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# Growth hormone regulation in two types of aerobic exercise of equal oxygen uptake

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**Summary.** Five normal men, aged 23 to 35 years, participated in two bouts of continuous aerobic cycling separated by five days. The first type of exercise (EI) was cycling at a pedalling frequency of 50 rev · min$^{-1}$ with a load which produced a steady state $O_2$ uptake of approximately 40% of the subjects' $\dot{V}_{O_{2\,max}}$. The second type of exercise (EII) was cycling at a pedalling frequency of 90 rev · min$^{-1}$ with a load such that an equal steady state $\dot{V}_{O_2}$ was reached and maintained. Both EI and EII lasted 40 min. GH levels increased in EI and EII, reaching their maximum at 8 min of recovery (245 and 300% of resting values, respectively).

No significant differences were observed between EI and EII in GH, lactate, glucagon, insulin, cortisol and glucose levels between the two exercises. While it has been reported earlier that GH levels were frequently related to lactate levels and/or decreased $O_2$ availability (Sutton 1977; Raynaud et al. 1981; Kozlowski et al. 1983; VanHelder et al. 1984a, b), this study suggests that the opposite is also valid, that is, different types of exercise of equal $\dot{V}_{O_2}$, duration and lactate production do not produce significantly different GH responses.

**Key words:** Aerobic exercise — Equal oxygen uptake — Growth hormone — Lactate — Insulin

## Introduction

It is well known that the growth hormone (GH) response to exercise is influenced by the duration, intensity and type of exercise (Galbo 1983; VanHelder et al. 1984a). Oxygen availability may also influence exercise evoked responses of this powerful anabolic and glucogenic hormone as hypoxia and acute exposure to high altitude augment the GH response to exercise (Sutton 1977; Raynaud et al. 1981). Other authors have suggested that, in two exercises of nearly equal $\dot{V}_{O_2}$, the exercise employing the smaller muscular groups produces higher levels of lactate and GH (Kozlowski et al. 1983).

Recent studies (VanHelder et al. 1984a, b) have shown that the GH response to exercise correlates with the anaerobic component of exercise, suggesting a possible relationship to oxygen demand. In the above studies, exercise-evoked GH responses were always found to be associated with higher lactate levels and/or a lower $O_2$ availability in the exercising muscle, provided that the exercises being compared were of approximately equal duration and work output.

To assess the hypothesis that comparable exercises differing in $\dot{V}_{O_2}$ are associated with different GH responses, we undertook to test the opposite hypothesis, that is, different types of exercise of equal $\dot{V}_{O_2}$, duration and lactate production will not produce different GH responses.

## Material and methods

Five normal men, ages 23 to 35 years, volunteered for this study. Their average weight, height and maximal oxygen uptake (mean ± SD) were 86.4 ± 10.7 kg, 178 ± 5 cm and 46.0 ± 4.8 ml · kg$^{-1}$ · min$^{-1}$, respectively. Maximal aerobic power was determined by a direct method, using a Monark cycle ergometer and continuous gas sampling and analysis (Ergo Oxyscreen, Jaeger).

Two types of exercise separated by five days were studied: a low frequency (50 rev · min$^{-1}$)/high load cycling exercise

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(EI); and high frequency (90 rev · min$^{-1}$)/low load cycling exercise (EII). Both exercises were under the anaerobic threshold (Davis 1985) and had identical $\dot{V}_{O_2}$'s.

*Exercise protocol*

During each exercise, subjects were required to breathe through an Otis-McKerrow valve which directed expirate through large bore tubing to a Parkinson-Cowan volumeter. The volumeter was coupled to a multi-channel recorder (HP-Sanborn) which provided a continuous record of expirate volume. Medical Gas Analysers (Beckman LB-2, $CO_2$ and OM-11, $O_2$) were used continuously to monitor gas composition of the expirate at the volumeter exhaust (isolated from ambient air by 30 cm of pipe).

Low frequency pedaling exercise (EI): The ergometer load for each subject was set so as to require a pedaling frequency of 50 rev · min$^{-1}$ in order to produce a steady state $O_2$ uptake of approximately 40% of his $\dot{V}_{O_{2\,max}}$.

High pedaling frequency (EII): For each subject a pedaling frequency was set to 90 rev · min$^{-1}$ to require only a small ergometer load to produce a steady state $\dot{V}_{O_2}$ of approximately 40% $\dot{V}_{O_{2\,max}}$.

Both EI and II cycle ergometer parameters were verified with respect to their corresponding $\dot{V}_{O_2}$ one week after their initial determination. Steady state $O_2$ uptakes in EI and EII were 18.9±2.6 and 19.0±2.7 ml · kg$^{-1}$ · min$^{-1}$ (mean±SD), respectively.

All subjects ate a standardized meal six hours before exercise started. An indwelling catheter was inserted in the antecubital vein of subjects 45 min prior to the beginning of exercise. Blood samples were taken three times, eight minutes apart, prior to the beginning of exercise and then every eight minutes during 40 min of continuous cycling (Monark Ergometer) and 24 min of recovery. Blood samples were centrifuged and the serum processed for analysis using commercially-available assay kits: GH (Diagnostics Products Corp. kit), insulin (Serono Diagnostics Inc., kit), glucagon (Cambridge Medical Diagnostics, Inc., kit), cortisol (Travenol-Genentech Diagnostics), glucose (Statzyme Glucose 50, Worthington Kit), and lactate (Sigma).

The experimental values were normalized with respect to control levels and compared by Student's *t*-test and Wilcoxon ranked test.

## Results

The design of the two exercise protocols (EI and EII) achieved the stated objective of identical $\dot{V}_{O_2}$ (EI — 18.9±2.6 ml · kg$^{-1}$; EII — 19.0±2.7 ml · kg$^{-1}$, mean±SD) and below the anaerobic threshold. Normalized experimental values for the different plasma constituents are shown in Figs. 1 to 3. Growth hormone (Fig. 1) increased during exercises EI and EII and reached maximal concentrations of approximately 245 and 300% over control values, respectively, eight minutes after cessation of exercise (48 min). There were no statistically significant differences between EI and EII by either parametric or nonparametric testing.

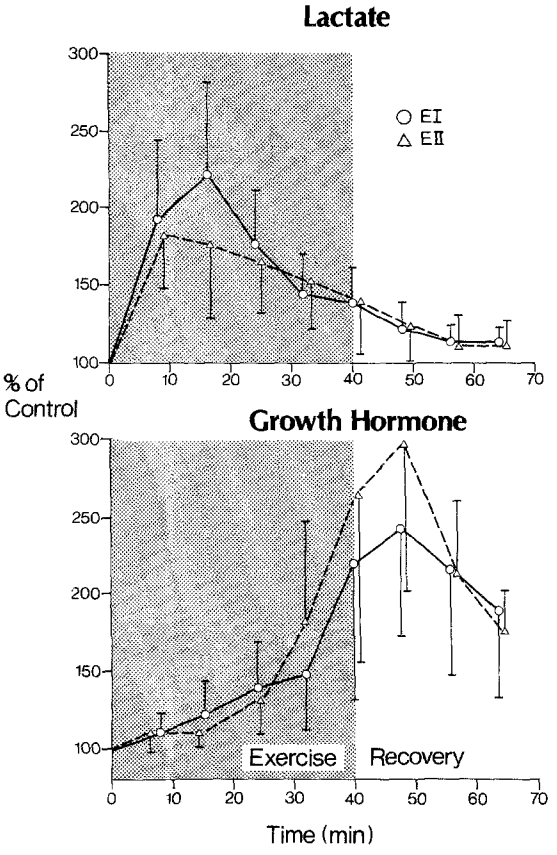


**Fig. 1.** GH and lactate serum concentrations in exercises I and II. Values are expressed as % of resting state (mean±SEM). Full symbols represent statistically significant difference ($P<0.05$) from the resting value

Lactate levels (Fig. 1) increased initially, peaking at 16 (15.±0.4 mmol · L$^{-}$1) and 8 min. (1.39±0.2 mmol · L$^{-}$1) (mean±SEM) of exercise in EI and EII, respectively, but decreased continually thereafter. No significant differences from control levels or between EI and EII were found. No significant differences (EI vs. EII) were found in insulin (Fig. 2), glucagon (Fig. 2), cortisol (Fig. 3) or glucose (Fig. 3).

## Discussion

Both types of exercise used in this investigation led to an increase in GH levels (245 and 300% above controls for EI and EII, respectively) (Fig. 1). However there was no significant difference in the GH responses between the two steady state exercises (EI & EII) which were of different frequency (50 and 90 rev · min$^{-1}$, respectively) and load, but of equal duration (40 min). Furthermore, both exercises were well under the anaerobic

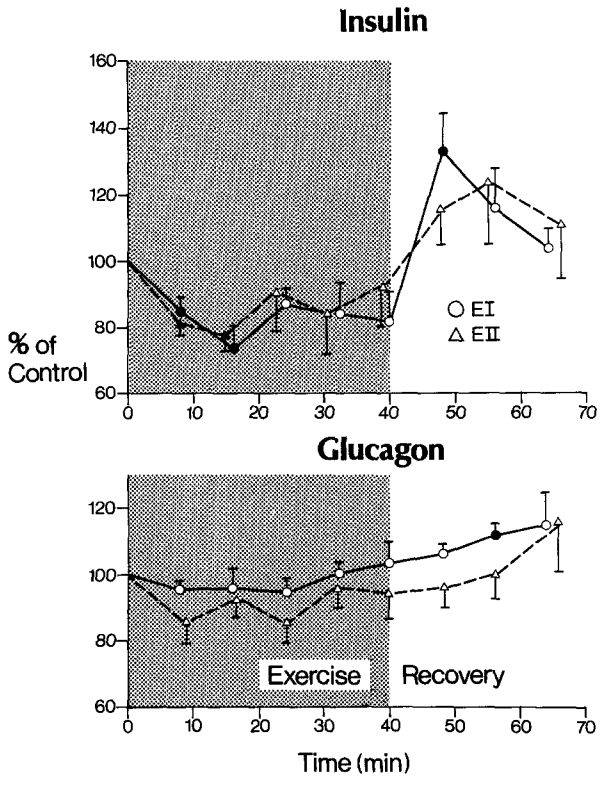


**Fig. 2.** Insulin and glucagon serum concentrations in EI and EII. Values are expressed as in Fig. 1

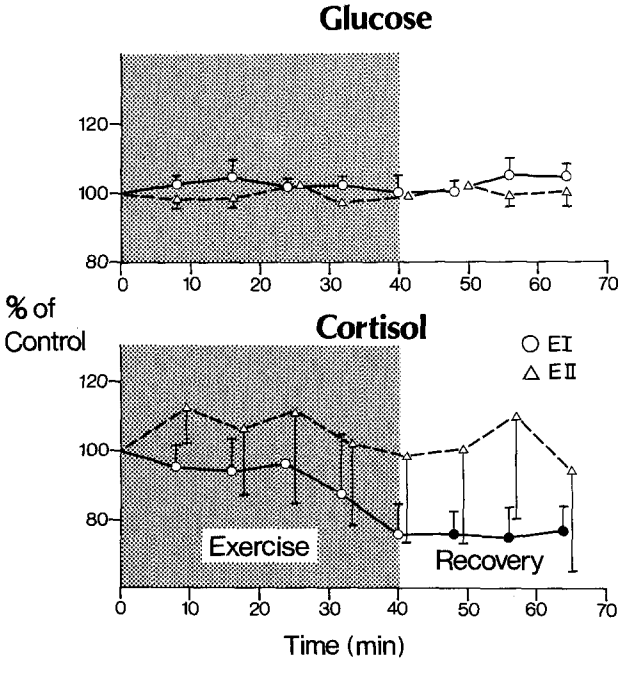


**Fig. 3.** Cortisol and glucose serum concentrations in EI and EII. Values are expressed as in Fig. 1

threshold as is evident from the changes in serum lactate (Fig. 1). The small initial increase in lactate in both exercises was associated with the initial oxygen debt induced by the onset of exercise.

Previous attempts to elucidate the regulators of GH response to various types of exercise have been confounded in many cases by comparisons of exercises of unequal aerobic ($\dot{V}_{O_2}$) and anaerobic (lactate) components, duration, and muscular groups employed. A better understanding of GH regulators might be achieved if these variables were standardized. Sutton (1977) reported that the GH response to 20 min of cycling was significantly higher in hypoxia ($P_IO_2 = 11$ kPa) than in normoxia and that the GH increase was associated with significantly higher lactate levels. Hypoxia had no effect on resting GH levels. Raynaud et al. (1981) described higher exercise-induced GH levels both in subjects acutely translocated to high altitude (2,850 m) and, on another occasion, after breathing a 15% $O_2$ gas mixture, as compared to normoxic exercise at sea level (60 min of cycling). Lactate levels were also higher during both of these hypoxic exercises.

Kozlowski et al. (1983) suggested that, at a similar $\dot{V}_{O_2}$, the exercise employing smaller muscular groups led to significantly higher lactate and GH levels. It was proposed that either muscle metabolic receptors (activated by lactate and/or decreased $PO_2$ in the working muscle) or efferent impulses from the CNS could be responsible for GH regulation. However, since these authors sampled blood only twice, before and after exercise, their suggestion of a linear increase of GH and lactate concentrations during exercise was based on only two samplings. These studies (Sutton 1977; Kozlowski et al. 1983) showed that decreased $O_2$ availability and increased anaerobiosis (reflected by lactate) were associated with higher GH levels in exercises of equal duration.

The present experiments demonstrate that the reverse is also valid; in two continuous exercises of different frequency and load but with carefully controlled (and near equal) $\dot{V}_{O_2}$ and lactate levels, no significant differences in GH concentrations were observed. This provides indirect support for the hypothesis that oxygen availability may be one of the regulators of GH release during exercise. This does not exclude the possibility that other regulators exist, such as: plasma glucose concentration (Merimee 1980); catecholamines secreted in hypothalamus rather than systemically (Hansen 1971); insulin availability preceeding the exercise (Galbo et al. 1979) which has recently

been shown to affect GH synthesis (Melmed et al. 1985); or intermittency of the exercise (VanHelder et al. 1984a). The fact that GH increased similarly both in EI and EII may be explained by the initial oxygen deficit (Lassare et al. 1974), an increased $\dot{V}_{O_2}$ over the resting state, or the existence of other unidentified factors. The lack of any change in cortisol levels would eliminate general stress as a stimulus for GH release in EI and EII. It is suggested that the oxygen availability may be one of the regulators of GH response to exercise.

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