

CLINICAL STUDY

Effect of hydration on exercise-induced growth hormone response

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Abstract

Design: Growth hormone (GH) has demonstrated water-retaining effects in subjects at rest, whereas other research has indicated that GH may stimulate sweating. Thus, the aim of this study was to investigate the effect of fluid intake on the exercise-induced GH response.

Methods: Seven healthy male volunteers (age: 27.4 ± 1.3 years, weight: 74.5 ± 1.1 kg, height: 179.3 ± 2.3 cm) performed a 40-min submaximal rectangular cycling exercise in two different sessions. The first session (Session 1) was performed without water intake, and the second (Session 2) involved the ingestion of spring water (four intakes) corresponding to the volume of water lost during the first session.

Results: In session 1, the water loss was 568 ± 32 ml. In Session 2, the volume of water loss was not significantly different from the volume of fluid intake (524 ± 16 versus 568 ± 32 ml respectively). The decrease in plasma volume was significantly reduced in Session 2 ($-6.69 \pm 1.59\%$ versus $-11.3 \pm 1.89\%$; $P < 0.05$). In Session 1, the GH concentration was significantly lower than that during Session 2 after 25 min (3.04 ± 1.05 versus 5.26 ± 1.81 ; $P < 0.05$) and after 40 min (13.7 ± 3.55 versus 17.60 ± 4.14 ng/ml; $P < 0.05$) of exercise. The total GH response was significantly lower in Session 1 than in Session 2 (136.6 ± 39.2 versus 202.4 ± 58.9 ng/ml.min; $P < 0.05$).

Conclusions: We conclude that the exercise-induced GH response decreases when exercise is performed without fluid intake.

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Introduction

The exact effects of growth hormone (GH) on body fluid remain unclear; discrepancies appear between the GH effect in subjects at rest and the effect in subjects during exercise. For subjects at rest, many studies have demonstrated the sodium- and water-retaining effects of GH (1–3). Body-fluid volume decreases in GH-deficient patients (3), but increases in acromegalic patients, who secrete excess GH (1, 4). In both groups of patients, body fluid is normalized after, respectively, GH treatment and surgical treatment of the hypophysis. In healthy men, the pharmacological administration of GH also raises body-fluid volume (2). The action of GH is thought to be mediated by aldosterone. Indeed, many studies have shown that GH stimulates aldosterone production both *in vivo* (5) and *in vitro* (6), whereas co-administration of GH and an aldosterone antagonist suppresses the body-fluid expansion induced by GH (7).

In contrast, recent investigations have suggested, that in subjects during exercise, GH facilitates water

loss by stimulating sweating (8–13). Juul *et al.* demonstrated a reduced sweating rate during exercise in GH-deficient patients relative to controls (10). Moreover, in a study performed on athletes, we showed that the water loss recorded during exercise was positively correlated with the GH secretion rate (14). In molecular research, specific receptors for GH have been found in the epithelium of sweat glands (15, 16).

Sweat-induced hypohydration during exercise will reduce plasma volume and increase plasma osmotic pressure in proportion to the level of fluid loss (17). The plasma volume decreases because it provides fluid for sweat, and osmolality increases because sweat is hypotonic relative to plasma. In order to preserve fluid homeostasis, arginine vasopressin and aldosterone secretion are stimulated (18). However, when exercise is performed with fluid replacement, secretion of arginine vasopressin and aldosterone is inhibited (19, 20).

Thus, the aim of this study was to test the effect of water replacement on GH response during exercise. We

hypothesized that if GH has the same water-retaining action during exercise as at rest, we would observe a lower GH response during exercise with fluid intake, in accordance with the well-documented abolished aldosterone response. In contrast, if GH stimulates water loss during exercise, as has been suggested in many studies, we suspected that we would observe a lower GH response during exercise without fluid replacement, as a result of feedback regulation of plasma homeostasis. To investigate these two possibilities, we studied the influence of hydration on the exercise-induced GH response in seven active, healthy men performing a rectangular exercise conducted without and with water intake.

Materials and methods

Subjects

Seven healthy male volunteers participated in this study. Their physical characteristics were as follows: age, 27.4 ± 1.3 years; weight, 74.5 ± 1.1 kg; height, 179.3 ± 2.3 cm; lean body mass, $82 \pm 1\%$ of total body weight. They performed regular physical activity, such as jogging and cycling, for approximately 2–3 h weekly and did not take any medication or nutritional supplements. We asked them to refrain from physical activities for 24 h before every test. They gave informed written consent and the procedures were approved by the local ethics committee.

Protocol

The study was conducted in two sessions with a 3-day interval. At 0800 h on each day, the subjects ate a standardized breakfast containing 2070 kJ and comprising 9.1% proteins, 27.5% lipids and 63.4% carbohydrates. The meal was composed of bread (80 g), butter (10 g), jam (20 g), milk (80 ml), sugar (10 g) and powdered coffee (2.5 g) or tea, with no additional water supplementation. This meal has been previously shown to restore normal glycemia in less than 2 h (21). Exercise testing was performed 2 h later. Room temperature was $25 \text{ }^\circ\text{C} \pm 0.5 \text{ }^\circ\text{C}$ with hygrometry at $54\% \pm 3\%$. A catheter was placed in the cubital vein for blood sampling, approximately 30 min before exercise. Subjects performed a submaximal rectangular exercise that lasted 40 min. The exercise test was performed on a cyclo-ergometer (Bodyguard; Jonas Oglænd A.S., Sandnes, Norway). The thermoregulatory response is directly linked to absolute intensity (22, 23). Because a part of our hypothesis was that GH could be implicated in thermoregulation, we chose to reproduce exactly the absolute intensity between the two sessions by using absolute workloads. Subjects were required to maintain a constant pedal speed (60 r.p.m.). A warm-up of 10 min, with 5 min at absolute workloads of 50 W and then 5 min at 100 W,

was followed by a constant period of 30 min at 150 W. The heart rate was monitored. Rectal temperature was measured, before and after exercise, with a medical thermometer.

Blood samples were drawn at 0, +10, +25, and +40 min of exercise. Because we studied the combined effects of fluid replacement and exercise on the different parameters, the value at time 0 was considered to be the reference value. Water loss was determined by the difference in weights measured before and immediately after exercise (Model F 150-S-F2, precision 1 g; Sartorius, Göttingen, Germany). For Session 1, we asked subjects to urinate before the weight measurements were taken, and they were weighed naked before the onset of exercise. Immediately after the end of the exercise session, the subjects were dried and then weighed naked. No water intake was allowed after the meal until the entire exercise test was over. For Session 2, we asked subjects to urinate before weight measurements were taken, and they were weighed naked before the first water intake at –15 min. For the 15 min preceding the onset of exercise, they remained on the cyclo-ergometer. In this session, a volume of spring water equal to the water loss recorded during the first session was ingested in four equal intakes at –15, –5, 0 and +20 min of exercise. Subjects reported not feeling much fluid in their stomach after exercise (24). In both sessions, subjects did not feel the need to urinate at the end of the exercise.

Plasma-volume variation

Changes in plasma volume (% Δ PV) during exercise were assessed from hematocrit changes by means of a formula previously used during moderate as well as maximal exercise (25–27). This formula does not need hemoglobin measurements and has been demonstrated to produce values in close agreement with values calculated from both hematocrit and hemoglobin measurements (25, 28). This formula can be reduced to:

$$\% \Delta \text{PV} = \frac{10\,000(H_0 - H)}{H_0(100 - H_0)}$$

where H_0 is the resting hematocrit value and H is the hematocrit value during exercise. The percentage of total plasma loss at end of the exercise was calculated with the hematocrit for time +0 and the hematocrit for time +40 min. The hematocrit was determined in duplicate with a small amount of blood collected from each blood sample in a microcapillary tube. This latter underwent microcentrifugation for 5 min at 15 000 *g*. Values were obtained using a specific standard scale. The coefficient of variation of the method was about 0.9%.

Plasma viscosity

Blood samples for plasma-viscosity measurements (7 ml) were drawn into a vacuum tube (Vacutainer, Belliver Industrial Estate, Plymouth, UK), with potassium EDTA as the anticoagulant. They were centrifuged for 15 min at 3000 r.p.m.; 1 ml plasma was collected in the viscometer-specific syringe, and the plasma viscosity was measured at a high shear rate (1000 s^{-1}) with a falling ball viscometer (MT 90; Mediatest, Saint Benoit, France) (29). The coefficient of variation of this method ranged between 0.6% and 0.8% (30).

GH assay

GH was assayed by immunoradiometry with the ELSA-hGH kit (CIS Bio International, Gif-sur-Yvette, France). The intra-assay and interassay coefficients of variation were 2.5% and 3.8% respectively, with a sensitivity of 0.1 ng/ml.

Statistics

The results are presented as means \pm the standard error of the mean (S.E.M.). A verification of a normal distribution for all values was performed with the Kolmogorov-Smirnov test. According to the results of this test, the effects of exercise and fluid replacement on plasma GH concentrations and plasma viscosity were evaluated with a two-way ANOVA for repeated measurements; changes in plasma volumes at the end of exercise, water loss, and rises in rectal temperature were compared using a paired *t*-test; and the GH area under the curve (GH AUC) was calculated using the trapezoidal rule; values were compared using a paired *t*-test. A value of $P < 0.05$ was considered to be significant.

Results

Water-loss differences between the two sessions did not reach a significant levels: $568 \pm 32 \text{ ml}$ versus $524 \pm 16 \text{ ml}$. Thus, the water intake during the session involving fluid replacement was not significantly different from the water loss. The change in plasma volume was significantly lower in the session involving fluid replacement (Session 2) than in the session with no fluid replacement (Session 1) ($-6.69 \pm 1.59\%$ versus $-11.3 \pm 1.89\%$ respectively; $P < 0.05$; Fig. 1). In Session 1, the plasma viscosities at time +25 and time +40 min were significantly higher than the pre-exercise value (time 0) ($1.45 \pm 0.02 \text{ mPa s}$ and $1.43 \pm 0.03 \text{ mPa s}$ versus $1.38 \pm 0.02 \text{ mPa s}$ respectively; $P < 0.05$). In Session 2, the plasma viscosities at +25 and +40 min were significantly higher than the pre-exercise value (time 0) ($1.41 \pm 0.02 \text{ mPa s}$ and $1.39 \pm 0.02 \text{ mPa s}$

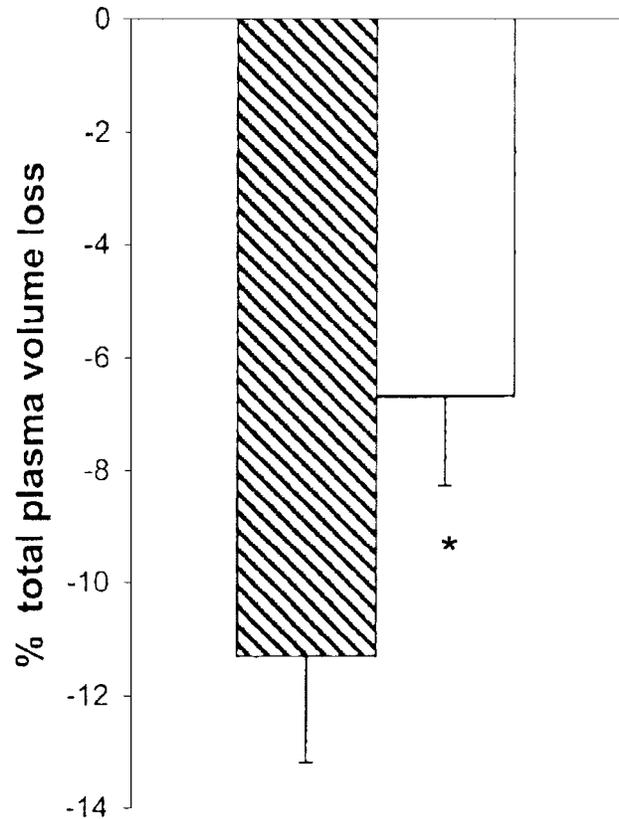


Figure 1 Percentage of plasma-volume loss over two sessions of a submaximal exercise. Session 1, hatched bar, without water intake; Session 2, open bar, with water intake. Values are means \pm S.E.M.; $n = 7$; *significant difference between two sessions, $P < 0.05$.

versus $1.34 \pm 0.02 \text{ mPa s}$ respectively; $P < 0.05$). The plasma viscosities were significantly lower at +25 and +40 min of exercise in Session 2 than in Session 1 ($1.41 \pm 0.02 \text{ mPa s}$ versus $1.45 \pm 0.02 \text{ mPa s}$, and $1.39 \pm 0.02 \text{ mPa s}$ versus $1.43 \pm 0.03 \text{ mPa s}$, respectively; $P < 0.05$; Fig. 2). In Session 1, the plasma GH levels at +25 and +40 min were significantly higher than the pre-exercise value (time 0) ($3.04 \pm 1.05 \text{ ng/ml}$ and $13.70 \pm 3.55 \text{ ng/ml}$ versus $0.30 \pm 0.15 \text{ ng/ml}$ respectively; $P < 0.05$). In Session 2, the plasma GH levels at +25 and +40 min were significantly higher than the pre-exercise value (time 0) ($5.26 \pm 1.81 \text{ ng/ml}$ and $17.60 \pm 4.14 \text{ ng/ml}$ versus $0.31 \pm 0.15 \text{ ng/ml}$ respectively; $P < 0.05$). The plasma GH concentrations were significantly lower at +25 and +40 min in Session 1 than in Session 2 ($3.04 \pm 1.05 \text{ ng/ml}$ versus $5.26 \pm 1.81 \text{ ng/ml}$ respectively, and $13.70 \pm 3.55 \text{ ng/ml}$ versus $17.60 \pm 4.14 \text{ ng/ml}$ respectively; $P < 0.05$; Fig. 3). The GH AUC was significantly lower in Session 1 than in Session 2 (136.6 ± 39.2 versus 202.4 ± 51.90 respectively; $P < 0.05$; Fig. 4). The rise in rectal temperature was significantly higher in Session 1 ($+1.2 \pm 0.1^\circ \text{C}$ versus $+0.9 \pm 0.1^\circ \text{C}$; $P < 0.05$).

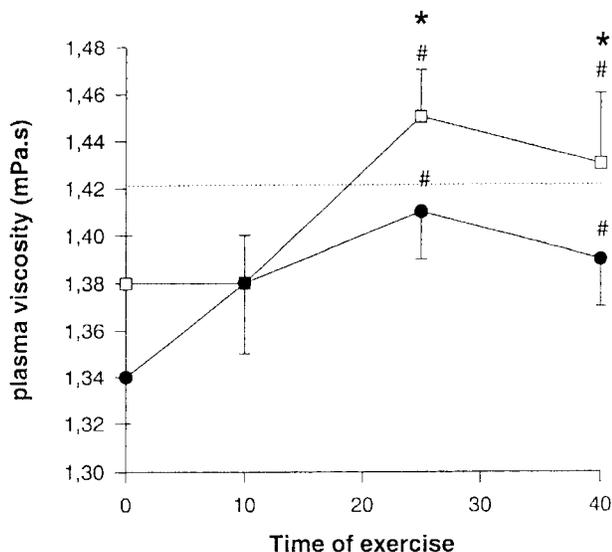


Figure 2 Plasma-viscosity kinetics during two sessions of a submaximal exercise. Session 1, without water intake (□); Session 2, with water intake (●). Values are means ± S.E.M.; *n* = 7; *significant difference between sessions, #significant difference from basal value (time 0); *P* < 0.05. The dotted line indicates the normal limit of plasma viscosity in subjects at rest.

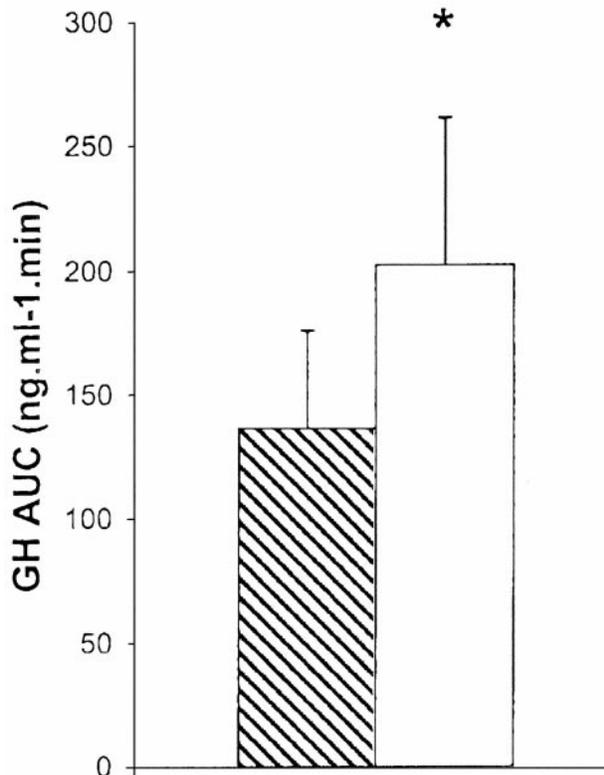


Figure 4 GH AUC during two sessions of a submaximal exercise. Session 1, hatched bar, without water intake; Session 2, open bar, with water intake. Values are means ± S.E.M.; *n* = 6, *significant difference between two sessions, *P* < 0.05.

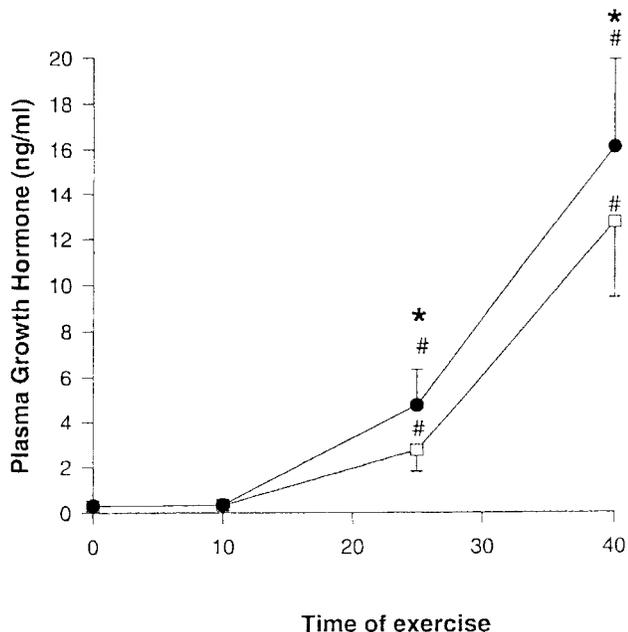


Figure 3 GH-concentration kinetics during two sessions of a submaximal exercise. Session 1, without water intake (□); Session 2, with water intake (●). Values are means ± S.E.M.; *n* = 7 (*n* = 6 for time 40); *significant difference between two sessions, *P* < 0.05; #significant difference from basal value (time 0), *P* < 0.05.

Discussion

We observed a significantly lower plasma GH concentration after +25 and +40 min of exercise, and a significantly lower GH AUC during the session without fluid intake.

The exercise-induced GH response is influenced by many factors such as age, gender, training status, and pre-exercise meal composition (31, 32). Thus, we performed the two sessions in the same subjects, all of whom had been given the same standardized meal. Moreover, the GH response is also influenced by other factors that are difficult to measure simultaneously, such as the levels of epinephrine, norepinephrine, acetyl choline and dopamine (33–35). Thus, the aim of this study was limited to determining if the effect of hydration on the exercise-induced GH response would indicate stimulation of water loss by GH, as previously described, or water retention – in accordance with its action in subjects at rest.

From a methodological point of view, an initial adaptive session with water-loss measurement, followed by two randomized experimental conditions with and without fluid intake, would have been interesting. However, an adaptation to stress in the second session

would not explain the higher GH concentrations in this session. Moreover, it has been assumed that the optimal rate of fluid ingestion is always that which equals the rate of fluid loss (36). In accordance, the volume of fluid intake given in the session conducted with fluid replacement did not differ statistically from the measured water loss. The significant decrease in plasma volume in spite of an ideal volume of fluid intake may be explained by the mineral-free nature of the spring water ingested (37, 38). However, the decrease in plasma volume in the session conducted with fluid intake was significantly lower than that in the session conducted without fluid intake. Moreover, plasma viscosity was also significantly lower in the session conducted with fluid intake, and remained in the normal range. The difference in plasma viscosity between the two sessions was in accordance with the previously described effect of water ingestion on plasma viscosity (39) and with the lower plasma-volume loss observed in the session involving fluid intake.

Convertino *et al.* (25) reported an exponential correlation between the variation of plasma osmolality and the variation of plasma volume calculated using the same formula used in this study. Our results showed a significantly higher plasma-volume variation in the session conducted without fluid intake than in the session involving fluid intake. In agreement with the previous report of Convertino, it could be suggested that there is higher plasma-osmolality variation in the session conducted without fluid intake, and, consequently, higher osmoreceptor activity. During this same session, the GH concentration was significantly lower after +25 and +40 min of exercise, as was the total GH response estimated by using the GH AUC. This might suggest that the mechanism regulating plasma homeostasis led to an inhibition of the GH response. These results support the previously suggested view that GH has a stimulating effect on water loss rather than the water-retaining action observed in subjects at rest. We hypothesized that the GH response is under the control of baroreceptors and osmoreceptors, though this remains to be demonstrated. In this study, the GH concentration was significantly lower in the session conducted without fluid intake than in the session conducted with fluid intake only after 25 min. As exercise was performed for only 40 min, this was probably not long enough to detect a GH effect on sweating, by means of weight measurement. Exercise requires the body to attempt to cope simultaneously with competing demands for thermoregulation and fluid homeostasis. Previous studies have demonstrated that the GH response during exercise is correlated with core-temperature variation, suggesting that the GH response is under the control of thermoreceptors, in accordance with its suggested role in stimulating water loss (40, 41). Our study suggests that the GH response is also under the influence of the negative feedback of osmoreceptors and baroreceptors. We hypothesize that

the GH response during exercise is partly the result of these two opposite influences. In this study, our subjects performed exercise in relatively thermo-neutral conditions, and the exercise-induced core-temperature rise was significantly higher in the session conducted without fluid replacement; however, from a physiological point of view, this rise was minor. Thus we assume that the GH response was more influenced by the inhibitory effect of osmoreceptors and baroreceptors, and, consequently, this response during the session conducted without fluid intake was lower. In contrast with our results, Saini *et al.* (42) showed a lower exercise-induced GH response with rehydration during exercise in the heat (36 °C) in healthy subjects. They suggested that the lack of a GH response with rehydration was the result of a decreasing physiological stress level. A predominant effect of thermoreceptors during exercise in the heat may explain the higher GH response under dehydration reported by Saini *et al.* (42).

It has been previously reported, moreover, that during longer exercise periods hypovolemia contributes to the reduction in sweat loss (43, 44). It remains to be determined if the effect of volemia on GH secretion during such exercise is involved in reduced sweat loss.

The aim of our study was to determine if GH, during exercise, acts to stimulate water loss, as previously described, or to contribute to water retention in accordance with its action in subjects at rest. Our results are in accordance with those studies which suggest a role for GH in the stimulation of water loss. The mechanisms by which the opposite effects of GH occur between rest and exercise nevertheless remain to be elucidated.

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