# Effects of excess post-exercise oxygen consumption and resting metabolic rate in energetic cost

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## ABSTRACT

The constant growth of obesity and overweight only goes to show the need of intervention to reverse those figures. In this context, physical activity can contribute with a double effect, through acute and chronic physiological changes: in the first condition one can find the energetic cost from exercising and recovery (EPOC – excess post-exercise oxygen consumption), and in the second, the resting metabolic rate (RMR). Thus, this revision's goal was to investigate the effect of EPOC and RMR as supporting factors in weight-control programs, willing to discuss the different results found in literature, concerning both magnitude and length of EPOC, as well as discussing the effects of exercising in RMR. Research shows, in general terms, that the most intense exercises are able to promote a bigger EPOC when compared to exercises of lower intensity, while a bigger EPOC was found in resistive exercises when compared to aerobic ones. Concerning RMR changes, the acute results show significant increase at it; however, long-term results are more discrepant, due to the difficulty in measuring this variable without overestimating it. In summary, literature points that periodicity of a training that can maximize both EPOC and RMR may be an important factor to weight-losing and, although energetic cost of these variables in a therapy session seem rather small, it can be significant in a long-term relation. However, new studies are important to confirm these evidences.


**Keywords:** Obesity. Weight loss. EPOC. RMR.

## INTRODUCTION

Daily energetic cost can be divided in three components: RMR (resting metabolic rate); food thermal effect and energetic cost associated with the physical activity[1]. Physical activities promote an increase of the total energetic cost both in an acute and chronic manner. The first condition refers to the energetic cost itself during the exercise performance and during the recovery phase; while the second refers to the alteration of the resting metabolic rate RMR[2].

Concerning the acute effect, it is well established that the post-exercise $O_2$ consumption immediately returns to the resting indices. Such energetic demand during the post-exercise recovery period is known as *"Excess Post-exercise Oxygen Consumption"* – EPOC[3].

The excess post-exercise oxygen consumption consists of a fast component and an extended component. The EPOC fast component occurs within 1 h[4]. Although the precise cause of these responses is not well clarified yet, it is probable that these factors contribute for: the ATP/CP resynthesis; ions behavioral redistribution (increase in the activity of the sodium and potassium bomb); lactate removal; tissue damage restoration; as well as recovery of the increase of the HR and the body temperature increase[4-8]. During the extended component, processes for the physiological homeostasis return continuously occur; however, in a much lower level. These processes may include the Krebs cycle with a larger utilization of free fatty acids[4,6]; effects of several hormones such as cortisol, insulin, ACTH, thyroid hormones and GH[5]; hemoglobin and myoglobin resynthesis[7]; increase of the sympathic activity[7]; increase of the mitochondrial breath by the increase of the norepinephrine concentration[9]; glycogen resynthesis and temperature increase[10].

Moreover, results of several studies concerning the EPOC magnitude and duration have been discrepant. While several studies show that the EPOC may last for hours[4-5,7,10-15], other have concluded that the EPOC is transitive and minimal[14,16-18].

Several studies have analyzed the EPOC contribution in weight loss programs, once it is a result of a negative daily energetic balance between consumption and energetic cost[19]. Bahr *et al.*[10] had considered the EPOC as an important factor in weight control, once exercising requires extra energy besides the one expected in physical activity. Other studies corroborate that the increased metabolism magnitude during recovery has an important implication in the prescription of ponderal reduction programs[9,12-13,19-20].

Ponderal reduction is also related with chronic alterations of physical activities, that is, of the resting metabolic rate (RMR). The RMR is defined as the energetic cost necessary to the physiological processes maintenance in the post-absorptive state, reaching up to 60-70% of the total energetic cost, depending on the physical activity level[21].

The high interest on ponderal reduction is due to the fact that obesity is currently considered by the World Health Organization a public health problem[22]. An estimate considering the current prevalence of obese individuals highlights that if such proportion remains, in the year 2230 100% of the North-American population will be obese[23]. Analyzing the Brazilian reality, Meirelles and Gomes[21] showed that in Rio de Janeiro 44% of the men and 33% of the women in the age group from 26 to 45 years present overweight or obesity.

Although obesity is related to several causes, namely genetic; physiological; metabolic; environmental; emotional and cultural ones, all the attention dedicated to the increase of the daily energetic cost is highly valid, once the energetic balance is partly determined by the energy consumption and partly by the energy cost[21,24]. Therefore, a positive energetic imbalance may lead to excessive energy accumulation stored as body fat.

Thereby, the aim of the present study was to investigate the EPOC and RMR effect in the energetic cost, trying to discuss the contrasting results found in the literature concerning the EPOC magnitude and duration, as well as to discuss the exercise effect in the RMR.

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## DEVELOPMENT

### EPOC effect in the increase of the total daily energetic cost with aerobic exercise

The disparities in the results related with the EPOC may reflect differences in many factors, such as: the muscular mass involved in the exercise; intensity and duration; training status; food ingestion (meal's thermal effect); sleep quality of the previous night; environmental conditions; the subject's familiarity with the protocol; temperature and catecholamines concentrations variations; metabolic cost of the lactate removal; substrate utilization and menstrual cycle phase for women[25], pre-exercise anxiety; circadian rhythm; the fact that the majority of the studies did not analyze the same individuals[20] and *overtraining* presence for athletes.

The EPOC increases linearly with the exercise duration[10]. However, the exercise intensity seems to affect both the magnitude and the duration of the EPOC, while the exercise duration affects only its duration[12].

Therefore, the studies have suggested that exercises with higher intensity produce a more extended increase in the EPOC than exercises of shorter intensities (when they have equivalent volume), since shorter intensities exercises cause greater metabolic stress, being necessary a larger energy cost in order to return to the homeostasis condition[5,13-14]. Moreover, a higher level of activity of the sympathic nervous system (stimulated by the catecholamines) can also contribute to the increase of the post-exercise metabolic rate, since the epinephrine and the norepinephrine stimulate the mitochondrial breath and the cellular function, facilitating the sodium and potassium passage through the cellular membrane, increasing the ATP production and the oxygen cost[7,9,26].

An increase of 20 to 35% in the lipolytic responsiveness in the adipocyte after the exercise is found[4,6]. However, the oxidation rate of lipids is even higher after high intensity exercise, once the glycogen synthesis is increased after exercise in order to replace the glycogen used during exercise. The lipids oxidation is also associated with the increase of the *turnover* of the free fatty acids concomitantly; the *turnover* increase of protein can also contribute to a higher EPOC[19]. Besides these factors, the higher intensity exercise is associated with a greater synthesis of hemoglobin and myoglobin[27], as well as to seem to be inversely associated with the obesity rates[28].

Some works have analyzed the EPOC and the energetic cost comparing continuous exercise and submaximal intervaled protocols[20,29] and submaximal continuous and supramaximal intervaled exercises[13]. All these studies demonstrated a greater energetic cost for the more intense exercises. Starting from the premise that it is possible to perform more minutes at high intensity with intermittent exercise if compared with continuous exercise[13], overweighed individuals can exercise for a shorter time at an intensity that produces a higher EPOC. Such fact occurs once in the majority of the times these individuals are not conditioned besides not being fond of physical activities.

Nonetheless, it is worth mentioning that the EPOC is relatively short after exercises of moderate intensity and duration, < 70% of $\dot{V}O_{2\ max.}$ and/or < 60 minutes[12,25,30], since the EPOC magnitude after aerobic exercise depends on both factors (exercise intensity and duration). Conversely, exercises with duration > 60 minutes and/or with an intensity > than 70% of $\dot{V}O_{2\ max.}$ seem to be related to an extended EPOC[4,7,10,12-13]. It has been also proposed that in individuals who want to lose weight, the EPOC magnitude (energetic cost) is more important than duration[25].

### EPOC effect in the total daily increase of the energetic cost with resistive exercise

The studies that compared the resistive exercise with the aerobic one[31-32] emphasized that the resistive exercise would probably cause a greater disturbance in the homeostasis than the aerobic one, suggesting that, due to high intensities involved, it could require a greater energetic cost both during exercise and recovery. Moreover, analyzing only the resistive exercise, it is also suggested that a more extenuate protocol is more efficient in the weight control[15].

Two factors have been attributed to the fact that the resistive exercise produces higher EPOC. The first factor refers to hormone responses that may alter metabolism, specifically catecholamines, cortisol and GH[33-34]. The second refers to the tissue damage followed by the stimulus for the tissue hypertrophy[34], since the protein synthesis is decreased during the exercise itself. However, after exercise there is a compensatory phenomenon, in which the protein *turnover* seems to be stimulated. Besides that, the protein synthesis process requires high energetic demand (6 ATP per mol of made peptide). Such mechanism can also contribute to a long stimulation of the energetic cost after exercise[35].

A study[36] followed 7 healthy men in two weight training circuits; one using 20'' of interval and the other using 60'' at 75% of 20 RM. The results demonstrated that the EPOC was significantly higher in the 20'' protocol, if compared with the 60'' one. The energetic cost in 1 h recovery was of 51 ± 2,84 Kcal x 37 ± 1,97 Kcal respectively. Such difference was also significant; however, the total energy cost (exercise + 1 h of recovery) was significantly higher in the 60'' interval protocol (277,23 Kcal) compared with the 20'' interval protocol (242,21 Kcal). The 20'' protocol resulted in higher fast EPOC component, which was expected once it is proportional to the exercise intensity[37].

There seems to be an optimum combination between intensity and duration in the resistive exercise in order to maximize the EPOC[15]. Nevertheless, this optimum combination still remains obscure. In this study[15], an EPOC for beyond 16 h after 38 minutes of resistive exercise was found. In addition to that, it was also verified that the $\dot{V}O_2$ constantly increased during the entire day, probably due to the accumulation of the effects caused by the daily activities; emotions and circadian rhythm. Although these authors have mentioned the circadian rhythm and emotional issues (which can alter the sympathic activity), they were not controlled in the study.

The effect of resistive exercise on the EPOC was verified in men of 22 to 40 years of age previously trained with resistive exercise. It was observed that after a 90-minute session, the metabolic rate remained high for more than 2 hours after the exercise[38]. Another study[39] investigated the effect of 45' of resistive exercise in trained women on the EPOC. After 120 minutes of recovery, an increase of 18,6% in the resting metabolism was found if compared with the control.

Conversely to the previously mentioned works, a study[8] analyzed 6 adult men for 42 minutes during weight lifting exercise (12 RM), and observed the recovery period for 60 minutes. The metabolic rate was significantly high at the end of the 60 minutes; however, only 19 additional Kcal were used. The authors highlighted that such recovery energy cost would have little effect in the weight regulation. Nevertheless, the fact that the oxygen consumption remained high after 1 hour control did not allow the authors to determine the real EPOC duration and the real energetic cost during this period. Within this context, the oxygen consumption could have returned to the resting values immediately after this period or remained high for hours.

### EPOC and training

There are several powerful mechanisms through which regular exercise could facilitate the body weight maintenance or reduction, which includes: the increase of the total daily energetic cost; appetite reduction; RMR increase; increase of the fat free mass; increase of the meal thermal effect; increase of the excess post-exercise oxygen consumption and increase of the mobilization and

fat oxidation rate[40]. A scheme of this mechanism is described in figure 1.

A group of 15 women was submitted to a weight and aerobic training, where it was verified if a higher energetic cost (EC) during EPOC in the resistive training occurred if compared with the aerobic one when the sessions were equivalent in $\dot{V}O_2$ and duration

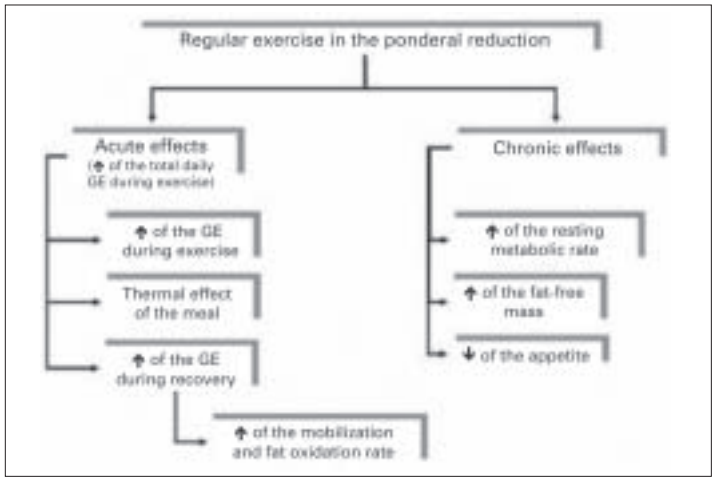


***Figure 1** – The function of regular exercise in the ponderal maintenance and reduction*

(95 Kcal and 64 Kcal respectively, in 30' of recovery)[32]. Nevertheless, these authors pointed that several factors contribute to the recovery from the resistive exercise which, on the other hand, do not occur after aerobic exercise. Studies show that hormonal alterations, particularly of the catecholamines, cortisol and GH, may be substantial, especially if the repetitions per series are high (> 5) and the interval between the series is shorter than 1 minute[29,34]. Moreover, the higher oxidation of lipids may be an important factor derived from the adaptation to training. Trained individuals use more fat in the recovery period than the non-trained ones[41].

### EPOC and obesity

Several studies mention the EPOC effect as a weight loss aid (table 1). Although the words weight loss and obesity have often appeared in the literature, few studies really analyzed such population. Poehlman *et al.*[42] analyzed the effect of aerobic and resistive training in obese women; however, they did not observe the post-exercise oxygen consumption. Such fact shows the importance of further studies which analyze the EPOC in this specific population.

These authors[42] still mention another relevant aspect to the obese subjects' context: the RMR was measured 10 days after the six-month training, and there were not significant chronic alterations in none of the groups.

### RMR: Acute and chronic effects of the exercise

The RMR is the major component of the daily energetic cost[31,43], being modified by several factors such as: time of the day; temper-

**TABLE 1**
**Works that mention EPOC in the ponderal control and reduction**

| Authors | Sample | Exercise | EPOC duration | EC | Comments |
|---|---|---|---|---|---|
| Maehlum *et al.*[55] | 4F, 4M | 80' at 70% of $\dot{V}O_{2max}$ (3 or 4 x at 10-30') | > 12 h | – | EPOC may continue up to 24 h |
| Bahr *et al.*[10] | 6M | 20, 40 and 76' at 70% of $\dot{V}O_{2max}$ | 76': > 12 h | – | 12 h: ↑ 15% in the RMR |
| Sedlock *et al.*[56] | 10M | 20' at 75%; 30' at 50% and 60' at 50% of $\dot{V}O_{2max}$ | Short EPOC | 29,4; 14,3 and 12,1 Kcal | High intensity and short duration exercise (> EPOC) |
| Kaminsky *et al.*[31] | 6F | 50' of running and 2 x 25' at 70% of $\dot{V}O_{2max}$ | 30' | 6, 39 Kcal x 13, 88 Kcal | Intervaled exercise: > EPOC and EC |
| Gore and Withers[12] | 9F | 20, 50 and 80' a 30, 50 and 70% of $\dot{V}O_{2max}$ | 8 h | – | No difference only for 30% of $\dot{V}O_{2max}$ |
| Laforgia *et al.*[13] | 8M | 30' at 70%; 20 x 1' at 105% of $\dot{V}O_{2max}$ | 1 h; 8 h | – | 1st work to use equal loads in the sub. and supramaximal |
| Dawson *et al.*[39] | 8F | 34' at 67%; 41' at 55% and 49' at 45% of $\dot{V}O_{2max}$ | 13,9 to 14,1' | – | Same total work (through the energetic cost) |
| Quinn *et al.*[9] | 8F | 20, 40 and 60' at 70% of $\dot{V}O_{2max}$ | > 3 h | 42,6; 59,6 and 89,2 Kcal | EPOC: ↑ of RMR in ~16,5% |
| Fukuba *et al.*[52] | 5F | 60' at 70% of $\dot{V}O_{2max}$ | 7 h | – | 4 diets in the 2 phases of the menstrual cycle |
| Brockman *et al.*[20] | 5F | 2 h at 25%; 10' at 81% 7 x 2' at 89% of $\dot{V}O_{2max}$ | > 1 h | – | ↑ of 12, 23 and 44% of metabolism |
| Imamura *et al.*[27] | 7F | 30 and 60' at 60% of $\dot{V}O_{2max}$ | 46' and 116' | – | EPOC magnitude: more import. To weight loss |
| Ryan *et al.*[33] | 15F | 12 weeks of Resistive train. | – | Additional of 50 Kcal/day | ↑ significant of RMR |
| Binzen *et al.*[41] | | 45' of resistive exercise | > 2 h | 29,2 Kcal | EPOC is insignificant fot these authors |
| Burleson *et al.*[34] | 15M | 27' (walking) and 27' (resistive exercise) | > 1:30 h | 30' of recov.: 64 and 95 Kcal | Train. Volume: resistive is very important |
| Haltom *et al.*[38] | 7M | Resistive train. (20 and 60'' of interval) | > 1 h | 10,3 and 7,4 Kcal | The EC of exercise + recov. was > in the 60" protocol |
| Melby *et al.*[40] | 2 x 6M | Resistive exercise | 2 h | – | ↑ of 4,7 of MRM in the following morning |

M = male; F = female; EC = energetic cost; ↑ = increase; train = training; dur = duration; sub = submaximal; RMR = resting metabolic rate; recov = recovery.

ature; food ingestion; caffeine ingestion; exercise type and stress[12]. The RMR decreases with age and body mass reduction. Such fact is partly due to the decrease of lean body mass and of the sympathic nervous system activity[31].

Concerning the acute effects of exercise, Osterberg and Melby[44] verified the resistive exercise increases the RMR for 16 hours after exercise in approximately 4,2%, suggesting an increase of approximately 50 Kcal/day in the RMR with physical exercise[33]. Another study[38] verified that the RMR in the following morning after a resistive exercise was 4,7% higher than the one measured in the morning prior to the exercise.

In order to analyze the chronic effect of exercise, obese women in the post-menopause were followed for 16 weeks of resistive training. The results demonstrated significant increase (approximately 4%) of the RMR and muscular mass in both obese and non-obese groups[31]. Moreover, the obese subjects obtained significant decrease of the body mass, fat mass and fat percentage, showing that the resistive exercise may be an important component integrated to weight loss programs in post-menopause women. This study adds an important aspect in the literature when demonstrates that the resistive training followed by body mass reduction did not result in RMR reduction. Corroborating this result, an increase of 7,7% of RMR was found in elder men with a similar protocol[45].

Nevertheless, some authors[40] analyzed the effects of 12 weeks (3 x per week) of aerobic and resistive exercise in the body weight, body composition and RMR in 18 elders (approximately 61 years) who had body mass reduction of 9 ± 1 Kg in 11 weeks. The results showed that none of the two types of exercise could revert the decrease of the RMR of approximately 15% (260 Kcal/day) or the fat oxidation as consequence of the previous body mass reduction. Likewise, 2 groups of non-obese women were trained for six months; one with resistive exercise and the other with aerobic exercise. The daily energetic cost was measured 10 days after the 6 months. It was concluded that the physical training benefits in the increase of the energetic cost is primarily derived from the exercise effect and not from a chronic increase of the daily energetic cost in no-obese women[42].

Nonetheless, it is worth mentioning that the data about the long term-training effects on the RMR are contradictory. This can probably occur due to the difficulty to quantify the exact time of recovery of a previous training, when one desires to measure only its chronic effect, excluding the acute effect of the last session (not to overestimate the RMR).

Genetic and physiological factors that influence the RMR were also investigated, namely the circulating levels of the thyroid hormones[46]; *turnover* of the *uncoupling 2 and 3 proteins*[47] and circulating levels of leptin[48]. These aspects of the resting metabolism show that physical activity may have two distinct effects in the RMR. Besides the training effect in the lean mass increase, a second effect may result from these physiological processes that influence the RMR[43].

The uncoupling protein (UCP3) was discovered in 1997[49]. These proteins transport some protons or anions of fatty acids through the mitochondrial internal membrane in order to produce ATP and increase the energetic cost. Nevertheless, there is also evidence that the primary physiological function of the UCP3 is not the regulation of the energetic cost; however, this phenomenon still remains obscure. It has been suggested[50] that the UCP3 may be involved with the adaptations induced by training in the energetic metabolism, causing a down-regulation in the UCP3, which consequently is associated with the increase of the efficiency mechanism in the energy profit. It is worth mentioning though, that in the acute exercise occurs up regulation of the UCP3[51], enabling better oxidation of fatty acids. However, with training the capacity to oxidate fatty acids could increase, reducing the need of high levels of UCP3.

**BMR in obese subjects and leptin**

Leptin, a hormone produced by the adipose tissue, is the major regulator of the food ingestion and the metabolic rate[48]. According to the same authors, the plasmatic level of leptin reflects in the energetic storage, once this hormone plays a negative feedback in the hypothalamus-pituitary-thyroid axis and hypothalamus-pituitary-adrenal axis.

The leptin action mechanism involves decrease of the food ingestion and the increase of the energetic cost, resulting in reduction of the body mass. Leptin also increases the sympathic activity, suggesting an increase of the thermogenesis in the brown adipose tissue (regulates the body weight after hyperphagia), which could be a mechanism in which leptin would regulate the body mass[52]. The results of this study showed decrease of 43% in the food ingestion with the administration of leptin. Moreover, it was also verified significant increase in the oxygen consumption, which was associated with the increase in the new UCP proteins (uncoupling proteins). The total concentration of UCP increased 131% with the leptin administration, promoting body mass reduction. The UCP activation increases the substrate oxidation rate and heat production. These authors concluded that leptin increases the norepinephine turnover in the adipose tissue, once it increases the sympathic activation, which increases the thermogenesis in the brown adipose tissue, increasing the UCP expression as well.

Within this context, it is important to mention that a recent study of our study group[53], demonstrated that the majority of the obese female adolescents who registered in our Multidisciplinary Program for obesity treatment, presented exacerbated hyperleptinemia. Such fact could suggest resistance to this hormone's action derived from alterations in their receptors, which would result in lower thermogenesis and MRM reduction of these patients. Besides that, these alterations suggest the need of long term-strategies in order to reestablish the leptin action. It could also be a factor that contributes to the discrepant results concerning the RMR of obese and non-obese individuals.

Conversely, in the same study previously metioned, it is verified that after a short term intervention period the hyperleptinemia was significantly reduced Nevertheless, the normality values were not reestablished, confirming thus, the need of long term treatment for the hormonal and metabolic adjustments which favor the MRM increase and consequently more easiness for the negative energetic balance obtaining and adipose mass reduction.

## FINAL CONSIDERATIONS

Therefore, although the exact combination is still unknown, the development of training programs which maximize the EPOC may be an important factor to the ponderal reduction[9,54]. Although the energetic cost of EPOC in a training session is small, its cumulative effect could have a positive impact in the obesity panorama. The same thought is valid for the RMR. Nonetheless, it is worth mentioning that the weight loss and EPOC have not been confirmed. Further studies are necessary in order to better understand this issue.

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