

We have demonstrated consolidation during sleep for complex, generalized skill acquisition. Previous research has shown that sleep affects perceptual^{4,7,8} and motor learning¹⁰, in tasks limited to specific patterns or spatial locations. In the present study, participants learned a new mapping from complex acoustical patterns to pre-existing linguistic categories, which generalized to new stimuli^{14,15}. This behaviour involves distinct processes¹⁷, the formation of specific memories associated with the learned words (episodic, declarative representations), and the establishment of a mapping defined over the set of learned words that supports generalization to new utterances (procedural learning). When participants are given equal amounts of training across days with a small set of repeated words or with entirely novel words, different patterns of learning are seen. Training on a set of repeated words produces near-perfect performance on those words, but very poor generalization to novel test items. Training on all novel items produces a much larger generalization effect on the post-test¹⁵. The generalization effect cannot be accounted for by memorizing acoustic patterns of phonemes, because different acoustic patterns may represent the same phoneme and the same pattern may represent different phonemes¹⁸, depending on context. These context-conditioned effects even span syllable boundaries¹⁹ and are used in perception²⁰. The acoustic patterns of phonemes in different phonetic contexts cannot be statistically inferred from the distribution of a sample of those utterances²¹, and rote memorization and linear interpolation across a small set of acoustic patterns for each consonant and vowel cannot explain human speech recognition²². To recognize new words, listeners must learn to generalize, predicting the acoustic consequences of different phonetic contexts.

Sleep has at least two separate effects on learning. Sleep consolidates memories, protecting them against subsequent interference or decay. Sleep also appears to 'recover' or restore memories. In the perceptual learning task we used, memories are sufficiently robust to last for up to six months¹⁴ or, in a comparable reading-acquisition task, even a year²³. Such robust memory represents a significant selective advantage that an organism might accrue from sleep-mediated processes. Learning can take place at any time during a waking period and any loss due to decay or interference will be restored by sleep. This also implies that a selective advantage of sleep is to enable organisms to learn opportunistically any time during the day without penalty as to robustness of learning.

We do not know if the reduction in performance observed after periods of wakefulness is due to decay of learned material, or to interference from listening to speech or other cognitive processing during the day. If performance is reduced by interference, sleep might strengthen relevant associations and/or weaken irrelevant associations, improving access to relevant memories. If performance is reduced by decay, sleep might actively recover what has been lost, presumably by an interaction between partially retained memories (words) and partially retained mappings that resulted from learning the word set. □

Methods

Participants listened to computer-generated monosyllabic consonant–vowel–consonant (CVC) words taken from a phonetically balanced (PB) list (approximating the distribution of phonemes in English)²⁴, and responded by typing the word. During training, a series of synthetic speech words were presented over headphones paired with the printed form of the word as feedback. After each training block, participants identified the trained words. A pretest and post-test were given before and after training, during which participants identified different sets of words without feedback. The pretest and post-test each consisted of 100 PB words, and the two training sessions each consisted of 150 PB words. The training sessions were structured into three blocks of 50 words. Participants rested between blocks. Word lists for testing and training were counterbalanced across participants. The 84 participants (all groups) were 20.3 ± 2.3 (mean \pm s.d.) years old; each participated in one group only. Stimuli were delivered through Sennheiser HD 570 headphones with an r.m.s. sound pressure level of 66.5 dB.

One group had two post-training periods of testing. In the second post-test, participants were tested with a further set of 100 PB words that no other group was trained or tested on. The consistency of the result for the second post-test argues against any materials effect and further emphasizes the robustness of the findings.

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1. Ficca, G., Lombardo, P., Rossi, L. & Salzarulo, P. Morning recall of verbal material depends on prior sleep organization. *Behav. Brain Res.* **112**, 159–163 (2000).
2. Smith, C. & Rose, G. Posttraining paradoxical sleep in rats is increased after spatial learning in the Morris water maze. *Behav. Neurosci.* **111**, 1197–1204 (1997).
3. Plihal, W. & Born, J. Effects of early and late nocturnal sleep on priming and spatial memory. *Psychophysiol.* **36**, 571–582 (1999).
4. Karni, A., Tanne, D., Rubenstein, B. S., Askenasy, J. J. M. & Sagi, D. Dependence on REM sleep of overnight perceptual skill. *Science* **265**, 679–682 (1994).
5. Karni, A. & Sagi, D. The time course of learning a visual skill. *Nature* **365**, 250–252 (1993).
6. Mednick, S. C. *et al.* The restorative effect of naps on perceptual deterioration. *Nature Neurosci.* **5**, 677–681 (2002).
7. Stickgold, R., James, L. & Hobson, J. A. Visual discrimination learning requires sleep after training. *Nature Neurosci.* **3**, 1237–1238 (2000).
8. Stickgold, R., Whidbee, D., Schirmer, B., Patel, V. & Hobson, J. A. Visual discrimination task improvement: A multi-step process occurring during sleep. *J. Cogn. Neurosci.* **12**, 246–254 (2000).
9. Brashers-Krug, T., Shadmehr, R. & Bizzeti, E. Consolidation in human motor memory. *Nature* **382**, 252–255 (1996).
10. Walker, M. P., Brakefield, T., Morgan, A., Hobson, J. A. & Stickgold, R. Practice with sleep makes perfect: sleep-dependent motor skill learning. *Neuron* **35**, 205–211 (2002).
11. Karni, A. *et al.* The acquisition of skilled motor performance: Fast and slow experience-driven changes in primary motor cortex. *Proc. Natl Acad. Sci. USA* **95**, 861–868 (1998).
12. Karni, A. *et al.* Functional MRI evidence for adult motor cortex plasticity during motor skill learning. *Nature* **377**, 155–158 (1995).
13. Karni, A. & Bertini, G. Learning perceptual skills: behavioral probes into adult cortical plasticity. *Curr. Opin. Neurobiol.* **7**, 530–535 (1997).
14. Schwab, E. C., Nusbaum, H. C. & Pisoni, D. B. Some effects of training on the perception of synthetic speech. *Hum. Factors* **27**, 395–408 (1985).
15. Greenspan, S. L., Nusbaum, H. C. & Pisoni, D. B. Perceptual learning of synthetic speech produced by rule. *J. Exp. Psychol. Learn. Mem. Cogn.* **14**, 421–433 (1988).
16. Johnson, M. P. *et al.* Short-term memory, alertness and performance: a reappraisal of their relationship to body temperature. *J. Sleep Res.* **1**, 24–29 (1992).
17. Gabrieli, J. D. E. Cognitive neuroscience of human memory. *Annu. Rev. Psychol.* **49**, 87–115 (1998).
18. Liberman, A. M., Cooper, F. S., Shankweiler, D. P. & Studdert-Kennedy, M. Perception of the speech code. *Psychol. Rev.* **74**, 431–461 (1967).
19. Ohman, S. E. Coarticulation in VCV utterances: Spectrographic measurements. *J. Acoust. Soc. Am.* **39**, 151–168 (1966).
20. Rudnicky, A. I. & Cole, R. A. Effect of subsequent context on syllable perception. *J. Exp. Psychol. Hum. Percept. Perform.* **4**, 638–647 (1978).
21. Nusbaum, H. C. & Pisoni, D. B. Automatic measurement of speech recognition performance: A comparison of six speaker-dependent recognition devices. *Comp. Speech Lang.* **2**, 87–108 (1987).
22. Pisoni, D. B. in *Handbook of Learning and Cognitive Processes Vol. 6 Linguistic Functions in Cognitive Theory* (ed. Estes, W. K.) 167–233 (Lawrence Erlbaum Assoc., Hillsdale, New Jersey, 1978).
23. Kolers, P. A. & Perkins, D. N. Orientation of letters and errors in their recognition. *Percept. Psychophys.* **5**, 265–269 (1969).
24. Egan, J. P. Articulation testing methods. *Laryngoscope* **58**, 955–991 (1948).

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Dissociable stages of human memory consolidation and reconsolidation

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Historically, the term 'memory consolidation' refers to a process whereby a memory becomes increasingly resistant to interference from competing or disrupting factors with the continued passage of time¹. Recent findings regarding the learning of skilled sensory and motor tasks ('procedural learning') have refined this definition, suggesting that consolidation can be more strictly

determined by time spent in specific brain states such as wake, sleep or certain stages of sleep^{2–8}. There is also renewed interest⁹ in the possibility that recalling or ‘reactivating’ a previously consolidated memory renders it once again fragile and susceptible to interference^{10–12}, therefore requiring periods of reconsolidation^{13–15}. Using a motor skill finger-tapping task, here we provide evidence for at least three different stages of human motor memory processing after initial acquisition. We describe the unique contributions of wake and sleep in the development of different forms of consolidation, and show that waking reactivation can turn a previously consolidated memory back into a labile state requiring subsequent reconsolidation.

We have previously shown that following practice of a specific motor sequence, delayed performance improvements only occur across a night of sleep, while waking periods of 4, 8 or even 12 hours offer no such performance enhancements^{7,8}. In the present work, we studied eight groups of subjects using the same motor skill finger-tapping task (Fig. 1). Subjects in group 1 demonstrated sleep-dependent enhancements in performance speed (Fig. 2a, right) and accuracy (Fig. 2a, left) when tested 24 hours after training, similar to earlier findings^{7,8}. However, when subjects in group 2 were trained on a second motor sequence immediately after the first, interference was seen, so that overnight improvement in accuracy only occurred for the second sequence, and not the first (Fig. 2b). Significant overnight improvement in speed was observed for both sequences.

It was possible that the second interference sequence immediately reversed the initial learning achieved during training on the first sequence. This was not the case, however, as subjects in group 3 showed no loss of speed or accuracy for either the first or second motor sequence when retested immediately after learning the second interference sequence, rather than after 24 hours (Fig. 2c). Thus, the interference effect seen in group 2 appeared to reflect a specific disruption of the subsequent consolidation process.

Although it was clear that 4–12 hours of waking did not enhance behavioural performance⁷, we did not know whether this waking time period could stabilize the motor memory. Subjects in group 4 were therefore trained on the first motor sequence at 10 a.m., but learned the second, interference sequence 6 hours later, at 4 p.m. In contrast to subjects in group 2, who underwent immediate interference training, subjects in group 4 now showed significant additional overnight gains in both speed and accuracy (Fig. 3). Thus it appears that 6 hours of waking can stabilize, but not enhance, this type of motor memory; the enhancement phase of consolidation requires sleep.

To determine the effects of post-training periods in excess of 24 hours, subjects in group 5 were trained only on one motor sequence on day 1 and retested 24 and 48 hours later (Fig. 4a). When retested after 24 hours, normal improvement in speed and accuracy was seen. At 48 hours, both speed and accuracy improved again, although the increase in accuracy was not significant. This is consonant with our previous findings⁸ that showed similar improvement in a group tested after 72 hours. Thus, there is improvement, rather than decay, of motor memory after a second and third post-training night of sleep.

As expected, when subjects in group 6 were trained on the first sequence on day 1, and learned a second sequence, 24 hours later, on day 2, performance for the first sequence suffered no effect of interference, improving significantly by day 3 (Fig. 4b). Therefore, the stabilization seen 6 hours after training is subsequently maintained for at least 24 hours.

But, to our surprise, this stabilization could be reversed. In contrast to group 6, subjects in group 7 (Fig. 4c) briefly rehearsed the first sequence immediately before learning the second, interference sequence on day 2, demonstrating improvement in both speed and accuracy. However, when retested again on the first sequence on day 3, learning had been reversed, with performance

accuracy decreasing significantly, by more than 50%, and speed showing a small but non-significant reduction. At the same time, performance on the second, interference sequence showed significant overnight improvements in both speed and accuracy, similar to normal 24 hours improvement. The performance decreases for the first sequence on day 3 were significantly different compared to both the initial improvements achieved on this sequence on day 2 (+24 hours) and the improvements for the second sequence on day 3 (+24 hours). Furthermore, the loss of learning observed in group 7 on day 3 was also significantly different compared to the increases in performance for the first sequence on day 3 in groups 5 and 6 (accuracy, $P < 0.030$; speed, $P < 0.006$) (Fig. 4a–c). Thus, recollection

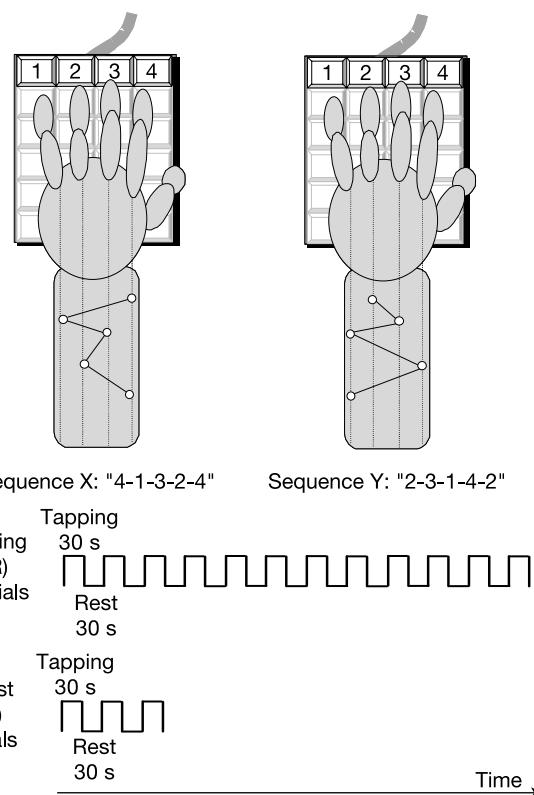


Figure 1 Procedural motor skill task and experimental protocol. One hundred right-handed subjects, aged 18–27 (s.d. \pm 2.1) and without neurological, psychiatric or sleep-disorder histories, were assigned to eight experimental groups (see Figs 2–4). Subjects performed a sequential finger tapping motor skill task (for detailed methods see ref. 7). The task involves pressing four numeric keys using the fingers of the left (non-dominant) hand, repeating a five-element sequence “as quickly and accurately as possible” for a trial period of 30 s, followed by 30 s of rest. Two motor patterns containing completely unique grammars (sequence X: 4-1-3-2-4; sequence Y: 2-3-1-4-2) were used in a balanced order as either the first or second sequence learned. Subjects were also retested on the first or second sequence in a balanced order to eliminate potential retest-ordering effects. Training (TR) consisted of 12 contiguous trials, while retesting (RT) involved three contiguous trials. Performance measures were the number of complete sequences achieved (‘speed’), and the number of errors made relative to the number of correct sequences (‘accuracy’). Comparisons within each experimental group were performed using repeated measures analysis of variance (ANOVA) and paired t -tests (two-tailed). Comparisons between groups were performed using unpaired t -tests (two-tailed). Retest values were compared to the final three trials of training, allowing true evaluation of between session, time-dependent learning, instead of the mean training value across all 12 trials. There were no significant differences between the initial learning curves during training for the first sequence compared to the second sequence learned in all groups that performed two sequences (average of groups 2–6 and 8; speed: ANOVA, $P = 0.423$; accuracy: ANOVA, $P = 0.408$).

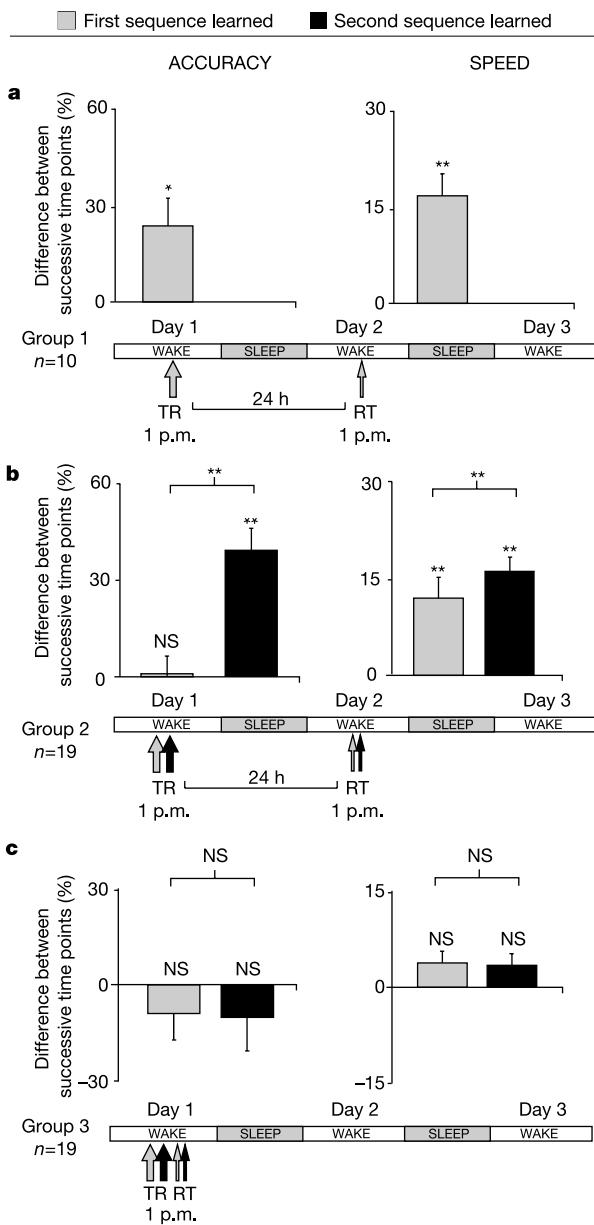


Figure 2 Changes in motor learning in groups 1–3. **a**, Group 1: following training on a single motor sequence on day 1 (thick arrow; TR), overnight increases in performance speed ($P < 0.001$) and accuracy ($P = 0.023$) were seen at the 24-hour retest on day 2 (thin arrow; RT). **b**, Group 2: immediately after learning the first motor sequence on day 1, subjects now learned a second motor sequence. When retested on day 2, improvements in accuracy occurred only for the second sequence ($P < 0.001$; filled black bar), while no such improvement developed for the first sequence ($P = 0.90$; grey bar). Improvements in speed ($P < 0.001$) were observed for both sequences. **c**, Group 3: subjects were again trained on the second motor sequence directly after the first. When retested after 5 min, performance indicated retention of initial training improvements for both sequences, but without the improvements in speed or accuracy seen 24 hours later. Error bars are s.e.m. Asterisks represent significance (P): * ≤ 0.05 ; ** ≤ 0.005 ; NS, non-significant. Same for all subsequent figures.

tion or reactivation of the first sequence memory during retesting on day 2 returned it to a labile state, making it once more susceptible to interference.

To confirm that interference following reactivation specifically disrupted subsequent reconsolidation rather than immediately reversing prior learning, an additional group of subjects (group 8) were retested a second time directly after the interference training

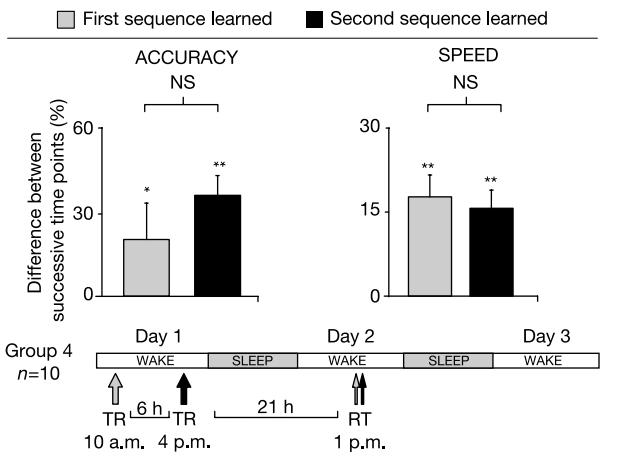


Figure 3 Changes in motor learning in group 4. Subjects initially learned the first motor sequence at 10 a.m. on day 1, and following a 6-hour waking interval, learned the second motor sequence. In contrast to group 2 who learned the second sequence immediately after the first, group 4 revealed significant improvements in performance speed ($P < 0.005$) and accuracy ($P < 0.05$) for both sequences.

on day 2, rather than on day 3 as in group 7, and showed no decrease in either speed or accuracy compared to the earlier retest (Fig. 4d).

Taken together with previous findings^{7,8,16}, these results describe a highly complex process of post-training memory consolidation and reconsolidation: (1) during initial training, learning occurs, leading to significant performance improvements; (2) between 10 minutes and 6 hours later, without intervening sleep, the memory undergoes the first, stabilization phase of consolidation, making it resistant to interference from a competing memory, but producing no improvement in speed or accuracy; (3) during the subsequent night, a second, enhancement stage of consolidation occurs, increasing both performance speed and accuracy on the task; (4) despite now having undergone both stabilization and enhancement, brief periods of rehearsal return the memory to a labile state, rendering it once again vulnerable to interference from a competing motor pattern, and in need of reconsolidation.

These findings provide, to our knowledge, the first clear dissociation of memory consolidation into temporally distinct phases of wake-state stabilization and sleep-dependent enhancement. However, it is unlikely that these stages of memory formation are generalizable across all human memory domains, as time-dependent consolidation has not been found for all memory tasks¹⁷. It also remains to be determined if these unique forms of memory consolidation are the result of common or distinct neural mechanisms. But the contrasting neurochemical and neurophysiological characteristics found across wake-sleep brain states¹⁸, together with their remarkably different patterns of gene expression and protein synthesis¹⁹, would argue for uniquely different neural mechanisms.

Our findings of memory lability and reconsolidation in humans complement similar findings in animal and clinical studies^{10–15}. Yet in these earlier reports, interference was produced using either electroconvulsive shock or cerebral injection of protein synthesis inhibitors, leaving it unclear whether more natural interventions could also block reconsolidation of labilized memories in humans. Our findings demonstrate that the simple process of training on a new motor sequence can block the reconsolidation of similar memories that have been returned to a labile state by simple rehearsal, and strongly imply functional significance for this process.

Allowing motor memories to return to a labile state requiring subsequent reconsolidation may permit the continued refinement and reshaping of previously learned movement skills in the context

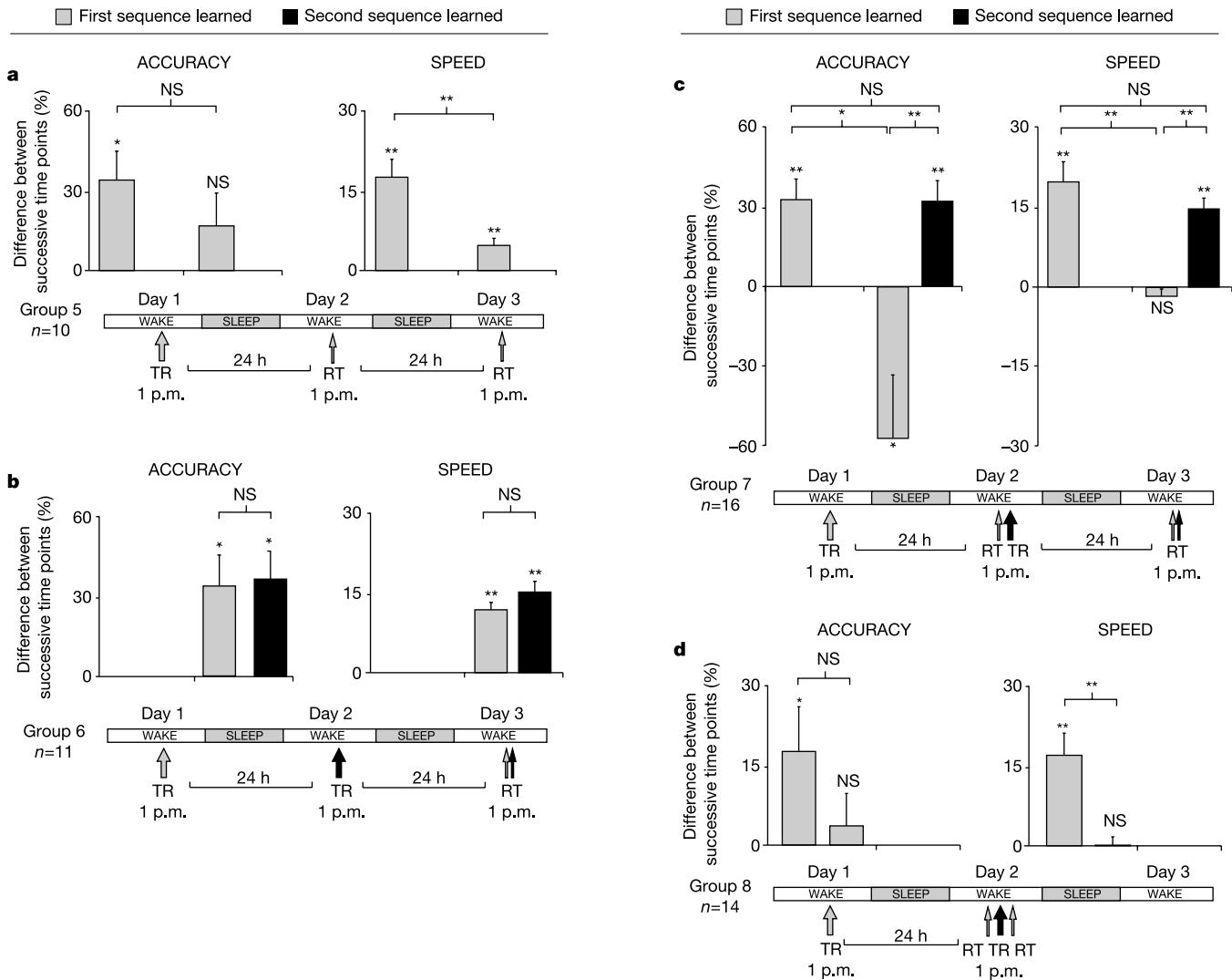


Figure 4 Changes in motor learning for groups 5–8. **a**, Group 5: subjects trained on only one sequence on day 1, and were retested once on day 2 and once on day 3. Significant improvements in accuracy ($P = 0.014$) and speed ($P < 0.001$) developed on day 2, with smaller but continued increases in speed ($P < 0.001$) and non-significant improvements in accuracy developing on day 3. **b**, Group 6: subjects were trained on the first sequence on day 1, and 24 hours later, trained on the second sequence on day 2. When retested on day 3, improvements in accuracy ($P < 0.015$) and speed ($P < 0.003$) had developed for both sequences equally. **c**, Group 7: subjects trained on the first sequence on day 1. On day 2, subjects were retested on the first sequence, demonstrating overnight improvements in speed ($P < 0.001$) and accuracy ($P < 0.001$) at the first retest. Immediately following this recall test on day 2, subjects learned the second sequence. When retested on this second sequence, overnight improvements in speed ($P < 0.001$)

and accuracy ($P < 0.001$) had developed on day 3, similar to those that developed for the first sequence on day 2. However, when subjects were now retested again on the first sequence on day 3 (+48 hours), the initial learning achieved on day 2 was lost, with accuracy decreasing significantly relative to day 2 ($P < 0.030$), and speed showing a non-significant reduction. **d**, Group 8: subjects trained on the first sequence on day 1 and were retested on day 2, showing significant overnight improvements in speed ($P = 0.001$) and accuracy ($P = 0.056$). Following recall of the first sequence on day 2, subjects learned the second interference sequence, but in contrast to group 7, were immediately retested on the first sequence again. At this second retest on day 2, subjects demonstrated maintenance, not loss, of the overnight improvements relative to the first retest (speed, $P = 0.947$; accuracy, $P = 0.447$).

of ongoing experience, and may even act as a process of extracting and integrating common elements from related complex movements. These results raise the possibility that similar mechanisms may also contribute to the integration of episodic memories and the revision of semantic knowledge based on newly acquired information. □

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- McGaugh, J. L. Memory—a century of consolidation. *Science* **287**, 248–251 (2000).
- Mueller, W. *et al.* Early consolidation in human primary motor cortex. *Nature* **415**, 640–644 (2002).
- Brashers-Krug, T., Shadmehr, R. & Bizzi, E. Consolidation in human motor memory. *Nature* **382**, 252–255 (1996).
- Karni, A., Tanne, D., Rubenstein, B. S., Askenasy, J. J. & Sagi, D. Dependence on REM sleep of overnight improvement of a perceptual skill. *Science* **265**, 679–682 (1994).
- Smith, C. & MacNeill, C. Impaired motor memory for a pursuit rotor task following Stage 2 sleep loss in college students. *J. Sleep Res.* **3**, 206–213 (1994).
- Stickgold, R., James, L. & Hobson, J. A. Visual discrimination learning requires sleep after training. *Nature Neurosci.* **3**, 1237–1238 (2000).
- Walker, M. P., Brakefield, T., Morgan, A., Hobson, J. A. & Stickgold, R. Practice with sleep makes perfect: Sleep dependent motor skill learning. *Neuron* **35**, 205–211 (2002).
- Walker, M. P., Brakefield, T., Hobson, J. A. & Stickgold, R. Sleep and the time course of motor skill learning. *Learn. Mem.* **10**, 275–284 (2003).
- Nader, K. Memory traces unbound. *Trends Neurosci.* **26**, 65–72 (2003).
- Misanin, J. R., Miller, R. R. & Lewis, D. J. Retrograde amnesia produced by electroconvulsive shock after reactivation of a consolidated memory trace. *Science* **160**, 554–555 (1968).
- Rubin, R. D. Clinical use of retrograde amnesia produced by electroconvulsive shock. A conditioning

- hypothesis. *Can. Psychiatr. Assoc. J.* **21**, 87–90 (1976).
12. Gordon, W. C. & Spear, N. E. The effects of strychnine on recently acquired and reactivated passive avoidance memories. *Physiol. Behav.* **10**, 1071–1075 (1973).
 13. Nader, K., Schafe, G. E. & Le Doux, J. E. Fear memories require protein synthesis in the amygdala for reconsolidation after retrieval. *Nature* **406**, 722–726 (2000).
 14. Debiec, J., LeDoux, J. E. & Nader, K. Cellular and systems reconsolidation in the hippocampus. *Neuron* **36**, 527–538 (2002).
 15. Milekic, M. H. & Alberini, C. M. Temporally graded requirement for protein synthesis following memory reactivation. *Neuron* **36**, 521–525 (2002).
 16. Fischer, S., Hallschmid, M., Elsner, A. L. & Born, J. Sleep forms memory for finger skills. *Proc. Natl Acad. Sci. USA* **99**, 11987–11991 (2002).
 17. Goedert, K. M. & Willingham, D. B. Patterns of interference in sequence learning and prism adaptation inconsistent with the consolidation hypothesis. *Learn. Mem.* **9**, 279–292 (2002).
 18. Pace-Schott, E. F. & Hobson, J. A. The neurobiology of sleep: Genetics, cellular physiology and subcortical networks. *Nature Rev. Neurosci.* **3**, 591–605 (2002).
 19. Hobson, J. A. & Pace-Schott, E. F. The cognitive neuroscience of sleep: Neuronal systems, consciousness and learning. *Nature Rev. Neurosci.* **3**, 679–693 (2002).

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Environmentally mediated synergy between perception and behaviour in mobile robots

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The notion that behaviour influences perception seems self-evident, but the mechanism of their interaction is not known. Perception and behaviour are usually considered to be separate processes. In this view, perceptual learning constructs compact representations of sensory events, reflecting their statistical properties^{1,2}, independently of behavioural relevance^{3,4}. Behavioural learning^{5,6}, however, forms associations between perception and action, organized by reinforcement^{7,8}, without regard for the construction of perception. It is generally assumed that the interaction between these two processes is internal to the agent, and can be explained solely in terms of the neuronal substrate⁹. Here we show, instead, that perception and behaviour can interact synergistically via the environment. Using simulated and real mobile robots, we demonstrate that perceptual learning directly supports behavioural learning and so promotes a progressive structuring of behaviour. This structuring leads to a systematic bias in input sampling, which directly affects the organization of the perceptual system. This external, environmentally mediated feedback matches the perceptual system to the emerging behavioural structure, so that the behaviour is stabilized.

One reason for the lack of progress in understanding the interrelationship of behaviour and perception is experimental intractability. An explanation of their coupling requires detailed analysis at both the behavioural and neuronal levels. Our approach was to bypass the animal experimental difficulty by using a mobile robot, for which it is possible to fully observe and quantify perception and

behaviour. The robot is controlled by a neural model, called distributed adaptive control (DAC), that includes mechanisms for perceptual and behavioural learning^{10,11}. The DAC architecture (see Fig. 1 and Methods) consists of three layers: ‘reactive’, ‘adaptive’ and ‘contextual’ control.

The reactive control layer implements a repertoire of basic reflex actions where low-complexity sensory events, unconditioned stimuli (US), trigger simple actions, unconditioned responses (UR), via an internal state (IS) representation. As a result of learning at the level of adaptive control, the purely reactive activation of the IS populations by US events is progressively replaced by acquired representations of sensory events, conditioned stimuli (CS), and the generation of conditioned responses (CR)^{11,12}. US events are the initial reinforcers of this learning process. The local learning mechanism that is used automatically generates a measure, D , of the discrepancy between expected and actual CS events (see Methods). When D falls below a specified transition threshold, θ_D , the contextual control layer is enabled. This layer is a behavioural learning system that constructs higher-order representations of the temporal order of the sensori-motor representations constructed by the adaptive layer (see Methods).

Representations of CS and CR events are stored in short-term memory (STM) when the adaptive layer triggers CRs. The content of STM is stored in long-term memory (LTM) when a goal state is reached, such as when a target is found. CS representations of the LTM of the contextual layer are matched to those generated by the adaptive layer. The best-matching CS representation at the level of contextual control will define the next action by projecting its CR representation onto the motor population M when the reactive layer is quiescent. Chaining through a LTM sequence is achieved through a biased competition mechanism (see Methods). DAC is a practical model of how different learning systems in the mammalian brain act together to generate adaptive goal-oriented behaviour, and is a standard in the field of new artificial intelligence and behaviour-based robotics^{13–16}. Moreover, it exhibits the regularities of bayesian decision-making that are thought to be one of the characteristics of human cognition^{17,18}.

We first investigated the hypothesis that the performance of the robot is enhanced through the contextual layer. To test this hypothesis, we used both simulated and real-world robots in a foraging task where collisions had to be minimized while the number of targets found had to be maximized. We distinguished

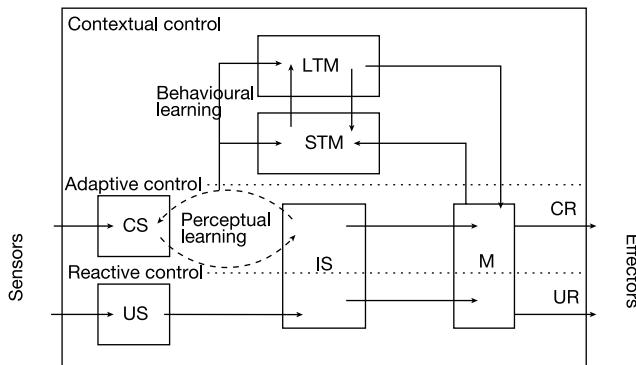


Figure 1 Distributed adaptive control. DAC is based on the assumption that adaptive behaviour results from three tightly coupled layers of control: reactive, adaptive and contextual control. Each box represents a neuronal population. Arrows indicate the connections between these populations. US, unconditioned stimulus population. CS, conditioned stimulus population. IS, internal state populations. M, motor neuron population. UR, unconditioned response. CR, conditioned response. STM, short-term memory. LTM, long-term memory. See text and Methods for explanation.