Effect of long-term strength training on glucose metabolism. Implications for individual impact of high lean mass and high fat mass on relationship between BMI and insulin sensitivity


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ABSTRACT. The aim of this study was to examine the independent effect of high lean mass on glucose metabolism, as well as its consequences on the classic relationship between BMI and insulin sensitivity (SI) in 3 groups: 1) 8 strength-trained males with BMI >27 kg/m² (athletes); 2) 10 sedentary males with BMI >27 kg/m² (obese); and 3) 12 sedentary males with BMI 22-25 kg/m² (control). Body composition was measured with impedance analysis. IV glucose tolerance test was performed at 09:00 h after overnight fast. Estimation of insulin sensitivity and glucose effectiveness by Minimal Model Approach. Plasma glucose and insulin determination by glucose-oxidase and RIA respectively. BMI and lean mass (LM) were greater in athletes than in controls, but there were no differences in fat mass (FM), basal glucose (Gb), basal insulin (lb), glucose tolerance (Kg), SI, glucose effectiveness (Sg), acute insulin response to glucose (AIRG) and leptin. Obese showed greater FM, leptin, lb and AIRG than athletes, while SI was lower; BMI, LM, Gb, Kg and Sg were similar. BMI, FM, LM, lb, AIRG and leptin were lower in controls than in obese, while SI index was greater; Gb, Sg and Kg were similar. We found that: 1) Resistance exercise does not modify glucose effectiveness, but can improve insulin sensitivity through FM reduction (LM augmentation alone has no effect on glucose metabolism); and 2) High BMI causes insulin resistance only if it depends on adipose tissue hypertrophy.


INTRODUCTION

The beneficial influence of physical activity on glucose metabolism has been known since the pioneering work published by Conard et al. in 1957 (1). More recently, since high accuracy methods to quantify insulin sensitivity (SI) have become available (2, 3) the effect of endurance exercise on glucose metabolism has been greatly studied, and the majority of Authors reported SI improvement with both single aerobic exercise bouts in non-trained subjects (4-7), as well as in long-term endurance training (8-11). However, there are few studies that deal with the action of resistance exercise (RE), a special kind of physical activity that provokes bw increase through lean mass (LM) hypertrophy (12-14). Until now, only 5 studies dealing with relationships between RE and SI have been published (15-19), but all of them showed fat mass (FM) reduction in the group of athletes, a circumstance that probably influenced the results (20). On the other hand, since bw increase in sedentary subjects is mostly related to FM hypertrophy, it is commonly assumed that BMI is a good indication of fat stores, and due to this, BMI is widely used in investigation projects as well as in routine clinical practice. In 1990, an inverse correlation between BMI and SI was observed from a threshold of 26.8 kg/m² (21). However, this finding has been linked to FM hypertrophy in the absence of knowledge with regard to the impact of LM increase on glucose metabolism in subjects with high BMI.
The purpose of the present work was to investigate the independent effect of high LM on glucose metabolism, as well as its consequences over the classic relationship between BMI and SI. Moreover, since the population with high BMI due to LM hypertrophy is represented by long-term strength trained athletes, we also investigated the metabolic impact of such sports practice, paying specific attention to the effect of high LM, as this is the first study that includes the non-existence of FM differences between athletes and controls as a necessary condition.

SUBJECTS AND METHODS

Subjects

Three groups of volunteers were studied. Group 1 (athletes): 8 healthy young males (age 25.38±5.95 yr) males with BMI≥27 kg/m² who undertook RE in a regular manner (5 bouts of 3 h per week on average) over a period of at least 2 yr before the onset of the study. Group 2 (obese): 10 healthy and sedentary young males (age 25.80±8.12 yr) males with BMI≥27 kg/m². Group 3 (control): 12 healthy and sedentary young males (age 26.00±3.19 yr) with normal BMI (21-24 kg/m²). The design of the study included the non-existence of differences in BMI between athletes and obese, as well as the non-existence of differences in FM between athletes and controls (see Results, Table 1) as necessary conditions. None of the volunteers consumed substances that could affect glucose metabolism, and none had first degree relatives with diabetes mellitus. All subjects received detailed information about the nature of this study and gave informed consent approved by the Ethical Committee of the Hospital Virgen del Cristal.

Methods

Body composition was measured through monofrequency tetrapolar bioelectric impedance (Bodystat (f)). Bodystat Ltd., Douglas, IM99. IDQ. U.K.). During the recruitment phase body composition was measured in several candidates and thereafter only those results coincided with the inclusion criteria, were recruited. In order to confirm previous data, body composition was analyzed again, these results were used for the study. At 09:00 h (after a 12 h nocturnal fasting) an iv glucose tolerance test (IVGTT) was performed (29 samples at times -20, -15, -10, -5, 0, 2, 3, 4, 5, 6, 8, 10, 12, 14, 16, 19, 22, 25, 30, 40, 50, 60, 70, 80, 100, 120, 140, 160, 180). An iv catheter was inserted in the right forearm for sampling, and another catheter was placed in the left forearm to inject a 300 mg/kg dextrose (50%) bolus at zero time. Samples were collected in heparinized crystal tubes that were maintained in ice until the end of the IVGTT. For at least 3 days prior to the IVGTT subjects consumed a diet with 300 g of carbohydrates. In order to avoid as much as possible any acute effect of exercise (6, 7), athletes performed no physical activity during the 36-h period prior to the IVGTT. Plasma glucose concentration was determined (triplicate) by the glucose oxidase method in an autoanalyzer (Beckman Instruments, AC). Insulinemia and leptinemia were determined (dupliciate) by RIA (LINCO Research, St. Charles, U.S.A.). Bergman’s Minimal Model Approach was used to calculate the insulin sensitivity index SI and the glucose effectiveness index Sg (3, 22). Acute insulin response to glucose (AIRG) was calculated as the mean of plasma insulin concentration increment over the basal in the times 2, 3, 4, 5, 6, 8, 10 (11). The iv glucose tolerance index Kg was calculated as previously described (7).

Statistics

Data are shown as mean±SD. Normal distribution was proven through Kolmogorov-Smirnov test. Significance level of means differences was estimated by t test for non-paired samples. Correlations were studied with Pearson test. Significance was considered when p<0.05.

RESULTS

Athletes vs controls

According to the design of the study BMI and LM were greater in athletes, but there were no differences in any other variable: FM, basal glucose (Gb), basal insulin (lb), Kg, SI, glucose effectiveness (Sg), AIRG and lep (Table 1).

Athletes vs obese

Once again following the study design, BMI was similar in both groups. On the other hand, FM, lep, lb and AIRG were lower in athletes, while SI was higher. No differences were observed in LM, Gb, Kg and Sg (Table 1).

Controls vs obese

BMI, FM, LM, lb, AIRG and lep were lower in controls, while SI was greater. Gb, Sg and Kg were similar (Table 1).

Correlations

LM did not show significant correlation with any other variable. Widely reported correlations between BMI, FM, lb, AIRG, SI and lep were also observed in this study (Table 2).

DISCUSSION

The beneficial effect of endurance exercise on SI is well known (4-11). However, there are few studies focused on glucose metabolism changes induced by RE, a special kind of sports practice that typically provokes a notable weight increase due to LM hypertrophy (12-14). Until now only 5 studies on relationships between RE and SI have been published (15-19), but all of them showed FM reduction in the group of athletes, a circumstance that probably influenced the results (20). In contrast, the present study analyzed, for the first time, the metabolic impact of RE, attending exclusively
to high LM effect: following the experimental design, although athletes showed greater BMI than controls, FM was similar, while LM was higher (an average difference of 7.60 kg). However, (and clearly in disagreement with data available in the literature) in this work no differences in SI or Sg in any other variable were observed between athletes and controls. At times cross-sectional comparisons based on BMI are difficult to interpret, as one group may have a greater absolute amount of FM or LM (excess or deficiency of fat) than another, but when expressed relative to bw, the fat or lean mass may be similar between the groups. In the present study, expressing SI or Sg per unit FM or per unit LM did not provide a different interpretation of the results (data not shown). In 1983, Yki-Järvinen et al. (15) studied 8 RE athletes and 7 sedentary subjects employing the clamp technique, finding in the former group a greater glucose clearance rate, but also a notably lower FM percent. In 1994, Miller et al. (16) enrolled 11 healthy elderly males in 16-week RE program that
Table 2 - Correlations.

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AIRG: acute insulin response to glucose; FM: fat mass; Gb: basal glucose; lb: basal insulin; Kg: glucose tolerance index; LM: lean mass; Sg: glucose effectiveness; SI: insulin sensitivity.

lead to LM increase (1.2 kg) and FM reduction (1.2%). In this study the glucose clearance measured by clamp both before and after training increased 24% and 22% at low and high insulin doses respectively. In 1996, Zachwieja et al. (17) performed a similar experiment, but using the minimal model of the clamp instead, and they found significant changes in body composition (LM rose 2.1 kg and FM fell 1.9 kg) accompanied by a SI increment that did not achieve statistical significance (p=0.06). On the other hand, neither Sg and Kg index nor insulin secretion was modified by RF training. In 1998, Ishii et al. (18), compared 8 non-obese Type 2 diabetic patients with 9 similar subjects recruited for a 6-week RE program using the clamp technique. While there were no metabolic changes in sedentary patients, the trained group showed glucose clearance rate increase (49%), as well as FM reduction (2%) that did not achieve statistical significance (p value not shown). Finally, also in 1998, Fujitani et al. (19) with the minimal model compared 11 long-term RE trained males with 20 sedentary ones, and they observed that athletes had lower FM (5.2%) and higher values of SI and Kg; likewise, trained subjects also showed higher Sg, albeit as a consequence of basal insulin effect (Sg component directly related to SI, and not via glucose effect at zero insulin (Sg component related to non-insulin mediated glucose uptake). In the light of the present study, we think that these data from the literature confirm the well-known and fundamental impact of fat stores on insulin action (20). Moreover, although from some lines of evidence (23, 24) a possible effect of LM hypertrophy on glucose clearance has been suggested (25), the data from the present study do not enable us to confirm this hypothesis, especially as neither Zachwieja et al. (17) nor ourselves observed that RE training leads to changes in Sg index, in spite of LM increment. Thus, although Fujitani et al. (19) observed a Sg value that was 33% higher in athletes than in controls, the Authors of that study consider this phenomenon to be dependent on unknown factors that are able to modify insulin-independent glucose uptake, since no correlation between Sg and LM was observed (as occurred in the present work).

The inverse relationship between obesity and SI has been known for almost 3 decades, when Sims et al. (26) observed in volunteers held in jail that bw increase (obtained by way of a hypercaloric diet) lead to hyperinsulinemia and insulin action impairment. Thereinafter, insulin resistance due to obesity has been widely studied and lipid hyperoxidation derived from abdominal adiposity has been identified as a fundamental pathogenetic factor (20). Thus, it is well known that Type 2 diabetes mellitus is due to the confluence of defective insulin secretion and insulin resistance, and obesity, as the first cause of insulin resistance, is essential in the pathogenesis of this highly prevalent disease (27-29). Since bw increase in sedentary subjects is mostly related to FM hypertrophy, it is commonly assumed that BMI is a good reflex of fat stores, and therefore BMI is widely used in investigation projects as in everyday clinical practice. Thus, although in 1990 a inverse correlation between BMI and SI was observed from a threshold of 26.8 kg/m² (21), this finding has been linked to FM hypertrophy in the absence of knowledge about the impact of LM increase on glucose metabolism in subjects with high BMI. In the present work we attempt to clarify the independent effect of high LM and high FM over the classic relationship between BMI and SI through the study of long-term RE trained subjects, since such athletes are the only population with high BMI due to LM hypertrophy. Our results showed the absence of differences in all glucose metabolism variables between athletes and controls, in spite of the higher values of BMI and LM, indicating that high BMI due to LM hypertrophy does not give rise to metabolic changes. With the aim of confirming this interesting finding, we also compared the RE trained subjects with sedentary volunteers selected to exhibit similar BMI than athletes (obviously having higher FM than controls), and finally, in order to increase the reliability of the present study, controls were compared to obese (contrasted with obese subjects). Since similar results were obtained when contrasting obese subjects to athletes as when obese subjects were contrasted with controls, we believe that these data confirm sufficiently that high FM is the only pathogenetic factor (or at least the fundamental
one) of the glucose clearance impairment observed in subjects with high BMI. Finally, according to the literature (30-32) and especially in agreement with a recent study carried out by our group (33), the data from the present study confirm that leptinemia is not influenced by physical training in absence of changes in FM, varying mainly as a consequence of fat stores.

In conclusion, our results indicate that: 1) Long-term RE training does not modify visceral sensitivity to glucose, but it can improve SI if it leads to a reduction in fat stores (high BMI by itself has no effect on glucose clearance); and 2) High BMI causes insulin resistance only if it depends on adipose tissue hypertrophy.

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REFERENCES