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From Wake to Sleep

The Effects of Acute Exercise on Sleep: A Quantitative Synthesis

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Summary: We used meta-analytic methods to examine the influence of acute exercise on sleep. Thirty-eight studies were reviewed yielding 211 effects on 401 subjects. Mean effect sizes were calculated for sleep onset latency (SOL), stage 2, slow-wave sleep (SWS), rapid eye movement (REM) sleep, REM latency (REM-L), total sleep time (TST), and wakefulness after sleep onset (WASO). Moderating influences of subject fitness, heat load, exercise duration, time of day, associated light environment (i.e. indoor or outdoor), sleep schedule, and the scientific quality of the studies were examined.

Effect sizes for SWS, REM, REM-L, and TST were moderate [0.18–0.52 standard deviation (SD)] and their associated 95% confidence intervals did not include zero. Exercise duration and time of day were the most consistent moderator variables. In contrast with previous hypotheses, heat load had little influence on sleep.

The results of our quantitative synthesis of the literature are inconsistent with previous narrative reviews (1,2) which suggested that exercise elicits larger changes in sleep than those quantified in this meta-analysis. A major delimitation of published studies on the effects of acute exercise has been an exclusive focus on good sleepers. Hence, the effects we report herein may be underestimates of the efficacy of exercise for enhancing sleep among people with sleep disturbances. **Key words:** Exercise—Physical activity—Sleep—Meta analysis—Duration—Time of day—Temperature—Bright light.

It is generally expected that acute daytime exercise will enhance sleep. This expectancy is commonly expressed in our daily conversation and may arise from personal experience (3). This expectancy has also been reinforced over the years by an apparent consensus in the sleep community (1,4,5) by primary care physicians, by exercise scientists (6,7), by numerous articles in the lay literature, and by traditional theories regarding the function of sleep [e.g. body restitution (8) and energy conservation (9,10)]. Indeed, surveys indicate that few behaviors are as closely linked with enhanced sleep as exercise (3,11). However, experimental evidence supporting the idea that exercise enhances sleep is not compelling, and recent reviews have challenged the robustness and generalizability of exercise effects on sleep (12,13). For example, though it is supposed that acute exercise increases slow-wave sleep (SWS), Trinder et al. (13) noted that the majority of studies have failed to show a significant increase in SWS following exercise. Moreover, the practical relevance of effects of exercise on sleep (e.g. upon daytime sleepiness or cognitive functioning) has not been established. Furthermore, little is known about whether features of the exercise stimulus such as duration or timing moderate the effects of exercise on sleep.

The discrepancy between anecdotal evidence that exercise promotes sleep and the accumulated experimental evidence may be attributed mainly to two factors. First, many studies of the effects of exercise on sleep have been of poor methodological quality. Second, because research in this area has been driven predominantly by theories regarding the function of sleep rather than the question of the efficacy of exercise for enhancing sleep, research has focused exclusively on good sleepers who may have little room for improvement (floor effects). On the other hand, it is possible that the beneficial effect of exercise on sleep is a myth perpetrated by researchers' expectancies, a selective focus on supporting data, and the assumption that mus-

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cular fatigue induced by exercise will result in better sleep.

Because exercise is associated with reduced cardiovascular and all-cause mortality (14) and improved mental health (15) it may be an attractive alternative or adjuvant to other sleep treatments (16). The use of sleeping pills is associated with negative side effects (17) and increased mortality (18); whereas other non-pharmacological treatments for insomnia (e.g. relaxation therapy) conceivably could be more labor intensive and expensive than an exercise program. Therefore, it is important to establish with more certainty whether exercise promotes sleep.

Meta-analysis, which has been used in recent years in both the sleep (19,20) and exercise science (21,22) literatures, provides a means of objectively summarizing study results while avoiding several limitations inherent in narrative reviews (23). The purpose of this study was to use meta-analytic methods to 1) examine the influence of *acute* exercise on sleep and 2) to explore possible moderating factors that might explain how the acute-exercise effects on sleep vary according to features of people and exercise or its setting.

METHODS

Literature search

Exercise studies involving sleep were located from personal retrieval and computer searches of the English literature from 1965 to August 1995 using *Medline, Eric, Psychlit, Sleep Research,* and *Dissertation Abstracts International* databases, and bibliographic searches of each reference. Key words used were exercise, physical activity, sleep, and polysomnography. Criteria for inclusion were: 1) the independent variable contrasted acute exercise with a non-exercise control condition; 2) the dependent variable(s) were sleep variables measured with polysomnography, in which standard visual-scoring methods were used; and 3) a minimum number of four subjects was tested. A total of thirty-eight studies (total subjects = 401, total number of effects = 211) that met these criteria were located and included in the meta-analysis (6,24–58). (An appendix that describes each study is available on request.) Ten studies of acute effects of exercise on sleep that did not meet these criteria were excluded.

The following sleep variables were examined: sleep onset latency (SOL), stage 2 sleep, SWS, rapid eye movement (REM) sleep, REM latency (REM-L), total sleep time (TST), and wakefulness after sleep onset (WASO). Other sleep variables were not examined because of an insufficient number of studies done on these variables to warrant quantification. Likewise, the effects of chronic exercise on sleep have been assessed by few studies and were not included in the meta-analysis.

Effect size calculations

All located studies involved within-subject designs. Effects sizes (*d*) were calculated as follows (59): (Mean minutes following exercise − Mean minutes following control)/Standard deviation of control condition. According to this formula, a negative-effect size for a sleep variable would indicate that the mean number of minutes following the exercise condition was reduced compared to the control condition. Thus, negative-effect sizes for SOL and WASO would suggest better sleep (i.e. shorter SOL, less WASO) following exercise compared to control. The standard deviation (SD) of the control condition was used in the denominator rather than the pooled SD of exercise and control conditions because the control SD was considered a better estimate of the population variance under non-manipulated conditions. This method helped protect against bias from the correlated scores of repeated measures (60). For studies in which precise mean data were not presented (n = 6), effect sizes were estimated from reported *F* tests (n = 1), *t* tests (n = 2), or from presented graphs (n = 3). For studies that reported only mean sleep data (n = 8), the median SD for each sleep variable gathered from the literature was used as a surrogate standard deviation. For studies in which identical exercise and/or control conditions were repeated over ≥2 days (n = 9), the pooled SDs within all conditions were used for the meta-analysis.

Approximately 50% of the studies reported sleep stage data in minutes, whereas 50% reported these data as a percentage of total sleep time. For our analysis, percentage data were converted to minutes whenever possible, i.e. when total sleep time was reported. In the few cases in which conversion to minutes was not possible, percentage data were used.

Mean effect sizes were calculated according to Rosenthal (23) via Schwarzer's Meta 5.3 software (61). The software included two corrections of the effect size data. First, because *d* is a biased estimator of the population d, *d* scores were simultaneously converted to Hedges and Olkin's *g* (59) for all effect sizes to account for differences in sample size. Second, a correction for relative unreliability of sleep variables was made. This correction tends to yield larger effect sizes because it assumes a greater proportion of the variability in the data can be attributed to measurement error rather than sampling error. Based on reports in the literature (62,63), the reliability of the sleep variables was assumed to be 0.70. The random effects model (59) was used to combine effect sizes for each sleep variable. Adjustment of *g* for a within-subject

design (64) did not alter the results. Effect sizes that exceeded the mean effect size by 2.5 SD were excluded as outliers from the meta-analyses. These outliers never involved more than 3% of the studies. A test of the homogeneity of effect sizes (Q) was made for each dependent variable using the method of Hedges and Olkin (59). The Q statistic provides a test of the consistency of the magnitude and direction of the effect sizes. A potential bias in any literature is that "positive" findings are likely to be published. For every significant mean effect size, we calculated the number of additional null findings that would reduce the estimated population values to a non-significant effect (23).

Inferences from meta-analysis results are analogous to those that can be drawn from original data in that statistical significance derived from multiple data points does not necessarily indicate that the effects are practically meaningful. The practical significance of an effect varies according to its associated clinical outcome. Nonetheless, Cohen (65) has proposed that 0.2, 0.5, and 0.8 SD are statistical guideposts for judging effects as small, moderate, and large, respectively.

Moderator variables

Analysis of moderator variables provides a way to test whether or not effect sizes vary systematically across differing levels of variables that are posited to influence the dependent measures. Moderator variables can explain effect size heterogeneity. We attempted to categorize the moderator variables in a manner that was theoretically valid and that divided the number of effect sizes equally across the moderator levels. Comparisons of moderator variables were conducted using SPSS (4.0) (66) analysis of variance (ANOVA), trend analysis, and planned contrasts when literature or theory justified a priori hypotheses. Hedges and Olkin's g was derived for each individual effect in the moderator analysis using Johnson's DSTAT 1.10 meta-analysis software (64). In cases in which significant moderator effects were found, the proportion of variance in the sleep variables accounted for by the moderators was determined (Ω^2).

Fitness of subjects. Fitness may moderate the effects of exercise on sleep (1,36) by enabling a quicker recovery of sympathetic nervous system arousal following exercise (67) or a greater ability to perform exhaustive or temperature-elevating exercise that may be necessary to enhance sleep (1). Fitness was coded into three levels based on the information provided. Subjects were labeled "unfit" if they were described as having low or average fitness, if they did not perform regular aerobic exercise, or if they possessed low peak oxygen uptake ($\dot{V}O_{2peak}$) values (<50 or <40 $ml \cdot kg^{-1} \cdot minute^{-1}$ for men and women, respectively) in response to an exercise test. Subjects were labeled "moderately fit" if they engaged in regular aerobic exercise (three times per week for ≥20 minutes) or if they had VO_{2peak} values above 50 and 40 $ml \cdot kg^{-1} \cdot minute^{-1}$ for men and women, respectively. "Very fit" subjects were competitive endurance athletes.

Heat load. Consistent with passive-heating studies (41,68) and the hypothesis that sleep may serve a brain-cooling function (69), Horne and Moore have provided evidence that temperature elevation mediates exercise effects on sleep by showing that increases in SWS following exercise could be reversed by attenuating temperature increases during exercise (i.e. via body cooling) (39). Heat load was coded into three levels: low, moderate, and high. Temperature increases linearly as a function of relative exercise intensity (i.e. % of peak oxygen uptake) (70), and this effect is largely independent of ambient temperature at ranges from approximately 20–30°C for exercise durations <1 hour (67,71). Thus, for most studies, the heat load levels corresponded with estimated relative intensities of <50% $\dot{V}O_{2peak}$, 50–70% $\dot{V}O_{2peak}$, and >70% $\dot{V}O_{2peak}$, respectively. These estimates were made based on available information (e.g. exercise duration for a given distance). Walking was considered to be a low-heat load. Other information relevant to heat load such as ambient temperature and humidity (71), fitness and heat acclimation of the subjects (72), and clothing (73) was usually not available. However, adjustments were made for conditions involving 1) moderate-intensity exercise performed in an extremely hot environment (high-heat load), 2) maximal exercise of short duration (≈15 minutes) in which core body temperature does not have time to rise dramatically (moderate-heat load), and 3) moderate exercise (at least 50%) over prolonged durations (>2 hours, high heat load).

Duration. Exercise duration may also be an important moderator variable, particularly with regard to an energy conservation theory of sleep. Three levels of duration were coded: 1) <1 h, 2) 1–2 hours, and 3) >2 hours.

Time of day. Typical sleep hygiene recommendations that sleep is likely to be promoted, inhibited, or unaltered by late afternoon, late evening, and early morning exercise, respectively (4), have not been adequately explored. The time variable was divided into three levels: exercise performed 1) >8 hours before bedtime, 2) 4–8 hours before bedtime, and 3) <4 hours before bedtime.

Light exposure. Bright-light exposure can enhance sleep (74,75). Since adults receive only about 20 minutes of daily bright-light exposure (76) (i.e. outdoors, >2,500 lux), outdoor exercise is likely to comprise at least half of a subject's daily bright-light exposure. To

TABLE 1. *Mean effect size g (95% confidence interval), number of effects, mean minutes after exercise and control conditions, and mean difference (minutes) following exercise compared with control conditions. Note that negative-effect sizes for SOL and WASO indicate better sleep (i.e. fewer minutes)*

Variable	n (# of effects)	Mean effect size ± 95% confidence interval	Mean ± SD minutes after exercise	Mean ± SD minutes after control	Mean difference (minutes)
SOL	34	−0.05 (−0.24, 0.15)	14.3 (7.3)	16.2 (8.6)	−1.4
Stage 2	34	0.18 (−0.03, 0.39)	198.6 (80.6)	188.4 (84.2)	5.2
SWS	49	0.19 (0.04, 0.33)	92.0 (31.7)	89.3 (32.5)	4.2
REM	42	−0.49 (−0.72, −0.26)	86.4 (25.0)	94.1 (24.2)	−7.4
REM-L	28	0.52 (0.27, 0.78)	90.6 (26.0)	77.1 (8.1)	13.1
TST	26	0.42 (0.17, 0.68)	458.6 (46.7)	444.8 (39.6)	9.9
WASO	20	0.07 (−0.23, 0.37)	13.2 (9.5)	10.9 (8.4)	2.1

SOL, sleep onset latency; WASO, wake after sleep onset; SD, standard deviation; SWS, slow-wave sleep; REM, rapid eye movement; REM-L, rapid eye movement latency; TST, total sleep time.

test this factor, the effects were coded according to whether the exercise was performed outdoors or indoors.

Sleep schedule. The potential promoting effects of exercise on sleep may be limited in many previous studies by fixed-sleep-period length and timing. For example, several studies involving college students have used fixed bedtimes of 2300 hours or earlier without considering that bedtimes are commonly delayed in this population. This moderator was tested by comparing exercise effects for studies that allowed sleep ad libitum with those that used fixed-sleep schedules.

Study quality. The scientific quality of studies was rated (poor, fair, good) according to the number of following criteria that were met: 1) description of subject fitness levels and/or activity history, 2) description of sleep history, 3) measuring of sleep on the night before experimental condition(s) (i.e. to minimize "first night" influences), 4) counterbalancing or randomization of experimental conditions, 5) standardization or description of exercise stimulus, 6) control for napping, caffeine, and exercise on experimental days, 7) blind scoring of data, 8) adherence to standard procedures for recording and scoring sleep, 9) bedtimes set according to customary bedtimes, and 10) an adequate number of experimental nights in each condition (≥2). These criteria were weighted equally.

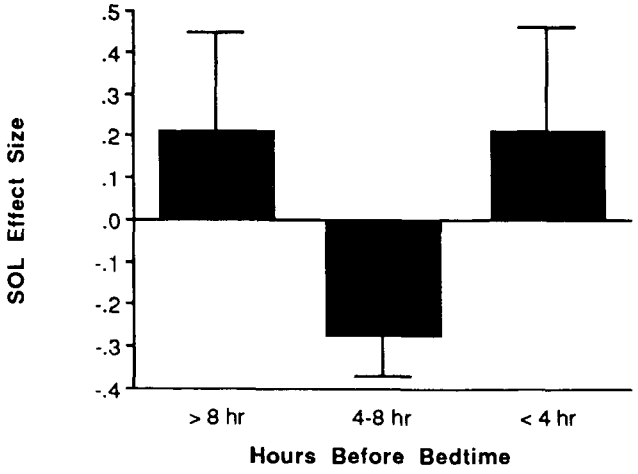

FIG. 1. Sleep onset latency (SOL) mean effect size ± standard error (SE) following exercise compared to control conditions completed >8 hour (n = 4 effects), 4–8 hours (n = 14), and <4 hours (n = 6) before bedtime.

RESULTS

SOL

Exercise had no significant effect on SOL (g = −0.05; p = 0.10) (Table 1). This effect was homogeneous [$Q(33)$ = 43.8; p = 0.15]. The median difference between exercise and control was −1.0 minutes. A significant quadratic trend for time-of-day effects on SOL was found [$F(1,21)$ = 6.61; p = 0.018, Ω^2 = 0.17]. Exercise completed 4–8 hours before bedtime (n = 14 effect sizes) decreased SOL compared to control conditions (g = −0.27), whereas exercise completed >8 hours (n = 4) and <4 hours (n = 6) before bedtime increased SOL (g = 0.21 and 0.22, respectively) (Fig. 1). A significant effect for light environment was found [$F(1,31)$ = 5.64; p = 0.024, Ω^2 = 0.12]. Exercise performed indoors (n = 20 effect sizes) decreased SOL compared to control conditions (g = −0.17); whereas exercise performed outdoors (n = 13) increased SOL (g = 0.20) (Fig. 2). No other significant moderator effects were found for SOL (Table 2).

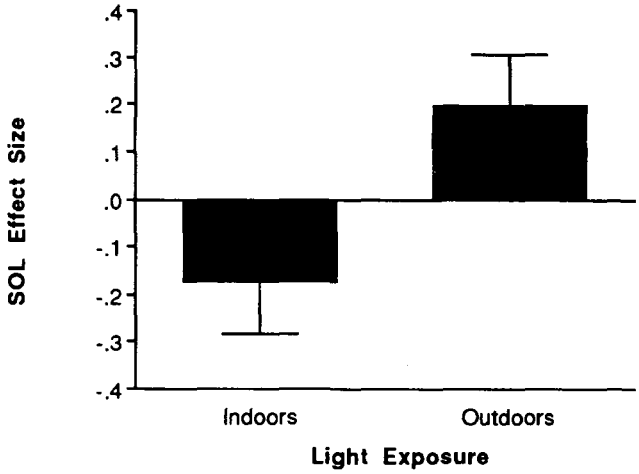

FIG. 2. SOL mean effect size (±SE) following exercise performed indoors (n = 20 effects) and outdoors (n = 13) compared to control conditions.

Stage 2

The mean effect size for stage 2 sleep was statistically significant (g = 0.18; p = 0.04) (Table 1). This effect was homogeneous [$Q(33)$ = 42.6; p = 0.12]. The median increase in stage 2 sleep following exercise compared to control was 1.7 minutes. A planned comparison revealed that stage 2 sleep was increased significantly in the very fit subjects (n = 12 effect sizes) compared to the fit and unfit subjects (n = 19) [$F(1,28)$ = 4.28; p = 0.037, Ω^2 = 0.08] (Fig. 3). Stage 2 sleep increased significantly more following exercise when sleep was permitted ad libitum (n = 16 effect sizes) compared to when the sleep–wake schedule was fixed (n = 18) [$F(1,32)$ = 4.50; p = 0.04, Ω^2 = 0.09] (Fig. 4). Stage 2 sleep was not significantly influenced by any of the other moderator variables (Table 2).

SWS

A significant mean effect size for SWS was found (g = 0.19; p = 0.005). This effect was homogeneous [$Q(48)$ = 40.7; p = 0.76]. The median difference in SWS following exercise compared to control conditions was 1.4 minutes, and nearly as many studies found decreases (n = 19) as found increases in SWS (n = 30). No significant differences between moderator levels were found (Table 2). It is noteworthy that no influence of heat load on SWS was found. Mean effect sizes (±SD) for low (n = 10 effect sizes), moderate (n = 16), and high (n = 16) heat loads were 0.12 ± 0.26, 0.18 ± 0.34, and 0.24 ± 0.53, respectively (Table 2). Moreover, no effect of heat load on SWS was found even when data derived from morning/early afternoon exercise (i.e. >8 hours before sleep) and from unfit individuals were excluded from analysis: mean effects (±SD) for low-, moderate-, and high-heat loads were 0.12 ± 0.26; 0.23 ± 0.30, and 0.23 ± 0.55, respectively.

REM

A significant reduction in REM was found following exercise compared to control conditions (g = −0.49; p < 0.001). This effect was heterogeneous [$Q(41)$ = 69.4; p < 0.01]. The median difference was −6.0 minutes. A planned comparison showed that exercise >2 hours duration (n = 14 effect sizes) elicited significantly greater reductions in REM compared to exercise of shorter duration (n = 21) [$F(1,32)$ = 6.36; p = 0.02, Ω^2 = 0.13] (Fig. 5). REM was not significantly influenced by the other moderator variables (Table 2).

REM-L

Exercise significantly delayed REM-L compared to control conditions (g = 0.52; p < 0.001). This effect was heterogeneous [$Q(27)$ = 41.0; p = 0.04]. The median difference was 11.6 minutes. REM-L was not significantly influenced by the moderator variables (Table 2).

TST

Exercise increased TST compared to control conditions (g = 0.42; p < 0.001). This effect was heterogeneous [$Q(25)$ = 45.1; p = 0.01]. The median difference was 10.0 minutes. A near-significant linear trend for exercise duration was found [$F(1,20)$ = 3.84; p = 0.06, Ω^2 = 0.06]. Greater increases in TST occurred with longer exercise durations (Fig. 6). TST was not influenced by any other moderators (Table 2).

WASO

Mean WASO was unchanged by exercise compared to control conditions (g = 0.07; p = 0.302). This effect was heterogeneous [$Q(19)$ = 30.8; p = 0.04]. The median difference was 2.0 minutes. A significant linear trend for heat load was found [$F(1,15)$ = 5.45; p = 0.03, Ω^2 = 0.16] such that WASO increased following exercise with increasing heat loads (Fig. 7). A significant quadratic trend for the effect of time of day on WASO [$F(1,11)$ = 7.55; p = 0.02, Ω^2 = 0.35] was also found. Exercise completed 4–8 hours before bedtime (n = 7 effect sizes) resulted in a mean WASO effect size of −0.36 (i.e. relatively less after exercise) compared to control conditions; whereas exercise completed within 4 hours of sleep (n = 3) and >8 hours

TABLE 2. *Mean effect sizes g (95% confidence interval) across levels of moderators, n = number of effects. Note that negative-effect sizes for SOL and WASO indicate better sleep (i.e. fewer minutes)*

Moderator variable	SOL	Stage 2	SWS
Fitness			
1) Unfit	−0.07; n = 7 (−0.60, 0.47)	0.01; n = 9 (−0.09, 0.12)	0.20; n = 14 (−0.03, 0.43)
2) Fit	−0.25; n = 8 (−0.68, 0.19)	−0.01; n = 10 (−0.46, 0.45)	0.22; n = 10 (−0.11, 0.54)
3) Very fit	0.07; n = 14 (0.17, 0.31)	0.40; n = 12 (0.07, 0.73)	0.15; n = 19 (−0.05, 0.34)
Heat load			
1) Low	−0.25; n = 8 (−0.80, 0.31)	0.13; n = 6 (−0.55, 0.81)	0.12; n = 10 (−0.06, 0.31)
2) Moderate	−0.02; n = 8 (−0.40, 0.35)	0.12; n = 12 (−0.18, 0.42)	0.19; n = 17 (0.01, 0.36)
3) High	0.02; n = 15 (−0.17, 0.22)	0.24; n = 13 (−0.08, 0.56)	0.24; n = 16 (−0.04, 0.52)
Duration			
1) <1 hour	0.05; n = 8 (−0.40, 0.50)	0.26; n = 8 (−0.09, 0.62)	0.11; n = 15 (−0.12, 0.34)
2) 1–2 hours	−0.07; n = 10 (−0.32, 0.19)	0.12; n = 10 (−0.07, 0.31)	0.29; n = 15 (0.07, 0.50)
3) >2 hours	−0.08; n = 11 (−0.46, 0.30)	0.36; n = 10 (−0.10, 0.83)	0.13; n = 12 (−0.14, 0.41)
Endtime			
1) >8 hours before bedtime	0.21; n = 4 (−0.57, 0.99)	−0.08; n = 3 (−0.40, 0.24)	0.16; n = 7 (−0.31, 0.62)
2) 4–8 hours	−0.27; n = 14 (−0.49, −0.06)	0.14; n = 16 (−0.17, 0.46)	0.24; n = 26 (0.07, 0.42)
3) <4 hours	0.22; n = 6 (−0.42, 0.85)	0.21; n = 5 (−0.30, 0.71)	0.22; n = 6 (0.02, 0.41)
Light			
1) Indoor	−0.17; n = 20 (−0.40, 0.05)	0.19; n = 21 (−0.03, 0.41)	0.28; n = 26 (0.11, 0.45)
2) Outdoor	0.20; n = 13 (−0.03, 0.44)	0.19; n = 12 (−0.18, 0.56)	0.05; n = 21 (−0.11, 0.21)
Schedule			
1) Ad libitum	0.03; n = 16 (−0.18, 0.24)	0.36; n = 16 (0.10, 0.62)	0.14; n = 20 (−0.05, 0.34)
2) Not ad libitum	−0.08; n = 18 (−0.34, 0.18)	0.01; n = 18 (−0.22, 0.25)	0.21; n = 28 (0.06, 0.36
Quality			
1) Poor	−0.06; n = 7 (−0.40, 0.28)	−0.05; n = 6 (−0.73, 0.62)	0.18; n = 17 (0.02, 0.35)
2) Fair	−0.04; n = 13 (−0.28, 0.37)	0.22; n = 14 (−0.01, 0.45)	0.16; n = 18 (−0.05, 0.37)
3) High	0.00; n = 13 (−0.23, 0.23)	0.30; n = 13 (0.00, 0.61)	0.20; n = 14 (−0.07, 0.46)

before sleep (n = 4) elicited mean effect sizes of −0.12 and 0.27, respectively (Fig. 8). No other significant moderator effects on WASO were found (Table 2). [A separate meta-analysis, excluding studies in which standard deviations were not reported, yielded the following mean effect sizes: SOL (−0.16), stage 2 (0.11), SWS (0.21), REM (−0.37), REM latency (0.42), TST (0.59), and WASO (0.05).]

DISCUSSION

The mean effect sizes for slow-wave sleep, REM, REM-L, and total sleep time were small to moderate as judged by Cohen's statistical guideposts (65); moreover, their associated confidence intervals did not include zero. Accordingly, acute exercise has a demonstrable effect on these sleep variables. The biological or behavioral meaning of exercise-associated changes of this magnitude are unclear. It is emphasized, however, that the present analysis was limited to an exclusive focus on good sleepers.

REM sleep

Moderate-effect sizes were found for REM and REM-L. These were the most consistent effects. De-

TABLE 2. *Extended*

REM	REM-L	TST	WASO
−0.15; n = 12	0.21; n = 8	0.15; n = 5	−0.13; n = 5
(−0.46, 0.16)	(−0.30, 0.73)	(−0.57, 0.87)	(−0.74, 0.49)
−0.30; n = 9	0.53; n = 7	0.34; n = 5	−0.02; n = 5
(−0.71, 0.11)	(−0.05, 1.10)	(−0.37, 1.04)	(−0.90, 0.87)
−0.56; n = 15	0.23; n = 9	0.44; n = 12	0.23; n = 5
(−0.95, −0.16)	(−0.05, 0.51)	(0.17, 0.71)	(−0.26, 0.72)
−0.26; n = 9	0.48; n = 6	0.39; n = 7	−0.52; n = 2
(−0.69, 0.16)	(−0.11, 1.07)	(−0.20, 0.98)	(−5.66, 4.63)
−0.24; n = 13	0.27; n = 10	0.33; n = 6	−0.12; n = 8
(−0.55, 0.07)	(−0.23, 0.78)	(−0.20, 0.86)	(−0.44, 0.20)
−0.58; n = 16	0.39; n = 11	0.39; n = 11	0.15; n = 8
(−0.96, −0.20)	(0.13, 0.65)	(0.04, 0.74)	(−0.13, 0.43)
−0.31; n = 9	0.23; n = 10	0.07; n = 6	−0.13; n = 5
(−0.59, −0.02)	(−0.17, 0.63)	(−0.27, 0.42)	(−0.71, 0.46)
−0.10; n = 12	0.40; n = 11	0.45; n = 9	−0.01; n = 5
(−0.43, 0.23)	(0.05, 0.75)	(0.07, 0.83)	(−0.28, 0.25)
−0.72; n = 14	0.55; n = 5	0.61; n = 8	0.03; n = 7
(−1.16, −0.29)	(−0.28, 1.38)	(0.11, 1.11)	(−0.47, 0.52)
−0.49; n = 7	0.32; n = 4	0.64; n = 2	0.27; n = 3
(−1.16, 0.18)	(−0.30, 0.95)	(−2.98, 4.27)	(−0.17, 0.70)
−0.34, n = 18	0.35; n = 15	0.53; n = 11	−0.36; n = 7
(−0.55, −0.12)	(0.04, 0.67)	(0.20, 0.87)	(−0.71, 0.00)
−0.39; n = 7	0.76, n = 5	0.53; n = 5	−0.12; n = 4
(−1.02, 0.25)	(0.13, 1.38)	(−0.60, 0.86)	(−0.17, −0.06)
−0.35; n = 25	0.45; n = 20	0.44; n = 16	−0.08; n = 11
(−0.57, −0.14)	(0.18, 0.71)	(0.14, 0.75)	(−0.31; 0.16)
−0.51; n = 14	0.14; n = 7	0.22; n = 10	0.00; n = 7
(−0.94, −0.07)	(−0.23, 0.51)	(−0.07, 0.51)	(−0.49, 0.49)
−0.59; n = 15	0.20; n = 11	0.34; n = 15	0.18; n = 6
(−0.95, −0.23)	(−0.15, 0.55)	(0.05, 0.64)	(−0.21, 0.57)
−0.31; n = 27	0.51; n = 17	0.38; n = 11	−0.07; n = 13
(−0.53, −0.09)	(0.23, 0.79)	(0.03, 0.73)	(−0.37, 0.24)
−0.48; n = 10	0.71; n = 7	0.15; n = 4	0.09; n = 6
(−0.91, 0.04)	(0.29, 1.12)	(−0.73, 1.03)	(−0.43, 0.61)
−0.37; n = 18	0.27; n = 12	0.25; n = 9	−0.07; n = 9
(−0.68, −0.06)	(−0.09, 0.62)	(−0.19, 0.69)	(−0.35, 0.21)
−0.47; n = 13	0.16; n = 8	0.44; n = 12	0.41; n = 3
(−0.83, −0.11)	(−0.16, 0.48)	(0.16, 0.72)	(−0.61, 1.41)

creases in REM and increases in REM-L were found in 83% and 79% of the studies that examined these variables, respectively; whereas increases or decreases in the other sleep variables occurred in 55–63% of the studies located. The implications of these effects with regard to sleep quality are unclear. The REM data were surprising because REM changes following exercise have been largely ignored in reviews of the literature. The timing of REM and REM-L is regulated by the circadian system (77). Thus, the REM and REM-L data might reflect circadian rhythm phase delays. Nocturnal exercise has been found to elicit circadian rhythm phase delays in humans (78,79); however, a complete phase–response curve to exercise in humans has not yet been elucidated. The time of day at which exercise ended was not found to be a statistically significant moderator of REM or REM-L. Nevertheless, when exercise ended <4 hours before sleep, the magnitude of the REM-L effect size (0.76) was more than twice as large as when exercise was performed at other times (0.32–0.35). A reduction in REM might be expected if the circadian timing of REM was delayed, but subjects awoke at the same time in exercise and control conditions. Alternatively, the reductions in REM may represent an inhibitory influence of increases in non-REM (NREM) sleep. The demonstrated antidepressant effects of both REM-depriving drugs and ther-

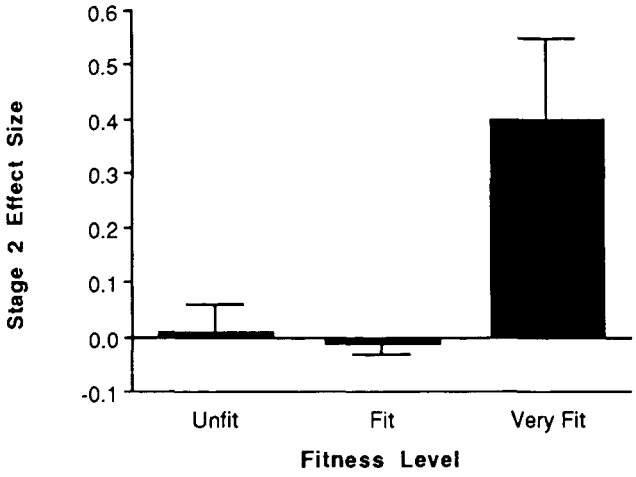

FIG. 3. Stage 2 mean effect size (±SE) following exercise compared to control conditions in unfit (n = 9 effects), fit (n = 10), and very fit (n = 12) subjects.

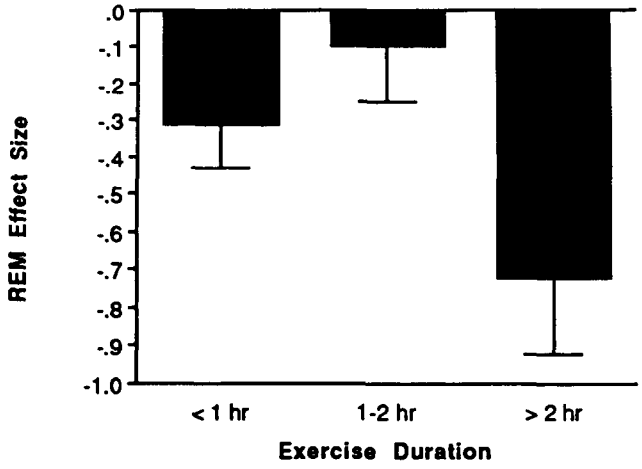

FIG. 5. Rapid eye movement (REM) mean effect size (±SE) following exercise compared to control conditions across durations of <1 hour (n = 9 effects), 1–2 hours (n = 12), and >2 hours (n = 14).

apies (80) and exercise (81) suggest that the influence of exercise on REM should be further explored as a potential antidepressant mechanism.

SWS

The SWS data, though statistically significant, indicate that a single bout of exercise does not substantially increase SWS in good sleepers. This observation contrasts with other prior reviews (1), but is consistent with the review of Trinder and colleagues (13). In light of current theory and empirical evidence regarding passive heating and thermogenic effects of exercise on SWS (39), it was surprising that no moderating influence of exercise heat load on SWS was found. Indeed, the only influence of heat load found in the meta-analysis was a significant linear trend for increased sleep disruption (i.e. WASO) with increasing heat load. Inferences regarding heat load were limited by a failure of many studies to report ambient conditions, fitness/acclimation of the subjects, and clothing worn.

Our meta-analysis is inconsistent with the findings of Horne and Moore (39), which showed that increases in SWS following exercise were reversed by the blunting of increases in body temperature. One limitation of that study, however, was a failure to assess either body temperature during sleep or the associated heat-loss mechanisms that are thought to interact with sleep mechanisms. Because exercise was completed approximately 6 hours before bedtime, it is unlikely that body temperature or related thermoregulatory mechanisms differed between conditions at bedtime. A recent study found no differences in rectal temperature at bedtime or throughout the sleep period following exercise (1

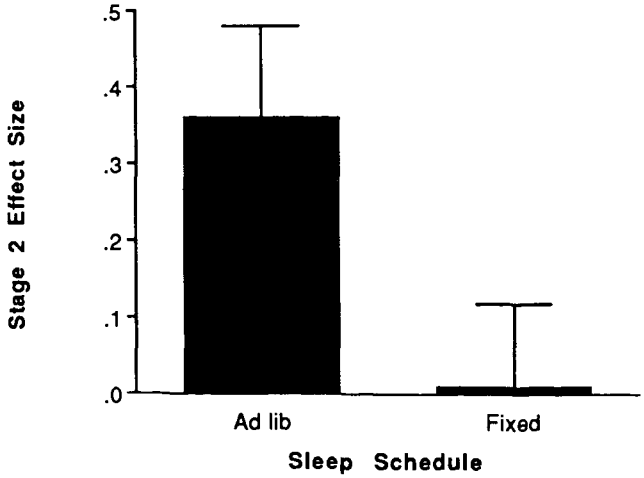

FIG. 4. Stage 2 mean effect size (±SE) following exercise compared to control conditions in which sleep was permitted ad libitum (n = 16) or was restricted to a fixed schedule (n = 18).

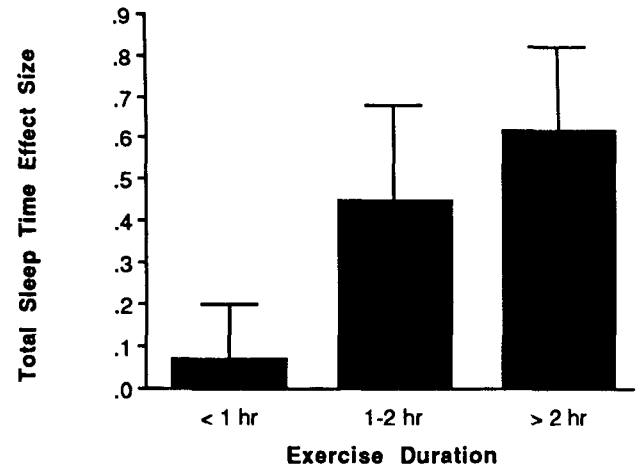

FIG. 6. Total sleep time (TST) mean effect size (±SE) following exercise compared to control conditions across durations of <1 hour (n = 6 effects), 1–2 hours (n = 9), and >2 hours (n = 8).

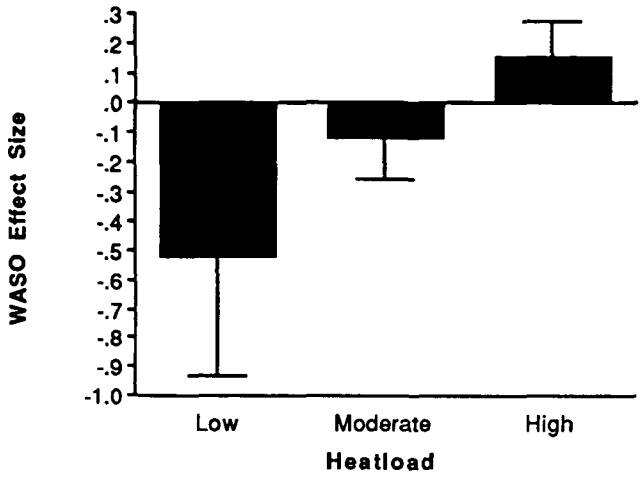

FIG. 7. Wake after sleep onset (WASO) mean effect size (±SE) following exercise compared to control conditions across low (n = 2 effects), moderate (n = 8), and high (n = 8) heat loads.

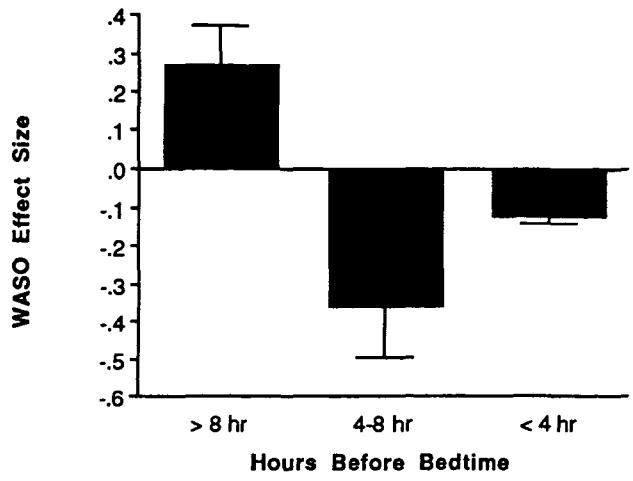

FIG. 8. WASO mean effect size (±SE) following exercise compared to control conditions completed >8 hours (n = 3 effects), 4–8 hours (n = 7), and <4 hours (n = 4) before bedtime.

hour at 60% $\dot{V}O_{2peak}$ 5–6 hours before bedtime) compared to a non-exercise control condition (57). McGinty and Szymusiak (69) have proposed that sleep mechanisms may possess a "memory" of daytime exposure to thermogenic stimuli that might explain delayed thermogenic effects of exercise on sleep. This "memory", it is argued, may be mediated by accumulation of putative sleep factors that are associated with body heating (e.g. prostaglandins). We are unaware of any direct tests of this hypothesis.

A corollary theme in the exercise literature has been that increases in SWS following exercise occur only in individuals fit enough to perform the vigorous, prolonged exercise thought necessary to elicit thermogenic effects on SWS (1). However, our analysis indicates that fitness, exercise heat load, or exercise duration did not moderate the effects of exercise on SWS.

Different results might be expected for analyses of the first two NREM–REM cycles, in which SWS is most prevalent and the rate of decline in body temperature is greatest. This notion is supported by the positive-effect size for REM-L. However, the few exercise studies (n = 11) that reported cycle or partitioned sleep data (26,35) found mixed results. Therefore, strong conclusions about this issue cannot be drawn.

TST

The effect of exercise on TST tended to be greater for exercise that was >1 hour duration. It is noteworthy that all three studies that were deleted as outliers from the TST analyses involved competitions of very long duration (marathon or ultramarathon races) (35,46,51). Although two of these studies found extraordinary increases in TST, the third study found a profound decrease in TST that might be attributed to muscle soreness. A potentially important factor mediating these results might be that sleep was permitted ad libitum in the studies finding TST increases; whereas the sleep schedule was fixed in the study that found negative results. Although the sleep schedule factor did not significantly moderate the effects of exercise on TST or any of the other sleep variables, it is possible that sleep duration is greatly increased by very prolonged, intense exercise when sleep is permitted ad libitum.

Besides the fact that effect sizes from these three studies exceeded our outlier criterion, the decision to exclude these studies was justified on other grounds. First, one limitation of these studies was the failure to assess sleep on the night preceding the races. Because sleep prior to competition might be impaired by anxious anticipation and first-night effects (i.e. sleeping in a hotel), sleep enhancement following a race may only be a reflection of sleep rebound. Second, other studies involving marathon/ultramarathon races failed to show dramatic changes in TST. The resolution of the influence of exercise duration on TST will require more research. Inferences about the clinical significance of the TST data are difficult to make because sleep stage data were not reported in all studies.

Given the relatively small number of studies included in the analyses presented above, it is likely that there is sampling bias in our estimates of effect size for each sleep variable. The number of additional null findings that would reduce the estimated population values to a non-significant effect (23) for stage 2, SWS, REM, REM-L, and TST are 18, 28, 453, 297, and 330, respectively.

Moderator variables

Fitness. Fitness generally did not moderate the influence of exercise on sleep. This finding contrasts with previous reviews (1) but is consistent with anecdotal reports from both relatively sedentary and physically active individuals.

Duration. Duration was a more consistent moderator than other variables. However, the relative strength of this association may have resulted from a clearer description of duration or relevant information (e.g. running distance) in the literature compared to other moderator variables. Because reliable effects of exercise on sleep were seen only for exercise exceeding a duration of 1 hour, the practical usefulness of exercise for many segments of the generally sedentary population is unclear.

Time of day. The significant quadratic time-of-day effects on SOL and WASO are consistent with common sleep hygiene recommendations that exercise will enhance or impair sleep when performed in the late afternoon or late evening, respectively (4). However, this issue has not been adequately addressed in the literature because the vast majority of exercise conditions were completed at least 4 hours before sleep. Only one study involving high-heat load was performed within 4 hours of sleep (33). It is noteworthy that two recent studies have failed to find impairment in behavioral measures of sleep (i.e. response to auditory stimulus and actigraph recording) following vigorous exercise (60–70% $\dot{V}O_{2peak}$) completed 30 minutes before sleep [(82) and Youngstedt et al. 1996, unpublished]. Disruptions in sleep following exercise conducted close to bedtime may be dependent upon fitness, which influences the quickness of physiologic recovery from exercise (67). Fitness, however, generally did not influence the sleep variables examined. The issue of whether chronic exercise, which may increase fitness, improves sleep was not addressed in the meta-analysis.

Light exposure. Illumination generally did not influence sleep. However, compared to studies involving indoor exercise, outdoor exercise studies had less experimental control and standardization (e.g. intensity and duration). Moreover, no information was provided regarding the level of illumination before or after the conditions.

Sleep schedule. One of the most robust moderating effects showed that stage 2 sleep was greater when sleep was permitted ad libitum. These data combined with TST outliers involving ad libitum sleep suggest the need for further exploration of this variable. Fixed sleep schedules in many experiments may be a primary reason for the discrepancy between anecdotal reports and experimental data.

Study quality. The scientific quality of studies did not moderate the sleep variables. However, our categorization of quality was arbitrary, and the degree to which sleep data are compromised by specific weaknesses in the quality of studies is not clarified by our quantitative analysis.

Methodological and interpretive issues

Although the clinical impact of an acute bout of exercise on sleep implied by our meta-analysis appears at first glance to be uncompelling, a delimitation of the literature reviewed herein has been an exclusive focus on good sleepers. Conclusions regarding the potential sleep-promoting effects of exercise for insomnia patients, as recommended by many sleep clinicians, cannot be made because data are simply not available on this topic. Analogous conclusions regarding the efficacy of other behavioral insomnia treatments would not be acceptable had they been given exclusively to young, healthy individuals who slept 8 hours each night, had good sleep hygiene, and were without daytime sleepiness. Indeed, even sleeping pills can have little effect on the sleep of good sleepers (83–85). Guilleminault et al. (86) recently reported that 4 weeks of exercise combined with sleep hygiene education resulted in actigraphically defined increases in TST as well as decreases in sleep latency and WASO in individuals with psychophysiological insomnia. Although the benefits of exercise were not statistically significant, effect sizes for SOL ($g = -0.36$) and WASO ($g = -0.16$) were larger than the average acute effects we report herein. The extent to which the sleep improvements could be attributed to exercise or the adoption of other sleep hygiene habits was unclear in the Guilleminault et al. study.

Inferences regarding causal effects of the moderator variables we report are also limited because the levels of the moderators were not manipulated in most studies. Also, for several of the moderator analyses, the number of effects for the different levels were small and unequal. Hence, powerful tests of the independent or interactive influences of the moderator variables on the mean effect for each sleep variable could not be conducted, a common limitation in meta-analysis. The moderating variables we examined did not explain variability in several sleep variables for which the mean effect of exercise was heterogeneous (23). Hence, other moderators may warrant investigation.

Notwithstanding the focus on good sleepers in the extant literature on acute exercise and sleep, greater effects of exercise on sleep might be expected in light of anecdotal reports and epidemiological evidence from the general population (3,11). One explanation for the contrary findings might be the generally poor

methodology of studies that have examined exercise effects on sleep. For example, most of the studies failed to randomize or counterbalance the order of experimental conditions. Other methods for quantifying sleep, for example slow-wave activity (87), may provide a better marker of homeostatic sleep drive and may show greater changes following exercise compared to standard sleep stage variables (88). Quantitative sleep electroencephalograms (EEGs) have received scarce attention in exercise studies.

Few studies have experimentally examined putative mechanism(s) mediating the effects of exercise on sleep. One plausible mechanism supported by anecdotal reports, but overlooked by most experimental studies, is that of anxiety reduction. Sleep improvements following exercise in normal sleepers may be restricted to days in which individuals are experiencing significant anxiety. These effects may be lost when testing subjects over a few experimental nights in laboratory conditions removed from their usual daily stressors. Techniques for experimentally inducing insomnia (57) may offer a particularly fruitful way to examine the mechanisms of exercise effects on sleep.

CONCLUSIONS

Our quantitative synthesis of the literature indicates that acute exercise was associated with consistent small-to-moderate effects on SWS, REM, REM-L and TST. At the present time, the mechanisms, as well as the biological or behavioral significance, of these effects are unclear. Since this body of research has focused exclusively on good sleepers, future research is needed to determine if the magnitude of the effects of exercise on sleep differ among people with sleep problems.

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