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Sleep Deprivation and the Effect on Exercise Performance[1]

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Summary

Sleep deprivation or partial sleep loss are common in work conditions as rotating shifts and prolonged work hours, in sustained military operations and in athletes competing in events after crossing several time zones or engaged in ultramarathon or triathlon events. Although it is well established that sleep loss has negative effects on mental performance, its effects on physical performance are equivocal. This review examines the latter question in light of recent studies published on this problem.

Sleep deprivation of 30 to 72 hours does not affect cardiovascular and respiratory responses to exercise of varying intensity, or the aerobic and anaerobic performance capability of individuals. Muscle strength and electromechanical responses are also not affected. Time to exhaustion, however, is decreased by sleep deprivation. Although ratings of perceived exertion always increased during exercise in sleep-deprived (30 to 60 hours) subjects compared with normal sleep, this is not a reliable assessment of a subject's ability to perform physical work as the ratings of perceived exertion are dissociated from any cardiovascular changes in sleep deprivation.

Examination of the various hormonal and metabolic parameters which have been measured in the studies reviewed reveals that the major metabolic perturbations accompanying sleep deprivation in humans are an increase in insulin resistance and a decrease in glucose tolerance. This may explain the reduction in observed time to exhaustion in sleep-deprived subjects. The role of growth hormone in mediating altered carbohydrate metabolism may be of particular relevance as to how sleep deprivation alters the supply of energy substrate to the muscle.

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Whereas sleep in humans appears essential for normal functioning, sleep deprivation or sleep loss is known to adversely influence certain aspects of psychological and/or physiological performance. Sleep deprivation or partial sleep loss can occur in many fields of human endeavour. It is often encountered in work conditions that require shift work or prolonged work hours, sustained military operations, and in athletes travelling across time zones or engaged in an ultramarathon or triathlon type of competition.

The effects of sleep and its function on the organism are still unresolved and are vigorously debated (Adam & Oswald 1983; Horne 1983, 1985). Sleep can be divided into 5 distinct stages. Stages 1 through 4 constitute non-rapid eye movement sleep and are usually followed by a rapid eye movement (REM) stage. Stages 3 and 4, characterised by waves which have a high amplitude and a slow frequency, are often referred to as δ or slow wave sleep (Rechtschaffen & Kales 1968). The REM sleep stage usually follows stage 4 sleep. REM and non-REM sleep repeat in cycles of approximately 90 minutes throughout the night, depending on variables such as age and amount of prior sleep.

It is known that secretion of growth hormone increases at night during sleep, independent of plasma glucose levels, and is dependent on sleep onset (Takahashi et al. 1981; Weitzman 1976; Weitzman et al. 1974). Specifically, peaks in growth hormone secretion occur during slow wave sleep (Sassin et al. 1969a,b; Takahashi et al. 1981). When a subject is sleep deprived, growth hormone secretion is diminished (Takahashi et al. 1981). It is tempting to speculate that, in view of the sleep-dependent growth hormone secretion, slow wave sleep plays an important role in body protein restoration. The occurrence of growth hormone secretion during slow wave sleep is, in part, the basis for the restorative theories of sleep, which suggest an anabolic recuperation during sleep to counter the catabolic processes that have occurred during the day (Adam & Oswald 1983). On the other hand, it has been also postulated (Horne 1983) that the restorative role of sleep does not occur at peripheral tissue sites as the muscle, but rather in the brain. Unfortunately, there is no conclusive evidence to support a theory of increased protein synthesis during sleep, especially when one considers that subjects are essentially in a fasting state while asleep.

Anyone who has stayed awake for more than 24 hours can, from personal experience, identify several subjective effects of sleep deprivation, such as increased sleepiness, mood changes, decreased vigilance and decreased accuracy in performance of cognitive tasks. There is now broad agreement among researchers that as little as 20 hours sleep deprivation negatively affects various parameters of mental performance (Bugge et al. 1979; Haslam 1982; Ryman et al. 1985; Vidacek et al. 1986). However, the effects of sleep loss on subsequent physical performance and accompanying physical changes are still equivocal (Buguet & Lonsdorfer 1987; Martin 1981; Martin & Gaddis 1981). These are re-examined below in light of recent studies published on the subject.

1. Problems of Physical Performance Measurements During Sleep Loss

Published studies of the effects of sleep deprivation on physical performance are contradictory and often not comparable (Martin & Gaddis 1981; Opstad et al. 1980; Plyley et al. 1987). One of the reasons for this is the difficulty in reproducing the same experimental conditions between various laboratories. This problem is inherent to the facilities available in individual laboratories. For example, are subjects isolated during a sleep deprivation experiment from other people that work in the same building or are they allowed to interact with the outside world? If so, how do these interactions influence the overall psychological stimulation of the subjects in terms of novelty and anticipation and how does this affect the subjects' secretion of stress hormones such as cortisol, catecholamines and growth hormone? Another potential problem exists in the interaction of the subjects

within the sleep-deprived group itself and also their interaction with the investigators. This is especially important during determination of physical performance, where the subjects are asked to produce a maximal effort, be it handgrip, $\dot{V}O_{2max}$ or anaerobic power output. In all of these measurements, mental stimulation plays an important role. Another problem in a comparison of sleep deprivation effects on physical performance arises from the various periods of sleep deprivation studied. These may vary anywhere between 24 and 240 hours. It appears that sleep deprivation has an additive 'dose'-response effect on mental performance (Angus et al. 1985), but little or no changes have been observed in various physical parameters (Horne & Pettitt 1984; Symons 1988). Thus, the length of sleep deprivation is critical when examining its effect on mental and physical performance. A corollary to this problem is the amount of supervision exercised over the subjects (or lack thereof) and the ability of investigators to prevent any occurrences of even very short naps. Another problem in comparing sleep deprivation studies is in their varying experimental designs. Almost every study employs a different experimental protocol. In some studies, the subjects remain sedentary except for their physical performance testing, while in other studies subjects are continuously active. In the latter the amount and intensity of the activity differs from one study to another.

Clearly, a well-controlled experimental environment must be provided for the subjects in order to study the response of stress hormones without the introduction of confounding variables into the experimental design which may also modify the effects of sleep deprivation, either adversely or beneficially. Therefore, it is very difficult to compare the results from one experiment to another in any meaningful manner.

2. Perceived Exertion During Exercise After Sleep Deprivation

Several investigators have studied the effects of sleep deprivation on the ratings of perceived exertion using the Borg scale (Borg 1970). In 3 different sleep deprivation and exercise protocols, 60 hours of sleep deprivation appeared to affect ratings of perceived exertion only during exercise bouts lasting at least 8 minutes (Myles 1985). Perceived exertion and fatigue were not affected by exercise bouts of short (30 seconds) duration during sleep deprivation.

In another study (Martin & Haney 1982), subjects after 30 hours of sleep deprivation, although rating themselves as more fatigued and sleepy, nevertheless still selected virtually identical work loads to induce the same level of perceived exertion as prior to sleep deprivation. Moreover, no changes in $\dot{V}O_2$ and $\dot{V}CO_2$ during exercise were detected and the heart rate was actually lower during the exercise after sleep deprivation. Subjects in this study adjusted the treadmill elevation themselves to provide an appropriate work load to correspond to a 'very hard' perceived exertion rating on the Borg scale. Changes in mood and sleepiness could not be linked to perceived exertion during an exercise bout after 30 hours of sleep deprivation. Thus, it appears that the increased sleepiness, fatigue, lower vigour and lower scores on profiles of mood states brought about by sleep loss do not affect the changes in perceived exertion.

Similarly, after 30 hours of sleep loss (Martin & Gaddis 1981) the ratings of perceived exertion were not significantly increased after light (25% $\dot{V}O_{2max}$) exercise lasting 8 minutes, but increased significantly during a moderate (50% $\dot{V}O_{2max}$) and heavy (75% $\dot{V}O_{2max}$) exercise of the same duration as the light exercise. Yet, no significant physiological differences (heart rate, $\dot{V}O_2$, $\dot{V}_E$, $\dot{V}CO_2$ and mean blood pressure) were detected at any of the 3 intensities of exercise between 0 hours and 30 hours of sleep deprivation. It was postulated that the increased ratings of perceived exertion after moderate and heavy exercise were possibly a self-fulfillment of the subjects' expectations rather than an expression of the usual physiological indices, and that this increased rating during the moderate and heavy exercise may be one of the limiting factors of submaximal exercise tolerance in sleep-deprived subjects.

An increase in the rating of perceived exertion

in sleep-deprived subjects has been observed during a walk of 1 hour on a treadmill at 28% $\dot{V}O_{2max}$ (Plyley et al. 1987). These subjects exercised one out of every 3 hours during the sleep loss period. The rating of perceived exertion was significantly elevated after only 15 hours of sleep loss and this trend continued with significant increases every 15 hours to the end of the experiment at 60 hours of sleep deprivation. As in the study of Martin and Gaddis (1981), an increased rating of perceived exertion was observed in face of an essentially stable heart rate, supporting a possible dissociation between normally observed rating changes and increases in heart rate (Borg 1970) and suggesting that subjects may unwittingly fulfil their expectations of what the sleep deprivation effects on physical performance are supposed to be. Interestingly, significant changes in the rating of perceived exertion were seen during light exercise (28% of $\dot{V}O_{2max}$) in contrast to the exercise of 25% $\dot{V}O_{2max}$ in the Martin anad Gaddis (1981) experiment. However, Plyley et al. (1987) performed their rating of perceived exercise measurements after 30 minutes of walking. It seems that not only the intensity of exercise but also its duration probably affect the rating measurement in sleep-deprived subjects. In conclusion, it appears that the rating of perceived exertion is not always a reliable measurement of a subject's performance during sleep deprivation because of increases in the rating which occur despite stable heart rates. Thus, the changes in the rating of perceived exertion may not reflect the physiological ability of sleep-deprived subjects to perform physical work.

3. Core Body Temperature During Sleep Deprivation

Thermoregulation is affected by sleep deprivation. Both hand and core temperature decreased in subjects deprived of sleep for 123 hours (Ax & Luby 1961), suggesting a peripheral vasoconstriction in order to conserve body heat and prevent any further cooling of the sleep-deprived subjects. During 40 hours of sleep deprivation, oral temperatures have been reported to follow a normal circadian pattern with a peak in the late afternoon (1500 to 1900 hours) and a trough in the early morning (0200 to 0700 hours) [Lubin et al. 1976]. Although sleep deprivation did not alter the circadian fluctuation of oral temperature, sleep deprivation and exercise produced the largest decline in oral temperature. Bedrest and sleep deprivation produced a somewhat smaller decline, but this decrease was still significantly lower than the one observed in the condition which included regular naps. Similar results were found in both normal and insomniac elderly subjects (mean age = 62 and 65 years, respectively) during 64 hours of sleep deprivation (Bonnet 1985). The decline in nocturnal oral temperature during sleep deprivation was significantly lower than in the baseline condition.

Similar patterns have been found in sedentary subjects deprived of sleep for 60 hours (Horne et al. 1983) and 72 hours (Horne & Pettitt 1985). Oral temperature decreased during sleep deprivation, but maintained its circadian rhythm. Subjects were deprived of sleep for approximately 72 hours and oral temperature was measured every 4 hours. Following a recovery sleep, the oral temperature returned back to the baseline levels (Horne & Pettitt 1985).

A study of the effects of 50 hours of sleep deprivation on subjects exercising in thermal comfort at an ambient temperature of 0°C showed that resting rectal temperature was significantly lower in the sleep-deprived condition than in the control condition (Kolka et al. 1984). On the other hand, rectal temperature increased significantly faster in the first 15 minutes of exercise in the sleep-deprived condition than in the control condition. No significant differences in rectal temperature during the exercise were observed after 15 minutes of exercise. Finally, there were no significant differences between the 2 conditions in the metabolic rates and heart rates during the exercise or on the chosen work intensity and duration of the exercise. It seems that the limiting factor in the exercise termination was the temperature discomfort rather than fatigue. These results are in agreement with the studies of Horne and Pettitt (1984) in terms of core temperature, heart rate and the work intensity selection.

It seems that core temperature (expressed as oral temperature) in sleep-deprived subjects maintains its circadian variation until at least 130 hours of sleep loss. The only effect of sleep deprivation may be an overall lowering of the mean daily oral temperature. This could possibly explain an anecdotal observation that sleep-deprived subjects often complain of feeling cold even though the room temperatures are maintained at levels they would usually find 'comfortable'. The mechanism for this apparent decline in core temperature is not known. It is, therefore, difficult to estimate the possible effects of this factor on physical performance, since on one hand a decreased core temperature may be beneficial in delaying a heat stress that accompanies a prolonged exercise, but on the other hand lower core temperatures may reflect a decreased rate of metabolism which could adversely affect physiological performance.

4. Cardiorespiratory Response to Exercise After Sleep Deprivation

The effects of sleep deprivation on heart rate and respiratory rate in sedentary subjects have been investigated in several studies. No significant changes in heart rate and respiratory rate were found after 62 hours of sleep deprivation (Horne 1977). However, significant increases in variations of both the heart and respiratory rates occurred after sleep deprivation. Significant correlations were found between the heart rate and respiratory rate variation scores suggesting that sleep deprivation influences the heart and respiratory rate variations and these are perhaps a more sensitive means of measurement of cardiorespiratory responses to sleep deprivation.

In another study, 36 hours of sleep deprivation reduced the subjects' ability to perform a treadmill walk to exhaustion at 80% of their $\dot{V}O_{2max}$ (Martin 1981). Sleep deprivation also decreased significantly the time to exhaustion by 11%, whereas oxygen uptake ($\dot{V}O_2$), carbon dioxide production ($\dot{V}O_2$), ventilatory rate ($\dot{V}_E$), heart rate and the respiratory exchange ratio remained unaltered after 36 hours of sleep loss.

An important finding in this study was a high variability in each subject in the time to exhaustion. Two subjects actually increased their time to exhaustion after the sleep deprivation, while 4 subjects showed relatively little change (less than 5%). Another 4 subjects showed a large (at least 15%) decline in their tolerance to exercise to exhaustion. Thus, sleep deprivation of 36 hours decreased the ability of subjects to work until exhaustion, but this decline was apparently dependent on some as yet unspecified factors that produced the observed intrasubject variability. This decreased ability was accompanied by increased ratings of perceived exertion in the absence of changes in heart rate or metabolic rate. Thus, there appears to be a physiological difference between the responses of 'resistant' and 'susceptible' subjects to the effects of sleep loss.

Martin and Gaddis (1981) measured various physiological parameters during exercise at 25%, 50% and 75% of $\dot{V}O_{2max}$ in subjects before and after 30 hours of sleep loss and after 1 night of unlimited recovery sleep. No significant changes were found in oxygen uptake, carbon dioxide production, minute ventilation, heart rate, mean blood pressure or maximal oxygen uptake after sleep loss. However, the peak exercise heart rate during the maximal test was significantly lower in the sleep-deprived subjects. This suggests some limited shift in heart stimulation from sympathetic dominance to parasympathetic control to explain the observed bradycardia during the exercise after sleep deprivation.

72 hours of total sleep deprivation in untrained subjects exercising at 40%, 60% and 80% of $\dot{V}O_{2max}$ had no significant effect on heart rate, $\dot{V}O_2$, $\dot{V}CO_2$, and the respiratory quotient, except for an increase in gross mechanical efficiency compared with non-deprived subjects (Horne & Pettitt 1984). The observed increases in gross mechanical efficiency after sleep deprivation were not due to a procedural or measurement error, or a training effect.

Similarly, 24 hours of sleep deprivation had no influence on the exercising and recovery heart rate during a 20-minute bout of exercise (McMurray & Brown 1984). Although ventilation rate and oxy-

gen uptake were also not significantly different between control and sleep-deprived subjects, the respiratory quotient and postexercise glucose levels were significantly higher during exercise after sleep deprivation. In this study, the major effect of 24 hours of sleep deprivation was on the postexercise recovery parameters.

In order to assess in part whether physical activity during a sleep loss period may ameliorate the effects of 60 hours of sleep deprivation on mental performance (Angus et al. 1985), subjects walked on the treadmill at 25 to 30% of their $\dot{V}O_{2max}$ for 1 hour out of every 3 hours during sleep deprivation. No significant differences were found between the exercise and sedentary groups in fatigue, mood and sleepiness ratings, heart rate, respiratory volume or oxygen consumption over the sleep-deprivation period showing that exercise during sleep deprivation did not improve or worsen the fatigue, mood or sleepiness perceptions of subjects. Sleep deprivation for 36 hours had no effect on oxygen uptake, minute ventilation, heart rate or rectal temperature during mild exercise (30% $\dot{V}O_{2max}$) for 3 hours or heavy exercise (65% $\dot{V}O_{2max}$) for 30 minutes, although ratings of sleepiness, mood and fatigue were all significantly higher in the sleep-deprived subjects (Martin et al. 1986).

Cardiorespiratory functions have been studied in subjects exercising 1 hour out of every 3 on a treadmill at approximately 28% of their $\dot{V}O_{2max}$ during 64 hours of sleep deprivation (Plyley et al. 1987). Sleep deprivation significantly decreased the $\dot{V}O_{2max}$ by 3.8 ml/kg/min and minute ventilation ($\dot{V}_E$) in both the exercise and the sedentary conditions. Maximal heart rate was significantly lower after sleep deprivation in the exercise trial but remained similar to the presleep deprivation values during the sedentary trial. Significant increases of plasma volume were also reported in both the exercise and sedentary trials. The decrease in $\dot{V}O_{2max}$ was not attributed to a decline in the subjects' motivation as the majority of the subjects reportedly reached an oxygen plateau. The decrease in $\dot{V}O_{2max}$ may have been due to the apparent haemodilution, since a decrease in oxygen carrying capacity of the blood can be a major factor in the decline of maximal oxygen intake. Time of sleep deprivation may also be a factor as most studies reporting no change involved 30 hours of sleep deprivation.

5. Other Parameters of Physical Performance

The effects of sleep deprivation on various other physical performance parameters have been examined. A sleep deprivation of 64 hours had no effect on the times for the 40-metre dash, or isometric strength and balance (Takeuchi et al. 1985), but significant decreases were found in the height of the vertical jump and isokinetic strength during the knee extension at 60°/sec. Furthermore, sleep deprivation combined with exercise (1 hour of walking out of every 3 hours) also decreased the knee extension torque at 60 and 180°/sec. It appears that sleep deprivation results in a decrement of explosive power and isokinetic strength. The reason for this decrease may be attributed to diminished motivation and arousal of the subjects.

Partial sleep deprivation (2.5 hours of sleep every night over 3 nights) [Reilly & Deykin 1983] did not affect hand grip, the broad jump, ratings of perceived exercise during the treadmill run, lung functin measures (vital capacity and forced expiratory volume) and endurance capacity. However, anaerobic power decreased as well as the 2-choice visual reaction time. Thus, partial sleep deprivation does not affect gross motor functions which include muscular strength, lung power and treadmill endurance running. On the other hand, decrements in psychomotor functions are altered after only the first night.

Recently, the effects of 60 hours of sleep deprivation and exposure to prolonged physical activity have been extensively examined in subjects exercising on average 5 hours every day for 3 days with a normal night's sleep and during sleep deprivation (Symons et al. 1988a,b). The following physical tests were not affected by sleep deprivation: blood lactate response to 20 minutes of cycle exercise at 70% of $\dot{V}O_{2max}$; maximum isometric strength in forearm flexors and leg extensors; muscular endurance; heart rate, heart rate recovery, or

rating of perceived exertion during 4 bouts of intermittent cycle exercise at 80% of $\dot{V}O_{2max}$; total reaction time (defined as the time between light stimulus and an increase in force generation over 5N upon a force transducer by the subject); peak power output; mean power output; fatigue index; blood lactate during the Wingate anaerobic test; maximal voluntary contraction in both the arm and the leg; the rate of force development; the electromechanical response times expressed as premotor time (the time from the light stimulus to the onset of EMG activity); electromechanical delay (the time between the onset of EMG activity); maximal voluntary contraction (MVC); the rate of force development and the times required to produce 25%, 50%, 75% and 100% of MVC; and the electromechanical response times. Thus, sleep deprivation of at least 60 hours does not impair the capability for aerobic or anaerobic physical performance, or electromechanical response times and muscle strength.

6. Sleep Deprivation and Hormonal Response

Sleep deprivation of more than 24 hours has long been thought to produce a stress in human subjects. In the past 10 years or so, it has become evident from studies of the effects of sleep loss on plasma levels of various stress hormones, such as adrenocorticotrophic hormone (ACTH), growth hormone (GH), prolactin, thyroid-stimulating hormone (TSH), triiodothyronine (T3), cortisol and catecholamines, that sleep deprivation is not the classic type of stress (Selye 1955).

The majority of the studies, which have described plasma profiles of the above-mentioned hormones throughout the sleep-deprivation period, or before and after sleep loss, have proven to be either inconclusive or contradictory. Some studies have reported increased catecholamines, ACTH and cortisol levels, while others have not detected any significant changes in hormone secretions. Thus, at the present time, there is no agreement between research groups on the basic question as to whether sleep deprivation produces a recognisable stress in humans.

Cortisol, an important glucocorticoid, has been implicated in its response to various kinds of specific and nonspecific stress (Davies & Few 1973; Mason 1975), and the pattern of cortisol secretion during sleep deprivation has been investigated in great detail (Murawski & Crabbe 1960). However, plasma and urinary levels of cortisol and 17-hydroxycorticosteroid do not change during sleep deprivation of up to 80 hours when compared with the baseline state (Kollar et al. 1966; Martin et al. 1986; Rubin et al. 1969). Furthermore, it appears that cortisol maintains its secretory rhythmicity even during sleep deprivation (Hellman et al. 1970). More recently, under a closely standardised sleep-deprivation period experiment of 60 hours with multiple sampling significant increases in cortisol secretion were found as sleep deprivation progressed, although the secretory rhythmicity of cortisol secretion was maintained (Hart 1983).

Whereas increased cortisol levels are a relatively chronic response to a long-lasting stimulus, high catecholamine levels reflect an immediate sympathetic system response to a short-acting stress such as a high intensity exercise (Euler & Hellner 1952; Jezova et al. 1985). Some authors (Fiorica et al. 1968; Froberg et al. 1975) have found that catecholamine levels do not change during sleep deprivation, whereas others (Kuhn et al. 1973; Opstad et al. 1980) have reported increased levels of catecholamines after sleep deprivation. Since the subjects in Opstad's study were military cadets observed during a Rangers course and the subjects in the Kuhn study were kept awake by '... simple physical activity...', the discrepancy between the cited studies may possibly be due to the different levels of physical activity of the subjects while they were sleep deprived. It would appear that sleep deprivation along with physical activity results in elevated catecholamine levels, suggesting that either the physical activity alone or a synergistic action between these 2 conditions produces a stressful environment for the subjects.

The effects of energy deficit and sleep deprivation together with heavy continuous physical activity on the secretion of catecholamines, plasma renin activity and aldosterone levels have been

studied in 3 groups of subjects (Opstad et al. 1985). One group did not receive any extra sleep or food; another was compensated for their energy deficit by additional food supplements; and the third was allowed to nap. The first and third groups had a significantly higher plasma renin activity and aldosterone levels than the second group. Catecholamine levels increased significantly throughout the experiment in all 3 groups but in group 2, which received a special diet to compensate for the energy expenditure, the response was significantly lower after 3 and 4 days than in the other groups. Sleep deprivation alone did not exert a major influence on the renin-aldosterone mechanism. On the other hand, prolonged exercise did stimulate this mechanism. Opstad et al. (1985) suggested that energy deficiency, rather than sleep deprivation, stimulated catecholamine secretion.

The effects of anticipation and the stress of actual sustained operations on the secretion of catecholamines and cortisol were studied in a group who was not aware of the total duration of the sleep deprivation period, and a second group who knew beforehand that the sleep deprivation would not exceed 42 hours (Francesconi et al. 1978). The first group showed significantly higher levels of both urinary 17-hydroxycorticosteroids and plasma catecholamines than the second group, suggesting that both the sympathicoadrenomedullary and adrenocortical activities were affected by the anxiety and uncertainty of the experiment rather than by sleep deprivation *per se*.

A comparison of the sympathoadrenal response to treadmill exercise in subjects receiving normal sleep and subjects sleep deprived for 50 hours showed that sleep loss had no effect on oxygen uptake, carbon dioxide production, minute ventilation, heart rate, blood lactate, plasma adrenaline (epinephrine), dopamine and noradrenaline (norepinephrine) after 12 minutes of exercise (Martin & Chen 1984). The only significant difference between the sleep-deprived and control conditions was a 20% decrease in the exercise time to exhaustion. Although sleep deprivation of 50 hours decreased the ability of subjects to exercise to exhaustion, this decrease was not accompanied by any metabolic, ventilatory, thermal or peripheral sympathetic nervous system responses to exercise.

Sleep deprivation for 64 hours in sedentary subjects isolated from external cues decreased or obliterated the circadian rhythmicity of heart rate and blood pressure (Ahnve et al. 1981). Urinary adrenaline and noradrenaline levels did not show any correlation with the cardiovascular variables. A significant correlation, however, was seen between urinary adrenaline levels and the heart contractility. This study differs from other studies surveyed in this review in that the researchers isolated their subjects from any 'Zeitgebers'.

Immediately after a 24-hour endurance run or a 10-hour triathlon, cortisol levels were significantly elevated to the same degree (Fellmann et al. 1988). Following 24 hours of recovery, the cortisol levels returned to baseline levels after both races. On the other hand, noradrenaline levels increased after the 24 hours' race and remained virtually unchanged for the next 24 hours, whereas the adrenaline and dopamine levels were not significantly elevated. In the 10-hour triathlon race, an increase in dopamine levels was found without any change in adrenaline and noradrenaline levels. It was suggested that the changes observed in these hormones were not due to a circadian rhythm oscillation.

Growth hormone is an anabolic hormone promoting protein synthesis and gluconeogenesis and is secreted during both specific and nonspecific types of stress (Schalch 1967; Selye 1955). Some typical examples of a specific stress inducing growth hormone secretion are insulin-induced hypoglycaemia (LeBlanc et al. 1982; Roth et al. 1963), submaximal exercise (Sutton & Lazarus 1976) and prolonged hypolipidaemia (Quabbe et al. 1972). Growth hormone is also secreted in increased quantities during nonspecific stress stimuli such as surgery (Noel et al. 1972; Schalch 1967; Sowers et al. 1977), psychological stress, such as school examinations or parachute jumping (Mikulaj et al. 1975; Schalch 1967), and during prolonged sleep deprivation combined with continuous physical activity, cold exposure and energy deficit of 29,000 kJ to 42,000 kJ (Aakvaag et al. 1978; Opstad &

Aakvaag 1981, 1983; Opstad et al. 1980). The magnitude of the growth hormone response to continuous physical activity is largely determined by the physical fitness of the subjects, trained subjects exhibiting a lower growth hormone response to psychological stress (Mikulaj et al. 1975) and exercise stimulus (Bloom et al. 1976) than untrained subjects. Conversely, trained subjects respond with a significantly larger growth hormone secretion in insulin-induced hypoglycaemia than untrained ones, due to a greater severity of hypoglycaemia caused by the higher insulin sensitivity in trained subjects (LeBlanc et al. 1982).

Most research on growth hormone secretion has been performed in subjects with normal sleep, selective slow-wave sleep deprivation, or changes in sleep-wake cycles, while very few results are reported for total sleep-deprivation condition. In humans, growth hormone is secreted mainly during slow wave sleep, and does not exhibit a circadian rhythm-dependent secretion (Sassin et al. 1969a,b; Takahashi et al. 1968; Weitzman et al. 1974). In rats, an injection of growth hormone results in an increased amount of REM sleep (Drucker-Collin et al. 1975), suggesting that the growth hormone released during slow-wave sleep (stages 3 and 4) allows the onset of REM sleep, during which time brain protein synthesis appears to be sharply increased. Higher growth hormone levels have been reported in dogs during a recovery sleep after 6 and 12 hours of forced wakefulness (Takahashi et al. 1981). This elevated secretion was almost always preceded by slow-wave sleep. No changes in growth hormone levels were reported while the dogs were kept awake. This evidence supports the view (Adam & Oswald 1983) that brain and general protein synthesis, cellular proliferation, and repair and growth of the tissues are increased during all stages of sleep. This view is opposed by others (Horne 1983), who claim that there is no conclusive evidence to suggest that tissues, with the exception of brain, are in any different resting state than when the subject is awake. In fact, sleep-related fasting is detrimental to protein synthesis.

The hormonal responses to the combined effects of sleep deprivation, psychological stress, energy deficit, physical activity and cold have been examined in a group of Norwegian Military Academy cadets undergoing a 5-day Rangers course (Aakvaag et al. 1978; Opstad & Aakvaag 1981, 1983; Opstad et al. 1980). Aakvaag et al. (1978) divided subjects into 3 groups: one group had no organised sleep whatsoever; the other two groups had 3 and 6 hours of sleep between days 3 and 4 of the course, respectively. All 3 groups were involved in strenuous physical activity throughout the whole course and experienced cold exposure (5 to 15°C) and an energy deficit of 29,000 to 42,000 kJ/day. Increased plasma levels of growth hormone were found in the morning in all 3 groups, with the highest levels in the group which had had no organised sleep. These elevated growth hormone levels were attributed to the stress caused by sleep deprivation and energy deficit. It is difficult in this experiment to identify the factor, or factors, responsible for the observed elevated growth hormone levels, since there were no controls for many of the variables.

Opstad et al. (1980) exposed similar subjects to the same conditions as mentioned above, but a standard exercise test of 30 minutes' cycling at approximately 50% $\dot{V}O_{2max}$ was introduced prior to the trial and on days 3 and 5 of the course. The growth hormone response to such an exercise was significantly higher on days 3 and 5 than prior to the sleep deprivation, but there were no differences in response between days 3 and 5. Cortisol levels were also elevated during the exercise in the sleep-deprived condition compared with the control. Conversely, insulin levels significantly decreased during the exercise in the sleep-deprived state on day 3. These investigators proposed that the elevated growth hormone levels helped to shift energy metabolism from carbohydrate to free fatty acid substrates. However, due to the experimental design, it was impossible to determine whether the major factor contributing to these changes was sleep deprivation, energy deficit, physical activity, cold exposure, psychological stress or a combination of any of these factors.

In a subsequent study (Opstad & Aakvaag 1981), the role of energy deficit as a contributing factor

to the elevated growth hormone levels was assessed by comparing 2 groups, an isocaloric and a low-energy group, under conditions similar to their previous studies cited above. Although growth hormone was elevated in both groups during the Rangers course, the isocaloric group had significantly lower elevations in growth hormone than the low calorie group. Cortisol was also lower in the isocaloric group, while insulin levels were lower in the low calorie group. It appears that the main stress factor in these experiments was the prolonged physical strain, with energy deprivation being only a moderate contributing stressor.

To assess the contribution of sleep deprivation to growth hormone secretion, another study from the same laboratory (Opstad & Aakvaag 1983) examined the effect of introducing a sleep period during the Ranger course. One group of subjects was allowed 3 hours of sleep a night, but it is not stated whether they actually slept, whereas a second group had no organised sleep throughout the length of the course. The first group showed higher levels of growth hormone during the sampling periods than the sleep-deprived group. Cortisol levels were also significantly higher in the sleep group. On the other hand there were no differences in the catecholamines levels between the 2 groups. The authors attributed this finding to the fact that blood samples were taken within 30 minutes of the subjects awakening in the morning and that the growth hormone and cortisol levels were influenced by the prior sleep period.

A sleep deprivation of 50 hours together with prolonged physical activity increased significantly the growth hormone secretion in response to a cycle exercise at 70% of $\dot{V}O_{2max}$ compared with sleep-deprived sedentary or normal conditions (Van-Helder 1988). High growth hormone levels found in the sleep-deprived, physically active subjects were not accompanied by any corresponding changes in plasma insulin, cortisol, glucose or lactic acid. Thus, it seems that sleep deprivation together with physical activity potentiates a higher growth hormone response to an exercise bout.

Several studies suggest that sleep deprivation gives rise to the development of insulin resistance in subjects. An elevated and protracted hyperglycaemia, possibly mediated by increased catecholamine levels, have been found during an oral glucose tolerance test in subjects sleep deprived for 100 hours than in control conditions (Kuhn et al. 1969).

Similarly, significantly lower insulin and significantly higher glucose levels were found during an oral glucose tolerance test in cadets on day 5 of a 5-day Ranger course compared with precourse control levels (Opstad & Fonnum 1983). The low insulin levels were not due to inhibition by high catecholamine levels, since the low insulin levels persisted even after the catecholamines levels had returned to baseline. These results suggested that prolonged physical activity combined with an energy deficit and sleep deprivation may result in decreased pancreatic β-cell sensitivity to a glucose stimulus or possibly an insulin exhaustion resulting in impaired carbohydrate metabolism. The significantly elevated plasma glucose levels suggest that the subjects may have reached a decreased glucose tolerance state.

Similar conclusions were reached using a different approach from previous investigators (Vondra et al. 1981). Subjects were deprived of sleep for 120 hours, apparently under laboratory conditions. It was not reported, however, how the subjects occupied themselves during this sleep-deprivation period. Subjects underwent a muscle biopsy before and after 120 hours of sleep deprivation and the samples were assayed for various enzyme activities. A decrease in citrate synthase and malate dehydrogenase activity was found, indicating a decreased working capacity of the Krebs cycle and therefore a decrease in the aerobic ATP formation capacity of the muscle. The hyperglycaemia observed and the limited probability of pyruvate entering the Krebs cycle suggested that a decreased aerobic metabolic state existed after 120 hours of sleep deprivation, supporting the findings of Opstad and Fonnum (1983).

In a study of oral glucose tolerance performed on a group of subjects in 3 conditions (sleep and physical activity, sleep deprivation and physical activity, sleep deprivation and sedentary), the

highest insulin response was found in the sleep deprivation and sedentary condition (VanHelder 1988). Insulin secretion to a glucose load stimulus was also significantly higher in the sleep deprivation and physical activity condition than in the sleep and physical activity condition. It was suggested that sleep deprivation of 60 hours decreased insulin sensitivity and/or responsiveness to a glucose load. This decrease was further enhanced by the physical inactivity of the subjects in the sleep deprivation sedentary condition but could be countermanded by physical activity during sleep deprivation.

7. Conclusions

Although sleep deprivation has long been considered to be a stressful condition (in the Selyean sense), the actual findings from sleep-deprivation studies do not seem to bear this conclusion out (Horne 1983). To date, it seems that the main adverse effects of sleep deprivation are evident in the mental performance of sleep-deprived subjects. The physiological parameters such as heart rate, $\dot{V}O_{2max}$, systolic and diastolic pressure, and respiration all fail to show any consistent significant changes during sleep deprivation. The inability to maintain a high level of physical performance during sleep deprivation can be attributed primarily to psychological fatigue as witnessed by increased rating of perceived exertion scores in face of unchanged heart rates.

The effects of prolonged sleep deprivation on growth hormone secretion sensitivity and carbohydrate metabolism deserve further attention. It is not known why large amounts of growth hormone are secreted primarily during sleep. Also, the mechanism of altered growth hormone secretion sensitivity during sleep deprivation is unknown. It appears that increased growth hormone levels during slow-wave sleep (Sutton & Lazarus 1976) are not accompanied by increased levels of blood glucose (Takahashi 1968). Furthermore, it is not clear whether the growth hormone sleep-related secretion is of a permissive nature to maintain glucose levels throughout the night fasting period at levels sufficient for CNS activities (Adam & Oswald 1983), or whether the secretion also plays an important role in brain protein synthesis (Drucker-Collin et al. 1975). Concomitantly, it is also not known how and why prolonged sleep deprivation of more than 40 hours appears to induce insulin resistance and glucose intolerance (Kuhn et al. 1969; Opstad & Fonnum 1983; VanHelder 1988; Vondra et al. 1981).

The studies carried out on military cadets (Aakvaag et al. 1978; Opstad & Aakvaag 1981, 1983; Opstad & Fonnum 1983; Opstad et al. 1980) cannot be truly classified as pure sleep-deprivation experiments. As was previously mentioned, in all of these studies, the subjects were exposed to a combination of stressful stimuli. Although some of these stimuli were eliminated at one time or another to examine the combined effect of the remaining ones, there is still one outstanding deficiency in all of these experiments; in none of these studies was the subject totally deprived of sleep for more than 24 hours. In every experimental protocol, the subjects had had some sleep even if it was for only a few hours. Therefore, these studies, while suggestive, leave the specific effect of sleep loss *per se* entirely unknown.

Most of the studies reported cannot be compared with each other in any meaningful fashion since the experimental conditions and methods of assessing the endocrine responses were so diverse. The effects of sleep deprivation on physiological responses of the subjects are difficult to compare between various studies because of the different lengths of sleep loss employed. Because of the apparent additive effects of sleep loss, it is difficult to extrapolate and predict what may happen after, for example, 100 hours of sleep deprivation based on the data available from a 60-hour sleep-deprivation experiment. On the other hand, it is unlikely that any sport or work event would demand more than 60 hours of continuous activity. Thus, this problem is of limited interest to the athletic community.

It does appear that athletes do not have to be concerned about their physical performance following a sleep loss of one or even two nights if

sufficiently mentally motivated. One of the limitations in all presently available studies is that none of them have examined élite athletes. It is therefore difficult to predict whether the changes observed in the subjects tested are applicable to highly motivated elite athletes. However, athletes competing in sports where both their mental and physical acuteness have to be at their peak throughout the whole performance, such as in fencing or the biathlon, may be affected by sleep deprivation. Although the metabolic changes brought about by disturbances in hormonal secretion after sleep loss do not appear to influence the physical performance of average subjects, it is not clear how these changes may affect the peak performance of elite athletes.

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