Attentional suppression of activity in the human visual cortex

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We have used fMRI to examine the nature of the changes that occur in the human visual cortex when an observer attends to a particular location in the visual image. Previous studies have shown that the magnitude of the response to a visual stimulus is increased when the observer attends to the stimulus. We show that, in addition, attention to a particular location results in a widespread suppression of activity levels at all other locations. This suggests that a key mechanism of attentional modulation may be that spontaneous (baseline) levels of neural activity are adjusted in a position-dependent manner across the entire visual field. *NeuroReport* 11:271–277 © 2000 Lippincott Williams & Wilkins.

Key words: Attentional modulation; fMRI; Visual cortex

INTRODUCTION

It has been known for some time that shifts in attention between different parts of a visual image can modulate the magnitude of excitatory responses to visual stimuli in primate cerebral cortex. It has recently become clear that, in humans, such modulation occurs not only in parietal and extrastriate cortex but also in the primary visual cortex (area V1). For example, in an elegantly simple experiment, Gandhi *et al*. [1] used fMRI to estimate activity levels in V1. The visual stimulation was essentially constant over time, but the subject periodically shifted attention between two similar images presented one on each side of a fixation spot. The magnitude of V1 activation caused by the invariant visual stimuli was found to vary systematically as the subject switched attention from left to right, the attended stimulus always eliciting more activity than the unattended. Other recent fMRI studies [2–5] have reached similar conclusions. In all cases, the activation in V1 caused by visual stimuli was increased when the subject attended to those stimuli.

These studies provide a firm foundation for future experiments designed to establish the nature of attentional modulation in the visual cortex. They are consistent with the notion that focal attention causes a change in the gain of the excitatory responses of sensory neurons to stimuli at the attended location. On this view, neurons do not change their response selectivities (orientation tuning, direction sensitivity, etc.) but the stimulus-related response derived from the retina is simply multiplied by some factor that depends on attention. This gain modulation could be intrinsic, occurring within the visual cortex, or extrinsic, depending on feedback from higher cortical areas.

Neurophysiological studies involving attentional modulation of single visual neurons in awake primates have also emphasized modulation of the magnitude of excitatory responses to stimuli to which the neuron is sensitive [6–11]. In this domain too, attentional modulation has been considered as a simple change in response gain [12].

Here we use fMRI methods to show that attentional modulation in the visual cortex is not confined to modulating the magnitude of the response to a visual stimulus. We show that directing attention to one location causes a decrease in activity at all other locations throughout the visual field. This suggests that attention modulates the spontaneous (resting) discharge of cortical neurons. Focusing attention causes suppression of activity over most of the visual field, ameliorated only by enhanced responsiveness at one restricted location.

MATERIALS AND METHODS

Our methods have been described fully elsewhere [13] and are described only briefly here.

MRI: Imaging was performed using a 1.5 T Siemens Magnetom (Vision) scanner, which has 25 mT/m gradients with a 0.3 ms rise time. The subject was positioned with the head in an RF receive–transmit head coil. The head was stabilized by means of a vacuum cap. Functional images were twelve contiguous T2*-weighted EPI slices (TE = 66 ms, TR = 3 s, flip angle = 90°, 128 × 128 matrix, voxel size = 2 × 2 × 4 mm), oriented approximately parallel to the calcarine sulcus and encompassing the occipital and posterior parietal lobes. Each experimental run consisted of 54 volume acquisitions, each 3 s in length, giving a total run time of 162 s. The run was divided into six blocks of 27 s (see Fig. 3a). In the first block the screen was uniform except for the fixation spot. In the second block a visual stimulus was presented continuously for 27 s with the fixation spot superimposed. These two blocks were then repeated three times. The mean luminances of the patterned and unpatterned periods were the same. At the end of each experimental session, a whole-head anatomical scan was acquired (sagittal T1-weighted images with 1 mm^3 voxels).

The functional data were first motion-corrected using imreg, part of the AFNI package [14]. Thereafter the data were analyzed using our own in-house software, BrainTools (http://www.pc.rhbnc.ac.uk/vision/BrainTools.html). Spatial smoothing was applied (Gaussian kernel with FWHM = 4 mm) and the timecourse of each voxel was corrected for any linear trend artefact. A correlation analysis [15,16] was then performed. This involved correlating the temporal activity profile of each voxel with an ideal response profile consisting of a squarewave representing the on/off stimulus cycle which is retarded in phase by the expected haemodynamic delay of 6 s and smoothed with a Gaussian kernel (s.d. = 3 s). The timecourse of each voxel was smoothed with the same Gaussian, in order to improve the signal-to-noise ratio. Cortical activation was then estimated for each 162 s run as the stimulus-correlated (or correlation-weighted) variance, which is the product of the correlation coefficient for the voxel and its variance calculated over the entire run [15].

Activation levels could be visualised by superimposing them on anatomical slices in any arbitrary plane. To aid visualization, the grey matter of the occipital lobe was unfolded and flattened in simulation to yield a two-dimensional map of the cortical surface, using algorithms developed by Engel and colleagues [17,18]. In each hemisphere, a circular patch of grey matter centred midway along the fundus of the calcarine sulcus was flattened. Functional activations for each voxel in the corresponding 3D acquisition volume were then overlaid as colours. To quantify the results, regions of interest (ROIs) were defined within the 2D surface and the mean activation of voxels within the ROI calculated. No thresholding was applied; means reflected all voxels in the ROI whether significantly correlated or not.

Visual stimuli: All visual stimuli were generated by an Apple Macintosh 7600 computer and displayed by an LCD projector (Panasonic LT562E). A rear projection screen was mounted across the open rear end of the bore of the scanner. The screen was viewed by the subject via a mirror mounted on the headcoil, above the eyes. This arrangement gave a sharp, approximately circular image (diameter 30°, mean luminance 35 cd m^{-2}). Visual stimulus presentation was initiated by a synchronization pulse provided by the computer controlling the scanner.

The stimuli were one-dimensional sinusoidal luminance modulations that drifted continuously in one direction or, in a few cases (Fig. 2d), were sinusoidally counterphased. Various spatial and temporal frequencies were used. The gratings were spatially windowed by a static 2D Gaussian contrast envelope (s.d. = 3.7°) to give a circular stimulus patch. The area surrounding the grating was unpatterned and its luminance was the same as the mean luminance of the grating. A central fixation spot (0.25° diameter) was continuously present. In some conditions, a task designed to maintain attention at the fovea was used. In these conditions the central fixation spot randomly changed colour at a rate of 3.3 Hz. Eight easily discriminated colours were defined. Every 305 ms, one colour was selected at random and applied to the fixation spot. The subject was instructed to fixate and attend to the coloured spot and to count the occurrences of one particular colour, say green. The total number of occurrences was reported at the end of the run. The colour update rate chosen was sufficiently high to occupy attention fully. Subjects typically made estimates that varied within ±5% of the correct figure.

Procedure: In Experiment 1, the attention task was not employed. The fixation spot was continuously yellow and the subject was simply instructed to fixate. Periods in which the grating was absent alternated with periods when it was present. Each period lasted 27 s. In Experiment 2, gratings were again presented and the attention task was applied in four ways in four separate conditions. These are shown in Fig. 3a. In each time phase (A, no grating; B, grating present) the colour of the central fixation spot was either continuously yellow (unshaded time periods) or changed colour every 305 ms (shaded periods). The grating was always a high-contrast, 0.4 c/deg sine grating drifting at 5 Hz. In one condition the subject performed the task throughout the three-minute run. In two further conditions, the fixation spot colour flickered only during one stimulus phase, either the on or the off phase of the grating stimulus, and was continuously yellow in the other phase. The subject was instructed to fixate throughout and to perform the task whenever the colour was seen to be changing. In the fourth (control) condition, the spot was yellow throughout the run, as it was in Experiment 1. In Experiment 3, two of the attentional conditions of Experiment 2 were applied, but this time in the absence of grating stimuli. In Experiments 2 and 3, each subject performed each condition twice and the results were averaged. In all experiments, the different conditions were conducted in random order within a single session.

Subjects: A total of eight subjects participated, including two of the authors (AS and KS) and six volunteers who were paid for their time and were unaware of the purpose of the experiments. Most subjects participated in more than one experiment.

RESULTS

Experiment 1: Several retinotopically organized visual areas have been identified in human occipital cortex,

including V1, V2, V3 and V4 [17,19,20]. Within these areas, each location in the visual field is associated with a specific location in the cortex. It is, therefore, possible independently to study regions of cortex associated with different spatial locations. By simulated flattening of the grey matter of the occipital cortex, such regions are revealed in an orderly, two-dimensional map of visual space. An example is shown in Fig. 1a,b.

Figure 1c shows, for one subject, that a simple, centrally fixated drifting grating stimulus causes a coherent region of activation (shown in red) around the foveal representation, as expected. In those parts of cortical areas V1 and V2 that represent unstimulated regions of the visual field beyond the edge of the stimulus, activation is reduced (shown as blue). That is, these peripheral regions of cortex are more active when the central stimulus is absent than when it is present. This is true over a large area, extending to the edge of the flattened region of cortex, which represents almost the entire visual field. Figure 2a shows the same phenomenon in the form of a graph. The data are from the hemisphere shown in Fig. 1c. Figure 2b,c show, for the same subject, the temporal profile of activation in representative voxels in the foveal and peripheral parts, respectively, of the visual field. In the periphery (Fig. 2c), activity is negatively correlated with the on/off cycle of the grating. Figure 2d shows (for a different subject) that suppression of activity in the peripheral visual field occurs for a wide variety of different grating stimuli.

Similar results were obtained for five other subjects, but with considerable variability in the magnitude of the suppression in the periphery. On average, the suppression is of the order of 20% of the excitation caused by the grating stimulus in the centre; in the example illustrated in Fig. 2a the effect is even more pronounced, but in some other hemispheres it is less so.

One interpretation of this phenomenon is that attentional resources are diverted away from unstimulated regions when a stimulus is presented. Although there were no instructions to attend (only to fixate), a prominent, moving stimulus presented in an otherwise unpatterned field tends to draw the subject's attention. When the stimulus is absent, attention will be more diffuse and neurons representing the peripheral visual field may be more active because peripheral parts of the field receive greater attention. If this interpretation is correct, attentional modulation does not, in this instance, take the form of a change in gain of excitatory responses to a stimulus at one location. Instead, it reflects an altered level of baseline or spontaneous activity, in the absence of visual stimulation, all over the field of view. However, other interpretations are possible, for example long-range inhibition of unstimulated cortex by active neurons in the stimulated region, or even diversion of oxygenated blood from regions of low demand to regions of high demand.

Experiment 2: To test the attentional interpretation of the result obtained in Experiment 1 and to explore it in more detail, we introduced a task designed to control the spatial locus of attention (see Materials and Methods). Four subjects were studied. The results for one hemisphere in each of three subjects are shown in Fig. 3b–d. Similar results were obtained in the other hemisphere of each

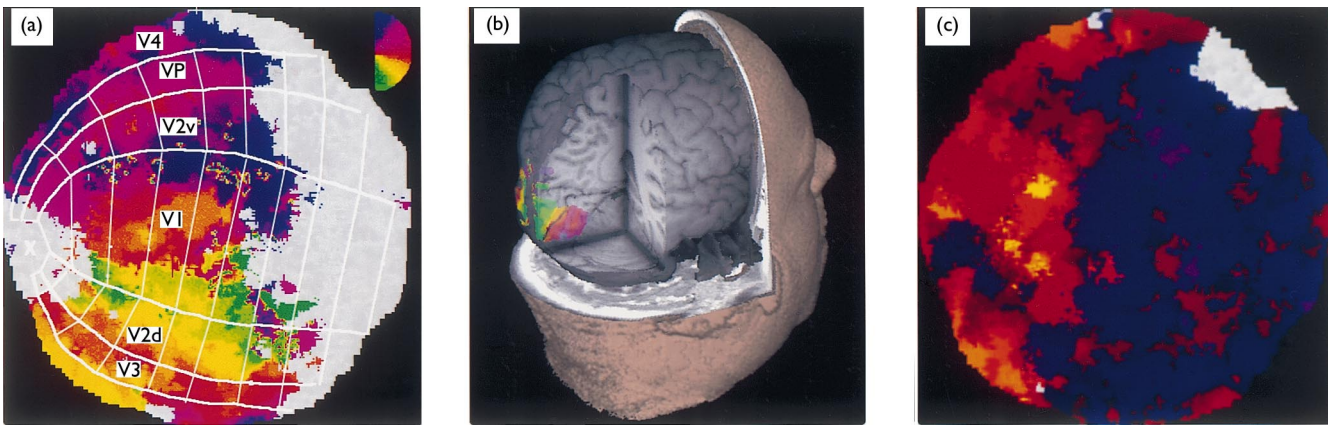

Fig. 1. The effect of a visual stimulus in the central visual field on neural activity in parts of the visual cortex representing unstimulated visual field locations. (**a**) A computer-generated representation of the occipital cortex of the left hemisphere of a healthy human subject (one of the authors), flattened to produce a 2D map of the grey matter. Every point in the map corresponds to a unique point in the original 3D brain. Superimposed on the 2D map are colours representing visual field location, obtained by measuring the temporal phase of the fMRI (BOLD) response elicited by a wedge-shaped flickering checkerboard pattern which slowly rotates about a central fixation point [13–15]. The key (top right) shows the relationship between colour and visual field location. The boundaries between visual areas V1, V2 and V3/VP are given by the locations at which the smooth shift in phase (colour) reverses direction. The foveal representation is marked on the left; eccentricity increases with distance from this point. The boundaries between areas are drawn by eye and each region is divided into sections of approximate width 1 cm along the cortical surface. (**b**) A 3D-rendered view of the same brain, cut away to reveal the visual cortex. Some of the colours representing visual field location in (a) are shown in their true positions in the 3D brain. Colours corresponding to the lower and upper visual fields are visible above and below the calcarine sulcus, respectively. The calcarine has been artificially darkened to make it clearer. (**c**) The flattened map of occipital cortex in (a) is shown again, this time with responses to a centrally fixated grating stimulus superimposed. The grating had a spatial frequency of 2 c/deg and drifted at a rate of 1 deg/sec (2 Hz). The colours now reflect cortical activation (correlation-weighted variance). Red and yellow indicate that activation was positively correlated with the on/off stimulus cycle; blue and purple indicate negative correlations, zero correlation is indicated by black. To the left is a large area of activation corresponding to the location of the stimulus. To the right is a large, predominantly blue area representing parts of the visual field beyond the edge of the stimulus. The negative correlation means that this area is less active when the stimulus is present than when it is absent.

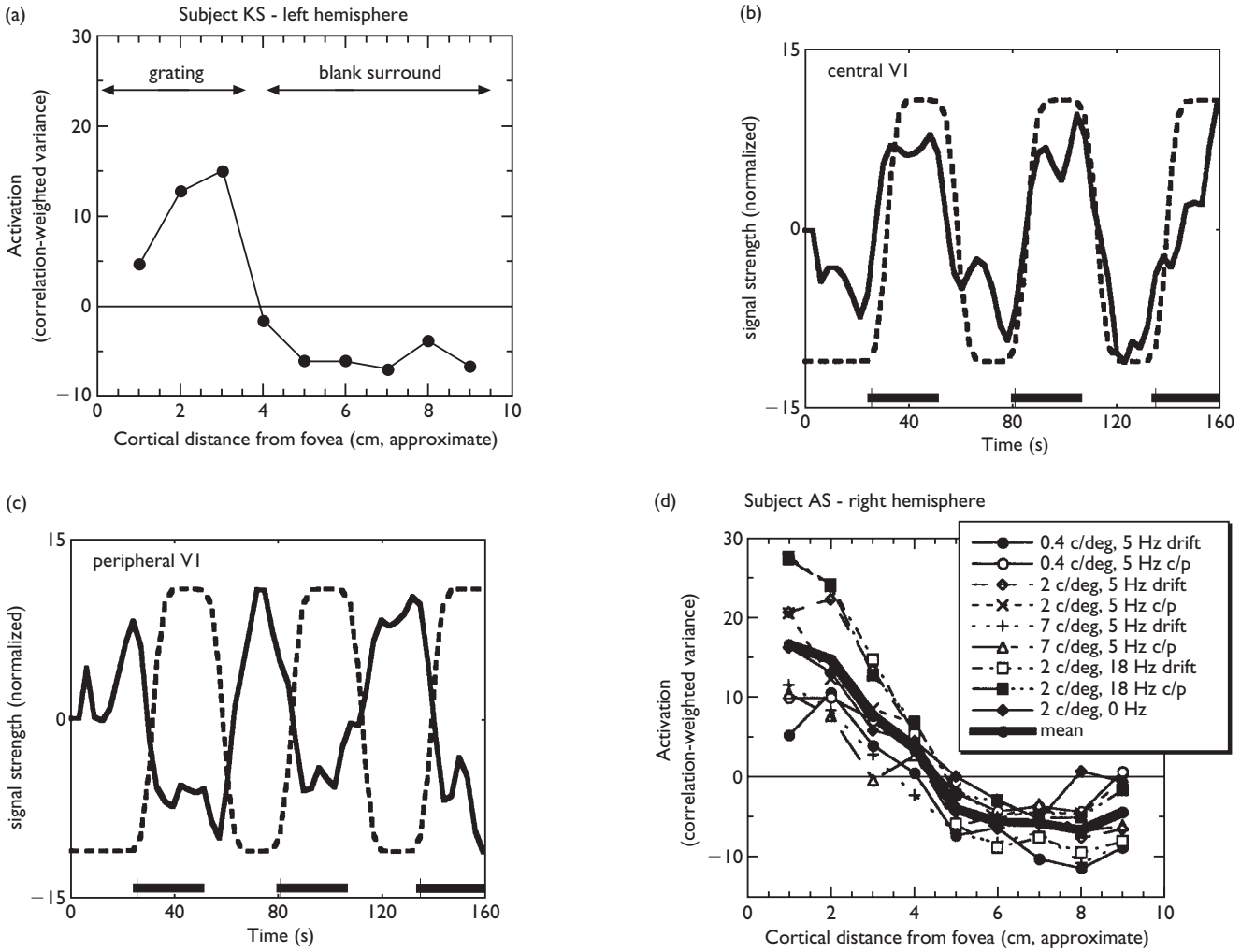

Fig. 2. Graphs quantifying the result shown in Fig. 1c. (**a**) Activation caused by a grating stimulus is shown as a function of eccentricity. Area V1, as defined in Fig. 1a, was divided into regions corresponding to strips of cortex ~1 cm in width. For each strip, the average activation of all voxels in the 3D brain whose locations fell within the strip was calculated and plotted as one point on the graph. The negative activation at peripheral locations is clear and extends to the edge of the measured region. (**b**,**c**) The timecourse of activity. Periods when a grating was present are marked with a solid bar above the abscissa. Each plot shows the observed activity (continuous line) averaged over a 6×6 array of voxels in one slice, together with the ideal, or expected, response to a stimulus (a smoothed and phase-retarded squarewave, dotted line). In (b) the measured voxels are in the central visual field representation of V1 (near the occipital pole) and the activity is positively correlated with the ideal waveform. In (c) the voxels are slightly more anterior, in the peripheral visual field representation, and the correlation is clearly negative. The two functions were recorded simultaneously in response to the same visual stimulation. (**d**) A similar plot to (a), based on fMRI data from a different subject, showing that the phenomenon is robust and occurs for a wide range of grating spatial frequencies and temporal frequencies and for both drifting and counterphasing (c/p) gratings.

subject and in both hemispheres of the fourth subject. In the control condition (labelled ''neither'' in the key) the result is the same as in Experiment 1. In the region of cortex corresponding to the stimulus, activity is positively correlated with the on/off stimulus cycle. Elsewhere, the correlation is negative (less activity when the stimulus is present than when absent). When the subject is forced by the colour task to attend closely to the fixation spot throughout the run (the A + B condition), positive activation in the central visual field is still present but the negatively correlated activity in peripheral regions is no longer seen. Instead, stimulus-related activation in the periphery is close to zero. This is consistent with the notion that negative activation in the periphery arises from an attention-related baseline change and has little to do with the grating stimulus *per se*. When attention is held constant, no baseline change occurs in the peripheral field representation as the central stimulus appears and disappears, so the correlation between activity and the stimulus cycle is zero.

If the colour task is performed only when the grating is absent (the A only condition), there is again no negative activation in the periphery. This is presumably because attention is focused centrally during stimulus-absent phases because of the colour task, as well as during grating phases because of the grating. In fact activation in the periphery tends to be positive (the effect is reversed), presumably because the colour task focuses attention more

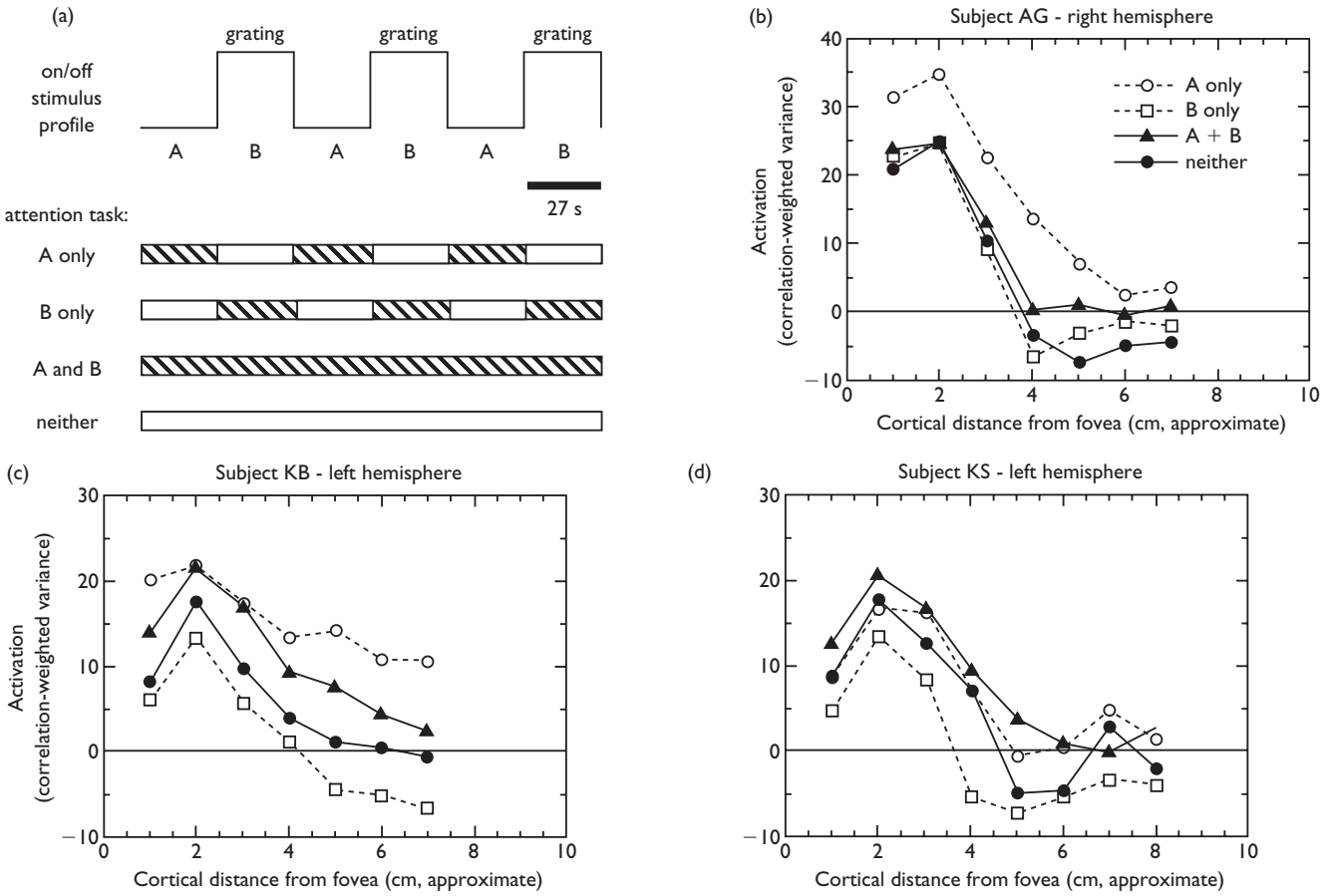

Fig. 3. (**a**) The stimulus profile and the various attention conditions used in Experiment 2. (**b**–**d**) Results obtained using the four attention tasks, for each of three subjects. Activation is plotted as a function of distance from the fovea, as in Fig. 2a. Negative activation is seen in the peripheral visual field representation, but only in the "B only" and "neither" conditions. Subject KS is one of the authors, the other two are paid volunteers who were unaware of the purpose of the experiment.

effectively than the mere presence of a grating with no task.

If the task is performed only when the grating is present (B only), the negative activation re-appears. If anything, it is greater than in the baseline condition. In this condition, the attention task only serves to reinforce the spontaneous attentional modulation, ensuring central attention with grating present while leaving attention diffuse with grating absent.

Thus all four conditions give results that fit the hypothesis that focal visual attention causes a change in baseline neural activity levels in unstimulated, as well as stimulated, regions of the visual cortex. Other explanations (long-range inhibition, blood stealing) predict that the phenomenon should occur in all conditions.

Experiment 3: In this experiment, we removed the grating stimulus altogether. Again, four subjects were used. Activations were measured in two conditions. One was the B only condition of the previous experiment (see Fig. 3a), now used as a control, in which subjects performed the attention task only when the grating was present. This condition was chosen because it gives the most pronounced attention-related baseline change in the periphery (Fig. 3b–d). The other condition was identical except that the grating was removed and the only stimulus was the fixation spot itself. Figure 4 shows the result for one subject. Similar results were obtained in the other subjects. With the grating present, the familiar result is repeated; positive activation is seen in the central region of the field and negative activation in the periphery. With the grating absent, the entire visual field (apart from the foveal representation) shows negative activation. That is, a small flickering spot, coupled with a simple task designed to focus attention, lowers the level of baseline activity over the entire visual cortex.

DISCUSSION

We have presented a striking demonstration that baseline neural activity levels are reduced across the entire non-foveal visual cortex (several square centimetres of grey matter containing many millions of neurons) when previously diffuse attention becomes focused in the fovea. This result suggests that focal attention involves changes in baseline activity across the entire visual cortex, not just

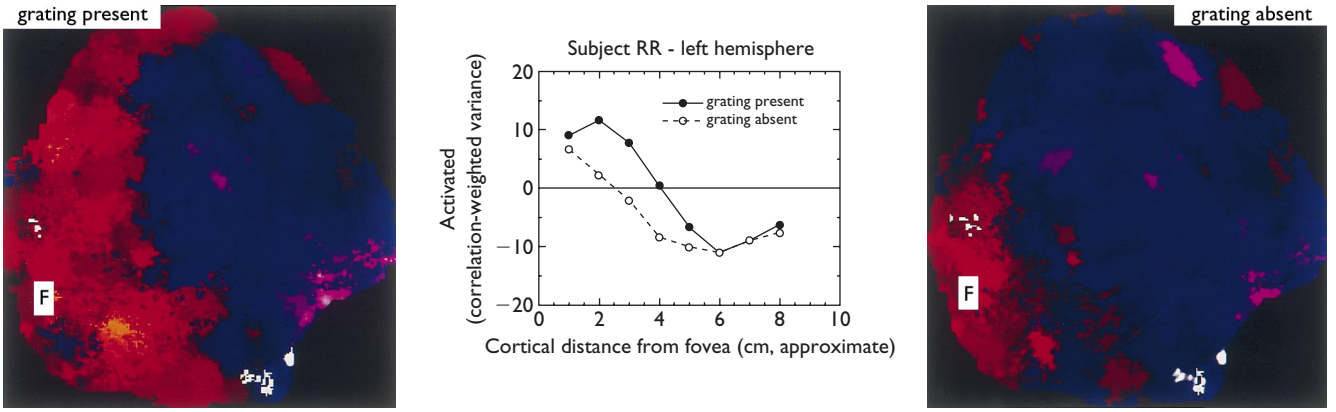

Fig. 4. The results of the Experiment 3, in which no visual stimulus, other than the fixation spot, was presented. (Left) A flattened representation of the occipital cortex of the left hemisphere of one subject. The estimated position of the fovea is marked with an F. Superimposed as a coloured overlay is the activation elicited by a grating stimulus (0.4 c/d, 5 Hz drift), presented while the subject performed attention task B only of Fig. 3a. The appearance is similar to Fig. 1c, with positive activation in the central visual field and negative activation in the periphery. (Right) Activation in the same hemisphere, under the same attention task, but without presentation of a grating. Activation is confined to a small region around the fovea, where the coloured spot is present. The rest of the visual field shows negative activation. (Centre) A plot showing the results quantitatively. The cortex was divided into strips as previously described. Strong negative activation is seen in the periphery in both conditions.

enhancement of response gain in regions where attended stimuli are present.

There are two distinct aspects to this conclusion: (i) that attention involves changes in baseline activity rather than (or as well as) response gain and (ii) that these changes involve the entire visual field. One other fMRI study has provided evidence consistent with an account of focal visual attention in terms of baseline changes, although only at the attended location. Kastner *et al.* [21] showed that the expectation of stimulus presentation at an attended location is sufficient to increase activation levels at that location. In that study, as much as 50% of the activation elicited by actual presentation of a stimulus was in evidence during the expectation period immediately prior to stimulus onset. It is difficult to know the cause of this large anticipatory effect (whether, for example, the subject was imagining a stimulus and the activation was related to imagery), but the interpretation favoured by the authors is an attention-related change in the baseline activity of neurons with receptive fields in the attended region. In Kastner's study, activity at non-attended locations was not examined.

If attention reflects a baseline change then this should be apparent in neurophysiological studies of single neurons in awake primates performing attentional tasks. Not only should a cell's excitatory response to a stimulus in its receptive field increase when the animal attends to the stimulus, as is well documented, but also changes in spontaneous discharge should occur, in the absence of visual stimulation, as the animal shifts the locus of its attention. In fact the neurophysiological evidence on this point is sparse and contradictory. Luck *et al.* [22] report attention-related changes in spontaneous activity in neurons of macaque V2 and V4. In this study, spontaneous activity increased when the animal attended a location within the cell's receptive field and decreased when it attended elsewhere. However, in a more detailed quantitative analysis, McAdams [12] found that baseline activity levels in V1 and V4 are unaffected by shifts in spatial attention. It is not clear how this discrepancy is best explained. Perhaps, again, the expectation, memory and imagery components of the task need to be considered alongside attentional demands.

There is considerable behavioural evidence for suppression of non-attended objects (distractors) [23]. There is also some suggestion from reaction time studies that unstimulated parts of the field can be suppressed [24], although the evidence on this point is not clearcut. Our work represents the first clear demonstration of widespread, as opposed to distractor-related, suppression.

Figure 5 illustrates three possible models of the neural modulation associated with focal attention. In the first, the response to a stimulus at the attended location is enhanced and activity at other locations is unaffected. The enhancement reflects a multiplicative gain change, as suggested by physiological studies. This model is prevalent but is incompatible with our results. The second model assumes that the gain of the response to a stimulus at the attended location is unchanged. The observed modulation is entirely due to a baseline change which is positive at the attended location and negative elsewhere. This model is consistent with our data, but it predicts that responses to all attended stimuli will be elevated by a fixed increment, which is not in accord with physiological data [25]. The third model combines the first two. The baseline varies in a position-dependent manner and the excitatory response that is added to the baseline has attention-dependent gain. This model is compatible both with the existing literature concerning response gain changes and with our new findings.

CONCLUSION

Focal attention modulates activity at all spatial locations, not just at the attended location.

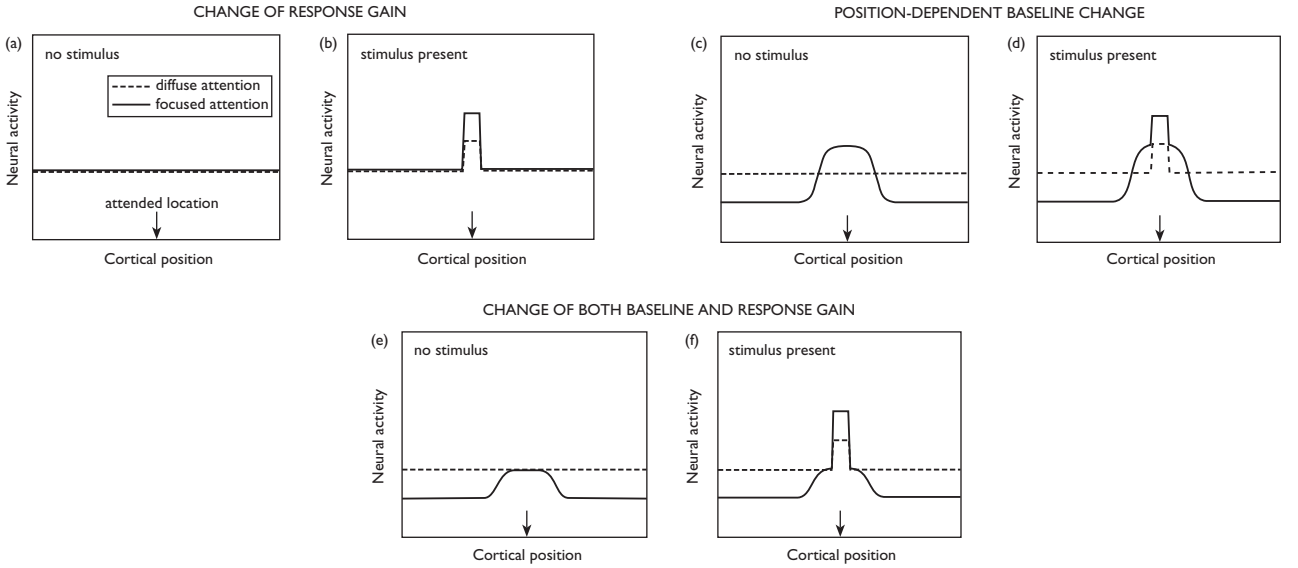

Fig. 5. Three possible models of attentional modulation. In each plot, neural activity (summed across many neurons) is shown as a function of location in the visual cortex, which corresponds directly to position in the visual image. The three models all produce the same response to a stimulus, both when it is attended and when it is not. (**a**,**b**) The conventional approach, in which attention to a particular location in the visual field leads to an increase in the gain (shown here as a doubling) of the response to a stimulus at that location. Activity levels at unattended, unstimulated regions are unchanged. (**c**,**d**) An alternative approach, in which baseline activity levels decrease at all locations other than the attended location when attention becomes spatially focused, as suggested by our results. The gain of the response to a stimulus at the attended location is unaffected. The model requires an increase in baseline at the attended location, as well as a decrease elsewhere, in order to produce an increased response. (**e**,**f**) A combination model, in which both position-dependent baseline changes and stimulus-related gain changes occur. In this case, it is not necessary to invoke an increase in baseline activity at the attended location as well as a decrease elsewhere.

REFERENCES

1. Gandhi S, Heeger D and Boynton G. *Proc Natl Acad Sci USA* **96**, 3314–3319 (1999).
2. Watanabe T, Hamer AM, Miyauchi S *et al. T Proc Natl Acad Sci USA* **95**, 11489–11492 (1998).
3. Brefczynski JA and DeYoe EA. *Nature Neurosci* **2**, 370–374 (1999).
4. Somers D, Dale A, Seiffert A and Tootell R. *Proc Natl Acad Sci USA* **96**, 1663–1668 (1999).
5. Martinez A, Anllo Vento L, Sereno MI *et al. Nature Neurosci* **2**, 364–369 (1999).
6. Moran J and Desimone R. *Science* **229**, 782–784 (1985).
7. Haenny PE and Schiller PH. *Exp Brain Res* **69**, 225–244 (1988).
8. Motter BC. *J Neurophysiol* **70**, 909–919 (1993).
9. Steinmetz MA, Connor CE, Constantinidis C and McLaughlin JR. *J Neurophysiol* **72**, 1020–1023 (1994).
10. Treue S and Maunsell JHR. *Nature* **382**, 539–541 (1996).
11. Connor CE, Preddie DC, Gallant JL and Van Essen DC. *J Neurosci* **17**, 3201–3214 (1997).
12. McAdams C and Maunsell J. *J Neurosci* **19**, 431–441 (1999).
13. Smith AT, Greenlee MW, Singh KD *et al. J Neurosci* **18**, 3816–3830 (1998).
14. Cox R. *Comp Biomed Res* **29**, 162–173 (1996).
15. Bandettini PA, Jesmanowicz A, Wong EC and Hyde JS. *Magn Reson Med* **30**, 161–173 (1993).
16. Friston K, Holmes A, Poline J *et al. NeuroImage* **2**, 45–53 (1995).
17. Engel SA, Glover GH and Wandell BA. *Cerebr Cortex* **7**, 181–192 (1997).
18. Teo P, Sapiro G and Wandell B. *Trans Med Imaging* **16**, 852–863 (1997).
19. Sereno MI, Dale AM, Reppas JB *et al. Science* **268**, 889–893 (1995).
20. Tootell RBH and Taylor JB. *Cerebr Cortex* **1**, 39–55 (1995).
21. Kastner S, Pinsk MA, De Weerd P *et al. Neuron* **22**, 751–761 (1999).
22. Luck SJ, Chelazzi L, Hillyard SA and Desimone R. *J Neurophysiol* **77**, 24–42 (1997).
23. Tipper SP. *Q J Exp Psychol* **37A**, 571–590 (1985).
24 Hughes, HC and Zimba LD. *Neuropsychologia* **25**, 5–18 (1987).
25. Treue S and Trujillo JCM. *Nature* **399**, 575–579 (1999).