EFFECT OF A PROGRAM OF MODERATE PHYSICAL ACTIVITY ON MENTAL STRESS-INDUCED INCREASE IN ENERGY EXPENDITURE IN OBESE WOMEN

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SUMMARY

Background: Energy restriction and physical activity are major components of weight reduction programs. Energy restriction is known to affect the sympathetic nervous system activity and to reduce several components of energy expenditure, including the stimulation of energy expenditure elicited by mental stress. The effect of physical activity on this parameter remains unknown.

Methods: This study was designed to assess the effect of physical training on the stimulation of energy expenditure during mental stress. Seven obese women (age 33 ± 3 years, BMI 34.9 ± 1.4 kg/m²) were studied before and after a 6 week program of aquagym (2 sessions of 50 min/week). Energy expenditure was measured by means of indirect calorimetry under hyperinsulinemic conditions before and during a 30 min mental stress. O₂ max was measured before and after physical training by means of the test of Balke.

Results: O₂ max was 23.4 ml/kg/min before physical training, and increased to 26.1 ml/kg/min at the completion of the training program (p < 0.05). Body weight, resting energy expenditure and insulin-mediated glucose disposal [oxidative and non oxidative] were not modified after training. Mental stress increased energy expenditure by 12.3% before physical training (p < 0.002 vs resting conditions) and by 12.6% after physical training (p < 0.002 vs resting conditions). NS vs before physical training).

Conclusion: Physical training per se does not alter the stimulation of energy expenditure induced by mental stress.

Key-words: Basal metabolic rate, sympathetic nervous system, exercise, insulin resistance.

RéSUMÉ - Effet d’un programme d’activité physique modérée sur l’augmentation de la dépense énergétique induite par un stress mental chez la femme obèse.

Contexte : Restriction calorique et l’activité physique sont les principales modalités thérapeutiques de l’excès pondéral. La restriction calorique diminue plusieurs composantes de la dépense énergétique, y compris les dépenses énergétiques induites par un stress mental. Le but de cette étude était d’évaluer l’effet de l’entraînement physique sur le compositant des dépenses énergétiques.

Méthode : Sept femmes obèses (âge 33 ± 3 ans, IMC 34.9 ± 1.4 kg/m²) ont été étudiées avant et après avoir achevé un programme d’activités physiques modérées (aquagym à raison de 2 séances par semaine pendant 6 semaines). Leur dépense d’énergie a été mesurée par calorimétrie indirecte en conditions d’hyperinsulinémie au repos et au cours d’un stress mental. L’effet de l’entraînement physique sur la capacité aérobie a été évalué en mesurant la O₂ max au moyen d’un test de Balke.

Résultats : La O₂ max s’est élevée de 23,4 ml/kg/min avant entraînement physique à 26,1 ml/kg/min à la fin du programme d’entraînement (p < 0.05). Le poids corporel et le métabolisme du glucose en hyperinsulinémie n’ont pas été modifiés. Le stress mental a augmenté les dépenses d’énergie de manière semblable avant (+ 12,3 %) et après entraînement physique (+ 12,6 %).

Conclusion : Cette étude indique que l’entraînement physique modéré ne diminue pas la stimulation des dépenses d’énergie induite par un stress mental.

Mots-clés : métabolisme basal, système nerveux sympathique, exercice, résistance à l’insuline.
The development of obesity results of a positive energy balance, i.e. an energy intake exceeding energy expenditure. Both an increased energy intake and a decreased energy expenditure may possibly be involved [1]. Recent evidence indicates that energy expenditure can increase significantly during overfeeding [2,3] and that individuals who are able to increase the most their energy expenditure resist to weight gain [3]. Of interest, this increase in 24 hours in energy expenditure is observed while basal energy expenditure, diet-induced thermogenesis, and the energetic yield of physical activity are not changed. It has been attributed to stimulation of non exercise activity thermogenesis, or fidgeting [3]. We have recently reported that sympathetic activation induced by a mental stress acutely increases energy expenditure by about 20% above basal energy expenditure [4]. This stimulation is mediated by activation of $\beta$ adrenoreceptors, since it is abolished by treatment with the $\beta$ antagonist propranolol. Sympathetically-mediated stimulation of energy expenditure may possibly contribute to this increase in energy expenditure elicited by overfeeding.

Obesity is known to be associated with alterations of sympathetic nervous system activity. Although data collected in various studies are somewhat discordant, it is generally observed that whole body sympathetic nervous system activity is low in obese patients [5-7]. It is however recognized that sympathetic nervous system is more complex than previously thought and that its activity is differentially regulated in different tissues or origins. Of interest, activity of sympathetic nerve targeted to the skeletal muscle of obese individuals was shown to be increased in fasting conditions [8], but poorly responsive to hyperinsulinemia [9]. In obese female patients, the stimulation of energy expenditure during mental stress was however found to be of the same magnitude as that observed in lean females, suggesting that this specific sympathetic pathway was not primarily affected by obesity.

The treatment of obesity requires induction of a negative energy balance which can be attained by energy restriction and/or physical activity [10-12]. Energy restriction leads to an adaptive decrease in energy expenditure, which affects primarily diet-induced thermogenesis. We have also recently observed that decreases the stimulation of energy expenditure induced by a standardized mental stress [13]. The effects of physical activity and endurance training on sympathetically-mediated energy expenditure remain unknown. Endurance training is known to alter the sympathetic nervous system activity [14-17] and hence may possibly decrease energy output. We therefore investigated the effect of a moderate endurance training on basal energy expenditure and on the increase in energy expenditure induced by mental stress in a group of obese women. The stimulation of energy expenditure induced by a 30-min mental stress was measured before and after 6 weeks of physical training (aquagym). Since physical training can also affects insulin sensitivity and the nature of food oxidized, measurements were performed during an hyperinsulinemic euglycemic clamp.

**METHODS**

**Subjects**

Seven obese female subjects were enrolled to participate in this study. Their anthropometric characteristics and their body fat mass calculated from their skinfold thickness [18] are shown in Table I. All were in good health except an excess body weight and took no medication.

Aquagym training: All participants followed a physical activity program consisting in 50-min aquagym sessions 2 times/week during 6 consecutive weeks. Aquagym consisted in aerobic exercises of mild intensity and were performed in groups of 6-10, under the supervision of a trained physical educator.

Assessment of physical fitness: $O_2$ max was assessed by modified Balke test [19] i.e. walking at 4 km per hour, with increasing slope by 1% each minute until exhaustion.

**Metabolic investigations**

Each participant took part to a 4 1/2 hour metabolic study twice, before and after physical training. Each study started in the morning after an overweight fast. At their arrival in the metabolic laboratory, subjects took place in a bed in a recumbent position and remained quietly lying during the next 4 1/2 hours, time during which they watched TV. One indwelling venous cannula was inserted into a vein of one forearm for infusion of a 20% dextrose solution and of insulin. A second cannula was inserted into a wrist vein of the contralateral arm for periodic withdrawal of blood samples. Plasma glucose concentrations were measured with a Beckman glucose Analyzer II (Beckman Instruments, Palo Alto, CA); plasma insulin concentrations were measured with a fluoride method. Glucose and insulin rates of appearance (Ra) and disappearance (Rd) were estimated by a two compartment model. The Ra of free fatty acids (FFA) were measured by a spectrophotometric method.

**Table I. Characteristics of study participants.**

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Mean ± SEM</th>
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<tbody>
<tr>
<td>Age (years)</td>
<td>36 ± 3</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>93.0 ± 4.8</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>34.9 ± 1.4</td>
</tr>
<tr>
<td>% fat mass</td>
<td>40.6 ± 1.5</td>
</tr>
<tr>
<td>Waist/hip ratio</td>
<td>0.84 ± 0.04</td>
</tr>
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All values are expressed as mean ± 1 SEM.
centrations were measured by radioimmunoassay using a kit from Linco (St Charles, MO). Plasma norepinephrine concentrations were measured by HPLC with electrochemical detection [20]. Energy expenditure and net substrate oxidation rates were monitored throughout the experiments by means of open circuit indirect calorimetry, as previously described [21]. The equations of Livesey and Elia were used to calculate substrate oxidation. After 120 min baseline measurements, an hyperinsulinemic (0.4 ml insulin/kg/min) euglycemic clamp was started, and lasted for a total of 150 min. After two hours, a 30-min mental stress (consisting in 5-min periods of mental arithmetics altering with 5 min periods of Stroop’s test) was applied, as described previously [4]. During this period, there was a significant hyperventilation, resulting in a respiratory quotient higher than 1.0. Calculation of substrate oxidation rates were therefore not valid during this period, and the sole O₂ was used as an index of energy expenditure. Heart rate and blood pressure were monitored every 30 min in basal conditions and every 5 min during the mental stress.

Statistical analysis

All results are expressed as mean ± 1 SEM. Mean values obtained after physical training were compared to pretraining values by means of paired t-tests.

RESULTS

Physical training resulted in a significant increase in O₂ max, from 23.4 ± 1.6 ml/min/kg (range 20.1-27.2) before to 26.1 ± 1.1 ml/min/kg (range 24.1-30.1) after training (p = 0.04). Anthropometric parameters were not changed after training (Table I).

Training did not alter significantly basal glucose and insulin concentrations (Fig. 1). During the two metabolic studies, plasma glucose was efficiently clamped at about 5.6 mmol/l whereas plasma insulin concentrations were increased to about 300 pmol. The glucose infusion rate required to maintain euglycemia was about 9 μl/kg/min and was no different before and after physical training (Fig. 1). The substrate oxidation rates are shown in figure 2. Physical training did not alter basal or insulin-stimulated carbohydrates and lipid oxidation rate. Mental stress increased plasma norepinephrine concentrations by 7% before and 8% after training (Fig. 2). It increased heart rate from 71.9 ± 3.6 bpm to 79.0 ± 2.6 bpm before, and from 71.1 ± 3.3 to 76.9 ± 2.1 after physical training, and mean blood pressure from 81.3 ± 1.4 to 88.1 ± 1.2 (p < 0.01) and from 83.0 ± 2.7 to 83.9 ± 2.0 (p = 0.69). It increased energy expenditure by 12.3% before physical training (p = 0.002), and by 12.6% after physical training (p = 0.003) (Fig. 4).

DISCUSSION

In this study, a group of obese non diabetic patients followed a program of physical activity of moderate intensity. This resulted in an improved fitness, as documented by an 11.5% increase in O₂ max. This indicates that a) this program corresponded to a significant physical training, and b) participants were compliant to participate in these exercise sessions.

Our major observation is that the stimulation of energy expenditure induced by mental stress was not altered by physical training. Endurance training is known to alter the sympathetic nervous system activity. The effects of training remain however controversial. In one study, training inhibited the norepinephrine increase induced by exercise [16] In another study, basal muscle sympathetic nerve activity was shown to be lower in trained than in untrained individuals [17]. It was also observed that the exercise-induced increase in heart rate was blunted in trained individuals, consistent with a lower sympathetic activation during exercise [22]. In contrast, Poehlman et al. observed an increased norepinephrine appearance rate in elderly trained individuals [14]. Alterations of sympathetic activity induced by training may therefore have affected the response to mental stress. Our observations clearly disprove this hypothesis. Mental stress elicited similar increases in heart rate and in plasma norepinephrine concentrations before and after physical training, indicating that activation of the sympathetic nervous system was not altered. Furthermore, the increase in energy expenditure during mental stress was also not affected, indicating that the overall response to sympathetic stimulation remained intact. The mechanisms responsible for the increase in energy expenditure elicited by mental stress remain unknown but are known to be secondary to stimulation of β adrenoreceptors.

Physical training also did not modify resting energy expenditure both in basal conditions and during mild hyperinsulinemia, nor the fuel mix oxidized. This absence of effects of physical training on energy expenditure contrasts with those induced by energy restriction which significantly decreases basal energy expenditure [23] and blunts the stimulation of energy expenditure during mental stress [13]. Energy restriction also decreases the thermic effect of food and 24-hour energy expenditure in healthy individuals [24] whereas physical training does not significantly affect these parameters. This indicates that, although both energy restriction [25] and physical training [17] may decrease the basal sympathetic tone the effects on energy expenditure are widely divergent This suggests that the neural circuits and sympathetic nerve fibers affected by physical training differ from those altered by energy restriction. Alternatively, decreased sympathetic activity after physical training may be associated with increased responsiveness of adrenergic re-
In addition to its effects on energy expenditure, mental stress also increases skeletal muscle blood flow in healthy lean individuals. This effect acutely increases whole body insulin-mediated glucose disposal, demonstrating the efficiency of a muscle vasodilation to increase insulin sensitivity [4]. Interestingly, this...
Effect is abolished in obese female patients although the effects of mental stress on energy expenditure are well preserved [4]. This has been attributed to an endothelial dysfunction in obese subjects, possibly secondary to increased plasma free fatty acid concentrations [26]. An additional aim of our study was to evaluate whether this endothelial cell dysregulation in obese patients might be improved by physical training. Our data suggest that it was unfortunately not the case since mental stress failed to significantly increase whole body insulin-mediated glucose disposal after endurance training. It remains possible that a program of exercise of greater intensity would be required to improve endothelial function in such patients.

The physical activity performed by our patient was of moderate intensity, and failed to increase insulin sensitivity over the 6-week observation period. During training, the patients were on an ad libitum uncontrolled diet during this period and did not significantly change their body weight. This indicates that any increase in total energy expenditure induced by physical activity was fully compensated by an increased energy intake. This design allowed to evaluate the effects of physical training per se, i.e. irrespective of changes in body weight or body fat mass. Such a physical training program will however have to be associated with energy restriction in order to decrease body weight and/or increase insulin sensitivity. Further studies will be required to evaluate whether physical training prevents the decrease in energy expenditure induced by energy restriction.

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REFERENCES


