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Administration of triazolam prior to recovery sleep: effects on sleep architecture, subsequent alertness and performance*

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Abstract. The effects of triazolam (0.125, 0.25, and 0.5 mg) versus placebo on recovery sleep staging, subsequent alertness and psychomotor performance were evaluated in humans. Forty-five healthy male subjects were deprived of sleep for 24 h, then administered a single dose of triazolam or placebo using a double-blind procedure. Subjects then attempted to obtain recovery sleep under non-sleep-conducive conditions (sitting upright in a well-lit, crowded chamber) for the next 6 h, followed by 18 more hours of sleep deprivation. During all sleep deprivation periods subjects were tested bihourly on a performance assessment battery which included symbol digit modalities tests (SDMT), four-letter search (FLS), logical reasoning (LR), time estimation (TE), visual vigliance (VV), and short term memory (STM) tasks. Sleepiness levels were measured objectively with multiple sleep latency tests (MSLT) and subjectively with the Stanford Sleepiness Scale (SSS). Compared to placebo, all doses of triazolam resulted in increased amounts of stage 3–4 sleep, and the 0.5 mg dose significantly reduced awakenings ($Ps < 0.05$). Although subjects receiving triazolam averaged 21–42 min more total sleep time (TST) than subjects receiving placebo, differences in TST were not statistically significant. Apparent triazolam-mediated benefits to sleep qualitiy resulted in no obvious improvements in performance or alertness levels during subsequent sleep deprivation. It was concluded that the increases in stage 3–4 sleep amouts were most likely due to triazolam-mediated increases arousal thresholds, and the triazolam mediated changes in sleep parameters obtained in the present study were not indicative of substantial changes in the recuperative value of sleep.

Key words: Triazolam – Sleep deprivation – Psychomotor performance – Sleep stage

Typically, hypnotic medications such as triazolam are used by insomniacs to increase control over the timing and duration of night-time sleep periods. Increasingly, they are also used by normals who wish to hasten adaptation to new time zones. Thus, triazolam is used to promote or improve sleep when circumstances dictate that sleep quality would otherwise be poor. For these purposes triazolam has proven to be both safe and effective (e.g., Roth et al. 1976; Seidel et al. 1984; O'Donnell et al. 1988). However, sleep medications are generally needed only in the absence of a large sleep debt. That is, travelers can facilitate adaption to new time zones by depriving themselves of sleep beforehand. Likewise, persons suffering from insomnia can sometimes exert increased control over sleep timing by judicious use of sleep restriction and other behavioral techniques (Bootzin and Nicassio 1978).

Although the mechanisms by which sleep effects recuperation are unknown, it is known that the recuperative value of sleep is determined primarily by two sleep parameters: sleep duration and sleep continuity (Bonnet 1985, 1986, 1987; Downey and Bonnet 1987). Both triazolam and prior sleep loss impact positively upon these sleep parameters – triazolam improves sleep by both increasing sleep duration (e.g., Seidel et al. 1984; Walsh et al. 1984) and promoting sleep continuity (Seidel et al. 1984; O'Donnell et al. 1988), as does prior sleep loss (Williams et al. 1964; Kales et al. 1970). However, triazolam and prior sleep loss have divergent effects on sleep architecture. Specifically, triazolam has been reported to suppress sleep stages 3–4 (Kales et al. 1976; Spinweber and Johnson 1982; Mitler et al. 1984) and REM (Vogel et al. 1975; Kales et al. 1976; Spinweber and Johnson 1982) and increase the total amount of stage 2 sleep (Vogel et al. 1975; Kales et al. 1976; Mitler et al. 1984; Walsh et al. 1984), whereas prior sleep loss generally results in increases in stages 3–4 and REM (e.g., Williams et al. 1964). Because it is not known whether sleep stages 2, 3, 4, and REM have differential recuperative value (Johnson et al. 1974; Lubin et al. 1974) the significance of these triazolam- and sleep loss-induced changes in sleep architecture is unknown. Currently, studies which suggest neutral or negative next day effects of triazolam on alertness and performance (e.g., Nicholson and Stone 1980) outnumber those that suggest positive effects (e.g., Mitler et al. 1984) but there have been no studies specifically designed to compare the recuperative value of drug-induced versus natural sleep in normals.

In most previous studies triazolam has been administered shortly before the normal bedtime (usually at night) and subjects subsequently slept in chambers which were quiet, dark, and comfortable – simulating the bedroom con-

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ditions under which most people choose to sleep. However, the environmental and situational conditions under which individuals sometimes must try to sleep can vary widely. For example, during military operations soldiers may suffer from chronic sleep restriction, obtaining recovery sleep on an irregular and unpredictable schedule, under environmental conditions which are not conducive to sleep (e.g., in crowded and noisy airplanes or trucks). Typically, sleep deprived normals experience little difficulty initiating and maintaining sleep since sleep loss not only hastens sleep onset but also raises the sleepers' threshold to the disruptive effects of external stimulation (Williams et al. 1964). However, even sleep deprived individuals are not completely immune to the alerting effects of the environment and may experience environmentally-mediated sleep disruption. The purpose of the present study was to determine whether any benefits to sleep, subsequent performance, or alertness are derived from administration of triazolam to sleep-deprived normals who are trying to sleep under non-sleep-conducive environmental conditions.

Material and methods

Subjects. The subjects were 45 healthy non-smoking male volunteers, aged 18–39 years ($\bar{x}=23.3$ years). Potential subjects were excluded if they had any of the following characteristics: current illness, presence of heart murmur, blood pressure >140/90, history of: impaired renal or hepatic functions, pulmonary insufficiency, organic heart disease, sleep disorder, in-patient psychiatric therapy, caffeine use in excess of 500 mg per day, or the use of benzodiazepine compounds, major tranquilizers, or antidepressants. Subjects were instructed to abstain from drug and alcohol use for 48 h prior to the study, and compliance was checked with a urine drug screen on the last day of the study. They were paid a base rate of $ 200 for completing the study, with the possibility of a $ 50 performance bonus as described below.

Apparatus. A soundproof, shielded chamber (3.2 m × 4.7 m × 2.5 m high) was used as the sleeping area. This chamber was kept well lit, the temperature was 80° F, and subjects were required to sit upright in comfortable, cushioned chairs while attempting to sleep. In addition to the four subjects, a staff member was present at all times during the sleep period. A separate, darkened, quiet chamber containing comfortable beds was used for multiple sleep latency tests (described below) and for recovery sleep at the end of the study.

Psychophysiologic sleep data were collected through continuous recording on 9 channel Medilog recorders using EEG electrodes on C3 and C4 sites, submental EMG, and EOG from the outer canthus near each eye. All polysomnographic data were scored manually, using the guidelines of Rechtschaffen and Kales (1968), by experienced sleep scores who were blind to drug condition.

Sleepiness measures. Multiple sleep latency tests (MSLTs) were used to objectively determine sleepiness in subjects (Richardson et al. 1978). In this test subjects were asked to try to fall asleep while reclining on a comfortable bed in a darkened, quiet room with their eyes closed for 15 min. Sleep latency was operationally defined as the time from lights off until the first 60 s of stage 2 sleep. A computerized version of the Stanford sleepiness scale (SSS), a seven-point self-rating scale of sleepiness (Hoddes et al. 1973), was administered as part of the bi-hourly performance assessment battery.

Symbol Digit Modalities Test (SDMT). The integration of verbal and nonverbal mental processes were measured using the written version of the symbol digit modalities test (SDMT). The SDMT substitution process involved identifying by code number each of nine geometric designs which appear on the page repeatedly (in irregular sequences) as quickly and accurately as possible, within a 90 s time limit. The test consists of 120 geometric designs, 10 of which are practice, and the subject was instructed to complete as many items as possible, as accurately as possible, within the allotted 90 s. This test is described in detail by Smith (1973). A different, equivalent form of the test was administered during each test session, to eliminate the possibility of learning effects.

Four-Letter Search (FLS). This was a visual search and recognition task in which four target letters were presented at the top of the screen, along with 20 letters at the middle of the screen. The subject's task was to indicate by keypress, as quickly as possible, whether the four target letters were present in the group of 20. Ten trials were administered during each test session.

Logical Reasoning (LR). This task required interpretation of transformational grammar. The letter pair "AB" or "BA" was presented along with a statement that either correctly or incorrectly described the order of the letters within the pair (e.g., "A is not preceded by B"). The subject's task was to indicate by keypress, as quickly as possible, whether the sentence accurately described the relationship of the presented letter pair. Each test session contained 50 trials, which were scored for both speed and accuracy.

Time Estimation (TE). In this task, the subject viewed a dot of light that descends from the top of a computer screen to a point one-third of the way from the bottom of the screen. At that point, the light appeared to pass behind a barrier; the subject's task was to depress the spacebar of the computer keyboard when he believed the dot (no longer visible) had reached a designated line at the bottom of the barrier. Ten trials per administration of the test were given.

Visual Vigilance (VV). In this task subjects were required to watch the display of a computer monitor as single letters were presented serially, and to respond by pressing the spacebar on a computer keyboard whenever the letter "A" was followed by the letter "F". Within each of eight trial blocks, subjects were presented 128 letters in a pseudorandomized order. Of the 128 letters presented in each block, 16 "A's" were presented which were not followed by "F", 16 "F's" were presented which were not preceded by "A", and 16 "A–F" combinations were presented. The remaining 32 presentations were "C", "E", "H", "G", "I", "O", "P", or "S". Each letter was presented for 400 ms with a 700 ms interstimulus interval. The subjects were required to respond within the 700 ms interstimulus interval.

Short Term Memory (STM). This task tested the ability of subjects to recall the serial order in which letters were

presented on a screen. Subjects were presented seven letters, one at a time, with each letter displayed for 500 ms. After a 1-s delay, the "target" letter, which was one of the seven preceding letters, was displayed and the subject had 3 s to type the number (1–7) corresponding to the target letter position within the previously presented string. A sequence presentation plus a response constituted a trial. Subjects received four blocks of 30 trials, separated by 30-s rest periods.

Together, these measures constituted the performance assessment battery, and took 90 min to complete. The battery was administered at 1500, 1700, 1900, 2100, 2300, 0100, 0300, and 0500 hours on both days of the study. Within each 90-min test session, the order of test administration was: MSLT, SDMT, SSS, FLS, LR, TE, VV, then STM. The SSS, FLS, LR, and TE tests are components of the Walter Reed Performance Assessment Battery (Thorne et al. 1985) and were presented using IBM XT computer systems. Subjects received four training session on each of these measures on the morning of day 1. The VV and STM tests were developed at the Sleep Laboratory at Bowling Green State University, and were presented on Apple IIe computer systems. Subjects received one training session on each of these tests on the morning of day 1.

Multiple forms of an addition test were administered at 1400–1430 hours on both days to examine the possible effects of triazolam on sleep inertia effects. These data will be discussed elsewhere.

To help maintain high levels of motivation and maximize performance throughout the study, subjects were told that they could earn a $ 50.00 bonus if their performance on the psychomotor tests exceeded an unnamed criterion. The criterion was, in fact, easy to exceed – maintenance of 70% accuracy on the FLS test during the first three test sessions – and all subjects were expected to earn the bonus.

Procedure. Subjects were usually run in groups of four although occasional no-shows resulted in some sessions with only two or three subjects. Subjects reported to the laboratory at 0800 hours on day 1, at which time they received training on the performance measures and had electrodes attached for continuous recording of EEG (from C_3 and C_4 sites on the scalp), EOG from the outer canthus near each eye, submental EMG, and EKG. At 1400 hours, subjects were administered multiple forms of an addition test and sleepiness rating scales. Starting at 1500 hours, subjects were administered the test battery and modified multiple sleep latency test at bi-hourly intervals. Each test session took 90 minutes so subjects received a 30 min rest period every 2 h during which they were allowed to watch videotaped movies, read, talk, and eat ad lib. (except no food was allowed after midnight). They were not allowed to sleep during the 30 min breaks. This schedule was maintained until 0700 hours of day 2, when indwelling catheters were inserted in the forearm of each subject, using the non-dominant arm when possible. Drugs (placebo, 0.125, 0.25, or 0.5 mg triazolam) were administered orally using a double-blind procedure at 0800 hours. Immediately after drug administration subjects were escorted to a well-lit chamber where they were instructed to try to sleep while sitting upright in cushioned chairs. Lights remained on during the 6-h sleep period, and 5 ml blood samples were collected immediately before drug administration and at 30, 45, 60, 75, 90, 120, 150, 180, 240, 300, 360, 420, 540, and 660 min post-drug administration – thereby constituting a potentially significant source of sleep disruption. At 1400 hours subjects were awakened (if asleep). The performance assessment battery and sleep latency tests were administered on the same schedule as day 1, at bi-hourly intervals starting at 1500 hours and continuing until 0630 hours the following morning, when subjects obtained at least 6 h of recovery sleep (in a separate, dark, quiet chamber containing four bunk beds) before having electrodes removed, being debriefed, and being released from the study.

Analyses. Data from the 6-h daytime sleep period were analyzed using a multivariate analysis of variance to compare mean values of sleep parameters (minutes of stages 1, 2 3–4, REM; number of minutes of wake time after initial sleep onset; latency to 60 s of stage 2 sleep; total sleep time) of the four Drug groups. Because of their importance in determining the recuperative value of sleep, planned comparisons (F tests for the individual components of variation) of total sleep time (TST) and number of minutes of wake time after initial sleep onset (WAKES) were performed to determine differences between drug groups. Post hoc comparisons, when appropriate, were made using Duncan's multiple range tests.

Performance and sleep latency data were analyzed using a 4X2X8 analysis of variance, with Drug level as the first factor, Day as the second factor, and Time of Day as the third factor (repeated measures on the latter two factors). For each measure the effects of interest were (a) the main effect of Day, which indicated whether overall performance was affected by the sleep manipulations (i.e., sleep deprivation followed by daytime recovery sleep), (b) the Day X Time of Day interaction effect, following which post hoc Duncan's multiple range tests were employed to determine whether the sleep manipulations had differentially affected performance across the various testing times (c) the Drug X Day interaction effect, which indicated whether the drug levels differentially affected performance after recovery sleep, with post-hoc Duncan's tests to determine which drug levels were associated with performance changes across days, and (d) the DrugX DayX Time of Day interaction, with post hoc Duncan's tests to determine whether differences in performance existed between drug levels for each test on each day. A probability level of 0.05 was used as the criterion for statistical significance in all analyses. For repeated measures effects the Greenhouse-Geisser criterion was used. Slight variations in degrees of freedom in the following analyses are due to occasional loss of data because of technical problems (e.g., detached electrodes or power failures during computer-administered performance measures).

Results

Six-hour sleep period

Multivariate analysis of variance of the 6-h sleep period immediately following drug administration revealed no significant effects of drug group on total sleep time (TST) or the number of minutes of mid-sleep awakenings (WAKES), though planned comparisons showed that WAKES was greater in the placebo group (mean = 78.8 min) than in the 0.5 mg triazolam group (mean = 37.6 min). For the placebo, 0.125, 0.25 and 0.5 mg triazo-

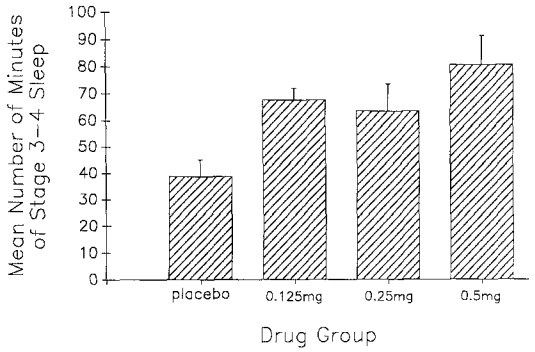

Fig. 1. Mean amounts of stage 3–4 sleep obtained during the 6-h recovery sleep period for each drug group

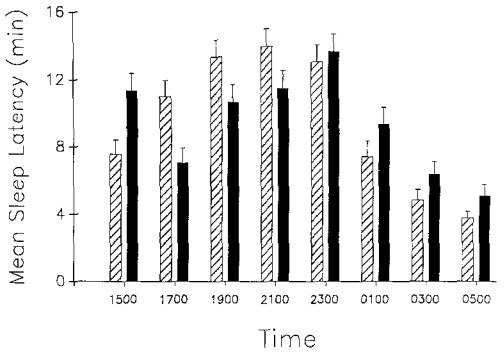

Fig. 2. Comparison of mean sleep latencies on day 1 versus day 2, during each bi-hourly test session. □ Day 1; ■ day 2

lam groups TST values were 257.5, 299.5, 278.6, and 298.9 min, respectively. But despite the relatively lower mean TST of the placebo group, planned comparisons failed to reveal significant differences. With respect to sleep architecture, the only significant effect due to drug group was found for minutes of stage 3–4 [$F(3,39)=4.51$, $P=0.0087$]. Post hoc Duncan's multiple range tests revealed that subjects in the placebo group obtained less stage 3–4 (mean = 38.7 min) than subjects in the 0.125, 0.25, and 0.5 mg triazolam groups (means = 67.4, 63.3, and 80.3 min, respectively – see Fig. 1).

Multiple sleep latency test

As expected, analyses revealed a significant effect of Time of Day on sleep latency [$F(7,245)=104.23$, $P<0.0001$], indicating circadian fluctuations in alertness, and a significant Day X Time of Day interaction [$F(7,245)=5.23$, $P<0.0001$], indicating the effects of the sleep loss followed by daytime recovery sleep on subsequent alertness levels. Post-hoc Duncan's tests showed that the mean sleep latency at 1500 hours was longer on day 2 than on day 1, but latencies at 1700 and 1900 hours were shorter on day 2 than day 1. Although no other differences were statistically significant, there appeared to be a trend for latencies to be longer on day 2 after 2300 hours (see Fig. 2). Analyses revealed no drug group-related differences in latencies or minutes of stages 1, 2, 3–4, and REM during the bi-hourly sleep latency tests. There was, however, a significant main effect of Time of Day on minutes of REM [$F(7,252)=4.90$, $P<0.0076$]. Post-hoc Duncan's tests revealed that significantly more REM occurred during the 0500 naps than at any other

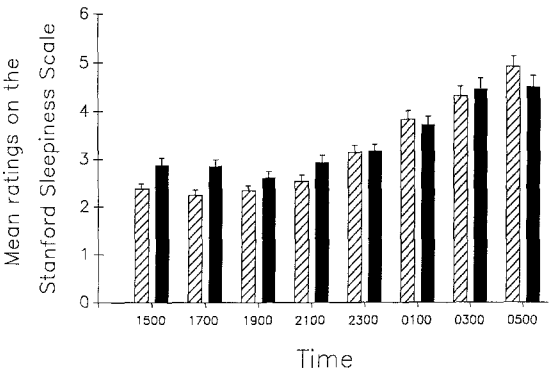

Fig. 3. Mean ratings on the Stanford Sleepiness Scale across days 1 and 2. □ Day 1; ■ day 2

times ($P<0.05$). Similarly, there was a main effect of Time of Day for minutes of stage 3–4 [$F(7,252)=5.50$, $P<0.0025$]. Post-hoc analysis revealed that more stage 3–4 was obtained during the early morning hours (0300 and 0500) than at any other times. But for both REM and stage 3–4, the mean amounts obtained during any sleep latency test were small – always less than 1 min. The analysis also revealed a main effect of Day, indicating that more stage 3–4 was obtained during the sleep latency tests on day 1 (mean = 0.15 min) than on day 2 (mean = 0.05 min) [$F(1,36)=6.10$, $P<0.0184$].

Stanford sleepiness scale

A significant main effect of Time of Day [$F(7,245)=104.23$, $P<0.0001$] indicated circadian fluctuations in self-rated alertness. Mean SSS ratings from 1500, 1700, 1900, 2100, 2300, 0100, 0300, and 0800 hours were 2.63, 2.64, 2.48, 2.73, 3.16, 3.79, 4.39, and 4.72, respectively. Post hoc analyses revealed that self ratings of sleepiness increased significantly every 2 h after 2100 hours. However, there were no significant differences between ratings obtained at 2100 hours or earlier. As in the sleep latency data, the Day X Time interaction was also significant [$F(7,245)=5.23$, $P=0.0004$], indicating that the sleep deprivation and the subsequent sleep periods significantly altered subjective alertness (see Fig. 3). Post-hoc Duncan's multiple range tests revealed that across days, self-rated sleepiness was greater at 1500 hours on day 2 than on day 1, and was less at 0500 hours on day 2 than on day 1.

Psychomotor tests

Significant results from analyses of the psychomotor tests comprising the performance assessment battery are summarized in Table 1. Main effects of Time and Day were found for many measures, indicating circadian fluctuations in performance and the effects of the sleep manipulations, respectively. For example, Fig. 4 shows the mean number of errors of omission on the VV task during each test session, across both days of the study. This figure clearly shows the circadian rhythmicity of performance and the trend toward poorer performance (more errors of omission) on day 2. (The one exception in this figure is the relatively improved performance on day 2 at 0500 hours – which could be attributed to end-spurt effects). These findings suggest that the psychomotor tasks administered in the

Table 1. Summary of significant main and interaction effects obtained with psychomotor performance measures

Task	Significant effects	*df*	*F* value	*P* level
Symbol digit Modalities SDMT (number correct)	Time	(7,259)	21.51	0.0001
	DayX Time	(7,259)	4.75	0.0010
Visual Vigilance (errors of ommission)	Time	(7,245)	15.32	0.0001
	Day	(1,35)	5.15	0.0295
	DayX Time	(7,245)	7.48	0.0001
	TimeX Drug	(21,245)	2.03	0.0487
Visual Vigilance (errors of commission)	Time	(7,245)	2.40	0.0214
	Day	(1,35)	5.46	0.0253
Logical Reasoning (number of errors)	DayX Time XDrug	(21,245)	1.68	0.0490
4-Letter Search (FLS) (response speed)	Day	(1,35)	31.51	0.0001
	DayX Drug	(3,35)	3.83	0.0179
Time Estimation	none	N/A	N/A	>0.05
Short Term Memory (number of incorrect responses)	Day	(1,34)	12.21	0.0013
	Time	(7,238)	12.24	0.0001
	DayX Time	(7,238)	2.98	0.0250
Short Term Memory (number of failures to respond within 3 s time limit)	Time	(7,238)	8.00	0.0002
	DayX Time	(7,238)	2.97	0.0270
	DayX Drug	(3,34)	3.46	0.0269

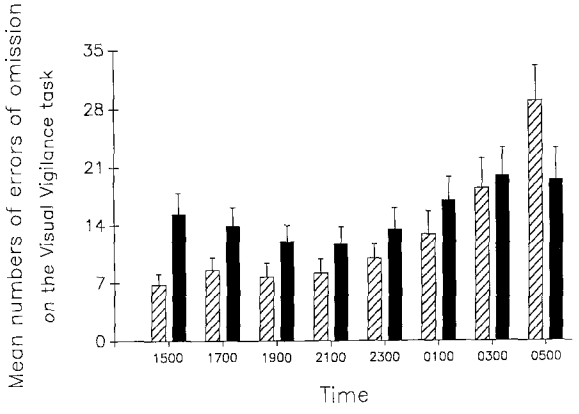

Fig. 4. Mean number of errors of omission (failures to respond within the allotted 700 ms) on the visual vigilance task for days 1 and 2, during each bi-hourly test session. □ Day 1; ■ day 2

present study are sensitive to variations in alertness across the day, sleep loss, and recovery sleep.

However, there were relatively few measures in which differential effects due to drug level were found. A significant DayX Drug interaction was obtained with the Four-Letter Search task (mean speed of responding). As shown in Fig. 5, there was an apparent trend toward faster responding on day 2 within each drug group, but only the 0.125 mg triazolam group responded significantly faster on day 2 ($P<0.05$). As shown in Table 1, a significant DayX Drug interaction was obtained with the Short Term Memory task (mean number of failures to respond within the allotted 3 s), and a significant TimeX Drug interaction was obtained with the Visual Vigilance task (mean number of errors of omission). Also, the DayX Time of DayX Drug interaction was significant on the Logical Reasoning task

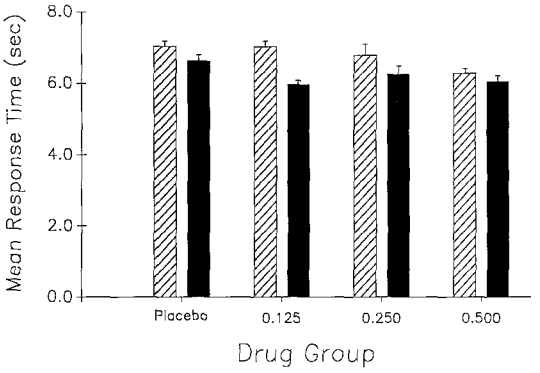

Fig. 5. Mean response time on the four-letter search (FLS) task on day 1 versus day 2, for each drug group . □ Day 1; ■ day 2

(mean number of errors). However, post-hoc analyses revealed no potentially relevant differences in these measures (e.g., post hoc analyses of the DayX Drug interaction in the STM data revealed no significant within-group differences across days).

Discussion

The results suggest that the qualitiy of recovery sleep under non-sleep-conducive conditions was measurably improved by triazolam, but there were virtually no apparent benefits of this improved sleep on subsequent psychomotor performance and alertness measures. All groups receiving triazolam obtained significantly more stage 3–4 sleep than the placebo group, a finding which was surprising since previous studies have shown that triazolam suppresses stages 3–4 and REM. However, since it has also been shown that triazolam administration raises auditory arousal threshold during sleep (Spinweber and Johnson 1982), it is likely that increased amounts of stage 3–4 following triazolam administration in the present study were due to the combined effects of sleep loss-mediated increases in pressure to stage 3–4 and triazolam-mediated increases in arousal threshold. That is, triazolam may have helped protect the subjects from the arousing effects of extant environmental stimuli, thus allowing subjects to enter and maintain stage 3–4 sleep more effectively. This interpretation is supported by the finding that sleep continuity (as measured by amout of mid-sleep wake time) was improved in the 0.5 mg triazolam group compared to the placebo group.

Although subjects in the placebo group averaged 21–42 min less TST than subjects receiving triazolam, analyses revealed no significant between-group differences in TST. Nevertheless, it remained possible that these non-significant drug group differences in TST, in combination with significant differences in sleep continuity and stage 3–4 amounts, might result in improved ability to withstand the deleterious effects of subsequent sleep deprivation. However, subjective (SSS) and objective (sleep latency) measures of alertness revealed no differentially beneficial effects of triazolam-induced recovery sleep during the subsequent sleep deprivation period. Likewise, the apparent drug-mediated differences in the daytime sleep period did not result in improved psychomotor performance during subsequent sleep deprivation, except for the finding that response latency on the FLS task was significantly improved on day 2 in the 0.125 mg triazolam group. Although this finding

might tend to suggest differential recuperative or inoculative effects of triazolam- versus placebo- induced sleep, little weight should be accorded this solitary finding because: (a) this effect was only significant for the 0.125 mg triazolam group, (b) the trend suggested faster responding on day 2 even in the placebo group (see Fig. 5) and (c) similar effects were not found in the other psychomotor tasks in which speed of responding was measured – in fact, significant drug-related interactions were obtained for only a few of the psychomotor tasks, and post hoc analyses failed to reveal any other potentially meaningful differences between means.

Overall, in the present study triazolam administration prior to recovery sleep (under non-sleep-conducive conditions) resulted in positive effects on stage 3–4 amounts, sleep continuity, and (non-significantly) total sleep time. However, these effects on recovery sleep were not convincingly translated into inoculative effects on measures of alertness or psychomotor performance during subsequent sleep deprivation. There are several possible explanations for the failure to find a relationship between daytime sleep parameters and subsequent alertness and performance in the present study: (a) It is possible that the performance and alertness measures used in the present study were not sensitive enough to detect the improvements resulting from the drug-mediated alterations in sleep. However, this does not seem likely since the measures were sensitive to sleep loss and circadian effects. As an example, circadian trends in alertness and performance are clearly visible in Figs. 3 and 4. (b) It is possible that the study was terminated too early – had the post-recovery sleep deprivation period been extended (e.g., to 48 h or longer) perhaps beneficial, inoculative effects of triazolam-induced changes in recovery sleep would have emerged. (c) It is also possible that all groups (including placebo) obtained fairly adequate recovery sleep, and the drug-mediated differences in recovery sleep parameters found in the present study did not represent important differences in recuperative value. This interpretation is consistent with Horne's (1988) notion that only a small portion of sleep is actually necessary for restitution ("core" sleep) and the rest of the sleep that is obtained is "optional" sleep, in that it serves no (or a greatly diminished) restitutive function. In future studies, recovery sleep periods of varying durations should be used to delineate the relationship between sleep duration and restitution. Comparisons of performance following natural versus drug-induced sleep may prove to be more sensitive as sleep durations are shortened, and the percentage of core sleep is increased.

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