INVITED REVIEW

Lift weights to fight overweight
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Summary

Although resistance training (RT) has long been accepted as a means for developing and maintaining muscular strength, endurance, power and muscle mass, its beneficial relationship with health factors and chronic disease has only recently been recognized in the scientific literature. Prior to 1990, resistance training was not a part of the recommended guidelines for exercise training and rehabilitation for either the American Heart Association or the American College of Sports Medicine (ACSM). In 1990, the ACSM recognized resistance training as a significant component of a comprehensive fitness programme for healthy adults of all ages, a position subsequently confirmed few years after. At present, even though interest in clinical applications of RT is increasing, there are still some concerns, among physicians, about the use of this exercise methodology in weight control programmes. This review aims to explore the metabolic effects of RT and its efficacy and feasibility in overweight subjects.

Introduction

Health organizations report a worldwide increase in the prevalence of overweight and obesity (Olshansky et al., 2005). This increase is a great source of concern considering the fact that obesity and in particular abdominal obesity is one of the principle risk factors for cardiovascular disease, and it is strongly related to dyslipidaemia, hypertension, diabetes and metabolic syndrome (Koh-Banerjee et al., 2004). Diet and exercise are considered the main tools to control body weight and body fat (Paoli et al., 2008), but nevertheless the exact ‘dose’ and ‘quality’ of diet (Paoli et al., 2013a,b) and exercise is still a subject of debate (Paoli et al., 2012a,b). The correct dose and quality of exercise, that is, the knowledge of how and how much should be suggested to patients is of importance for clinicians and medicine practitioners. The main question about the effect of exercise on body weight regards the influence it has on energy expenditure. Overall, daily energy expenditure may be briefly divided into three different components that can be categorized as (i) resting metabolism, (ii) thermic effects of food and (iii) the energy expenditure of physical activity associated with exercise and non-exercise movement (Jequier, 2002). It has been demonstrated that in Western countries, the mean ratio of daily energy expenditure and resting energy expenditure (REE) is 1·66; in other words, only 40% of energy is expended on activity while the remaining 60% is expended at rest (Black et al., 1996). Despite the recommendations of national and international health organizations for at least three 30-min sessions of vigorous exercise per week, it has been calculated, as a general example, that this would increase energy demands by only $\approx 1000$ Kcal per week (i.e. $\approx 5\%$ of the average weekly expenditure about 20 000 Kcal per week) (Black et al., 1996). Notwithstanding these considerations, clinical practice and data from the literature consistently point to the beneficial effect of exercise on fat loss (Jakicic & Gallagher, 2003). The only way to reconcile this apparently contradictory evidence between experimental and clinical data is to hypothesize the existence of other exercise-related factors involved in the fat loss effect other than the simple increase in energy expenditure during exercise.

Mechanisms of physical activity’s effects on body weight control

Three fundamental mechanisms have been taken into consideration to explain the effects of physical exercise on body weight control, and it may (i) reduce hunger, (ii) improve fitness levels and consequently might change behaviour related to non-exercise activity thermogenesis such as walking and stair climbing, (Jakicic & Gallagher, 2003) (iii) exert a positive effect on resting metabolism. Regarding the latter, resting energy expenditure (REE) is the largest component of the
daily energy budget and, consequently, any increase in REE in response to exercise could potentially have a great impact on health promotion and weight control. Even though there is considerable individual variation in REE, it is well established that fat-free mass (lean body mass) is the main factor that influences REE, accounting for 50–70% of the individual variance (Westerterp et al., 1992; Zhang et al., 2002). There is a linear relationship between lean body mass and resting energy metabolism but with broad interindividual variance (Wang et al., 2000; Muller et al., 2002). It is evident that muscle mass represents the majority (≥88%) of metabolically active fat-free mass (FFM) with the metabolically active organs representing only 12% of FFM, while the former provides an energy expenditure of approximately only 15 Kcal kg⁻¹ per day, the energy expenditure of the latter is 468 Kcal kg⁻¹ per day. Thus, muscle accounts for only a minor part (less than 20%) of total daily energy expenditure. Nevertheless, many human studies with long-term exercise protocols, separating the effects of training and nutrition, have demonstrated that the positive effect of training on REE is mainly mediated by the increase in free fat mass (i.e. muscle) (Byrne & Wilmore, 2001). Interestingly, these studies also found that the combination of endurance and resistance training leads to an overall decrease in REE (Westerterp et al., 1992; Byrne & Wilmore, 2001), but that this exercise-related decrease was evident with endurance training, while a net increase in REE was found after RT (Hunter et al., 2008). These results explain why in recent years there has been a lot of interest in the specific effects of RT on weight control, and it is also the case that resistance training (RT) has only been incorporated recently as an important component of exercise protocols for weight control in some guidelines (Kraemer & Ratamess, 2005; Donnelly et al., 2009).

**Resistance training and metabolism**

Resistance training acts in a substantially different way compared with endurance training (ET), and it increases muscle mass in the long term (Paoli et al., 2010, 2013a,b) but also increases excess postexercise oxygen consumption (EPOC) immediately after the training session (Schuenke et al., 2002). In 1984, Gaesser and Brooks defined the recovery period in which an increase in oxygen uptake is observed as ‘excess postexercise oxygen consumption’ (EPOC). EPOC can be divided into two phases: short term (due to the so-called oxygen debt or fast component of excess postexercise oxygen consumption) and long term (slow component of excess post-exercise oxygen consumption due to various mechanisms not yet completely understood but most likely linked to the restoration of homeostasis). As a matter of fact, after intense or prolonged exercise, oxygen consumption remains increased for several hours and is referred to as the slow component of excess postexercise oxygen consumption (EPOC). This elevated postexercise metabolism plays a part in the energy cost of exercise and in the overall effect of exercise in body weight control. Even though the beneficial effect on health and weight control of a 24-h increase in metabolism is evident (Hunter et al., 2000), the optimal amount and type of exercise routine remains to be established. We recently discussed that resistance training should be investigated more thoroughly and rigorously by taking into account the variables involved including (i) muscle action used, (ii) type of resistance used, (iii) volume (total number of sets and repetitions), (iv) exercises selected and workout structure (e.g. the number of muscle groups trained), (v) the sequence of exercise performance, (vi) rest intervals between sets, (vii) repetition velocity and (viii) training frequency (Paoli, 2012; Paoli & Bianco, 2012; Paoli et al., 2012a,b). It can be argued that, as Knuttgen asserted, if EPOC increased exponentially as a function of exercise intensity, whereas it increased linearly as a function of exercise duration (Knuttgen, 2007), a high-intensity resistance training programme could affect positively resting metabolism. Thus, RT could act through two different ways: increase in lean body mass (chronic/long-term effect) and transient increase in metabolism (EPOC). In fact, there are several reports that weight training may require more recovery energy and a longer duration EPOC (Haddock & Wilkin, 2006) compared with endurance training. Despite this, there are surprisingly few studies published on the specific influence of high-intensity resistance training on metabolism.

**Effects of exercise intensity on weight control**

Some studies reported that higher-intensity resistance exercise generates greater EPOC than lower-intensity resistance exercise (Haltom et al., 1999; Thornton & Potteiger, 2002), and the reason for this greater EPOC could be attributed to a greater perturbation of energy homeostasis (Melanson et al., 2002, 2005; Schuenke et al., 2002). In this regard, we recently demonstrated that a high-intensity resistance training programme can induce a greater EPOC in the 24 h after the training session (Paoli et al., 2012a,b) (Fig.1) and that this kind of RT, mixed with a high-intensity endurance interval training, can improve body composition and blood lipids (Paoli et al., 2013a,b). It is clear that the intensity of exercise is a keystone of postexercise energy expenditure not only in endurance exercise such as cycling (Little et al., 2010) but also in resistance training. The mechanisms underlying these effects are not still clear, but we can raise some hypotheses:

1. The greater increase in blood lactate (which is evidence of a major metabolic stress derived from high-intensity resistance training and may reflect the utilization of lactate as fuel in the aerobic pathway) imposes an increase in postexercise energy expenditure (Binzen et al., 2001). Lactate removal, though, may only be part of the process, in fact, if lactate is infused during the postexercise period, it does not elicit a further increase in EPOC (Barnard & Foss, 1969). However, lactate may explain, together with an increase in body temperature (Gaesser & Brooks, 1984), only the short-term component of EPOC;
2) an increase in β-adrenergic system activity (Hunter et al., 2006);
3) hormonal variations: in response to exercise-induced trauma, an increase in metabolic hormonal concentration is seen (e.g. cortisol, catecholamines and thyroid hormone), which could increase metabolism (Schuenke et al., 2002);
4) the increased protein resynthesis due to postexercise muscle damage is energy expensive (approximately 20% increase in resting metabolism) (MacDougall et al., 1995) and could contribute to greater EPOC after high-intensity resistance training (Binzen et al., 2001; Gasier et al., 2012);

Moreover, an important effect of high-intensity exercise is the improvement in utilization of fatty acids instead of glucose that could lead to a greater utilization of fat stores. This effect can be highlighted by a decrease in respiratory exchange ratio (RER). The respiratory exchange ratio (i.e. the ratio between CO₂ expired and O₂ consumed) is a good way to identify the origin of energy substrates: when RER is close to 0.7, it means that the major energy source is lipids, while when the ratio is near 1, carbohydrates are the main source of energy. It is clear that an improvement in fat oxidation can help weight control. Fasting or very low carbohydrate diet can lead to a decrease in RER (Paoli et al., 2012a,b, 2013a,b), but also training can have the same result (Paoli et al., 2011, 2012a,b). The increase in the rate of fatty acid oxidation (i.e. the reduction in RER) indicates that triacylglycerol/fatty acid cycling is an important supporter of the energy cost in the prolonged component of EPOC as it is an index that the organism is using fatty acids rather than glycogen to satisfy the energy cost of exercise (Bahr et al., 1990) and seems to be a compensatory sparing of glycogen after any kind of resistance training (Poehlman & Melby, 1998).

Several causes could explain the RER lowering after resistance training (see Fig. 2):
1) glucose metabolic pathways are directed to replenish glycogen stores first instead of being used for energy supply. This means that glucose and all gluconeogenic precursors will be spared from further oxidation and will be converted to glucose and glycogen, so lipids become the preferred oxidation substrate (Borsheim & Bahr, 2003);
2) the AMP kinases/acetyl CoA carboxylase (AMPK/ACC) pathway. It has been demonstrated that intense exercise increases AMPK (Gibala, 2009); thus, AMPK can phosphorylate ACC, decreasing its activity; the decreased ACC activity leads to a decrease in the rate of the synthesis of malonyl CoA, and consequently, there is a release of inhibition of carnitine palmitoyltransferase I (CPT1) activity leading to an increase in lipid oxidation (Winder, 2001);
3) an increase in atrial natriuretic peptide (ANP) stimulated by exercise could play a role in the increased rate of lipid oxidation; production of ANP is related to the intensity of exercise (Moro et al., 2008; de Almeida et al., 2012), it has been demonstrated that ANP increases lipolysis (Souza et al., 2011), and this pathway appears to be more suitable than the increase in

Figure 1 Changes in resting energy expenditure (REE) and respiratory exchange ratio (RER) at basal condition and 22 h after a session of traditional resistance training or high-intensity interval resistance training. ** P<0.005. (Modified from Paoli et al., 2012b)

Figure 2 Factors involved in resistance training effects on weight control.
catecholamines that have a very short half-life and appears not to be related to lipolysis after RT (Ormsbee et al., 2009); 4) growth hormone increase also could explain a part of the increase in lipid oxidation; it was demonstrated by Bottaro et al. et al. (Bottaro et al., 2009) that intense exercise with incomplete recovery might stimulate GH production in a significant manner; 5) there is some new evidence that suggests how some cytokines and other peptides (myokines) that are produced and released by muscle fibres can exert autocrine, paracrine or endocrine effects that might influence the metabolic effect of exercise (Pedersen, 2011). A recent paper describes a new polypeptide hormone, irisin, which is regulated by PGC1-α; it is secreted from muscle into the bloodstream and may activate thermogenic mechanisms in adipose tissue – this might also play a role in short time reported to lower RER (Bostrom et al., 2012).

Some concerns could be raised about the feasibility of RT and in particular high-intensity RT in unfit overweight subjects (LaForgia et al., 2006), but there is experimental evidence supporting the suitability of RT in such individuals (Sothern et al., 2000; Bouchard et al., 2009; McGuigan et al., 2009; Ibanez et al., 2010; Idoate et al., 2011; Kreider et al., 2011; Thornton et al., 2011; Willis et al., 2012). More recently, our group has demonstrated the safety and feasibility, after a familiarization period, of high-intensity resistance training in sedentary and overweight subjects (Paoli et al., 2010, 2013a,b).

Conclusions

Taken together, these recent findings suggest that resistance training could positively affect body composition and that it could usefully be included in lifestyle weight control programmes. In our opinion, the general medicine practitioner should become familiar with this kind of training and its metabolic effects in order to recommend it to patients with weight problems.

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Resistance training and overweight, A. Paoli et al. 5


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