

Adipose Tissue Lipolysis is Upregulated in Lean and Obese Men During Acute Resistance Exercise

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Objective. To investigate the effect of acute resistance exercise on adipose tissue triacylglycerol lipase activity (TGLA) in lean and obese men.

Research Design and Methods. Nine lean and 8 obese men performed 30 min of circuit resistance exercise. Adipose tissue and blood were sampled during exercise for TGLA, metabolite, and hormone determinations. Respiratory exchange ratio (RER) was measured throughout exercise.

Results. Energy expenditure of exercise relative to body mass was higher in lean; RER was higher in obese suggesting lower fat oxidation. TGLA increased 18-fold at 5 min of exercise in lean and 16-fold at 10 min in obese. The delayed lipolytic activation in obese was reflected in serum non-esterified fatty acid and glycerol concentrations. Plasma insulin increased in obese but did not change in lean.

Conclusions. Resistance exercise upregulated adipose tissue lipolysis and enhanced energy expenditure in lean and obese men, with a delayed lipolytic activation in the obese.

The American Diabetes Association endorses resistance exercise (RE) as a means of improving body composition and metabolic control in diabetes (1). Limited information exists regarding the effect of RE on adipose tissue lipolysis. We monitored adipose tissue triacylglycerol lipase activity (TGLA), along with metabolic and hormonal responses to RE, in lean and obese men.

RESEARCH DESIGN AND METHODS

Participants were 9 lean and 8 obese healthy untrained men (Appendix 1) with stable weight and no pharmacological or nutritional intervention during the last 6 months. Fasting plasma glucose was below 5.55 mmol l⁻¹. Written informed consent was obtained and procedures were in accordance with the Declaration of Helsinki and Institutional Review Board.

Following anthropometric, ergometric, and dietary assessment (2) participants performed 30 min of circuit RE in the fasted state (Appendix 2). Blood and adipose tissue were sampled at 0, 5, 10, 20, and 30 min of exercise. Respiratory exchange ratio (RER) was monitored breath-by-breath via portable gas-exchange analyzer.

Blood lactate, glucose, non-esterified fatty acids (NEFA), and glycerol were determined spectrophotometrically; catecholamines, by high-performance liquid chromatography; insulin, glucagon and cortisol, by standard immunoassays. Homeostasis model assessment for insulin resistance (HOMA-IR) was calculated (3). Adipose tissue needle biopsies from the buttock were analyzed for TGLA as described (4).

RESULTS

Groups had similar HOMA-IR and VO_{2max} (Appendix 1). Energy expenditure of RE was higher in the obese in absolute terms, in the lean relative to body mass, and in the obese relative to lean body mass. Relative energy

and macronutrient intakes were similar (Appendix 3).

Groups had similar resting VO₂ and RER (Appendix 4). Both parameters increased with exercise and remained elevated throughout; RER was higher in the obese throughout exercise. Blood lactate increased similarly in lean and obese. Glucose tended to increase with time ($P=0.053$).

Adipose tissue TGLA (Figure 1) peaked at 5 min in the lean (rising from 0.32±0.27 to 5.82±1.32 mmol kg⁻¹ min⁻¹) and at 10 min in the obese (from 0.36±0.23 to 5.88±2.32 mmol kg⁻¹ min⁻¹). Thereafter, TGLA declined without reaching baseline. Groups did not differ in area under the curve (78.1±12.4 vs. 95.3±16.0 mmol kg⁻¹, respectively). Plasma insulin did not change in the lean but increased in the obese following the first exercise cycle and remained elevated thereafter. In the lean, NEFA tripled and glycerol doubled at 5 min, maintaining a plateau thereafter, whereas, in the obese, NEFA tripled and glycerol quadrupled at 10 min. Catecholamines (Appendix 4) increased similarly in both groups. Glucagon increased only in the lean and cortisol increased only in the obese following the last two cycles.

CONCLUSIONS

Resistance exercise elevated adipose tissue TGLA 16- to 18-fold within 5-10 min in lean and obese men and increased energy expenditure in both groups. Although it can be estimated that only a small fraction of the fatty acids released by lipolysis could be actually oxidized, our data suggest that RE caused fat mobilization and might therefore be considered as part of interventions toward body weight/fat reduction. Interestingly, the lipolytic response to RE was similar to the response to aerobic exercise (4). In agreement with our findings, a 78% increase in glycerol concentration of a dialysate collected during RE from a probe inserted in abdominal

adipose tissue was reported (5). However, the fat biopsy technique applied in the present study provides direct evidence on the lipolytic rate at the intracellular level and permits a higher time resolution.

Lipolysis was apparently stimulated during RE by the progressive catecholamine rise. The increase in TGLA may be due to both activation of hormone-sensitive lipase and its increased attraction to lipid droplets in adipocytes because of perilipin phosphorylation (6). Our assay is sensitive to the latter effect, since it employs the natural substrate of triacylglycerol lipase in adipose tissue and mild homogenization to preserve the morphology of lipid droplets, in contrast to other assays that employ artificial emulsified triacylglycerols. The attenuation of lipolytic activity following the 5-10 min peak, despite the maintenance of the exercise stimulus, may be attributed to β -adrenergic receptor desensitization (7).

Triacylglycerol lipase demonstrated a delay in peak activation in the obese, although the overall lipolytic response did not differ between groups. The NEFA and glycerol responses showed a similar delay. Since the two groups had similar HOMA-IR and sympathoadrenergic stimulation, the delay in the RE-induced lipolytic activation in the obese may be attributed to the increase in insulin, although additional factors (e.g., growth hormone, cytokines, and β -adrenergic receptors) may have mediated this effect. While circulating insulin declines during endurance exercise, contributing to the stimulation of adipose tissue lipolysis, evidence suggests that insulin is non-responsive to RE (8). The insulin surge in the obese may be due to a reduced responsiveness of their β -cell α_2 -receptors (9) and may have activated phosphodiesterase (6), which degrades cAMP, thus slowing down the catecholamine-induced cAMP rise that leads to lipolytic stimulation. Nevertheless, the magnitude of the insulin rise (just $6 \mu\text{U ml}^{-1}$

at 10 min compared to baseline) may have been insufficient to blunt the catecholamines' lipolytic effect. On the other hand, the insulin increase in the obese could have resulted in increased glucose uptake and metabolism, especially in skeletal muscle, leading to higher carbohydrate oxidation, as evidenced by the higher RER.

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Figure 1. Effect of resistance exercise on adipose tissue triacylglycerol lipase activity (A) plasma insulin concentration (B), serum non-esterified fatty acid concentration (C), and serum glycerol concentration (D) in the two groups. Error bars represent SE. a, significantly different from the respective baseline; b, significantly different from the respective value at 5 min; c, significantly different from lean ($P < 0.05$), as detected by LSD test following 2-way (group-by-time) analysis of variance with repeated measures on time.

