Published in final edited form as:
Brain Res Bull. 2008 August 15; 76(6): 605–611.

Sleep improves the variability of motor performance

Sean Hill[1], **Giulio Tononi**[2], and **M. Felice Ghilardi**[3]

[1] IBM T.J. Watson Research Center, Yorktown Heights, NY & Brain Mind Institute, EPFL, Lausanne, Switzerland [2] Dept. of Psychiatry, University of Wisconsin, Madison, WI [3] Dept. of Physiology & Pharmacology, CUNY Medical School, New York, NY

Abstract

Sleep after learning often enhances task performance, but the underlying mechanisms remain unclear. Using a well-characterized rotation learning paradigm implemented both behaviorally and in computer simulations, we compared two main hypotheses: the first, that off-line replay during sleep leads to further potentiation of synaptic circuits involved in learning; the second, that sleep enhances performance by uniformly downscaling synaptic strength. A simple computer model implemented synaptic changes associated with rotation adaptation (30°), yielding a reduction in mean directional error. Simulating further synaptic potentiation led to a further reduction of mean directional error, but not of directional variability. By contrast, simulating sleep-dependent synaptic renormalization by scaling down all synaptic weights by 15% decreased both mean directional error and variability. Two groups of subjects were tested after either two rotation adaptation training sessions or after a single training session followed by sleep. After two training sessions, mean direction error decreased, but directional variability remained high. However, subjects who slept after a single training session showed a reduction in both directional error and variability, consistent with a downscaling mechanism during sleep.

Keywords

consolidation; performance enhancement; directional error; reaching movements; visuo-motor adaptation; human; modeling

Introduction

There is growing evidence that sleep after learning can enhance performance [1–3,5,12–15, 20,22,23,26,29,32]. Several studies, using both implicit and explicit learning paradigms, have shown that performance can improve after a night of sleep, or even after a nap, but not after an equivalent period of wakefulness.

The mechanisms by which sleep may lead to performance enhancements are presently unknown. One plausible idea is that neural activity during training potentiates local synaptic

---

Corresponding author: M. Felice Ghilardi, Department of Physiology & Pharmacology, Harris Hall H-210, CUNY Medical School, 160 Convent Avenue, New York, NY, 10031; Telephone: 212 650 5814; FAX: 212 650 7726, e-mail: lice.mg79@gmail.com.

There is no conflict of interest.

circuits during wakefulness, that similar patterns of neural activity are replayed during sleep, and that off-line replay may lead to a further potentiation of synaptic circuits [1,31,33,37].

An alternative possibility is that sleep may enhance performance by uniformly downscaling or renormalizing synaptic strength [34,35]. This mechanism would not produce further learning, but merely an increase in signal-to-noise ratios because local synapses unrelated to the newly learned task are reduced to sub-threshold strength and cease to influence performance.

To compare these two these general mechanisms, we took advantage of a rotation adaptation paradigm that shows sleep-dependent performance enhancement [12]. This motor task is a form of implicit learning that is well-characterized kinematically and from a neuroimaging perspective [9,16–19]. In addition, this task permits accurate parameterization of both performance improvement and noise reduction. As subjects gradually adapt their movements to a systematic rotation imposed to the perceived cursor trajectory, the directional error of movement trajectories as well as their variance are progressively reduced.

Here we first introduce a schematic computer model of rotation learning in which both mechanisms can be easily implemented and compared, leading to differential predictions. In the simulations, after a training session modeled as synaptic potentiation at an offset corresponding to the imposed rotation (30°), the test block showed, as expected, a reduction in mean direction error. To simulate the occurrence of further synaptic potentiation, whether occurring on-line during wakefulness or off-line during sleep, synaptic strength was further increased at the same offset. This led to further reduction of mean directional error, but directional variability did not improve. By contrast, to simulate sleep-dependent synaptic renormalization, all synaptic weights after the first training session were scaled down by the same fraction (e.g. 15%). After downscaling, the test block showed a decrease in both mean directional error and variability.

To test the predictions obtained from the model, we turned to behavioral experiments. One group of subjects underwent a first training session during the day. After a short interval, they underwent a second, additional training session followed by a testing session. A second group of subjects went to sleep after the first training session, and then underwent a testing session without further training. Performance tests showed that after a second training session during the day, mean direction error had decreased, but directional variability was still high. By contrast, subjects who had slept after the first training session showed both a reduction in mean directional error and in variability, consistent with a downscaling mechanism during sleep.

Material and Methods

1. Neural network model

The model describes at the level of individual neuron elements, a visuomotor mapping from 2D visual space to 2D motor output space (Figure 1). The model consists of 360 neuron-like elements in each of two layers: 1. a visual input layer (V) and 2. a motor output layer (M). Each element in V represents 1° of 360° of visual space in hand-centered coordinates, while each element in M represents 1° of 360° possible hand movement directions. A visual target presentation activates elements in V which project to elements in M, thereby signaling a motor output direction.

Network elements—Each element in V is activated by the presence of a target in the corresponding portion of the visual field, and projects with normalized weighted connections to a subset of elements in M. The probability of firing [36] for an individual motor cell is determined by the suprathreshold incoming synaptic weight as given by the following equation:

$$P_{firing}=\sum_{i}(w_{ij}x_i-\theta)/n \tag{1}$$

where n is a normalization factor equivalent to the maximum suprathreshold input for all motor units. This normalization assumes that motor units respond with the same probability of firing to an equal amount of synaptic input.

Connections: Default mapping—The map from visual inputs to motor outputs is defined by the weights of the connections from V to M. The default mapping of the weight w from an element i in V to an element j in M is given by the following Gaussian:

$$w_{ij}=c_{\alpha}\mathrm{e}^{-(\mathrm{j}-\frac{\mathrm{x}_{\alpha}}{2})^2/\sigma_{\alpha}} \tag{2}$$

which is centered at x_{α} with a maximal strength given by $c_{\alpha}$and a width σ_{a}.

Network output—The output movement direction is determined by taking the mean of the movement angle represented by the active units in M during a given trial. With this population coding scheme, the activity of the output units in M can code for a motor output in any direction.

Learning the visuomotor shift—Learning in the model is implemented as the addition of synaptic weight to the connections between V and M. The added weights follow a Gaussian profile centered at x_{β}, with independent parameters c_{β} and σ_{β} governing the peak and width of the added weights, respectively. Therefore, the distribution of weights after learning is given as

$$\omega_{ij}=c_{\alpha}\mathrm{e}^{-(\mathrm{j}-\frac{\mathrm{x}_{\alpha}}{2})^2/\sigma_{\alpha}}+c_{\beta}\mathrm{e}^{-(\mathrm{j}-\frac{\mathrm{x}_{\beta}}{2})^2/\sigma_{\beta}}. \tag{3}$$

Before the visuomotor shift x_{α}=0°, and x_{β} =0° and after the visuomotor shift x_{α} =0°, and x_{β} =30°. The indices i and j each cover the range 0–360°.

Measuring performance—Performance of the model was evaluated by simulating 88 trials of 8 movements each. The directional error X was determined by the difference between the output direction and the target direction. The mean and variance of the directional error were computed per trial and then averaged over all trials. These measures were used to evaluate the performance of the model network. To assess performance enhancement, we compared the two learning measures, mean directional errors and variances, between groups *(Sleep and Downscaling*) and testing times (*Test 1 and Test 2*) with one-way ANOVA.

2. Behavioral experiment

Subjects—24 right-handed subjects (13 men, 11 women, aged 25–36 years) participated in the study. All were naïve to the purpose of the experiments and signed a consent form. Subjects were randomly assigned to one of two groups. One group (*Extra-Training*, n=12) was tested in a single day. The other one (*Sleep*, n=12) was tested over two successive days.

Experimental procedure—As in previous experiments [9,16], subjects sat facing a computer monitor, held a screen cursor with their right hand and moved it on the surface of a digitizing tablet. An opaque shield prevented subjects from seeing their hand or arm. Targets were eight radially arrayed circles at 4.2 cm from a starting point, separated by 45° and displayed on a computer monitor. Targets appeared randomly at a rate of 1/s. Subjects were instructed to make straight out-and-back movements reversing direction within the target and to move as fast as possible without making in-flight corrections. Cursor location was visible on the screen at all times. Targets were presented in blocks of 11 cycles of 8 targets resulting in 88 movements over 90 seconds. Subjects were given 30 seconds to rest between two consecutive blocks.

All subjects performed a first training session in the morning. This consisted in a baseline block of movements at 0° rotation, two *training* blocks during which the direction of the screen cursor was rotated 30° counterclockwise, and one *testing* block at 30°. Fifteen minutes after the first session, subjects in the *Extra-Training* group underwent a second training block with 30° rotation, followed by a testing block. Subjects in the *Sleep* group underwent a testing block 24 hours after the first training session, without further training.

Data analysis—Movement onsets, peak velocity and reversal points were calculated as reported previously [9]. The directional error for each movement was taken as the difference between the direction of the target and the direction of the hand at the peak outward velocity from the initial hand position [9].

To estimate the degree of learning achieved, we computed the mean directional error for each testing block of the first and second sessions. For each testing blocks, we also computed the mean variance of directional error, as the average of the variances around the error mean for each of the eight targets. Thus, this measure of variable error represents the variability in directional error after the systematic and direction-dependent variance has been accounted for [7].

To assess performance enhancement, we compared the two learning measures, mean directional errors and variances, between groups *(Sleep and Extra-Training* Groups) and testing times (*First and Second* sessions) with mixed model ANOVA and post-hoc tests (Bonferroni-Dunn). Differences were considered significant at a value of $p< 0.05$ with the appropriate correction for multiple comparisons.

Results

The model performance was tested in the baseline condition (0° shift) by presenting a visual stimulus at each of the 360° of visual space (Figure 2). The mean directional error of the motor output in the baseline condition was 0.05±0.3° and the variance was 18.2±1.6°. For each condition the number of trials = 88.

Simulated learning produces a decrease in directional error and an increase in variability

Learning was simulated by the addition of synaptic weight (c_β =0.5; σ_β =10) to the connections from visual units to motor output cells with a preferred direction shifted by 30° (Figure 2). When the 30° shift was introduced, the directional error of the motor output shifted by 30° to 29.9±0.3° with variability of 16.8±1.4°. After learning, the mean directional error improved to 5.9±0.9° but with an increase in the variance of directional error to 69.2±13.3° (one-way ANOVA; F=5072.6; p<0.001).

Simulating extra learning produces an improvement in the mean directional error but no improvement in the variability

To simulate the effects of additional learning, either due to a second training session or to off-line replay followed by synaptic potentiation, additional synaptic weight (c_β =0.5; σ_β =10) was added to the same offset location (x_β). This doubled the amount of synaptic weight due to learning while the mean directional error decreased by 28.4% to 4.2±.9° (F=14.35, p<0.001) while the variability did not change significantly (69.2±13.3° vs. 68.2±12.1°; F=211.0; p=0.77).

Simulating downscaling produces a significant reduction in the variability of the directional error

To test the hypothesis that downscaling can improve performance, the individual weights of all connections from the visual to the motor map were scaled down by 15% of their value (Figure 2). The immediate effect of downscaling was to reduce the postsynaptic input to the motor output cells by 15%. This caused a large fraction of the synaptic input to fall below the firing threshold. The remaining suprathreshold input was now strongest to motor output units with the greatest total synaptic weight, which in this case were those representing the direction of the recently learned shift. The result was that the mean directional error fell by 34.8% to 3.8 ±.6° (F=32.6; p<0.001) while the variance decreased 60% to 26.6±3.5° (F=67.3; p<0.001).

The model thus predicts that downscaling improves performance significantly, while additional learning has a relatively minor impact on the mean and variability of the directional error. The greatest improvement produced by downscaling is seen primarily in the reduction of variance of directional error accompanied by a significant improvement in the mean error. Results shown reflect a downscaling factor of 15 %, which is in the range observed in recent in vivo experiments [36]. In the model, varying the specific value of the downscaling factor consistently produces an improvement in performance, unless downscaling is so large that synaptic input falls below firing threshold. In vivo, such a scenario is unlikely: downscaling is thought to be effected by sleep slow waves, which are in turn sensitive to net synaptic strength. Therefore, while synaptic strength reaches physiological baseline levels, as would be the case at the end of the night, slow waves are small enough that any further downscaling would cease, thereby implementing a safe, self-limiting process [34,35].

2. Behavioral experiments

In the baseline “no-rotation” blocks, the means and variances of directional errors were the same in the two groups (Figure 3, p=0.44).

By the end of the first training session with an imposed 30° rotation, directional error had decreased to 7.1°±2.6° (all subjects combined), with no significant difference between the two groups (*Sleep*: 6.9°± 2.6°; *Extra-Training*: 7.5°±2.6°, F(1,44)=0.94, p=0.9). In the testing block of the first session, the mean and variance of directional errors were similar in the two groups (Figure 3, mean: F(1,22)=0.8, p=0.4; variance: F(1,22)=0.17, p=0.7).

In the testing block of the second session, the mean of directional errors decreased further, compared to the first session testing block, in both groups (Figure 3, effect of session: F(1,44) =9.97, p=0.003). Although the improvement was more evident in the *Sleep* group (31.5% versus 23.1%), there was not statistical difference between the two groups (Figure 3, effect of group: F(1,44)=2.7, p>0.1). On the other hand, the variance of the directional error decreased significantly from the first to the second session (F(1,44)=5.48, p=0.02) only in the *Sleep* group (Figure 3, *Sleep* group: F(1,22)=10.44, p=0.001; *Extra-Training* group: F(1,22)=0.48, p=0.5, decrease of 43.3% versus 8.6%). Thus, in the second session, the variance of the *Sleep* group at test reached the value range of the baseline “no-rotation” block (p>0.05) and was significantly lower than that of the *Extra-Training* group (p=0.001).

Discussion

Two competing frameworks have been proposed to account for the improvements in performance that are frequently observed after sleep. The *reactivation-consolidation hypothesis* suggests, in its most schematic version, that off-line replay during sleep of neuronal firing patterns occurring during learning leads to a further potentiation of memory traces, which would explain the improvement after sleep [1]. This hypothesis is supported by evidence for off-line reactivation of neurons and brain areas involved in the initial learning, obtained using both multi-unit recordings in animals and neuroimaging in humans. It should be noted that direct evidence for synaptic potentiation during sleep is currently lacking, but it is assumed that it may occur due to changes in calcium dynamics [4]. It is possible that synaptic potentiation during sleep may also lead to a migration of memory traces to a set of regions different from the ones initially involved during the learning task [1,15,21,26–28].

An alternative hypothesis – the *synaptic homeostasis hypothesis* - suggests that during sleep, synapses would be renormalized or downscaled, meaning that their efficacy or weight would decrease by a similar percentage [34,35]. The main purposes of downscaling would be to counteract the increased costs in terms of energy, space, cellular supplies, and saturation in the ability to learn, which would be caused by a net increase in synaptic strength during learning. Under certain circumstances, as exemplified by rotation learning tasks, downscaling might also result in improved performance due to an increase in signal-to-noise ratios. Evidence for this hypothesis comes from molecular and electrophysiological studies suggesting a net potentiation of synapses in the cerebral cortex of animals who had been mostly awake, and a net depression in the cortex of animals who had been asleep [36]. Additional evidence comes from studies demonstrating that rotation learning, as well as manipulations promoting synaptic potentiation, result in a local increase in slow wave activity during sleep, presumably due to increased local synchronization caused by synaptic strengthening [12].

In this work, we have attempted to compare the alternative accounts of sleep-dependent performance enhancements offered by the reactivation-consolidation hypothesis and the synaptic homeostasis hypothesis. Sleep-dependent performance improvements have been reported with various kinds of learning tasks [1,12–15,20,22,23,26,29,32] and have been ascribed to different sleep stages or to different aspects of brain activity during sleep. For the present purposes we chose rotation learning because it is a well-characterized task, easy to parametrize, and it is mostly implicit, not requiring any cognitive strategy: indeed, for rotation up to 30° subjects do not report any awareness either of the rotation or how the learning occurs. The imposition of a visuomotor rotation causes a mismatch in the alignment of the visually- and proprioceptively-perceived hand positions. Learning, in this case, is a gradual process involving the formation of new visuomotor maps and of new internal models [6,8,10,11]. Classic studies of motor and procedural learning predict that, initially, subjects should adapt to the imposed perturbation at the expense of a large increase in variability. Later on learning asymptotes and variability returns within the normal range. Here we focused on the variability associated with learning new visual-proprioceptive-motor mappings, excluding from consideration a component dependent on target direction that is related to limb dynamics [30].

The present results confirm that, after the first learning session, directional error improves but response variability is high. Later, if subjects are allowed to sleep, performance improves further and variability returns to the normal range. Instead, an additional learning session does not improve variability. Thus, these results support the notion that sleep is important for achieving skilled, low-variability performance, an effect that is not achieved through additional training sessions. Of course, we cannot discard the possibility that spaced training or more intensive training occurring twelve hour later, might produce a decrease in performance variability similar to the one observed after sleep. It has been also reported that repeated training, without intervening sleep, induces a worsening in a visual discrimination learning task [23–25]. By contrast, in our motor task, the extra-training session did not induce significant deterioration in either mean performance or its variability.

The results obtained with a simplified model of rotation adaptation suggest that synaptic downscaling, as postulated by the synaptic homeostasis hypothesis, can account in a parsimonious manner for the changes in directional error and variability observed in behavioral experiments. It may at first appear counterintuitive that a proportional reduction in synaptic strength should lead to better performance. However, the simulations indicate that synaptic downscaling, by reducing postsynaptic input, decreases the variability of postsynaptic firing, in that output units with the greatest pre-downscaling synaptic input become the only ones to fire post-downscaling. Assuming that output units represent movement vectors, the result is that movement variability returns within the baseline range, just as observed in behavioral experiments. Interestingly, imaging experiments in humans show that during initial learning brain activation is at first diffuse and bilateral [18]; in expert learners, instead, activity is confined to more restricted foci of activation in parietal regions [9], consistent with increased signal-to-noise ratios. Thus, our simple model illustrates how the combination of synaptic downscaling with a simple threshold mechanism can produce significant improvements in the signal (mean direction) to noise (variability of mean direction) ratio.

By contrast, our simulations suggest that further strengthening of circuits involved in learning rotation adaptation, as might occur due to additional training or through off-line replay, can lead to an improved directional error but not to a substantial reduction of response variability. To the extent that an additional learning session during wakefulness does indeed produce a further strengthening of the relevant circuits, the predictions of the model are confirmed, in that response variance did not improve significantly. Altogether, the behavioral results conform to the simulated results when these implemented synaptic downscaling and not when they implemented additional synaptic strengthening

Of course, caution is needed to interpret the present findings and to extend these conclusions to other categories of learning. Our computer model of rotation learning is purposely as simple as possible, and we have assumed that synaptic potentiation is a homogeneous process. It is possible that sleep may be associated with synaptic potentiation, but through mechanisms that may differ considerably from those associated with learning during wakefulness. For example, due to changes in the level of certain neuromodulators, or due to the frequent hyperpolarization of cortical cells during slow wave sleep, synaptic potentation could be restricted to a much smaller set of connections, thereby possibly reducing variability. Alternatively, potentiation may occur in circuits downstream of those involved in waking plasticity, potentially leading to different behavioral outcomes. Also, synaptic potentiation and downscaling might occur together during sleep: the strongest synapses could be potentiated further, while weaker synapses would go through a process of downscaling, yielding an even larger decrease in performance variability. More studies and direct cellular evidence are obviously needed to define the precise mechanisms responsible for the reduction of variability after sleep. Moreover, it remains to be seen to what extent sleep improves performance variability in other learning tasks: for instance, implicit and explicit learning tasks may show different patterns of improvements and sleep-dependence. Nevertheless, precisely because our model is simple and makes minimal assumptions, it provides a starting point for developing more elaborate accounts of sleep-dependent improvements in performance.

Acknowledgements

supported by NIH grant NIH R01 NS054864 (MFG); NIH R01 NS0 (GT); McDonnell foundation grant (GT, MFG), Pioneer grant (GT). We thank Dr. Clara Moisello for helpful comments.

References

1. Born J, Rasch B, Gais S. Sleep to remember. Neuroscientist 2006;12:410–424. [PubMed: 16957003]
2. Cipolli C, Fagioli I, Mazzetti M, Tuozzi G. Consolidation effect of the processing of declarative knowledge during human sleep: evidence from long-term retention of interrelated contents of mental sleep experiences. Brain Res Bull 2005;65:97–104. [PubMed: 15763174]

3. Cipolli C, Fagioli I, Mazzetti M, Tuozzi G. Consolidation effect of repeated processing of declarative knowledge in mental experiences during human sleep. Brain Res Bull 2006;69:501–511. [PubMed: 16647579]
4. Destexhe A, Sejnowski TJ. Interactions between membrane conductances underlying thalamocortical slow-wave oscillations. Physiol Rev 2003;83:1401–1453. [PubMed: 14506309]
5. Ferrara M, Iaria G, De Gennaro L, Guariglia C, Curcio G, Tempesta D, Bertini M. The role of sleep in the consolidation of route learning in humans: a behavioural study. Brain Res Bull 2006;71:4–9. [PubMed: 17113921]
6. Ghez C, Gordon J, Ghilardi MF, Christakos CN, Cooper SE. Roles of proprioceptive input in the programming of arm trajectories. Cold Spring Harb Symp Quant Biol 1990;55:837–847. [PubMed: 2132861]
7. Ghez C, Gordon J, Ghilardi MF. Impairments of reaching movements in patients without proprioception. II. Effects of visual information on accuracy. J Neurophysiol 1995;73:361–372. [PubMed: 7714578]
8. Ghilardi MF, Gordon J, Ghez C. Learning a visuomotor transformation in a local area of work space produces directional biases in other areas. J Neurophysiol 1995;73:2535–2539. [PubMed: 7666158]
9. Ghilardi MF, Ghez C, Dhawan V, Moeller J, Mentis M, Nakamura T, Antonini A, Eidelberg D. Patterns of regional brain activation associated with different forms of motor learning. Brain Res 2000;871:127–145. [PubMed: 10882792]
10. Gordon J, Ghilardi MF, Cooper SE, Ghez C. Accuracy of planar reaching movements. II. Systematic extent errors resulting from inertial anisotropy. Exp Brain Res 1994;99:112–130. [PubMed: 7925785]
11. Gordon J, Ghilardi MF, Ghez C. Accuracy of planar reaching movements. I. Independence of direction and extent variability. Exp Brain Res 1994;99:97–111. [PubMed: 7925800]
12. Huber R, Ghilardi MF, Massimini M, Tononi G. Local sleep and learning. Nature 2004;430:78–81. [PubMed: 15184907]
13. Karni A, Tanne D, Rubenstein BS, Askenasy JJ, Sagi D. Dependence on REM sleep of overnight improvement of a perceptual skill. Science 1994;265:679–682. [PubMed: 8036518]
14. Karni A. When practice makes perfect. Lancet 1995;345:395. [PubMed: 7845149]
15. Korman M, Doyon J, Doljansky J, Carrier J, Dagan Y, Karni A. Daytime sleep condenses the time course of motor memory consolidation. Nat Neurosci 2007;10:1206–1213. [PubMed: 17694051]
16. Krakauer JW, Ghilardi MF, Ghez C. Independent learning of internal models for kinematic and dynamic control of reaching. Nat Neurosci 1999;2:1026–1031. [PubMed: 10526344]
17. Krakauer JW, Pine ZM, Ghilardi MF, Ghez C. Learning of visuomotor transformations for vectorial planning of reaching trajectories. J Neurosci 2000;20:8916–8924. [PubMed: 11102502]
18. Krakauer JW, Ghilardi MF, Mentis M, Barnes A, Veytsman M, Eidelberg D, Ghez C. Differential cortical and subcortical activations in learning rotations and gains for reaching: a PET study. J Neurophysiol 2004;91:924–933. [PubMed: 14523069]
19. Krakauer JW, Ghez C, Ghilardi MF. Adaptation to visuomotor transformations: consolidation, interference, and forgetting. J Neurosci 2005;25:473–478. [PubMed: 15647491]
20. Laureys S, Peigneux P, Perrin F, Maquet P. Sleep and motor skill learning. Neuron 2002;35:5–7. [PubMed: 12123601]
21. Maquet P, Peigneux P, Laureys S, Boly M, Dang-Vu T, Desseilles M, Cleeremans A. Memory processing during human sleep as assessed by functional neuroimaging. Rev Neurol (Paris) 2003;159:6S27–29. [PubMed: 14646796]
22. Maquet P, Schwartz S, Passingham R, Frith C. Sleep-related consolidation of a visuomotor skill: brain mechanisms as assessed by functional magnetic resonance imaging. J Neurosci 2003;23:1432–1440. [PubMed: 12598632]
23. Mednick S, Nakayama K, Stickgold R. Sleep-dependent learning: a nap is as good as a night. Nat Neurosci 2003;6:697–698. [PubMed: 12819785]
24. Mednick SC, Nakayama K, Cantero JL, Atienza M, Levin AA, Pathak N, Stickgold R. The restorative effect of naps on perceptual deterioration. Nat Neurosci 2002;5:677–681. [PubMed: 12032542]
25. Mednick SC, Arman AC, Boynton GM. The time course and specificity of perceptual deterioration. Proc Natl Acad Sci U S A 2005;102:3881–3885. [PubMed: 15731350]

26. Orban P, Rauchs G, Balteau E, Degueldre C, Luxen A, Maquet P, Peigneux P. Sleep after spatial learning promotes covert reorganization of brain activity. Proc Natl Acad Sci U S A 2006;103:7124–7129. [PubMed: 16636288]
27. Peigneux P, Laureys S, Fuchs S, Destrebecqz A, Collette F, Delbeuck X, Phillips C, Aerts J, Del Fiore G, Degueldre C, Luxen A, Cleeremans A, Maquet P. Learned material content and acquisition level modulate cerebral reactivation during posttraining rapid-eye-movements sleep. Neuroimage 2003;20:125–134. [PubMed: 14527575]
28. Peigneux P, Orban P, Balteau E, Degueldre C, Luxen A, Laureys S, Maquet P. Offline persistence of memory-related cerebral activity during active wakefulness. PLoS Biol 2006;4:e100. [PubMed: 16602824]
29. Roth DA, Kishon-Rabin L, Hildesheimer M, Karni A. A latent consolidation phase in auditory identification learning: time in the awake state is sufficient. Learn Mem 2005;12:159–164. [PubMed: 15805314]
30. Sainburg RL, Ghilardi MF, Poizner H, Ghez C. Control of limb dynamics in normal subjects and patients without proprioception. J Neurophysiol 1995;73:820–835. [PubMed: 7760137]
31. Sejnowski TJ, Destexhe A. Why do we sleep? Brain Res 2000;886:208–223. [PubMed: 11119697]
32. Smith C, MacNeill C. Impaired motor memory for a pursuit rotor task following Stage 2 sleep loss in college students. J Sleep Res 1994;3:206–213. [PubMed: 10607127]
33. Tononi G, Cirelli C. Some considerations on sleep and neural plasticity. Arch Ital Biol 2001;139:221–241. [PubMed: 11330203]
34. Tononi G, Cirelli C. Sleep and synaptic homeostasis: a hypothesis. Brain Res Bull 2003;62:143–150. [PubMed: 14638388]
35. Tononi G, Cirelli C. Sleep function and synaptic homeostasis. Sleep Med Rev 2006;10:49–62. [PubMed: 16376591]
36. Vyazovskiy VV, Cirelli C, Pfister-Genskow M, Faraguna U, Tononi G. Molecular and electrophysiological evidence for net synaptic potentiation in wake and depression in sleep. Nat Neurosci. in press
37. Walker MP. A refined model of sleep and the time course of memory formation. Behav Brain Sci 2005;28:51–64. [PubMed: 16047457]discussion 64–104

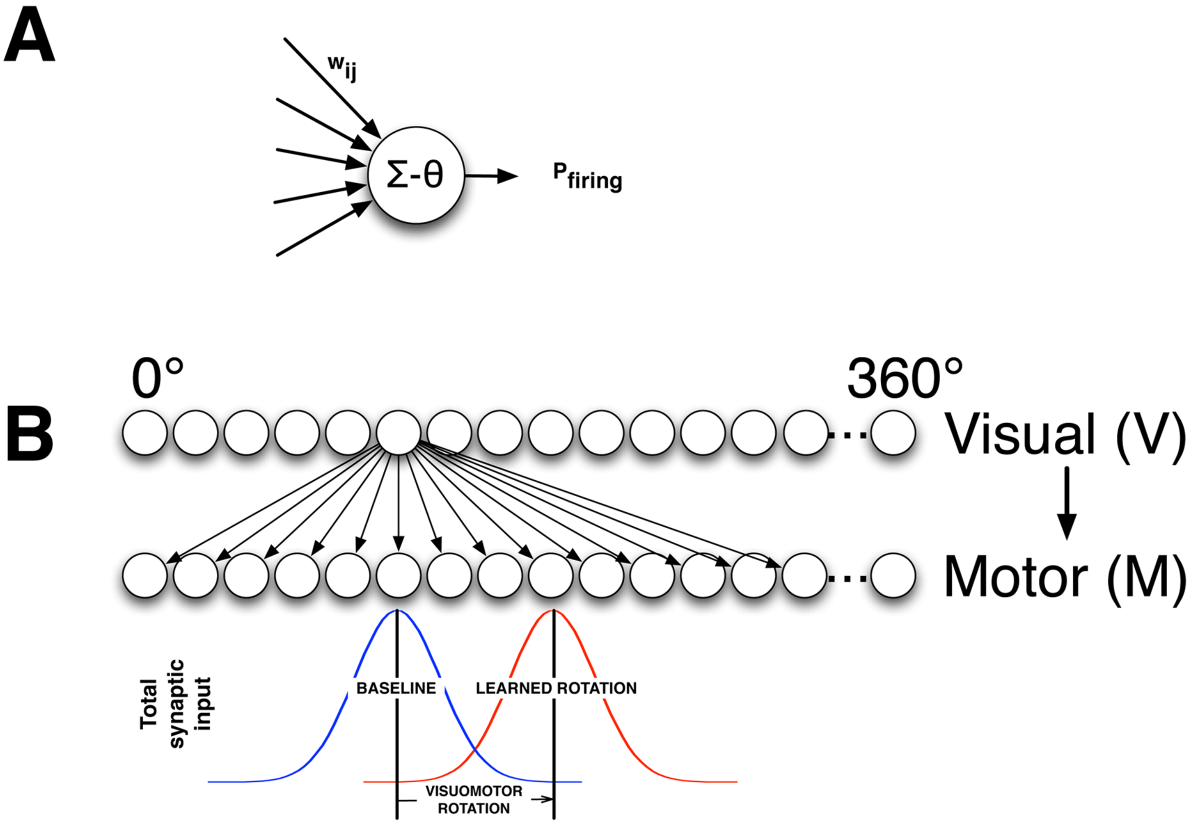

Figure 1.

Model of rotation adaptation. A. Individual neuron-like network elements sum synaptic inputs to determine the probability of firing. B. The model contains a Visual input layer (V) with network elements representing a 360° hand-centric visual coordinate space and a Motor output layer (M) with neurons representing 360° of output movement directions. Weighted connections from V innervate M to determine the output movement direction given a particular visual stimulus in V. Visuomotor learning is simulated by adding synaptic weight to these connections.

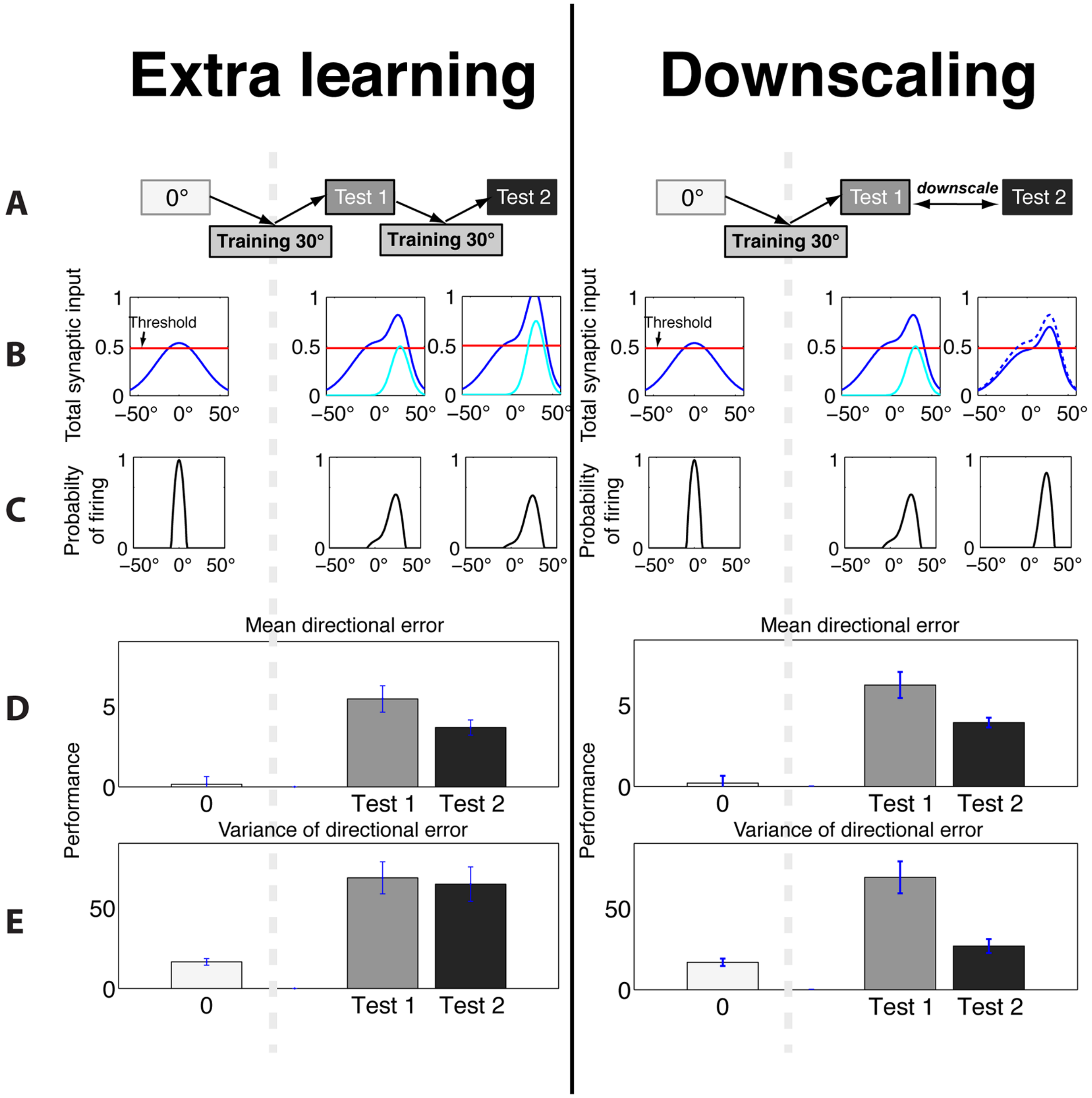

Figure 2.

Comparison of model performance with extra learning and downscaling. A. The learning and testing process starts at 0° rotational shift, training then occurs with a 30° shift in the visuomotor mapping at which time Test 1 occurs. In the case of extra learning, Test 2 occurs after an additional training period at 30°. In the case of downscaling, Test 2 occurs after synaptic weights are scaled down by 15% (downscaling). B. Total synaptic input to M (from V) is shown. The red line indicates the threshold above which the model neuron may fire. During training additional synaptic weight is introduced (indicated by the cyan line) resulting in a change in the distribution of synaptic input (dark blue line). In the case of extra learning, there is a further increase in synaptic input. In the case of downscaling, the synaptic input is scaled down and a portion of the synaptic input is reduced to below firing threshold. C. The probability of firing for network elements in M when visual input is presented at different locations in V. In the case of additional extra learning, the probability distribution widens slightly. In the case of downscaling, the probability distribution narrows, sharpening the newly learned target and removing the trace of the original target location. D. The mean directional error improves after both extra learning and downscaling. E. The variance of the directional error does not decrease significantly with extra learning, while it is significantly reduced after downscaling.

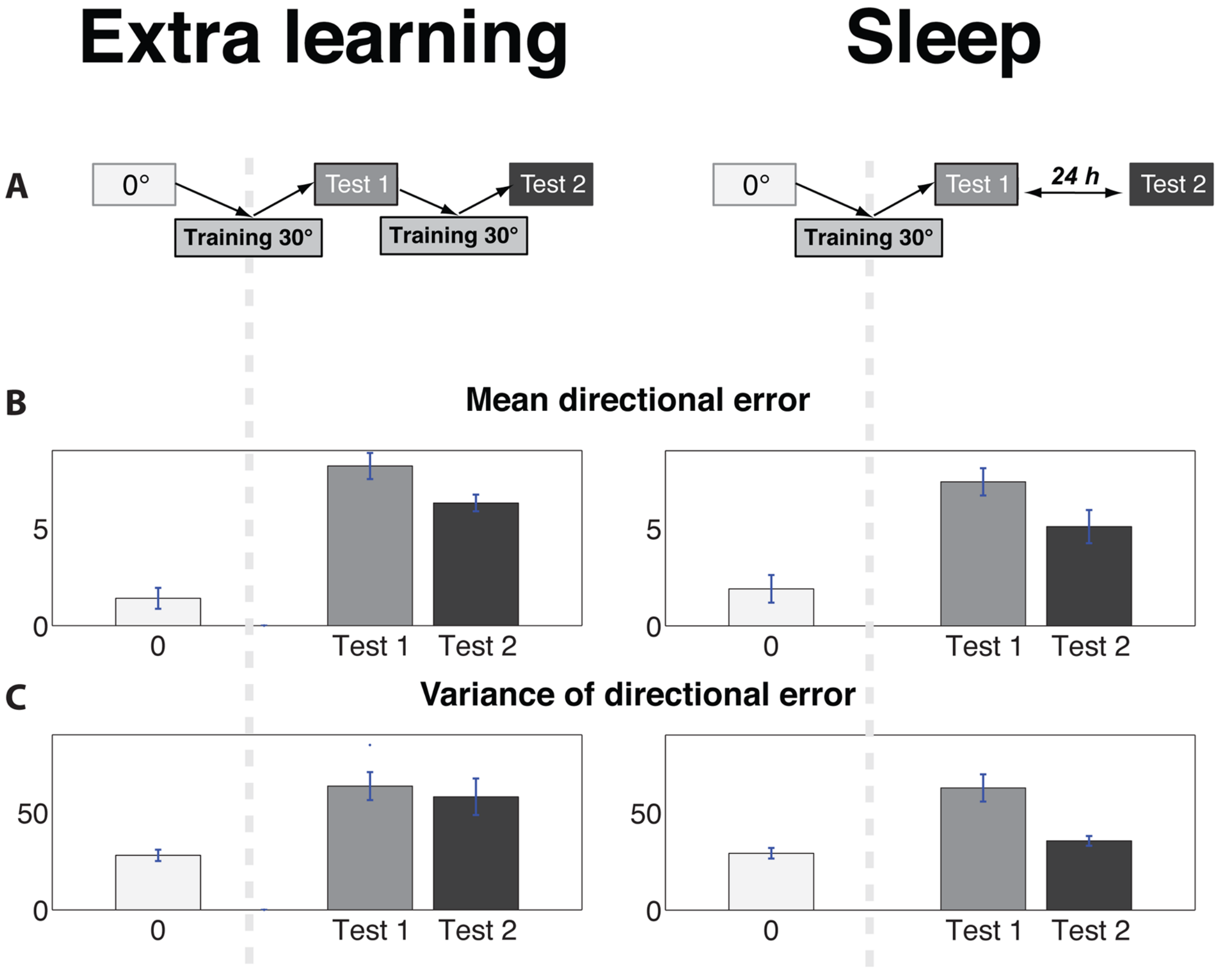

Figure 3.

Behavioral experiment. A. Two groups of subjects adapted to a rotated display and were tested either after extra learning or after sleep. B. The mean directional error improves after both extra learning and sleep. E. The variance of the directional error does not decrease significantly with extra learning, while it is significantly reduced after sleep.

Table 1

Model parameter values

Parameter	Value	Parameter	Value
σ_α	40	σ_β	15
c_α	0.535	c_β	0.5
θ	0.5		