Alterations in the morning plasma levels of hormones and the endocrine responses to bicycle exercise during prolonged strain. The significance of energy and sleep deprivation

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Abstract. The relative significance of physical exercise, energy and sleep deprivation for the morning levels of hormones and the endocrine response to short-term bicycle exercise were investigated in 24 male cadets during a 5-day military training course. Significant increases in the morning levels of noradrenaline, adrenaline, and dopamine, and a decrease in PRL were ascribed mainly to physical strain. Cortisol and hGH increased, whereas insulin and glucose decreased mainly due to energy deficiency. Pulse rate after the bicycle test was unchanged and similar in all groups in spite of increased catecholamine responses. The increased catecholamine response was mainly due to physical strain. The cortisol response to the bicycle test was increased in all groups, and energy deficiency caused slower postexercise recovery. The incremental hGH response to the exercise test was unchanged in the energy-deficient subjects but abolished in the well-fed subjects. The results suggest that the endocrine responses during long-lasting exhausting strain were mainly due to physical exertion and energy deficiency, whereas sleep deprivation did not play any major role.

Physical exercise is known to elicit a multitude of endocrine and metabolic responses appropriate for energy mobilization and cardiovascular adaptation. Thus, there is a gradual increase in the plasma levels of noradrenaline, adrenaline, dopamine and pulse rate with increasing exercise intensity (1,2). The serum levels of cortisol and growth hormone are known to increase when a certain workload is exceeded (3-6). Short-term physical exercise has variable effects on serum prolactin (7-10), and a decrease has been shown during long-term exercise (11). Serum insulin decreases during short-term exercise, and glucose is slightly increased (10,12).

Only moderate peripheral endocrine and metabolic alterations have been found during sleep deprivation (13). Cortisol, hGH and PRL have been shown to increase during night-time, but the distinction between the effect of sleep and the natural circadian rhythm is difficult. It seems, however, as if sleep as such increases the serum levels of hGH and cortisol (13,14).

In contrast to sleep deprivation, energy deficiency causes profound changes in the metabolic and endocrine state of the human body (15-17). There are alterations both in the secretion and degradation of hormones and metabolites. The action of hormones on target tissues is altered as are the energy stores. hGH and cortisol have been shown to increase during starvation, whereas the thyroid function is decreased (18,19).

Previously, considerable endocrine and metabolic alterations have been documented during a 5-day military training course with heavy physical exercise, sleep and energy deficiency (10-12,14,20,21).

Some of these results were found to be dependent on sleep and/or nutritional factors as studied in separate courses where the cadets were given either extra amounts of sleep or additional food supplies. In these courses, blood samples were drawn immediately after the sleep period, which
may have resulted in the detection of hormonal changes induced by sleep itself.

In the present investigation the effects of extra food or 3 hours additional sleep on an extended number of endocrine responses were compared in the same experiment. Immediate effects of sleep were avoided by organizing the extra sleep period 6-8 hours before blood sampling. Alterations in the morning levels of hormones during the training course were measured. In addition, one main object of the study was to determine the significance of nutrition and extra sleep for the endocrine responses to a standardized exercise test after prolonged exhausting stress.

Subjects and Methods

Training course
The subjects were 24 first year cadets of the Norwegian Military Academy who participated in a ranger training course for 5 days as a part of their school programme. The subjects were randomly divided into three groups. Group 1 consisted of 9 subjects with a mean age of 22 years (range 21-25). They were exposed to continuous physical exercise (35% of maximal oxygen uptake as a mean) representing a daily energy consumption of about 35 000-45 000 kJ. The subjects did not get any scheduled sleep during the course. By observation and heart rate recordings we found that they slept for a total of 1.3 h during the entire course. Group 2 consisted of 7 cadets with a mean age of 24 years (range 22-27) who differed from Group 1 in that they received a specially composed diet. Group 3, consisting of 8 cadets with a mean age of 25 years (range 22-25), had 3 h of sleep each night, usually between 21.00 and 03.00 h. While this group slept, the others had activities only enough to keep them awake.

Diet
The daily basic food intake for all the cadets consisted of approximately 60 g of proteins, 40 g of fat, 100 g of carbohydrates, and 1-2 g of NaCl, representing about 4000-5000 kJ/24 h. Group 2 was in addition given a specially composed diet representing about 30 000-35 000 kJ/24 h for each cadet. This diet contained 100 g of proteins, 150 g of fat, 1400 g of carbohydrates, and 20-50 g of NaCl. The extra diet was given as soup, orange juice, cocoa, and milk shake. The high carbohydrate content was achieved by adding a preparation easily soluble in water, nearly tasteless and colourless (maltodextrin). Because of this special diet the subjects of Group 2 consumed about 6 l liquid each day. The cadets were drinking water and liquid from their canteens, and reported the amount of liquid consumed. This amount was roughly the same in all groups. Because of the high energy diet, Group 2 did not have any significant loss of weight during the course. In Groups 1 and 3 with an energy deficit of 30 000-40 000 kJ/24 h, each cadet had a weight loss of about 4 kg, mostly fat.

Blood sampling
All tests and blood sampling were performed between 06.00 and 09.00 h daily during the course. All blood samples were obtained from a Veneflon® cannula in the antecubital vein, which was inserted 5-10 min before blood sampling. The amount of blood taken each time was about 40 ml. Blood for preparation of plasma was centrifuged immediately in a refrigerated centrifuge, whereas blood for the preparation of serum was allowed to clot for about 30 min, and then centrifuged in a refrigerated centrifuge. Aliquots of serum and plasma were frozen immediately on dry ice and kept frozen at -80°C.

Bicycle exercise
The maximal oxygen uptake (VO₂ max) was estimated for each cadet before the course by measuring the heart rate at three separate submaximal workloads on a bicycle ergometer. The mean value of estimated VO₂ max from the Aastrand & Rynning nomogram was used. On day 3 during the course and in a control experiment some months after the course, the cadets were tested by 15 min ergometer bicycle exercise of approximately 60% of maximal oxygen uptake (workload 100-130 W). The control experiment was performed indoors with a room temperature of about 21°C. During the course, the exercise test was performed outdoors in the training area at about 500 m altitude. The weather was fairly good with a temperature of about 20°C. Blood samples were drawn just before the bicycle exercise with the subjects sitting on their bicycles, at the end of the 15 min of exercise, and with the subjects recumbent in their sleeping bags after 15 and 25 minutes of recovery.

TRH test
The TRH test was performed on day 1 just before the start of the course and on day 4 during the course, both days between 06.00 and 08.00 h in the morning with the subjects recumbent in sleeping bags. Blood samples were taken just before and furthermore 30 and 60 min after the iv injection of 0.4 mg TRH (Hoechst, FRG).

Chemical analysis
The catecholamines were analysed with a radioenzymatic method (22). Radioimmunoassays were used for the other hormone analyses. Cortisol was determined with the GammaCoatKit from Clinical Assays, (MA, USA). hGH, insulin and prolactin were measured with kits from Immuno Nuclear Co (MN, USA). Glucose was analysed using the hexokinase method (Boehringer, FRG).
Statistics

The results are presented as means ±SEM. An analysis of variance for repeated measures was used to test alterations within the same group and between different groups (Manova, SPSS). The t-test was used to identify the significant differences and day-to-day differences. Pearson’s correlation coefficient was calculated using SPSS. Non-parametric statistics were applied to the growth hormone results, since these data did not show normal distribution; Friedman’s two-way ANOVA to test alterations within the same group and Kruskal-Wallis’ one-way ANOVA to test differences between the different groups (SPSS). Wilcoxon rank-sum and sign test were used to identify significant differences.

Results

Plasma noradrenaline levels (nmol/l) increased about 10-fold during the course, from 1.1±0.2 to 11.4±1.2, from 1.1±0.1 to 12.0±1.4, and from 0.9±0.2 to 10.5±1.2 in Groups 1, 2 and 3, respectively (F3,126=112.4, p<0.0005) (Fig. 1). Using an overall analysis of variance for repeated measures (Manova, SPSS) Group 2 was significantly (F12,126=3.17, p=0.001) different from Groups 1 and 3, which did not differ. The t-test showed significantly lower levels for Group 2 only on day 4 (T22=2.28, p=0.033). The absolute noradrenaline response to the bicycle test was increased in all subjects during the course (Fig. 2) (F3,63=8.85, p<0.0005), whereas the relative response was unchanged. The noradrenaline response during the exercise test (F3,63=83.87, p<0.0005) was not significantly different between the three groups, either in the control experiment or during the course. The noradrenaline concentrations were unaffected by TRH iv (Table 1).

Plasma adrenaline (nmol/l) (Fig. 1) increased 3-5 fold during the course from 0.33±0.04 to 1.31±0.19 in Group 1, from 0.30±0.04 to 1.31±0.27 in Group 2, and from 0.18±0.04 to 1.40±0.19 in Group 3 (F3,126=33.4, p<0.0005). There were no significant differences between the three groups. The adrenaline response to the bicycle test (F3,63=55.8, p<0.0005) was increased during the course (F3,63=3.8, p=0.014) (Fig. 2), without significant differences between the three groups. Plasma adrenaline was not influenced by TRH stimulation iv (Table 1).

Plasma dopamine concentrations (nmol/l) (Fig. 1) increased from 0.16±0.04 to 1.03±0.12 in Group 1, from 0.18±0.05 to 1.01±0.21 in Group 2, and from 0.18±0.05 to 1.29±0.15 in Group 3 (F3,126=46.83, p<0.005). Significantly lower levels were found for Group 2 compared with the others (F12,126=2.48, p=0.006) only on day 4 (T22=2.79, p=0.011). A small but significant increase (F3,63=13.18, p<0.005) was also seen during the exercise test, without significant differences between the three groups (Fig. 2). TRH stimulation iv did not affect the plasma concentrations of dopamine.

Pulse rate (beats/min) (Fig. 2) was increased in all three groups during the course (F3,63=9.79, p<0.0005), whereas the same pulse rate was reached after 15 min of exercise (F3,63=728.98, p<0.0005) both during the course and in the control experiment. There were no significant differences between the three groups.

Serum cortisol concentration (nmol/l) (Fig. 3) increased about 2-fold during the course (F6,90=13.40, p<0.0005), from 400±51 to 787±70

Fig. 1.

Alterations in the morning levels of noradrenaline, adrenaline and dopamine during a 5-day military training course. The basal (B) values were obtained two weeks before the course. Another baseline was obtained after 24 days of recovery with ordinary school activities. The subjects in Group I (N=9) were exposed to continuous physical activities combined with energy and sleep deprivation. The subjects in Group 2 (N=7) were compensated for the energy deficiency, and Group 3 (N=8) was given 3 h of scheduled sleep each night. The day-to-day variations significant at p<0.01 are shown by thick lines, and variations not significant are shown by dotted lines. The values are given as means ±SEM.
in Group 1, and from 392±36 to 850±75 in Group 3. A small but significant increase was found during the course in Group 2 (F_{6.36}=2.47, p<0.042) which had significantly lower levels throughout the course than Groups 1 and 3 (F_{12.126}=3.1, p=0.001). During the bicycle exercise test, a significant decrease was found for plasma cortisol in the control experiment (F_{6.63}=4.86, p=0.004), whereas a significant increase (F_{6.63}=10.52, p<0.0005) was found during the course in all groups (Fig. 4). In the energy-deficient groups the increase continued for 10 min into the recovery phase, and levels had not fallen to pre-exercise values even 25 min after exercise (F_{6.03}=2.36, p=0.041). In Group 2, the cortisol level decreased gradually during the recovery period and pre-exercise levels were obtained after 25 min of recovery.

**Human growth hormone** (Fig. 3) concentrations (μg/l) increased 5-10 fold during the course, from 1.7±0.4 to 9.1±2.0 in Group 1 (CS(chi square)=30.0, p<0.0005), and from 1.6±0.2 to 7.2±0.9 in Group 3 (CS=33.58, p<0.0005). A small but significant increase was also found in Group 2 (CS=15.95, p=0.014). No significant difference was found between Group 1 and 3. Group 2 had significantly lower plasma levels of growth hormone during the course than Groups 1 and 3 when comparing integrated values by Kruskal-Wallis' one-way ANOVA (CS=7.73, p=0.0209). Differences between the groups for each day were: day 2: CS=6.39, p=0.041; day 3: CS=11.03, p=0.004; day 4: CS=8.83, p=0.0121; day 5: CS=12.06, p=0.0024. In spite of higher basal levels, the incremental hGH response to the exercise test was not altered in Groups 1 and 3 during the course (Fig. 4). The response in Group 2 was completely abolished. hGH in serum was not affected by TRH iv (Table 1).

**Serum prolactin** concentrations (μg/l) (Fig. 3) decreased during the course, from 25.4±3.6 to 12.5±1.8 in Group 1, from 21.8±2.7 to 10.1±1.5 in Group 2, and from 23.0±4.0 to 13.1±2.2 in Group 3 (F_{6.63}=16.93, p<0.0005). The subjects in Group 2 had significantly lower levels than the others (F_{12.126}=2.84, p=0.002). No significant alterations were found during the bicycle test (Fig. 4). The PRL response to TRH stimulation (F_{2.16}=65.44, p<0.0005) was significantly increased during the course in Group 1 (F_{2.16}=6.45, p<0.009). No significant alterations were found in the PRL response to TRH for Groups 2 and 3 during the course (Table 1).

**Serum** insulin levels (mU/l) (Fig. 3) decreased during the course in the energy-deficient groups (F_{6.84}=18.42, p<0.0005), from 15.7±1.3 to 10.1±0.5 in Group 1, and from 16.0±1.6 to 10.5±1.0 in Group 3, whereas no significant alterations were seen in Group 2. During the exercise test (Fig. 4) the insulin levels decreased, with a subsequent increase after 10 min of recovery (F_{5.68}=36.51, p<0.0005). Pre-exercise levels were obtained after 25 min of recovery. The same response pattern was found during the course, but at

![Fig. 2.](image-url) Noradrenaline, adrenaline, dopamine, and pulse rate during bicycle exercise with 60% of VO₂ max (0-15 min) and during the following recovery (15-40 min). The bicycle experiment was performed after prolonged strain (ΔΔ) and in a control experiment (○○) some months after the course. For details, see Fig. 1.
significantly lower levels ($F_{1,20} = 17.89, p < 0.0005$). Serum insulin levels were not affected by TRH iv.

**Serum glucose** concentration (mmol/l) (Fig. 3) decreased during the course in the energy-deprived subjects ($F_{6,60} = 16.80, p < 0.0005$), from 4.2±0.3 to 3.2±0.2 in Group 1 and from 4.2±0.1 to 2.9±0.1 in Group 3, whereas no significant alterations were found in subjects receiving the high-calorie diet. During the bicycle test (Fig. 4) no significant alterations were seen in serum glucose concentrations in the control experiment or during the course in the subjects receiving the high-calorie diet. In contrast, a significant decrease was found in the energy-deficient subjects during the course ($F_{3.45} = 12.87, p < 0.0005$) (Fig. 4).

**Correlations**

During the course, significant correlations were found between the noradrenaline and adrenaline response on day 2 ($R = 0.70, p = 0.0005$), day 3 ($R = 0.49, p = 0.007$), day 4 ($R = 0.50, p = 0.007$), and day 5 ($R = 0.58, p = 0.001$). Significant correlations were also found between the alterations in nora-drenaline and dopamine on day 2 ($R = 0.47, p = 0.010$), day 4 ($R = 0.61, p = 0.001$), and day 5 ($R = 0.52, p = 0.005$), and between adrenaline and dopamine on day 4 ($R = 0.61, p = 0.001$) and day 5 ($R = 0.52, p = 0.005$).

**Discussion**

This study shows that during prolonged multifactorial strain, there is an increase in the plasma levels of catecholamines and a decrease in prolactin mainly owing to physical strain. Cortisol and growth hormone increased, and glucose and insulin decreased mainly owing to energy deficiency. The study further shows that sleep deprivation seems to play only a minor role in these alterations, when blood sampling takes place several hours after the sleep period. During a previous course, when the blood samples were drawn just after the

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sleep period, significantly higher levels were found for plasma cortisol, hGH and testosterone, indicating that sleep itself might stimulate the release of these hormones (14).

During the present course, 3 hours of sleep each night was chosen because it was the amount allowed by the Military Academy in order not to hamper the military training programme. However, even though 3 hours of sleep each night is still sleep deprivation, the most serious deterioration in behaviour, performance and clinical symptoms appears when sleep is reduced from 3 hours each night to no sleep at all. Almost all cadets totally sleep-deprived are hallucinated during the course. In contrast, hallucinations are rare in subjects allowed 3 hours of sleep each night (23). The most profound endocrine alterations owing to sleep deprivation should therefore be expected when the amount of sleep is reduced to below 3 hours/24 hours.

Previously we have shown that the catecholamine response to the bicycle exercise test was strongly increased after prolonged stress (10). The present investigation with identical workload in the control experiment and the stress experiment shows that this increased response was mainly due to preceding physical strain.

In the rat it has been shown that fasting reduces noradrenaline turnover in most tissues except the liver (17,24,25). Fasting in humans has been shown to decrease urinary excretion of noradrenaline and increase the urinary excretion of adrenaline (24).

Fig. 3.
Alterations in the morning levels of cortisol, hGH, PRL, insulin, and glucose during a 5-day military training course. For details, see Fig. 1.

Fig. 4.
Cortisol, hGH, PRL, insulin and glucose responses to the exercise test before and after prolonged strain. For details, see Figs. 1 and 2.
Conversely, refeeding increases the urinary excretion of noradrenaline and reduces the excretion of adrenaline. These results indicate a suppression of sympathetic activity and stimulation of the adrenal medullary activity during fasting. On the other hand, others have shown that fasting increases the basal plasma levels of noradrenaline but does not change the basal plasma levels of adrenaline, whereas the noradrenaline and adrenaline response to bicycle exercise both are increased (8,26). On this background it is surprising that there is no significant difference between the energy-deficient and the well-fed subjects during the present course. A possible explanation is that all the subjects were in a state of starvation after 6-8 hours without food, and undergoing strenuous activities. Another explanation is a metabolic adaptation to prolonged physical strain. The increased catecholamine levels during the course and the increased response to the exercise test in all groups show that the effect of prolonged strenuous exercise dominates over the effect of energy deficiency.

Even if plasma noradrenaline, adrenaline and dopamine often respond to the same environmental factors, exercise mostly induces noradrenaline secretion, whereas hypoglycemia elicits adrenaline release. The role for dopamine is not so well established. The significant correlation between the different catecholamines indicates a 20-30% common activation of the three catecholamines during prolonged physical stress.

It has been shown that plasma catecholamines are increased in salt-deficient subjects (27,28). The subjects in Groups 1 and 3 got only 1-2 g of salt each day, whereas the subjects in Group 2 had 20-30 g of salt each day in their diets. It is therefore noteworthy that this difference in salt intake was not reflected in the plasma catecholamine levels.

In spite of the increased catecholamine response to the bicycle test there was no corresponding increase in the pulse rate response, indicating an adrenergic desensitization. This desensitization during the course may be due to a downregulation of the adrenergic receptors. Other explanations might be that the plasma levels of noradrenaline do not reflect the release of noradrenaline in the sympathetic nerve terminals in the heart, or that there is a concomitant activation of the parasympathetic nervous system, which counterbalances the increased sympathetic activation.

In accordance with previous investigations (5,6,10), cortisol showed a slight decrease during 15 min of bicycle exercise with 60% of VO2 max in the control experiment. Cortisol is known to promote gluconeogenesis, and the increased response to the bicycle test during the course may indicate that the subjects activated the gluconeogenesis earlier during the course than in the control experiment. Even in subjects in approximate energy balance the plasma cortisol increased during the exercise test after prolonged strain. The main consequence of energy deficiency on the cortisol level is the slower postexercise recovery.

Growth hormone is known to increase during exercise, and has glucose-sparing effects (10,29,30). The incremental hGH response to short-term bicycle exercise was, however, not increased during the course despite increased basal levels. The abolished hGH response to the bicycle test in the subjects receiving the high-energy diet is in accordance with results by others (31-33) who have shown that the hGH response to exercise is reduced by prior glucose injection, whereas the cortisol response is less affected.

The decrease in serum prolactin during the course was mainly due to physical exercise with a very small contribution from energy deficiency, whereas sleep deprivation did not have any significant influence. PRL did not change during short-term bicycle exercise in contrast to the findings by Sowers et al. (7) and Galbo et al. (31).

The fact that the PRL response to TRH was slightly increased during the course in the subjects in Group 1, and not in those in Groups 2 and 3, indicates a small effect of sleep and energy deprivation on the PRL response to TRH. This is in accordance with observations by Carlson et al. (34), but in contrast to the blunted PRL response to TRH after 36 hours of fasting found by Vinik et al. (35).

Other hormones investigated were not influenced by TRH stimulation. The increased noradrenaline concentrations found by Morley et al. (36) may be explained by the sensitivity of plasma catecholamines to all sorts of environmental and experimental conditions.

As expected, plasma glucose decreased during the course, and during the bicycle exercise test only in the energy-deficient subjects. In the control experiment, insulin decreased during the exercise test followed by an increase 10 min post exercise (10). The same response pattern was observed during the course as in the control experiment, although at significantly lower levels.
In conclusion, this investigation shows that the large endocrine and metabolic alterations found during prolonged strain combined with sleep and energy deficiency were due to a combination of physical exercise and energy deficiency, whereas sleep deprivation was of only minor significance.

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