debate; moreover, this compensatory effect seems to be asymmetric, with more intense forces resisting weight loss compared to those acting in response to weight gain.

Therefore, a linear relationship between the prescribed energy deficit and the achieved weight loss should not be assumed. The regulation of the energy balance seems to be a complex and dynamic process, wherein a disturbance in one component can trigger changes in one or more components of energy expenditure and/or food intake. The relationship between individual characteristics (age, sex and nutritional status) and the variables of physical exercise (type of exercise, frequency, intensity and duration) with the compensatory mechanisms requires investigation, as well as the physiological mechanisms responsible for these changes.

The discussion needs to go beyond “eat less and exercise more.” Rather, the focus should be on understanding the causal factors and mediating mechanisms for the relationship between physical exercise (and/or diet) and weight loss. Identifying factors that resist weight loss maintenance in some individuals, thereby “disrupting” the entire (or partial) effort, is necessary. Based on the compensatory effects discussed here, the expectation of reducing obesity with programs based exclusively on physical activity should be reduced.⁸ However, regardless of the achieved weight loss response, the health benefits associated with regular physical activity are quite significant, as reported in the literature, and the adoption of an active lifestyle with a decrease in sedentary behaviors should be categorically encouraged.

Long-term clinical trials are still necessary for a better understanding of the effect of physical exercise on energy expenditure, food intake, and body weight, and to identify factors (physiological and behavioral) related to the different responses presented by the individuals. The key question is no longer “if” the compensatory effect occurs, but rather “when,” “how,” and “what” are the individual characteristics that increase the susceptibility to this phenomenon.

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