The restorative effect of naps on perceptual deterioration

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Human performance on visual texture discrimination tasks improves slowly (over days) in the absence of additional training. This 'slow learning' requires nocturnal sleep after training and is limited to the region of visual space in which training occurred. Here, we tested human subjects four times in one day and found that with repeated, within-day testing, perceptual thresholds actually increased progressively across the four test sessions. This performance deterioration was prevented either by shifting the target stimuli to an untrained region of visual space or by having the subjects take a mid-day nap between the second and third sessions.

As life in our culture becomes more demanding, average nightly sleep is decreasing in all segments of the population (Stein, M., National Sleep Foundation Poll, 2001, www.sleepfoundation.org/PressArchives/lessfun_lesssleep.html). 'Power naps'—brief periods of daytime sleep lasting an hour or less—improve alertness, productivity and mood[1,2], especially under sleep-deprived conditions[3,4], during nightshift work[5] and during prolonged periods of driving[6]. Although naps have been shown to enhance psychomotor speed as well as short-term memory acquisition[7,8], the effect of daytime naps on previously learned information is not known. The finding that power naps are common among people reporting daily information overload indicates that napping supports a previously unknown mechanism of off-line information processing, perhaps related to that which normally occurs during nocturnal sleep[9–16].

We investigated the phenomenon of information overload at the perceptual level. Typically in visual perception tasks, fast learning happens in the first minutes to hours of training[17,18]. Previous studies using a visual texture discrimination task (TDT)[19] show that a slower phase of perceptual learning also exists, which depends on nocturnal sleep after training[9,14,19–22]. The slow phase of improvement becomes evident only after at least six hours of nocturnal sleep[14], and sleep deprivation the night after training eliminates the normal post-sleep improvement, even when measured after two full nights of recovery sleep[15]. The improvement seen in subjects who sleep for eight hours during the night after training correlates with the proportion of deep, slow wave sleep (SWS) in the first quarter of the night and with the proportion of rapid eye movement sleep (REM) in the last quarter[14]. These results indicate that a full night of sleep is important for maintenance and consolidation of experience-dependent learning, and that without at least six hours of sleep, this potential consolidation is lost. These studies do not, however, address the question of how power naps of an hour or less could aid in such information processing.

Here we show that perceptual performance declined on the TDT with repeated, within-day training. In the context of this deterioration, we found that (i) a daytime nap, but not an equivalent period of rest without visual input, reversed the deterioration, (ii) the deterioration was retinotopically specific and (iii) neither an increase in subject motivation nor a decrease in task difficulty improved performance.

Results

Can too much practice be detrimental?

To investigate whether repeated within-day testing on a perceptual learning task can impair performance, subjects were tested on the TDT four times in a single day (at 9 a.m., 12 p.m., 4 p.m. and 7 p.m.). Each session lasted approximately 60 minutes. For each session, the speed of perceptual processing was calculated as the threshold target-to-mask interstimulus interval (ISI) needed to achieve 80% accuracy. Thirty subjects were randomly assigned to one of three groups: control, long nap or short nap. Control subjects ($n = 10$) showed a 52% slowing in perceptual processing across the four test sessions (**Fig. 1**, filled circles; $P = 0.0003$, repeated measures analysis of variance (ANOVA) and *post hoc* tests). Thus, with each successive session, subjects needed increasingly longer exposures to the stimuli to reliably identify targets. Performance deteriorated despite all testing being done within 12 hours of morning awakening, a time when one would not normally expect to see cognitive impairment, and without prior sleep deprivation. Subjects averaged 6.92 ± 0.77 (s.d.) hours of sleep on the night before testing.

Can daytime sleep reverse perceptual deterioration?

As nocturnal sleep is known to enhance alertness and to consolidate TDT learning[9,14,19,22], we asked whether a daytime nap might stop or even reverse the process of deterioration seen with repeated within-day testing. The remaining 20 subjects were randomly assigned to a long (60-minute) or short (30-minute) nap condition. All subjects, including no-nap controls, performed the task four times during the day; experimental subjects took a

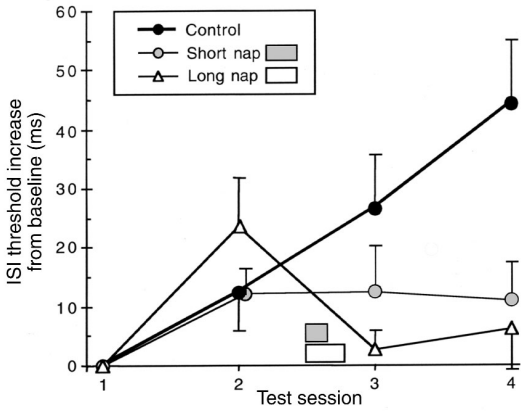

Fig. 1. Beneficial effect of napping. Performance of control and both short and long nap groups across same-day sessions. ISI thresholds from the first session defined baseline performance, and were subtracted from thresholds for subsequent sessions to determine relative change in performance.

nap at 2 p.m.—midway between the second and third test sessions. As predicted, napping significantly affected subsequent performance ($P = 0.001$, group × session interaction, mixed-model ANOVA): short naps prevented the normal deterioration that was seen in test sessions 3 and 4 (**Fig. 1**, open circles) and long naps reversed the deterioration seen in the second test session (**Fig. 1**, open triangles). Thus, whereas controls showed a 14.1-ms increase in ISI threshold between the second and third sessions, the short nap group showed no change (<1-ms increase) and the long nap group showed a 20.9-ms decrease ($P = 0.03$, paired *t*-test). The short nap group showed no significant change in ISI thresholds across the last three sessions ($P = 0.94$), but did show significantly lower thresholds in the fourth session than did controls ($P = 0.01$). The long nap group showed significantly better performance than controls on both the third and fourth sessions ($P = 0.03$). We compared the distributions of sleep stages in long naps versus short naps (**Table 1**) and found no significant differences in the amount of time spent in stage 1 or stage 2 sleep. There was, however, a difference for SWS and REM sleep: the long naps contained 4.1–4.4 times more SWS and REM sleep than the short naps did. This matches the SWS and REM dependency reported for overnight improvement on the task[14], suggesting that processes occurring during SWS and/or REM underlie sleep-induced perceptual recovery.

We compared naps taken on the day of TDT testing with baseline naps taken on a different day (see Methods) and found that the long nap group spent significantly more time in SWS on the test day (27.4 versus 20.9 minutes, $P < 0.05$, one-tailed paired *t*-test) at the expense of time in stage 2 sleep (17.9 versus 25.0 minutes, $P = 0.06$) (**Fig. 2**). This large increase in SWS in test-day naps (31% over baseline) suggests that SWS is crucial for post-nap performance, perhaps through stabilizing and consolidating plastic neuronal changes from earlier in the day. Such a function has previously been proposed for nocturnal SWS (refs. 23–25).

The role of REM, however, is less clear. The increase in time spent in REM during test-day naps compared with baseline naps (60% increase) was larger than that seen for SWS, but was not statistically significant (**Fig. 2**). Similar post-training REM sleep increases have been reported in animal studies of sleep-dependent learning[26]. In the short nap condition, there was no significant change in SWS (9.2 minutes on test day versus 6.7 minutes baseline) or REM (0.8 minutes on test day versus 2.0 minutes baseline) duration. Nap duration was controlled in both groups and neither group showed a significant difference in nap duration between baseline and test-day naps ($P > 0.27$).

Sleep versus rest

To test whether the benefits of naps resulted from the absence of visual input rather than from sleep itself, subjects ($n = 9$) repeated the long nap protocol; but instead of sleeping, they rested quietly while blindfolded. Wake–sleep state was continuously monitored physiologically to ensure maintained wakefulness. The hour of rest without visual input did not have a restorative effect: subjects still showed performance decrements at 4 p.m. and 7 p.m. ISI thresholds increased by an average of 29.0 ms between the second and fourth session ($P < 0.05$), a performance decrement nearly identical ($P = 0.31$) to the 32-ms increase seen in controls (**Fig. 1**).

Motivation and task difficulty

To test whether a decrease in motivation contributed to the performance deterioration, we informed subjects ($n = 10$) after their second session that their performance had worsened, and that they would receive a cash bonus if they subsequently returned to their baseline performance. Even with this incentive, none of the subjects regained baseline performance during the third or fourth sessions, and mean ISI thresholds were 32.2 ms longer on the fourth session compared to the first session ($P = 0.001$).

To test whether the performance decrement resulted specifically from exposure to more difficult trials, we tested ten subjects throughout the day in four sessions, with all blocks in the first three sessions run at the longest ISI (400 ms). Subjects were then tested in the fourth session with the standard 25 blocks of decreasing ISIs, and their performance in the fourth session did not differ from that of controls ($P = 0.50$). Thus, despite having an easier task in the first three sessions, subsequent performance still declined.

What aspect of processing is impaired?

Several mechanisms might underlie this deterioration. A generalized fatigue effect, mediated by a decrease in alertness or attentional resources, is one possibility that is consistent with our data. Alternatively, we propose that specific neural networks in primary visual cortex gradually become saturated with information through repeated testing, preventing further perceptual processing. This would cause a training-specific deterioration in per-

Table 1. Characteristics of short and long naps.

	S1 (min)	S2 (min)	SWS (min)	REM (min)	ΔThreshold (ms)
Short naps	6.2 ± 1.1	14.3 ± 1.5	6.6 ± 2.1	2.0 ± 1.4	4.5 ± 10.7
Long naps	5.5 ± 0.7	17.9 ± 3.0	27.4 ± 3.8	8.9 ± 3.4	20.8 ± 7.6
P value	0.85	0.22	0.0001	0.07	

The mean times spent in each sleep stage during short (30-min) and long (60-min) naps are presented as min ± s.e.m. S1, stage 1; S2, stage 2; SWS, slow wave sleep (stages 3 and 4); REM, rapid eye movement sleep. *P* values from unpaired *t*-test comparing times in each stage for long versus short naps. ΔThreshold, the difference between ISI thresholds on the second (pre-nap) and third (post-nap) tests, expressed as ms ± s.e.m.

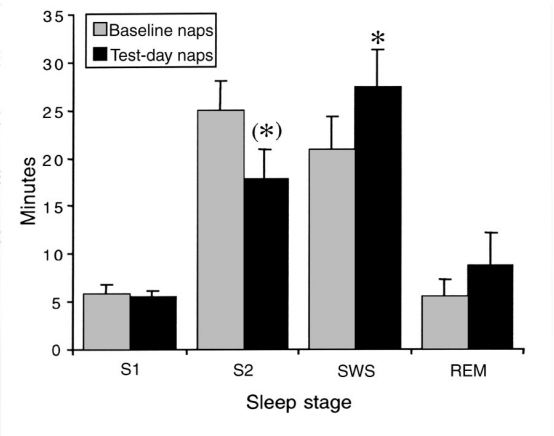

Fig. 2. Comparison of test-day and baseline long naps. The number of minutes spent in stage 1 (S1), stage 2 (S2), rapid eye movement (REM) and slow wave sleep (SWS; stage 3 and 4) during baseline (gray bars) and test-day (solid bars) naps. * $P < 0.05$; (*) $P = 0.06$.

ceptual processing. Whereas the generalized fatigue hypothesis predicts that decrements in performance would be widespread and largely task-independent, our hypothesis of a training-specific deterioration predicts that the performance decrements would be restricted to behaviors mediated by the specific neural networks previously involved in processing the target stimuli. We reasoned that because TDT learning does not transfer to untrained portions of the visual field[20], there should be no training-specific deterioration if stimuli are presented to an untrained region of the visual cortex.

To test this hypothesis, we trained 24 subjects as before, but for half of them we switched the target stimuli to the contralateral visual field for the fourth and final same-day test session. Performance of the switch group did not differ significantly from that of the control group across the first three sessions, but, unlike the control group, the switch group showed significant recovery in the fourth session (**Fig. 3**, $P = 0.002$, ANOVA group × session interaction and *post hoc* test). Performance during the switch condition was not significantly worse than it was during the first session, indicating that the behavioral deterioration observed in the trained visual quadrant did not transfer to the untrained contralateral quadrant. These results strongly support the training-specific deterioration hypothesis, and are contrary to the predictions of the generalized fatigue hypothesis.

Further evidence against the generalized fatigue hypothesis comes from the dissociation between improved performance and subjective levels of sleepiness. If the steady decrease in performance throughout the day in the control group resulted from a general fatigue effect, then one should see a parallel increase in reported sleepiness. But no such increase was seen, and mean levels of subjective sleepiness on the first and last tests were identical ($P = 0.45$, repeated measures ANOVA). Similarly, the switch group showed no significant change in sleepiness across sessions ($P = 0.49$). In contrast, sleepiness decreased from the first to the last session in the nap groups ($P < 0.03$, ANOVA and *post hoc* tests). Thus, the switch group showed the same improvement in performance as the nap groups did, but without a similar decrease in subjective sleepiness; compared to controls, the switch group showed the same maintenance in degree of sleepiness, but no deterioration in performance.

Data from the switch group also eliminated another possible explanation, that of a strictly circadian effect. Although the control data could have been explained as a circadian rather than repetition effect, the fact that shifting the stimulus to the contralateral visual field for the last session reversed this decrease eliminates this possibility. Thus, when subjects were tested at 7 p.m. with stimuli in an untrained region of visual space, they performed as well as they had at 9 a.m. the same morning.

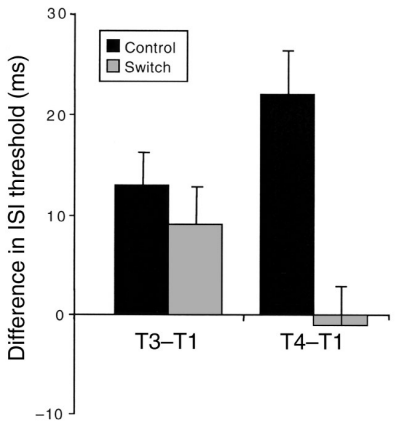

Fig. 3. Beneficial effect of shifting stimulus location. Solid bars, deterioration in performance of control subjects during the third (T3) and fourth (T4) sessions of the day. Gray bars, deterioration in performance of experimental 'switch group' subjects during T3 followed by recovery during T4 when stimulus location was shifted.

Discussion

Two learning components occur with TDT testing: a fast, within-session component and a slow, sleep-dependent component[16]. Our study identified a third consequence of TDT training: with repeated, same-day training, people require progressively longer ISIs for texture discrimination. Such perceptual deterioration has not previously been reported for repeated same-day testing on other visual tasks. Task procedure may contribute to this difference, such as whether the task measures vernier acuity[27], resolution acuity[27] or texture discrimination[19], whether stimuli are presented foveally[17,18], parafoveally[27] or at more peripheral eccentricities[19], and whether stimulus presentations are long (100–150 ms)[17,18,27] or short (17 ms)[19]. Furthermore, a number of perceptual learning protocols train subjects over several days[17,18,27–29] rather than within-day, as in the current study, making it unclear whether fast or slow learning is occurring. With these caveats in mind, the present study shows that some forms of neural plasticity that require sleep for subsequent consolidation and improvement of perception may actually hinder performance before sleep.

Our three main findings—that there was a normal decline in TDT performance across the day with repeated exposure to the task, that this decline was specific to previously trained regions of visual space and that performance was restored by daytime napping—have important implications. First, as circadian influences have been ruled out, the performance decline must result from specific neuronal changes induced by the initial testing period. Second, as brain regions involved in higher levels of visual

processing lack retinotopic specificity, the critically affected neurons are most likely located in early visual processing areas. Finally, these initially affected neural networks must be further altered during napping to reverse the performance decrement.

We propose that the performance decrement seen here was a direct consequence of a mechanism for preserving information that has been processed but has not yet been consolidated into memory by sleep. As this hypothetical limited-capacity mechanism becomes saturated with task-specific information, the local neural network's ability to process on-line information during task performance worsens, resulting in the performance decrement. Several findings support this relationship between information processing and the performance decrement. First, the retinotopic specificity of the performance decrement is consistent with previous studies showing that TDT learning is similarly retinotopic[19] and dependent on early stages of visual processing[19,30]. Second, post-training sleep is known to be critical for stabilization and consolidation of TDT learning[9,14,15] and we now have shown that a 60-minute nap reverses the performance decrement.

Although we cannot exclude a function for REM in this process, it seems most likely that SWS has the central role. Although both SWS and REM have previously been implicated in the nocturnal, sleep-dependent consolidation of this task[9,14], the REM-dependent period appears four hours after the SWS-dependent period has ended[14]—well beyond the timeframe of these naps. We posit that during SWS, mechanisms of cortical plasticity lead to secondary changes in the TDT-trained neural networks. Thus SWS serves as the initial processing stage of experience-dependent, long-term learning and as the critical stage for restoring perceptual performance. Roles for SWS in memory consolidation have been proposed by others[9,20,23–25].

This example of a training-induced deterioration in performance has several additional implications. First, it indicates that the cognitive benefits of sleep can be studied over a very short time period and do not require sleep deprivation or overnight sessions of sleep. This provides a more favorable set of conditions to study the involvement of sleep in information processing and performance. Second, it suggests that the psychological sensation of 'burnout,' described anecdotally as increased irritation and frustration along with decreased effectiveness after prolonged cognitive effort, may not reflect a general mental fatigue, but rather the specific need of an overused local neural network to enjoy the restorative benefits of sleep.

Methods

A total of 129 undergraduates gave informed consent to participate in the study, which was approved by the Harvard University Department of Psychology internal review board. All subjects had normal or corrected-to-normal vision and no history of neurological, mental or physical illness. Each subject was tested on the TDT four times in one day: at 9 a.m., 12 p.m., 4 p.m. and 7 p.m. Tests normally lasted 60–75 min and included 1,250 trials. Participants were asked to discriminate the shape of a target in one of the lower quadrants of the display at 2.5–5.0° eccentricity from the center. Either a 'T' or an 'L' appeared in the center, where fixation was maintained. (Except where noted, target arrays always appeared in the lower left quadrant.) The target consisted of a horizontally or vertically oriented array of three diagonal bars against a background of horizontal bars. For each trial, the following sequence was shown: target screen for 16 ms, blank screen for a variable period of time, and then a mask for 16 ms. After each trial, subjects reported both the letter (T or L) at the central fixation point and the orientation of the diagonal bar array (horizontal or vertical). For each session, the speed of perceptual processing was calculated as the threshold target-to-mask ISI needed to achieve 80% accuracy. The ISI threshold provides a measure of the minimal effective stimulation needed for perception of the target. This task was carried out as previously described[14,19]. To examine the amount of change from baseline, difference scores were calculated by subtracting the threshold for the first session from those of the second, third and fourth. Subjects in all groups kept sleep logs for the week before testing, and no significant between-group differences were found in sleep patterns.

Thirty subjects were randomly assigned to one of the nap conditions or to the previously described control condition, permitting statistical comparisons between all three groups. Naps began at 2 p.m. and were recorded polysomnographically, with standard electroencephalographic (EEG), electro-oculographic (EOG) and electromyographic (EMG) measures. Subjects were allowed to sleep until they had completed either a full half-hour or a full hour of polysomnographically identified[31] sleep and then were woken by the experimenter. Sleep stages were subsequently rescored off-line. On re-scoring, one subject in the short nap group was found to have slept for only 9.5 min, and was excluded from all analyses. Subjects were recorded during naps on two separate days: the TDT test day and a control day ('baseline nap') either one week before or one week after the test day, with the order balanced across subjects. Subjects in the short nap group averaged 29.1 ± 2.9 (mean ± s.d.) min of sleep on the test day; subjects in the long nap group averaged 59.6 ± 5.5 min.

For the quadrant switch study (24 switches; 22 controls), peripheral targets were presented in the lower left or lower right quadrant (balanced across subjects) for the first three test sessions. Then, for the fourth session, the targets were switched to the opposite lower quadrant for the switch group. For the quiet rest condition, nine subjects followed the long nap protocol but were instructed not to sleep during the nap hour. Subjects were blindfolded to prevent visual stimulation and listened to an audio tape of short stories. Each subject's wake state was monitored with the Nightcap[32] (HealthDyne Technologies, Marietta, Georgia, USA) and subjects were alerted at the first indication of impending sleep. At the start of the third session, subjects ($n = 10$) in the motivation protocol were offered a bonus of $25 if they could regain their initial performance level during the next two sessions. Subjects ($n = 12$) in the fixed ISI protocol performed the standard protocol but with the ISI set to 400 ms for all 25 blocks during test sessions 1–3. For the fourth session, subjects followed the standard protocol with decreasing ISIs across the 25 blocks. All subjects rated their sleepiness using the Stanford sleepiness scale[33], which rates subjective sleepiness on a seven-point scale.

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Competing interests statement

The authors declare that they have no competing financial interests.

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