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Exercise-induced growth hormone secretion and hemorheology during exercise in elite athletes

C. Peyreigne [a], D. Bouix [a], J.P. Micallef [b], J. Mercier [b], J. Bringer [b], C. Préfaut [b] and J.-F. Brun [b,*]

[a] *Service Central de Physiologie Clinique, Centre d'Exploration et de Réadaptation des Anomalies du Métabolisme Musculaire (CERAMM), CHU Lapeyronie, 34295 Montpellier Cédex 5, France*
[b] *Département de Physiologie, Institut de Biologie, Faculté de Medecine, INSERM U103, 34060 Montpellier, France*

1. Introduction

Sweating is induced during hyperthermia by cholinergic sympathetic inputs triggered by thermoreceptor-mediated CNS activation. In this process, plasma filtration is increased in cutaneous sweat glands [1–3]. This water loss results in a decrease in both blood volume and blood fluidity [4–8] and is thus likely to exert negative effects on performance.

Recent research suggests that growth hormone (GH) may be involved in the mechanism of sweating. Studies in GH-deficient subjects compared to controls demonstrate a reduced sweating rate during exercise that could increase the risk of hyperthermia [9–14]. On the other hand, acromegalic patients suffer from excess sweating [15]. Specific receptors for GH have been evidenced in the epithelium of sweat glands [16,17]. However, studies on the effects of GH on sweating are difficult for several reasons. First, animal models are not relevant to this chapter of human physiology [18]. For instance, exercise reduces GH release in rats while it increases it in humans. On the other hand, experimental alterations in GH status cannot be performed in humans for ethical reasons, unless in deficient subjects undergoing treatment. Actually, there is a wide physiological range of GH responses and GH-related growth factors levels in humans according to the training status and body composition. Furthermore, sweating rates are quite different among humans of various fitness and training conditions [19–22]. To our knowledge, a correlational study of sweat, hemorheology, and GH has not yet been reported.

Thus, the scope of this work was to investigate interrelationships among water loss, blood rheology, and GH response during exercise in healthy athletes.

*Correspondence to: Dr. Jean-Frédéric Brun, M.D., Ph.D., Département de Physiologie, Institut de Biologie, Faculté de Medecine, 34060 Montpellier, France. Fax: +33 4 6733 8963.

Table 1
Characteristics of study subjects with their adaptation to exercise and their water loss (mean ± SEM)

	Age (years)	Weight (kg)	Height (cm)	Surface (m^2)	Fat mass (%)	Lean mass (%)	$VO_{2\,max}$ (ml/min/kg)	W_{170} (W/kg)	Water loss (g/m^2)
Mean	18.50	80.10	190.20	2.08	10.58	89.42	47.55	2.68	191.02
SEM	0.21	2.13	2.22	0.04	0.41	0.41	2.87	0.16	25.65

2. Subjects and methods

2.1. Subjects

10 young athletes (football and volleyball players) involved in international matches underwent an exercise-test during a medical check-up. Their characteristics are shown in Table 1.

2.2. Exercise test

No alimentary restriction was imposed; however, subjects were asked to fast for 12 h before commencement of the test at 8:30 a.m. A cannula was placed in the cephalic vein at the level of the cubital fossa for blood sampling at various times. The exercise-test was performed on cycloergometer (Bodyguard, Jonas Oglaend A.S., N4301 Sandnes, Norway). It consisted of a 25 min cycling session with the first 10 min being a warm-up period at 50 W followed by a 15 min plateau at 85% of the theoretical maximal heart rate given by the tables of the American Heart Association. Pedal speed was kept constant at 60 rpm by the subjects. Physical working capacity W_{170} was calculated, this being the work in watts that subjects were able to perform at a heart rate of 170 b min^{-1} [23]. $VO_{2\,max}$ was also indirectly calculated from these submaximal steps with a home-made software using the classical Astrand's nomograms [34] (Table 1). In order to standardize GH response, as previously indicated [35] the exercise-test was performed at the same hour in the morning (10:30 a.m.) exactly 120 min after a standardized breakfast [36]. They ate a standardized breakfast containing 2070 kJ with 9.1% proteins, 27.5% lipids, and 63.4% carbohydrates. The meal was composed of bread (80 g), butter (10 g), jam (20 g), skimmed concentrated milk (80 ml) from Gloria SA, 14 rue de Bassano, 75783 Paris Cédex 16, sugar (10 g) and powder coffee (2.5 g). The average time for consuming the meal was 6 min.

2.3. GH

Growth hormone was assayed by immunoradiometry with the kit ELSA-hGH of CIS Bio International, France. Since some of the apparent GH increase during exercise results from hemoconcentration, we corrected values at time +10 and +25 min for plasma volume contraction. Changes in plasma (% ΔPV) during exercise were evaluted from hematocrit changes with a formula developped at the NASA-Ames Research Center [24–26] and that has been demonstrated to be valid in moderate as well as maximal exercise:

$$\%\,\Delta PV = 100/(100 - H_0) \times 100[(H_0 - H)/H_0],$$

where H_0 is resting hematocrit and H hematocrit during exercise.

2.4. IGF1

Insulin-like growth factor 1 (somatomedin C) was measured by radioimmunoassay with the kit INCSTAR IGF1-RIA of INCSTAR Corporation, Stillwater, MN, USA.

2.5. Water loss

Dehydration was evaluated by precision weighing (Sartorius model F 150-S-F2, France). Cutaneous surface (S^2 (skin)) was calculated from height (H (cm)) and weight (W (kg)) with the formula of DuBois and DuBois [27]:

$$S^2 \text{ (skin)} = H^{0.725} \text{ (cm)} \times W^{0.425} \text{ (kg)} \times 71.84 \times 10^{-4} \quad \text{(Table 1)}.$$

2.6. Plasma viscosity

Blood samples for hemorheological measurements (7 ml) were drawn with potassium EDTA as the anticoagulant in a vacuum tube (Vacutainer). Plasma viscosity was measured at high shear rate (1000 s^{-1}) with a falling ball viscometer (MT 90 Medicatest, F-86280 Saint Benoit) [28,29]. Accuracy of the measurements was regularly controlled with the Carrimed Rheometer 'CS' (purchased from Rhéo, 91120 Palaiseau, France) [30]. The coefficient of variation of this method ranged between 0.6 and 0.8% [31].

2.7. Statistics

Results are presented as mean ± the standard error of the mean (SEM). A value of $p < 0.05$ was considered as significant. Comparisons were made with nonparametric tests. Linear correlations were tested by least-square fitting. GH concentrations corrected for blood volume contraction (GHc) and plasma viscosity (η_{pl}) at the 10th and the 25th min of exercise were compared with the Wilcoxon rank sum test for paired data. Integrated concentrations of GH were calculated with the trapezoidal rule. Partial correlation analysis was used to compare correlations among changes in GH, η_{pl} and water volume.

3. Results

Water loss was 191 ± 25.5 g/m^2 (Table 1). There was a significant increase ($p < 0.01$) in plasma GH corrected for plasma volume contraction (GHc) at the 10th and the 25th min of exercise compared to baseline (Fig. 1). There was also a significant increase ($p < 0.05$) in η_{pl} at the same times (Fig. 2). We observed a significant correlation ($r = 0.72$, $p < 0.01$) between the total GH increase during exercise (area under the curve of GHc) and water loss (Fig. 3). There was another correlation ($r = 0.819$, $p < 0.01$) between water loss and the change in η_{pl} between 0 and 25 min (Fig. 4) and a correlation between η_{pl} and GHc ($r = 0.776$, $p < 0.01$, see Fig. 5). Partial correlation analysis suppressed the correlation between changes in GH and η_{pl} when the variable "water loss" was kept constant, since at fixed water loss became 0.404 (non-significant). Neither GH, η_{pl} nor water loss were correlated to the total amount of work (area of power output plotted against time) that was quite similar among subjects (185.25 ± 2.82 kJ, range: 165–195 kJ). No significant correlation between basal IGF1 and this total GH increase was found.

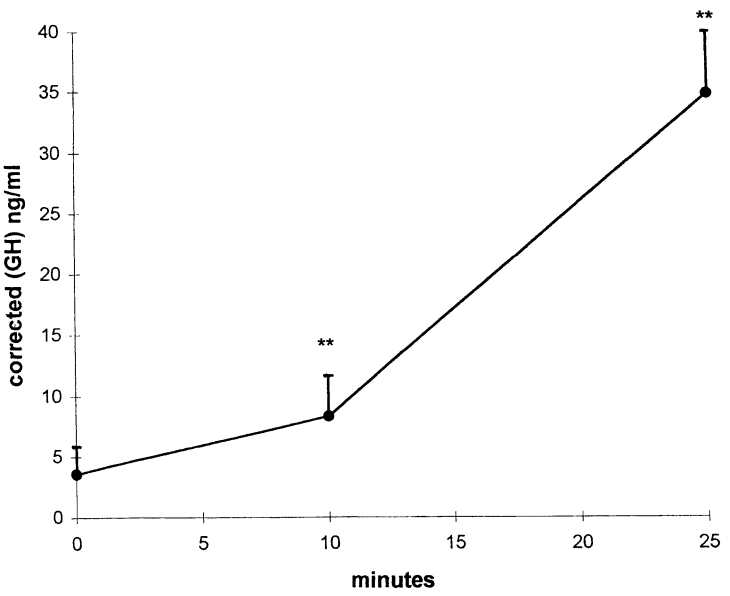

Fig. 1. Kinetics of growth hormone (corrected for hemoconcentration) during exercise. $^{**}p < 0.01$.

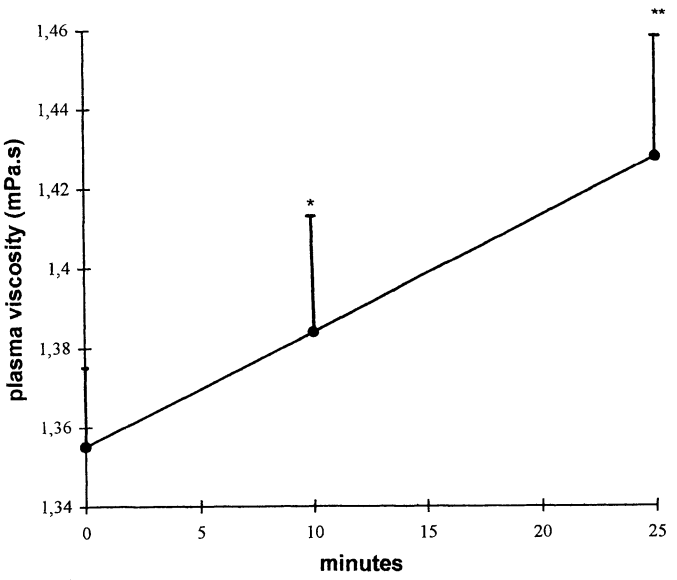

Fig. 2. Kinetics of plasma viscosity during exercise. $^{*}p < 0.05$, $^{**}p < 0.01$.

4. Discussion

Results of this study are in agreement with our working hypothesis of exercise-induced GH release being involved in hemorheologic changes via a role of GH in water loss by sweat.

Correlations among parameters that increase during exercise should always be interpreted with caution, since most of the apparent changes could be explained by hemoconcentration. For this reason, we corrected GH values for plasma volume contraction and used only these corrected values (GHc) in our

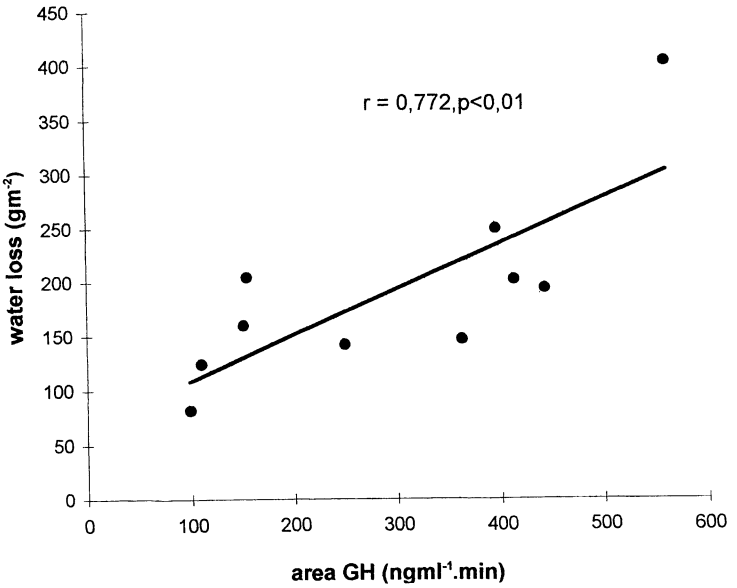

Fig. 3. Correlation between the area under the curve of corrected GH concentration and water loss.

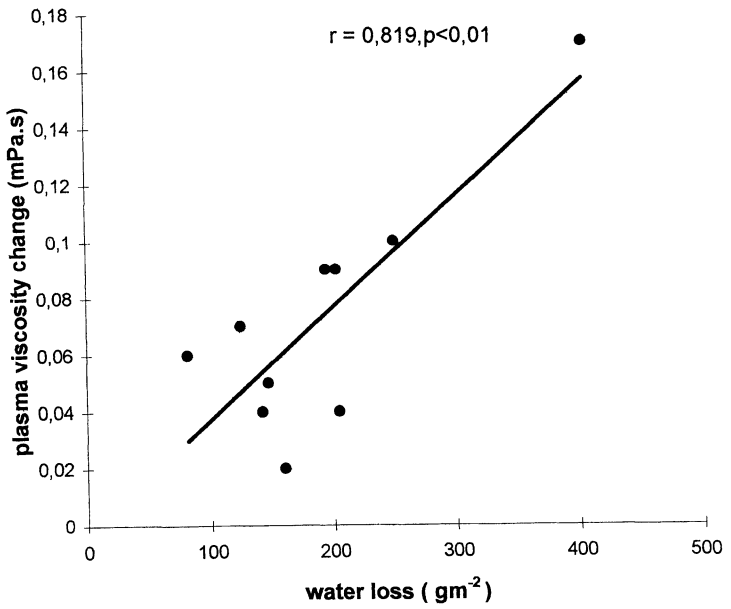

Fig. 4. Correlation between water loss and the change in plasma viscosity between 0 and 25 min.

interpretation of results. Even after this correction, GH response is correlated with the increase in plasma viscosity. Since both parameters are also correlated with water loss, results support the assumption that GH may be a determinant of water loss in the sweat, this water loss being itself a factor involved in the increase in plasma viscosity [32]. Partial correlation analysis further supports this hypothesis, since the correlation between GH response and η_{pl} changes appears to be dependent on water loss. Thus, water loss may represent a causal link between GHc and η_{pl} during exercise.

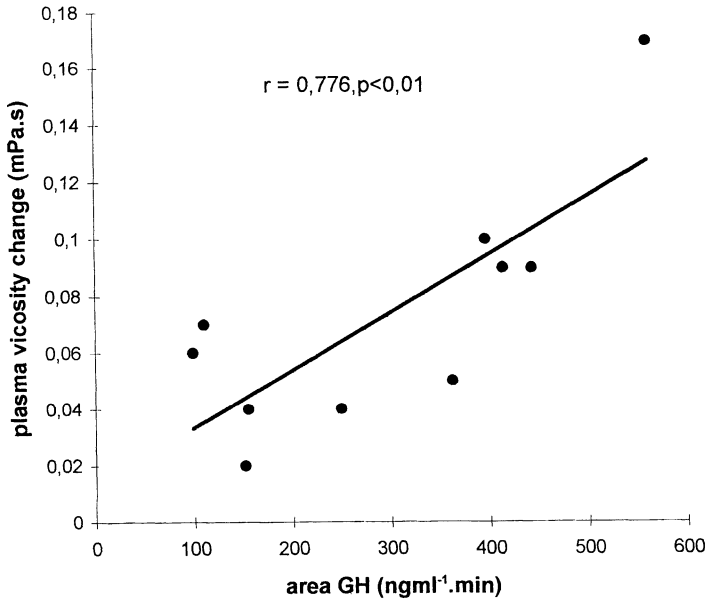

Fig. 5. Correlation between the change in plasma viscosity and the area under the curve of corrected GH concentration.

Our group of subjects including elite young volleyball players is characterized by some very high GH responses, with plasma GH levels higher than the quite unusual level of 70 ng/ml. Such a sample of subjects provides a unique opportunity for investigating the biological correlates of GH response. The wide range of GH responses was worthwile for such a correlative study. Besides, all these sportsmen were submitted to a strenuous regular aerobic training which was likely to make them a rather homogeneous group with respect to training effects. It is clear from our results that the stronger is the GH response, the greater is the water loss, with at the level of plasma a more pronounced increase in viscosity.

Since the total work output is a major determinant of both GH response [33] and heat elimination by sweat [4] during exercise, we ruled out the alternative hypothesis, i.e., our findings are explained by a correlation of GH and sweat volume with the amount of work performed. In this sample, which includes trained sportsmen with a high aerobic capacity, the power output at 85% of the theoretical maximal heart rate was almost the same, so that work did not significantly explain the variability in GH and sweat loss. Thus, we assume that the correlations we report in this sample are not dependent upon differences in work output among subjects.

The previous studies of Juul and coworkers [9,10,12] demonstrated that GH deficient patients were characterized by a low sweating capacity during exercise. However, these investigators could not rule out that the low sweat rate of these patients could be the result of an irreversible atrophy due to the low IGF1 levels before GH therapy. Obviously, in our study, these healthy young sportmen had normal IGF1 levels. Even in that case, sportmen with the greatest GH secretion during exercise had also the greatest water loss, ie, our results are in agreement with Juul's concept of a role of GH in sweating during exercise.

Whether this relationship is explained by chronic effect of increased GH and GH-related IGF production (that is reflected by a high postexercise GH peak) or by an acute rise in GH itself is less clear. While the first hypothesis seems more likely, if one looks at the current literature on GH physiology, an acute dynamic effect of the exercise-induced GH surge on sweat glands during exercise can also be hypoth-

esized, since GH receptors have been detected on the epithelium of sweat glands [16,17]. Whether GH may have a direct effect on ion and fluid shifts, at this level remains unknown.

On the whole, this study is in agreement with the recent data that suggest an influence of the somatotropic function on exercise-induced sweating, even in normal subjects. Accordingly, via these putative effects on fluid shifts during exercise, GH could be hypothesized to influence blood rheology.

This study was presented as an oral communication at the XVII Congress of the French Society of Sports Medicine (SFMS), Caen, France, 19–21 June 1997.

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