PHYSICAL FITNESS AND SLEEP

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ABSTRACT

Physically fit athletes have been found to have elevated slow wave sleep (SWS), longer sleep duration and decreased sleep onset latency (SOL) compared with unfit sedentary individuals. It has been hypothesized that the critical variable in these effects is the subject's level of fitness resulting from habitual exercise. Certain negative findings, however, cast doubt on this interpretation. The aim of the current series of experiments was to further investigate the effects of physical fitness on electroencephalographic (EEG) and hormonal aspects of sleep. These investigations are of theoretical interest as they potentially provide information regarding the functional significance of sleep and SWS in particular. Those theories which hypothesize a relationship between peripheral metabolism and sleep, for example, the restorative and energy conservation theories, would predict that chronic physical exercise would promote SWS and sleep-related anabolic hormones. In contrast, those theories which do not propose a direct relationship between peripheral metabolism and sleep, for example, the cerebral restitution and immobilization theories, would predict no effect of physical fitness on sleep.

By using independent group designs, previous studies have potentially confounded aerobic fitness with other characteristics of athletic individuals. Therefore, the first experiment in this thesis assessed the sleep of proficient athletes on two occasions; initially when they were unfit and subsequently when aerobically fit. In addition, the sleep of athletes was compared with that of

an unfit, non-athletic, sedentary group. The athletes tended to sleep longer and had elevated SWS and non-rapid eye-movement (NREM) sleep compared with non-athletes. These differences were independent of the aerobic fitness of the athletes. Thus, it was concluded that aerobic fitness was not a critical factor but, rather, had been confounded with a more enduring variable associated with physical fitness.

A secondary issue addressed in Experiment 1 was the relationship of daytime exercise, physical fitness and SWS. It has been hypothesized that a facilitative effect on SWS of a single exercise session is dependent on the subjects being physically fit. This hypothesis was tested by assessing the effect of exercise on sleep in the athletes when they were unfit and subsequently when they were fit. The hypothesis was not supported as no effect of exercise on sleep was observed. It is possible, however, that fitness may increase the probability of an exercise effect occurring when one or more other conditions are met.

Experiments investigating the effects of physical fitness on sleep have typically concentrated on EEG variables. A number of factors have also suggested that hormonal aspects of sleep may be influenced by physical fitness. Experiment 2, therefore, was designed to examine the effect of physical fitness on the night-time secretion of human growth hormone (hGH), prolactin and cortisol. In addition, the relationship between the secretion of these hormones during the night and body composition was assessed.

Two groups of 17 subjects, one of fit athletes and the other of unfit non-athletes, were selected so that the groups were matched for weight, height, lean body mass (LBM) and fat levels. Subjects slept in a sleep laboratory for 3 non-consecutive nights; an adaptation night and two experimental nights. On one experimental night blood samples were collected, while on the other, baseline sleep was assessed and the catheter was not inserted. Weight and height were measured and LBM assessed by 24hr urinary creatinine. The effect of physical fitness was tested by a comparison of the two groups, while the effect of body composition was assessed by correlation analyses.

Physical fitness did not have a significant effect on either sleep or hormone levels, though in the latter case the results were marginal and are worthy of further investigation. Body composition was related to hGH level, percentage LBM being positively correlated with hGH levels. These results were significant for all subjects combined and for the fit group, though not the unfit group alone. Consistent with the findings of Experiment 1, it was concluded that physical fitness is not a critical factor influencing sleep variables and that previous studies may have confounded it with other variables.

Since body composition is related to physical training it was hypothesized that differences in SWS observed between athletes and non-athletes may be related to differences in body composition. This hypothesis was tested by comparing sleep and anthropometric variables of

fit athletes and unfit non-athletes. Two sets of data were analysed. One from the Hobart laboratory included subjects from Experiment 1 plus others run in other experiments at that time (designated Experiment 3a), while the other consisted of the subjects from Experiment 2 (designated Experiment 3b). Twenty-five fit and 22 unfit subjects were run in Experiment 3a and 17 fit and 17 unfit in Experiment 3b. LBM and fat were estimated using a different method in each experiment. The results showed percentage LBM was negatively correlated to SWS in fit subjects while the amount of LBM and weight were negatively related in the unfit groups. When all subjects were combined within each experiment, significant negative correlations were found between SWS and both LBM and percentage LBM in Experiment 3b. The results, therefore supported the hypothesis that body composition influences SWS levels.

Different types of physical training develop different anthropometric characteristics and other physiological attributes. Thus, it was considered possible that different types of training would influence sleep. This hypothesis was tested in Experiment 4. The sleep of four groups of 10 young male subjects who differed with respect to the type of athletic training in which they habitually engaged, was compared on two consecutive, non-exercise nights. The groups were : aerobically trained endurance runners, power trained weight lifters and bodybuilders; athletes with mixed anaerobic, aerobic and power training; and an unfit, non-athletic, sedentary, control group.

Pre-planned comparisons showed that the control group did not differ from the combined athletic groups on any sleep variable. However, the aerobic group had more SWS and NREM sleep, slept longer and had shorter SOLs than the power group. The mixed group was intermediate on each of these variables. The data show that the type of physical training in which athletes engage has substantial effects of their sleep. It was not possible to determine from Experiments 3a, 3b and 4 if the effects of type of training and body composition are related. The results, however, demonstrate that variations in peripheral physiological factors are related to sleep architecture; particularly to SWS.

The results of the experiments reported in this thesis clearly show that aerobic fitness has no direct effect on the sleep variables assessed. They do, however, indicate that peripheral factors associated with physical training are related to aspects of sleep architecture. The results also have implications for theories of sleep. While sleep may serve cerebral restitution and immobilization functions it also appears to be influenced by peripheral factors and thus the findings are inconsistent with present formulations of the cerebral restitution and immobilization theories of sleep. However, despite finding peripheral effects on sleep, the data were not consistent with a general restorative view as the direction of the results was largely incompatible with this theory. Finally,

in relation to the energy conservation theory of sleep as applied within species or within individuals, the data indicate that athletes as a broad group do not use the elevation of SWS or TST to compensate for high energy expenditure. A subgroup of athletes (endurance athletes), however, may use this method to balance their energy intake and expenditure.

CHAPTER 1.

A REVIEW OF THE EMPIRICAL LITERATURE ON THE EFFECTS OF PHYSICAL FITNESS AND EXERCISE ON SLEEP

In Baekeland and Lasky's (1966) original experiment investigating the consequences of exercise and sleep in humans, two independent effects emerged. Firstly, afternoon exercise resulted in elevated levels of slow wave sleep (SWS) during the subsequent nights sleep, indicating an immediate facilitative effect on SWS of a particular exercise session; an exercise effect. Secondly, regardless of the exercise condition, the athletes used in this experiment had higher levels of SWS than would have been anticipated on the basis of normative data. It was later proposed (Griffin and Trinder, 1978) that such a sustained elevation of SWS may be the result of elevated physical fitness, derived from habitual exercise; a fitness effect. It is the latter effect which is the primary experimental focus of this thesis. The main aim was to determine the derivation of the SWS fitness effect. Other empirical issues addressed include the nature of the interaction between fitness and exercise effects and the relationships between physical fitness, other sleep variables and sleep related hormone secretions. In this chapter the experimental literature related to these issues will be reviewed.

Physical Fitness and SWS

A relatively small number of experiments are directly relevant to the question of the relationship between fitness

and sleep, so these will be described in some detail. As stated, the first indication that physical fitness, or a related factor, may affect sleep was reported by Baekeland and Lasky (1966). They used 10 male college students as subjects, who were described as being "accustomed to participate in strenuous athletics of various kinds (track, swimming, basketball etc.) at least three times a week ..." (Baekeland and Lasky, 1966, p.1204). Sleep was recorded on three non-consecutive experimental nights, once in each of three conditions, a no exercise control and an afternoon and evening exercise condition. Baekeland and Lasky (1966) observed that the mean level of SWS on the control night of 32.5% in the first 6 hrs of sleep, was considerably higher than the mean SWS level of 20.9% of total sleep time (TST) obtained by Williams, Agnew and Webb (1964) in a normative study on a similar age group.

Similar findings have been reported in two subsequent studies (Buguet, Roussel, Angus, Sabiston and Radomski, 1980; Zloty, Burdick and Adamson, 1973) investigating the effects of daytime exercise on sleep. Buguet et al.(1980) examined the sleep of six cardiovascularly fit, young, male volunteers (mean age = 20.1 yrs; mean maximal oxygen uptake (VO_{2max}) = 62.3 ml/kg x min; (details of fitness assessment are presented in Chapter 3)), during a five day control phase, a six day exercise phase and a five day recovery phase. The authors note that control levels of SWS (117.07 min or 27% TST) and Stage 4 (94.38 min or 22% TST) were higher than those reported in normative studies by Williams et al. (1964) and Feinberg and Carlson (1968). (In the latter study

Stage 4 represented approximately 12% of TST). Zloty et al. (1973) measured the sleep of 16 male distance runners with a mean age of 24 yrs. Sleep was recorded on three consecutive nights and during the experimental period subjects continued their regular running programmes. SWS accounted for a mean of 23% or 104.7 min of TST; again, a higher level than reported by Williams et al. (1964).

Since subjects were selected on the basis of proficient athletic performance or high VO_{2max} these studies suggest the possibility that some aspect of physical fitness is related to higher SWS. The major problem with this interpretation is that no unfit, sedentary control groups were used. Further, there were considerable differences in experimental and scoring procedures between studies. Thus, both Zloty et al. (1973) and Buguet et al. (1980) used the scoring criteria described by Rechtschaffen and Kales (1968) while Williams et al. (1964) and Feinberg and Carlson (1968) used the scoring criteria of Dement and Kleitman (1957). Comparison of results between laboratories is therefore somewhat unreliable. However, two recent experiments, (Griffin and Trinder, 1978; Trinder, Bruck, Paxton, Montgomery and Bowling, 1982a), in which unfit sedentary subjects acted as controls, supported earlier speculations.

Griffin and Trinder (1978) compared the sleep of 8 fit subjects (mean age = 23.3 yrs) and 8 unfit subjects (mean age = 23.5 yrs). There were four males and four females in each group and cardiovascular fitness was determined using a submaximal bicycle ergonometer fitness test. Each subject slept in the laboratory on two non-consecutive experimental

nights, one following late afternoon exercise and the other following a day of no exercise, other than that required to carry out their daily routine. The order of the two conditions was counterbalanced. The exercise consisted of a 7.3 km run over a hilly course. An analysis of variance showed fit subjects had significantly higher amounts of SWS (119.7 min vs. 94.0 min) regardless of exercise condition. This difference was entirely due to differences in the Stage 3 component of SWS (60.4 min for the fit and 37.8 min for the unfit group).

A second study by Trinder et al. (1982a) provided further support for a fitness effect on SWS but also indicated certain limitations. It had been suggested (Baekeland and Lasky, 1966; Griffin and Trinder, 1978) that the apparently sustained elevation of SWS in fit subjects may not be related to physical fitness but rather to a residual exercise effect resulting from exercise on days immediately preceding the non-exercise testing night. It was also proposed (Trinder et al.,1982a) that since SWS levels fall as a function of age, the fitness effect may be larger in an older population.

To test these hypotheses the sleep of four groups of six subjects was measured. Two groups contained aerobically fit athletes by VO_{2max} criteria, who trained a minimum of three times a week. Two groups contained unfit subjects, with low VO_{2max} scores, who did not exercise regularly in any way. The two groups within each fitness category differed as to ages, the mean age of the younger fit and unfit and older fit and unfit subjects being 21.67, 22.33, 31.83 and 31.83 yrs respectively. Three measurements of sleep were made on each subject over a five night period, on nights 1 (N1), 3 (N3),

and 5 (N5). On the afternoon of N1 fit subjects exercised at the level of a normal training session but during the subsequent 4 days they did no training. Unfit subjects performed no exercise other than that involved in their usual daily activities.

The results of this study provided support for the existence of a fitness effect in the younger age group but not in the older one. Thus, an analysis of variance revealed a significant interaction between fitness and age. SWS was significantly elevated in the young fit group (124.5 min) compared to the young unfit group (93.3 min) but there was no significant difference between the two fitness levels in the older groups (87.9 min for fit and 100.1 min for unfit groups). The elevated SWS in the younger fit group was due to contributions from both Stages 3 and 4. Total non-rapid eye movement (NREM) sleep showed a similar trend but did not reach significance.

This experiment also indicated that the effect of fitness is a sustained one rather than a result of an elevation in SWS in response to exercise on the previous day, as no interaction between fitness and nights of sleep recording was observed. SWS was still elevated on the fifth night after the exercise session in the young fit group. The mean SWS levels for the young fit subjects were 120, 128 and 125 min for nights 1, 3, and 5 respectively, while for the unfit group the equivalent values were 92, 91 and 97 min. As the physiological effects of the exercise would have dissipated by this time the data show the independence of the fitness effect from the exercise effect.

While the experiments so far described indicate a sustained elevation of SWS in fit young adults, two papers reporting unexpectedly low levels of SWS in fit populations (Paxton, Montgomery, Trinder, Newman and Bowling, 1982; Walker, Floyd, Fein, Cavness, Lualhati and Feinberg, 1978) suggest the phenomenon is not unequivocal. Paxton et al. (1982) described two studies designed to investigate the effects of exercise of varying intensity on sleep, one in unfit and the other in fit subjects. In the first experiment 24 aerobically unfit subjects with the mean age of 21.67 yrs, were used. Subjects slept in the laboratory for 4 non-consecutive experimental nights. The mean SWS level in this sample was 98.99 min, comparable with other unfit groups studied in this laboratory (Griffin and Trinder, 1978; Trinder et al., 1982a). Different exercise regimes did not influence SWS.

In the second experiment (run concurrently with the first experiment in this dissertation), 11 fit male subjects with a mean age of 19.5 yrs were used. Each subject slept in the laboratory on 8 non-consecutive nights in four different exercise conditions. Since there was no effect of exercise conditions on SWS, SWS levels for each subject on the 8 nights were averaged, the mean for all subjects being 82.9 min. This is lower than might have been anticipated on the basis of other experiments from the same laboratory (Griffin and Trinder, 1978; Trinder et al., 1982a). However, in the absence of a control group, between experiment differences in scoring criteria cannot be discounted.

Walker et al., (1978) have also failed to find evidence

for a fitness effect on SWS. Again the study was designed primarily to investigate the effect of exercise on the subsequent nights sleep, but matched athlete and non-athlete groups were also used. Subjects were 10 male cross-country or distance runners (mean age = 19.3 yrs) and 10 non-athlete control subjects (mean age = 20.2 yrs). The control subjects did not engage in any sustained exertion or training.

Both groups had mean heights of 178cm, however the runners were lighter (mean weight = 63.1kg) than the non-runners (mean weight = 66.2kg). Each subject slept 4 consecutive nights in the laboratory, the first being an adaptation night. Athletes exercised every afternoon except the last, while non-athletes performed no exercise except for a short run on the 4th afternoon.

The difference in SWS between the runners and non-runners was not significant (mean SWS for runners being 88.3 and 87.3 min for no exercise and exercise conditions respectively, and for non-runners 82.5 and 79.4 min for no exercise and exercise respectively). However, it is notable that runners showed significantly higher levels of NREM sleep than non-runners regardless of exercise condition (mean NREM for runners being 341.7 and 347.0 min and for non-runners being 313.0 and 313.4 min for no exercise and exercise conditions respectively).

Of the experiments reviewed a sufficient number find that young fit athletes have greater amounts of SWS than unfit non-athletes to indicate the possibility of a factor which influences SWS level. However, the contradictory reports suggest that physical fitness itself may not be the

relevant component. This conclusion is supported by the fact that in both the studies finding positive results and those finding negative ones, the subjects appear to be quite similar in cardiovascular fitness and athletic proficiency. Experiment I (Chapter 4) was designed to determine if indeed cardiovascular fitness is the critical factor in the higher levels of SWS observed in athletic subjects.

Physical Fitness and Other Sleep Variables

The experiments investigating the relationship between fitness and sleep have been designed primarily to observe effects on SWS. As a result other effects may sometimes have been masked by laboratory procedures (for example limiting TST by restricting time in bed (TIB)), or not analysed or reported. In particular, there is evidence that sleep onset latency (SOL) is decreased and sleep duration increased in fit subjects. Despite limiting TIB to 8 hrs and matching runners and non-runners for estimated sleep length, Walker et al. (1978) found runners to have a greater mean TST than non-runners ($.05 > p > .01$) in the exercise condition. A more definite indication that TST is elevated in physically fit subjects comes from a re-analysis of five experiments conducted at this laboratory (Montgomery, Trinder and Paxton, 1982)[1]. The procedures used in these experiments enabled considerable variation in sleep time as subjects selected their own bedtimes and rising times, and therefore normal sleep durations are likely to have been reflected in the laboratory recordings. Each of the experiments involved subjects who were exercising regularly and were physically fit and/or subjects who did not habitually exercise and were

1. One of the experiments in this analysis was Experiment 1 (Chapter 4) of this thesis.

unfit. Both groups were typically assessed following both exercise and no exercise days. While the individual experiments did not show a significant difference in TST between fit and unfit groups, when considered together the results of the five experiments clearly indicate that TST is higher in fit than in unfit subjects. In each of the three experiments in which both groups were run, fit subjects slept longer. Further, there was no overlap between the two groups over the five experiments, TST for each of the fit being higher than each of the unfit groups. There was, however, no consistent effect of exercise on TST across the experiments. An analysis of the data, treating groups as replications showed a significant effect of fitness but no effect of exercise or interaction between the two. There appeared to be two main sources of elevated TST in fit subjects. They tended to have a longer TIB (though this was not significant) and a significantly shorter SOL.

NREM-REM cycle length has been examined by Trinder, Stevenson, Paxton and Montgomery (1982b) in 18 fit and 24 unfit young adult subjects under no exercise and moderate daytime exercise conditions. Sleep cycle length was found to be significantly shorter in the fit subjects but was unaffected by exercise condition. The effect of physical fitness was independent of the position of the cycle during the night. No other studies comparing fit and unfit subjects have reported significant sleep cycle or REM latency differences between the two groups. Walker et al (1978) however, observed a trend ($.05 > p > .01$) towards a lower percentage of REM sleep and significantly smaller amounts of eye movement activity in

runners compared to non-runners regardless of exercise condition.

Daytime Exercise : EEG Sleep Variables and Sleep-Related Hormone Secretion

The numerous experiments examining the consequences of physical exercise on the subsequent nights sleep have recently been reviewed (Horne, 1981; Shapiro, 1981; Torsvall, 1981). In the present section this literature is summarized and general issues discussed. Those studies directly relevant to the design of Experiment I and those published subsequent to the running of this experiment are discussed in the Introduction and Discussion respectively of Chapter 4.

Daytime Exercise and EEG Sleep Variables in Humans : Daytime exercise in human subjects has been observed to increase subsequent SWS (Baekeland and Lasky, 1966; Maloletnev, Telia and Tchatchanashvili, 1977; Shapiro, Bortz, Mitchell, Bartel and Jooste, 1981; Shapiro, Griesel, Bartel and Jooste, 1975; Shapiro and Verschoor, 1979) and sleep duration (Buguet et al., 1980; Shapiro et al., 1975; Shapiro et al., 1981; Walker et al., 1978; Zir, Smith and Parker, 1971) and decrease SOL (Browman, 1980; Buguet et al., 1980). These findings, however, are not consistently reported and numerous experiments have failed to find effects of exercise on any of these variables (Adamson, Hunter, Ogunremi, Oswald and Percy-Robb, 1974; Baekeland, 1970; Bonnet, 1980; Browman and Tepas, 1976; Desjardins, Healey, and Broughton, 1974; Hauri, 1968; Horne and Porter, 1976; Paxton et al., 1982). Further examination

of these experiments suggests several factors may be important in the observation of exercise effects on SWS. They are 1. the fitness of the subjects, 2. the time of day the exercise is performed and 3. the intensity of the exercise performed.

1. All the experiments which have found an elevation in SWS as a consequence of exercise have used subjects either involved in regular exercise or of above average aerobic fitness, while those which have not found an exercise effect have generally used subjects unselected on the basis of physical condition. Thus, Griffin and Trinder (1978) hypothesized that fitness was a critical factor in determining whether an exercise effect would be found. This hypothesis received tentative support from their experiment in which the sleep of fit and unfit subjects was compared following a moderate intensity afternoon run. Stage 3 was elevated in fit subjects and decreased in unfit subjects following exercise, this interaction being significant. However, the post-exercise level of Stage 3 compared to the no exercise control level in fit subjects was not significant by itself. No significant interaction between fitness and exercise was observed for either Stage 4 or total SWS. Four experiments provide results which conflict with this hypothesis. Bonnet (1980), Buguet et al. (1980), Paxton et al. (1982) and Walker et al. (1978) used fit or trained subjects and failed to observe changes in SWS following moderate intensity exercise. The role of fitness in determining the occurrence of an exercise effect on SWS is explored further in Experiment 1.

2. Baekeland and Lasky (1966) compared the effects on sleep of afternoon and evening exercise with a no exercise condition. They found that SWS was significantly elevated following afternoon exercise, compared with no exercise levels, However, there was no significant difference between evening and no exercise conditions. In addition, evening exercise produced higher levels of wake and Stage 1 than afternoon exercise. It was suggested that the evening exercise acted as a "stressor, producing CNS activation opposing a more general (perhaps metabolic) effect of exercise tending to increase delta (SWS) sleep requirements" (Baekeland and Lasky, 1966, p.1205). Consistent with this data, Hauri (1968) also failed to find an effect of evening exercise on the first 3½ hrs of sleep, although untrained subjects were used. If Baekeland and Lasky's (1966) interpretation of these studies is correct, in order to observe a facilitation of SWS following exercise, the exercise must be performed sufficiently early to allow such stress to have subsided before sleep.

3. A third factor thought to influence the likelihood of observing an exercise effect is the intensity of the exercise. Horne (1981) has speculated that the higher the intensity of exercise and the nearer it approaches a subject's physiological limits, the more likely a SWS increase will be observed. There is some evidence to support this view. Firstly, low intensity exercise has never been reported to produce changes in SWS. Secondly, those studies which have found elevations in SWS have used moderate to very intense exercise in trained subjects.

An alternative view is that the relationship between exercise intensity and SWS is an inverted U function such that SWS is inhibited if the exercise becomes too stressful. A number of conditions indicative of stress have been reported to be associated with a failure to find an exercise effect. These are disturbed sleep (Adamson et al., 1974; Baekeland and Lasky, 1966; Griffin and Trinder, 1978), increased REM latency (Baekeland, 1970; Browman and Tepas, 1976), decreased REM sleep (Bonnet, 1980; Desjardins et al., 1974), increased heart rate (Bonnet, 1980; Hauri, 1968; Browman and Tepas, 1976; Walker et al., 1978) and increased cortisol secretion during the exercise day (Buguet et al., 1980). As unfit subjects are more likely to be stressed by exercise than trained subjects, this concept is consistent with the observation of an exercise effect in the latter but not the former populations.

Two studies (Shapiro et al., 1975; 1981) report both increases in SWS and indications of stress (elevations in disturbed sleep and decreases in total REM). They used fit subjects, however, and the stress may not have been sufficient to counteract the SWS facilitation. Shapiro et al. (1975) examined the effects of exercise of graded intensity and found that SWS increased as exercise intensity increased except in the final most exhausting conditon where SWS declined slightly. One paper which does not provide support for an inverted U shape function of exercise on SWS is that of Paxton et al. (1982). In two experiments, one using unfit and the other fit subjects a range of

exercise intensities, from light to moderately severe, were used. Neither changes in SWS, nor indications of stress were reported. Possibly the highest exercise conditions in these experiments were still inadequate to produce either SWS elevation or stress effects.

Exercise and the Sleep of Animals : A number of experiments have examined the effects of exercise on the sleep of animals, primarily cats and rats. The EEG manifestations of sleep differ between species, however, and the sleep of cats and rats is generally only divided into NREM and REM sleep. Thus, while the effects of exercise on animals may point to possible similarities in man, direct comparisons of animal and human sleep cannot be made. Another factor relevant to the interpretation of some of the animal studies is that extended periods of exercise are also often associated with sleep deprivation. The recovery from the sleep deprivation may overwhelm the effects of exercise. The stress of unusual exercise may also have the same effect.

Of the six animal studies using either cats or rats, three used both relatively mild exercise and mild sleep deprivation (Boland and Dewsbury 1971; Hobson, 1968; Matsumoto, Nishisho, Suto, Sadahiro and Miyoshi, 1968). These studies reported the earlier onset of NREM sleep, an elevation in NREM sleep and a longer latency to REM sleep in the exercise condition. Three further studies (Borbély and Neuhaus, 1979; Friedman, Bergmann and Rechtschaffen, 1979; Šušić and Kovaćević - Ristanović, 1980) used more severe sleep deprivation regimes and manipulated

the level of exercise performed during the deprivation period. While one (Šušić and Kovaćević - Ristanović, 1980) found NREM sleep to be increased and REM decreased in the higher exercise condition, two (Borbély and Neuhaus, 1979; Friedman et al., 1979) found that higher levels of exercise did not enhance the recovery levels of NREM sleep.

Exercise and Sleep Deprivation in Human Subjects : Two studies (Moses, Lubin, Naitoh and Johnson, 1977; Webb and Agnew, 1973) have examined the effects of different exercise conditions during a period of sleep deprivation on recovery sleep in humans. Webb and Agnew (1973) found no differences in recovery sleep following sleep deprivation with bedrest or sleep deprivation with intermittent exercise. In contrast, Moses et al., (1977), in a similar experiment, found elevated TST following sleep deprivation and exercise. As with the animal studies, the effect of sleep deprivation may mask any independent effect of exercise.

Exercise and Sleep-Related Hormone Secretion : Exercise and sleep experiments have mainly concentrated on EEG sleep variables. However, the effects of exercise on the night-time secretion responses of the hormones human growth hormone (hGH) and cortisol have also been examined (Adamson et al., 1974; Zir et al., 1971). Adamson et al., (1974) compared the night-time secretions of hGH and corticosteroids in young adult males following moderate, early afternoon exercise and no daytime exercise conditions. While no changes in EEG sleep variables were observed, hGH secretion was elevated following exercise as indicated by a significant

elevation in the area under the curve between 2400 hrs and 630 hrs, and also a significant increase in peak hGH secretion. Plasma corticosteroid levels showed a significant decrease during the post-exercise night.

In a similar study Zir et al.(1971) found contrasting results. There was a significant increase in mean TST after exercise. However hGH secretion was not consistently influenced by exercise either in terms of peak secretion or area under the curve. Thus there is some suggestion that a single exercise session may effect night-time secretion of hGH and corticosteroids but this is inconclusive. The possibility of physical fitness influencing these hormone secretions has not been examined.

Conclusion

Chapter 1 has reviewed the empirical background to the present series of experiments. In particular, it appears that SWS is generally, though not universally, elevated in aerobically fit subjects. Whether this is a consequence of high aerobic fitness per se or a related factor was the major issue addressed in Experiment I. A second issue investigated in the first experiment was the role of fitness in determining the occurrence of an exercise effect on SWS. This experiment specifically explored the hypothesis that the facilitative effect of exercise was dependent on the subjects being physically fit. In Experiment 2 the effects of fitness on the sleep-related secretions of the hormones hGH, prolactin and cortisol were examined; a hitherto unexplored area.

Experiment I indicated that aerobic fitness was not the critical variable in the observation of elevated SWS in athlete samples. Therefore two further variables related to athletic activities, body composition and type of training were examined in Experiments 3 and 4.

CHAPTER 2.

THE FUNCTION OF SLEEP : A LIMITED REVIEW

Experiments which have investigated the effects on sleep of exercise and physical fitness have generally been conducted to test the view that sleep is related to peripheral metabolism. Theories which take this view, such as restorative and energy conservation theories, would be supported by an increase in SWS and sleep duration as a consequence of physical exercise. In contrast, theories which argue that sleep is unrelated to metabolism, such as the immobilization theory, or related to cerebral metabolism, predict sleep will be unaffected by exercise. Those theories for which the empirical findings of the sleep and exercise studies have particular relevance are reviewed in this chapter.

The Restorative Theory of SWS

A general restorative or repair function has traditionally been ascribed to sleep and more recently to SWS particularly. This traditional view has been elaborated and clarified by Adam and Oswald (Adam, 1980; Adam and Oswald, 1977; 1983; Oswald, 1980) who interpret restoration in terms of protein synthesis and in recent versions of the theory make no distinction between central nervous system and peripheral restoration. They argue that while protein synthesis and degradation proceed all the time, during sleep generally, and during SWS especially, anabolism is favoured. Increases in both TST and SWS following increases in daytime catabolism are therefore regarded as evidence in support of this theoretical position.

Adam and Oswald (1977) believe that several factors operate to provide an optimal environment for protein synthesis during SWS. One is the prevailing cellular energy charge (EC). The EC is a measure of the balance between adenosine triphosphate (ATP), adenosine diphosphate (ADP) and adenosine monophosphate (AMP) such that a relatively high EC is indicative of a high level of chemical energy available in the form of ATP (Atkinson, 1968). When EC is high, synthetic activity is favoured. As a consequence of reduced activity and responsiveness compared to waking levels, the EC of cells is high during SWS. The rise in EC appears, in fact, to be sleep dependent (Durie, Adam, Oswald and Flynn, 1978).

In addition to a favourable energy state in cells for protein synthesis, the hormonal environment appears conducive to anabolic processes. In this context, the SWS dependent peak secretion of hGH is of particular interest. By acting directly on some tissues or via the mediators, the somatomedins, hGH has a wide range of effects which are anabolic in nature. Human growth hormone stimulates protein synthesis in muscle and liver tissue by increasing amino acid uptake into cells, enhancing the activity of the ribosome template units engaged in protein synthesis and promoting the multiplication of ribosomes (Kostyo and Nutting, 1974). Human growth hormone is also important in the stimulation of bone and cartilege growth.

This effect, however, is mediated by the somatomedins. Thus, indirectly, it stimulates the uptake of sulphate, increases uptake of amino acids and their incorporation into protein and promotes mitogenesis and cell replication in skeletal tissue (Cheek and Hill, 1974; Kostyo and Nutting, 1974; Tepperman, 1980). In addition, hGH has metabolic effects. It is released in response to low blood glucose (for example, during exercise) and acts to increase circulating free fatty acids, making more energy available to cells from this source thus sparing both glucose and protein (Berger, 1982; Tepperman, 1980).

As indicated, hGH has a nychthemeral secretion rhythm with the daily maximum rising and declining in the first half of the sleep period (Parker, Rossman, Kripke, Hershman, Gibson, Davis, Wilson and Pekry, 1980; Takahashi, Kipnis and Daughaday, 1968). The major peak is in fact dependent on the occurrenc of SWS (Pawel, Sassin and Weitzman, 1972; Sassin, Parker, Mace, Gotlin, Johnson and Rossman, 1969). The restorative theory proposes that the close relationship apparent between SWS and the anabolic hormone hGH is strong evidence in favour of an anabolic function for SWS.

Similarly, the secretion pattern of the hormone prolactin is also consistent with this proposal. Prolactin is another anabolic hormone with diverse functions, many of which are not clearly understood. In females it is primarily known for its lactogenic effect as it stimulates the synthesis of casein and lactose synthetase (Horrobin, 1980; Tepperman, 1980). In males prolactin is involved in

the control of male reproductive function influencing gonadotropin release, growth of male accessory reproductive glands and indirectly increasing testosterone production (Bartke, 1980; Hermanns and Hafez, 1981; Smith, 1980). In addition, prolactin appears to have some similar growth promoting metabolic effects as hGH and may also enhance the production of the somatomedins (Horrobin, 1979; 1980). Like hGH, prolactin shows a nychthemeral secretory rhythm and the night-time elevation in prolactin is sleep (though not SWS) dependent, occurring about 2 hours after sleep onset (Parker et al., 1980). The relationship between prolactin secretion and sleep is seen as additional support for the restorative theory.

Adam and Oswald (1977) also note that two other hormones with anabolic consequences, luteinizing hormone (LH) and testosterone (T), are sleep dependent. Luteinizing hormone, a gonadotropin which regulates testosterone production (Parker et al., 1980), demonstrates a nychthemeral variation in pubertal boys (e.g. Boyar, Finkelstein, Roffwarg, Kappen, Weitzman, and Hellman, 1972; Parker, Judd, Rossman and Yen, 1975) and the maximum appears sleep related (Kapen, Boyar, Finkelstein, Hellman and Weitzman, 1974). Parker et al. (1980) have also observed a low amplitude rise in LH across the night in recently post-pubertal men. However, they observe that the consensus of a number of studies is that adult males do not show nychthemeral variations in LH concentrations. Testosterone secretion in males reaches its peak near the end of sleep (Parker et al., 1980) and also seems sleep

related, the normal rise being aborted if sleep is missed and the daily maximum shifts immediately if the sleep period is shifted (Parker, Judd and Yen, 1977).

In contrast, the catabolic hormone cortisol reaches its lowest level of secretion during the early part of the night and rises to a peak at the end of sleep. It has also a truly circadian rhythm, the secretion being independent of waking or sleeping (Parker et al., 1980). The main functions of cortisol are to stimulate protein breakdown into glycogen and sugar and to inhibit glucose uptake and oxidation in many cells in order to make energy available for working cells (McMurray, 1977).

Adam and Oswald (1977) suggest that the high levels of some anabolic hormones complemented by low levels of catabolic hormones, during sleep, contribute to an optimal environment for protein synthesis. In addition, the fact that the peak secretions of a number of the anabolic hormones are sleep or SWS dependent, implicates protein synthesis as an important function of sleep.

Findings by Rudman, Freides, Patterson and Gibbas (1973) support this view. They observed that, in subjects deficient in hGH, injections of hGH at 23.00 hrs, prior to sleep, resulted in a greater rise in nitrogen retention and thus presumably protein synthesis, than at the same dose at 8.00 hrs, prior to breakfast. They concluded that this difference was probably due to the morning elevation in cortisol countering the anabolic action of hGH. Further studies reflecting rates of protein synthesis are consistent

with the restorative theory including : 1. amino acid incorporation into protein in rats is inhibited during exercise but enhanced during the resting/sleeping period (Adam, 1980), 2. mitosis, a process dependent on synthesis, has been shown in a large number of mammals to occur at maximal rates during the time of rest and sleep and to be inhibited by exercise (Adam, 1980), and 3. sleep deprivation results in elevated nitrogen excretion probably reflecting enhanced protein and/or amino acid catabolism (Schrimshaw, Habicht, Pellet, Piche and Cholakos, 1966).

Finally, Adam (1980) and Oswald (1980) have drawn support for the restorative theory from observations of the SWS and TST responses to different metabolic demands which appear to indicate a positive relationship between metabolic requirements and amount of SWS and TST. They argue that the higher the metabolic rate and consequent degradation during the activity period, the longer is the period required for compensatory synthesis at night. While the authors of the studies do not necessarily make the same theoretical interpretations, the following findings are cited as consistent with this view.

1. There is a positive correlation over mammalian species between daily metabolic rate and SWS (Zepelin and Rechtschaffen, 1974).

2. Children have higher levels of TST and SWS than adults (Roffwarg, Muzio and Dement, 1966).

3. A group of habitually long sleepers (9.5 - 10.5 hrs) have been shown to have higher mean daytime body

temperatures than a group of average sleepers (7 - 8 hrs) (Taub and Berger, 1976).

4. Hypothyroid patients have low SWS (Kales, Heuser, Jacobson, Kales, Hanley, Zweizig and Paulson, 1967) while hyperthyroid patients have a relatively high proportion of SWS and TST (Dunleavy, Oswald, Brown and Strong, 1974).

5. Following sleep deprivation, TST is increased and the proportion of SWS is elevated (Berger and Oswald, 1962; Williams, Hammack, Daly, Dement and Lubin, 1964).

In a similar manner, studies of the effects of exercise on sleep have been cited in support of the restorative theory. The manipulation of exercise is regarded as a method of varying metabolic demands and requirements for synthetic activity. In this context those experiments which have found the following results are regarded as consistent with this theory.

1. Slow wave sleep is elevated after exercise (Chapter 1, p.10).

2. Slow wave sleep is elevated in athletes compared to non-athletes (Chapter 1, pp.1-5).

3. Human growth hormone is increased and corticosteroids decreased following exercise (Chapter 1, pp. 15-16).

4. Sleep duration is elevated following exercise (Chapter 1, p 10).

5. Sleep duration is elevated in athletes compared to non-athletes (Chapter 1, pp.8-9).

While it has been argued that optimal conditions are provided for anabolic processes during SWS and consequently SWS can be considered to promote tissue restoration (Adam and Oswald, 1977), the theory has been criticised on a number of grounds. In general the issues raised involve the interpretation of empirical findings. The main points concerned are; 1. the level of protein synthesis during sleep; 2. the role of night-time hGH secretions; 3. the nature of the effects of exercise and fitness on sleep; 4. the relevance of sleep deprivation to peripheral restoration; 5. the necessity of sleep for restorative functions. These points will now be elaborated.

1. Recent investigations by Waterlow, Garlick and Millward (1978) and Garlick, Clugston, Swick and Waterlow (1980), suggest that sleep is not an ideal time for net protein synthesis (Horne, 1981; 1983). The main stimulus to protein synthesis appears to be food intake and because of man's usual night-time fast, protein synthesis during sleep would therefore be expected to be low. Garlick et al. (1980) measured protein turnover rates during the sleep period, and found that whilst the rate of breakdown of protein into amino acids dropped slightly during the night, the rate of protein synthesis fell by 33% from daytime to night-time and protein oxidation fell by 62%. Night-time levels only rose with re-feeding. Therefore, there is a net decrease in protein during sleep. Horne (1981) also

notes that while studies of protein synthesis in the rat indicate that synthesis is high during the sleep period, there is no information on protein breakdown or oxidation and possibly turnover rates are high. In addition, rodents do not fast during the sleep period but rather wake regularly to eat.

It has been pointed out that,

> "a high EC does not cause such an increase (in anabolism), but only promotes it, given adequate levels of intra-cellular amino acid substrate. Because of the probability that in human sleep these levels are reduced owing to the fast, and that cells cannot store amino acids to any extent without converting them to protein, then any EC increase in human sleep, if it were to be found, would be unlikely to affect protein metabolism" (Horne, 1981, p.270).

A further factor arguing against a high rate of protein synthesis in humans during sleep is the low metabolic rate at this time. As the energy cost of protein synthesis and precursory activities is appreciable, metabolic activity may be expected to rise above normal resting levels to meet the extra demand. Measures of oxygen consumption during sleep show this is not the case but rather metabolic rate drops between 5-10% (Horne, 1979).

2. The significance of the sleep-related release of hGH has been questioned. Horne (1979; 1981; 1983) suggests

the primary role of hGH during sleep is unclear, the instigation of protein synthesis being only one of the hormone's functions. The actions of the somatomedins during sleep are also unknown and, in fact, the secretion of one of them, somatomedin C, falls at sleep onset and remains low during sleep. Horne (1983) also notes that a GH release during SWS is not observed in most mammals investigated; an unexpected finding if GH secretion is an important aspect in a restorative process occuring during sleep. In addition, while there is a peak in mitotic rates in some human tissues during sleep, these peaks are not sleep dependent and therefore are unrelated to the sleep related hGH release, and in fact may be a consequence of the sleep independent low levels of corticosteroids. Further there is no information regarding cell death rates during sleep in humans. These may also be elevated, resulting in no net gain in tissue cell numbers. Cell turnovers of this kind could still be considered restorative, though not a sign of growth (Horne, 1981).

3. As discussed earlier (Chapter 1), while some experiments have shown higher levels of SWS or TST after exercise in athletes, others have failed to do so. This inconsistency cannot readily be incorporated into the restorative view of SWS. While the failure to observe an exercise effect may be related to the intensity of the exercise or variations in experimental conditions (Chapter 1, pp.12-13), the restorative theory cannot account at present for the observation of exercise effects in

athletes but not in non-athletes. It has been proposed that the increase in SWS may be related to heat stress and the resultant increase in brain temperature (Horne, 1981; Horne and Staff, 1983). It is suggested that unfit subjects may seldom reach a sufficient degree of exertion to result in such an elevation in brain temperature. Consistent with the heat stress view are the findings that in animals hypothalamic warming causes increases in NREM sleep (Heller and Glotzbach, 1977; Sakaguchi, Glotzbach and Heller, 1979) and that both high intensity exercise and passive heating (in a hot bath) in fit subjects resulted in increases in SWS (Horne and Staff, 1983).

4. Meddis (1975) has argued that the discomforts of sleep deprivation and the subsequent increase in SWS and TST are the result of interference with the mechanisms inducing and maintaining sleep rather than with sleep functions. It has also been proposed that the main disturbances during a period of sleep deprivation are related to central nervous system functioning rather than peripheral, somatic functioning as would be predicted by the peripheral restorative theory of sleep (Horne, 1978).

5. Finally, whether sleep or SWS is actually essential for restorative processes to proceed in the periphery has been questioned. Meddis (1975, pp. 682-3) comments regarding experiments which have shown SWS to be responsive to metabolic changes,

> "... we have no direct evidence that the role played by SWS in metabolic functioning is in any way vital ... Until then (further research

> conducted) it may be better to assume that these functions have merely become temporally associated with SWS".

Horne (1979) also proposes that recovery processes may begin at the termination of exercise and intrude into sleep, although they may not be specific to sleep. Notably, however, the restorative theory of SWS does not exclude anabolic processes occuring during wake time but rather proposes that SWS provides an optimal environment for their occurence (Oswald, 1980).

The Energy Conservation Theory of Sleep

The energy conservation theory proposes that the major function of sleep is to enforce rest, thereby limiting metabolic requirements and conserving energy supplies; SWS, being a time of especially low energy usage is regarded as particularly efficient in this function (Allison and Van Twyver, 1970; Berger, 1975; Snyder, 1966; Walker and Berger, 1980; Zepelin and Rechtschaffen, 1974). Not only do the lack of activity and arousal reduce energy requirements, they enable a slight decline in body temperature to occur. During sleep this reduction is in the order of $2^{o}C$ (Heller and Glotzbach, 1977). This decrease in temperature is associated with a drop in metabolic rate sufficient to result in considerable energy savings (Heller, Walker, Florant, Glotzbach and Berger, 1978).

The phylogenetic development of SWS supports the hypothesis that energy conservation is a major function of sleep. Reptiles, which are ectotherms and as such

have relatively low metabolic rates by comparison to endotherms of the same size (Walker and Berger, 1980), do not appear to have the equivalent of mammalian SWS (Allison and Van Twyver, 1970; Berger 1975; Walker and Berger, 1980). In contrast, however, the complete electrophysiological and behavioural manifestations of sleep (SWS and increased arousal thresholds) are present in the endotherms, the birds and mammals. The endotherms generate heat through high rates of metabolism (Walker and Berger, 1980) and while homeothermy enables animals to survive in more extreme environmental conditions, it imposes a very high energy cost (Allison and Van Twyver, 1970). Considering the high energy demands and the fact that energy supplies are very often at a premium, energy conservation whenever possible is clearly adaptive. As Walker and Berger (1980, p.257) describe,

> "Species survival depends not only upon effective reproduction and parental care but also upon the effective utilization of energy resources. Endotherms are generally specialized to forage during either the light or the dark portion of the day and are inactive during the other portion. During periods of inactivity, when the search for and ingestion of food is not possible, any reduction in basal metabolism is energetically adaptive, given the finite amount of energy available at each trophic level in an ecosystem ".

That sleep has an energy conservation function is also indicated by the relationship between sleep, hibernation and torpor, as these latter states of dormancy clearly have this role during periods when energy supplies are scarce. Walker and Berger (1980) have reviewed studies relating to these states, and propose that they are qualitatively and functionally the same but vary on a continuum of arousal and energy conservation processes. In regard to the level of arousal,

> "This continuum extends from waking behavioural inactivity, with immediate capabilities for rapid arousal, through sleep to shallow torpor and hibernation with their greatly diminished capabilities for arousal " (Walker and Berger, 1980, p.257).

Likewise, there is a continuum from sleep, to torpor to hibernation in degree of energy conservation. The drop in body temperature during sleep enables energy to be conserved, however, the far greater drop during hibernation (to as low as $10^{o}C$), and to a lesser extent during torpor (around $25^{o}C$), reduces energy demands so effectively as to enable animals to survive food shortages for prolonged periods.

Observations of EEG characteristics of torpor and hibernation indicate these states as qualitatively related and on a continuum with sleep. While there are variations between species it has been reported that :

1. Prior to hibernation TST increases and the proportion of SWS increases (Walker, Glotzbach, Berger and Heller, 1977).

2. Hibernation is entered from a state of sleep (South, Breazile, Dellman and Epperly, 1969).

3. EEG events characterizing SWS occur during the entrance into hibernation (Walker et al., 1977).

4. While the amplitude of the EEG during hibernation becomes very low the frequency is also low in a manner more characteristic of SWS than REM or wakefulness (Walker, et al., 1977).

5. The exit from hibernation is characterized by EMG and EEG activity resembling emergence from sleep (Walker et al., 1977).

6. In shallow hibernation and torpor the EEG is composed almost entirely of SWS (Harris, Walker and Berger, 1979; Walker, Garber, Berger, and Heller, 1979).

In addition to EEG variables, thermoregulatory processes and mechanisms suggest sleep, hibernation and torpor are related. The drop in body temperature during hibernation is a continuation of the decrease which occurs during sleep (Walker, et al., 1977). Further, the length of dormancy, whether sleep torpor or hibernation has been observed to be inversely proportional to the body temperature of squirrels (Twente and Twente, 1965; Walker and Berger, 1980). Finally, the same thermoregulatory mechanisms appear to be involved in both sleep and hibernation

(Walker and Berger, 1980). Walker and Berger (1980, p.270) conclude,

> "Because of the thermoregulatory, electrophysiological and behavioural continuity among the above 3 states of dormancy (sleep, torpor and hibernation), it is reasonable to assume that there is also a continuity of biological significance among these states, viz. energy conservation ".

The relationship between metabolic rate and sleep variables observed in mammalian species by Zepelin and Rechtschaffen (1974) can be interpreted within the energy conservation framework. They found a positive correlation between SWS and TST, and metabolic rate. They suggest this signifies that species whose waking activity in terms of metabolic rate, is relatively expensive tend to spend a greater proportion of the day asleep and therefore have less time in which to engage in activity. Thus sleep sets a ceiling on metabolic expenditures by limiting the time available for activity and consequently limits metabolic requirements.

The phylogenetic development of SWS and homeothermy appear to be paralleled in ontogeny (Berger, 1975). In mammals in which SWS is not evident at birth (eg, the rat, dog, rabbit and oppossum) but develops within the first few weeks of life, there is also a lack of complete thermoregulation at birth, and SWS and homeothermy develop more or less at the same time. In addition, in man both brain and body metabolism decline with increasing age and

so too do TST and SWS. The decreases in these sleep variables may reflect a reduction in the need for energy conservation.

Further, experiments indicating a relationship between metabolic rate and SWS and TST are consistent with the energy conservation theory. Johns, Masterton, Paddle-Ledinek, Winikoff and Malinek (1975) found a positive correlation between SWS levels and the Free Thyroxine Index (which is positively related to metabolic rate) in six human subjects. Taub and Berger (1976) have observed a higher mean daytime body temperature in a group of long sleepers compared to a group of average length sleepers, possibly reflecting compensatory energy conservation in subjects of higher daytime metabolic expenditure. In contrast, Eastman and Rechtschaffen (1979) manipulated metabolic rate in rats over a six week period by the administration of thyroxine and found that despite an increase in metabolic rate there were no changes in sleep stages or TST.

Varying exercise level is a natural way to manipulate energy expenditure within individuals. Thus the early studies of the immediate effects of exercise on SWS (Baekeland and Lasky, 1966; Hobson, 1968; Matsumoto et al., 1968) were interpreted as compatible with the energy conservation hypothesis (Berger, 1975). This no doubt would also be the case for the findings of high levels of SWS in fit athletes who habitually expend high levels

of energy (See Chapter 1, pp.1-5). The effects of exercise and fitness on TST have also been examined from an energy conservation view. Only one study (Shapiro et al., 1975) has found an increase in TST following a single exercise session and one has found a trend ($.05 < p < .01$) for fit athletes to have more sleep than non-athletes (Walker et al., 1978). However, as Montgomery et al.,(1982) point out, laboratory procedures and experimental designs in the exercise and sleep experiments may have masked changes in TST. Further, TST has not always been analysed or reported. As described in Chapter 1(pp.8-9), Montgomery et al. (1982) looked at a large sample of young male subjects in five different experiments. They found that TST was significantly higher in the physically fit compared to the unfit subjects, although there was no consistent effect of a single exercise session. They propose that, compatible with the energy conservation model of sleep, sleep length is adaptive to relatively sustained, although not short-term, variations in the level of energy expenditure. Nonetheless, two factors mitigate against the interpretation of this result within the energy conservation theory.

> "First, the magnitude of the demonstrated effect was insufficient to account for the assumed difference in energy expenditure between the two groups (fit and unfit) of subjects. Thus it is probable that changes in TST in response to variations in physical exercise are only of minor relevance to total energy expenditure within individual members

> of a species ... Second, none of the present studies included an assessment of energy balance. Thus food intake data were not available, ... it cannot be stated with certainty that the total waking-period energy expenditure of the exercise subjects was greater than the sedentary groups ... (and) it is not known if metabolic rate during sleep varied between the two groups" (Montgomery, et al., 1982, p.167).

In conclusion, there is substantial support for the energy conservation theory of SWS and TST. Phylogenetic and ontogenetic data suggest that species may have adapted their sleep duration and SWS levels to balance the general level of energy expenditure and energy supply. However, the experimental literature indicates that TST and SWS may be unresponsive to variations in energy expenditure within species or individual members of a species, although the data on this issue is, at present, inconclusive.

The Cerebral Restitution Theory of SWS

While the restorative and energy conservation theories propose general restorative functions for SWS, an alternative view is that SWS serves exclusively a cerebral restitution role (Feinberg, 1974; Horne, 1978; 1979; 1981; 1983). Evidence for the cerebral restitution theory is derived both from basic descriptive features of sleep and experimental data.

Feinberg (1974, p.297) hypothesizes that sleep has the function of reversing some "as yet unknown neuronal - metabolic consequences of waking brain activity ". SWS is ascribed the predominent role in this function and REM acts to maximize the occurrence of SWS perhaps by producing a cofactor or other substrate required by SWS for this process. Substantial progress is made in this conversion in the first SWS period, but the process is not completed. REM then occurs to produce more of the vital factor which permits SWS to occur again. This cycle continues until the reversal of the neuronal-metabolic consequences of waking is complete. This model accounts for why SWS take precedence over REM in a nights sleep and in recovery from sleep deprivation. It also presents an explanation of the cyclic nature of sleep.

Amongst the data Horne (1979) regards as consistent with a cerebral restoration view is the finding that there is a relative abundance of 5-HT in the brain during NREM sleep and this neurotransmitter substance appears to promote anabolic activity in brain tissue (Laborit, 1972). During sleep there also appears to be an increase in brain levels of ATP and a decrease in lactate - conditions favourable for cerebral restitution, it is unclear, however, whether these circumstances are exclusive to NREM or REM (Durie et al., 1978; Reich, Geyer and Karnovsky, 1972; Van den Noort and Brine, 1970). Horne (1978) also points out that the main effects of sleep deprivation appear to relate to psychological and ophthalmological functioning, aspects mainly under cerebral control, rather than physiological functioning. There are some EEG changes as a consequence of sleep deprivation and Horne (1978) argues that the EEG

only measures cortical activity and does not reflect the functioning of the rest of the body. In addition, pointing to a cerebral role for SWS, high learning situations, as in a high waking visual load, have been observed to significantly increase SWS while REM sleep is unaffected (Horne, 1976; Horne and Walmsley, 1976).

Horne (1983, p.575) speculates that sleep and particularly SWS,

> "allows some form of cerebral shut-down, increased thresholds of responsiveness to sensory input, and release from the constraining state of quiet readiness, which is typical of waking ".

Such a period may be required, it is suggested, for periods of "off line" restitution in the cerebrum.

Clearly, a theory which proposes SWS has a role solely for central mechanisms, does not predict any alteration in SWS as a direct function of either exercise or fitness. However, as described earlier (Chapter 2, p.28) Horne (1981; 1983) accommodates findings of increased SWS following exercise into the cerebral restitution theory by proposing that extreme exercise produces a rise in brain temperature during the exercise, and it is this which causes a rise in SWS. Horne (1981) suggests that the temperature change may stimulate an energy conservation response similar to that described by Berger (1975) and Walker and Berger (1980). An alternative view is that the SWS increase following a single exercise session is a response to an increase in

brain metabolism and a corresponding requirement for brain restitution resulting from the brain warming (Horne, 1981). The latter hypothesis is most consistent with the cerebral restitution theory. This interpretation provides an explanation of increases in SWS following exercise. However, it does not account for findings of chronic elevations in SWS in fit athletes.

The Immobilization Theory of Sleep

Finally, Webb (1971; 1974) and Meddis (1975; 1977) have proposed a function of sleep which rejects any recovery/restoration role. They view sleep as a behavioural adaptation, the prime function of which is to maintain immobility at times when immobility might be expected to improve an animal's chances of survival. All mammals have periodic rest - activity cycles, organized on a circadian basis which, it is suggested, maximize activity periods most favourable to the species. Thus food gathering is restricted to those times of the day when food is most available and when the species' physiological attributes are most effective. In contrast, the rest period occurs when the environment is most hostile and has least to offer in terms of food. During the rest phase the animal would be least vulnerable to predation and accident, and least wasteful of energy, if it is immobile and in a secure place. The immobility theory of sleep proposes that sleep and associated behaviours ensure these conditions are met. Sleep acts to maintain the integrity of the rest phase by reducing spontaneous behaviour and elevating response thresholds, and other instinctive

behaviours ensure that sleep occurs in maximum security sites whenever possible.

The major evidence in support of the theory is derived from phylogenetic data which indicate that there is a positive correlation between species' sleep time and the security of their sleeping arrangements, and a significant negative correlation between species' sleep time and the amount of time required for food gathering (Allison and Van Twyver, 1970).

As stated earlier (Chapter 2, pp.28-29) Meddis (1975) does not consider that SWS has any vital metabolic function. However, the behavioural function postulated by the immobilization theory, like the cerebral restitution theory, predicts no effect of fitness, exercise or any form of increased daily metabolism on SWS in man.

Conclusion

The experiments on the effects of physical fitness on both EEG and hormonal aspects of sleep are of particular theoretical interest as they potentially provide information regarding the functional significance of SWS. Those theories which hypothesize a relationship between peripheral metabolism and sleep, i.e. restorative and energy conservation theories, predict that physical exercise would promote SWS and sleep related anabolic hormones. It would be further expected that chronic exercise would result in sustained elevations in these aspects of sleep. As yet, however, the literature is inconclusive and does not unequivocably support any theoretical position.

CHAPTER 3.

ENERGY METABOLISM AND PHYSIOLOGICAL RESPONSES TO EXERCISE AND TRAINING

The interpretation of the experiments on sleep and fitness within the restorative and energy conservation theories of sleep are dependent on the present knowledge of exercise physiology. Thus an understanding of the consequences of exercise, both acute and chronic, for energy metabolism and synthetic processes is valuable. An outline of these areas is presented in this chapter. Since the effects of fitness on sleep is a dominant issue in this series of experiments, the nature and rationale of aerobic fitness assessment will also be described.

Energy Metabolism

"The physiology of muscular work and exercise is basically a matter of transforming bound energy into mechanical energy" (Åstrand and Rodahl, 1977). The source of the energy is the chemical bonds within the carbohydrates, fats and proteins of digested food. These nutrients are metabolized via different pathways to produce the energy rich compound ATP from ADP, phosphate (P) and energy. When ATP reacts with water (hydrolysis) the outermost phosphate bond is broken resulting in ADP, P, and energy. ATP is the immediate origin of energy for all the biologic work of cells.

$$ATP + H_2O \rightarrow ADP + P + 7.3 \text{ kcal per mole.}$$

Only relatively small amounts of ATP are present in the cell

at any one time. However, energy to recreate ATP from ADP may be stored in the cell in the high energy phosphate bond of creatine phosphate (CP).

$$CP + ADP \rightarrow ATP + C$$

Thus there is a cycle of energy transforming activities in the cell; 1. to form and conserve ATP from the potential energy in food and 2. to use the chemical energy in ATP for biologic work (McArdle, Katch and Katch, 1981).

Carbohydrates are the primary source of energy for cellular work. Initially they are broken down to glucose in the liver. Some glucose is stored as glycogen or converted to fat in the liver, while the remainder is transported to tissue cells where it may be used immediately in energy production or stored as glycogen. There are two phases of glucose degradation in the cell. The first involves the breakdown of a glucose molecule to two molecules of pyruvic acid (glycolysis). Oxygen is not required for glycolysis. Pyruvic acid may be further degraded, releasing energy for ATP formation with or without the use of oxygen. The former case is described as aerobic and the latter anaerobic metabolism.

In aerobic metabolism pyruvic acid is converted to acetyl-CoA which is then degraded in the Krebs' cycle, carbon dioxide and hydrogen atoms being the end products. The Krebs' cycle involves ten different chemical reactions and oxygen is required for each of them. The main purpose of the cycle is to form hydrogen atoms which are then oxidized to liberate large quantities of ATP. All the chemical reactions beyond the conversion to acetyl-CoA that require oxygen occur in the

mitochondria, where all the necessary enzymes are present. The oxidation of the hydrogen atoms produce about 90% of the ATP formed in aerobic metabolism. About 56% of the energy formed, during the breakdown of glucose is used for oxidative processes and released as heat. The remaining ATP formed is available to provide energy for cellular work (Berger, 1982).

When oxygen is not available pyruvic acid may be converted to lactic acid releasing energy for the formation of ATP. This occurs in the sarcoplasm of the cell. The energy yield is relatively low however, when compared to aerobic metabolism and when the accumulation of lactic acid becomes too high the process ceases.

Fat is the body's major source of potential energy. Some fat is stored in all cells but the main reservoir is adipose tissue. Transfer of fat from adipose tissue to active tissues is achieved by its conversion to free fatty acids (FFA) which diffuse rapidly into the circulation and are transported to active cells for energy metabolism. Within the active tissues, fatty acids are degraded to acetyl-CoA which then enters the Krebs' cycle and follows the same pathway as the aerobic metabolism of carbohydrates. Fats are both a readily available and rich energy source.

Protein is a relatively minor source of energy under normal circumstances. To provide energy, protein is transported to the liver where the nitrogen group is removed (deaminated) and the remainder is converted to glucose (gluconeogenisis). The glucose is then released into the blood and delivered to cells requiring energy. The protein alanine appears partic-

ularly important in this form of energy metabolism (McArdle et al., 1981).

The chemical reactions involved in energy release, as with most other cellular activities, are facilitated by the presence of highly specific protein catalysts, or enzymes. By regulating enzyme synthesis and breakdown, cells can regulate the rate of their metabolism. The major factor controlling energy production in cells is the relative balance between ADP and ATP, or, as described earlier (Chapter 2, p. 19) the energy charge (EC) of the cell. Thus if ADP levels are low and ATP high, i.e. a high EC prevails, low rates of energy metabolism will exist, as there is an excess of energy available for immediate cellular activities. On the contrary, if ADP is high and ATP low, i.e. EC is low, there will be high rates of energy metabolism to restore cellular energy supplies and to supply immediate cellular requirements. It will be recalled (Chapter 2, p. 19) that high EC conditions are conducive to protein synthesis, an energy demanding activity, as surplus energy is available to channel in this direction after more immediate cellular requirements have been met. Adam and Oswald (1977) point out that high EC conditions prevail during sleep and especially during SWS as general demands on energy supplies are then at their lowest. As a consequence they propose that there is an optimal energy environment for anabolism during SWS and thus, assuming substrates are available and end products in short supply, protein synthesis will be promoted (Adam, 1980; Adam and Oswald, 1977).

Energy Transfer During Exercise

Exercise requires very high levels of energy. For different kinds of exercise and at different times during exercise the main energy providing system relied upon by muscles varies. At the very beginning of exercise, or in very short duration high intensity exercise, muscles use the energy already available in cells in ATP and CP. At the commencement of even low levels of exercise there is a time lag between the demand for oxygen for energy metabolism, and its supply. Consequently during this period energy is produced anaerobically. Exercise relying on anaerobic metabolism could only be sustained one or two minutes as lactic acid builds up and prevents further metabolism of this kind. However, during the first minutes of exercise oxygen supply rises rapidly until the energy produced aerobically meets the energy requirements of the cell. At this point oxygen consumption plateaus and a 'steady rate' of oxygen consumption is attained.

When exercise is very strenuous aerobic metabolism again cannot provide all the energy required and anaerobic metabolism becomes substantial. This situation, however, cannot be maintained for more than a few minutes. Even steady rate exercise cannot be maintained indefinitely despite oxygen supply being adequate as other factors (e.g. fluid loss and depletion of electrolytes and energy reserves) intervene to reduce the capacity for exercise.

Physiological Responses During Exercise

Many physiological changes occur during exercise to

facilitate energy metabolism within working cells. They are directed primarily towards mobilizing energy stores, the supply of oxygen for aerobic metabolism and the removal of waste products which inhibit metabolism. Important among these responses are those related to neuroendocrine, cardiovascular and respiratory functioning.

Neuroendocrine Responses : A general arousal of the sympathetic nervous system occurs during exercise with the effect of quickening heart rate, vasoconstriction of blood vessels in the abdomen but vasodilation of coronary blood vessels, mobilization of liver glycogen, secretion of epinephrine in the blood and increased sweating, amongst other responses (Thomas, 1975).

Hormonal changes during exercise are particularly important in the mobilization of energy supplies. Secretion of cortisol results in increased blood glucose by promoting the breakdown of protein in muscle cells, and subsequent gluconeogenesis in the liver. In addition, cortisol facilitates the removal of FFA from adipose tissue into the blood, making available to muscle cells an alternative energy source than glycogen. The catecholamines also affect the metabolism of carbohydrates and fats. Norepinephrine is a particularly powerful stimulator of FFA mobilization and is secreted both during short, intense and prolonged moderately heavy muscular work. Again the mobilization of FFA spares blood glucose supplies. At the same time, the catecholamines activate the breakdown of glycogen to glucose in the liver and skeletal muscles, for energy metabolism. Catecholamines are

also the neurotransmitter substances involved in the sympathetic nervous system and their neural secretion results in the sympathetic nervous system arousal described above.

Growth hormone, which is important in controlling normal growth and metabolism (see Chapter 2, pp.19-20) is secreted in response to exercise, especially at a moderate level. When blood glucose levels are low it stimulates processes to elevate them. It stimulates the breakdown of adipose tissue, increasing levels of FFA available to cells for energy metabolism. At the same time growth hormone inhibits the entry of glucose in the cells and therefore exhibits a glucose sparing function. Several other hormones are released during exercise. Aldosterone increases blood pressure and insulin regulates glucose levels. Thyroxine turnover rates are increased though the specific function of this during exercise is unclear. Finally, androgens, especially in males, are secreted in response to power training. One of these, testosterone, causes muscle tissue to increase in size by stimulating amino acid uptake, and also causes bones to thicken (Berger, 1982).

Respiratory and Cardiovascular Responses : Sources of energy, oxygen, waste products and heat are carried to or from active cells in the blood. The respiratory and cardiovascular systems therefore adjust to meet the elevated demand for their supply and removal. To facilitate the movement of larger quantities of oxygen into the blood, and carbon dioxide out of it, pulmonary ventilation is increased by elevations in tidal volume and/or respiratory

rate, depending on the severity of the exercise. During aerobic exercise, there is also greater diffusion between blood and air of oxygen and carbon dioxide such that the proportion of the two gases in the blood stays much the same despite changes at a cellular level. This is probably the result of an increased number of pulmonary capillaries being opened to the alveoli (Berger, 1982).

To supply the demands of working cells the amount of blood circulating to them is itself increased. To this end, heart rate (HR) increases due to a reduction in stimulation of the vagi nerves to the heart and an increase in sympathetic nervous activity. Sympathetic cholinergic stimulation causes a dilation of arterioles in the periphery, increasing their blood flow, while sympathetic adrenergic stimulation results in constriction of the blood vessels in the viscera. Veins also constrict. In the exercising skeletal muscles, blood flow is increased as a direct result of arteriolar dilation and the opening of capillaries within the active tissue. These effects are caused by the metabolic activity of the cells themselves and are probably related to the local changes in pH, oxygen, carbon dioxide and lactate levels, and potassium and hydrogen ions. This local control of blood flow is the most important factor ensuring adequate blood supplies specifically to the exercising muscles. In addition, there is vasodilation of the superficial venous and arterial blood vessels to divert blood transporting excess heat to the skin, thus facilitating heat loss by radiation and conduction from the skin and by evaporation through sweating. The major consequence of the cardiovascular changes, plus the pumping action of working muscles

and respiratory movements, is a greater circulation of blood to exercising muscles and the periphery at the expense of the viscera (Åstrand and Rodahl, 1977).

Recovery from Exercise

While the body is recovering from exercise, metabolic rate remains elevated. There are two factors which act to maintain metabolic rate above normal levels for a considerable period following moderately intense exercise; the use of energy to actively restore pre-exercise conditions; and the stimulating effect of exercise itself which requires time to dissipate.

Restoration and Repair Following Exercise : The following processes of restoration and repair are most notable.

1. There is a resynthesis of ATP and CP within cells to meet future energy demands, a large proportion of which occur in the first minutes after the termination of exercise.

2. Readily available oxygen supplies are replenished. There is resaturation of oxygen in tissue water, venous blood, blood in muscle and myoglobin in muscle cells. About one half of the total resaturation of oxygen occurs within 30 secs of the termination of exercise (McArdle et al., 1981).

3. There is redistribution of calcium, potassium, and sodium ions which may take a few minutes (Simonson, 1971).

4. Lactic acid is removed by oxidation and resynthesis into glycogen. After exhausting exercise, blood and muscle lactate can return close to resting levels within an hour (Åstrand and Rodahl, 1977).

5. Following the consumption of carbohydrates there is a restoration of energy stores in the form of glycogen and fat. Restoration of muscle glycogen may take 12-24 hrs following the termination of exercise with adequate carbohydrate intake.

6. Proteins are resynthesized and damaged tissue repaired. Tissue repair may take a considerable time as, for example, damage indicated by muscle soreness may take several days to repair (Åstrand and Rodahl, 1977; McArdle et al., 1981; Simonson, 1971). It is in this last anabolic aspect of recovery from exercise that Adam and Oswald (1977) propose sleep has a role. They argue that SWS is a time of intensified rates of protein synthesis as there are few competing demands for energy, and hormonal conditions are favourable. Adam and Oswald (1977) suggest that when anabolic requirements increase as a consequence of protein catabolism and tissue damage during exercise, the period of optimal conditions for protein synthesis is extended. Thus the time spent in sleep, and particularly SWS is increased.

As all the restoration activities just described require oxygen, there is, even after exercise, an elevated load on respiratory and cardiovascular activity. Consequently the heart and respiratory muscles continue working above resting levels and therefore also require higher levels of oxygen than prior to exercise (McArdle et al., 1981).

The Residual Effect of Exercise on Metabolic Rate : Exercise has a stimulating effect on metabolic rate for two further reasons.

1. Neuroendocrine factors which stimulate catabolism during exercise may remain predominant for a period of a few hours following exercise (Berger, 1982; McArdle et al., 1981).

2. Body temperature may rise 2 or 3°C during vigorous exercise and may also take several hours to return to normal. The elevation in temperature catalyzes the rate of chemical reactions in the body and there is consequently an increase in metabolic rate (Berger, 1982). It is this rise in body temperature during exercise which Horne (1981) and Horne and Staff (1983) suggest stimulates subsequent rises in SWS, as it also produces an elevation in brain metabolism. They propose this triggers an energy conservation response or, alternatively, requires compensatory brain restitution, conditions which may be associated with SWS increases (See Chapter 2, pp. 38-39).

Physiological Adaptations To Training

Different sports rely predominantly on different aspects of physiology and different energy pathways. In terms of muscular development and energy metabolism, sports can be divided into three broad categories. Firstly, there are sports which depend primarily on muscular strength and power. They also require energy to be readily available in the muscle cells in the form of ATP and CP. These activities involve brief (up to 6 secs) but intense bursts of energy as in putting the shot, weight lifting, high jumping and sprinting. Secondly, there are sports which mainly use anaerobic energy pathways, and muscle strength though to a lesser extent than the first category. These include a

200-400 metre dash and 100 metres swim. Activities in this category may be sustained up to 1½ mins. Finally, there are those sports which depend on aerobic metabolism and endurance and to a minor extent on muscular strength, e.g. distance running (Åstrand and Rodahl, 1977; McArdle et al., 1981).

Power Training : Training in a specific exercise results in adaptations in the particular aspects of physiology of primary importance in the performance of that exercise. The main effects of training in exercise requiring brief bursts of power and strength are structural changes in muscles, bones, ligaments, cartilege and tendons which increase a muscle's ability to generate tension. Largely in response to the testosterone secreted during heavy work muscle fibers become larger (hypertrophy) due to increases in the protein that constitutes the contractile elements (myofybrils) (McArdle et al., 1981). Thus, myofybrils thicken and increase in number. There is particular development of fast-twitch muscle fibers which are high in ATP ase and glycolytic enzymes and are therefore, adapted to supply immediate energy via short-term non-oxidative energy pathways (Berger, 1982). In addition to changes in musculature there are increases in bone girth and density to meet higher demands for sturdiness; an increase in the thickness of cartilege which provides a better cushioning effect between joints; and an elevation in the inter-cellular substances of tendons and ligaments, enhancing their tensile strength (Åstrand and Rodahl, 1977; Berger, 1982). It would be consistent with the restorative theory

of SWS for people performing strength training to have high levels of SWS to provide extended periods conducive to these anabolic activities.

Anaerobic Training : Many of the changes resulting from anaerobic training are similar to those which occur in power training. In addition there are increases in the quantity and activity of key enzymes controlling the anaerobic phase of glucose catabolism, particularly in fast-twitch muscle fibers. Further, there is an increase in the capacity for levels of blood lactic acid during intense exercise. These factors contribute to the greater availability of energy during the first 1½ min of exercise and consequently facilitate performance of anaerobic sports (McArdle et al., 1981). As in power training, muscular development is important in most anaerobic exercise and thus the restorative theory of SWS would predict elevated levels of SWS during anaerobic training.

Aerobic Training : Since endurance sports depend almost entirely on aerobic energy the major effects of training are related to oxygen transport and utilization. At a cellular level, endurance training increases the capacity of muscle to oxidize pyruvic acid, carbohydrates and fatty acids to form ATP via aerobic metabolism, largely by the rise in the activity of mitochondrial enzymes, enabling an elevation in oxygen uptake. There is an associated increase in the number and volume of mitochondria (Vihko, Sarviharju, Havu, Hirsimaki, Salminen, Rahkila and Arstila,

1975). Aerobic training also results in a rise in myoglobin in skeletal muscle. It appears that myoglobin is involved in oxygen transport through the cytoplasm and an increase in myoglobin facilitates oxygen transport through the cell to the mitochondria (Berger, 1982; McArdle et al., 1981). It has been estimated that approximately 50% of the increase in oxygen consumption due to training is owing to the increase in myoglobin and mitochondrial activity (Berger, 1982).

The cellular adaptations during aerobic training, in contrast to strength training, occur mainly in slow-twitch muscle fibers. These cells break down glucose relatively slowly compared with fast-twitch muscles, partly as they contract slower; are surrounded by more capillaries and are thus supplied with more glucose and oxygen; have more mitochondria and myoglobin enabling them to obtain most of their energy from aerobic metabolism; derive more energy from free fatty acids; and are less easily fatigued (Berger, 1982). Clearly, the accentuation of the development of these muscle fibers is adaptive for endurance performance.

Changes occur in the balance of the sources of energy and the efficiency of their metabolism during exercise as a consequence of habitual aerobic exercise. Thus the amount of fat used by muscle for energy, increases over that used by untrained, power, or anaerobically trained individuals. There is a greater release of free fatty acids from adipose tissue stimulated, in part, by higher levels of growth hormone secretion during exhaustive

exercise. There is also a greater ability of muscles to metabolize fat due to an increase in fat metabolizing enzymes. As a consequence, glycogen stores are conserved, reducing the likelihood of hypoglycemia during prolonged exercise (Berger, 1982). There is also, however, an increase in the muscle's ability to oxidize and store glycogen, again elevating the availability of energy (McArdle et al., 1981).

The increased efficiency in energy utilization during exercise is partly a function of changes in endocrine responses to exercise. In unfit people hGH secretion at first rises during moderately intense exercise but then declines as exhaustion is approached. In trained subjects hGH remains elevated as exercise becomes exhaustive (Hartley, 1975; Hartley, Mason, Hogan, Jones, Kochen, Mougey, Wherry, Pennington and Ricketts, 1972). Norepinephrine is lower in trained subjects at any absolute level of work than in untrained subjects. However, it is the same when work is described in terms of a percentage of VO_{2max} (Hartley, 1975). Similarly, cortisol secretion is lower at rest and during submaximal work in trained than untrained individuals, but in exhaustive work cortisol levels are about the same in the two groups (Berger, 1982).

In addition to changes in cellular metabolism, aerobic training causes changes in the respiratory and cardiovascular systems. The net effect is an improvement in the transport of oxygen and energy supplies to active cells and the removal of lactic acid, carbon dioxide and

heat. The major adaptations of this kind are summarized in the following section.

1. There is an increase in the capacity of oxygen and carbon dioxide diffusion between the alveoli and capillaries. This is due primarily to an increase in lung volume and the consequent increase in the surface area between the alveoli and capillaries.

2. There is an elevation in blood volume and total haemoglobin, improving the amount of oxygen that can be provided to working muscles.

3. The weight and volume of the heart is greater following training, improving its ability to pump blood around the body.

4. The heart's stroke volume increases and associated with this is an elevation in maximal cardiac output.

5. During exercise, blood is more effectively diverted to working muscles.

6. Oxygen extraction by muscles is improved, and, as mentioned earlier, oxygen transport through the muscle cells is more efficient due to an increase in myoglobin.

7. Finally, as a consequence of these adaptations which improve the efficiency of oxygen transport from the lungs to the mitochondria, the heart is not required to pump at such a high rate during submaximal exercise to provide adequate levels of oxygen. Thus heart rate at any specific work load (including rest) is lower following aerobic training (Åstrand and Rodahl, 1977; Berger, 1982;

McArdle et al., 1981; Vander, Sherman and Luciano, 1980)

While aerobic training does not involve the same degree of skeletal development as power and anaerobic training, the cardiovascular and respiratory adaptations of endurance training require heightened protein synthesis. It would, therefore, be in accordance with the restorative theory of SWS if endurance athletes also had higher levels of SWS than sedentary individuals.

Training and Recovery from Exercise :

The rate of recovery from the specific exercise for which an individual is trained is more rapid than in unfit individuals (Åstrand and Rodahl, 1977). The adaptations which promote energy production during exercise also enable a faster replacement of oxygen and diminished energy sources following exercise. Similarly, lactates, other metabolites (Simonson, 1971) and excess body heat (McArdle et al., 1981) are removed more rapidly by athletes than non-athletes. Consequently, the return to pre-exercise conditions is generally faster in trained individuals.

It would be consistent with the restorative theory of SWS if SWS levels were chronically higher in trained than untrained individuals to meet the higher demands for anabolism created by the higher daytime catabolism and tissue damage. Thus chronically high levels of SWS may facilitate recovery from exercise in people who habitually exercise.

Training and Body Composition

Normative studies of body composition factors indicate that total fat usually accounts for 15% and 27% of body fat for young adult males and females respectively (Behnke and Wilmore, 1974; Durnin and Womersley, 1974). Behnke and Wilmore (1974) estimate that, in males, 3% and in females, 12% fat can be regarded as essential fat without which physiological well being is jeopardized, while the rest is storage fat used for energy reserves. For males and females respectively, muscle accounts for approximately 45% and 36%, bone for 15% and 12% and the remainder, 25% and 25% of total body mass (Behnke and Wilmore, 1974). Total body mass less total fat is known as lean body mass (LBM). Training, however, generally has the effect of reducing levels of body fat. It is usually true to say that physically active individuals have a higher proportion of LBM to fat than physically inactive individuals of the same age, height and weight (Pařizková, 1977; Pike and Brown, 1967) and that training programmes result in increased LBM (Boddy, Humes, King, Weyers and Rowan, 1974; Bjorntorp, 1980; Gwinup, 1975; Leon, Conrad, Hunninghake and Serfass, 1979).

The precise effect of training on body composition, however, as with other physiological factors, depends on the nature and intensity of the training. Thus endurance training tends to reduce body fat considerably to enhance heat dissipation and to lower the amount of unproductive mass to be carried (McArdle et al., 1981). The percent fat for Olympic marathon and distance runners is generally around 3% for males and 15% for females (McArdle et al., 1981).

At the other extreme, in some sports both weight and muscular strength are an advantage and under these circumstances percent fat may be considerably above average. Olympic weight throwers (shot, discus and hammer) have body fat levels of around 30% for both males and females (McArdle et al., 1981). Weight as well as strength is also an advantage in body contact sports like Australian Rules Football, American Football, and Rugby. In these sports weight is particularly relevant for particular positions in the team. For example, the mean body fat of 32 professional defensive linemen has been shown to be 18.2% (Wilmore , 1976). As indicated by the body compositions of male Olympic athletes, the optimal level of fat for sprinters is approximately 10%, for swimmers 12%, for basketballers 13% and for rowers 14% (McArdle et al., 1981).

Energy Expenditure During Rest and Exercise

Resting Metablic Rate : The minimum level of energy required to sustain the body's vital functions is known as the basal metabolic rate (BMR) or, more recently resting metabolic rate (RMR). This terminology shift has reflected the fact that in practice basal conditions can never be completely achieved. The term RMR will be used throughout this thesis and is generally assessed when a person is awake but resting in an environment at thermoneutrality after 12 hrs fasting (Dauncey, 1979). Generally oxygen consumption is measured to determine the rate of energy expenditure

(indirect calorimetry). The alternative method used is to measure the body's heat output (direct calorimetry) (Dauncey, 1979).

The RMR of the average man and average woman is approximately 1.1kcal x min^{-1} and 1.0 kcal x min^{-1} (Åstrand and Rodahl, 1977). Most of this energy is expended by the heart, kidney, liver and brain (Vander et al., 1980). Differences in RMR between individuals are largely related to four factors; body composition, age, sex and dietary influences.

Generally, larger individuals have higher RMR's than smaller ones. This reflects, however, the greater amount of fat free tissue or LBM, rather than a higher RMR per kilogram. Greater physical size will not be associated with a higher RMR if the additional mass is adipose tissue as this is metabolically quite inactive and therefore contributes little to the energy expenditure of an individual (McArdle et al., 1981).

RMR declines with increasing age. It is particularly high in children relative to their size due to the large amount of energy used in the formation of new tissue (Berger, 1982). In adults the age related decline in RMR is unrelated to the activity levels of specific tissues. Rather, it is associated with a decrease in the proportion of LBM to body fat which occurs with age (Keys, Taylor and Grande, 1973; Tzankoff and Norris, 1977). A similar factor accounts for females usually having lower RMRs than males even when body weight is taken into account. Thus females have a considerably higher percentage of

inactive fat tissue than males (McArdle et al., 1981).

One factor which influences RMR per kilogram is energy balance. Underfeeding has been shown to result in a decline in RMR, presumably as an adaptation to conserve energy (Dauncey, 1979; Garrow, 1974; Sims, 1976). There is also much speculation that a high food intake causes an elevation in RMR. This effect, known as diet-induced thermogenesis, would provide for the dissipation of excessive energy supplies (Dauncey, 1979; James and Trayhurn, 1981; Miller, 1975; Rothwell and Stock, 1983; Sims, 1976).

Resting Metabolic Rate and Training : The effect of physical training on RMR has been examined in a number of studies which have been reviewed by Cureton (1971). Training does not consistently have any direct effect on RMR. However, it is possible that training has a number of indirect effects. Two possible indirect effects are those of body composition and energy balance.

As described earlier (Chapter 3, pp.58-59) training results in changes in body composition. Increases in LBM result in a proportionate increase in RMR (Thomson, Jarvie, Lahey, Cureton, 1982). It is also possible that the phase in a training programme may have consequences for RMR. Just as children have high RMRs associated with growth, during phases of training where high levels of tissue development are occurring (as in the earlier phases of weight training before growth plateaus), RMR may be elevated.

Intense training may indirectly lead to a reduction

in RMR by contributing to the creation of an underfeeding situation where energy intake does not meet the high energy output. There is some evidence to suggest that, in males, exercise has an appetite suppressing effect (Thompson et al., 1982) which may also produce an underfeeding situation and thus a reduction in RMR. This is purely speculative, however, and there is no firm evidence to show that any kind of training affects energy balance and consequently, RMR.

Total Energy Expenditure, Exercise and Training : As implied above, physical activity results in a great increase in energy expenditure compared with resting levels. In fact, most people are able to increase metabolic rate by ten times resting levels by exercising (McArdle et al., 1981). The energy cost of exercise can be seen in Table I which shows the approximate amount of energy required by a person weighing 70 kgs to perform a range of physical activities. Clearly if moderate exercise is performed for a sustained period there can be a large increase in total daily energy expenditure.

McArdle et al., (1981) estimate that the average American male, aged 19-22 yrs expends 3,000 kcals a day while the average female in the same age range expends 2,100 kcals a day. By comparison, however, elite male athletes in sports like cross-country skiing, distance running or swimming, soccer and basketball, may expend in excess of 5,600 kcals a day (McArdle et al., 1981). Clearly, athletes in training will have consistently high levels of energy expenditure. Over time, as proposed by

TABLE I

Energy required to perform a range of physical activities by a person weighing 70 kg (Morehouse and Miller, 1976).

Activity	Energy Expenditure kcal x min^{-1}
Sleeping	1.21
Lying awake	1.30
Sitting	1.67
Standing	1.83
Walking (3.5 mph)	4.83
Bicycling (rapid)	6.92
Running (5.7 mph)	12.00
Running (11.4 mph)	21.67
Running (15.8 mph)	65.17

the energy conservation theory of sleep, such high levels of total energy expenditure may provoke a compensatory energy conservation response in the form of elevated SWS or TST to assist in the maintenance of energy balance.

Assessment of Aerobic Fitness

Direct measurement of performance on any particular physical task generally indicates the level of proficiency in that task. However, performance is influenced by factors other than physiological potential, such as motivation and tactics (Åstrand and Rodahl, 1977) so tasks of performance may inaccurately estimate physical ability. To avoid this problem and for laboratory convenience, measures of aerobic power, or fitness, based on oxygen consumption levels have been devised.

Tests of aerobic fitness of this kind are based on the fact that ability to perform aerobic work is limited by the amount of oxygen per kilogram which can be taken up by the body in a given period of time. People who can potentially uptake large amounts of oxygen per body weight have a greater capacity to perform aerobic exercise than those who have a lower maximal oxygen uptake (VO_{2max}). Consequently VO_{2max} (expressed either in litres/min or millilitres/kilogram x minute) provies an indication of aerobic fitness.

VO_{2max} can be assessed by directly measuring oxygen uptake at increasingly high work loads until oxygen consumption no longer increases despite further elevations

in work load. It may also be indirectly estimated by the measurement of heart rate at a submaximal work load. This is possible for several reasons.

1. Oxygen uptake is linearly related to heart rate.

2. Oxygen uptake (and therefore heart rate) is linearly related to level of work load, when the work load is submaximal.

3. For a particular age and sex maximal heart rate does not vary greatly.

Thus, if heart rate is measured at a submaximal work load an extrapolation can be made to determine the work load which could be performed at a maximal heart rate and the VO_{2max} this would represent. Åstrand (1960) has devised a nomogram to facilitate the estimation of VO_{2max} by this method. The VO_{2max} value attained using the nomogram should then be corrected for the age and weight of the subject (Åstrand and Rodahl, 1977).

Estimation of VO_{2max} from heart rate is not as accurate however, as direct measurement of oxygen consumption. There are a number of sources of error.

1. The increase in heart rate with increase in oxygen uptake is not always exactly linear.

2. The subject's maximal heart rate may differ from the value used on the basis of normative data.

3. The mechanical efficiency on the particular exercise task (usually bicycle ergonometer or treadmill) may vary slightly between subjects (Åstrand and Rodahl, 1977).

Finally, it should be noted that VO_{2max} level assessed using a submaximal work test does not necessarily indicate a person's state of training as constitutional factors play an important role in determining VO_{2max} (Åstrand and Rodahl, 1977).

CHAPTER 4.

EXPERIMENT I : AEROBIC FITNESS AND SWS

Baekeland and Lasky (1966) commented on the unusually high levels of SWS of the physically fit athletes used in their experiment on the effects of exercise on human sleep. Similar findings were later reported by other authors (Buguet et al., 1980; Zloty et al., 1973) who also used fit subjects in investigating the immediate effects of exercise. Further suggestion that fitness or a related factor may influence sleep came from two studies in which young aerobically fit athletes were found to have more SWS than unfit sedentary controls (Griffin and Trinder, 1978; Trinder et al., 1982a). In addition to differences in SWS, aerobically fit individuals have been reported to have more NREM sleep (Walker et al., 1978), longer sleep durations and shorter sleep onset latencies (Montgomery et al., 1982). In contrast, however, high levels of SWS were not observed in young fit athletes by Paxton et al. (1982), nor in a slightly older group of fit subjects (mean age = 31.8 yrs) (Trinder et al., 1982a), nor in a sample of middle distance runners compared with a non-athletic control group (Walker et al., 1978).

The inconsistency in the observation of a fitness effect suggested the hypothesis that aerobic fitness was not a critical factor, but rather, in some experiments had been confounded with a variable associated with physical fitness, or with athletic activities in general. As each of the experiments reported above used independent group designs they would not have identified relatively permanent characteristics of individuals which might predispose them to athletic activities,

or be a consequence of a long period of athletic activity. The main aim of this experiment was to eliminate these variables by using a within subject design. Thus, the sleep of a group of proficient athletes was assessed on two occasions. It was assessed first when the athletes were aerobically unfit after an enforced non-training period, and second, when they were physically fit following training. The sleep of the athletes was also compared with that of an unfit, non-athlete, sedentary control group. It was hypothesized that if aerobic fitness was a critical factor in determining SWS levels, there would be no difference between the amount of SWS of the unfit athletes and the unfit non-athletes. However, it would be anticipated that the SWS of the athletes would increase as a function of aerobic fitness and therefore be higher on the second occasion of testing than SWS levels for either unfit athletes or unfit non-athletes. In contrast, if the relevant variables in determining SWS level are more enduring characteristics of athletes, their SWS level would be independent of aerobic fitness, though presumably different from the non-athlete control group.

It has been proposed that the observation of a facilitative effect on SWS of a single exercise session - an exercise effect, is dependent on the subjects being physically fit (Griffin and Trinder, 1978). In accord with this view the exercise effect has been confined to experiments using fit subjects (Baekeland and Lasky, 1966; Maloletnev and Telia, 1976; Maloletnev et al., 1977; Shapiro et al., 1975; Shapiro and Verschoor, 1979). In addition, an exercise effect has never been observed in experiments using untrained

subjects (Adamson et al., 1974; Browman, 1980; Browman and Tepas, 1976; Desjardins et al., 1974; Hauri, 1968; Horne and Porter, 1975; Zir et al., 1971). Griffin and Trinder (1978) found a significant interaction between level of fitness and exercise for amount of Stage 3 sleep. A number of experiments, however, using fit subjects, have failed to find an increase in SWS following exercise (Bonnet, 1980; Buguet et al., 1980; Paxton et al., 1982; Walker et al., 1978). Further, only two experiments (Griffin and Trinder, 1978; Walker et al., 1978) have compared the effect of exercise on sleep in both trained and untrained subjects within the same experiment, with one (Griffin and Trinder, 1978) providing tentative support for the hypothesis and the other (Walker, et al., 1978) finding negative results.

No experiment has examined the interaction between exercise and degree of fitness within subjects. Thus a second aim of this experiment was to investigate the hypothesis that an exercise effect on SWS is dependent on the level of fitness of the individual by assessing sleep in athletes, first when unfit and later when fit in two different exercise conditions, a no exercise control and a moderate afternoon exercise condition. The sleep of the unfit and fit athletes in the two exercise conditions was also compared with the sleep of an unfit non-athlete control group following a no exercise and a moderate afternoon exercise condition.

METHOD

Subjects and Design

The sleep of two groups of subjects was assessed. One group of eight male subjects (mean age = 20.25 yrs; SD = 1.85) was selected on the basis of athletic ability (athlete group), while the other group of nine male subjects (mean age = 20.67; SD = 1.63) consisted of sedentary, non-athletic individuals (non-athlete group). The athlete group was tested at a time when they were physically unfit and subsequently when fit. Six of the nine subjects in the non-athlete group were tested twice to control for any sequential effects. Subjects in this group were unfit on each occasion. One adaptation night and four non-consecutive experimental nights were run at each phase of the experiment. Two of the experimental nights followed afternoon exercise and two were non-exercise days.

Athletes were initially selected according to three criteria : a high achievement level in their selected sport; at least 6 months abstinence from competition and training (in general subjects had missed one season due to study commitments, injury or overseas travel); and a VO_{2max} value of less than 44 ml/kg x min. The subjects were largely participants in team sports such as field hockey, rowing and the various football codes. The non-athletes were selected on the basis of a sedentary lifestyle and a VO_{2max} value of less than 44 ml/kg x min. All potential subjects were screened for medical disorders generally and sleeping difficulties in particular.

Thirteen athletes began the experiment. Five, however, were discarded when they failed to continue their training program. In one instance this was due to injury, in a second, illness, and in the remaining three cases the subjects failed to return for fitness tests. The two sleep assessment occasions were separated by 3.6 months for the athletes and 3.2 months for the six non-athletes.

Procedure

Aerobic fitness levels were assessed by a submaximal bicycle ergonometer test (Åstrand & Rodahl, 1977), in which heart rate (HR) at the end of 6 min of riding on the ergonometer at a specified, steady work load was used to estimate VO_{2max}. Average VO_{2max} values are shown in Table 2. The two groups were of similar fitness levels during the initial phase of the experiment. These values were comparable with those of unfit groups used in previous studies (Griffin and Trinder, 1978; Trinder et al., 1982a). Athlete subjects were preferred if they expressed an intention of returning to competition. Thus, the improvement in their fitness was achieved by the predominantly aerobic training programs required by their respective sports. During this period, each subject's fitness was regularly assessed and the results graphed and shown to subjects at each visit. The sleep of the athletes was reassessed when their individual fitness levels asymptoted, though all subjects were given a minimum target. The mean gain for this group was 15.31 ml/kg x min and 1.1 l/min. The fit values for the athletes were equivalent to that of fit groups in previous studies (Griffin

TABLE 2.

Average physical fitness values for athletes and non-athletes on the first and second occasions of testing. Standard deviations are shown in brackets. On the 30 steps/min Step Test five subjects in both the athlete unfit and non-athlete unfit groups reached a representative HR of 180 bpm during the test, at which point testing was terminated. Therefore mean HR data is unavailable and variance data is not meaningful.

	Athletes		Non-Athletes	
	Unfit (1st Test) n = 8	Fit (2nd Test) n = 8	Unfit (1st Test) n = 9	Unfit (2nd Test) n = 6
Bicycle Test				
VO_{2max} 1/min	2.86 (.39)	3.96 (.58)	2.56 (.45)	2.53 (.41)
VO_{2max} ml/kg x min	37.88 (2.59)	53.19 (4.55)	38.17 (4.67)	38.92 (4.52)
Step Test - 20 stp/min				
HR	158.4 (15.68)	135.63 (10.80)	161.1 (114.70)	162.3 (15.08)
Step Test - 30 stp/min				
HR	180+	162.75 (12.73)	180+	180+

and Trinder, 1978; Trinder et al., 1982a). As can be seen from Table 2, the average fitness levels of the non-athletes on the two occasions of testing was essentially unchanged.

In addition to the bicycle ergonometer test two supplementary fitness assessment procedures were administered. They were two step tests in which HR was measured while subjects stepped onto and down from a 40cm step at a rate of 20 steps/min and 30 steps/min for 5 min. These tests were given to all subjects on each occasion that their sleep was assessed. Mean HR values for each group are presented in Table 2. The data confirmed the effects observed in the bicycle ergonometer test.

The subjects' weight, height, lean body mass (LBM) and fat were measured on each of the two testing occasions. The Quetelet's Index (weight/height^2 x 100) (Khosla and Lowe, 1969) was also computed. Mean values for these variables are shown in Table 3. Height data was unavailable on two unfit subjects and LBM information was unavailable on one unfit subject. LBM and fat were determined according to procedures described by Durnin and Womersley (1974) from skinfold measurements at four sites, biceps, triceps, subscapular and suprailiac. These measurements provided an assessment of the effects on body composition of both group differences and the training program in the athletes. However, it was not possible to measure other factors such as lifestyle, or diet, which may have differed as a function of groups, or training.

On each testing occasion subjects slept in the sleep

TABLE 3.

Anthropometric measures for the unfit non-athletes on the first occasion of testing and the athletes when they were both unfit and subsequently fit. Standard deviations are shown in brackets.

	Athletes		Non-Athletes
	Unfit (1st Test) n = 8	Fit (2nd Test) n = 8	Unfit (1st Test) n = 9
Weight (kg)	75.4 (9.4)	75.1 (8.4)	67.0 (6.4)*
Height (cm)	180.6 (8.5)	180.6 (8.5)	180.1 (2.4)
Quetelet's Index	.2307 (.0204)	.2301 (.0185)	.2022 (.0173) *
LBM (kg)	63.0 (6.1)	63.2 (6.1)	58.2 (5.6)
Fat (kg)	12.4 (4.1)	11.9 (3.4)	8.8 (3.6)
% LBM	83.6 (3.5)	84.2 (3.0)	86.9 (4.4)

* Athletes significantly higher than non-athletes ($p < .05$).

laboratory on five non-consecutive nights, all within a two week period. The first was an adaptation night. The four experimental nights consisted of two nights following afternoon exercise and two following non-exercise days arranged in an ABBA design. Athelete and non-athlete subjects were run simultaneously, and further, the study took over 12 months to complete. As a consequence circannual effects would not have influenced the results.

The exercise for the athletes consisted of their usual training program at an intensity sufficient to be exhausting but not stressful. The exercise performed by the non-athletes was a supervised run of between four and six km depending on their individual capacities. The intent of the method used to determine the level of exercise was for the subjects to maximise the amount of exercise without it becoming sufficiently stressful to disturb sleep. It was thought that when only a single exercise level is to be used, this could be most effectively achieved by having the subject determine this level, though the procedure does have the disadvantage that it is not quantifiable. The aims of the procedure were explained to each subject.

Subjects went to bed at their usual bedtime and were awakened at their usual rising time. The sleep records were obtained and scored blind according to standardised procedures (Rechtschaffen & Kales, 1968). The two scorers had an inter-rater agreement of greater than 90 percent. Sleep variables were calculated according to the definitions proposed by Williams, Karacan and Hursch (1974). The exception was sleep onset which was defined as the first Stage 2 epoch. Sleep period time (SPT) was them computed from the first Stage 2 until

morning awakening. As a consequence, SPT and total sleep time (TST) have a variable relationship to each other depending on the amount of wake time during the night and Stage 1 before sleep onset.

RESULTS

Average SWS values for the four conditions are shown in Table 4. The data were analysed using a 2 x 2 x 2 ANOVA with repeated measures on the latter two factors. The factors were athletes vs non-athletes, exercise vs no exercise and the first vs the second occasion of testing. The sample size for the athletes was eight on both testing occasions. The sample size for the non-athlete group was nine with three missing values on the second occasion. The two identical nights within each cell were averaged before the statistical analyses were performed. The significance level was set at $p < .05$. (It should be noted that a second analysis using only those 6 subjects run on the second occasion gave identical results).

The data clearly indicate that the athletes had more SWS than the non-athletes irrespective of their level of aerobic fitness. The analysis showed a significant main effect of groups for both minutes of SWS ($F(1/15) = 7.06$, $MSe = 2833$) and SWS as a percent of TST ($F(1/15) = 5.42$, $MSe = 142$). Both Stage 3 and 4 contributed to the higher SWS in the athletes. The difference was significant for Stage 3 ($F(1/15) = 6.27$, $MSe = 631$) and approached significance for Stage 4 ($F(1/15) = 4.32$, $MSe = 1431$). The effect of testing occasion ($F(1/12) = .43$, $MSe = 232$) and the interaction ($F(1/12) = .30$, $MSe = 232$) were both non-significant for total SWS, though they were significant for

TABLE 4.

Average values in minutes for selected sleep variables as a function of experimental group and occasion of testing. Standard deviations are shown in brackets.

	Athletes				Non-Athletes			
	Unfit (1st Test) n = 8		Fit (2nd Test) n = 8		Unfit (1st Test) n = 9		Unfit (2nd Test) n = 6	
Time in bed	468	(15.8)	466	(30.2)	446	(35.7)	454	(36.0)
Sleep period time	448	(14.6)	448	(25.1)	416	(42.3)	426	(37.9)*
Total sleep time	443	(17.3)	448	(23.0)	420	(43.6)	430	(37.8)
Total time awake	25	(16.0)	18	(15.1)	26	(21.5)	24	(22.3)
Stage 1 + MT	41	(15.0)	37	(16.5)	47	(18.5)	34	(37.8)
Stage 2	197	(27.2)	196	(32.4)	199	(26.6)	201	(26.8)
Stage 3	38	(16.6)	46	(21.3)	27	(7.2)	28	(9.2)**
Stage 4	78	(11.4)	75	(21.5)	57	(25.5)	68	(21.4)
SWS (3 + 4)	116	(24.9)	121	(36.7)	84	(25.8)	96	(18.0)*
NREM (2 + 3 + 4)	313	(19.2)	317	(27.8)	283	(31.0)	297	(32.5)*
REM	89	(8.9)	94	(14.9)	90	(19.0)	99	(19.1)
Sleep onset latency	17	(12.2)	16	(10.7)	28	(20.2)	24	(20.7)
Stage 3 latency	12	(4.9)	11	(4.5)	14	(9.9)	12	(3.4)
REM latency	110	(39.5)	106	(30.8)	77	(24.1)	87	(27.7)*

* Main effect of groups significant ($p < .05$)

** Significant effect of groups, occasion of testing and interaction ($p < .05$).

Stage 3 ($F(1/12) = 4.69$, $MSe = 45$; $F(1/12) = 4.63$, $MSe = 45$ respectively). As can be seen from Table 4, Stage 3 was elevated in athletes when they were fit. However, Stage 4 showed a compensatory effect in the opposite direction. Because of the opposite effects found in Stages 3 and 4 the Stage 3 effect is not viewed as strong evidence for a fitness facilitation of SWS, particularly as it was Stage 3 which was elevated and Stage 4 which was suppressed.

Total NREM sleep (Stages 2 + 3 + 4) and sleep duration tended to be longer in the athletes. The effects were statistically significant for NREM sleep ($F(1/15) = 5.97$, $MSe = 2606$) and SPT ($F(1/15) = 4.67$, $MSe = 2975$) though not for TST ($F(1/15) = 2.68$, $MSe = 3214$) or TIB ($F(1/15) = 2.45$, $MSe = 2388$). Sleep onset latency was shorter in the athletes than non-athletes and showed the same pattern as SWS and total sleep duration, though the main effect of groups was not significant ($F(1/15) = 2.01$, $MSe = 910$).

With the exception of REM latency, which was elevated in the athletes ($F(1/15) = 7.72$, $MSe = 2024$), no other total night sleep variable was affected by any of the independent variables. As might be expected the longer REM latency in the athlete group was associated with a higher level of SWS in the first cycle of the night ($F(1/15) = 9.59$, $MSe = 891$). However, there were no significant effects as a function of the exercise, either over the whole night, or within the first cycle. Specific analyses of the exercise effect in the athletes when fit, also failed to show significant differences (122 and 119 minutes for exercise and non-exercise respectively).

As shown in Table 3, while athletes and non-athletes had almost identical mean heights, athletes weighed significantly more than non-athletes ($t(15) = 2.17$, $p < .05$). Consequently, athletes also had a significantly higher mean Quetelet's Index ($t(13) = 2.79$, $p < .05$). However, no differences in LBM and fat variables between the two groups were significant and there were no changes in body composition as a function of aerobic fitness in the athlete group.

DISCUSSION

The hypothesis that aerobic fitness was the critical factor contributing to elevated SWS levels in fit athletes reported in previous papers, was not supported by the results of this experiment. Athletes had more SWS than non-athletes regardless of their level of fitness. This finding supports the hypothesis that the difference observed in SWS between athletes and non-athletes is related to a more enduring variable. A similar pattern of results was observed for measures of sleep duration, in particular SPT, again suggesting the effect is a function of a more enduring variable.

While there are a number of possible candidates for such a factor e.g. genetic or dietary influences, a potential area for closer examination is suggested by this experiment. The athletes were significantly heavier although of a similar height, than the non-athletes. Also the athletes had a tendency to a higher proportion of fat to LBM. The possibility of a relationship between SWS and anthropometric variables was therefore examined in subsequent experiments.

It is notable that the failure to observe an effect of fitness itself on SWS is not consistent with a recent study in which SWS, TST, aerobic fitness and LBM were found to increase as a function of basic training in a sample of eight new army recruits (Shapiro, Trinder, Paxton, Oswald, Warren, Catterall, Flenley, East and Harvey, 1981). There were a number of important differences between the studies. These included the use of proficient athletes as opposed to an unselected, largely non-athletic group; the use of aerobic training compared with a more comprehensive programme in the case of the army recruits and differences in the method of measuring body composition. However, the most likely explanation was that since the baseline recording period was during the recruits first week in the army, SWS levels were depressed due to the stress of the situation. The magnitude of this possible stress effect could not be assessed as it was not possible to run the necessary control group. In view of these factors the results of this experiment should be treated with caution.

A facilitatory effect on SWS of exercise of moderate intensity was not evident in this experiment in either athletes or non-athletes, or within athletes as a function of degree of aerobic fitness. There is, therefore, no evidence in this experiment to support the hypothesis that the occurrence of an exercise effect on SWS is dependent on the subjects being physically fit. This conclusion is reinforced by the fact that a within subjects design was used in which exercise was given to subjects when they were both unfit and fit. It remains possible, however, that

fitness may increase the probability of an exercise effect occurring when one or more other conditions are met. A sufficiently high intensity of exercise, i.e. both a high rate and total output of energy expenditure, may be such a condition (Horne, 1981).

The exercise level used in the present study was selected to be equivalent to that used in previous experiments in which an exercise effect has been reported (Baekeland and Lasky, 1966; Shapiro et al., 1975). Subsequent to the completion of this experiment five studies have been reported using still more intense exercise (e.g. treadmill running to exhaustion and marathon running) in fit subjects. Three of these have found an exercise effect (Bunnel, Bevier and Horvath, 1983; Horne and Staff, 1983; Shapiro, Bortz, Mitchell, Bartel and Jooste, 1981). These studies suggest that the likelihood of observing the effect is a function of the intensity of the exercise (both rate and amount) and that physical fitness allows extreme intensity levels to be achieved (Horne, 1981). The level of exercise used in this experiment, though equivalent to others which found positive effects, may have been too low to reliably produce the effect.

Two of the recent experiments which used very intense exercise have not reported increases in SWS (Montgomery, Trinder, Paxton, Fraser, Meaney and Koebin, 1984; Torsvall, Akerstedt and Lindbeck, 1984). However, both of these studies used older subjects and reported stress effects as a result of the exercise. Thus, the sleep of

the subjects was characterised by an increase in total time awake, sleep onset latency and REM latency, and a reduction in total REM. In addition, cortisol secretion during the day and night of the marathon increased in the experiment of Montgomery et al. (1984) and catecholamine excretion during the sleep period and HR at bedtime and rising were elevated after a marathon in Torsvall et al.'s (1984) study. It is likely that, despite the high levels of fitness of the subjects, the interaction of age and exercise intensity produced a stress effect that offset a facilitatory effect on SWS. In this context it is important to note that Montgomery et al. (1984) did not find a SWS rebound on nights following the marathon when cortisol levels and sleep had returned to the baseline values.

Thus, a number of studies are consistent with an inverted U shape relationship between SWS level and exercise intensity (Chapter I, pp.13-14). However, it is unclear what determines the optional level of intensity, although recent experiments suggest it generally needs to be quite severe. The decline in SWS level may be determined by the degree of physiological stress. The weight of present evidence suggests that fitness is a necessary but not sufficient factor to produce an exercise effect. It appears exercise intensity is also an important factor, and, considering the illusive nature of the effect, it is probable that other specific conditions are required.

CONCLUSION

Differences in the nature of sleep in aerobically fit athletes compared with non-athletes have now been reported in a number of experiments (Griffin and Trinder, 1978; Montgomery et al., 1982; Trinder et al., 1982; Walker et al., 1978) though the SWS effect in particular is not always observed. The data in Experiment I indicate that these effects are not due to aerobic fitness per se, but rather to some more enduring characteristic of the subjects used. The significant differences in weight between the athletes and non-athletes in this experiment suggest that an examination of the effects of the body composition variables may reveal the nature of this characteristic. The specific determinants of an effect of exercise on SWS are unclear although they seem quite restrictive. While fitness may be a necessary factor it is insufficient alone to produce an increase in SWS following exercise. It is most likely that its role is to allow extreme levels of exercise to be carried out (Horne, 1981). It is also likely that extreme exercise disturbs sleep, an effect which is promoted by factors such as low fitness levels and age.

Experiment I pursued two issues; the effect of physical fitness on sleep and its interaction with exercise. Following the completion of Experiment I it was decided to pursue the former phenomenon and thus exercise was not included as a variable in subsequent experiments.

CHAPTER 5.

EXPERIMENT 2 : AEROBIC FITNESS AND NIGHT-TIME HORMONE SECRETIONS

In the second experiment, run concurrently with the first, the night-time secretions of the hormones hGH, prolactin and cortisol were examined in fit and unfit populations. The research in the area of sleep in athletes and non-athletes has concentrated on differences in the EEG manifestations of sleep. A number of factors however, indicated that the hormonal aspects of the sleep of these two groups could also be of particular interest.

Firstly, as described in Chapter 1.(pp.15-16) there is a tentative indication that moderate daytime exercise elevates hGH and decreases corticosteroid secretions during the night. These findings were reported by Adamson et al., (1974). In contrast, however, Zir et al., (1971) failed to find an increased peak hGH secretion, following moderate or light daytime exercise. If a single exercise session modifies sleep-related hormone secretions it might be anticipated that habitually high exercise levels, reflected by high fitness, would have a chronic effect on hormone levels during the night.

Secondly, training results in hormonal adaptations to exercise (Chapter 3., p.55). In particular, as a consequence of training hGH secretion remains at a high level during moderately intense exercise maintained to exhaustion. In contrast, in untrained individuals there is a marked decline in hGH as the exercise continues

(Hartley, 1975; Hartley et al., 1972). In addition, cortisol levels are lower following training both at rest and during submaximal exercise but not in exhaustive exercise (Berger, 1982). Since training influences hGh and cortisol secretions during exercise it would be of interest to examine if training also affects the night-time secretion of these hormones.

Finally, the close relationship early in the sleep period between the peak secretion of hGH, the trough in cortisol, and the occurence of SWS has been viewed as consistent with the restorative theory of SWS (Adam, 1980; Adam and Oswald, 1977; Oswald, 1980). The high level of SWS in fit athletes reported in a number of experiments (Baekeland and Lasky, 1966; Buguet et al., 1980; Griffin and Trinder, 1978; Trinder et al., 1982a; Zloty et al., 1973) has also been interpreted as consistent with this theory on the basis that the extra demands for anabolism created by training are met by increasing the amount of time in SWS when conditions are optimal for protein synthesis (Oswald, 1980). Similarly, it could be predicted that night-time hGH may be elevated and cortisol secretion decreased as a consequence of training to further enhance an environment favouring protein synthesis.

For the reasons described above, Experiment 2 was designed primarily to assess the relationship between physical fitness and the night-time secretion of hGH and cortisol. Assays for prolactin, whose peak secretion like hGH is sleep-related (Parker et al., 1980) were

also available. Thus, although not of central interest in this thesis, prolactin levels were analysed and are reported here.

In addition to these variables, LBM and fat levels were assessed in all subjects. The main reason for this was methodological as there is reason to expect that hGH, SWS and body composition may be interrelated. Sleep-related growth hormone is known to be low in the obese (Hunter, Friend and Strong, 1966; Kalucy, Crisp, Chard, McNeilly, Chen and Lacey, 1976; Mims and Lopez, 1976; Quabbe and Helge, 1967; Sims and Horton, 1968) and one report has shown it to be related to fat levels, as measured by height/weight ratios in a normal sample (Othmer, Levine, Malarkey, Corvalan, Hayden-Otto, Fishman and Daughaday, 1974). The same study found a moderate, though non-significant, negative correlation between hGH and SWS. Finally, the possibility that SWS levels may be influenced by body composition has been considered (Chapter 4). It has been proposed that the difference in SWS observed between fit and unfit populations may not be a consequence of fitness but rather some related, more endurable variable like body composition.

In view of the potential interrelationships between these variables, for methodological reasons, it was necessary to control body composition while assessing the effect of physical fitness on hormone secretions and sleep. This was achieved by using two independent groups of fit and unfit subjects which were matched for the relevant anthropometric variables.

An additional benefit derived from these body composition measurements was that it was possible to investigate the relationship between hormone levels, and sleep and body composition variables by means of correlation analyses. The results from these analyses are reported in this chapter. A more detailed analysis of the relationship between sleep and body composition is reported in Chapter 6.

METHOD

Subjects

Two groups of subjects were used, each consisting of 17 paid male volunteers, predominantly students at the University of Edinburgh. One group consisted of physically fit athletes and the other of unfit non-athletes. The fit athletes were selected primarily on the basis of their sporting proficiency and amount of training. All fit subjects were participating at the top club level, and in some cases international level, in their respective sports. Each trained a minimum of four times a week. Aerobic fitness measures were also obtained on all subjects, though, for methodological reasons given below, less importance was attached to this measure. The athletes had an average VO_{2max} of 3.46 1/min (SD = .67) compared with 2.37 1/min (SD = .21) for the non-athletes. This difference was highly significant ($t(32) = 6.11$, $p < .001$). Of the 17 subjects in the athlete group, 6 were swimmers, 5 played rugby or soccer, 2 hockey, 3 were distance runners, and 1 canoed competitively. The second group, the non-athletes,

were selected on the basis of a history of non-participation in sport.

The groups were matched for age, weight, and height, the mean values being presented in Table 5. As a consequence of matching for height and weight the groups were in retrospect equivalent with respect to LBM and fat levels. In addition, subjects were interviewed to ensure that they had regular sleeping habits and were not on medication. While it was not possible to control for dietary practices, no subjects had unusual patterns of food intake. The subjects were informed as to the nature of the experiment and their consent obtained prior to their participation. The procedures were approved by the Ethics Committee of the Royal Edinburgh Hospital.

Procedure

Each subject slept three non-consecutive nights in the sleep laboratory over one week. The first night was an adaptation night, while the second and third were experimental nights. On one experimental night sleep recordings only were collected, while on the other, both sleep recordings and blood samples were taken. Nine fit and eight unfit subjects were scheduled to have their sleep recording night prior to their blood sampling night. However, the blood sample night was repeated in four unfit subjects to obtain a satisfactory number of blood samples with the result that ten unfit subjects had sleep recordings first. Subsequent analysis of the sleep data showed no effect of the order of the two conditions.

TABLE 5.

Average age, maximal oxygen uptake (VO_{2max} l/min) and anthropometric measures for the fit and unfit samples (n = 17 in each group except for LBM and fat measures for the fit group where n = 15). LBM and fat were derived from creatinine excretion measures. Standard deviations are shown in brackets.

	Fit Athletes	Unfit Non-Athletes
Age (yrs)	20.7 (.93)	20.1 (.99)
VO_{2max} (l/min)	3.46 (.67)	2.37 (.21)
Weight (kg)	73.3 (8.21)	72.8 (5.81)
Height (cm)	181.6 (5.97)	180.8 (5.33)
Quetelet's Index	.22 (.017)	.22 (.023)
LBM (kg)	53.96 (7.43)	54.81 (5.70)
Fat (kg)	19.18 (6.13)	18.02 (4.59)
% LBM	73.88 (7.33)	75.29 (5.66)

Subjects came to the laboratory 1½ hrs before their usual bedtime, electrodes were attached, and on adaptation and blood sampling nights the catheter was inserted. On the other experimental night sleep data only was collected and the catheter was not inserted. Lights were turned out as close to each subject's usual bedtime as possible, and subjects were woken, or allowed up, at their usual rising time. During the period of the study subjects were required to refrain from alcohol, daytime naps and irregular sleeping hours. On experimental days and on each day preceding, subjects in both groups performed no exercise beyond minimum daily requirements.

Blood samples were taken by means of an indwelling venous catheter inserted in the forearm approximately one hour before retiring. An extension which allowed the subject freedom of movement in bed passed from the bedroom to the adjacent control room so that samples could be taken without disturbing the subject. The catheter and extension were filled with a heparinised saline solution which was drawn out with a syringe and discarded before each blood sample was taken. A 10 ml blood sample was drawn into a syringe then the tube and catheter were refilled with the saline. The 10 ml blood samples were collected every 20 mins from lights out until morning rising.

Blood samples were immediately centrifuged, the plasma separated and stored at -20^{o}C until assayed. The samples were analysed for hGH, prolactin and cortisol by specific double antibody radio-immuno-assays.

The results for hGH and prolactin are expressed in ng/ml; 1ng standard hGH is equivalent to 2 μIU/ml WHO 66/217 and 1ng standard prolactin is equivalent to 32.5 μIU/ml WHO 75/504. The cortisol levels are expressed in μmol/1. The intra-assay coefficient of variation ranged between 2.5 and 7%, and the inter-assay coefficient of variation ranged from 6 to 13.5% with only minor differences between the various hormone assays.

In the majority of subjects all samples were collected on schedule. The loss rate was low and is reported in the Results section. Difficulties were experienced in obtaining blood samples from 6 non-athletes and 2 athletes. In each case the condition was repeated, being successful for 4 non-athletes. As discussed above, this had consequences for the counterbalancing of conditions. The remaining subjects, 2 from each group, were excluded from the hormone data analyses, leaving 15 in each group. These exclusions did not alter the age or height matching and improved the weight matching between the two groups.

Sleep recordings and electrode placements were in accordance with standardised procedures (Rechtschaffen and Kales, 1968). The sleep records were scored blind by two scorers with an inter-rater reliability of greater than 90%. Each scorer scored the records from half the athletes and half the non-athletes.

The proportion of LBM to fat was determined from 24 hourly creatinine excretions. Creatinine is a nonreusable metabolite of muscle cells which is eliminated quickly into urine. The amount excreted varies positively as a function

of muscularity and is relatively constant from day to day within the same individual (McMurray, 1977). Thus urinary creatinine excretion can be used as an indirect measure of muscle mass.

For the collection of urine, subjects were instructed to void last thing before going to bed on the night before the collection day, noting the time. Subjects collected the first morning urine excretion at home, before coming into the laboratory where they stayed all day until the bedtime of the previous night. All urine excreted during this period was collected and thus one total days urine was obtained. The volume of urine excreted was recorded and samples frozen for subsequent analysis. Subjects were required to perform minimal exercise the day prior to and on the urine collection day and beginning with the evening meal of the previous day maintained a non-meat diet until the end of the urine collection period.

Creatinine levels were obtained on all but 2 fit subjects. In these two cases collection errors were made. The two groups were found to be closely matched for creatinine levels, the average creatinine coefficients being 20.85 (SD = 3.61) and 21.55 (SD = 2.75) for the fit and unfit groups respectively. LBM and %LBM were derived using the regression equation of Miller and Blyth (1952) LBM = 20.97 + .5161 (mgms creatinine excreted per hour). It is notable that creatinine levels and consequently creatinine coefficients and LBM estimates in this study were low in comparison with several other studies (e.g. Miller and Blyth, 1952; Talbot,1938).

This could have been due either to the method of assay, or to restrictions placed on subjects activity levels and dietary intake. As a consequence absolute LBM values derived from the available equations for converting creatinine levels to LBM, including the Miller and Blyth equation, were underestimates. However, as the formula simply applies a constant, relative levels were meaningful and thus correlational analyses were valid. The advantage of applying the formula, rather than using creatinine coefficients was that percentage LBM values were computed and used in the correlational analysis.

Difficulties were experienced in obtaining appropriate aerobic fitness measures on the subjects as neither a treadmill nor a bicycle ergonometer was available. The test selected was a modification of the Harvard Step Test for which standardised tables were available (Åstrand and Rodahl, 1977). Subjects were required to step up to and down from a 40cm step at the rate of 20 steps per min for 6 min and then following a recovery period at 30 steps per min for 6 min. Heart rate (HR) at the termination of the test was recorded and the rate for the 30 steps per min test was used to determine VO_{2max} values. In unfit non-athletes whose HR exceeded 180 during the 30 step per min test the rate during the 20 step per min test was used to estimate their VO_{2max}. However, the step test has a number of disadvantages which result in imprecise measurements (Åstrand and Rodahl, 1977). In particular, it was noticeable that shorter athletes tended to score lower than would have been exptected on the basis of their competitive success and training routine. For this

reason subject selection was primarily on proficiency and training. Nevertheless, as indicated above, the groups were discriminated with respect to VO_{2max} values, though the average for the fit group is probably an underestimation.

RESULTS

Hormone Data

Growth Hormone : Plasma concentration values for each 20 min sample for the two groups over the first 4 hrs of sleep are shown in Figure 1. They appear to indicate higher levels of hGH in the athletes. However, statistical analyses do not support this conclusion. The data were initially analysed by means of a 2 x 12 ANOVA where the first factor consisted of the two groups, athletes and non-athletes, and the second of 12 blood sample trials from each subject. Since hGH secretion is sleep dependent (Parker et al., 1980) in order to synchronize subjects the first sample used in the analysis was the first after sleep onset. After 12 consecutive trials (4 hrs), hGH concentrations were so low as to be unmeasurable in virtually all subjects so analysis was not continued beyond this point. Missing data were 2.8% of all samples.

Neither the main effect of groups nor the groups by trials interaction were significant[1] ($F(1/28)=2.44$, $MSe = 159.22$; $F(11/308)=1.60$, $MSe=26.84$ respectively) though there was a significant main effect of trials ($F(11/308)=30.88$, $MSe=26.84$) reflecting the rhythmic secretion of hGH. Further

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[1] As in all the analyses in Experiment 2, the significance level was set at $p < .05$.

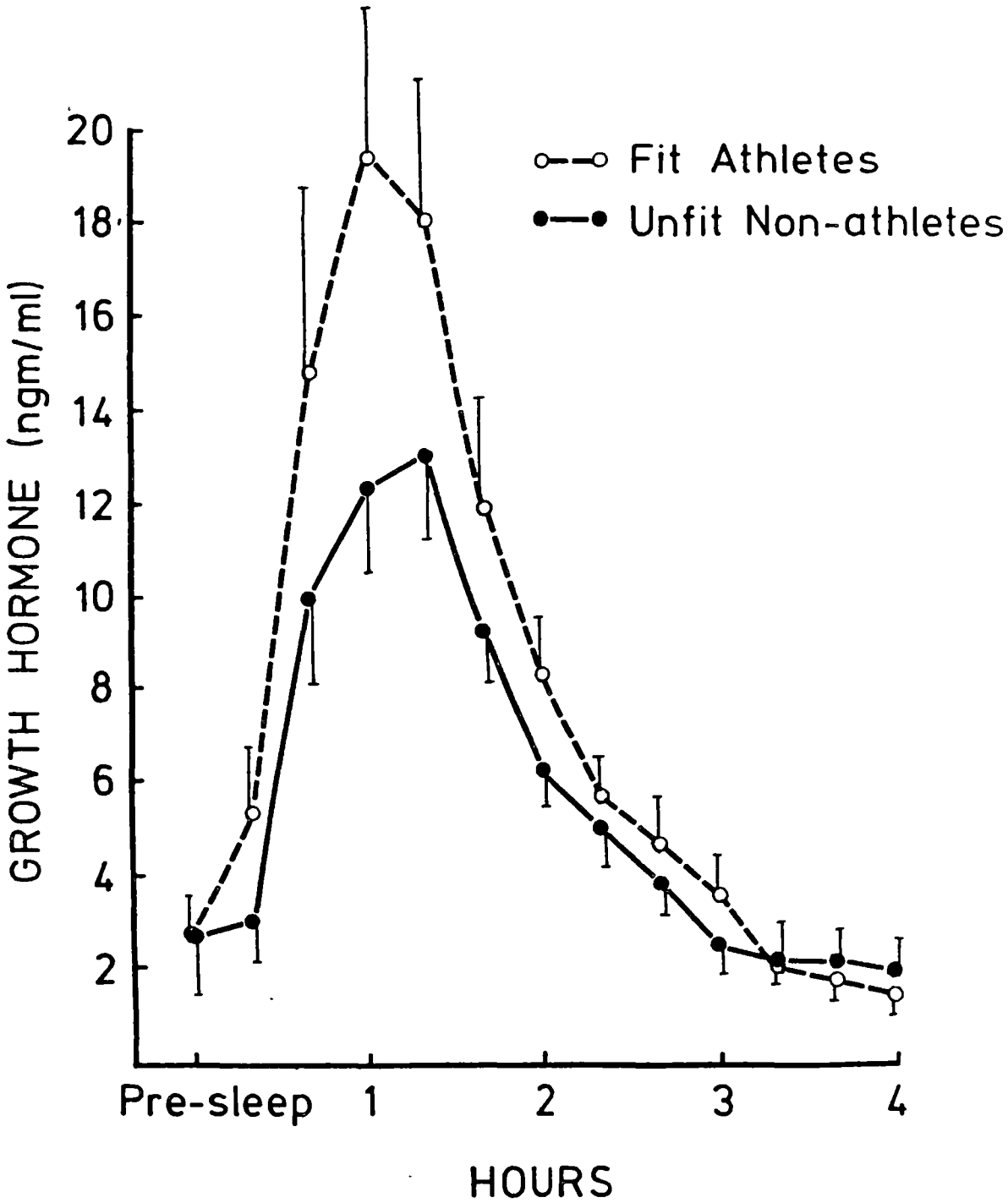

FIGURE 1. Mean plasma growth hormone concentrations in fit and unfit groups over the first 4 hrs of sleep, referenced to sleep onset. The pre-sleep value is the final sample before sleep onset. The standard error of the mean is represented by the vertical line at each data point.

comparisons of peak plasma concentration and area under the curve for the first four hours between the groups were not significant ($t(28) = 1.66$, $p > .10$ and $t(28) = 1.29$, $p > .10$ respectively). Non-parametric analyses of the latter two measures were also non-significant. Individual values for the peak response are shown in Table 6.

Prolactin : The prolactin concentrations are illustrated in Figure 2. As in the case of hGH the group differences were not significant. Thus a 2 x 18 ANOVA (groups x 20 min samples), which was referenced to sleep onset (Parker et al., 1980) was initially conducted. Missing data were 5.4% of all samples.

The analysis indicated there was no significant difference between prolactin secretion levels in the athlete and non-athlete groups. Neither the interaction between groups and trials ($F(17/476)=.77$, $MSe=5.86$) nor the main effect of groups ($F(1/28)=.39$, $MSe=211.41$) was significant. There was, however, a main effect of trials ($F(17/476)=6.57$, $MSe=5.86$) due to the elevation in prolactin secretion about 2 hrs after sleep onset. Comparison of both peak plasma concentration and area under the curve for the first six hours failed to reveal a significant difference between the two groups ($t(28)=.65$, $p > .10$ and $t(28) = .49$, $p > .10$ respectively). Again non-parametric analyses produced the same results. Individual values for the peak response are shown in Table 6.

Cortisol : The cortisol secretion data are illustrated in Figure 3. They were analysed using a 2 x 21 ANOVA. As

TABLE 6.

Hormone concentrations for individual subjects. Values for the peak response (20 min sample) are shown for hGH and prolactin while both the minimum and maximum values during the sleep period are shown for cortisol. Peak responses were selected to illustrate the data as the units are more meaningful than area under the curve. Nevertheless, as indicated in the text, the results were identical for each form of the data.

	hGH (ng/ml) Peak Response		Prolactin (ng/ml) Peak Response		Cortisol (umol/l) Minimum		Maximum	
	Fit	Unfit	Fit	Unfit	Fit	Unfit	Fit	Unfit
1.	14.5	4.9	9.6	15.5	44.1	52.2	652.3	389.2
2.	5.8	14.1	29.8	15.9	49.6	40.5	695.9	602.1
3.	15.0	17.4	13.5	14.5	61.2	71.2	406.5	499.2
4.	37.6	12.4	26.2	15.0	37.0	93.3	465.8	547.7
5.	18.6	15.1	11.6	16.4	37.6	72.8	534.8	592.4
6.	5.1	18.2	6.3	12.7	27.6	63.8	416.2	515.3
7.	24.2	25.7	9.8	8.8	41.8	35.0	459.3	426.9
8.	11.7	19.9	12.1	11.4	27.6	29.5	59.9	356.9
9.	33.4	8.5	13.2	9.1	50.2	38.6	534.8	449.2
10.	16.2	31.9	6.7	15.2	50.9	27.6	463.6	326.7
11.	17.2	10.7	13.9	5.6	53.5	36.0	674.9	409.7
12.	17.9	13.9	10.3	8.8	31.5	55.1	508.9	616.7
13.	54.2	19.1	19.8	11.5	37.3	65.8	459.3	304.9
14.	39.8	19.2	15.6	23.2	34.7	66.4	418.0	394.6
15.	24.2	6.0	15.5	10.2	43.4	31.2	495.9	211.3
$\bar{x}$	22.4	15.8	14.3	12.9	41.9	51.9	483.1	442.9
SD	13.6	7.2	6.6	4.3	9.8	19.7	149.4	118.8

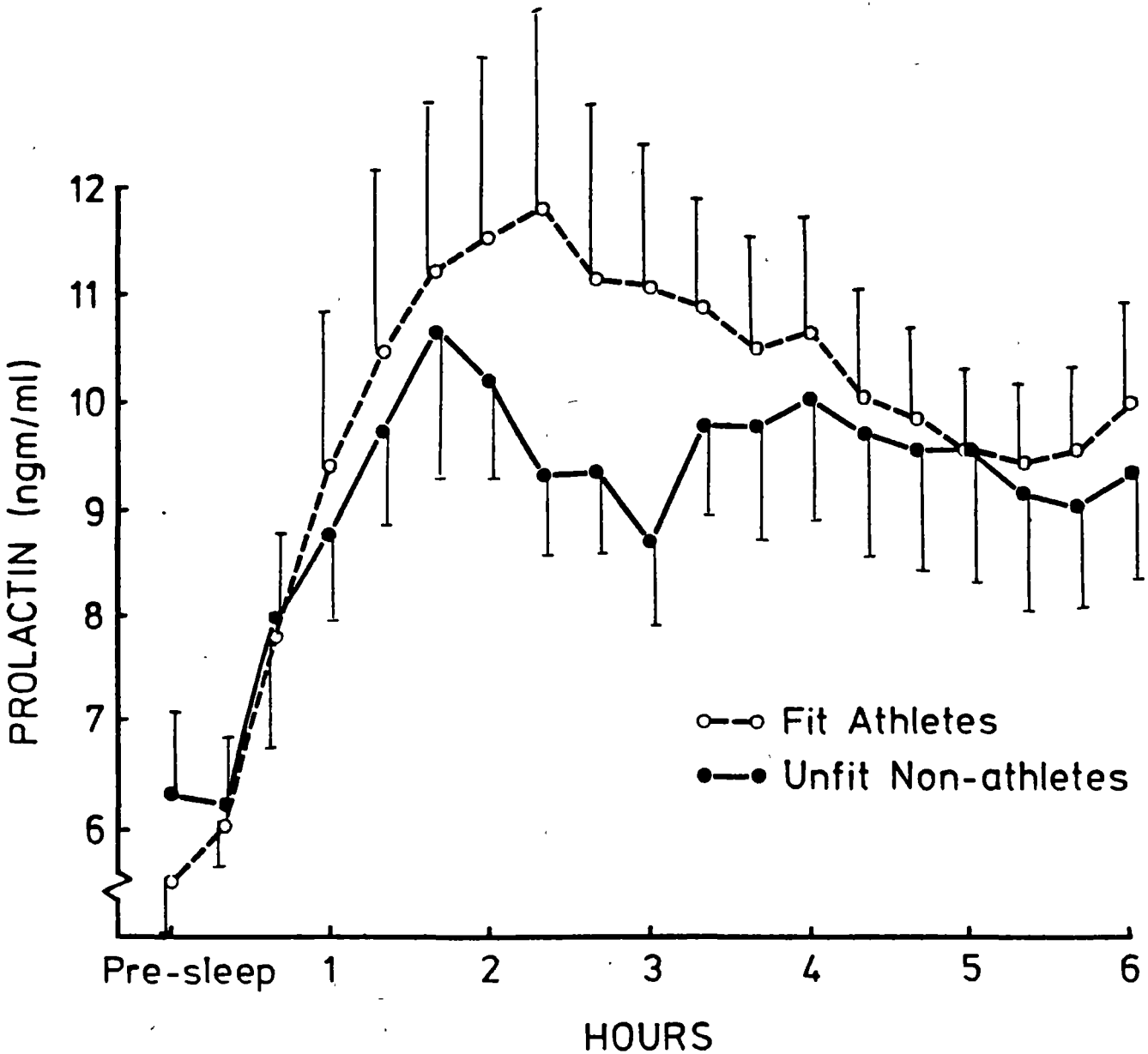

FIGURE 2. Mean plasma prolactin concentrations in fit and unfit groups over the first 6 hrs of sleep, referenced to sleep onset. The pre-sleep value is the final sample before sleep onset. The standard error of the mean is represented by the vertical line at each data point.

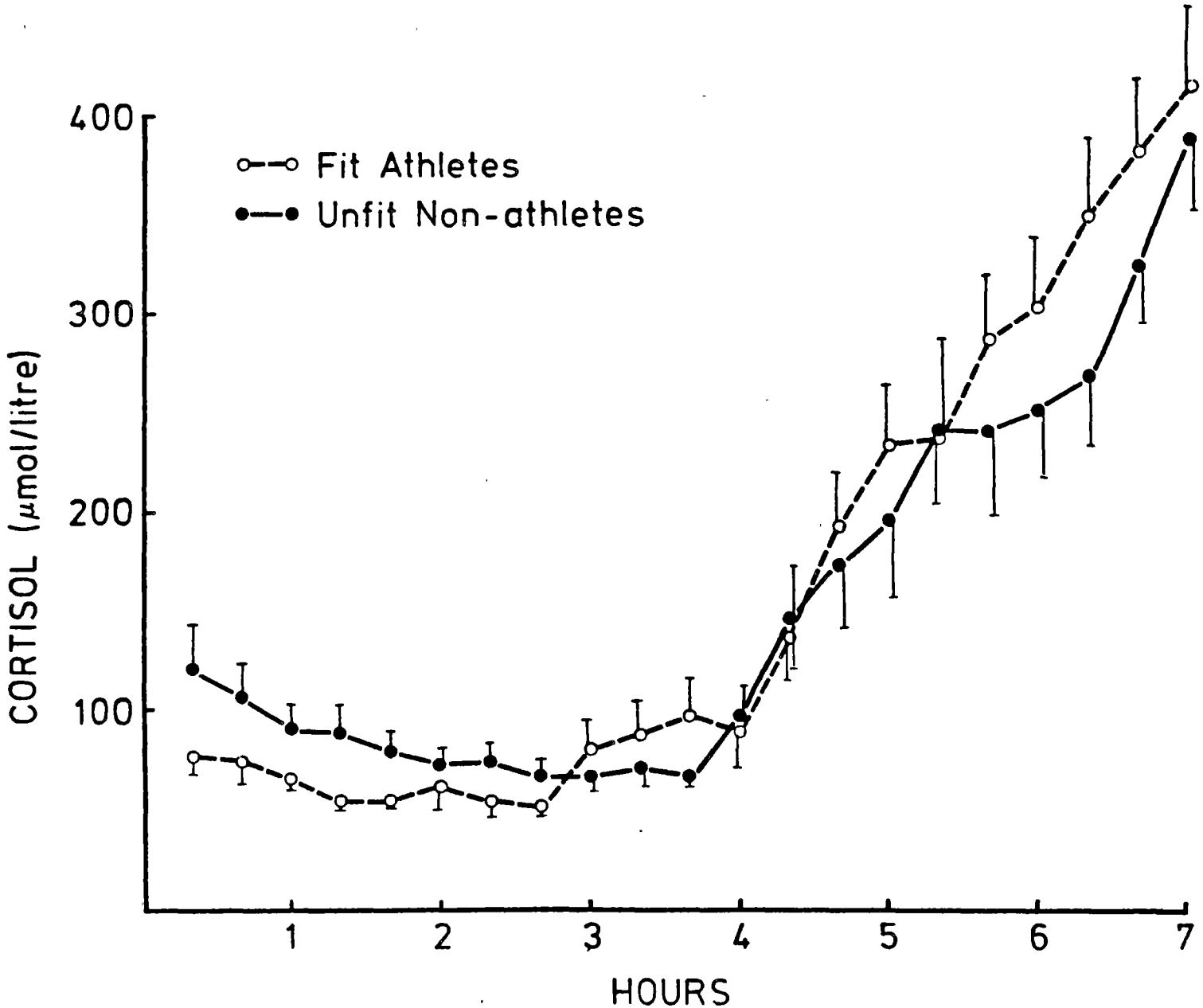

FIGURE 3. Mean plasma cortisol concentrations in fit and unfit groups over the first 7 hrs, referenced to time of going to bed. The standard error of the mean is represented by the vertical line at each data point.

cortisol has a true circadian rhythm (Parker et al., 1980), the first sample of the analysis was the first taken after lights out as this was as close to the subject's usual bedtime as possible and was thought to be the best method of synchronizing subjects' circadian rhythms. The analysis was discontinued after 21 trials as missing data became an appreciable proportion. Missing data were 6.0% of all samples. The analysis of variance was performed on a logE transformation of the raw data as the transformed data better conformed to the assumptions of the analysis of variance.

The results revealed a significant groups by trials interaction ($F(20/560) = 2.31$, $MSe = .19$) such that cortisol levels were lower in athletes than non-athletes early in the night, but were higher in athletes later in the night. There was no significant effect of groups ($F(1/28) = .05$, $MSe = 3.15$), though as would be expected, there was a significant effect of trials ($F(20/560) = 62.62$, $MSe = .19$) reflecting the increase in cortisol secretion across the night. Further examination of the interaction effect revealed that it was not as strong as the initial analysis had indicated. Thus an ANOVA based on averaged hourly concentrations failed to reach significance ($F(6/168) = 2.09$, $MSe = .16$, $p = .057$). In addition, while post hoc analyses based on the ANOVA showed that the athletes had significantly lower cortisol values on each of the first 7 trials, a non-parametric test (Mann-Whitney U) was significant only on trials 4 and 7. The differences late in the night were not significant. The maximum and minimum sample values are shown for each subject in Table 6.

In order to investigate further the relationship between hGH and cortisol, the logE of the ratio of cortisol to hGH (logE(cortisol/ hGH)) was determined for each subject for each trial during the period of hGH secretion. The value of this ratio represented the relative levels of cortisol and hGH, such that a high value indicated relatively high levels of cortisol compared with hGH, while a low value indicated the opposite. A comparison between groups at the lowest value for each subject (a method equivalent to that of comparing peak responses) showed that the fit subjects were significantly lower (relatively high hGH) than the unfit ($t(28) = 2.36$, $p < .05$). Similarly, the ratio for the fit subjects was significantly lower at the time of the peak hGH response approximately an hour following sleep onset ($t(28) = 2.16$, $p < .05$ and $t(28) = 2.27$, $p < .05$ for trials 3 and 4 respectively). In other words, this analysis supports the impression conveyed by Figures 1 and 3 that the combination of high hGH and low cortisol during the early part of the night is more extreme in fit athletes.

Sleep Variables

The sleep data were analysed using a sample size of 17 subjects in each group and a 2 x 2 ANOVA where the first factor consisted of the two groups, athletes vs non-athletes and the second consisted of the two sleep conditions, base-line (no catheter) vs sleep recording plus catheter. Table 7 shows the mean values for the most relevant EEG sleep variables for the two groups.

TABLE 7.

Selected mean sleep variables as a function of group and condition. All values are given in minutes and standard deviations are shown in brackets. n = 17 for each group.

	Fit		Unfit		
	Baseline	Catheter	Baseline	Catheter	
Time in bed	495 (26.7)	461 (30.4)	489 (25.4)	462 (24.1)	*
Sleep period time	480 (26.8)	442 (38.5)	463 (51.7)	433 (34.2)	*
Total sleep time	475 (28.6)	423 (34.1)	449 (49.5)	414 (39.1)	*
Total time awake	20 (15.7)	38 (31.6)	40 (49.0)	48 (32.5)	*
Stages 1+MT	52 (15.5)	41 (17.8)	58 (19.1)	40 (11.3)	
Stage 2	218 (29.8)	195 (35.1)	194 (38.7)	193 (21.4)	
Stage 3	33 (12.9)	28 (7.2)	30 (9.7)	27 (9.6)	
Stage 4	59 (21.8)	67 (18.7)	68 (22.6)	70 (21.9)	
SWS (3+4)	92 (22.0)	95 (21.0)	98 (24.6)	97 (28.8)	
NREM (2+3+4)	310 (23.0)	290 (20.7)	292 (49.2)	290 (31.5)	
REM	113 (21.1)	92 (19.9)	99 (25.3)	84 (17.8)	*
Sleep onset latency	19 (14.8)	22 (20.8)	35 (41.4)	34 (37.7)	
Stage 3 latency	11 (3.2)	10 (3.4)	10 (3.4)	11 (3.9)	
REM latency	66 (16.7)	74 (26.0)	76 (23.9)	104 (58.6)	**

* Main effect of night (baseline vs catheter) significant ($p < .05$)

** Main effect of group (athlete vs non-athlete) significant ($p < .05$)

The data analysis indicated there was no significant difference in SWS between athletes and non-athletes ($F(1/32) = .30$, $MSe = 936.34$). In fact, there were no significant differences in sleep variables between the groups except in REM latency ($F(1/32) = 6.03$, $MSe = 1078.53$). There is no reason in the literature to anticipate this result, so, considering the number of analyses performed, this is most likely a chance result.

There were no significant interaction effects between the groups and conditions, indicating that neither group was more affected by the blood sampling procedures than the other. Sleep condition, in fact, was not shown to affect sleep patterns in any major way. All effects of the catheter can be related to a shortened TIB (mean TIB for sleep night = 492 min, catheter night = 461.5 min) and a slightly greater wake time on catheter nights (mean wake time for sleep night = 30 min, for catheter night = 43 min). This was almost entirely due to procedural factors as it took longer to organize subjects for bed on blood sampling nights. Thus TIB, TST and SPT were significantly shorter on catheter nights ($F(1/32) = 37.64$, $MSe = 336.42$; $F(1/32) = 37.69$, $MSe = 817.95$; $F(1/32) = 49.67$, $MSe = 314.48$ for TIB, TST and SPT respectively) and consequently REM sleep in minutes was significantly lower ($F(1/32) = 13.42$, $MSe = 398.05$). However, REM as a percentage of TST was not significantly lower on catheter nights ($F(1/32) = 3.25$, $MSe = 18.25$) and an analysis of the first 6 hours of sleep show that time in REM was essentially identical in the two conditions (sleep = 68.4 min and catheter = 67.7 min; $F(1/32) = .03$, $MSe = 273.81$).

Individual Difference in Hormones, Sleep and Body Composition

In order to assess, within subjects, the relationship between plasma hormone levels and both sleep variables and body composition, a series of correlation analyses were conducted. The hormone variables entered into the analyses were hGH during the first 4 hours of sleep (area under the curve), hGH peak response, prolactin during the first 6 hours of sleep (area under the curve), the maximum and minimum cortisol values and the average sleep time cortisol value. The sleep variables were minutes in SWS, REM as a percent of TST, TST, and a measure of disturbed sleep (wake + movement time + Stage 1 as a percentage of TIB). The body composition variables were weight, height, weight/height ratio, Quetelet's Index (w/h^2 x 100), LBM and fat weight, and LBM and fat percentages. Finally, VO_{2max} values were also included. Spearman rank order correlation coefficients were computed as some variables were markedly skewed. Independent analyses were conducted for all subjects combined and for fit and unfit subjects separately.

Human growth hormone, SWS and percentage LBM were interrelated when analysed over all subjects combined. Thus, there were a significant negative correlation between hGH and total SWS, a significant positive correlation between hGH and percentage LBM and a significant negative correlation between SWS and percentage LBM. (The correlations between hormone variables and both sleep and body composition variables are shown in Table 8. The correlations between sleep and body composition measures are shown in Table 12 in Chapter 6.

TABLE 8.

Spearman rank order correlation coefficients between hormone concentrations and physical fitness, body composition and selected sleep variables. hGH was represented by the area under the curve for the first 4 hrs following sleep onset, prolactin by the area under the curve for the first 6 hrs following sleep onset and cortisol by the average nightly concentrations (ng/ml).

	All Subjects			Fit Subjects			Unfit Subjects		
	hGH	Prolactin	Cortisol	hGH	Prolactin	Cortisol	hGH	Prolactin	Cortisol
VO_{2max} (l/min)	.03	-.06	.00	-.46	-.13	-.06	.02	-.29	.07
Weight (kg)	-.27	-.06	-.06	-.25	-.01	-.06	-.34	-.09	-.06
Height (cm)	.15	.01	-.35	.02	.19	-.27	.26	-.28	-.48
W/H	-.27	-.06	.02	-.29	-.11	-.08	-.28	.03	.20
Quetelet's Index	-.29	-.03	.12	.22	-.28	.02	-.35	.14	.31
LBM (kg)	.02	-.07	.01	.09	-.10	.18	.08	-.01	-.08
FAT (kg)	-.53**	-.06	-.09	-.64*	-.08	-.41	-.43	-.02	.21
% LBM	.47*	.05	.09	.72**	.08	.36	.37	.00	-.26
% FAT	-.47*	-.05	-.09	-.72**	-.08	-.36	-.37	.00	.26
SWS (min)	-.39*	-.12	.07	-.65**	-.16	-.17	-.08	.01	.25
REM (% TST)	.20	.35	.15	.23	.50	.11	.06	.11	.16
TST (min)	.10	.06	.17	-.19	-.15	.03	.34	.18	.23
DIST. SLEEP	-.22	-.18	-.21	-.24	-.04	-.09	-.05	-.22	-.27

* $p < .05$

** $p < .01$

Though significant over all subjects, the effects were more marked in the fit subjects, failing to reach significance in the unfit group alone, although the unfit group showed significant negative correlations between SWS and total body weight, and SWS and LBM kgs. The results for hGH peak response were the same as for the first 4 hrs. Prolactin and all cortisol measures showed no significant correlations with either sleep or body composition variables. Finally, as would be expected from the earlier analyses, physical fitness did not correlate significantly with either hormone levels or SWS.

The critical factor in the relationship between hGH and percentage LBM may be the effect of body fat on the relative proportions of LBM and fat as there was a significant negative relationship between fat weight and hGH but the absence of any relationship between LBM weight and hGH.

DISCUSSION

The data did not support the hypothesis that physically fit athletes have a different sleep period hormone secretion from unfit individuals. While some comparisons were marginally significant, notably the low cortisol and elevated hGH during the first 4 hrs of sleep, the results in general were not strong enough to allow the rejection of the null hypothesis. However, it is recognized that alternative interpretations of these data could be made.

Body composition appears to be a better predictor of hGH than physical fitness. In particular there is a

positive relationship between hGH and proportion of LBM but a negative relationship between hGH and total body fat. These data are similar to an earlier report of Othmer et al. (1974) who found a significant positive correlation between the height/weight ratio and hGH during the first 4 hrs of sleep. It is of some interest that it is fat levels rather than total weight, or LBM which predicts hGH levels. This result is similar to that reported by Kalkhoff and Ferrou (1972) using an insulin induced hypoglycemia test in awake subjects. They reported that low hGH secretion was related more to body fat accumulation than excess lean tissue mass. The body composition/hGH data are more consistent with the suggestion that hGH secretion at night is related to the regulation of fat metabolism rather than protein synthesis (Horne, 1979).

The stronger relationship in fit athletes may be a result of a hormonal adaptation in energy metabolism following physical training (see Chapter 3). Thus, in these individuals fat levels may be more directly influenced by hGH activity. Further, the heterogeneity of the sporting activities of the athlete group, in contrast to the homogeneity of physical activity in the unfit group, could also have contributed to the observed pattern of correlations.

The negative correlation found between SWS and hGH was unexpected and may be an artifact of the relationship of each with body composition. Further, the comparison of all night SWS with hGH, which occurs during the early sleep period, may be inappropriate. Therefore analyses comparing

hGH levels during the first 4 hrs of sleep with SWS levels during this period were conducted. Under these conditions the correlation coefficients were not significant (-.28, $p > .05$ for all subjects; -.46, $p > .05$ for fit subjects; -.19, $p > .05$ for unfit subjects). It is possible that the overall negative relationship is unimportant.

Nevertheless, it should be noted that the generally accepted positive relationship between hGH and SWS is based on hGH's dependence on SWS for its occurrence, and the observation that certain stimuli have similar effects on each (e.g. hyperthyroidism (Dunleavy et al, 1974)). Neither of these findings necessarily exclude the possibility that within normal subjects chronic levels of hGH and SWS are negatively related.

The sleep patterns of the two groups were very similar. In particular there was no significant difference in SWS between athletes and non-athletes. Further, the correlation analysis revealed no relationship between SWS and VO_{2max}. On the basis of earlier literature (Baekeland and Lasky, 1966; Buguet et al., 1980; Griffin and Trinder 1978; Trinder et al., 1980; Zloty et al., 1973) it might have been anticipated that SWS would be elevated in fit athletes. However, present data are consistent with those of the first study in this thesis which indicate that some factor other than aerobic fitness is involved. As in Experiment I, body composition is implicated in the second experiment as a possible factor since the matching of body composition variables appears to have eliminated the SWS

differences observed in the first study between athletes and non-athletes. An additional indication that body composition variables may be related to SWS is the finding of a negative relationship between SWS and percentage LBM. This issue is further discussed in Chapter 6 where more detailed results are presented.

Minor differences were observed in sleep between baseline and catheter nights. These differences were most likely due to procedural factors rather than to the catheter itself. This conclusion is contrary to a recent report (Adam, 1982). In this study the sleep of subjects aged 53 to 63 showed a number of changes as a function of blood sampling via an indwelling catheter. The differences of outcome between the studies are most likely due to age.

CHAPTER 6

EXPERIMENT 3 : BODY COMPOSITION AND SWS

The results of Experiment I indicated that the differences in sleep observed between fit and unfit groups are not a consequence of fitness. The results of Experiment 2 were consistent with this conclusion. It therefore appears likely that previous differences between fit and unfit groups were due to one or more other variables which are systematically related to physical activity. The possibility that physical fitness was confounded with other variables had been previously proposed (Griffin and Trinder, 1978); that body composition might be a critical variable was considered when designing Experiment I. Thus, anthropometric variables were measured in subjects in Experiment I and in subjects being run in other experiments in the laboratory at the time. Similarly, while it was necessary for methodological reasons relating to hormone levels to control for height and weight in subjects in Experiment 2, the addition of LBM measurements also allowed an analysis of the relationship between body composition and sleep. While aspects of these data have been presented in Experiments I and 2 a full analysis and presentation of the body composition data has been with-held to this point to facilitate the clarity of the presentation.

There are, in fact, few reports in the literature which have examined the relationship between body composition and sleep variables. Othmer et al. (1974) in a study on sleep-related growth hormone secretion and SWS, reported that SWS

was not significantly related to weight, height, height/weight ratio or deviation from ideal body weight while Nakazawa, Hasuzawa, Ohkawa, Sakurada and Nonako (1978) found a significant negative relationship between SWS % and height, but no relationship between SWS and weight or weight/height ratio. In addition, there have been a number of studies which have reported on the sleep of mildly obese older subjects (Adam, 1977a,b), clinically obese subjects before and after weight loss (Crisp, Stonehill, Fenton & Fenwick, 1973; Ho, Fekete-Mackintosh, Resnikoff & Grinker, 1978; Ogilvie & Broughton, 1977) and anorexia nervosa patients during weight gain (Lacey, Crisp, Kalucy, Hartmann & Chen, 1975). No consistent relationship between weight and sleep has been found. These studies, however, have not reported anthropometric data other than weight and height.

This chapter presents the analyses of body composition and sleep data from subjects run in Experiment I and other studies conducted in this laboratory during approximately the same period and from subjects run in Experiment 2. The data from these two sources were analysed separately and are designated Experiments 3 (a) and 3 (b) respectively. In each case there were two independent groups, fit athletes and unfit sedentary individuals.

METHOD

EXPERIMENT 3(a)

Subject and Design

Anthropometric data were collected on two groups of male volunteers, 25 aerobically fit and 22 aerobically unfit subjects. All subjects were participants in other experiments which were being run in the laboratory. The anthropometric measurements were collected in addition to other data relevant to each experiment. This procedure was commenced when it became apparent that body composition could be an important factor. Subjects were mainly drawn from the University of Tasmania student population and three quarters of the subjects were paid for their participation. The average age for each of the groups was 20.2 and 20.1 yrs for the fit and unfit groups respectively.

Fit subjects were required to be training a minimum of 3 times a week and to perform at a proficient level in a sport. Runners, rowers, and football and hockey players were represented in this group. The unfit subjects were selected if they had low aerobic fitness, were not involved in athletic activities and had not done so in the past other than compulsory activities at school. Average VO_{2max} levels for the two groups were assessed. The value for fit subjects was 3.97 1/min (57 ml/kg x min) while for the unfit group it was 2.58 1/min (37 ml/kg x min). Weight, height and skinfold thickness measurements were collected. From these a number of variables were subsequently derived and used in the analyses. These were : weight, height, Quetelet's index, kilograms LBM, kilograms fat, percentage LBM and

percentage fat. Mean values for anthropometric variables for the fit and unfit groups are shown in Table 9. Subjects slept a minimum of three experimental nights in the sleep laboratory and sleep variable data from each subject on all available experimental nights were averaged.

Procedure

Subjects slept either 3 (9 subjects), 4 (21 subjects), 6 (11 subjects) or 8 (6 subjects) nonconsecutive experimental nights in the sleep laboratory over a period of 2-3 weeks. An adaptation night preceded the experimental nights. Subjects came into the sleep laboratory one hour before their usual bedtime and electrodes were attached. They went to bed at their usual time and were woken at their usual rising time. During the experimental period subjects were instructed to maintain regular sleeping, eating and exercise patterns and to abstain from taking any alcohol. The original experiments in which the subjects participated were concerned with the effect of various levels of exercise on sleep. As these manipulations did not have any significant effect, sleep values were obtained by averaging over all experimental nights. Not all subjects from these experiments have been included in the present analysis. Subjects were excluded if they were female, not between ages 18 and 25 or if anthropometric data were unavailable.

Weight was available for all subjects not excluded by the above criteria, while height and consequently height dependent information were available on 20 fit and 11 unfit subjects. The proportion of LBM and fat were determined by a skinfold thickness method. Skinfold thickness was

TABLE 9.

Average age, maximal oxygen uptake and anthropometric measures for the fit and unfit groups in Experiment 3(a). As n is different for different variables it is indicated in the table. Standard deviations are shown in brackets.

	Fit		Unfit	
Age (yrs)	20.24 (1.76)	n=25	21.14 (1.58)	n=22
VO_{2max} (l/min)	3.97 (.53)	n=25	2.58 (.38)	n=22
VO_{2max} (ml/kg x min)	57 (6.3)	n=25	37 (4.6)	n=22
Weight (kg)	72.44 (6.63)	n=25	68.08 (6.97)	n=22
Height (cm)	180.50 (6.62)	n=20	178.55 (4.97)	n=11
Weight/Height	.41 (.029)	n=20	.38 (.030)	n=11
Quetelet's Index	.23 (.018)	n=20	.21 (.018)	n=11
LBM (kg)	62.81 (5.22)	n=19	58.70 (5.73)	n=12
Fat (kg)	10.91 (2.67)	n=19	9.53 (3.11)	n=12
%LBM	85.32 (2.59)	n=19	86.09 (4.07)	n=12

* $p < .05$

measured at four sites; biceps, triceps, subscapular and supra-iliac and converted to percentage fat following procedures described by Durnin and Womersley (1974). These data were available on 19 fit and 12 unfit subjects.

Aerobic fitness levels were assessed using a standard submaximal bicycle ergometer test (Åstrand & Rodahl, 1977). Sleep recordings were obtained and scored according to the standardized procedures described by Rechtschaffen and Kales (1968). Sleep records were scored blind by two scorers with an inter-rater agreement of greater than 90 percent.

EXPERIMENT 3(b)

Subjects and Design

Subjects used in Experiment 3(b) were those who took part in Experiment 2. Thus, there were two groups of male volunteers who were predominantly students at the University of Edinburgh and were all paid. One group consisted of 17 fit athletes (average age = 20.7 yrs) while the other consisted of 17 unfit non-athletes (average age = 20.1 yrs). Selection procedures and further subject information are provided in Chapter 5.

In contrast to Experiment 3(a) the fit and unfit groups were matched for weight and height, the mean values for each being presented in Table 5. The other anthropometric variables assessed in this experiment were the same as in Experiment 3(a). All subjects slept two experimental nights in the sleep laboratory and sleep data from both nights were averaged.

Procedure

As described in Chapter 5 subjects slept three non-consecutive nights in the sleep laboratory over the period of one week. The first night was an adaptation night, while the second and third were experimental nights. Subjects came into the laboratory 1½ hrs before their usual bedtime, went to bed at as close to their usual time as possible and were woken at their usual rising time. During the period of the experiment subjects were required to maintain regular sleeping and eating patterns and refrain from taking alcohol. On experimental days and on the day preceding, subjects in both groups performed no exercise beyond minimum daily requirements.

On one of the experimental nights, (the order was counter-balanced), blood samples were taken at 20 min intervals by means of an indwelling venous catheter, without disturbing the subject. Comparison of the non-catheter and catheter nights indicated there were only minimal effects of blood sampling procedures on sleep variables and there was no difference in the effect of the catheter on the fit and unfit groups. Therefore, the mean of the data obtained from the non-catheter and catheter nights was used in the present analysis.

Sleep recordings and electrode placements were in accordance with standardized procedures (Rechtschaffen and Kales, 1968). Sleep records were scored blind by two scorers with an inter-rater reliability of greater than 90%. Each scorer scored the records from half the athletes and half the non-athletes.

Weight and height information were available on all subjects in this study. In contrast to Experiment 3(a) LBM and fat were determined from 24 hourly creatinine excretions according to statistical procedures described by Miller and Blyth (1952). The values obtained by this method appear consistently low in comparison to other studies (see Chapter 5, pp. 92-93). However, as the formula to derive LBM from creatinine simply applies a constant, relative levels were meaningful and therefore correlation analyses were valid.

To obtain the 24 hour urine sample, subjects were instructed to void last thing before going to bed on the night before the collection day, noting the time. Subjects collected the first morning urine excretion at home, before coming into the laboratory where they stayed all day until the bedtime of the previous night. All urine excreted during this period was collected and thus one total days urine was obtained. The volume of urine excreted was recorded and samples frozen for future analysis. Subjects were required to perform minimal exercise the day prior to and on the urine collection day and beginning with the evening meal of the previous day maintained a non-meat diet until the end of the urine collection period. Creatinine levels on all subjects were obtained using this method. However, creatinine data from the two fit subjects were discarded because of collection errors.

RESULTS

In a preliminary analysis the fit and unfit groups were compared on both anthropometric and sleep variables. In Experiment 3(a) the fit subjects were significantly heavier ($t(45) = 2.19$, $p < .05$) and showed a slightly higher proportion of fat tissue, particularly as indicated by Quetelet's Index which was significant ($t(29) = 2.46$ $p < .05$). Mean body composition data for athletes and non-athletes are shown in Table 9. The differences between groups in sleep in Experiment 3(a), while generally not significant, were consistent with the trend of previous reports as fit subjects had more SWS ($t(45) = 1.65$, $p > .05$), had less disturbed sleep (W + 1 + MT as a % of TIB) ($t(45) = 1.93$, $.1 > p > .05$) and shorter sleep onset latencies ($t(45) = 4.09$, $p < .01$). Selected mean sleep variables for athletes and non-athletes are shown in Table 10. In Experiment 3(b) in which the fit and unfit groups were matched for height and weight, there were no significant differences in body composition or major sleep variables (see Chapter 5, p.88, p.101-103). Mean values for selected body composition and sleep variables are shown in Tables 5 and 7 respectively.

In order to test the hypothesis that individual differences in sleep are related to body composition, Spearman Rank Order correlation coefficients were computed between sleep and anthropometric variables both within fitness groups and by combining all subjects within experiments. The rank order test was chosen as several variables in each experiment were significantly skewed. The significance level was set at $p < .05$. The coefficients for all subjects combined and fit and unfit groups separately are shown for Experiments 3(a) and 3(b) in Tables 11 and 12 respectively.

TABLE 10.

Selected mean sleep variables of fit and unfit groups in Experiment 3(a). All values are given in minutes and standard deviations are shown in brackets. No differences between the fit and unfit groups were significant.

	Fit		Unfit	
	n = 25		n = 22	
Time in bed	458	(32.9)	457	(30.1)
Sleep period time	440	(26.1)	426	(43.3)
Total sleep time	439	(29.2)	432	(39.0)
Total time awake	19	(14.4)	25	(17.8)
Stage 1 + MT	39	(13.5)	45	(14.0)
Stage 2	206	(34.4)	201	(26.4)
Stage 3	37	(13.9)	34	(11.0)
Stage 4	65	(18.5)	58	(23.0)
SWS (3+4)	102	(28.5)	92	(25.3)
NREM (2+3+4)	308	(21.2)	293	(29.3)
REM	92	(15.7)	94	(17.1)
Sleep onset latency	14	(7.9)	24	(14.9)
Stage 3 latency	11	(4.2)	14	(6.9)
REM latency	91	(22.4)	78	(22.9)

TABLE 11.

Spearman rank order correlation coefficients between the anthropometric measures and selected sleep variables in Experiment 3(a).

	SWS			REM %			TST			W + M + 1 (% TIB)		
	Fit	Unfit	Total	Fit	Unfit	Total	Fit	Unfit	Total	Fit	Unfit	Total
Weight	-.07	-.53*	-.23	.12	.32	.14	-.03	-.07	-.03	-.08	.04	-.07
Height	.03	-.67*	-.11	-.14	.40	-.09	-.25	-.04	-.23	.12	-.32	-.09
Quetelet's Index	.22	.02	.16	.08	.15	-.03	.24	.63*	.29	-.28	.04	-.21
LBM (kg)	-.02	-.55	-.18	-.02	.13	-.09	-.31	.25	-.08	-.02	-.23	-.17
FAT (kg)	.41	-.17	.24	-.03	.67*	.12	.09	.54	.22	-.50*	-.04	-.33
LBM (%)	-.54*	-.01	-.33	.00	-.62*	-.13	-.03	-.41	-.24	.52*	-.02	.29

* $p < .05$

** $p < .01$

TABLE 12.

Spearman rank order correlation coefficients between the anthropometric measures and selected sleep variables in Experiment 3(b).

	SWS			REM %			TST			W + MT + 1 (% TIB)		
	Fit	Unfit	Total	Fit	Unfit	Total	Fit	Unfit	Total	Fit	Unfit	Total
Weight	.14	-.63**	-.22	-.04	.21	.05	.39	-.36	.02	.23	.49*	.32
Height	.08	.02	.03	.11	.28	.23	.16	.05	.09	-.09	-.35	-.19
Quetelet's Index	.05	-.55*	-.28	-.24	.01	-.12	.46	-.34	-.02	.31	.54*	.40*
LBM (kg)	-.17	-.68**	-.44*	-.46	.36	-.04	.22	-.20	-.04	.15	.25	.21
FAT (kg)	.38	.02	.22	.24	-.09	.06	.14	-.19	.02	.06	.25	.06
LBM (%)	-.54*	-.33	-.41*	-.23	.18	-.04	-.03	.00	-.03	-.19	.00	.00

* $p < .05$

** $p < .01$

The results of this analysis indicated that SWS was negatively related to LBM. The finding was consistent, being observed in each of the four samples. However, there were differences between the fit and unfit groups in the nature of this relationship. In fit subjects SWS was negatively correlated with the proportion of LBM, while in unfit subjects the association was with weight and total LBM. When all subjects were combined within each experiment, negative correlations were found between SWS and both total LBM and the proportion of LBM. These were significant in Experiment 3(b), and Experiment 3(a) showed a trend in this direction.

No other sleep variable showed a consistent relationship across experiments with any body composition factor. The results for percentage REM, TST and disturbed sleep are shown in Tables 11 and 12. They demonstrate that the significant correlations for these variables were unsystematic and isolated. The absence of significant correlations cannot be explained by low variability between subjects. The average standard deviations for the four groups were 23.89, 33.02, 3.06 and 5.67 for SWS (min), TST, REM % and disturbed sleep respectively. The high TST variance was a consequence of time in bed primarily determined by the subjects usual sleep duration.

As the anthropometric measures were not independent of each other, partial correlation coefficients were computed partialling out each measure in turn. In the fit subjects the relationship between SWS and percentage LBM was shown to

be independent of any other variable. In contrast, the relationship between total LBM and SWS in the unfit subjects was a composite effect of total weight, height and total LBM in Experiment 3(a) and total weight and LBM in Experiment 3(b). Values for partial correlation coefficients are shown in Table 17 (Appendix A, p.176). Multiple Regression Analysis indicated that in fit subjects no variable added to the variance explained by the proportion of LBM, while in unfit subjects no combination of variables did better than the individual variables alone.

DISCUSSION

The data provide solid evidence for a relationship between body composition and SWS. However, the nature of the relationship appears to vary as a function of physical fitness; SWS being negatively related the the proportion of LBM in fit subjects and body size in the unfit. While this difference was not anticipated, the relationships are relatively strong and robust, the identical pattern being found in two independent studies using different methods of assessing LBM.

The results do not offer direct support for the possibility that previously reported differences in SWS between fit and unfit individuals were entirely due to differences in body composition. Nevertheless, they are compatible with this hypothesis in that differences between groups could have occurred as a function of the particular anthropometric characteristics of each group. The finding in Experiment I that athletes had higher levels of SWS than sedentary individuals irrespective of the physical fitness of the athletes, and the absence of a difference in

Experiment 2 when height and weight were matched are both consistent with the body composition view.

The small number of studies which have directly, or indirectly, investigated the relationship between anthropometric and sleep variables have generally reported contradictory or negative results (see Introduction of this chapter). It is likely that this has been because most studies have measured only weight and because the groups studied have typically been heterogeneous with respect to factors such as age, sex and physical fitness. These are likely to affect sleep directly and to interact with body composition. The present results suggest that in a homogeneous population body composition has an effect on SWS.

CHAPTER 7

EXPERIMENT 4 : THE EFFECTS OF AEROBIC vs POWER TRAINING ON SLEEP

In experiments examining the effect of physical fitness on sleep, athletes have generally been selected on the basis of above average aerobic fitness or performance levels rather than the nature of the subject's training. While these selection procedures have most likely favoured subjects with a substantial aerobic component in their training routine there has been considerable heterogeneity both within and between studies as to the sports in which subjects have participated and therefore with respect to the training programs they would have followed. However, different training regimes develop different physiological attributes (see Chapter 3). It is possible that variations in physiological adaptations to different training routines may contribute to the nature of sleep in athlete groups and that differences between studies in the composition of fit athlete groups in regard to type of training may have produced the discrepant findings reported in the literature.

The differential development in skeletal musculature as a result of different forms of training illustrates this point. Power training results in muscle hypertrophy particularly in fast-twitch fibres, and increases in immediately available energy. Anaerobic training also develops fast-twitch muscle fibres but in addition enhances the ability to produce energy via anaerobic pathways. In contrast, aerobic or endurance training causes a reduction in muscle

size and body fat and an increase in the aerobic energy producing capacity of slow-twitch muscle fibres. In addition, there are many other diverse changes in factors such as cardiovascular functioning, energy metabolism and hormone secretion patterns which occur as a result of different forms of athletic training (see Chapter 3).

Power, anaerobic and aerobic training are not mutually exclusive however, and many sporting activities require the development of all areas over sedentary levels, although superior performance in one area is not compatible with superior performance in another.

A closer examination of the subjects used in those studies which have examined the effect of physical fitness on sleep, reveals a wide variety of sporting activities and thus presumably, of training regimes. Baekeland and Lasky (1966) used athletes involved in sports such as track, swimming and basketball. All the experiments from this laboratory (Griffin and Trinder, 1978; Paxton et al., 1982; Trinder et al., 1982a; Experiment I of this thesis) and Experiment 2 conducted in Edinburgh, have used mixed groups of athletes, including distance runners, hockey players, swimmers, rowers and football players from various codes. Although the source of fitness in Buguet et al.'s (1980) experiment is not described, the high mean VO_{2max} (62.3 ml/kg x min) would suggest a predominance of aerobic training. Zloty et al. (1973) used distance runners while Walker et al. (1978) used cross-country or distance runners although their training programme at the time of the experiment

(interval training for middle distance events) would have developed a combination of anaerobic and aerobic capacity.

It is possible therefore, that the variety in the types of training of athletes used in the different experiments may have contributed to the inconsistency of results in the fitness and sleep literature. Therefore, Experiment 4 was designed to examine the effects of different training regimes on sleep. In addition, body composition variables for the different training groups were assessed and compared.

METHOD

Subjects and Design

The sleep of four groups of 10 paid male volunteers was assessed. The groups were matched as closely as possible for age, the mean age for each group being shown in Table 13. The average age for all subjects combined was 21.98 yrs (S D = 2.29).

The groups varied according to the degree of aerobic vs anaerobic and power training performed by the subjects. Subjects in the first group were endurance athletes, whose training was almost entirely aerobic. A second group contained weightlifters and bodybuilders whose training was almost entirely of the power variety. The third group consisted of subjects whose training consisted of aerobic, anaerobic and power components. These subjects were predominantly football (Australian Rules) and hockey players. The final group consisted of unfit sedentary individuals who were not involved in any training.

Athletes were selected for the aerobic training group if they met three criteria; high competitive achievement in distance running events; high weekly training mileage (mean of 74km/wk, SD = 31.96) and a high VO_{2max} (minimum of 4.0 1/min and 58ml/kg x min). There was one exception. This subject was performing very successfully and training a minimum of 70km/wk but had an unexpectedly low VO_{2max} (3.2 1/min, 44ml/kg x min). It was assumed for reasons described earlier (Chapter 3, p.65) this was an inaccurate estimation of the subjects maximal oxygen uptake. These subjects concentrated almost entirely on aerobic training, in the form of distance running. Three subjects did a small amount of anaerobic training in the form of interval work, and two did some power training (arm strength), but this was in each case a minor component of training.

Subjects were selected for the power training group if they were performing high levels of power training. Half the subjects were weightlifters and the remainder were body-builders. Eight were actively involved in competitions. These subjects were required to be training a minimum of 2 hrs, 5 days a week, and to have been doing so for a minimum of 12 months. There was one exception, a subject who trained 1 hr daily. Subjects in this group trained an average of 12.1 hrs a week (SD = 2.81). VO_{2max} was assessed in this group but not used as a selection criterion. Mean VO_{2max} level is shown in Table 13. While training was predominently weightlifting, there was some development of anaerobic capacity as training regimes generally involved sets of repetitions. There was no aerobic component to training.

TABLE 13

Average age, maximal oxygen uptake and anthropometric measures for the aerobic; mixed aerobic, anaerobic and power; power; and no training groups. Standard deviations are shown in brackets. n = 10 for each group.

	Aerobic		Mixed		Power		No Training	
Age (yrs)	22.30	(2.26)	21.10	(2.18)	22.50	(2.64)	22.00	(2.16)
VO_{2max} (l/min)	4.71	(.82)	4.18	(.653)	3.61	(.743)	2.66	(.245)
VO_{2max} (ml/kg x min)	67.95	(11.44)	55.20	(7.93)	45.85	(6.39)	37.05	(5.76)
Weight (kg)	69.48	(5.78)	75.59	(6.09)	77.95	(9.47)	72.76	(9.61)
Height (cm)	178.20	(5.63)	180.30	(6.49)	176.25	(5.02)	178.05	(6.67)
Weight/Height	.390	(.025)	.419	(.025)	.442	(.049)	.409	(.049)
Quetelet's Index	.2188	(.0124)	.2325	(.0131)	.2509	(.0265)	.2295	(.0278)
LBM (kg)	61.09	(5.58)	65.76	(5.74)	67.15	(7.83)	59.50	(6.50)
Fat (kg)	8.39	(1.67)	9.83	(2.80)	10.80	(2.50)	13.26	(4.98)
% LBM	87.92	(2.39)	87.00	(3.58)	86.14	(2.25)	81.78	(4.85)
$(LBM/Height^2)$ 100	.1924	(.013)	.2023	(.0141)	.2162	(.0225)	.1877	(.0144)

TABLE 14

F and MSe values for comparisons between groups on anthropometric variables

	MSe	Main Effect of Groups df (3,36)	Athletes vs. Non-Athletes df (1,36)	Linear Trend in Athletes df (1,36)	Quadratic Trend in Athletes df (1,36)
VO_{2max} (l/min)	.42	17.92**	39.75**	13.99**	.00
VO_{2max} (ml/kg x min)	66.95	26.14**	41.66**	36.48**	.29
Weight (kg)	63.14	2.12	.29	5.68*	.37
Height (cm)	35.89	.76	.00	.53	1.76
Weight/Height	.00	3.15*	.34	9.07**	.23
Quetelet's Index	.00	3.93*	.32	11.40**	.07
LBM (kg)	42.07	3.20*	4.79*	4.36*	.45
Fat (kg)	10.43	4.02*	9.25*	2.78	.04
% LBM	11.78	5.57**	15.52**	1.20	.00
(LBM/Height²)100	2.75	5.81**	7.11*	10.26**	.07

* $p < .05$

** $p < .01$

Subjects were selected for the mixed training group if they were training or playing a minimum of four times a week at a high competitive level in a sport promoting aerobic and anaerobic metabolism and muscular development. Thus, 4 played Australian Rules Football, 3 hockey, 1 basketball, 1 sprint running and 1 sprint swimming. In addition, relatively high VO_{2max} levels were required (a minimum of 3.50 1/min, 45 ml/kg x min). Training in this group consisted of a combination of power exercises e.g. lifting weights and push-ups; anaerobic exercise e.g. interval sprints; and aerobic endurance running in addition to training in the specific skills of the sport.

Subjects in the final group were selected on the basis of a sedentary lifestyle and a history of non-participation in sport. The maximum VO_{2max} for this group was set at 3.0 1/min and 44 ml/kg x min).

All potential subjects were screened for medical disorders generally and sleeping difficulties in particular.

The sleep of the athletes was assessed on two consecutive nights. On experimental days subjects did not train. In addition to these two nights, as part of another experiment, the athlete subjects slept in the laboratory on two other consecutive nights following daytime training. The non-exercise and exercise conditions were separated by a number of intervening nights the order being counterbalanced across subjects. The unfit, non-athletes slept in the laboratory on four experimental nights. These consisted of two groups of two consecutive nights separated by several non-experimental days.

These subjects did not perform any exercise. In the statistical analyses data from the first two consecutive nights were used for half the subjects and the second two consecutive nights for the other half. Thus, the experimental design consisted of a groups (4) x nights (2) factorial with repeated measures on the second variable. All subjects were given one adaptation night several nights preceeding the experimental sessions.

Procedure

The aerobic fitness level of each subject was assessed, as in Experiment I and 3(a), by a sub-maximal bicycle ergonometer test (Åstrand and Rodahl, 1977). Average VO_{2max} values are shown in Table 13.

Anthropometric data were collected on all subjects. The measures recorded were weight, height, weight/height ratio, Quetelet's Index, LBM, body fat and LBM/height^2 x 100. These measurements were all made during the period of sleep assessment. LBM and fat were determined using procedures described by Durnin and Womersley (1974). Measurements of skinfold thickness were made at four sites, tricep, bicep, subscapular and supra-iliac. From these, percent LBM and fat were derived. Unfortunately, it was not possible to measure muscle fibre structure, although, it was anticipated that assumptions could be reasonably made as to the predominant muscle fibres and energy pathways on the basis of the nature of the subjects training.

It was not feasible to measure accurately the energy expenditure of subjects. On the basis of the information

on sporting activities however, there is no doubt that all athlete groups expended considerably more energy than the non-athlete control subjects. Further, considering the high mileage of the aerobic group and the high number of training hours of the power group it is quite likely that the average energy expenditures of these groups were approximately equivalent and exceeded that of the mixed training group, although this is only speculative.

On sleep assessment nights subjects came into the laboratory at 8.00pm. Following the attachment of electrodes and a number of other laboratory procedures, they were put to bed at approximately 9.00pm. Lights were turned out at a time of the subject's own choosing and they were allowed to read , study, listen to the radio, or watch T.V. in bed. Subjects remained in bed in the morning until they requested to get up, they had been awake for 30 consecutive minutes after 7.00am or it was 9.00am. This procedure was followed in order to provide a better estimation of the total sleep requirements of the subjects than that which is available from laboratory procedures which prescribe a constant lights out period.

The sleep records were obtained and scored blind according to standardised procedures (Rechtschaffen and Kales, 1968). Two scorers scored each record independently. Epochs over which there was disagreement were rescored by a third scorer whose assessment was final. The usual sleep variables were then derived from the scored records.

The statistical analyses were arranged to test two hypotheses. First, did the sleep of the unfit non-athlete group differ from that of the athletes ? Second, did the sleep of the athlete subjects differ as a function of the type of physical training ? The hypotheses were tested using pre-planned comparisons. The unfit non-athletes were compared with the three athlete groups (Hypothesis I), and the linear and quadratic trends across the three athlete groups along a continuum from power but no aerobic training; power, anaerobic and aerobic training combined; to aerobic training only, were determined (Hypothesis 2). Thus the analysis of the sleep data consisted of a 4 x 2 factorial (groups x nights) in which the three degrees of freedom from the main effect of groups were used as described above. The anthropometric data were analysed using a single factor design as in the groups factor above. Separate analyses were conducted for the total lights out period and the first 8 hrs after lights out. The pattern of results was unchanged so only the data for the total period are reported. In addition some variables were analysed for the first 150 mins after lights out and these are reported where appropriate. Group means for selected sleep variables are presented in Table 15. F values and significance levels for the analyses for the main effect of groups and the three planned comparisons for the major sleep variables are presented in Table 16. F values for the analyses of anthropometic variables are shown in Table 14. The significance level was set at $p < .05$.

RESULTS

Anthropometric and Fitness Variables

As would be expected on the basis of selection procedures, there was a main effect of groups for both VO_{2max} measures, with athletes having significantly higher levels than non-athletes. In addition, there was a significant linear trend within athlete groups such that the power training group had the lowest and aerobic training group the highest VO_{2max}. Anthropometric measures also varied between groups in a manner predicted by the usual physiological adaptations to specific types of training, or lack of training. Thus, analyses of body composition indicated that non-athletes had significantly lower total LBM, percent LBM and (LBM/height^2)100, but higher total fat, than athletes. Within athlete groups there was a significant linear trend for weight, despite the groups being approximately the same height, the power training group being the heaviest and the aerobic training group the lightest. Compatible with this, significant linear trends were also observed for weight/height and the Quetelet's Index. There was no difference between the athlete groups on percent LBM. There were significant linear trends, however, for total LBM and (LBM/height^2)100, aerobic athletes being the lowest and power athletes the highest on these measures. The main effect of groups was significant for weight/height, Quetelet's Index, LBM (kg), fat (kg), % LBM and (LBM/height^2)100.

Sleep Variables : Athletes vs Non-Athletes

The initial pre-planned comparison revealed no significant differences between non-athletes and athletes

on any sleep variable. Percent REM was the only variable to approach significance ($p = .086$) in this set of analyses, non-athletes having a tendency towards higher percent REM than athletes. This was most likely due, however, to a suppression in REM in the aerobic and power training groups which is discussed later.

Sleep Variables : Effects of Type of Training

In contrast to the athletes vs non-athletes comparisons substantial differences were revealed in comparisons between groups of different type of training (see Table 16).

A significant main effect of groups was found for SWS. Of particular interest however, was a significant linear trend between SWS level and degree of power and aerobic training such that the greater the power component in training the lower the level of SWS, while the higher the aerobic component the higher the level of SWS. The mixed group, which had elements of power, anaerobic and aerobic training had intermediate levels of SWS. Consistent with total SWS, the linear trend for SWS as a percentage of TST was also significant.

There was no significant difference between the groups for latency to SWS, although aerobically trained subjects had higher levels of SWS during the first 150 mins of the night than the power athletes, as indicated by a significant groups effect and a significant linear trend in the three training groups.

The difference in SWS level between the groups was attributable almost entirely to differences in Stage 4 rather

TABLE 15

Means for selected sleep variables for the aerobic; mixed aerobic, anaerobic and power; power; and no training groups. All values are given in minutes and standard deviations are shown in minutes. n = 10 for each group.

	Aerobic	Mixed	Power	No Training
Time in bed	606 (56.7)	591 (45.3)	546 (101.8)	576 (47.6)
Sleep period time	575 (60.6)	557 (42.3)	517 (113.8)	550 (50.3)
Total sleep time	550 (64.7)	535 (50.5)	506 (108.1)	533 (47.2)
Total time awake	56 (40.6)	56 (42.1)	40 (49.8)	43 (27.2)
Stages I + MT	64 (30.5)	61 (15.4)	71 (40.5)	65 (16.2)
Stage 2	278 (49.8)	280 (43.8)	271 (55.1)	269 (35.3)
Stage 3	34 (18.2)	34 (17.3)	32 (11.4)	30 (10.2)
Stage 4	53 (14.6)	35 (27.5)	29 (31.0)	44 (18.7)
SWS (3 + 4)	87 (26.0)	69 (21.7)	61 (33.0)	74 (22.8)
NREM (2 + 3 + 4)	365 (45.0)	349 (41.9)	332 (77.5)	343 (31.4)
REM	121 (27.1)	125 (28.0)	103 (34.1)	125 (23.7)
Sleep onset latency	17 (8.1)	26 (25.5)	33 (52.0)	25 (19.2)
Stage 3 latency	13 (3.4)	12 (4.1)	21 (27.3)	15 (6.5)
REM latency	88 (46.2)	73 (19.7)	91 (44.7)	81 (32.3)

TABLE 16

F and MSe values for comparisons between groups on selected sleep variables. Sleep variables were analysed in minutes except where otherwise indicated. (* = $p < .05$; ** = $p < .01$)

	MSe	Main Effect of Groups df (3,36)	Athletes vs. Non-athletes df (1,36)	Linear Trend in Athletes df (1,36)	Quadratic Trend in Athletes df (1,36)
Time in bed	4280	3.11*	.02	8.57**	.69
Sleep period time	5355	2.21	.02	6.31*	.32
Total sleep time	5192	1.33	0	3.84	.14
Sleep efficiency (TST/TIB)	.006	.34	.02	.68	.09
Total time awake	1672	1.03	1.08	1.50	.51
Stage I + MT	761	.53	.24	.67	.67
Wake + I + MT (in TIB)	2535	.25	.45	.30	.02
Wake + I + MT (% TIB)	73	.16	.41	.05	.01
Wake + I + MT (in SPT)	1718	.10	.02	.23	.05
Wake + I + MT (% SPT)	58	.05	.01	.01	.19
Stage 2	2375	.22	.27	.22	.20
Stage 3	227	.12	.04	.22	.11

TABLE 16 Contd.

	MSe	Main Effect of Groups df (3,36)	Athletes vs. Non-athletes df (1,36)	Linear Trend in Athletes df (1,36)	Quadratic Trend in Athletes df (1,36)
Stage 4	667	3.62*	1.17	8.94**	.81
SWS (3 + 4)	800	3.08	.04	8.83**	.40
SWS (% TST)	24	2.40	.08	6.77*	.36
SWS (1st 150 min)	302	3.97*	.23	10.99*	.78
NREM (2 + 3 + 4)	2623	1.45	0	4.37*	.00
REM	854	2.89*	2.06	3.86	2.88
REM (% TST)	15	2.80*	3.12	1.88	3.78
Sleep onset latency	953	.96	.01	2.85	.01
Stage 3 latency	200	1.53	.30	2.86	1.42
REM latency	1358	.96	.15	.07	2.81

than Stage 3 as can be seen from Table 15. The main effect of groups and the linear trend analysis were significant for Stage 4. Thus, as with SWS, while there was no effect of training vs no training, of the subjects in training, those with the highest aerobic component had the highest level of Stage 4. Comparisons between groups for Stage 3 found no significant differences.

Similarly, a linear trend within athlete groups was observed for total NREM sleep, aerobic athletes having the highest and power athletes the lowest NREM sleep. Again the difference between the groups in Stage 4 was the main contributing component to this effect and no significant differences between the groups were found for Stage 2.

Experiment 4 also showed different training groups to vary according to sleep duration, with the aerobic group having the largest and the power group the shortest sleep duration. A significant main effect of groups and linear trend were found for TIB (where TIB was the lights-out period). SPT also showed a significant linear trend and TST showed a strong but non-significant tendency ($p = .058$) in this direction.

Sleep onset latency showed relatively large group differences with the power group having delayed onset. While the main effects were not significant there was a significant interaction between nights and the linear trend over athlete groups ($F(1/36)=5.01$, $MSe = 110$). The interaction was primarily due to the aerobic group being lower on night 1 than 2, though they had the shortest SOL on each night.

REM variables showed some minor differences between groups. There was a significant main effect of groups for total REM and both the linear ($p = .057$) and quadratic ($p = .087$) trends approached significance. Non-athletes had the highest total REM. Of the athletes, the power training group had the lowest and the mixed athletes the highest REM, with the aerobic training group having a REM level closest to the mixed group. These results were however influenced by the different TSTs and when REM is analysed as a percent TST the main effect of groups approaches significance ($p = .054$) and the difference in REM between non-athletes and athletes becomes more apparent ($p = .086$). Considering the high TST of the aerobic and mixed groups this result appears to be due more to a REM suppression in athletes than an elevation in non-athletes. The quadratic trend also approaches significance ($p = .056$) in athlete groups in a pattern similar to total REM. There were no differences between the groups for latency to REM or amount of REM in the first 150 mins and first 8 hrs. Thus, there are some tentative indications that training may affect REM sleep, however, the data are generally non-significant.

Finally, there were no significant differences between groups in amount of disturbed sleep (wake + MT + Stage I) in minutes, as a percentage of TIB or as a percentage of SPT. In addition there was no significant difference between groups in sleep efficiency (TST/TIB).

Sleep Variables : Effect of Nights

There were three significant differences involving the comparison of the first and second night. They were an

increase in Stage 3 on night 2 ($F(1/36) = 4.78$, $MSe = 67.8$), a significant interaction between nights and the athlete vs non-athlete comparison for minutes of Stage I + MT ($F(1/36) = 4.60$, $MSe = 217$) and, as mentioned, an interaction between nights and the linear trend over athlete groups for SOL. With the exception of the SOL effect, these effects do not appear to be of particular importance.

DISCUSSION

Experiment 4 clearly shows that the sleep of athletes with different training regimes differs in a number of major respects. The aerobically trained endurance runners had the highest levels of SWS and NREM sleep, slept longer and tended to have shorter SOLs than those subjects who emphasized power training. The mixed power, anaerobic and aerobic training group were intermediate with respect to each of these variables. These differences were not accompanied by differences in disturbed sleep, though SOL might be so interpreted. The non-athlete level of SWS lies between the mixed training and aerobic training group while for NREM, sleep duration and SOL the non-athlete and mixed training groups were approximately equivalent.

The results are consistent with the analysis of body composition variables reported in Chapter 6 in showing the relevance of peripheral physiology to sleep; particularly to SWS and NREM. The data have demonstrated effects of both body composition, notably amount and proportion of LBM, and the type of physical training. Experiment 3 showed the proportion of LBM to be negatively related to SWS levels in a group of athletes from various training backgrounds, while

the amount of LBM and total weight were negatively related to SWS in sedentary individuals. In the present data the type of training was shown to affect both SWS and sleep length, while the proportion of LBM was held constant between athlete groups. However, the data do not provide any information as to the possible interaction effects between body composition and type of athletic training, nor is there any evidence as to the relative strengths of these two factors. Further, it could not be determined with any certainty whether the pattern of results in the present experiment were due to variations in level of aerobic training, power training, both, or some other variable, though some conclusions are possible.

In accord with the findings of Experiment 1 and 2, VO_{2max} does not seem to be a critical factor. There was no difference in SWS or sleep duration between the athlete groups and the sedentary group though there was a significant difference between these two groups on VO_{2max} measures. It is not possible to be as certain as to the role of power training and, because an independent assessment of the consequences of such training was not available, it is unclear as to the position of the control group with respect to each of the athlete groups.

The role of weight or amount of LBM is unclear. In the present study it was associated with, and presumably a consequence of, type of athletic training. It is therefore possible that, of the various adaptations to the training routines, it is weight or amount of LBM which is the critical variable. However, in the body composition data reported in

Chapter 6, weight was unrelated to sleep in athletes, although it did show a relationship with SWS in sedentary individuals.

Variations in energy expenditure are unlikely to account for differences in SWS or sleep duration. As discussed earlier, energy expenditure was not measured, However there is little doubt considering their training regimes that athletes expended considerably more energy than non-athletes though the athlete vs non-athlete comparison showed no difference in sleep between the two groups. In addition, it was estimated that the aerobic and power athletes probably expended an equivalent amount of energy yet these two groups had SWS and sleep duration levels at opposite extremes.

It is of interest that when subjects were encouraged to sleep as long as they felt they needed, in general they slept over an hour longer than in either Experiment 1 or 2. It is unclear whether this reflects a response to a minor degree of sleep deprivation or just the ability to sleep longer given the opportunity. It also appears that the experimental procedures followed enabled differences between the groups in sleep duration to be revealed while it is quite probable that a restricted sleep length would have masked this effect.

There is some suggestion of REM suppression in both the aerobic and power training groups. This may be a consequence of physiological or psychological stress from the particularly heavy training schedules.

As noted earlier these results suggest that some peripheral factors are capable of affecting SWS. While not yet clearly identified these may include factors known to be affected by different forms of physical training such as body composition, muscle development, metabolism or hormone secretions. Many of these factors are also related to VO_{2max}. Thus, the selection of subjects according either to aerobic fitness criteria or the performance of athletic activities in general, may have produced quite heterogeneous samples in terms of the crucial factor(s) affecting SWS. Thus, subject selection procedures may have accounted for the inconsistency of outcomes across studies of the sleep of athletes.

CHAPTER 8

CONCLUSIONS

Summary of Empirical Findings

The experiments reported in this thesis are concerned with the effects of physical fitness on sleep. The available literature at the time these experiments were commenced indicated that physically fit individuals have more SWS (Baekeland and Lasky, 1966; Buguet et al. 1980; Griffin and Trinder, 1978; Trinder et al. 1982a; Zloty et al., 1973), have more total NREM (Walker et al., 1978), sleep longer and have shorter SOLs (Montgomery et al., 1983), than unfit individuals. However, there were also contrary findings (Paxton et al., 1982; Walker et al., 1978). These experiments defined physical fitness in terms of aerobic fitness though few studies actually reported VO_{2max} values. It was considered possible that the contradictory nature of the literature could have occurred because the critical factor affecting sleep was a variable associated with aerobic fitness or athletic activities. If this factor showed a variable relationship with aerobic fitness, for example, the conflicting results might then be accounted for.

Experiment 1 tested this hypothesis by varying the level of aerobic fitness within subjects who were proficient athletes. Thus the sleep of the athletes when unfit was compared with their sleep when they were aerobically fit. Their sleep was unchanged though they had more SWS, more NREM sleep and longer sleep durations (as defined by SPT) on both testing occasions than an unfit, non-athletic,

sedentary control group. This finding was consistent with the view that aerobic fitness was not the critical variable.

There was a large literature which had looked at the effects of exercise on the sleep of the immediately following night (reviewed by Horne, 1981; Shapiro, 1981; Torsvall, 1981). As with the physical fitness effect it was contradictory, particularly regarding the major finding of an increase in SWS following exercise. It had been hypothesized that the contradictions were a consequence of the "exercise effect" being confined to the physically fit subjects (Griffin and Trinder, 1978). This issue was investigated in Experiment 1. The athlete subjects were tested following both exercise and non-exercise days when they were both unfit and fit. The exercise level used was "their usual training program at an intensity sufficient to be exhausting but not stressful" (Chapter 4, p. 75). No evidence for an exercise effect at either level of fitness was obtained. In retrospect the exercise level may not have been sufficiently intense (see Horne, 1981). However, this issue was not pursued further in the studies reported in this thesis.

Experiment 2 was primarily concerned with the effect of physical fitness on the night-time plasma levels of the hormones hGH, prolactin and cortisol. These hormones were assessed in a group of fit athletes and unfit non-athletes. Human growth hormone secretions during the first four hours of sleep did not differ significantly between groups, either in terms of area under the curve or peak plasma concentration although there was a marginal trend in this direction ($p < .10$).

Analyses of cortisol secretion revealed no main effect of groups. It did indicate a groups by trials interaction such that cortisol concentrations were lower in athletes than non-athletes early in the night but, higher in athletes later in the night. This was not a particularly powerful effect however, as the interaction failed to reach significance for averaged hourly concentrations. In addition, while post hoc analyses based on the original ANOVA showed athletes to have significantly lower cortisol values for the first 7 trials a non-parametric test was significant for trials 4 and 7 only.

Human growth hormone and cortisol considered independently did not show strong effects. However, the combination of high hGH and low cortisol during the early part of the night was more extreme in athletes than non-athletes. No differences were observed between groups for prolactin secretion. Thus, while the effects observed were not strong enough to enable the rejection of the null hypothesis, their marginal nature encourage further investigation of the hypothesis. In retrospect, in view of the results of Experiment 4, a stronger effect may have been achieved using an endurance rather than a mixed group of athletes. This possibility is supported by the sensitivity of the hGH response to exercise in endurance trained athletes (see Chapter 3, p. 55).

An investigation of the relationship within subjects between night-time hormone secretions and both sleep and body composition variables was made. It revealed a significant

negative correlation between hGH in the first 4 hrs of sleep and total SWS. This relationship disappeared however, when hGH in the first 4 hrs and SWS during the first 4 hrs were correlated, so the relevance of the overall relationship is unclear. A significant positive relationship between hGH and proportion of LBM and a significant negative relationship between hGH and total body fat were found. There was no relationship between hGH and LBM. Thus, it appears that fat level is the most critical of the body composition variables assessed in predicting night-time hGH secretion. This finding is consistent with that of Kalkoff and Ferrou (1972) who found, using an insulin induced hypoglycemia test in awake subjects, that low hGH secretion was related more to body fat accumulation than excess LBM. As hGH is involved in energy metabolism, increasing concentrations of FFA when blood glucose is low, the relationship between fat and night-time hGH appears more consistent with the possibility proposed by Horne (1979) that hGH secretion during sleep may be related to fat metabolism rather than protein synthesis.

Experiment 2 was commenced before the data from Experiment 1 were available, but after the trend of the results was evident. At this time it was considered possible that body composition might be the critical factor rather than aerobic fitness. But body composition is known to be related to physical fitness and it was considered possible that it was related to sleep. In accordance with this hypothesis body composition measurements were taken on subjects being run in various experiments in the Hobart laboratory at that

time. In addition, similar measurements were collected on subjects in Experiment 2. The two sets of data are reported in Chapter 6. The data were in part, consistent with the hypothesis. In each experiment SWS was systematically related to LBM. In physically fit athletes the proportion of LBM was negatively correlated with SWS while in unfit non-athletes total LBM and weight were negatively correlated with SWS. This pattern was the same in the two experiments.

The results of these studies clearly demonstrated an effect of body composition on SWS. Other sleep variables such as SOL and sleep duration were not related to body composition. While the data suggested the possibility that variations in the anthropometric characteristics of subject groups run in previous experiments could account for variations in outcomes there is no direct evidence to support this view as little anthropometric data have been reported in previous studies. However, the high SWS levels of athletes in Experiment 1 could have been a result of the relatively low LBM of this group. It should be remembered though, that subjects in this experiment were part of the sample analysed in Experiment 3a (Chapter 6). The failure to find a SWS difference in Experiment 2 in which the athletes and non-athletes were matched for anthropometric characteristics could also be considered evidence for this view. However, as the body composition analysis has identified a different variable for fit and unfit subjects it is not clear to what extent true matching has been achieved. Thus, the percentage LBM in fit athletes equivalent to a partic-

ular weight in unfit non-athletes is unknown. In summary, body composition has been shown to affect SWS. However it is unlikely that body composition entirely accounts for the previously reported effects of physical fitness on sleep.

In view of these and other considerations the final experiment assessed the effect of type of athletic training on sleep. As in the case of body composition, this factor had been allowed to vary unsystematically, both within and between previous studies. Accordingly the sleep of four groups of subjects was compared : aerobically trained endurance runners, power trained weight lifters and body builders, athletes who engaged in a combination of aerobic, anaerobic and power training and an untrained sedentary control group.

The results were clear. The athletes did not differ from the non-athletes on any sleep variable. In contrast, there were a number of differences between the athlete groups. The aerobically trained group had more SWS and NREM sleep, slept longer and went to sleep more rapidly (though the data on this latter point are less clear) than the power trained athletes. The group with the more mixed training routine were intermediate in each variable. These differences were not accompanied by differences in disturbed sleep.

These data are consistent with those of Experiment 3 in that they emphasize that variations in peripheral factors affect sleep architecture; particularly SWS. However, these

experiments show apparently independent effects of body composition and type of athletic training. It is not possible to determine from these studies if the two effects are related in any way; whether they may interact; or be influenced by a common mechanism.

It could not be determined what effect of training is the critical one regarding the differences observed in sleep. Variation in weight is a possibility suggested by Experiment 4 but not supported by the correlational analyses in athletes in Experiment 3. VO_{2max} does not appear to be crucial as in Experiment 4 the unfit control group did not differ from the athlete groups and were approximately equivalent to the mixed group on all sleep variables, despite their aerobic fitness levels being markedly different. This is also compatible with the findings of Experiment 1 and 2. Variation in energy expenditure also appears not to be a critical factor as there was no difference in SWS levels between all athletes combined and non-athletes, despite there being, most probably, large differences in average daily energy expenditure between these two groups. Moreover, while aerobic and power athletes probably expended similar amounts of energy there were large discrepancies in their levels of SWS, NREM sleep, sleep length and SOL.

Other factors related to training were not assessed. Thus their influence could not be determined. As discussed in Chapter 3 however, many other aspects of physiology change with training of different types and one of these could therefore be an important influence on sleep. These include the proportion of different energy pathways used in metabolism,

especially during exercise; RMR; muscle fibre, and skeletal development; and patterns of hormone secretion. Future research may shed more light on this issue.

As in the case of the body composition data a retrospective analysis of the literature in the light of the effects of training on sleep is difficult because of insufficient information as to the nature of the training routines the subjects used. Nevertheless, some insight into the literature is possible. The positive finding of Zloty et al. (1973) (though it should be remembered that the study did not use an unfit control group) and the negative results of Walker et al. (1978) may be a consequence of the former using long distance runners and the latter using runners trained for middle distance and who, as a consequence would have had a substantial anaerobic capacity. In our own laboratory, although, we have typically used subjects from a number of different sports, each study has included some distance runners and we have always stressed aerobic fitness in our subject selection procedures (Griffin and Trinder, 1978; Trinder et al., 1982a). However, we have also failed to find elevated SWS in fit subjects (Paxton et al., 1982). Thus while some of the literature is interpretable in terms of type of training effects this form of retrospective analysis cannot be pursued with confidence.

Theoretical Implications of the Present Results

The early experiments investigating the effects of physical fitness and exercise on sleep were conducted to test theories that sleep and peripheral metabolism were related. The present results from experiments investigating the relationships between aerobic fitness, body composition and type of training on sleep variables contribute to the current debate regarding the functions of sleep.

Restorative Theory : The restorative theory proposes that SWS is an optimal time for protein synthesis (Adam, 1980; Adam and Oswald, 1977; Adam and Oswald, 1983; Oswald, 1980). The cellular energy charge conditions and the hormonal environment appear particularly favourable for anabolic processes to predominate. Amongst evidence cited in support of the restorative theory are observations which suggest a positive relationship between metabolic requirements and amount of SWS and TST. Oswald (1980) argues that the higher the metabolic rate and consequent degradation during the day, the larger is the required period for compensatory synthesis at night. Therefore, the restorative theory would predict SWS to be elevated during periods of physical training. It would not discriminate between different types of training as they all result in increased anabolic requirements although these take place in different aspects of physiology (Chapter 3, pp.51-57).

Thus the restorative theory predicts that SWS will be higher in trained athletes than unfit sedentary subjects,

higher in athletes in training than out of training, and equally high in athletes in all types of training, if of approximately equivalent anabolic requirements. This was not found to be the case, however. Experiments 2 and 4 found SWS levels to be similar in unfit non-athletes and athletes in training; Experiment 1 indicated no difference in SWS between athletes when out of training and the same athletes when in training; and Experiment 4 found, despite equally heavy training schedules, aerobically trained athletes to have significantly more SWS than power trained athletes.

It would also have been anticipated by the restorative theory that secretion of hGH would be higher and cortisol secretion lower, early in the night, in fit athletes than unfit non-athletes. The data reported in Chapter 5 do not allow this conclusion to be confidently made as the difference in fit and unfit groups in hGH secretion, while in the predicted direction failed to reach significance and the interaction between groups and trials for cortisol secretion was not a particularly strong one. None-the-less, the marginal nature of the results suggest that future studies may reveal a relationship between training and night-time hormone secretion obscured in Experiment 2. Thus, while the restorative theory of SWS cannot be supported it certainly cannot be rejected on the strength of the hormone results.

A final aspect of the results which cannot be regarded as consistent with the restorative theory is the negative

relationship found between LBM (kg) and minutes of SWS in unfit subjects and % LBM and SWS in fit athletes. LBM being the active body tissue it would be predicted that greater LBM would require greater anabolic activity and consequently higher SWS. This clearly was not the case.

The restorative theory of SWS as presently conceptuallized is not supported by the findings in this series of experiments. However, the predictions made depend on the assumption that increased restoration will be reflected in increased duration of SWS and increased hGH secretion. This assumption is quite explicit in the literature and was certainly the basis of this series of experiments. If this were not the case the restorative theory could accommodate the present data using one or more of the following hypotheses.

1. Some qualitative aspect of SWS, such as intensity may be of greater relevance than number of minutes of SWS.
2. If greater protein synthesis is required more may occur early in the SWS period.
3. There may be some optimal or minimum level of hGH required for synthetic processes to occur and quantity of hGH secretion per se may not be the critical issue in maximising protein synthesis.
4. Sleep architecture and protein synthesis are parallel but independent processes, synthesis perhaps being under circadian control and entrained to sleep but not affecting it.

The theory that SWS provides an optimal environment for restorative processes cannot be rejected but present interpretations of the empirical consequences of the theory must

be reconsidered as the data from this series of experiments are contrary to the restorative theory as it is currently expressed.

Energy Conservation Theory : The energy conservation theory proposes that the major function of sleep, and SWS particularly, is to enforce rest, thereby limiting metabolic requirement and conserving energy supplies (Allison and Van Twyver, 1970; Berger, 1975; Berger, 1984; Snyder, 1966; Walker and Berger, 1980; Zepelin and Rechtschaffen, 1974). This theory postulates that one physiological adaptation to high metabolic expenditure is the elevation of SWS and TST or, in certain animals, the occurrence of hibernation or torpor. An alternative adaptation to high energy expenditure is the increase in caloric consumption. The particular mode of energy consumption most likely to be used by a particular species depends on ecological factors (such as food availability and predation status) and physiological factors (such as adiposity, body size and metabolic rate) (Berger, 1984).

While this theory has generally been applied to explain between species differences in sleep patterns, it has also been hypothesized that sleep may be responsive to variations in energy expenditure within species and within individuals. Therefore, the observation of higher levels of SWS and TST in athletes with high levels of energy expenditure over non-athletes of sedentary lifestyles, would be consistent with the energy conservation theory of sleep. However, in the absence of information regarding energy intake, the failure to observe such an effect is not necessarily inconsistent with the theory.

The results of the experiments reported in this thesis indicate that athletes as a broad group do not use the elevation of SWS and TST to compensate for high energy expenditure. Thus, in Experiment I athletes had similar levels of SWS both when in and out of training. Since it is probably reasonable to assume daily energy expenditure of fit athletes to be higher than unfit non-athletes the finding that these two groups have similar SWS levels in Experiment 2 also indicates that the athletes have not balanced their energy intake and output equation by increasing time in SWS or TST. Rather, it is most likely that athletes have increased their energy intake.

Results from Experiment 4 indicate that the method of balancing the energy intake/expenditure equation may vary depending on factors related to the type of training performed. As in the previous experiments, energy expenditure levels were not measured in Experiment 4 but as mentioned earlier (Chapter 7, p.133) it is most likely aerobic and power athletes expended approximately equivalent levels of energy. However, the aerobic training group had high while the power training group had low SWS and sleep duration measures. It could be speculated that the endurance athletes used increases in SWS and TST while power training athletes used dietary measures to achieve energy balance.

Certainly, some sports, especially power training, often encourage particular diets. Further, a number of studies have shown nutritional level to influence sleep

patterns (Crisp, 1980; Crisp and Stonehill, 1976). It may be hypothesized, therefore, that observed differences in sleep between different training groups are in fact related to differences in diet or energy intake. Such factors may also explain the intermediate levels of SWS in the non-athletes in Experiment 4, since neither differences in constitution variables nor energy expenditure appear to do so.

While it does not appear that SWS is increased in most trained athletes as a consequence of generally high energy expenditure, other design factors could have obscured energy balance effects on sleep. Experiments 2 and 4 assessed sleep on non-exercise nights only and Experiment 1 used quite moderate levels of exercise. If SWS and/or sleep duration respond to changes in energy balance created by extreme increases in energy expenditure during the immediately preceding day this would not have been revealed by the experimental designs used. A further design problem in Experiments 1 and 2 is related to the capacity of the experiments to reveal differences in sleep duration between groups and conditions. Sleep duration was not restricted but subjects determined their time in bed largely according to their perceptions of their usual sleep requirements. This may not have accurately reflected current sleep need. However, in Experiment 4 conditions were desinged to more accurately reveal genuine sleep requirements.

Predictions made regarding SWS and sleep duration on the basis of the energy conservation theory have, like

those from the restorative theory, assumed the length of time spent in SWS or sleep generally is the critical variable. Again, this may be incorrect. It may be, for example that a greater degree of energy conservation is achieved in fit athletes through a drop in core temperature rather than in an extension of SWS or sleep duration.

In conclusion, while the experiments described in this thesis do not test the energy conservation theory of sleep directly, they do indicate that athletes as a broad group do not use the elevation of SWS or TST to compensate for high energy expenditure. It is possible, however, that a sub-group of athletes (endurance athletes) may use this method to balance their energy intake and energy expenditure.

The Cerebral Restitution and Immobilization Theories : Two further theories propose that sleep has no peripheral function. One proposes that SWS serves an exclusively cerebral restitution role (Feinberg, 1974; Horne, 1978; 1979; 1981; 1983) perhaps by reversing some neuronal consequences of waking during a "shut-down" period (Horne, 1983, p.575). The second, the immobilization theory, views sleep as a behavioural adaptation, the prime function of which is to maintain immobility at times when immobility might be expected to improve an animals chances of survival e.g. during the dark period for man (Meddis, 1975; 1977; Webb, 1971; 1974). Neither of these theories predict any effect of peripheral metabolism per se on the nature of sleep and therefore predict no effect of exercise or training on sleep.

The present results would appear contradictory to both these theories as both training and body build, factors related to peripheral metabolism, are related to SWS. Since the effects of training were observed on days following no exercise these results cannot be attributed to a brain warming effect of exercise itself. Thus, while sleep may serve cerebral restitution and immobilizing roles, hypotheses that were not tested in this series of experiments, sleep also appears to be influenced by peripheral factors.

The experiments reported in this thesis commenced with the observation that SWS and sleep duration were elevated while SOL was decreased in fit athletes compared with unfit sedentary individuals. It had previously been hypothesized that the critical variable in these effects was the level of physical fitness resulting from habitual exercise. Findings from a number of studies, however, had cast doubt on this interpretation Data from this thesis indicates that variations in physical fitness and energy expenditure are not sufficient explanations to account for the sleep differences. Variations in body composition and type of physical training are more precise determinants of both sleep architecture and night-time secretion of hGH. The data clearly show the relevance of peripheral physiological factors on sleep although the particular mechanisms involved are not identified. These findings appear to have implications for a number of the theories concerning the function of sleep, although, since the mechanisms involved are not understood, theoretical interpretations remain speculative.

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APPENDIX A.

TABLE 17

Partial correlation coefficients for Experiments 3a and 3b showing the relationship between SWS and the anthropometric variables.

Anthropometric Variables	Fit		Unfit		Variable Partialed Out
	Exp.3a	Exp.3b	Exp.3a	Exp.3b	
Weight	-.07	.14	-.53*	-.63**	
Height	.03	.08	-.67*	.02	None
Fat (kg)	.41	.38	-.17	.02	
LBM (kg)	-.02	-.17	-.55	-.68**	
% LBM	-.54*	-.54*	-.01	-.33	
Height	.07	-.05	-.58*	-.16	
Fat (kg)	.57**	.36	.05	.46	
LBM (kg)	.10	-.38	-.22	-.51*	Weight
% LBM	-.58**	-.53*	-.06	-.53*	

Anthropometric Variables	Fit		Unfit		Variable Partialed Out
	Exp. 3a	Exp.3b	Exp.3a	Exp.3b	
Weight	-.09	.07	-.37	-.62**	
Fat (kg)	.42	.38	-.41	.04	
LBM (kg)	-.04	-.27	-.33	-.74**	Height
% LBM	-.54*	-.54*	.16	-.43	
Weight	-.44*	.00	-.51*	-.73**	
Height	-.10	-.02	-.72**	.04	
LBM (kg)	-.19	-.04	-.56*	.70**	Fat
% LBM	-.41	-.54*	-.46	.72**	
Weight	-.13	.37	-.13	-.58*	
Height	.05	.23	-.55**	.33	LBM
Fat (kg)	.45*	.39	-.21	-.38	
% LBM	-.54*	-.55*	.22	.29	
Weight	-.26	.07	-.53**	-.39	
Height	.02	.10	-.68*	.30	
Fat (kg)	-.18	-.38	-.46	-.61**	% LBM
LBM (kg)	.00	.18	-.59*	-.69**	

* $p < .05$

** $p < .01$

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