Attention deficits without cortical neuronal deficits

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The ability to process relevant stimuli selectively is a fundamental function of the primate visual system. The best-understood correlate of this function is the enhanced response of neurons in the visual cortex to attended stimuli1,2. However, recent results show that the superior colliculus (SC), a midbrain structure, also has a crucial role in visual attention3–5. It has been assumed that the SC acts through the same well-known mechanisms in the visual cortex3,5. Here we tested this hypothesis by transiently inactivating the SC during a motion-change-detection task and measuring responses in two visual cortical areas. We found that despite large deficits in visual attention, the enhanced responses of neurons in the visual cortex to attended stimuli were unchanged. These results show that the SC contributes to visual attention through mechanisms that are independent of the classic effects in the visual cortex, demonstrating that other processes must have key roles in visual attention.

Visual attention is a fundamental brain function that makes it possible to base perceptions and actions on the relevant parts of the environment. In the laboratory, visual attention is typically studied by asking subjects to respond to the properties of a cued stimulus while simultaneously ignoring the content of irrelevant, distracting stimuli. Twenty-five years ago, it was shown that in the primate visual cortex, the activity of neurons responsive to cued visual stimuli was higher than the activity evoked by un-cued distracters6. This finding, later termed ‘gain modulation’, has been subsequently observed in many different areas of the cerebral cortex7–9, in many variants of the cueing task6.

Visual attention is now understood to involve a network of areas, including the frontal and parietal cortex, as well as the visual cortex10, and gain modulation of sensory responses is commonly considered to be the keystone of the neuronal mechanisms of attention11. Correlates of visual attention are not restricted to the cortex and have also been found in subcortical structures such as the SC11–13 and thalamus12–14. Some of these effects could be inherited from the cortex. However, manipulation of neuronal activity in the SC alters or disrupts performance in tasks that test visual attention15–18, indicating that the SC has a causal role. In a recent study using pharmacologic inactivation of the SC, monkeys had to report the direction of motion in a stimulus at a cued location, while ignoring equivalent motion in an irrelevant ‘foil’ stimulus located elsewhere14. After SC inactivation, the animals showed profound deficits in visual attention: they largely failed to report the direction of motion of the cued stimulus when it was placed in the part of the visual field affected by SC inactivation, and instead reported the direction of motion of the foil stimulus. Activity in the SC is therefore not simply updated about visual attention but seems to be necessary for its normal operation.

Previous studies have generally assumed that the SC plays a part in attention by influencing the well-known mechanisms in the visual cortex19–21. If so, then disrupting visual attention by inactivating the SC should change attention-related effects in the visual cortex. We tested this hypothesis by recording the activity of single neurons in the middle temporal area (MT) and medial superior temporal area (MST)—two cortical visual areas well known for their roles in processing motion signals22—and their modulation by visual attention23—while monkeys performed a motion-change-detection task. We measured how neuronal activity was modulated by spatial cues before and during temporary pharmacological inactivation of SC. Contrary to the hypothesis, we found that attention-related effects in MT and MST remained intact even though SC inactivation caused major deficits in the visual attention task.

Two monkeys (J and M) performed a motion-detection task in which they were rewarded for pressing a button when they correctly detected a change in the direction of motion of the stimulus at the cued location and ignored changes in the direction of motion of a foil stimulus located diagonally opposite the cued stimulus (Fig. 1a). In trials in which the change occurred in the cued stimulus, the animals pressed the button correctly in about 50–60% of the trials (Fig. 1c, pre-injection ‘hit rates’ were 53 ± 26% for J and 57 ± 21% for M). Conversely, they correctly refrained from responding in most of the trials in which the change occurred in the distracter stimulus (pre-injection ‘false alarm’ rates were 9 ± 15% for J and 9 ± 7% for M).

To test the effects of SC inactivation on attention and sensory cortex activity during this task, we injected muscimol, a GABA_A (γ-aminobutyric acid type A) agonist, in the intermediate and deep layers of the SC (Fig. 1b). The extent of the neuronal inhibition caused by the injection was assessed at the beginning and end of each session, by measuring eye peak velocity during visually guided saccades24. Each session included two data-collection phases, one before and one during SC inactivation.

Consistent with previous results1, we found that SC inactivation caused large and spatially specific deficits in the ability of the animal to detect changes in the cued stimulus, with post-injection hit rates dropping to about 10–15% in the part of the visual field affected by SC inactivation (Fig. 1c–e and Supplementary Information). We then tested whether SC inactivation induced comparable changes in the cue-related modulation of activity in MT and MST.

Neurons were recorded in either the MT or MST area while the monkeys performed the task, during the same behavioural sessions documented above. The location and direction of motion of the stimuli were based on the tuning properties of the neurons, and the size of the motion patch was adjusted to the size of the receptive fields (see Methods). In brief, either the cued or the foil stimulus was placed in the receptive field of the neuron under study, and the direction of motion on each trial was set as the preferred or anti-preferred direction of the neuron, and was always opposite in the two stimulus patches. We recorded a total of 69 MST (monkey J, 31; monkey M, 38) and 44 MT (J, 34; M, 10) neurons before inactivation and 77 MST (J, 26; M, 51) and 55 MT (J, 47; M, 8) neurons during inactivation. Some of these neurons were isolated continuously throughout the experiment (n = 36 cells for MST and n = 18 cells for MT). We provide additional analyses for this particular set of neurons in Supplementary Information.

Before SC inactivation, as expected from previous studies demonstrating attention-related modulation of visual responses in MT and MST22, we found that neurons recorded in MST (Fig. 2c) and MT (Fig. 2g) showed higher discharge rates when the motion stimulus in their receptive field was cued (‘cue in’) than when it was not cued (‘cue out’). As in previous studies, we quantified this modulation by...
two sample experiments shown in Fig. 2. The arrows point to the data corresponding to the (contra.) and ipsilateral (ipsi.) to the injection before (x) and during (y) SC inactivation. Each dot corresponds to a different experiment and the grey lines show the 95% confidence interval (the computation of which is based on a method described in ref. 29). The arrows point to the data corresponding to the two sample experiments shown in Fig. 2.

Measuring the discharge rate during the period of the task (300–800 ms after motion stimuli onset), and computed a modulation index, defined as the difference in discharge rates between cue in and cue out conditions, divided by their sum. For the two sample neurons shown in Fig. 2, the modulation indexes were 0.16 and 0.07 for the MST and MT neurons, which corresponded to increases in the discharge rate of 39% and 15%, respectively.

During SC inactivation, this modulation was intact. Neurons in MST (Fig. 2d) and MT (Fig. 2h) continued to show higher discharge rates for the motion stimulus in their receptive field when it was cued than when it was not cued. The post-injection modulation indexes were 0.21 and 0.08 for the MST neuron and MT neuron, respectively, which were not significantly different from their pre-injection values, but remained significantly greater than chance (both \( P < 0.001 \), Wilcoxon rank-sum test, cue in versus cue out). This cue-related modulation in discharge rate was intact, despite the deficits in detection performance observed simultaneously during the SC inactivation (Fig. 1d).

To quantify the effect of SC inactivation across our population, we measured a modulation index for each neuron before and during inactivation. Pre-injection, the average modulation index in our sample of neurons was \( 0.075 \pm 0.029 \) (mean \( \pm 95\% \) confidence interval; median, 0.051) in MST and 0.061 \( \pm 0.023 \) in MT (median, 0.048; significantly greater than zero, Wilcoxon signed-rank test, all \( P < 0.001 \) (Fig. 3a) corresponding to average increases in the discharge rate of 24% and 15%, respectively. Post-injection, the average modulation index was 0.071 \( \pm 0.025 \) (median, 0.048) in MST and 0.057 \( \pm 0.022 \) in MT (median, 0.041) (Fig. 3a); these values remained significantly greater than zero (Wilcoxon signed-rank test, all \( P < 0.001 \), and were not different from the values before inactivation (Wilcoxon rank-sum test, \( P > 0.5 \); Bayesian posterior probability of the null (no-change) hypothesis \( (p(H_0)) \), MST, 0.99; MT, 0.985). Thus, SC inactivation produced no appreciable change in the cue-related modulation of the average discharge rate across our sample of MST and MT neurons.
results were found when the non-preferred stimulus was presented inside the receptive field (Supplementary Information).

We considered whether SC inactivation might have altered other aspects of cue-related changes in MST and MT neuronal activity. Although modulation of average discharge rate is the standard method for documenting attention-related changes in neuronal activity, it does not measure how noise or variability of discharge rate might change with attention.

To address this point, we computed three additional values for each neuron. First, we computed the area under the receiver operating characteristic (ROC) curve, which indicates how well an ideal observer could classify the condition based on the activity of the neuron; in our case, whether the cued or un-cued stimulus was in the receptive field of the neuron. Second, we computed the Fano factor (the ratio of the variance over the mean of the response), which has been found to be lower for cued stimuli than for un-cued stimuli, indicating that attention decreases the variability of neuronal activity. Third, we computed the noise correlation between pairs of simultaneously recorded neurons, which has recently been found to decrease with attention, improving the signal-to-noise ratio of visual signals across the population of neurons.

These additional measurements were also unchanged by SC inactivation. The ROC areas were significantly higher than chance for both MST and MT (Wilcoxon signed-rank test, all P < 0.001), and were not changed by SC inactivation (Wilcoxon rank-sum test, MST, P = 0.30; MT, P = 0.28; Bayesian p(H0), MST, 0.978; MT, 0.981); this result indicates that the ability of an ideal observer to discriminate the cued location was unchanged by SC inactivation.

The Fano factor index was significantly less than chance (Fig. 3c), both before and during SC inactivation in both MST and MT (Wilcoxon signed-rank test, all P < 0.001; MT, P = 0.007) and during (MST, P < 0.0001; MT, P = 0.04) inactivation, and not different from each other (MST, P = 0.66; MT, P = 0.23; Bayesian p(H0), MST, 0.988; MT, 0.979); this result shows that the ability to distinguish the variance in the discharge rate was reduced by spatial cueing both before and during SC inactivation.

The change in interneuronal correlation was significantly less than zero (Fig. 3d), both before (MST, P < 0.0001; MT, P = 0.0001) and during (MST, P = 0.0001; MT, P = 0.0005) inactivation, and not different from each other (MST, P = 0.76; MT, P = 0.71; Bayesian p(H0), MST, 0.996; MT, 0.995); this result indicates that spatial cues reduced the correlation in activity between neurons, and this reduction was unchanged by SC inactivation. Similar findings were made with a wide range of bin sizes used to compute the correlations (Supplementary Information).

Finally, we examined cue-related modulations in neuronal activity during other intervals in the task as well as changes in neuronal activity unrelated to the cue, and these were also unchanged during SC inactivation (Supplementary Information). We also confirmed that neuronal activity in the parts of MST we recorded were indeed necessary for the performance of the attention task (Supplementary Information).

In summary, we found that during SC inactivation, the enhanced responses of neurons in the visual cortex to attended stimuli were preserved despite large behavioural impairments in a covert attention task. This result was found in two visual areas well known for their roles in processing motion signals and their modulation by visual attention. Moreover, the attention deficit induced by SC inactivation not only preserved the cue-related changes in visual responses, but it also left intact the other known correlates of attention in the visual cortex: the ability of neurons to discriminate cued from un-cued spatial locations, the reliability of neuronal discharge (that is, Fano factor) and cue-related changes in noise correlations between neurons. These effects cannot be explained by a sensory impairment, because previous studies have shown that attention deficits during SC inactivation are not caused by changes in local motion perception. The effects also cannot be explained by a motor deficit, because the single-button response in our task was unimpared for stimuli outside the affected region of the visual field.

These findings demonstrate that the known modulations of activity in the visual cortex are not the only mechanisms involved in the control of attention and that other processes must have a key role. One possibility is that visual attention involves other aspects of neuronal activity in these same visual areas. For example, although we found no changes in correlations between nearby neurons, there could be changes between more distant sites or across different areas. A second possibility is that the crucial steps take place in other brain areas entirely, for example, in the parietal or prefrontal cortex, the SC or the basal ganglia. In particular, the frontal eye fields (FEF) exert effects on attention qualitatively similar to the SC. However, because of prominent feedback from FEF to the visual cortex, SC-induced changes in FEF might have been expected to also change responses in the visual cortex. Finally, it is possible that different circuits mediate different aspects of attention. For example, changes in the visual cortex might be important for feature-based attention and for regulating the perceptual appearance of stimuli, whereas the mechanism targeted by SC inactivation is important for the all-or-none aspects of spatial attention (for example, change blindness).

**METHODS SUMMARY**

We performed MT and MST neuronal recording and reversible inactivation of the SC in two adult rhesus monkeys (subjects J and M). The animals were prepared using standard surgical techniques described in detail in ref. 17. All experimental protocols were approved by the Institutional Animal Care and Use Committee and complied with US Public Health Service policy on the humane care and use of laboratory animals. The laboratory set-up for behavioral control and monitoring was identical to that described in ref. 17.

At the beginning of each inactivation session, we lowered a recording tetrode in a track selected on the basis of previous recording sessions. After identification of a good recording spot for MT and MST neurons, we mapped the receptive fields (see examples in Fig. 2a, b, e, f, 50–60 trials) and motion-direction tuning properties (Fig. 2a, b, e, f, 30–60 trials) of the isolated neurons and recorded them during performance of the attentional task (232–366 trials). After completion of the pre-injection data collection, an electrode was lowered into the intermediate and deep layers of the SC and muscimol was injected following a procedure described in ref. 4. Around 20 min after the beginning of the injection, the extent of the effect of the inactivation was evaluated on the basis of eye velocity during visually guided saccades (60–120 trials). MT and MST neurons were then recorded again during the receptive field and tuning-mapping procedures and during the attentional task.
Full Methods and any associated references are available in the online version of the paper.

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1. Desimone, R. & Duncan, J. Neural mechanisms of selective visual attention. Annu. Rev. Neurosci. 18, 193–222 (1995).
2. Reynolds, J. H. & Chelazzi, L. Attentional modulation of visual processing. Annu. Rev. Neurosci. 27, 611–647 (2004).
3. Müller, J. R., Philia-stides, M. G. & Newsome, W. T. Microstimulation of the superior colliculus focuses attention without moving the eyes. Proc. Natl Acad. Sci. USA 102, 524–529 (2005).
4. Lovejoy, L. P. & Krauzlis, R. J. Inactivation of primate superior colliculus impairs covert selection of signals for perceptual judgments. Nature Neurosci. 13, 261–266 (2010).
5. Cavanagh, J. Subcortical modulation of attention counters change blindness. J. Neurosci. 24, 11236–11243 (2004).
6. Moran, J. & Desimone, R. Selective attention gates visual processing in the extrastriate cortex. Science 229, 782–784 (1985).
7. Treue, S. Neural correlates of attention in primate visual cortex. Trends Neurosci. 24, 295–300 (2001).
8. Roelfsema, P. R., Lamme, V. A. & Spekreijse, H. Object-based attention in the primary visual cortex of the macaque monkey. Nature 395, 376–381 (1998).
9. Corbetta, M. & Shulman, G. L. Control of goal-directed and stimulus-driven attention in the brain. Nature Rev. Neurosci. 3, 201–215 (2002).
10. Ignashchenkova, A., Dicke, P. W., Haarmeier, T. & Thier, P. Neuron-specific contribution of the superior colliculus to overt and covert shifts of attention. Nature Neurosci. 7, 56–64 (2003).
11. O’Connor, D. H., Fukui, M. M., Pinsk, M. A. & Kastner, S. Attention modulates responses in the human lateral geniculate nucleus. Nature Neurosci. 5, 1203–1209 (2002).
12. Bender, D. B. & Youakim, M. Effect of attentive fixation in macaque thalamus and cortex. J. Neurophysiol. 85, 219–234 (2001).
13. Robinson, D. L. & Petersen, S. E. The pulvinar and visual salience. Trends Neurosci. 15, 127–132 (1992).
14. Rudolph, K. Transient and permanent deficits in motion perception after lesions of cortical areas MT and MST in the macaque monkey. Cereb. Cortex 9, 90–100 (1999).
15. Treue, S. & Maunsell, J. H. R. Attentional modulation of visual motion processing in cortical areas MT and MST. Nature 382, 539–541 (1996).
16. Treue, S. & Martinez Trujillo, J. C. Feature-based attention influences motion processing gain in macaque visual cortex. Nature 422, 4228–4235 (2006).
17. Hafer, Z. M., Goffart, L. & Krauzlis, R. J. Superior colliculus inactivation causes stable offsets in eye position during tracking. J. Neurosci. 28, 8124–8137 (2008).
18. Hanley, J. A. & McNeil, B. J. The meaning and use of the area under a receiver operating characteristic (ROC) curve. Radiology 143, 29–36 (1982).
19. Mitchell, J. F., Sundberg, K. A. & Reynolds, J. H. Differential attention-dependent response modulation across cell classes in macaque visual area V4. Neuron 55, 131–141 (2007).
20. Cohen, M. R. & Maunsell, J. H. R. Attention improves performance primarily by reducing interneuronal correlations. Nature Neurosci. 12, 1594–1600 (2009).
21. Mitchell, J. F., Sundberg, K. A. & Reynolds, J. H. Spatial attention decorrelates intrinsic activity fluctuations in macaque area V4. Neuron 63, 879–888 (2009).
22. Moore, T. The neurobiology of visual attention: finding sources. Curr. Opin. Neurobiol. 16, 159–165 (2006).
23. Redgrave, P., Prescott, T. J. & Gurney, K. The basal ganglia: a vertebrate solution to the selection problem? Neurosci. 389, 1009–1023 (1999).
24. Moore, T. & Armstrong, K. M. Selective gating of visual signals by microstimulation of frontal cortex. Nature 421, 370–373 (2003).
25. Wardak, C., Iilos, G., Duhamel, J.-R. & Olivier, E. Contribution of the monkey frontal eye field to covert visual attention. J. Neurosci. 26, 4228–4235 (2006).
26. Treue, S. & Martinez Trujillo, J. C. Feature-based attention influences motion processing gain in macaque visual cortex. Nature 422, 4228–4235 (2006).
27. Rensink, R. A., O’Regan, J. K. & Clark, J. J. To see or not to see: the need for attention to perceive changes in scenes. Psychol. Sci. 8, 368–373 (1997).
28. Ross, T. D. Accurate confidence intervals for binomial proportion and Poisson rate estimation. Comput. Biol. Med. 33, 509–531 (2003).
METHODS

Monkey preparation. We performed MT and MST neuronal recordings and reversible inactivation of the intermediate and deep layers of the SC in two adult rhesus monkeys (subjects J and M) that were 12–16 years of age and weighed 14–16 kg. The monkeys were prepared using standard surgical techniques described in detail in ref. 17. All experimental protocols were approved by the Institutional Animal Care and Use Committee and complied with US Public Health Service policy on the humane care and use of laboratory animals. The laboratory set-up for behavioural control and monitoring was identical to that described in ref. 17.

Attentional task. Trials began with the appearance of a central dot on which the monkey had to fixate during the whole trial duration. Achievement of fixation triggered the display of a peripheral stimulus, the cue, consisting of a 5°–7° wide patch of static dots. The actual size of the patch was chosen as not to exceed the size of the receptive fields of the neurons being recorded. On each trial, the cue could be displayed at one of two possible locations, chosen randomly. One of these locations was chosen to be in the centre of the receptive fields of the recorded neurons and the other one was the symmetric location across the fixation point. The cue was displayed for 133 ms and was followed by a 500-ms delay, during which only the fixation point was displayed. Two patches of moving dots were then displayed at the two previously described locations. The dots were moving in opposite directions in the two patches, one of which being the preferred direction of the neurons being recorded. The characteristics of the stimulus have been described elsewhere19. In the present case, the dots had an eight-frame lifetime (corresponding to 107 ms). The direction of motion of each dot was drawn from a normal distribution centred on the direction of motion of the patch and with a 16° standard deviation. The direction of motion of the patches remained constant for 800 ms plus a geometrically distributed delay of mean 480 ms (range, 0–3520 ms). This distribution allowed the hazard function to remain flat during the delay. After this delay, the direction of motion of one of the patches changed. The monkey had to press a button whenever the change in direction occurred at the previously cued location. The change varied from 16° to 20° and was adjusted on the basis of the performance of the monkey at the beginning of each session to keep a global performance of about 75%.

After the beginning of the change in direction, stimuli remained on the screen for 650 ms or until the response of the animal. Monkeys received a liquid reward only for correct responses in completed trials (button press after change occurred at cued location or absence of response when no change occurred or change occurred at un-cued location). If the monkey broke fixation midtrial, the trial was aborted and repeated later in the session. This paradigm has been referred to as a ‘filtering’ task because it requires the monkey to actively ignore stimulus changes at the un-cued location. The advantage of this task design is that correct performance requires the filtering out of signals from irrelevant distractor stimuli. This paradigm is similar to that used originally to demonstrate attentional modulation in areas MT and MST19 and more recently to show a causal role of the SC in this paradigm is similar to that used originally to demonstrate attentional modulation in areas MT and MST19 and more recently to show a causal role of the SC in this task.

Neuronal recordings. Recordings were conducted with a tetrode (Thomas Recording GmbH). Neuronal signals were amplified, band-pass filtered and digitized (Plexon recording system). Neurons were isolated during the experiment to allow for online mapping of their receptive fields and motion-direction tuning properties. In parallel, all waveforms passing a manually set threshold were stored for offline sorting. Offline sorting was conducted first automatically (Klustakwik sorting algorithm15) and then refined manually. On average, we recorded 7.5 neurons per experimental session.

Inactivation experiments. In the four-channel waveforms and interspike interval distributions of each neuron isolated before muscimol injection was correlated with the waveforms and interspike interval distribution of each neuron isolated after injection22. We then used these correlation values to identify the neurons that were putatively the same before and during inactivation (see also Supplementary Information).

Motion-direction tuning and receptive-field mapping. After isolation of the neurons, the motion-direction tuning of the cells was first evaluated, following a procedure similar to that described in ref. 33. In brief, the monkey had to fixate on a central dot while a whole-screen patch of dots was moving coherently in a direction changing on every frame, leading to a circular motion. The direction of rotation (clockwise or anticlockwise) was selected randomly on every trial. The response of the isolated neurons as a function of the direction of motion of the patch was used to determine their preferred direction of motion.

In order to estimate the probability of an absence of difference between the pre- and post-injection data, we computed the Bayes factor for the comparison between a model assuming a change in mean value during inactivation and a model assuming no change (H0). When necessary, data were transformed to achieve a normal distribution. We computed the Bayes factor by means of different methods: fractional Bayes factor25, Bayesian information criterion37 and Bayesian t-test based on the Savage–Dickey ratio test38. These different methods provided comparable results. We mention in the main text only the p(H0) computed with the fractional Bayes factor method.

Interneuronal correlations. Interneuronal correlations were computed following the same procedure as described in ref. 21. In brief, the delay period (between 300 and 800 ms following stimulus onset) was divided into non-overlapping bins (1.6, 6, 7, 11, 16, 22, 31, 45, 63, 83, 125, 250 or 500 ms long) in which spike counts were computed. The average spike count in each bin was subtracted out from the spike-count values to remove any stimulus-locked response variation. Similarly, the slow variation in discharge rate over consecutive trials was also removed by subtracting the Gaussian-weighted smoothing of spike-count changes (σ = five trials). Pearson correlations were computed for all pairs of units having a minimum discharge rate of five spikes per second (MT before inactivation, 122 pairs; MT during, 134 pairs; MST before, 194 pairs; MST during, 235 pairs).

We then estimated the effect of attention on interneuronal correlations by computing the difference in correlations (cue in minus cue out) for MST and MT. These differences are shown in Supplementary Material for all bin sizes. To illustrate these results in the main article, we chose a bin size of 31 ms (shown in Fig. 3), on the basis of the timescale of interneuronal correlations estimated in ref. 37. Because the spike counts obtained with this bin size were not always...
normally distributed, we also performed the same analysis using non-parametric Spearman correlations and obtained similar results.

30. Palmer, J. & Moore, C. M. Using a filtering task to measure the spatial extent of selective attention. *Vision Res.* **49**, 1045–1064 (2009).

31. Harris, K. D., Henze, D. A., Csicsvari, J., Hirase, H. & Buzsáki, G. Accuracy of tetrode spike separation as determined by simultaneous intracellular and extracellular measurements. *J. Neurophysiol.* **84**, 401–414 (2000).

32. Dickey, A. S., Suminski, A., Amit, Y. & Hatsopoulos, N. G. Single-unit stability using chronically implanted multielectrode arrays. *J. Neurophysiol.* **102**, 1331–1339 (2009).

33. Schoppmann, A. & Hoffmann, K. P. Continuous mapping of direction selectivity in the cat’s visual cortex. *Neurosci. Lett.* **2**, 177–181 (1976).

34. Berger, J. & Pericchi, L. in *Model Selection* Vol 38 (ed. Lahiri, P) 135–207 (Institute of Mathematical Statistics Lecture Notes – Monograph Series, 2001).

35. Wagenmakers, E. J. A practical solution to the pervasive problems of p values. *Psychon. Bull. Rev.* **14**, 779–804 (2007).

36. Wetzels, R., Raaijmakers, J. G. W., Jakab, E. & Wagenmakers, E. J. How to quantify support for and against the null hypothesis: a flexible WinBUGS implementation of a default Bayesian t test. *Psychon. Bull. Rev.* **16**, 752–760 (2009).

37. Bair, W., Zohary, E. & Newsome, W. T. Correlated firing in macaque visual area MT: time scales and relationship to behavior. *J. Neurosci.* **21**, 1676–1697 (2001).