ROLE OF BODY TEMPERATURE IN EXERCISE-INDUCED GROWTH HORMONE AND PROLACTIN RELEASE IN NON-TRAINED AND PHYSICALLY FIT SUBJECTS

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Objective. To study the role of physical fitness and hyperthermia in inducing growth hormone (GH) and prolactin (PRL) responses to exercise in physically fit and in non-trained healthy subjects.

Methods. Ten wrestlers preparing for international competition (mean age 19), and nine untrained healthy males (mean age 21), volunteered in the study. They were exposed twice to the exercise consisting of 27 min swimming, freestyle, in water of 29 or 36 °C, with last 3 min increased to maximal effort. Measurement of blood pressure, heart rate, sublingual temperature and sampling of blood was performed before exercise, immediately after and after a 30 min period of rest.

Results. Body temperature, heart rate, systolic blood pressure and plasma growth hormone (GH) were significantly elevated in both groups after swimming in water of either temperature ($P<0.01$). The difference between GH responses to swimming in water of 29 °C vs 36 °C was significant only in non-trained subjects and was associated with the changes of body temperature. A rise in PRL concentration was found only in exercise in warmer water ($P<0.01$). There were no statistical differences between athletes and controls in any response to swimming in water of the same temperature.

Conclusions. The augmented release of GH and PRL was the result of direct stimulation by increased body temperature.

Key words: Fitness – Swimming – Water temperature – GH – PRL – Wrestlers

Dynamic submaximal exercise induces hormonal responses including an elevation of GH and to a lesser degree also an increase of PRL (Oleshansky 1990; Brisson et al. 1991; Fischer et al. 1991). Neuroendocrine response to exercise depends on several factors such as the type, duration or intensity of exercise. However, also the environmental temperature and physical fitness are considered important components influencing the extent of hormonal response (Viru 1992; Radomski et al. 1998). Muscle work augments the production of heat and after the elevation of body temperature even the heat dissipation (Shimizu et al. 1998). Thus it provides a disadvantage for the exercising organism: there are increased and often conflicting demands on the cardiovascular system, since both working muscles and cutaneous circulation require large portions of the cardiac output. These increased requirements may result in greater neuroendocrine response.

Most hormones released during exercise are increased in plasma also during the heat exposure (Leppaluotto et al. 1986; Moller et al. 1989; Kaciuba-Uscilko et. al. 1992; Jezova et al. 1994; Brisson et al. 1991). The mechanism(s) of heat induced neuroendocrine activation are not fully understood. Direct stimulation was reported to be operative by signals generated in core thermoreceptors for growth

hormone (Christensen et al. 1984) and in skin thermoreceptors for catecholamines (Weiss et al. 1988), but even indirect influence via increased demands on the cardiovascular system during exercise hyperthermia cannot be excluded.

To elucidate the indirect participation of hyperthermia in augmentation of neuroendocrine responses to exercise, trained wrestlers and non-trained subjects were exposed to swimming in water of different temperatures. The study was based on the assumption that trained subjects could easier cope with increased demands on the cardiovascular system during exercise in higher environmental temperature and thus respond with lower neuroendocrine activation than non-trained subjects.

Subjects and Methods

Subjects. Ten highly trained young wrestlers (mean ± S.E., height 176±2 cm, weight 72±3 kg, age 19±0.6 years) of national rank were tested during their preparation for the competition season. They performed general fitness training and specific technique training twice a day. A group of nine healthy untrained males (181±3 cm, 76±3 kg, age 21±0.8 years) served as controls.

The investigation was approved by the Ethical Committee of the Institute of Experimental Endocrinology, Slovak Academy of Sciences. Written informed consent was obtained from all subjects before the study.

Design of the study. Volunteers fasting overnight arrived in the morning at 8.00 h. Each subject participated in two randomised experiments separated by 8-11 days. After 30 min rest in comfortable sitting position, blood pressure was measured with a sphyngomanometer, heart rate by auscultation and sublingual temperature with a mercury thermometer. Blood sample was withdrawn for determination of pre-exercise values of hormones. The subjects then entered the pool with water of 29 or 36 °C. They were swimming 24 min free style at a self-selected comfortable pace, followed by 3 min freestyle at maximal pace under verbal encouragement by the trainer. The subjects remained sitting in the pool, measurements were performed and blood samples were immediately withdrawn. Then the subjects left the pool and rested for 30 min in horizontal position, covered with a blanket. Finally the last measurements were performed and blood samples withdrawn.

Analyses. Blood samples cooled on ice were centrifuged and plasma aliquots were frozen until assayed. All samples were run in the same assay. Plasma GH and PRL were determined by RIA kits of Immunochem (Prague, Czech Republic) and glucose by the glucose-oxidase method (Boehringer, Germany).

Statistical evaluation. Two way ANOVA for repeated measures with consecutive post-hoc test was used to determine the differences from baseline within each group, between the groups and between different water temperatures.

Results

Control group. As shown in Fig. 1 (left panels), the exercise-induced increase of body temperature was significant after swimming in water of either temperatures ($P<0.01$), but the increment in 29 °C water reached less than half (0.5±0.1 °C) of the increment found in 36 °C (1.3±0.1 °C), and this difference was significant ($P<0.01$). A similar response was found in heart rate with significant elevation after swimming in water of either temperature ($P<0.01$), which was again significantly higher in 36 °C water ($P<0.05$). At both temperatures swimming induced the same increase in systolic blood pressure (<0.01). Diastolic blood pressure was not changed by swimming in cooler water, while it showed a significant decrease in 36 °C water (1.3±0.1 °C).

Pre-exercise plasma GH was the same on both days of investigation. Swimming induced significant elevations of plasma GH concentrations ($P<0.01$) with increments exceeding 20 ng/ml in 29 °C water and 30 ng/ml in 36 °C water (Fig. 2, left panels).

Swimming in water of 29 °C water did not induce any elevation of plasma PRL concentration, however in 36 °C water the increment exceeding 10 ng/ml was more than twofold of the pre-exercise plasma concentration ($P<0.01$) (Fig. 2, left panels). The difference between the post-exercise values of PRL in cooler and warmer water was significant ($P<0.01$).

Wrestlers. Fig. 1 (right panels) shows that body temperature was increased by swimming in either water ($P<0.01$), but the incements in cooler water (0.9±0.1 °C) and in warmer water (1.3±0.2 °C) were not statistically different. Swimming-induced eleva-

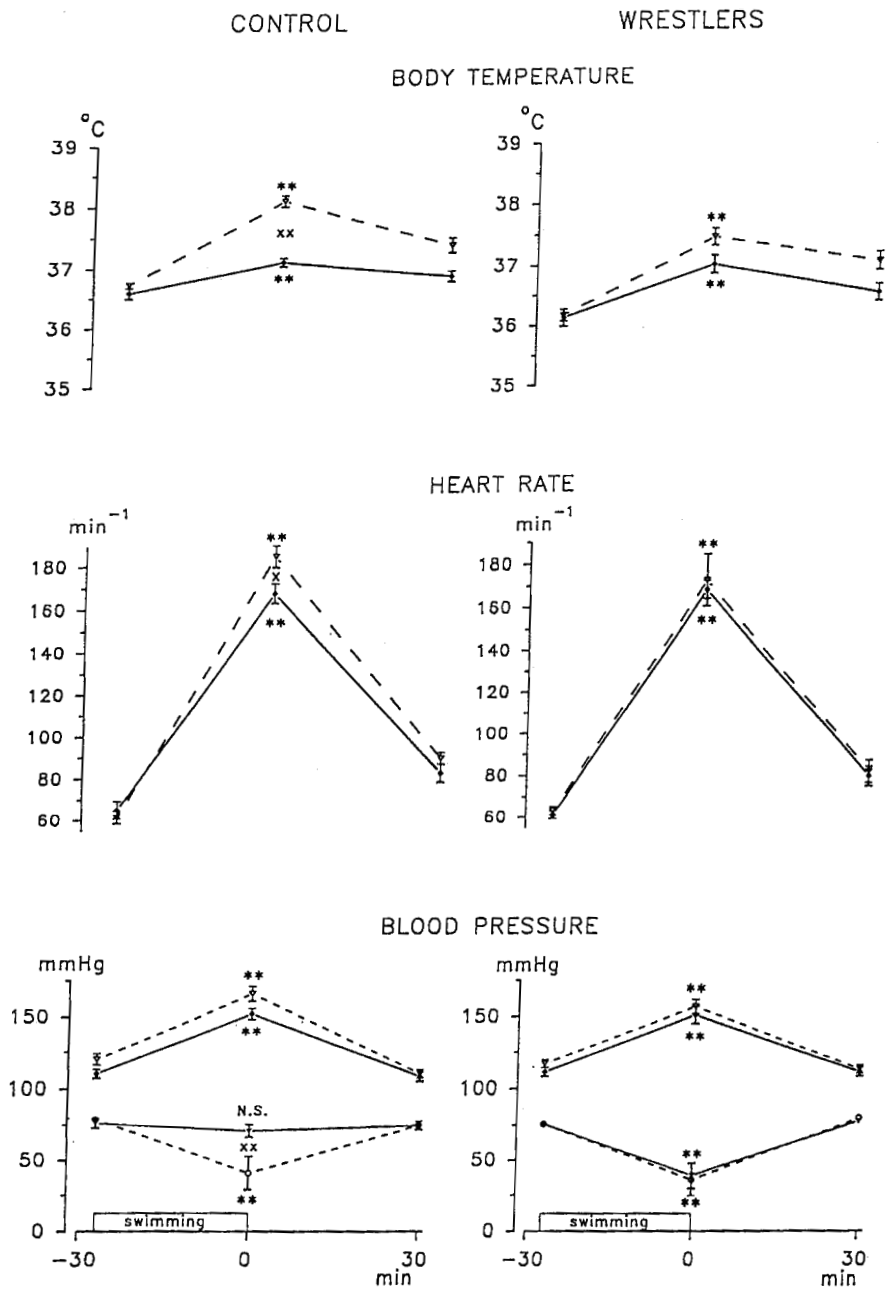

Fig 1 Body temperature, heart rate and blood pressure (mean±S.E.) before (-30 min), immediatelly after swimming (0 min) in water of 27 °C (full line) or 36 °C (interrupted line), and after the rest (+30 min) in 9 control subjects and 10 wrestlers. Statistical significance against the value at -30 min is indicated by asterisks (P<0.01) and that between the groups by crosses (´ P<0.01).**

tion of heart rate and systolic blood pressure, as well as decrease of diastolic pressure were comparable in water of 29 and 36 °C, and significant in comparison to pre-exercise values (P<0.01). There were no differences between cardiovascular responses in water of different temperatures.

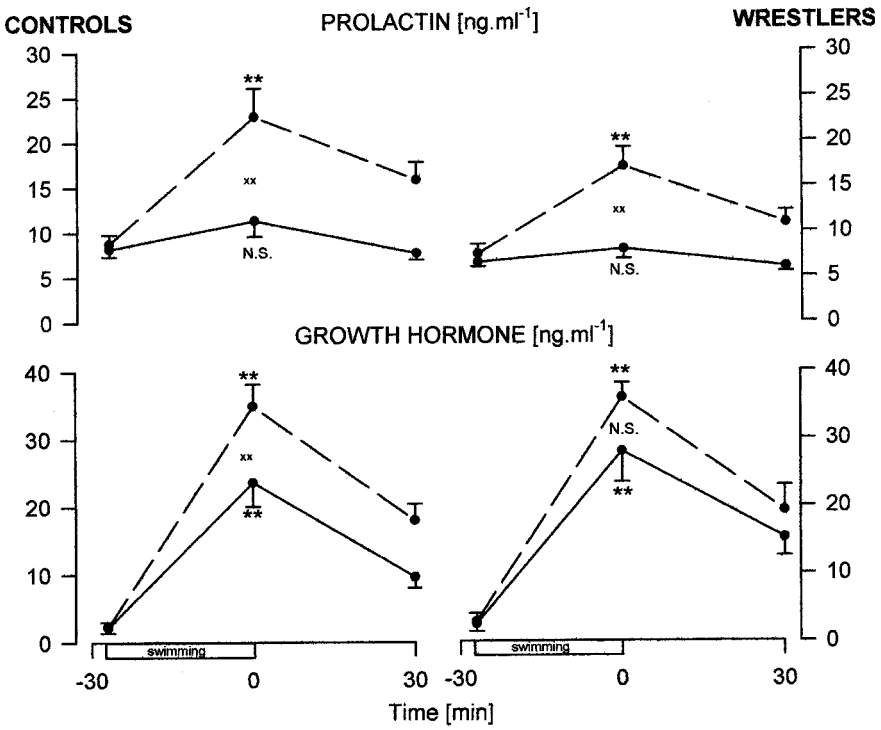

Fig 2 Plasma growth hormone and prolactin concentrations (mean S.E.) before (-30 min), immediately after swimming (0 min) in water of 27 °C (full line) or 36 °C (interrupted line), and after the rest (+30 min) in 9 control subjects and 10 wrestlers.
Statistical significance against the value at -30 min is indicated by asterisks (P<0.01) and that between the groups by crosses (x P<0.05, ´´ P<0.01).**

Plasma GH was increased after swimming in either water, though contrary to the control group, the tendency to more pronounced elevation in warmer water was not significant (Fig. 2, right panels). There was no PRL response to swimming in cooler water, while that in warmer water was significant (P<0.01). No differences between wrestlers and non-trained subjects in GH and PRL responses to swimming in water of the same temperature were observed.

Discussion

In our investigation, training of wrestlers during their preparation period for the competitive season consisted of dynamic power training, technique training, and general fitness training in which swimming was not included. Physical fitness of these professional-national rank athletes was supposed to be high (no direct measurement of $V_{O2\,MAX}$ was performed) in comparison with non-trained subjects. Swimming was chosen as a model of exercise to avoid the great advantage of athletes had they been exposed to work load for which they were trained, and to use to advantage the high thermoconductance of water allowing more effective application of different ambient temperatures. Exercise in water differs from the training of wrestlers due to the prominent arm work, water pressure, horizontal position and different heat dissipation (Viti et al. 1989). The skill of wrestlers for swimming was similar to that of non-trained subjects. Differences in individual swimming performances both in athletes and controls did not allow to use the speed or distance covered by swimming for evaluation of work output. However our previous studies (Kozlowski et al. 1983; Tatar et al. 1984) showed that during short dynamic exercise activation of the endocrine system was much more

affected by the intensity of the exercise than by its duration or the total work output.

The mechanism of exaggerated somatotrophic and lactotrophic responses during exercise in higher ambient temperature has not yet been completely elucidated. Exercise in a warm environment is followed by increase in skin blood flow. Mechanism of active cutaneous vasodilatation requires functional nitrous oxide synthase to achieve full expression (KELLOGG et al. 1998). This response may be different in athletes and non-trained subjects, as suggested by the significantly higher body temperature in controls. Increased demands on the cardiovascular system in blood supply of skin and working muscles during the exercise in hyperthermia may be one pathway for the increased activation of the endocrine system, including augmented GH and PRL release. Another possibility, supported by CHRISTENSEN et al. (1984) and BRISSON et al. (1987), would be that hyperthermia itself provides the impulse generating increased exercise-induced release of GH and PRL. These authors found no GH and PRL release during work load in cold or using an air-fan. On the other hand, a negative influence of cold on GH and PRL control cannot be excluded. In our previous studies, a clear-cut inhibition of PRL release was observed during insulin-induced hypoglycaemia at 4 °C (JEZOVA et al. 1995) and the basal secretion of GH was found to be decreased in cold (VIGAŠ et al. 1988).

The augmented concentrations in plasma GH and PRL in warmer water indicated greater stimulation during swimming in the milieu of higher temperature, however these increments were approximately equal in athletes and controls. Thus, the higher physical fitness of wrestlers failed to show a significant advantage in swimming, which requires higher movement co-ordination. Another reason for the comparability between athletes and non-trained subjects may have been the too short period of vigorous swimming which did not give athletes the opportunity to manifest their higher physical fitness. However, plasma noradrenaline and renin activity in wrestlers swimming in water of 29 °C were significantly reduced in comparison with non-trained subjects (VIGAŠ et al. 1998).

In conclusion, the results of this study indicate that the higher physical fitness in wrestlers did not result in reduced GH and PRL release during short-lasting vigorous swimming. Contrary to reduced plasma noradrenaline and renin activity after swimming in wrestlers versus non-trained subjects previously found, the secretion of GH and PRL during the exercise model used appears to be affected by body temperature rather than by physical fitness. The augmented responses of GH and PRL found in both groups at higher ambient temperature support the notion of regulatory role of body temperature in exercise-induced secretion of these hormones.

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