

## EXTENDED NIGHTS, SLEEP LOSS, AND RECOVERY SLEEP IN ADOLESCENTS

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### BACKGROUND

*"To answer the question of the 'why and how' of sleep, I studied the effects of prolonged deprivation of sleep, as well as the concomitants of the deep sleep that follows such an experimental period of wakefulness (p. 5)."*

This theme of sleep deprivation/recovery is one of many threads that pervade Professor Kleitman's research. Kleitman's experimental approach serves as an appropriate perspective for the present study, because our efforts, too, involve sleep deprivation and recovery sleep in an effort to understand developmental changes in human sleep. Professor Kleitman's work also included a strong developmental perspective, though chiefly focused on sleep of infants rather than – as is the case in the present research – adolescents. Kleitman's focus on the very young is understandable, given his burning interest as much in the state of wakefulness as in sleep. Thus, whereas the typical question of sleep researchers (and of parents) in early development concerns the longest episode of sustained sleep, Professor Kleitman's research was equally inspired to examine the developmental course of the longest episode of sustained wakefulness. Interestingly, Professor Kleitman's assessments of infant sleep were made using techniques designed to acquire the natural patterns of the infants' sleep with minimal social influence: "infants remained lying in their cribs, except when lifted to be fed and cared for (p. 115)" (14). One of the few descriptions of adolescent sleep in Kleitman's book contains an allusion to the observation of Terman and Hocking (18) that the percentage of children who spontaneously terminate sleep begins to decrease at age 11 and their suggestion that adolescence is associated with a "change from a vesperal to a matinal type of sleeper (p. 118)" (14).

The present study takes its cues from a number of these Kleitmanesque threads. Our long-nights protocol was designed specifically to isolate circadian patterns and nocturnal sleeping behavior from psychosocial influences (7) in the service of better understanding the switch to "matinal" sleep that occurs at adolescence. The imposition of a night of sleep deprivation and the focus on subsequent recovery sleep in these adolescents provides the kinds of insights Kleitman sought into the "why and how" of sleep, specifically the "why and how" of changing sleep patterns during adolescent development.

### INTRODUCTION

The nature of sleep need and recovery sleep in adolescents may be important for understanding changes in sleep patterns that occur at this maturational stage. By survey and daily self report, adolescents tend to go to bed later and to sleep later

than preadolescents (6). This pattern has typically been ascribed to psychosocial factors, such as increasing assertion of autonomy or as indicating increased academic and social responsibilities or opportunities. Terman and Hocking (18), for example, suggested that evening school work was an operational factor in the adolescent sleep delay. In recent years, we have noted evidence for a relationship between the behavioral sleep delay and maturational changes in the circadian timing system (7, 8). Other changes in the intrinsic mechanisms controlling sleep/wake processes may also be involved in delaying sleep onset during adolescent development, including processes characterized as involved in homeostatic sleep/wake regulation.

One way to examine this homeostatic sleep drive is to measure the response to sleep deprivation. In general, the process – referred to as Process S in some models (2) – is indexed by the amount of EEG power in the delta range during sleep. A surrogate for delta power is slow wave sleep (SWS) stages 3+4 (16), although this measure of slow EEG activity likely does not account for sleep-related delta power occurring other sleep stages. Length of sleep and amount of intercurrent wakefulness are also related to the homeostatic process, the latter inversely.

In the present study, we have the opportunity to examine these phenomena in adolescents studied in an unusual experimental protocol originally intended to examine circadian rhythms (7). This “long nights” protocol included three consecutive 18-hour nights. We have studied two groups of adolescents with this method, in one group following an “optimal” sleep schedule and in the other after a night of sleep deprivation. The present report details polysomnographically recorded sleep on these long nights.

## METHODS

*Participants.* - Participants were recruited with pamphlets distributed at schools and through newspaper advertisements for a study of sleep and circadian rhythms. The project took place during two consecutive summers (1995, 1996) and was approved by the hospital's Institutional Review Board (IRB) for the Protection of Human Subjects. Participants were paid for their time. Telephone and questionnaire screening were performed to exclude anyone with a personal or family (first-degree relative) history of psychopathology, narcolepsy, or sleep apnea syndrome; a sleep schedule that varied by greater than 3 hours across the week or indicated chronically insufficient sleep manifested as inappropriate daytime sleep episodes; more than one nap per week; chronic major illness; current illness, fever, or symptoms of respiratory infection or allergy; current use of psychoactive agents or other compounds affecting the sleep/wake system; history of head trauma or brain injury; physical handicap likely to interfere with testing; or evidence of learning disability.

Twenty-nine adolescents successfully completed the study. Seven boys (ages 11.3 to 14.1, mean age 13.0 years) and eight girls (ages 12.2 to 14.5, mean age 13.1 years) took part in the nonsleep deprived protocol, and seven boys (ages 12.1 to 14.4, mean 13.1 years) and seven girls (11.1 to 13.8, mean 12.5 years) took part in the protocol involving sleep deprivation. Tanner staging 17 was performed by two independent raters, and consensus ratings for pubic hair growth were used for comparisons in this report. For the nondeprived group, 4 boys and 3 girls were classified as Tanner stages 1 or 2 (pre- or early pubertal); the others were Tanner stages 3 or 4 (mid- or late pubertal). For the deprived group, 3 boys and 3 girls were Tanner stages 1 or 2 and the others Tanner stages 3 or 4. No child in either group was rated Tanner stage 5 (mature).

*Procedures.* - All participants began the study with one week of actigraphy, sleep logs, and telephone calls to the lab each morning while sleeping on their self-selected schedules. These data are not included in this report. For the subsequent 8 (nondeprived group) or 9 cycles (deprived group), participants were required to attempt to sleep at home only during the hours from 2200 to 0800 wearing opaque eye shades, to wear an actigraph, to keep a sleep/wake log, and to telephone the laboratory each morning to report bedtime and rising time. The nondeprived group (1995) then came to the lab at 1700, had one night of sleep on the home schedule and began the long nights protocol the subsequent day. Participants in the deprived group (1996) came to the lab at 1600 and immediately after electrode application (about 1800) entered a modified constant routine 9, 15 protocol in which they remained in bed at a 45° angle, awake, taking small meals at 2-hour intervals, completing forms (30 minute intervals), and engaging in the multiple sleep latency test and performance testing at 2-hour intervals. The long nights protocol for the sleep deprived group followed 24 hours of this constant routine procedure; hence, these participants were without sleep for 34 hours before the first long night, whereas the other group was awake for only 10 hours before the first long night.

Both groups of adolescents were studied for three consecutive long nights lasting from 1800 to 1200, during which they were requested to lie quietly in bed and not to resist sleep but to remain relaxed. Ambient light level during long nights was <1 lux, too dim to distinguish color or detail but enough light to see shapes or read very large print after full visual accommodation. In addition, a continuous tape loop of tree frog sounds (1) was played through speakers at the head of the bed at a level just above waking auditory threshold. The provision of a minimal level of light and sound was requested by the IRB to avoid "sensory deprivation". No participant complained that the level of light or sound interfered with sleeping.

On the two days intervening between the long nights, participants in the nondeprived group only were permitted to experience ambient outdoor light for about two hours between 1400 and 1600; otherwise, participants remained in the dimly lit (<40 lux) laboratory conditions. Family-style meals were served at 1230 and 1630 on these two days, with fluids available ad libitum during scheduled waking hours. The following restrictions for eating/drinking were applied during the long nights. Meal requests occurring within 3 hours of lights out, 60 minutes of lights on, or 3 hours since a previous meal were denied ("too soon for a meal"), and a 250 ml drink of milk or fruit juice was offered instead. Otherwise, a meal consisted of fluid (250 ml milk, water, fruit juice; one refill available); 4 ounces of fruit, carrots, or celery; sandwich (child's pre-elicited choice); a 4-ounce container of yogurt, pudding, or jello; 5/8 ounce bag of chips; 2-ounces of cookies. During any meal, the child remained in bed in <1 lux. Meal intervals were limited to 30 minutes from initial request to completion, and interactions with staff were minimized. Drink requests were met with 250 ml water, milk, or fruit juice unless within 3 hours of a previous request. Meal requests occurred on average less than one time per night.

Because of a concern that children might be unsettled by the long nights' procedure and become distressed if unable to fall asleep, a "comfort visit contingency" was implemented in the first long night. If a child lay awake for >60 minutes before sleep onset or >30 minutes after having been asleep, a staff member entered the room to inquire "is everything okay?", to remind the child to call out if he or she needs anything, and to remind the child to lie quietly and try to fall asleep or "just relax and think about fun and interesting stuff." These comfort visits were kept to minimum time and performed without raising light levels. Restroom trips were also accomplished quickly and without increasing light levels.

Sleep was continuously recorded at a chart speed of 10 mm/sec from electrodes attached to scalp and face to record EEG (C3/A2 or C4/A1 and O1/A2 or O2/A1), right and left outer canthus, and mentalis/submentalis EMG using Grass Instruments Model 7 polygraphs (Astromed/Grass; West Warwick, RI, USA). Records were scored in 30-second epochs according to the criteria of Rechtschaffen and Kales (16) and summarized using Somnibus software (Behavioral Cybernetics; Cambridge, MA, USA).

Group (nondeprived vs. deprived) by Sex by Tanner (1/2 vs. 3/4) by Night repeated measures multivariate analysis of variance was performed for each of the following variables derived from the sleep stage scoring across each night: minutes of stages 1, 2, 3 + 4 (SWS), and REM sleep; minutes of total sleep time (TST); minutes of total wake time (TWT); sleep latency (lights out

to first of 3 consecutive epochs of sleep); REM latency (sleep onset to the first epoch of REM sleep); and minutes of wakefulness after the final arousal (WAFAs). An alpha of .05 was used to determine statistical significance.

## RESULTS

No variable manifested a main effect for sex or Tanner stage. Table 1 gives means and standard deviations for each variable as a function of group and night. Significant main effects for Group and Night and significant interactions are described below.

Table 1. - Mean (standard deviation) sleep data for each long night and 3-night mean for participants in the nondeprived and deprived groups.

	Night 1	Night 2	Night 3	3-Night Mean
<b>Nondeprived (n = 15)</b>				
Stage 1 sleep minutes	70 (20)	57 (21)	55 (25)	60 (15)
Stage 2 sleep minutes	391 (52)	361 (51)	330 (49)	361 (44)
Slow wave sleep minutes	105 (37)	88 (25)	79 (15)	91 (22)
REM sleep minutes	173 (30)	151 (30)	142 (30)	155 (27)
Total sleep minutes (TST)	739 (59)	657 (55)	606 (57)	667 (46)
Total wake minutes (TWT)	341 (59)	419 (55)	470 (60)	410 (47)
Sleep latency minutes	19 (12)	102 (84)	192 (73)	104 (37)
WAFAs minutes	62 (71)	99 (95)	143 (87)	101 (58)
REM latency minutes	100 (64)	117 (82)	92 (55)	103 (53)
<b>Deprived (n = 14)</b>				
Stage 1 sleep minutes	43 (23)	49 (13)	53 (13)	48 (13)
Stage 2 sleep minutes	539 (67)	345 (57)	377 (47)	420 (47)
Slow wave sleep minutes	162 (43)	97 (33)	95 (27)	118 (31)
REM sleep minutes	192 (32)	144 (22)	152 (32)	163 (19)
Total sleep minutes (TST)	936 (52)	634 (65)	678 (60)	749 (44)
Total wake minutes (TWT)	141 (52)	445 (64)	401 (61)	329 (44)
Sleep latency minutes	2 (2)	112 (107)	68 (55)	61 (50)
WAFAs minutes	53 (65)	134 (90)	144 (70)	110 (54)
REM latency minutes	181 (81)	87 (72)	198 (123)	155 (60)

*Main effects of Group.* - Six of the sleep variables demonstrated a significant effect of group. Higher values for stage 2 sleep ( $F(1,21) = 10.2, p = .004$ ), SWS ( $F(1,21) = 6.4, p = .020$ ), and TST ( $F(1,21) = 24.0, p < .001$ ) indicate overall more sleep in the deprived than nondeprived group across the long nights. The deprived group on average had 7 hours of stage 2 sleep across all nights, versus 6 hours on average in the nondeprived group. For SWS, the three-night average for the deprived group was about 2 hours versus about 90 minutes in the nondeprived group; for total sleep, about 12.5 hours in the deprived group versus about 10 hours in the nondeprived group. Reflecting the concomitant reduction in time spent awake for the deprived group were shorter sleep latency ( $F(1,21) = 5.9, p = .025$ ) and lower TWT ( $F(1,21) = 22.9, p < .001$ ). The final variable showing a significant ( $F(1,21) = 6.7, p = .017$ ) overall difference as a function of group was REM

latency, for which the three-night average was 155 minutes in the deprived group versus 103 minutes in the nondeprived group.

*Main effects of Night.* - The analysis indicated significant Night effects for 7 variables: stage 2 sleep ( $F(2,42) = 88.2, p < .001$ ), SWS ( $F(2,42) = 57.7, p < .001$ ), REM sleep ( $F(2,42) = 24.1, p < .001$ ), TST ( $F(2,42) = 167.1, p < .001$ ), TWT ( $F(2,42) = 163.8, p < .001$ ), sleep latency ( $F(2,42) = 33.2, p < .001$ ), and Wafa ( $F(2,42) = 11.2, p < .001$ ). Inspection of the mean values presented in Table 2 indicates that these statistically significant differences in general reflect the longer and deeper sleep on Night 1. The sleep hypnograms in Figure 1 illustrate the Night 1 to Night 3 trends in a typical member of each group. Sleep on Night 1 started quickly and lasted longer than on subsequent nights for participants in both conditions. By Night 3, sleep occurred principally in the middle portion of the long night, with a considerable delay to sleep onset and a lengthy time spent awake at the end of the sleep episode. Note also that SWS accumulated early on each of these night and the NREM-REM cycle was intact on all nights.

Table 2. - Mean (standard deviation) sleep data for each long night and 3-night mean for all participants in both groups.

All Participants (N = 29)	Night 1	Night 2	Night 3	3-Night Mean
Stage 1 sleep minutes	57 (25)	53 (18)	54 (20)	54 (15)
Stage 2 sleep minutes	463 (95)	353 (53)	353 (53)	390 (54)
Slow wave sleep minutes	132 (49)	93 (29)	87 (23)	104 (30)
REM sleep minutes	182 (32)	148 (27)	147 (31)	159 (23)
Total sleep minutes (TST)	834 (114)	646 (60)	641 (68)	707 (61)
Total wake minutes (TWT)	244 (115)	432 (60)	437 (69)	371 (61)
Sleep latency minutes	11 (12)	107 (94)	132 (89)	83 (48)
Wafa minutes	58 (67)	116 (93)	144 (78)	106 (56)
REM latency minutes	139 (83)	102 (78)	144 (107)	128 (62)

*Interaction effects.* - The statistically significant interaction effects principally involved Group by Night interactions (see Table 1 for mean values and standard deviations). Stage 1 sleep declined across nights in the nondeprived group and increased across nights in the deprived group ( $F(2,42) = 3.7, p = .032$ ). Stage 2 sleep ( $F(2,42) = 38.1, p < .001$ ), SWS ( $F(2,42) = 14.1, p < .001$ ), and TST ( $F(2,42) = 41.7, p < .001$ ) each showed a shallow decline across nights in the nondeprived group while the deprived group manifested a steep decline from night 1 to night 2 with a plateau or increase on the third night. Sleep latency ( $F(2,42) = 10.3, p < .001$ ) and TWT ( $F(2,42) = 43.0, p < .001$ ) essentially mirrored this pattern, shorter on Night 1 and lengthening on subsequent nights, more so in the nondeprived group than in the deprived group. REM sleep latency also manifested a Group by Night interaction ( $F(2,42) = 5.4, p = .008$ ), showing a quite stable pattern across nights in the nondeprived group, while the deprived group pattern of REM latency was twice as long on Nights 1 and 3 as on Night 2.

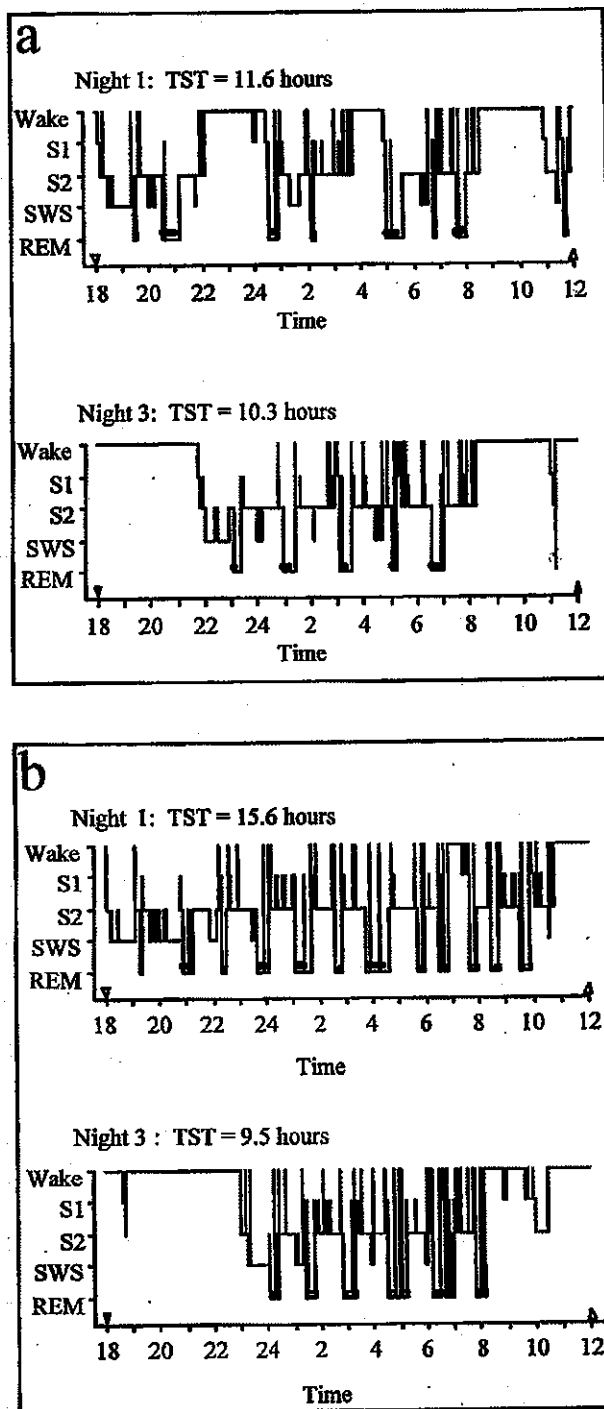


Fig. 1. - Two hypnograms are presented each for one participant in the nonsleep deprived group (a) and in the sleep deprived group (b).

Night 1 is the first long night and Night 3 is the third long night. Time is labeled along the abscissa in 2-hour intervals based upon 24-hour clock time; hence, 18 = 1800 (or 6:00 pm) and so forth. The downward pointing triangles represent lights out and the upward pointing triangles represent the time of lights on at the end of the night. Sleep stages are labeled on the ordinate as Wake = wakefulness, S1 = Stage 1 NREM sleep, S2 = Stage 2 NREM sleep, SWS = slow wave (Stages 3 + 4) NREM sleep, REM = REM sleep. The total amount of time scored as sleep for the entire night is labeled at the top of each graph as TST.

The final interaction occurred with regards to SWS. Figure 2 shows this significant Tanner stage group by Night interaction ( $F(2,42) = 3.8; p = .029$ ), in which SWS in the more mature group of children gradually declined across the three nights, whereas the less mature group had a higher amount of SWS on Night 1 and dropped to a stable level on Nights 2 and 3.

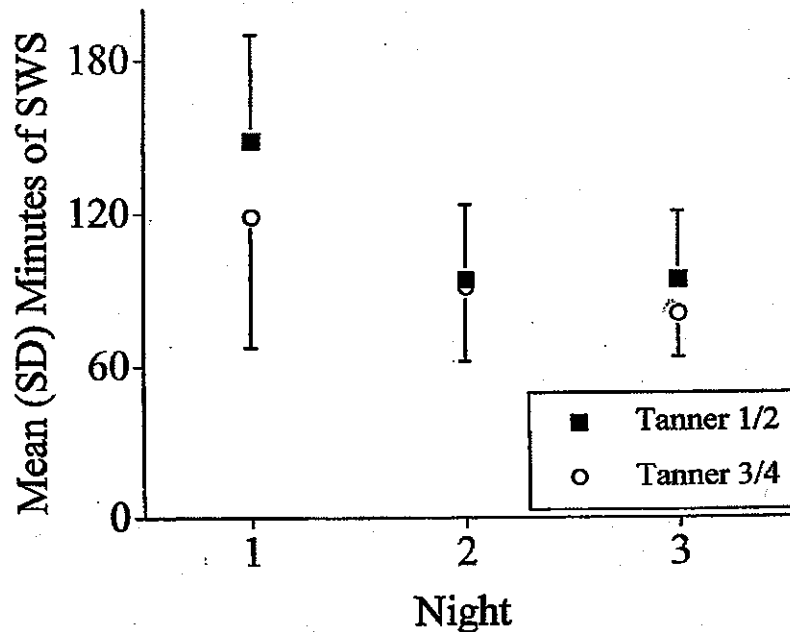


Fig. 2. - The mean (and standard deviation) of minutes of slow wave sleep (SWS) on each night of the study is depicted for those participants who were less mature (Tanner 1/2 = dark squares) versus those who were more mature (Tanner 3/4 = open circles).

A Tanner group by night interaction is carried by the increased amount of SWS on the first long night in the less mature participants.

A three-way interaction was found only for sleep latency. This finding involved a Group by Tanner by Night interaction ( $F(2,42) = 3.7, p = .033$ ), largely carried by a very lengthy (3.7 hour average) delay to sleep onset on Night 3 in the more mature, nonsleep deprived group versus a plateau on Night 3 at about 2.5 hours in the less mature nonsleep deprived group and a decline in sleep latency to about 90 minutes in the sleep deprived groups on Night 3.

*Patterns across the Night.* - The distributions of sleep stages and wakefulness on the three nights show patterns that highlight differences between sleep-deprived and nonsleep-deprived conditions and across nights. In order to illustrate these sequential changes, we summarized the cumulative minutes of wake or each sleep stage per hour as a function of time (i.e., hours after lights out) or by aligning data according to the beginning of sleep (i.e., hours after sleep onset). Most prominent among these summaries is the "recovery" night effect in the sleep-deprived group.

For example, Figure 3 illustrates that the sleep-deprived group sustained well consolidated sleep (less than 20 minutes total of wakefulness on average) across the first 14 hours of Night 1. Thereafter on that night, wakefulness accumulated at a rate greater than 15 minutes per hour. For the nondeprived group, sleep onset on Night 1 was somewhat delayed, and sleep was relatively consolidated only for the first two hours followed by a significant amount of waking. On all nights in both groups, the consistently consolidated sleep (<15 minutes of wake per hour) occurred from the fifth or sixth through the twelfth or thirteenth hours, corresponding to the hours spanning about 2300 to 0700.

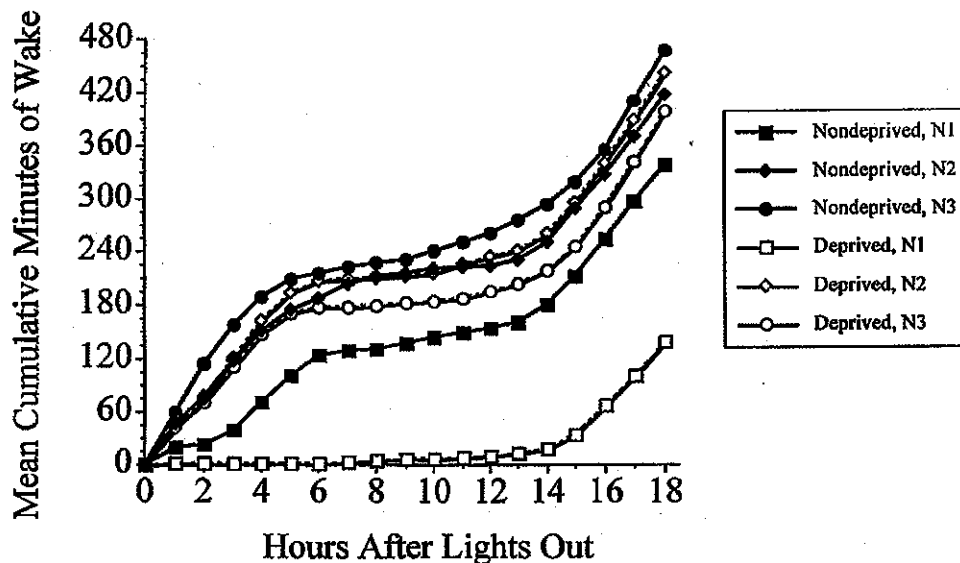


Fig. 3. - The mean number of cumulative minutes of wakefulness per hour since lights out is plotted for each of the groups for each night.

The dark symbols represent the nondeprived group; open symbols depict the deprived group; squares = Night 1, diamonds = Night 2, and circles = Night 3.

Marked Group differences were seen in the patterns of accumulation of SWS and REM sleep. Figure 4a illustrates the substantial amount of SWS occurring during Night 1 in the deprived group. From the third to the sixth hours of sleep, for example, SWS accumulation in the deprived group was double that of any other night for either group. Of interest, Figure 4a indicates no secondary increase of SWS after 12 hours, as might be anticipated by previous studies that indicated a biphasic distribution of SWS with extended sleep (3). Such a secondary "pulse" of SWS was seen neither when SWS was assessed as a function of sleep accumulation nor when assessed as related to time since sleep onset.

REM sleep accumulation showed no differences among the groups or nights for the first 12 hours of sleep (Fig. 4b). On Night 1 for both deprived and nondeprived groups, REM sleep continued to accumulate as sleep was prolonged, hence achieving greater quantities than on Nights 2 and 3, as confirmed by the significant effect



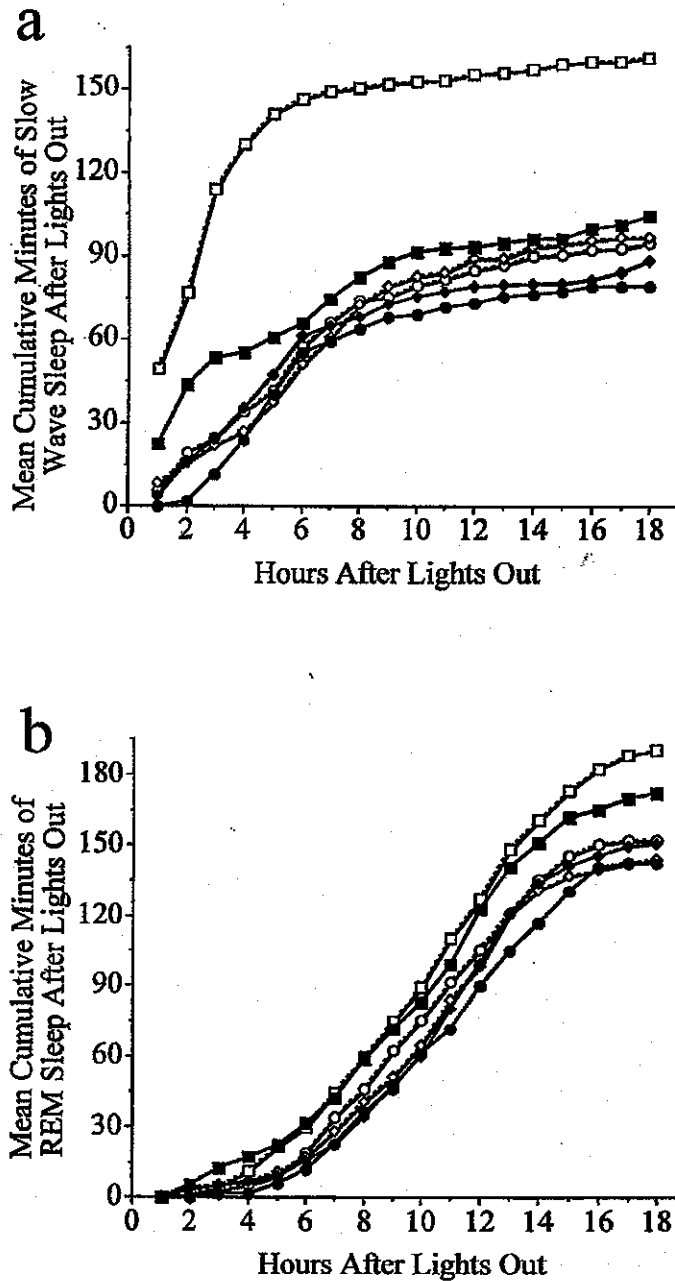


Fig. 4. - The mean number of cumulative minutes of SWS (a) and REM sleep (b) are plotted for each group each night as a function of time since lights out.

Symbols are as described for Figure 3. Distinctive among these curves is the rapid accumulation of high amounts of SWS in the deprived participants on Night 1.

of nights on REM sleep time in the analysis of variance. Figure 5 illustrates that the accumulation of REM sleep was affected by SWS accumulation; thus, REM sleep on the first night in the deprived group did not show an accelerated rate of increase until after about 2 to 2.5 hours of SWS, versus an accelerated increase after just an hour of SWS on the other nights in both groups.

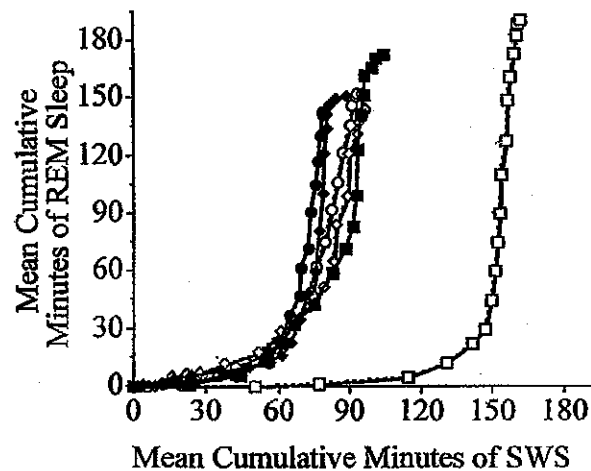


Fig. 5. - The cumulative minutes of REM sleep for each group and each night are plotted as a function of the cumulative minutes of SWS for that night.

Symbols are as described for Figure 3. Notable among these curves is the delay of REM sleep accumulation on Night 1 in the deprived group until after a large amount of SWS occurred.

#### DISCUSSION

This study of sleep on an atypical schedule of long nights in children who were either pretreated with "sufficient" sleep for a week or after a night of sleep deprivation provides a few important insights about the distribution and regulation of sleep. In the first place, the relative rapidity of sleep onset in the nondeprived group on the first long night is somewhat surprising, given that these youngsters were on a schedule involving 10 hours of scheduled sleep for over a week beforehand and that they had been awake for only 10 hours at bedtime. Whether this early sleep onset represents a residual sleep debt is uncertain. Clearly, the nondeprived group did not show an accelerated accumulation of SWS on Night 1 versus the other nights; however, total sleep on the first night ( $739 \pm 59$  minutes) was significantly longer than on Nights 2 and 3, even without prior sleep deprivation. In a similar way, some have suggested that the initial ability of adult participants to extend sleep in Wehr's study (20) of 14-hour nights indicated the dispersal of an accumulated sleep debt in adults who had slept about 8 hours per night during the weeks before the study. We have suggested elsewhere that adolescents require approximately 9 hours and 15 minutes for optimal sleep (21). Perhaps even at that level a chronic sleep deficit may accumulate. On the other hand, such a chronic low-level deficit may place a tonic load on the system providing a requisite tonic homeostatic pressure that optimizes sleeping and waking.

The occurrence of a significant difference in SWS as a function of Tanner stage and Night provides preliminary evidence that adolescent development may affect the homeostatic sleep/wake process. Such a relationship has previously been implied from the overall decline of SWS that occurs across the second decade (5). Yet, this developmental reduction of SWS may simply reflect the overall dampen-

ing of EEG amplitude both awake and asleep that occurs with age (11) rather than defining a process integral to sleep/wake homeostasis. Thus, the SWS response to sleep deprivation may be a more direct way to assess the homeostatic sleep process across age groups than simply examining SWS in the unperturbed state. The data presented here are not definitive on this issue, since we did not find a singular effect of sleep deprivation in the more mature than less mature participants. Nevertheless, our results point to an interesting trend that merits evaluation with more direct experimental approaches.

We were surprised not to see a return of SWS after 12 hours of sleep, as has been noted in young adults (12). Broughton and colleagues (4) propose that the 12-hour return of SWS indicates a semicircadian pattern in association with amplitude of the core temperature rhythm, and one might anticipate that this pattern would be more robust in younger individuals in whom SWS is more prominent overall. On the other hand, the studies of De Koninck and colleagues (13) indicate that the return of SWS may require a specific association between circadian phase and the timing of the extended sleep episode. Hence, the return of SWS in our participants might be expected to be 12 hours out of phase of the entrained sleep onset, i.e., near 1000-1200. By phase advancing sleep onset toward 1800, the temporal window for the return of SWS occurred at a time when our participants were most likely to be awake. Dijk and Czeisler (10) have noted a small but statistically significant circadian influence on slow wave EEG activity (SWA) in adults, as have we in adolescents (19). It remains to be determined, however, whether such a circadian influence occurs for slow wave sleep in adolescents and how a SWA or SWS circadian influence interacts with the timing of sleep.

#### SUMMARY

In summary, this study of sleep in adolescents on an atypical schedule of 18-hour nights showed marked but not unanticipated differences in sleep as function of prior sleep deprivation. Unanticipated was the evidence of "recovery" sleep in adolescents who not only were not sleep deprived, but who had been on a sleep "optimizing" schedule and had been awake for only 10 hours. Extended sleep beginning about 4 hours in advance of entrained sleep onset phase was not associated with a return of SWS, a finding coinciding with predictions from studies in adults. Finally, this study provides an indication that the homeostatic sleep/wake process becomes less robust or sleep responsive during adolescent development, a phenomenon that may influence the delay of sleep common in adolescents.

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