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3 Dynamics of GH secretion during incremental exercise in obesity, before and after a short period of
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5 training at different work-loads
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Summary

Background. Growth hormone (GH) is normally sensitive to physical exercise. Intensity and duration of exercise, fitness as well as age, all can influence GH response to exercise. In obesity, GH secretion is decreased both in basal conditions and in response to exercise.

Objective. To analyze the dynamics of GH response to a progressive cycloergometric test conducted up to exhaustion in adult normal subjects and obese patients, after a reconditioning program at different workloads.

Design and methods. We studied 8 lean subjects (4 males, mean age 34.3 yrs, range 26-47 yrs, mean BMI 22.1 kg/m²). GH was sampled at baseline and during the last 30 s of each power output increase. Anaerobic threshold (AT) was detected by the V-slope method. The same test was carried out in 16 obese subjects (7 males, mean age 39.1 yrs, range 20-59 yrs, mean BMI 35.8 kg/m²) and repeated after a 4-week reconditioning program consisting of aerobic workout (Group A, 8 subjects, 3 males, mean age 40.5 yrs, range 22-59 yrs, mean BMI 33.6 kg/m²), and aerobic plus anaerobic work (group B, 8 subjects, 4 males, mean age 37.6 yrs, range 20-56 yrs, mean BMI 38.0 kg/m²) for 6 days/wk, with no dietary restrictions.

Results. Mean exercise peak occurred at higher intensity in controls (140 vs. 110 W, $p < 0.05$) and AT exceeded at higher work outputs than in obese subjects (102 vs. 74 W, $p < 0.05$). In controls, GH response to exercise was prompt and further sustained after AT; in obese subjects, GH increased slowly and insignificantly before AT, thereafter it increased to lower levels than in controls ($p < 0.001$). Following the reconditioning period, both Group A and Group B of obese subjects failed to improve exercise performance as well as GH response to exercise before AT; beyond AT, a greater GH response to exercise occurred in Group B than Group A (7.59 ± 0.32 $\mu\text{g/l}$ at peak of exercise) with significantly different Delta AUCs following AT: 30.5 ± 12 $\mu\text{g}\cdot\text{min/l}$ in the Group A vs 124.2 ± 38 $\mu\text{g}\cdot\text{min/l}$ in the Group B, $p < 0.05$.

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3 **Conclusions.** Our results confirm the blunted GH response to exercise in obese adults when
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5 compared to lean counterparts. With obesity, aerobic training poorly increases the GH response
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7 beyond AT while supplemental anaerobic workload appears to increase GH response beyond AT.
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9 These observations may have implications for prescription of physical exercise, which is one of the
10
11 recommendations in the management of obesity.
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17 **Introduction**

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20 GH is one of the key regulators of body fat and lean tissue. Its pulsatile secretion is
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22 positively governed by GHRH and inhibited by somatostatin (1). A variety of physiological and
23
24 pathological conditions are known to affect GH secretion. They include age, gender, pubertal stage,
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26 sleep, nutritional status, body composition and temperature, fitness, gonadal steroids, insulin and
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28 IGF-I (1, 2, 3).
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32 GH secretion is responsive to bouts of physical activity (4) by an order of magnitude that
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34 depends on exercise intensity and duration, fitness and age (5). Several Authors suggested that GH
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36 response implies a threshold of exercise intensity, while others have documented that GH secretion
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38 increases linearly with increasing intensity of exercise (6, 7).
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42 Body fat and aerobic fitness act to control GH secretion with an inverse relationship existing
43
44 between relative abdominal fat and VO_2 peak, as well as with the 24-h integrated GH
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46 concentrations (2, 3). It is well known that abdominal visceral fat increases in obese subjects and
47
48 this has been claimed to explain why obesity dramatically reduces spontaneous and stimulated GH
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50 secretion (1, 2), decreases the 24h GH secretory bursts and accelerates GH disposal rates (8),
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52 supposedly due to a diminished GHRH or increased somatostatin tone (1).
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56 Importantly, obesity blunts the GH response to exercise (2).

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58 The American College of Sport Medicine recommends the addition of resistance exercise to
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60 a program of regular aerobic training (9) and only a few studies assessed the effect of concurrent
strength and endurance training on GH (4), particularly in obese subjects.

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3 In the present study we thought to examine the dynamics of acute GH secretion in a group of
4 adult obese and control subjects during a progressive cycloergometric test up to the maximal work
5 capacity and, subsequently, to evaluate the GH response to the same exercise protocol in obese
6 subjects following a 4-wk training period by aerobic or by aerobic plus anaerobic daily exercise,
7 without diet restrictions.
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17 **Subjects and experimental protocol**

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19 The protocol was approved by the Ethics Review Committee of our Institution and written
20 informed consent was given by all the subjects before participation, in accordance with the Helsinki
21 Declaration (1964, amended in 1975 and 1983) of the World Health Organization.
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27 The obese group consisted of 16 subjects (7 males; age 39.1 yrs, range 20-59 yrs, BMI 35.8
28 kg/m², range 30-42) while 8 untrained lean subjects (4 males; age 34.3 yrs, range 26-47 yrs; BMI
29 22.1 kg/m², range 17-25) served as controls (Tab. 1A). Exclusion criteria were smoking, physical
30 inability, hypertensive, diabetic or suffering from any other cardiovascular or metabolic disorders.
31 Physical examination, EKG, routine blood and urine analysis were carried out preliminarily to
32 exclude medical illnesses. Women were analysed during early follicular phase of the menstrual
33 cycle. The study protocol included anthropometric measurements after voiding and bioimpedance
34 analysis (BIA 101/S Akern, Florence, Italy) for measurement of fat (FM, kg) and fat-free mass
35 (FFM, kg). Patients with fluid overload according to vectorial analysis were excluded to minimise
36 errors in estimating FM and LBM in severe obesity. BMI was calculated as weight (kg)/height (m²).
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50 Before exercise testing, subjects were asked to restrain from strenuous activity for at least 24
51 h. In each subject a preliminary nocturnal O₂ saturation monitoring did not demonstrate the
52 presence of significant periods of pathological low values. Following an overnight fasting, at 8 am
53 individuals underwent an ergospirometric test in a thermo-regulated room on a bicycle ergometer at
54 a pre-set pedalling rate of 60 cycle/min and initial workload of 20 watt. Exercise intensity was
55 subsequently increased by 20-watt steps every 4 min until physical exhaustion occurred. Subjects
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3 were vocally encouraged to maintain a constant speed while the wheel resistance level was adjusted
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5 to preserve an increasing power output. At the beginning of recovery, subjects were invited to cycle
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7 for 2 more min effortlessly, and then were followed for up to 40 min while maintaining a sitting
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9 position. For analytical purposes, sampling was performed through an indwelling cannula inserted
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11 at the antecubital vein and kept patent by continuous slow saline infusion. Blood was sampled at
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13 baseline, during the last 30 s of each power output increase, and at first recovery (5 min) and late
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15 recovery (30 min).
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20 A cardiopulmonary exercise test station (Vmax 229, Sensor Medics, Yorba Linda, CA,
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22 USA) was used for continuous analysis of oxygen consumption (VO_2), CO_2 production (VCO_2) and
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24 minute ventilation (VE). Exercise capacity was assessed by determining the maximum work rate,
25
26 ventilatory anaerobic threshold (AT), and VO_2 -max defined as the VO_2 averaged over the last
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28 minute of exercise. AT was detected by the V-slope method (10). Calibrations were performed prior
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30 to each test. Heart rate (HR) and EKG signals were recorded by a CASE 6.5 (GE Medical System
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32 Milwaukee, WI, USA) and oxyhaemoglobin saturation was determined by a Radiometer
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34 percutaneous oxymeter. Blood haemoglobin was determined in all subjects at baseline.
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41 *Exercise training*

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43 Following the baseline evaluation, in-patient obese subjects were randomly assigned to 2
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45 subgroups each comprising 8 subjects matched by gender, age and BMI (Group A: 3 males and 5
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47 females; age 40.5 yrs, range 22-59 yrs; BMI 33.6 kg/m^2 , range 30-41; Fat mass 36.5 kg; Fat-free
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49 mass 52 kg; Group B: 4 males and 4 females; age 37.6 yrs, range 20-56 yrs, BMI 38.0 kg/m^2 , range
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51 34-42; Fat mass 48.1 kg; Fat-free mass 68.9 kg). Group B differed from Group A in a higher Fat
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53 mass ($p < 0.05$). Each subgroup underwent a 4-wk training protocol consisting of 2-daily 30 min
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55 cycloergometric exercise sessions for 6 days/wk. In Group A, the power output of each training
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57 session was exclusively aerobic, constantly attaining 70% of HR registered at AT. In Group B, the
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59 power output of each training session was aerobic (25 min) and anaerobic (5 min), attaining 70% of
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3 the HR registered at AT for the aerobic part, and 85% of the maximal HR for the anaerobic part.
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5 Increments of power output were imposed in view of the actual improvement by reconditioning,
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7 maintaining HR 70% of AT (Group A and B) and 85% (Group B) as the reference standards.
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9 Oxygen saturation and heart rate were continuously monitored during each training session. At the
10
11 end of each training period, each of the participants in Group A and Group B underwent a
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13 conclusive ergospirometric test according to the same protocol setting as the entry test.
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20 *Hormone measurements*

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22 GH levels were measured by chemiluminescence (Immulite 2000 Analyser, DPC, Los
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24 Angeles, CA) calibrated against WHO 1st IRP 80/50, having a sensitivity of 0.01 µg/l and intra- and
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26 inter-assay coefficients of variation (CVs) of 2.9-4.2% and 4.2-6.5%, respectively.
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32 **Statistical analysis**

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34 In controls and obese subjects all the values obtained at each step of the test were compared
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36 between groups by analysis of variance (ANOVA), transforming data to the natural logarithmic
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38 scale when requested (11). Among obese subjects and controls, integrated GH concentrations (area
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40 under the curve) were calculated during exercise and recovery by using the trapezoidal rule (12).
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42 ANOVA was employed to determine differences between groups and between pre and post training
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44 values.
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53 **Results**

54 *Exercise testing at study entry*

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56 Control subjects reached a mean maximal peak of exercise at 140 ± 12 W and exceeded AT
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58 at a mean value of 102 ± 11 W (Tab.1 A). VO_2 max was 1860 mL/min (28.52 mL/Kg/min, 81% of
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60 theoretical maximal VO_2) (13, 14). The obese subjects, on the whole (Tab. 1), reached a mean

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3 maximal peak at 110 ± 8 W ($p < 0.05$ vs normals) and exceeded AT at a mean value of 74 ± 5 W
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5 ($p < 0.05$ vs controls). VO_2 max was 1850 mL/min (18.03 mL/Kg/min, 77% of theoretical VO_2 max).
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8 In controls, GH significantly increased at AT ($p < 0.01$ vs. rest) and at peak exercise ($p < 0.01$
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10 vs rest, $p < 0.05$ vs AT), persisting at sustained levels during the first recovery and decreasing
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12 thereafter (Tab. 1 B). In obese subjects, GH did not significantly increase at AT but did so at peak
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14 exercise ($p < 0.05$) (Tab. 1 B). Between-group differences in GH response to exercise were clearly
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16 dichotomised when expressed as AUCs, these being significantly higher in controls than obese
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18 subjects (649.4 ± 3 vs 145.8 ± 0.6 $\mu\text{g/ml-min}$, $p < 0.001$).
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25 *Effects of a 4-wk aerobic training in obesity*

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27 At study entry, Group A achieved a mean exercise peak of 92.5 ± 9 W ($p < 0.01$ vs controls)
28
29 and exceeded AT at 64 ± 6 W ($p < 0.01$ vs controls). VO_2 max was 1625 mL/min (18.37 mL/Kg/min,
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31 79% of theoretical VO_2 max). At the end of the 4-wk aerobic training period, mean exercise peak at
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33 exhaustion and AT insignificantly rose to 110 ± 10 W and 74 ± 6 W, respectively. VO_2 max
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35 increased to 1720 mL/min (20.25 mL/Kg/min, 81% of theoretical VO_2 max). During this period,
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37 body weight and composition did not change significantly in any patient.
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41 GH response to exercise was null before AT while it increased at the peak exercise ($p < 0.01$
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43 vs rest, $p < 0.05$ vs AT) and persisted at higher levels than baseline at the first recovery, decreasing
44
45 thereafter (Fig. 1 B). The 4-wk aerobic training reconditioning program did not impact the pattern
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47 of GH secretion, which only significantly increased at the peak exercise ($p < 0.01$ vs rest, $p < 0.05$ vs
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49 AT), then decreasing during the late recovery (Fig. 1 B). GH values before and after the training
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51 period were similar at any time point.
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55 As shown in Fig. 1A, Group A achieved lower GH response to exercise than controls both at
56
57 study entry and after the training period at AT ($p < 0.01$ and $p < 0.05$, respectively), peak exercise
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59 ($p < 0.01$ and $p < 0.05$, respectively), first recovery ($p < 0.01$ for both) and late recovery ($p < 0.001$ and
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 $p < 0.05$, respectively).

Effects of a 4-wk aerobic plus anaerobic training in obesity

At study entry, Group B reached a mean peak at exhaustion at $127 \text{ W} \pm 9$ ($p = \text{NS}$ vs controls) and exceeded AT at a mean value of $85 \pm 7 \text{ W}$ ($p = \text{NS}$ vs controls). VO_2 max was 2080 mL/min (17.89 mL/Kg/min , 75% of theoretical VO_2 max). At the end of the aerobic and anaerobic training period, the mean peak at exhaustion rose up to $140 \pm 10 \text{ W}$ and AT up to $97 \pm 8 \text{ W}$ ($p = \text{NS}$ vs starting values). VO_2 max increased to 2165 mL/min (19.60 mL/Kg/min , 77% of theoretical VO_2 max). Similarly to Group A, weight and fat mass did not change significantly with training.

Exercise failed to evoke any significant GH response at AT, while this occurred at the peak exercise ($p < 0.05$ vs rest, $p < 0.05$ vs AT), persisted during the first recovery and decreased following (Fig. 1 B). At the end of the 4-wk aerobic plus anaerobic training program, GH response was weak at AT and became significant at the peak exercise ($p < 0.05$ vs rest, $p < 0.05$ vs AT), persisted at the first recovery and decreased at the late recovery (Fig. 1 B). Like in Group A, no significant difference occurred in GH values before and after the training period.

As shown in Fig. 1 B, GH was less responsive to exercise in Group B than controls before the training period at AT ($p < 0.05$), peak exercise ($p < 0.01$), first recovery ($p < 0.01$) and late recovery ($p < 0.01$). Unlike Group A, however, the combination aerobic plus anaerobic training program abrogated the previously documented differences in GH response to exercise between Group B and controls at any time but late recovery ($p < 0.01$) (Fig. 1 B).

Comparison between different training programs

Improvements in physical performances, albeit not significant, were obtained in any obese subjects after either training periods. When GH results were normalised according to power output achieved at AT ($\text{GH}/\text{W}_{\text{AT}}$) and peak exercise ($\text{GH}/\text{W}_{\text{PE}}$), both Group A and Group B showed significantly lower $\text{GH}/\text{W}_{\text{AT}}$ and $\text{GH}/\text{W}_{\text{PE}}$ values before training than controls. In normal subjects $\text{GH}/\text{W}_{\text{AT}}$ and $\text{GH}/\text{W}_{\text{PE}}$ were 0.098 ± 0.035 and $0.117 \pm 0.039 \text{ } \mu\text{g/l/watts}$ respectively; in Group A, pre-training $\text{GH}/\text{W}_{\text{AT}}$ and $\text{GH}/\text{W}_{\text{PE}}$ were, respectively, 0.028 ± 0.012 and $0.037 \pm 0.010 \text{ } \mu\text{g/l/watts}$

($p < 0.05$ vs normals for both), and after training these were 0.030 ± 0.014 and 0.036 ± 0.011 $\mu\text{g/l/watts}$ respectively ($p < 0.05$ vs normals for both), with a uniform pattern; in Group B pre-training $\text{GH}/\text{W}_{\text{AT}}$ and $\text{GH}/\text{W}_{\text{PE}}$ were, respectively, 0.030 ± 0.016 and 0.031 ± 0.009 $\mu\text{g/l/watts}$ ($p < 0.05$ vs normals for both) and after training these were 0.039 ± 0.015 and 0.053 ± 0.018 $\mu\text{g/l/watts}$ respectively ($p = \text{NS}$ vs normals for both), with a less uniform pattern compared to Group A and, specifically, showing an increment in 6 of 8 subjects.

To further analyse this dichotomised pattern, we also took in consideration GH AUCs following AT in group A and Group B (Fig. 2), showing a significant higher increase in the latter (Group A: from 92.3 ± 19.5 to 122.8 ± 17.7 $\mu\text{g}\cdot\text{min/l}$; Group B: from 136.5 ± 34.4 to 260.6 ± 71.2 $\mu\text{g}\cdot\text{min/l}$; Delta AUCs: 30.5 ± 12 vs 124.2 ± 38 $\mu\text{g}\cdot\text{min/l}$, $p < 0.05$).

Discussion

The stimulating effect of acute exercise on GH secretion has been extensively documented in non obese individuals (15). To our knowledge, no study has so far examined the effect of prospective exercise training on the GH response to acute exercise in obesity. Exercise is one of the most potent physiological stimuli to GH release. Oppositely, obesity dampens GH bursts caused by exercise, although the underlying mechanisms for this dysfunction are largely undetermined (1, 4). Our study sought to evaluate in obese subjects if aerobic, or aerobic plus anaerobic training could elicit a dichotomised response of GH to acute exercise. In agreement with previous studies (16), obese subjects achieved a reduced work capacity when compared to controls. Our data also confirm the known significant differences in GH response to exercise between obese and normal subjects. The latter, in fact, exhibited a greater post-training GH increment, which appeared to be steeper particularly beyond AT and persisted elevated at the peak exercise as well as during the whole recovery period. Pritzlaff et al. (17), found in young men and young women that the magnitude of GH release augmented linearly with increasing intensity of exercise. Our results in adult normal subjects thus seem to agree with a linear correlation existing between GH and increasing work

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3 outputs up to AT. Beyond AT, the increase seemed to be faster, according with the notion that
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5 resistance exercise protocols result in the greatest GH response (18). At variance with these results,
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7 obese individuals achieved a weak GH response before AT despite a clear-cut increase in the power
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9 output and, thereafter, the increase in GH levels failed to reach a magnitude similar to that obtained
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11 in control subjects. While it's known that GH secretion increases with obesity at a lower magnitude
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13 than in the lean state (1), our findings support the hypothesis that GH response to exercise in obesity
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15 is unrelated to exercise intensity before AT, while a measurable GH response could be documented
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17 following AT. A dose-response relationship thus appears to exist between exercise intensity and GH
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19 responsiveness in obesity after AT. Therefore, exceeding AT seems to act as a point for the
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21 modification in the GH response to exercise in obesity. The difference seen in the GH response
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23 seems authentic, as it also persisted when adjusted for external work rates, to correct for potential
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25 differences in physical performance among the different subgroups.
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31 Noticeably, GH levels appeared to be sustained in the post-exercise recovery period in
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33 controls, while in both obese groups GH levels returned rapidly to starting values. We speculate that
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35 this circumstance might confirm previous evidence of a decreased GH half-life (17), or an increased
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37 rate of disposal during exercise occurring with obesity by an increased clearance (1, 8).
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41 One main finding of our study resides, however, in the differential effects of a 4-wk exercise
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43 training comprising aerobic alone or aerobic plus anaerobic training on GH response to acute
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45 exercise. Training did not appear to change GH concentrations at rest and before AT significantly in
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47 either group. When expressed as areas under the curve, however, we documented that the obese
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49 group subjected to supplemental anaerobic training achieved greater GH increments beyond AT
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51 than in solo aerobic-trained subjects. A concomitant improvement in physical performance, though
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53 statistically not significant, was observed in both groups at the end of the training period, consisting
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55 in higher work rates at AT and greater maximal sustainable work capacity. It is difficult to
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57 discriminate if post-training change in GH response to exercise occurred as a function of anaerobic
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3 training or was simply due to a more intense training. Anaerobic training might reduce abdominal
4 visceral fat, and thus GH clearance.
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8 Contrary to previous studies, we found no between-gender differences in the post-exercise
9 dynamics of GH increase possibly due to a limited number of participants tested. On the other hand,
10 a recent report has noted no difference between genders in older adults (19).
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13
14 Some additional caveats should be considered in the interpretation of our results. Firstly,
15 aerobic fitness may influence GH release via effects on abdominal visceral fat and fasting insulin
16 concentrations (5). While we categorised obesity based on BMI values, a greater amount of visceral
17 fat may *per se* attenuate GH release more than overall obesity (1, 20). In line with this inference,
18 administration of rh-GH to obese adults has been shown to reduce visceral more than cutaneous
19 abdominal fat (21, 22). Thus, exercise training at an appropriate intensity should elevate GH release
20 (19) and reduce visceral abdominal fat. Secondly, the short length of the training periods may have
21 limited the significance of our observations. Being our aim to look for differences between aerobic
22 and anaerobic work, we wanted to make sure that the appropriate aerobic and anaerobic workloads
23 were carried out. So we enrolled exclusively obese in-patients, and the training period could thus
24 not be longer than four weeks. Again, each exercise was performed in the same thermo-regulated
25 room, but we had no core temperature before and after the test. Differences of it may condition
26 absolute GH levels (23). Group B had a significantly higher fat mass than Group A, nevertheless we
27 may suppose that this difference had little or no influence on GH response to training; its levels
28 before training were similar to those of Group A.
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50 In summary, single bouts of exercise seem to evoke a measurable GH responsiveness in
51 obesity only when exercise intensity overpowers AT. This also occurs after a short training period.
52 However, training intensity plays a key role in determining the magnitude of GH response, that is:
53 higher the intensity, higher the response. As such, a training program consisting of aerobic and
54 anaerobic workout may contribute to normalise GH responsiveness to exercise after AT in obese
55 subjects.
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3 Attenuation of GH responses in obesity at all exercise intensities, especially those below AT,
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5 should have implications for the application of exercise as a prescription, and higher exercise
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7 intensities might be required to stimulate an adequate GH release. Likely, studies in larger obese
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9 cohorts and following longer training are warranted to confirm and expand current results.
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3 **Figure's captions:**
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5 **Fig. 1** GH in the obese subjects during and after exercise testing before and after the period of
6 training. Group A: aerobic training (N. 8 subjects), Group B: aerobic plus anaerobic training (N. 8
7 subjects). Asterisks indicate a significant difference before and after the training period in obese
8 subjects vs controls (see the text) (* $p<0.05$, ** $p<0.01$). # significant difference after training in
9 obese subjects vs controls (# $p<0.05$).
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19 **Fig. 2** Histograms represents AUC within AT and late recovery, before and after the training
20 periods, in Group A and Group B of the obese subjects. (see the text).
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Table 1 A. Anthropometric and functional data of the samples

	Normal subjects	Obese patients	P Value ^a
No. of subjects	8	16	
Sex, M/F	4/4	7/9	NS
Age (years) ^b	34.3 ± 2.8	39.1 ± 2.9	NS
Weight (Kg) ^b	65.2 ± 5.3	102.7 ± 5.8	< 0.001
Height (cm) ^b	171 ± 3.4	168 ± 3.3	NS
BMI (Kg/m ²) ^b	22.1 ± 1.2	35.8 ± 1.1	< 0.001
Fat-free mass (Kg) ^b	50.0 ± 4.1	60.4 ± 4.2	< 0.01
Fat mass (Kg) ^b	15.6 ± 3.4	42.3 ± 2.6	< 0.01
Exercise at exhaustion (watts) ^b	140 ± 12	110 ± 8	< 0.05
AT (watts) ^b	102 ± 11	74 ± 5	< 0.02
Max VO ₂ (ml/min) ^b	1860 ± 200	1850 ± 90	NS
	(81% pred)	(77% pred)	

Table 1 B. Serum GH concentration (µg/l) during and after exercise testing

Rest ^b	1.83 ± 0.19	1.08 ± 0.08	NS
Free ^b	4.67 ± 0.30	1.36 ± 0.09	NS
20 watts ^b	5.00 ± 0.31	1.59 ± 0.09	NS
40 watts ^b	5.44 ± 0.32	1.53 ± 0.08	NS
60 watts ^b	6.13 ± 0.33	1.95 ± 0.09	NS
Anaerobic Threshold ^b	9.68 ± 0.35	2.03 ± 0.09	< 0.01
Peak ^b	16.38 ± 0.45	3.66 ± 0.10	< 0.001
Recovery 1 (5 minutes) ^b	16.69 ± 0.41	3.99 ± 0.10	< 0.001
Recovery 2 (30 minutes) ^b	8.36 ± 0.27	2.10 ± 0.07	< 0.001

^aBy two tailed analysis of variance.

^bValues are mean ± SEM.

Abbreviations: BMI=body mass index; AT=anaerobic threshold; Max. VO₂
=Maximum VO₂

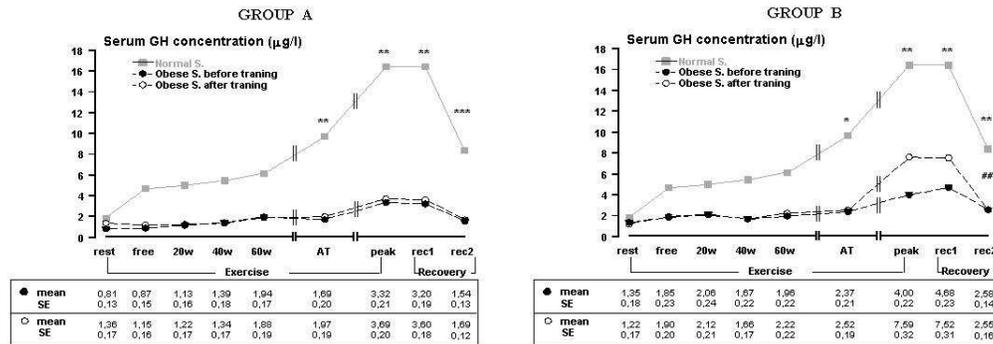


Fig. 1 GH in the obese subjects during and after exercise testing before and after the period of training. Group A: aerobic training (N. 8 subjects), Group B: aerobic plus anaerobic training (N. 8 subjects). Asterisks indicate a significant difference before and after the training period in obese subjects vs controls (see the text) (* p<0.05, ** p<0.01). # significant difference after training in obese subjects vs controls (# p<0.05).

90x31mm (300 x 300 DPI)

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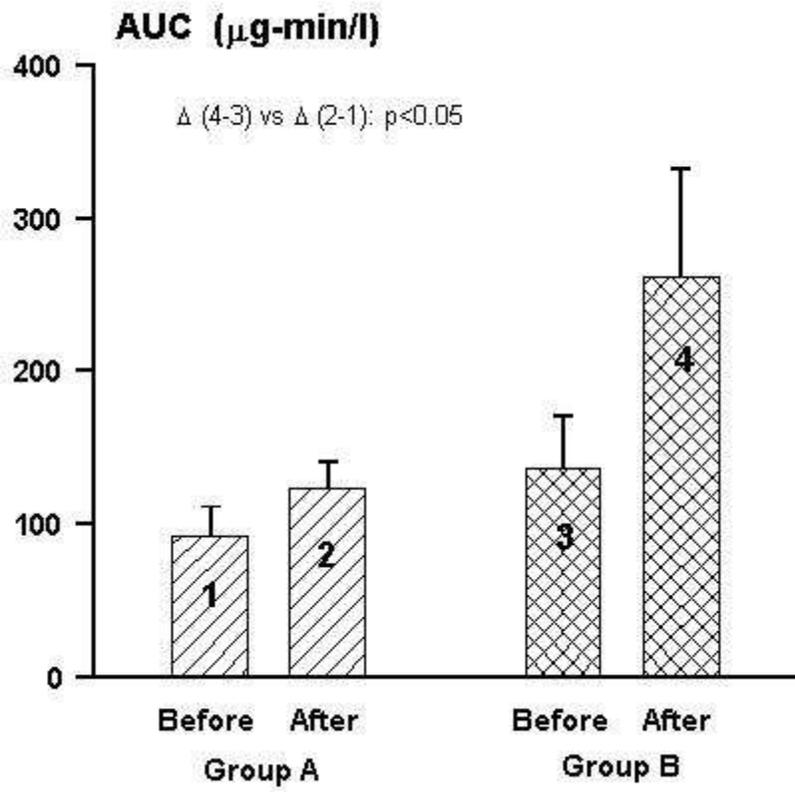


Fig. 2 Histograms represents AUC within AT and late recovery, before and after the training periods, in Group A and Group B of the obese subjects. (see the text).
75x72mm (150 x 150 DPI)

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