Plasma TSH, T₃, T₄ and cortisol responses to swimming at varying water temperatures

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The acute effect of 30-min swimming at a moderate speed, at three water temperatures (20, 26 and 32°C) on plasma thyroid stimulating hormone (TSH), free thyroxine (F.T₄), triiodothyronine (T₃) and cortisol concentrations was studied in 15 elite male swimmers. Blood was sampled before and immediately after the events. The heart rate, which was continuously monitored during exercise, had the highest response at 32°C and the lowest at 20°C. Blood lactate concentrations were found to be similar after the three tests. Plasma TSH and F.T₄ were found to be significantly increased (by 90.4% and 45.7% respectively) after swimming at 20°C, decreased at 32°C (by 22.3% and 10.1% respectively) and unchanged at 26°C. Exercise at these three water temperatures did not significantly affect T₃. Finally, plasma cortisol was found to be increased after swimming at 32°C (by 82.8%) and 26°C (by 46.9%), but decreased at 20°C (by 6.1%).

Keywords: Swimming, water temperature, TSH, thyroxine, triiodothyronine, cortisol

Increased activity of the pituitary-thyroid axis, as well as the adrenal cortex, plays a major role in adaptations to physical exercise. Moreover, it has been demonstrated that changes in their secretory activity in response to exercise are not only closely correlated with muscular work intensity, but also influenced by thermal stress^{1,2}.

However, few studies have been published on hormonal regulation during swimming³⁻⁷. In this paper we report on the changes in concentration of thyroid stimulating hormone (TSH), thyroxine (T₄), triiodothyronine (T₃) and cortisol in plasma after prolonged swimming at three different water temperatures.

Materials and methods

This study included 15 healthy and proficient boy swimmers. Their anthropometric and training data are given in *Table 1*. The athletes swam front crawl for 30 min at a moderate speed (submaximal effort) in three individual test sessions at random. Each swimming session took place in a 25-m indoor

Table 1. Characteristics of swimmers

Characteristic	Mean(s.d.)
Age (years)	16.7(0.9)
Height (m)	1.75(0.06)
Weight (kg)	69.2(5.3)
Body fat (%)	13.6(2.3)
Years of training	6.8(2.9)
Weekly training (h week ⁻¹)	41.8(5.9)
1500-m record (min)	16.9(0.1)

swimming pool with the water temperature at 20°C, 26°C and 32°C. The water temperature was accurately regulated each time. All tests were carried out early in the afternoon, after the subjects had fasted for the previous 8 h. Successive tests were separated by a day's rest. Measurements were made toward the end of the training season. Heart rate was continuously recorded during exercise by Polar Vantage XL telemetric heart rate monitors. Blood samples (20 ml) were obtained from an antecubital vein after resting in a supine position for 15 min before, and immediately (less than 30 sec) after each swimming event. Plasma TSH, T₃, T₄ and cortisol were assayed by radioimmunological test kits (Sorin RIA kits), while plasma lactate was measured enzymatically through a reagent kit from Boehringer-Mannheim.

Data analysis

Data are expressed as mean(s.d.). The heart rates, which were monitored during swimming at three water temperatures, were compared using repeated measures analysis of variance (Statistical package for the Social Sciences; Chicago, Illinois, USA). The data for the other measurements were analysed by a paired *t*-test. A probability of less than 0.05 was taken as being significant.

Results

The swimming distance, speed and plasma lactate concentration of the athletes in the three randomized swimming sessions are presented in *Table 2*. The values of the first two parameters were found to be significantly decreased only in the cold water (20°C).

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Table 2. Description of the swimming sessions

Variable	Water temperature (°C)		
	20	26	32
Distance (m)	2132(108)*	2274(112)	2276(139)
Speed (m sec ⁻¹)	1.17(0.06)*	1.25(0.007)	1.25(0.08)
Lactate (mmol l ⁻¹)	4.6(2.4)	5.2(2.1)	4.2(2.3)

Values are mean(s.d.); **P* < 0.05 at 20°C in comparison with the other two groups

Table 3. Heart rates recorded during swimming at three water temperatures

Time (min)	Heart rate (beats min ⁻¹)		
	20°C	26°C	32°C
0	67.7(4.9)	66.6(7.3)	61.9(4.6)
5	108.7(4.3)	118.0(5.1)	128.1(5.7)
10	117.8(3.2)	124.0(4.8)	139.9(5.4)
15	126.2(3.6)	135.4(5.3)	141.4(6.2)
20	133.1(3.5)	135.2(6.9)	141.5(3.2)
25	131.5(3.2)	137.2(5.0)	140.4(5.1)
30	138.5(3.7)	146.4(4.1)	147.9(4.8)
After*	129.1(4.1)	132.3(7.2)	137.3(3.8)

Values are mean(s.d.); *30 s after the finish

However, the plasma lactate concentrations were not found to be significantly different after the three tests. Heart rate recordings during each test are presented in Table 3. Highest responses to 30-min swimming were noted at 32°C (from 61.9(4.6) to 147.9(4.8) beats min⁻¹) and lowest at 20°C (from 67.7(4.9) to 138.5(3.7) beats min⁻¹). A comparison of the recorded values of heart rate (Figure 1) showed that there were statistically significant differences between the events at the three water temperatures (*P* < 0.05).

As shown in Figure 2, TSH was increased after swimming at 20°C by 90.4% (from 1.15(0.42) to 2.19(0.41) µU ml⁻¹, *P* < 0.05), was unchanged at 26°C (from 1.18(0.40) to 1.19(0.40) µU ml⁻¹) and decreased at 32°C by 22.3% (from 1.12(0.38) to 0.87(0.41) µU ml⁻¹, *P* < 0.05).

Figure 1. Changes of heart rate during swimming at varying water temperatures: ---, 20°C; ----, 26°C; —, 32°C

Figure 2. Changes of plasma TSH, T₄, and cortisol in swimmers at varying temperatures: ■, before; ▨, after

Plasma F.T₄ showed similar changes. After the test at 20°C it was increased by 45.7% (from 1.16(0.19) to 1.69(0.30) ng 100 ml⁻¹, *P* < 0.05), at 26°C it was increased only by 6.1% (from 1.14(0.10) to 1.21(0.23) ng 100 ml⁻¹, not significant), while at 32°C plasma T₄ was decreased by 10.1% (from 1.19(0.20) to 1.07(0.28) ng 100 ml⁻¹, *P* < 0.05). No significant changes in the levels of T₃ in plasma were detected after competition at these water temperatures (from 123(20) to 127(26) ng 100 ml⁻¹ at 20°C, from 128(19) to 126(22) ng 100 ml⁻¹ at 26°C and from 125(24) to 127(28) ng 100 ml⁻¹ at 32°C).

There was a highly significant increase (82.8%) in plasma cortisol after the test at 32°C (from 15.60(2.90) to 28.51(6.41) µg 100 ml⁻¹, *P* < 0.01). The concentration of cortisol in plasma was also elevated by 46.9% (from 15.81(3.01) to 23.22(3.61) µg 100 ml⁻¹, *P* < 0.05) at 26°C, while it was decreased by 6.1% (from 16.10(3.23) to 15.12(2.43) µg 100 ml⁻¹, not significant) at 20°C.

Discussion

The circulatory response, as well as the work capacity, during swimming at varying temperatures, are well known^{8,9}. Our data are in agreement with

these observations. The response in the heart rate during swimming at moderate intensity seemed to be related to water temperature, since it was highest at 32°C and lowest at 20°C. These differences can be explained mainly by alterations of the peripheral sympathetic vasoconstrictor activity at these temperatures. Lower heart rates were recorded during swimming in cold water because of the vasoconstriction of the skin in the cold, whereas warm water induces the opposite response^{4,8,9}. Although the swimmers tried to make the same effort under all three conditions, they swam slower in the cold water. This fact may be due to changes in the biochemical and functional processes of the working muscles in the cold⁹. The plasma lactate concentrations after the tests were similar. However, the values seem to indicate that the swimmers exceeded the anaerobic threshold (4 mmol l⁻¹). This finding can be explained by the fact that speed is usually increased at the end of each swim.

Heat production during swimming is related to work intensity and water temperature¹⁰. It has been reported that the changes of rectal, oesophageal and muscle temperatures are closely related to water temperature during swimming¹¹. The body tends to save heat during exercise in warm water and to lose heat during the same effort in cold water¹⁰. The two major factors determining this heat flux are subcutaneous fat and thermoregulatory control^{9,10}. It has been shown that thermal stress causes increase in some hormonal responses, such as catecholamines, glucagon, growth hormone and cortisol, to prolonged muscular exercise^{2,4,7,12}. It is also well known that these hormones are essential for the regulation of many functions during effort^{2,5,13}.

The plasma TSH and F.T₄ levels were found to be significantly increased after swimming at 20°C. This effect was not observed at 26°C, while a significant decrease of these hormones was noted at 32°C. These findings are in accordance with the results of others^{3,4,12}. Furthermore, Dulac *et al.* claim that T₄ release in plasma seems to be related to the duration of the stay in cold water, since the highest concentrations were noted in the slowest swimmers¹². During exercise in cold water the secretion of TSH is triggered by peripheral and central cold receptors¹³. It has also been reported that acute cold exposure leads to an elevation of TRH levels in plasma¹³. Therefore, it should be emphasized that these hormonal responses to cold, in addition to the demonstrated increase in blood epinephrine and norepinephrine, are considered to be a direct consequence of thermoregulation during swimming^{14,15}. Moreover, O'Connell *et al.*¹⁶ support the view that the exercise-induced changes in thyroid hormone levels could also be secondary to altered blood flow, plasma binding, fluid shifts or other effects of exercise.

There was no remarkable difference in the T₃ levels before and after effort which is in agreement with previous findings^{4,12}. Others have reported that during prolonged moderate exercise rT₃ and T₄ were increased and T₃ was decreased because of alterations in the concentration of plasma free fatty acids, glucose and other fuels^{3,16,17}.

The response of plasma cortisol to prolonged swimming appears to be augmented by temperature, since a significant rise in concentration was noted after the event at 26°C and an even greater increase at 32°C. No significant change in cortisol was observed after swimming at 20°C. There are some contradictory results regarding the cortisol response pattern to swimming at varying water temperatures. Galbo *et al.*⁴ found that plasma cortisol was increased after 60 min of swimming at 26°C or 32°C, but remained unchanged at 21°C. Furthermore, Collins and Weiner claim that a decrease of body temperature suppresses either the rate of steroid synthesis or their secretion from the adrenal cortex¹. On the other hand, Dulac *et al.* found that cortisol concentrations in blood were significantly increased during long-distance swimming in cold water¹².

It has been demonstrated, that the increase in core temperature during intense exercise leads to a sharp reduction of the cortisol-binding globulin and, thus, to an acute release of free cortisol¹⁸. Moreover, many investigators have reported that cortisol levels in blood are increased only after prolonged exercise secondary to an increase in pituitary-adrenocortical activity^{1,4,19,20,21}. In accordance with this hypothesis, it has been shown, that cortisol levels are decreased during exercise below 50% of the $\dot{V}O_{2\max}$ but increased when the workload exceeds 60% of the $\dot{V}O_{2\max}$ ^{2,19,22}. These bidirectional changes can be explained by the fact that the release of the pituitary hormone, corticotropin (ACTH), which is an important stimulator of cortisol secretion, is increased only during vigorous physical stress⁶. Furthermore, many investigators have reported that during submaximal swimming in cold water the oxygen uptake was higher than in warm water, while the $\dot{V}O_{2\max}$ was lower^{4,9,10,23}. Thus, the decreased levels of cortisol can be explained partly by the low workload in cold water².

Finally, it is known that glucocorticoids suppress secretion of thyroid hormones¹³. Therefore changes in cortisol concentration are shown to be related to the alterations of plasma TSH and thyroxine during swimming at varying temperatures.

References

- Collins KJ, Weiner JS. Endocrinological aspects of exposure to high environmental temperatures. *Physiol Rev* 1968; **48**: 785–839.
- Hartley LH, Mason JW, Hogan RP *et al.* Multiple hormonal responses to prolonged exercise in relation to physical training. *J Appl Physiol* 1972; **33**: 607–10.
- Tuomisto J, Mannisto P, Lamberg BA *et al.* Effect of cold-exposure on serum thyrotrophin levels in man. *Acta Endocrinol* 1976; **83**: 522–7.
- Galbo H, Houston ME, Christensen NJ *et al.* The effect of water temperature on the hormonal response to prolonged swimming. *Acta Physiol Scand* 1979; **105**: 326–37.
- Lovoie J, Cousineau D, Peronnet F *et al.* Metabolic and hormonal responses of elite swimmers during a regular training session. *Eur J Appl Physiol* 1983; **50**: 173–7.
- Lenz T, Weib M, Werle E *et al.* Influence of exercise in water on hormonal, metabolic and adrenergic receptor changes in man. *Int J Sport Med* 1988; **9** (Suppl): 125–31.
- Viti A, Lupo C, Lodi L *et al.* Hormonal changes after supine posture, immersion and swimming. *Int J Sports Med* 1989; **10**: 402–5.

- 8 Costill DL, Cahill PJ, Eddy D. Metabolic responses to submaximal exercise in three water temperatures. *J Appl Physiol* 1967; **22**: 628–32.
- 9 Holmer I, Bergh U. Metabolic and thermal response to swimming in water at varying temperatures. *J Appl Physiol* 1974; **37**: 702–5.
- 10 Nadel ER, Holmer I, Bergh U *et al.* Energy exchanges of swimming man. *J Appl Physiol* 1974; **36**: 465–71.
- 11 Saltin B, Hermansen L. Esophageal, rectal and muscle temperature during exercise. *J Appl Physiol* 1966; **21**: 1757–62.
- 12 Dulac S, Quirion A, DeCarufel D *et al.* Metabolic and hormonal responses to long-distance swimming in cold water. *Int J Sports Med* 1987; **8**: 352–6.
- 13 Reichlin S, Martin JB, Jackson JM. Regulation of thyroid-stimulating hormone (TSH) secretion. In: Jeffcoate SL, Hutchinson JSM, eds. *The Endocrine Hypothalamus*. London, UK: Academic Press, 1978, 237–43.
- 14 Christensen N, Galbo H, Hansen J *et al.* Catecholamines and exercise. *Diabetes* 1973; **28** (Suppl): 58–62.
- 15 McArdle W, Katch F, Katch V. *Exercise Physiology*. Philadelphia, USA: Lea and Febiger, 1991: 549–50.
- 16 O'Connell M, Robbins D, Horton E *et al.* Changes in serum concentrations of 3,3',5'-Triiodothyronine and 3,5,3'-Triiodothyronine during prolonged moderate exercise. *J Clin Endocrinol Metab* 1979; **49**: 242–6.
- 17 Vagenakis A, Burger A, Portnay G *et al.* Diversion of peripheral thyronine metabolism from activating to inactivating pathways during complete fasting. *J Clin Endocrinol Metab* 1975; **41**: 191–6.
- 18 Westphal U. Steroid-protein interactions. In: Gross F, Labhart A, Mann T, Samnells L, Zander J, eds. *Monographs on Endocrinology*. New York, USA: Springer-Verlag, 1971.
- 19 Bonen A. Effects of exercise on excretion rates of urinary free cortisol. *J Appl Physiol* 1976; **40**: 155–8.
- 20 Urhausen A, Kindermann W. Behaviour of testosterone, sex hormone binding globulin and cortisol before and after a triathlon competition. *Int J Sports Med* 1987; **8**: 305–8.
- 21 Jurimae T, Viru A, Karelson K *et al.* Biochemical changes in blood during the long and short triathlon competition. *J Sport Med* 1989; **29**: 305–9.
- 22 Davies C, Few J. Effects of exercise on adrenocortical function. *J Appl Physiol* 1973; **35**: 887–91.
- 23 Holmer I. Oxygen uptake during swimming in man. *J Appl Physiol* 1972; **33**: 502–9.

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