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TITLE

EXERCISE-INDUCED HYPERTHERMIA AND HORMONAL RESONSES TO EXERCISE

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Exercise-induced hyperthermia and hormonal responses to exercise[1]

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Abstract: Changes in plasma hormonal concentrations during exercise have been ascribed to the type, duration, and intensity of exercise, physical fitness of subjects, oxygen availability and debt, and acid–base balance. However, relatively few studies have examined the possible role of exercise-induced hyperthermia. This paper reviews previous studies on this subject and describes a series of experiments carried out in our laboratories to define the role of changes in body temperature in the release of hormones during exercise. In a first series of experiments, we studied the relationship between thermoregulatory and growth hormone responses to severe exercise at 23°C for 2 h in fit euhydrated subjects, controlling the core temperature increase to a maximum of 40°C by varying wind speed. Exponential relationships were found between increases in core temperature and plasma growth hormone, prolactin, and catecholamines during exercise, suggesting the existence of a thermal threshold for stimulation of hormonal release during exercise. The effect of endurance exercise with and without a thermal clamp (immersion in cold and warm water) on hormonal and leukocyte responses was examined. Again, a significant exponential relationship was found between increases in core temperature and hormonal responses. Thermal clamping significantly diminished the hormonal and the leukocytic responses to exercise, suggesting that an exercise-induced thermal threshold of ~38°C exists where hormonal responses are observed. Therefore, core temperature increases may be integrated in the controlling system of hormonal and leukocytic responses to exercise.

Key words: exercise, hyperthermia, body temperature, growth hormone, catecholamines, hormones.

Résumé : Les variations de la concentration plasmatique de différentes hormones au cours de l'exercice ont été attribuées au type d'exercice, à sa durée et à son intensité, à l'aptitude physique des sujets, à la disponibilité et à la dette en oxygène et à l'équilibre acido-basique plasmatique. Cependant, le rôle de l'hyperthermie d'effort a été peu étudié. Cette revue reprend les données de la littérature sur le sujet et décrit une série d'investigations réalisées dans nos laboratoires pour approfondir la relation entre l'augmentation de la température corporelle et la production hormonale pendant l'exercice. Lors d'un exercice sévère de 2 h à 23°C, l'hyperthermie étant limitée à 40°C en faisant varier les échanges thermiques convectifs, nous avons montré que les concentrations plasmatiques d'hormone de croissance, de prolactine et des catécholamines sont liées par une relation de type exponentiel à l'augmentation de la température centrale, suggérant l'existence d'un seuil thermique de stimulation de la sécrétion hormonale en cours d'effort. Ces relations ont également été observées au cours d'un exercice physique en eau chaude ou froide, celle-ci supprimant l'hyperthermie (« clampage » de la température centrale). Les réponses hormonale et leucocytaire sont atténuées en eau froide, suggérant l'existence d'un seuil de température interne avoisinant 38°C à partir duquel les réponses induites par l'exercice sont observées. En conclusion, l'augmentation de la température interne pourrait être un facteur de contrôle des réponses hormonale et leucocytaire lors de l'exercice physique.

Mots clés : exercice, environnement thermique, hormones.

Introduction

In addition to elevating the levels of blood and tissue hormones and metabolites, muscular activity increases the metabolic rate to produce heat and elevations in core temperature, which we will refer to in this paper as "exercise-induced hyperthermia" (EIH). Although many factors influence the regulation of hormonal release during exercise, such as the intensity and duration of the exercise, the physical fitness of the subjects, the oxygen debt and availability of the exercise, and changes in circulating metabolites and acidosis (Kjaer 1992; Macintyre 1987), one factor that has received relatively little attention is the accompanying EIH.

Body temperature is regulated at higher levels during exercise than at rest, and is dependent upon the work rate and metabolic rate, but is independent of environmental temperature (5–30°C) and duration of the exercise (Nielsen 1938; Berggren and Christensen 1950). Saltin and Hermansen (1966) have shown that the core temperature and probably muscle temperature responses to various levels of exercise are influenced by the relative work rate (25–75% $\dot{V}O_2$ max) but not by the absolute work rate. The relationship between

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Table 1. Exercise-induced changes in plasma hormones and core temperature in different thermal environments.

Investigator	Physical activity and ambient temperature	Core temperature and hormone response
Frewin et al. 1976	20-min run in 10°C air	T_{sl} +0.5°C, → GH
	20-min run in 40°C air	T_{sl} +1.15°C, ↑GH
Melin et al. 1988	1-h run at 50% $\dot{V}O_2$ max in 25°C air	T_{re} +2°C, ↑PRL, ↑NE
Buckler et al. 1973	20-min cycle @ 900 kpm at 4°C	T_{re} +0.92°C, ↑GH
	20-min cycle @ 900 kpm at 21°C	T_{re} +0.89°C, ↑GH
Raynaud et al. 1983	1-h cycle @ 47% $\dot{V}O_2$ max at 24°C	T_{re} +0.45°C, → GH
	1-h cycle @ 59% $\dot{V}O_2$ max at 24°C	T_{re} +0.6°C, ↑GH 10X
	1-h cycle @ 70% $\dot{V}O_2$ max at 24°C	T_{re} +0.93°C, ↑GH 20X
	1-h cycle @ 41% $\dot{V}O_2$ max at 33°C	T_{re} +0.55°C, ↑GH 40X
	1-h cycle @ 54% $\dot{V}O_2$ max at 33°C	T_{re} +0.6°C, ↑GH 60X
	1-h cycle @ 71% $\dot{V}O_2$ max at 33°C	T_{re} +1.05°C, ↑GH 80X
Christensen et al. 1984	40-min cycle @ 400 kpm at 4°C	T_{ty} +0.27°C, ↓GH
	40-min cycle @ 400 kpm at 22°C	T_{ty} +0.8°C, ↑GH
Dore et al. 1991	45-min cycle @ 70% $\dot{V}O_2$ max at 23°C	T_{re} +0.9°C, ↑GH
	45-min swim @ 70% $\dot{V}O_2$ max at 26°C	T_{re} +0.9°C, ↑GH
Kaciuba-Uscilko et al. 1992	4 × 30-min cycle @ 50% $\dot{V}O_2$ max at 22°C	T_{re} +0.96°C, ↑GH, ↑NE, ↑EPI, ↑COR, ↑Glucagon
Galbo et al. 1979	1-h swim @ 68% $\dot{V}O_2$ max at 21°C	T_{re} –0.8°C, → GH ↑NE ↑EPI
	1-h swim @ 68% $\dot{V}O_2$ max at 27°C	T_{re} +0.7°C, ↑GH ↓Insulin
	1-h swim @ 68% $\dot{V}O_2$ max at 32°C	T_{re} +1.3°C, ↑GH ↑NE ↑EPI

Note: T_{re}, rectal temperature; T_{sl}, sublingual temperature; T_{ty}, tympanic temperature; 10X, ten-fold; GH, growth hormone; NE, norepinephrine; COR, cortisol; PRL, prolactin; EPI, epinephrine.

core temperature and percentage of $\dot{V}O_2$ max is curvilinear over a range of exercise capacities up to 90% $\dot{V}O_2$ max, and the pattern of core temperature rise reflects a central controlling system rather than a failure in heat dissipation mechanisms (Davies et al. 1976). However, during exercise with a substantial combined metabolic and environmental heat load, or during periods of high humidity, overall core temperature responses are not independently governed, and may rise uncontrollably when heat loss mechanisms are impaired (Sawka and Wenger 1992).

GH responses to heat exposure

Induced hyperthermia has been used in clinical medicine, especially pediatric endocrinology, as a growth hormone (GH) stimulation test for assessing pituitary insufficiency. A positive relationship between GH secretion and increases in body temperature of 1–2°C has been shown during exposure to hot air (Okada et al. 1972), sauna baths (Weeke and Gundersen 1983), and hot water immersion (Buckler 1973). Jurcovicova et al. (1980) raised core temperature to 40°C in a hot water bath and found a positive correlation between plasma GH levels and core temperature. No increases in GH levels or core temperature occurred in an isothermic water bath at 30°C for the same time period, leading the authors to support the concept of a core temperature threshold for induction of GH responses.

Weeke and Gundersen (1983) attempted to separate peripheral thermal inputs from central thermal inputs by having subjects ingest ice while immersed at different water temperatures. During ice ingestion in a 31°C bath, core temperature decreased by ~1.0°C, remained stable in the 36°C bath, and still increased by 1.8°C in the 39°C bath. Body core cooling induced a significant suppression of GH secretion, whereas body core heating had the opposite effect. Plasma norepinephrine (NE) increased during both body core cooling and heating, with no responses in thyroid stimulating hormone, T3 and T4. In conclusion, central cooling suppressed GH secretion and increased NE secretion, whereas core heating significantly increased both GH and NE.

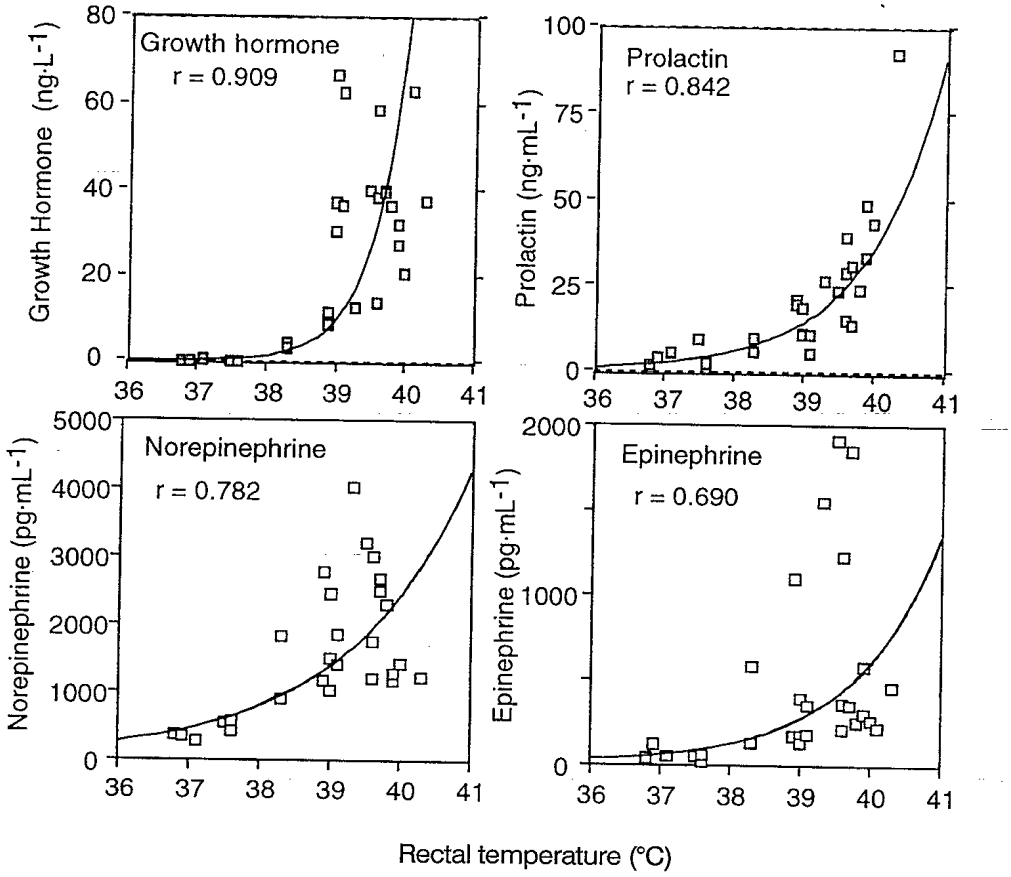
From these studies of passive body heating and cooling, it appears that core temperature plays an integral role in the regulation of various hormonal responses within the human body. However, the relationship between changes in core temperature and the hormonal responses that occur during physical exercise is not well established.

Core temperature and hormonal responses to exercise

Classically, three types of exercise (running, cycling, and swimming) have been studied to investigate the relationship between EIH and the concurrent metabolic and hormonal responses (Table 1). In all exercise types, whether performed in air or water, and at ambient temperatures ranging from 4°C to 40°C, if the core temperature increase during exercise did not exceed +0.5°C, no increases in GH were observed by the end of the exercise period (Frewin et al. 1976; Galbo et al. 1979; Raynaud et al. 1983; Christensen et al. 1984; Dore 1991). When core temperature increased by more than +0.6°C in ambient temperatures of 4°C to 40°C (Buckler 1973), however, significant increases in GH, prolactin (PRL), NE, and epinephrine (EPI) occurred by the end of the exercise.

Buckler (1973) demonstrated that the rise in rectal temperature (T_{re}) during exercise was related in timing and magnitude to the responses of circulating GH levels, and proposed that the rate of temperature change rather than the absolute core temperature was associated with the response of GH. If the rate of T_{re} increase was sufficiently slow, little

Fig. 1. Relationship between the changes in plasma growth hormone, prolactin, norepinephrine, and epinephrine levels during exercise and the corresponding rectal temperatures. All of the r values are significant at $p < 0.01$.



or no GH response would occur, but at rates of T_{re} change of 0.012°C·min^{-1}, the increase in GH levels would double. This may explain the lack of any GH response observed in exercises where the exercise intensity was too light, or short-acting, and where little or no increase in body temperature occurred.

Raynaud et al. (1983) examined exercises at three different submaximal intensities (40, 60, and 80% of the individual's $\dot{V}O_2$ max), performed at either 24°C or 33°C. Although core temperatures and GH levels at the three exercise intensities were significantly higher at 33°C than at 24°C, they discounted any relationship between GH and core temperature, attributing their findings to interindividual differences.

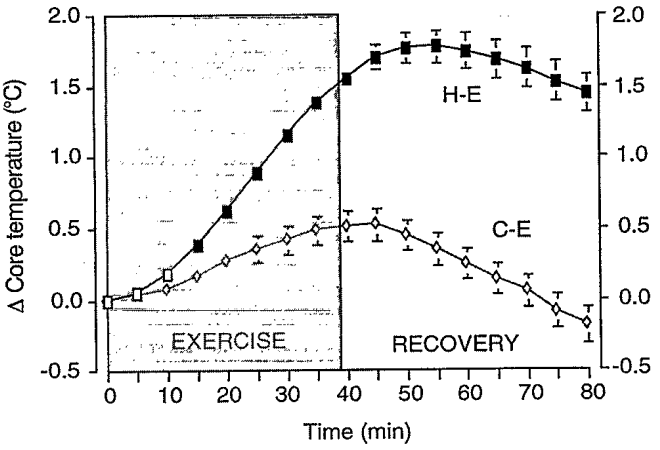
Melin et al. (1988) reported that both GH and NE increased significantly in an exponential manner as the T_{re} increased in subjects who were exercising to exhaustion at 50% of $\dot{V}O_2$ max and 35°C. They found a highly significant PRL–NE relationship, suggesting that the secretion of these hormones may be controlled by a common central noradrenergic activation.

Kaciuba-Uscilko et al. (1992) examined the metabolic, body temperature, and hormonal responses to repeated periods of prolonged cycle-ergometer exercise in a 23°C environmental chamber, finding concurrent increases in T_{re} and GH, cortisol (COR), glucagon, and catecholamines. Blood glucose and insulin decreased in consecutive exercise periods. They speculated that the changes in core temperature were the result of the release of thermogenic hormones such as GH, COR, and catecholamines, which were due, in part, to the decrease in circulating glucose levels in the exercising subjects.

Swimming represents a uniquely different thermal condition from exercise in air, as water presents a highly conductive and convective medium for heat loss. Galbo et al. (1979) investigated the effects of prolonged swimming at 68% of $\dot{V}O_2$ max for 60 min at three water temperatures, 21°C, 27°C, and 33°C. Core temperature increased during the swim in both 33°C and 27°C water (+1.3°C and +0.7°C, respectively), but decreased during exercise in the 21°C water (–0.8°C). The GH, COR, and glucagon concentrations increased in the 27°C and 33°C conditions, but failed to increase during exercise at 21°C. Insulin and glucose levels decreased throughout all three water conditions. Norepinephrine increases were higher in 33°C than in thermoneutral water at 27°C, whereas both NE and EPI increased during the 21°C exercise. This study supported the concept that the rise in core temperature during exercise enhances the exercise-induced increases in plasma levels of NE, COR, GH, and glucagon.

Unfortunately, many of the studies listed in Table 1 suffer from such methodological limitations as infrequent blood sampling, and infrequent and inadequate techniques for measurement of core temperature. In spite of these limitations, there is clear evidence that exercise-induced increases in

Fig. 2. Mean changes (± SEM) in rectal temperature during exercise and recovery in water at 38°C (H-E) and at 23°C (C-E). The solid squares for H-E represent significant differences ($p < 0.05$) from the corresponding values for C-E.



core temperature above a certain threshold initiate the secretion of various hormones into the plasma.

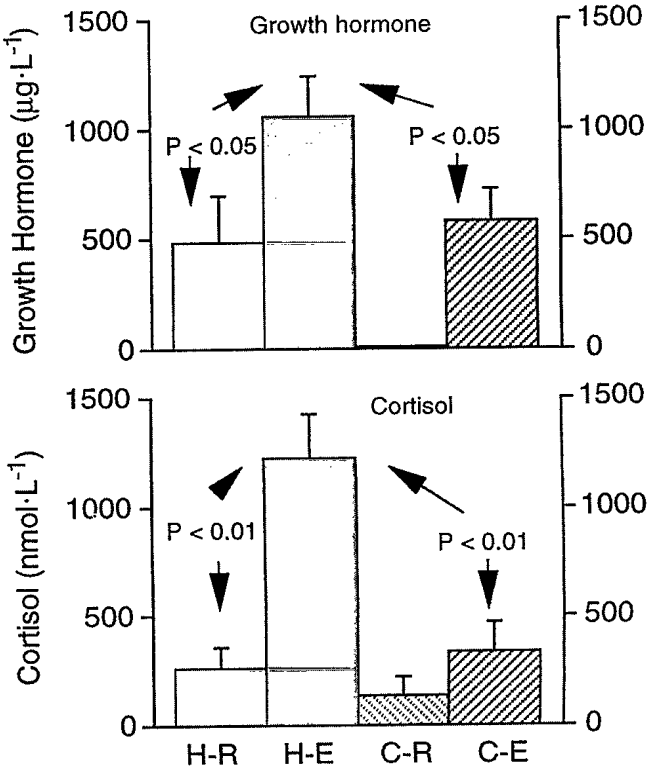
Controlled exercise-induced hyperthermia and hormone responses

To clarify further the relationship between thermal and hormonal responses to exercise, Radomski et al. (1994) designed an exercise protocol to produce a controlled increase in T_{re} up to 40°C. This was accomplished by exercising highly fit subjects at 65–70% of their $\dot{V}O_2$ max to exhaustion (~2 h) on a treadmill at an ambient temperature of 26°C. Core temperature increases were modified with convective and evaporative heat loss by increasing or decreasing wind speed and maintaining euhydration. On average, core temperature increased linearly over the first 30 min of exercise to 38.8°C at 0.050°C·min^{-1}, and at a rate of 0.013°C·min^{-1} over the last 90 min to a final T_{re} of 40°C. Growth hormone, PRL, and NE increased significantly as core temperatures exceeded 38°C in each subject, in a statistically significant exponential relationship (Fig. 1). This supported the concept of a thermal threshold in core temperature that must be exceeded during exercise to induce significant increases in certain hormones such as GH and PRL. A re-analysis of the data of Melin et al. (1988) revealed a similar exponential relationship between PRL, NE, and core temperature changes during exercise, supporting the concept of a thermal threshold for activation of these hormones. Among the various studies listed in Table 1, increases in the various hormones noted did not occur until increases in core temperature had exceeded approximately +0.6°C.

Thermal clamping of exercise-induced hyperthermia and corresponding hormonal responses

Given that the hypothalamus is the central regulator of both core temperature and hypothalamo–pituitary hormone secretion, it is realistic to hypothesize that the body thermal

Fig. 3. The total area under the curve for the growth hormone and cortisol responses for the four experimental conditions: H-R, resting in the water at 38°C; H-E, exercising in water at 38°C; C-R, resting in water at 23°C; C-E, exercising in water at 23°C.

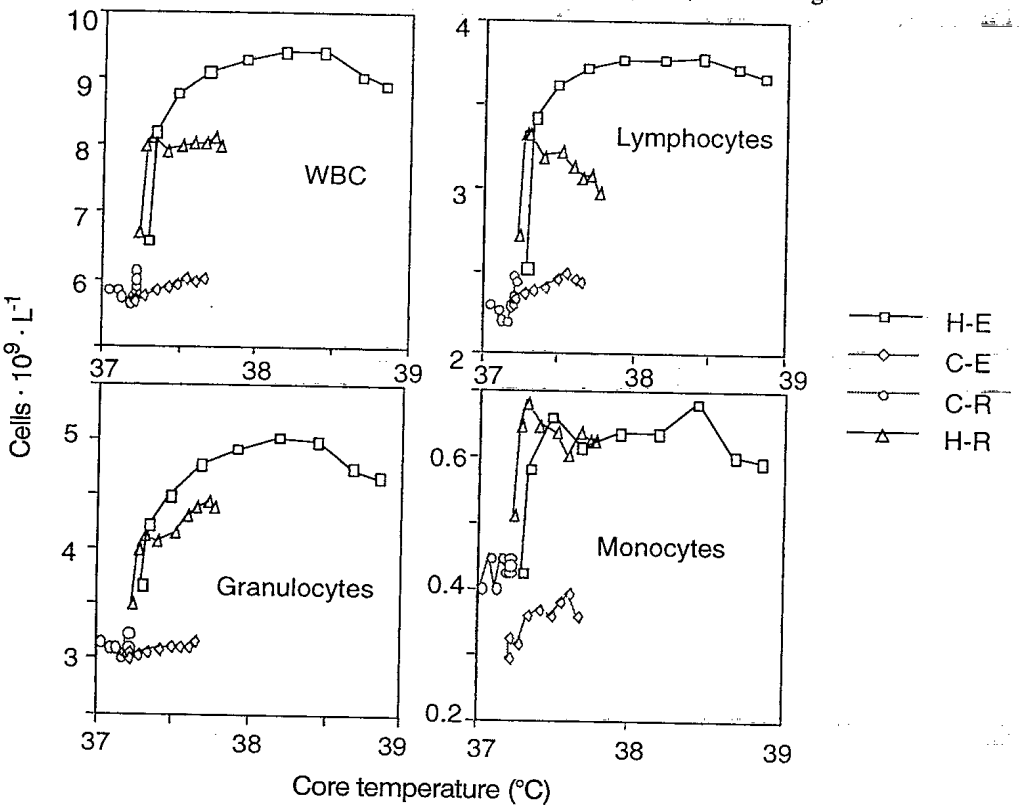


responses to exercise may be related causally to the induction of hormone release into the circulation during exercise. To examine this hypothesis further, Cross (1996) designed a study to clamp the increases in core temperature during intensive exercise and to examine hormonal responses in relationship to changes in core temperature. Subjects exercised at 65% of their $\dot{V}O_2$ max for 40 min while immersed to their mid-chest at two different water temperatures of 38°C and 23°C. Two other experimental conditions included resting in the water for the same time period. In preliminary studies, a variety of air environments (5–20°C, with varying windspeed and humidity) were also tried in air to attempt to clamp the increases in core temperature, but these measures proved ineffective.

Figure 2 shows the mean changes in T_{re} over the 40-min of exercise at the two different water temperatures. The core temperature increases observed during exercise in the hot water were significantly clamped by performing the same exercise in water at 23°C. A clamping of GH and COR (Fig. 3) responses as well as NE and EPI to exercise occurred during exercise at a water temperature of 23°C. A statistically significant exponential relationship ($p < 0.01$) was found between elevations in core temperature and GH, NE, EPI, and COR. Furthermore, a core temperature threshold for activation of hormone secretion appeared to exist in the vicinity of 38–38.5°C.

Employing this same thermal clamp technique, Cross et al. (1996) examined the effects of endurance exercise with and without a thermal clamp on leukocytes and leukocyte

Fig. 4. White cell (WBC), lymphocyte, granulocyte, and monocyte counts at the different rectal temperatures occurring during the four experimental conditions: H-E, hot-exercise; H-R, hot resting; C-E, cold exercise; C-R, cold resting.



subsets. Thermal clamping during exercise substantially reduced the rise in white cell (WBC), lymphocyte, and granulocyte counts, but not the increases in monocyte counts, observed during exercise without a thermal clamp. We replotted their changes in leukocytes and leukocyte subsets against the corresponding core temperature over the period of the exercise (Fig. 4). Exercise or resting in cold water did not induce any increase in any of the leukocyte subsets. However, immersion in hot water induced an immediate increase in all subsets with exercise inducing an additional increase in WBC, lymphocyte, and granulocyte counts, but not in monocytes. It is evident that both exercise and a rise in core temperature contribute to the changes in WBC and subset counts during the exercise. Cross et al. (1996) also found that cortisol and GH appeared to play significant roles in mediating these responses. Thus, the observed changes in leukocyte counts appear to reflect the interaction between core temperature, hormonal influences, and other effects of exercise.

In conclusion, this review has attempted to demonstrate that the increase in core temperature that occurs during exercise (EIH) plays a significant role in the induction of hormone release into the circulation. When such an EIH is modulated by clamping the increases in core temperature by performing the exercise in cold water, the release of GH, COR, NE, and EPI is significantly reduced. Presumably, if total clamping of the core temperature increase could be achieved by exercise in colder water than that employed by Cross (1996), the hormone secretion during exercise could be inhibited totally. Although increases in core temperature may not be the only factor inducing the release of hormones during exercise, there does appear to be a thermal threshold in core temperature that must be exceeded prior to the increase in the circulation of GH, COR, PRL, NE, and EPI.

A recent review of the interrelationships between heat exposure, exercise, and immune function has shown that the responses induced by hyperthermia alone are very similar to exercise-induced changes in immune function (Brenner et al. 1995). This review suggests that a substantial part of the exercise responses may actually be due to the associated elevations in core temperature. These immune changes include a leukocytosis, an increase in natural killer cell activity, either increases or decreases in mitogen-stimulated lymphocyte proliferation, and increased release of certain interleukins (IL) (IL-1, IL-2, IL-6, interferon, and tumour necrosis factor). It would be interesting to examine whether these EIH-induced changes will persist during a thermal clamp similar to that found for hormones by Cross (1996).

The relationship between the increase in body temperature related to muscular work and the increase in GH secretion may be triggered by an increase in inflammatory cytokines, such as interleukin-1 (IL-1). Interleukin-1 has been shown to enhance GH secretion (Rettori et al. 1987) and the growth hormone-releasing hormone (GHRH) has been suggested as a GH sleep-promoting mediator of IL-1 action (Krueger et al. 1995). In fact, GH secretion and sleep are related, possibly due to a common regulatory mechanism involving the stress-related secretion of GHRH, which is mainly secreted

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by the hypothalamic arcuate nucleus. This relationship holds in patients with sleeping sickness (Radomski et al. 1996), a disease that suppresses the circadian rhythmicity of sleep and wakefulness (Buguet et al. 1993). The thermal threshold described in our studies may therefore influence stress-induced sleep or wake responses after exercise (reviewed by Buguet et al. 1998), through an immune-arcuate nucleus (GHRH)–pituitary pathway, which is still relatively unknown. Other proinflammatory cytokines, such as tumor necrosis factor(TNF)-α or interferon-α (IFN-α) and IFN-β, which are known to induce fever (Blatteis 1993) and sleep (Krueger et al. 1995), may also be involved in the activation of the GHRH system.

As exercise-induced hyperthermia is an important factor limiting physical performance, and physical performance is better in colder than warmer environments, this review of the literature suggests that many of the hormonal and immune responses that occur during intense and prolonged exercise with an accompanying exercise-induced hyperthermia, such as the marathon, may be disadvantageous to performance and the health of the individual (Brenner et al. 1994). These changes may, in fact, be a reflection of a generalized stress response rather than exercise specific, and if so, may be detrimental to the individual.

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