Attention modulates responses in the human lateral geniculate nucleus

Daniel H. O'Connor, Miki M. Fukui, Mark A. Pinsk and Sabine Kastner

Department of Psychology, Center for the Study of Brain, Mind, and Behavior, Princeton University, Green Hall, Princeton, New Jersey 08544, USA
Correspondence should be addressed to S.K. (skastner@princeton.edu)

Published online 15 October 2002; doi:10.1038/nn957

Attentional mechanisms are important for selecting relevant information and filtering out irrelevant information from cluttered visual scenes. Selective attention has previously been shown to affect neural activity in both extrastriate and striate visual cortex. Here, evidence from functional brain imaging shows that attentional response modulation is not confined to cortical processing, but can occur as early as the thalamic level. We found that attention modulated neural activity in the human lateral geniculate nucleus (LGN) in several ways: it enhanced neural responses to attended stimuli, attenuated responses to ignored stimuli and increased baseline activity in the absence of visual stimulation. The LGN, traditionally viewed as the gateway to visual cortex, may also serve as a 'gatekeeper' in controlling attentional response gain.

Natural visual scenes are typically cluttered with many different objects that cannot all be processed simultaneously. Attentional mechanisms are needed to filter incoming stimuli for selective processing. Converging evidence from single-cell recording studies in monkeys and functional brain mapping studies in humans shows that selective attention modulates neural activity in the visual system in multiple ways[1–4]. At which stage of the visual pathway does such modulation first occur? Attentional response modulation was originally found in extrastriate but not in striate cortex[5]. Recent evidence has shown that neural activity in striate cortex can also be affected, depending on certain task-related factors, such as the attentional demands or the need to integrate contextual information[6–11]. Little is known, however, about the role of earlier, subcortical structures in attentional processing.

The lateral geniculate nucleus (LGN) is the thalamic component in the retinocortical projection and has traditionally been viewed as the gateway for sensory information to enter the visual cortex[12,13]. In addition to retinal afferents, the LGN receives input from other sources including the thalamic reticular nucleus, striate cortex and brainstem. The LGN therefore represents the first stage at which cortical top-down feedback signals could affect visual processing. The functional role of these top-down inputs to the LGN is not well understood[14]. Here we used functional magnetic resonance imaging (fMRI) to investigate attentional response modulation in the human LGN.

At the cortical level, selective attention affects visual processing in at least three different ways. First, neural responses to visual stimuli are greater when those stimuli are attended versus when they are ignored (attentional enhancement)[5,15]. Second, neural responses to ignored stimuli are attenuated depending on the load of attentional resources engaged elsewhere (attentional suppression)[16]. And third, directing attention to a location in the absence of visual stimulation and in anticipation of stimulus onset increases neural baseline activity (attention-related baseline increases)[17–20]. We investigated these effects of selective attention in the LGN in a series of three experiments.

It is difficult to obtain fMRI signals from subcortical nuclei, owing to their small sizes and deep locations[21,22]. We therefore optimized our experimental designs to evoke maximal responses in the human LGN. In different conditions, subjects attended to, ignored, or anticipated the onset of flickering checkerboard stimuli that were used in all experiments (**Fig. 1a** and **b**). LGN activity was enhanced when subjects attended to the stimulus, and it was suppressed when they ignored it. Further, directed attention to a spatial location in anticipation of the stimulus onset led to an increase of baseline activity in the LGN. We found qualitatively similar attention effects in visual cortex, confirming previous results[5,15–20]. These findings challenge the classical notion that selective attention is confined to cortical processing[23] and suggest an important role for the LGN in attentional control.

Results

Flickering checkerboard stimuli of high- or low-contrast were presented in alternation to the left or right visual hemifield while subjects maintained fixation; this activated the right or left LGN, respectively (**Fig. 1**). The locations of the functional activations were consistent within subjects across experiments and in close correspondence to the anatomical locations of the LGN, as determined on high-resolution anatomical images in each subject (**Table 1**). The mean activated LGN volume was 258 mm^3 (± 53 s.e.m.) in the right LGN and 239 mm^3 (± 35 s.e.m.) in the left LGN, averaged across all subjects and experiments, confirming previous studies[22] that used similar scanning parameters on a 4-tesla scanner. In visual cortex, areas V1, V2, V3/VP, V4, TEO, V3A and MT/MST were activated. Delineations of areas were determined on the basis of retinotopic mapping[24,25].

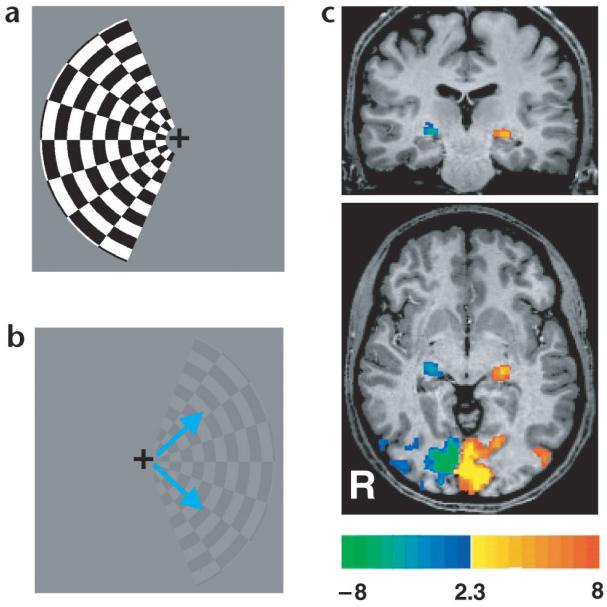

Fig. 1. Visual stimuli and experimental design. (**a**, **b**) High- or low-contrast checkerboard stimuli were presented to the left or right of a central fixation point (+). In experiments 1, 3 and 4, subjects covertly directed attention to a checkerboard arc (**b**, blue arrows) and detected randomly occurring luminance changes along that arc. The detection of luminance changes in low contrast and high-contrast checkerboard stimuli was not matched for task difficulty. (**c**) Axial slice showing activations of the right (green-blue) and left (yellow-orange) LGN and visual cortex evoked by the checkerboard stimuli. The coronal plane shows that activations in the thalamus were restricted to the LGN; no activations within the pulvinar were obtained. Scale indicates *Z*-score values of activations in colored regions. R, right hemisphere.

Enhancement of responses to attended stimuli

To investigate attentional response enhancement in the LGN (experiment 1), checkerboard stimuli of high- or low-contrast were presented to the left or right hemifield while subjects directed attention to the stimulus (attended condition) or away from the stimulus (unattended condition) in separate runs. In the unattended condition, subjects counted letters at fixation, which directed attention away from the stimulus. The letter-counting task ensured proper fixation and effectively prevented subjects from covertly attending to the checkerboard stimuli[26]. In the attended condition, subjects were instructed to covertly direct attention to the checkerboard stimulus and to detect luminance changes that occurred at random times and at 10° eccentricity (**Fig. 1b**). Behavioral performance was 59 ± 1% correct (mean ± s.e.m.) for letters and 76 ± 29% for luminance changes.

In our statistical model, stimulation of the left visual hemifield was contrasted with stimulation of the right visual hemifield. The analysis was thus restricted to voxels activated by the peripheral checkerboard stimuli and excluded foveal stimulus representations. Relative to the unattended condition, the mean fMRI signals evoked by the high-contrast stimulus increased from 0.74% to 1.06% in the attended condition (**Fig. 2a**). Similarly, activity evoked by the low-contrast stimulus increased from 0.26% to 0.41% (main effect of attention, $t_3 = 6.3$, $P < 0.01$). These results suggest that attention facilitates visual processing in the LGN by enhancing neural responses to an attended stimulus relative to those evoked by the same stimulus when ignored.

Suppression of responses to ignored stimuli

To investigate attentional load–dependent suppression in the LGN (experiment 2), high- and low-contrast checkerboard stimuli were presented to the left or right hemifield while subjects performed either an easy or difficult attention task at fixation and ignored the peripheral checkerboard stimuli. During the easy attention task, subjects counted brief, infrequent color changes of the fixation cross. During the hard attention task, subjects counted letters at fixation as in experiment 2. Behavioral performance was 99 ± 1% correct in the easy attention task and 54 ± 7% in the hard attention task ($t_3 = 7.98$, $P < 0.01$), reflecting the differences in attentional load.

Relative to the easy task condition, mean fMRI signals evoked by the high-contrast stimulus decreased from 0.98% to 0.85% and those evoked by the low-contrast stimulus decreased from 0.47% to 0.24% in the hard task condition (main effect of task, $t_3 = 2.8$, $P < 0.05$; **Fig. 2b**). These fMRI signals reflected activity evoked only by the peripheral checkerboard stimuli when processed under conditions of different attentional load; they were not confounded with activity evoked by the foveal stimuli. This finding suggests that neural activity evoked by ignored stimuli in the LGN is attenuated as a function of the load of attentional resources engaged elsewhere. According to accounts of attentional load[27], the degree to which ignored stimuli are processed is determined by the amount of spare attentional capacity. In our design, the low-load task presumably left a greater amount of spare attentional capacity than did the high-load task, which may account for the observed attenuation of responses evoked by the ignored stimuli during the high-load condition.

Attention-related increases of baseline activity

To investigate attention-related baseline increases in the LGN (experiment 3), subjects were cued to covertly direct attention to the periphery of the left or right visual hemifield and to expect the onset of the stimulus. After the expectation period, during which subjects attended to the periphery without receiving visual input, a high-contrast checkerboard was presented at the expected location. During the attended presentations, subjects counted the occurrence of luminance changes, as in experiment 1. During the expectation period, fMRI signals increased by 0.3% relative to the preceding blank period in which subjects were fixating but not directing attention to the periphery (main effect of expectation versus blank period: $t_3 = 6.4$, $P < 0.01$). This elevation of baseline activity was followed by a further response increase evoked by the visual stimuli (**Fig. 2c**). This finding suggests that neural activity in the LGN can be affected by attentional signals, even in the absence of visual stimulation.

Attention effects in the LGN and in visual cortex

Qualitatively similar effects of attention were found at the cortical level, as shown in the fMRI time series averaged across all activated areas in visual cortex (**Fig. 2d–f**) (experiment 1: main effect of attention, $t_3 = 6.2$, $P < 0.01$; experiment 2: main effect of task, $t_3 = 4.5$, $P < 0.05$; experiment 3: main effect of expectation versus blank period, $t_3 = 12.5$, $P < 0.001$). We compared the attentional effects found at the thalamic and cortical levels by normalizing the mean fMRI signal evoked in the LGN and in each activated cortical area. Index values that quantified the magnitude of attentional effects were derived for each experiment

Table 1. LGN Talairach coordinates and estimated volumes

Experiment	Subject	Right			Left			Volume (mm^3)	
		x	y	z	x	y	z	R	L
Anatomical	1	22	–21	–5	–23	–23	–3		
	2	23	–22	–6	–23	–22	–3		
	3	25	–22	–3	–24	–21	–6		
	4	21	–21	–6	–22	–21	–5		
	Average	**23**	**–22**	**–5**	**–23**	**–22**	**–4**		
Enhancement	1	23	–20	–3	–20	–23	–3	270	270
	2	28	–19	0	–23	–19	–4	324	108
	3	24	–19	–5	–19	–26	–5	216	81
	4	19	–22	–3	–22	–24	–2	135	162
	Average	**24**	**–20**	**–3**	**–21**	**–23**	**–4**	**236**	**155**
Suppression	1	20	–25	–3	–24	–26	–4	189	270
	2	22	–16	–6	–26	–18	–6	95	189
	3	22	–20	1	–24	–23	–3	216	189
	4	19	–23	–5	–22	–20	–3	189	135
	Average	**21**	**–21**	**–3**	**–24**	**–22**	**–4**	**189**	**135**
Baseline	1	25	–18	0	–23	–20	–3	270	729
	2	24	–20	–4	–18	–23	–3	243	189
	3	23	–19	–6	–24	–15	–9	351	189
	4	16	–25	–2	–22	–22	0	270	324
	Average	**22**	**–21**	**–3**	**–22**	**–20**	**–4**	**284**	**356**

R, right; L, left.

and for each area. For all indices, larger values indicate larger effects of attention. This analysis revealed two important results (**Fig. 3a–c**). First, and in accordance with previous findings[10,26,28,29], the attentional effects of enhancement, suppression and baseline elevations increased from earlier to later processing stages along both the ventral and dorsal pathways of visual cortex (**Fig. 3a–c**) (V1 versus V4: main effect of area, $F_{1,3} = 109.1$, $P < 0.01$; V1 versus MT/MST: main effect of area, $F_{1,3} = 308.7$, $P < 0.001$). Second, all three attentional effects tended to be stronger in the LGN than in striate cortex (main effect of area, $F_{1,3} = 13.1$, $P < 0.05$). This finding suggests that attentional response modulation in the LGN may be influenced not only by corticothalamic feedback from striate cortex, but also by sources such as the brainstem or the thalamic reticular nucleus[30].

Spatial specificity of attentional response enhancement

The attention experiments reported thus far were not designed to test for the spatial specificity of attentional modulation. Therefore, it may be argued that the attentional effects found in the LGN and in visual cortex reflect nonspecific attentional states such as arousal, rather than mechanisms of selective attention. General arousal affects the visual system in topographically nonspecific ways, whereas selective attention has been shown to be spatially specific[2,10,19,26]. To test for the spatial specificity of attentional enhancement in our results, we performed a control experiment in which high-contrast checkerboard stimuli were presented simultaneously to both hemifields. Subjects were instructed to direct attention either to the left or right checkerboard and to detect luminance changes as in experiments 1 and 3. The checkerboard stimulus in the respective contralateral hemifield was thus ignored during this time. Behavioral performance was 88 ± 6% correct. In the LGN, mean fMRI signals evoked by the checkerboard stimuli were 1.03% when ignored and 1.24% when attended (**Fig. 4a**; main effect of attention, $t_3 = 3.5$, $P < 0.05$). In visual cortex, mean fMRI signals evoked by the checkerboard stimuli increased from 1.65% to 1.97% during the attended relative to the unattended condition (**Fig. 4b**; main effect of attention, $t_3 = 3.1$, $P < 0.05$). The size of the LGN attentional effects obtained in experiments 1 and 4 was similar, suggesting that the attentional demands of the letter-counting task at fixation and the luminance detection task at the peripheral checkerboard location were comparable. These findings indicate that attentional response enhancement in the LGN and in visual cortex was spatially selective and rule out the possibility that the modulation was due to unspecific attention effects such as arousal.

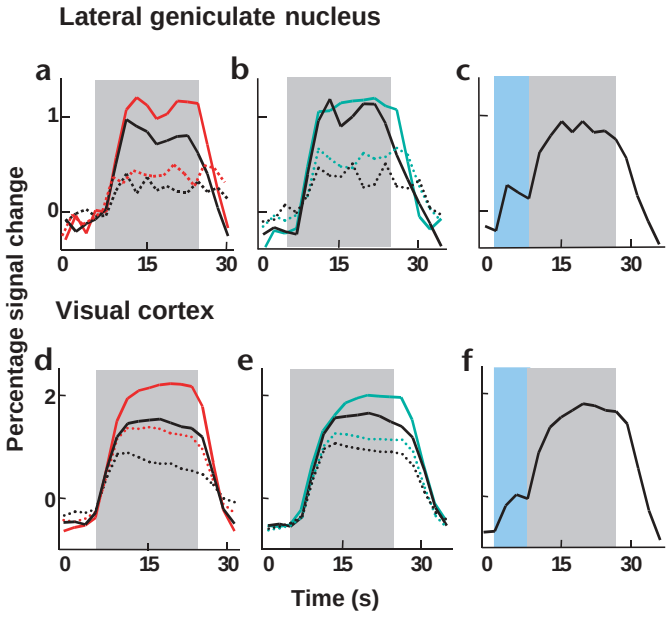

Fig. 2. Time series of fMRI signals in the LGN (**a**–**c**) and visual cortex (**d**–**f**). Group analysis (n = 4). Data from the LGN and visual cortex were combined across left and right hemispheres. Activity in visual cortex was pooled across areas V1, V2, V3/VP, V4, TEO, V3A and MT/MST. (**a**, **d**) Attentional enhancement. During directed attention to the stimuli (red curves), responses to both the high-contrast stimulus (100%, solid curves) and low-contrast stimulus (5%, dashed curves) were enhanced relative to an unattended condition (black curves). (**b**, **e**) Attentional suppression. During an attentionally demanding fixation task (black curves), responses evoked by both the high-contrast stimulus (100%, solid curves) and low-contrast stimulus (10%, dashed curves) were attenuated relative to an easier attention task at fixation (green curves). (**c**, **f**) Baseline increases. Baseline activity was elevated during directed attention to the periphery of the visual hemifield in expectation of the stimulus onset (blue). Gray shades indicate periods of checkerboard presentation.

Eye movement controls

To investigate the possibility that eye movements confounded the attentional response modulation found in the LGN and visual cortex, we carried out eye movement control experiments outside the scanner. The same attention tasks and visual displays were used as during scanning sessions, and behavioral performance was similar to that seen inside the scanner. Separate frequency histograms of horizontal and vertical eye position were derived for the conditions when subjects attended to the left or to the right in covert attention tasks (Fig. 5). The difference in mean horizontal eye position between these conditions was 0.09, 0.03 and 0.48 degrees in experiments 1, 3 and 4, respectively. None of these differences was significant (experiment 1, $t_3 = -0.21$, $P = 0.85$; experiment 3, $t_3 = 0.13$, $P = 0.91$; experiment 4, $t_3 = 1.73$, $P = 0.18$), indicating that there was not a tendency to shift gaze location along with attention.

During covert attention and fixation tasks in all experiments, fixation was well maintained and almost never strayed outside the blank region surrounding the fixation cross (with a radius of 1.5°). Of the horizontal position samples, 99.86% were within this region. For the rare samples that fell outside this region, there was no systematic relation between eye position and direction of attention, indicating that eye movements deviating from fixation were not correlated with task condition.

Although this analysis does not entirely rule out eye movements as a confounding factor because eye movement data was not available from scanning sessions, the results do show that subjects were able to perform all attention tasks over extended periods of time while maintaining fixation, with a standard deviation of eye position of only 0.17°, and that there was no systematic relation between the different attention conditions and eye movements. Assuming that our subjects performed similarly during scanning sessions, it is not likely that the modulation of neural activity in the LGN and visual cortex was significantly confounded by eye movements.

Discussion

Here we report evidence for attentional response modulation in the human LGN. Selective attention modulated neural activi-

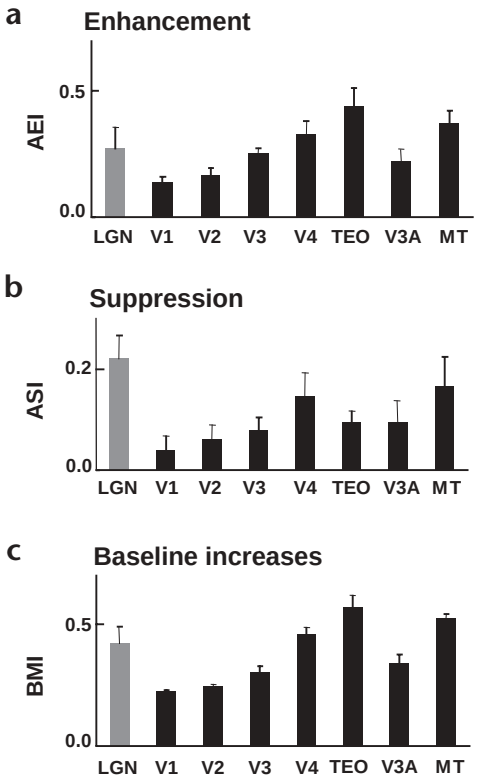

Fig. 3. Attentional response modulation in the LGN and in visual cortical areas V1, V2, V3/VP, V4, TEO, V3A and MT/MST. Attentional effects were quantified and normalized by defining several indices: (**a**) attentional enhancement index (AEI, experiment 1), (**b**) attentional suppression index (ASI, experiment 2) and (**c**) baseline modulation index (BMI, experiment 3). For all indices, larger values indicate larger effects of attention. Index values were computed for each subject based on averaged signals obtained in the different attention conditions and are presented as averaged index values from four subjects (see Methods). In visual cortex, attentional effects increased from early to later processing stages. Attentional effects in the LGN were larger than in V1. Vertical bars indicate s.e.m. across subjects.

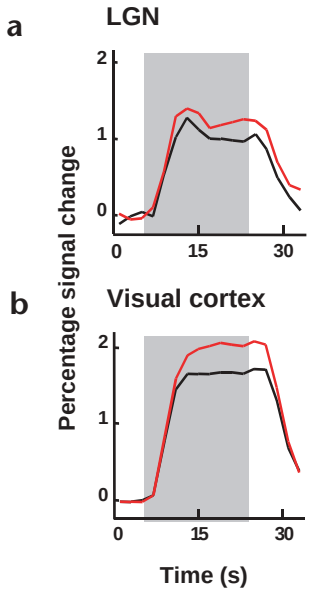

Fig. 4. Spatial selectivity of attention effects in the LGN (**a**) and in visual cortex (**b**). Group data (n = 4). Checkerboard stimuli were presented simultaneously to the right and left visual hemifield while subjects were instructed to attend either to the left or to the right checkerboard to detect randomly occurring luminance changes. FMRI signals evoked by the checkerboard stimuli on the attended side (red curves) were greater than signals evoked by the checkerboard stimuli on the unattended side (black curves). Other conventions as in **Fig. 2**.

ty in the LGN in multiple ways: by enhancing neural responses to attended stimuli, by attenuating those to ignored stimuli, and by increasing baseline activity in the absence of visual stimulation. These effects of attention were qualitatively similar to those obtained in visual cortical areas. The effects of attentional response enhancement were spatially specific, indicating that they were due to selective attention rather than to unspecific arousal. In behavioral studies outside the scanner, we found that eye movements were not systematically correlated with the different task conditions and were therefore an unlikely source for the response modulation found in the LGN and visual cortex. Taken together, our results indicate that the LGN may be the first stage in the processing of visual information that is modulated by attentional signals.

Previous studies have not found attentional modulation in the LGN. In monkey physiology studies[28,29,31], attentional modulation was investigated by comparing neural responses evoked by identical visual stimuli when attended or ignored across multiple areas of the visual system, including the LGN, striate and extrastriate cortex. Attentional modulation of neural responses was consistently found in cortical areas, but not in the LGN. These negative findings support the classical notion that selective attention affects neural processing only at the cortical level. We now challenge this view by using fMRI to demonstrate qualitatively similar attention effects in the LGN and visual cortex. Functional MRI measures neural activity at a population level that may be better suited to uncover large-scale modulatory activity. Small modulatory effects that cannot be reliably found by measuring neural activity at the single- or multi-unit level may be revealed when summed across large populations of neurons. Our results confirm and extend findings from a double-label deoxyglucose study[32] that showed the suppression of metabolic activity in regions surrounding an attended location in the magnocellular layers of the monkey LGN. This is in accordance with our finding that attention suppresses fMRI signals evoked by an ignored stimulus. Furthermore, we found that such attentional suppression depends on attentional load.

The magnitude of all attention effects—enhancement, suppression and baseline increases—increased from early to more advanced processing levels of visual cortex, confirming results from previous studies[10,26,28,29]. This is consistent with the idea that attention operates through top-down signals that are transmitted via corticocortical feedback connections in a hierarchical fashion. This way, later stages of visual cortical processing are more strongly controlled by attentional mechanisms than are early processing stages. According to this account, one would predict smaller attention effects in the LGN than in striate cortex. In contrast, we found that all attention effects tended to be larger in the LGN than in striate cortex. This raises the possibility that attentional modulation in the LGN may be due to factors other than corticothalamic feedback from striate cortex; modulatory influences may also come from sources such as the brainstem, the superior colliculus (SC) and the thalamic reticular nucleus (TRN). Because hemodynamic responses appear to reflect the synaptic input to an area[33], the large attention effects in the LGN may result from these multiple modulatory influences. Other possibilities that may explain the differences in magnitude of the modulation between the LGN and V1 include regional disparities under-

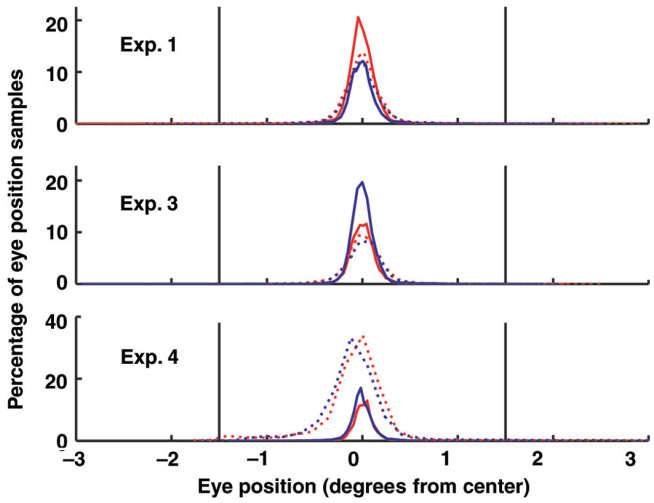

Fig. 5. Frequency histograms of eye position for experiments 1 (top), 3 (middle) and 4 (bottom). Group data (n = 4) from behavioral sessions outside the scanner. Subjects directed attention either to the left (red curves) or to the right (blue curves) of fixation. Horizontal eye position (solid curves) and vertical eye position (dashed curves) were both sharply peaked and centered on the fixation cross at zero degrees. Negative abscissa values indicate leftward deviations of eye position from fixation. Positive values indicate rightward deviations from fixation. The inner vertical lines indicate the inner margin of the checkerboard stimulus; a blank region surrounded the central 1.5° around the fixation cross (see **Fig. 1a** and **b**).

lying the blood oxygenation level–dependent (BOLD) signal or non-linearities in thalamocortical signal transmission.

Owing to its afferent input, the LGN may be in an ideal position to serve as an early gatekeeper in attentional gain control[34]. In addition to corticothalamic feedback projections from V1, the LGN receives inputs from the SC, the parabrachial region and the TRN. For several reasons, the TRN has long been implicated in theoretical accounts of selective attention[30,34,35]. First, all feedforward projections from the thalamus to the cortex, as well as their reverse projections, pass through the TRN. Second, the TRN receives inputs not only from the LGN and V1, but also from several extrastriate areas and the pulvinar. Thus, it may serve as a node where several cortical areas and thalamic nuclei can interact to modulate thalamocortical transmission through inhibitory connections to LGN relay cells[36]. And third, the TRN contains topographically organized representations of the visual field and can thus modulate thalamocortical or corticothalamic transmission in spatially specific ways. The SC is part of a distributed network of areas controlling eye movements, which have recently been shown to modulate the activity of LGN neurons[37]. The existence of multiple modulatory inputs to the LGN, which greatly outnumber the retinal inputs, is consistent with the idea that this structure is centrally involved in selective attention, as suggested by our findings.

The neural mechanisms underlying attention effects are not yet clear. Attentional response enhancement may result from an increase in neural activity at the attended location or a decrease in activity around the locus of selection[32]. Our result of attentional load–dependent suppression of neural activity evoked by ignored stimuli points to a suppressive mechanism. Other studies suggest that attentional response enhancement may be mediated by a push–pull mechanism that combines excitatory and suppressive components (ref. 9 and M.A.P & S. K., *Soc. Neurosci. Abstr.* **27**, 574.4, 2001). Alternatively, attentional response enhancement could result from an elevated baseline activity that is sustained during the visual stimulation period and simply adds to the visually evoked activity[17–20]. Increases in baseline activity during directed attention in anticipation of a visual stimulus have been interpreted, in the framework of the biased competition account of attention[1], to reflect a top-down feedback bias in favor of the attended location, enhancing synaptic efficacy[18,19]. Several findings counter the idea that attentional response enhancement simply reflects a baseline increase in activity. For example, attention-related baseline increases were demonstrated in early visual cortex without a concomitant increase in the visually evoked response to attended stimuli (for a more detailed discussion, see ref. 19). In the present study, we sought to determine the level at which attentional modulation first occurs in the visual system. The neural mechanisms underlying the attentional modulation reported here and in other studies[5–11,15–20,26–29], however, will require further study.

Much remains to be learned about the complex thalamic circuitry that subserves attentional modulation in the LGN. We conclude from our studies that the LGN appears to be the first stage in the processing of visual information that is modulated by attentional signals. Our findings challenge the idea that attention effects are confined to cortical processing, and suggest the need to revise the traditional view of the LGN as a mere gateway to the visual cortex. The LGN may, in fact, serve as a gatekeeper in attentional gain control. The precise nature of the gain mechanism remains to be explored.

Methods

Subjects, visual stimuli and tasks. Four healthy subjects (three men, 22–38 years old, normal or corrected-to-normal visual acuity) gave written informed consent for participation in the study, which was approved by the Institutional Review Panel of Princeton University.

High- or low-contrast checkerboard stimuli (**Fig. 1a** and **b**) that reversed contrast at 7.5 Hz were presented in alternation either to the left or the right visual hemifield in blocks of 16–18 s. The subjects' central fixation point was separated from the checkerboard stimuli by 1.5°; regions along the vertical meridian were spared. In experiment 4, the checkerboard stimulus was simultaneously presented to both hemifields in blocks of 18 s interleaved with blank periods of the same duration. In the attended conditions of experiments 1, 3 and 4, subjects covertly directed attention to the checkerboard arc at 10° eccentricity (**Fig. 1b**) and detected randomly occurring luminance changes along that arc. Covert attention shifts were indicated by briefly presenting an arrow at fixation that pointed to the left or right hemifield 1 s before the onset of the stimuli. In the unattended conditions of experiments 1 and 2, subjects performed a letter-counting task at fixation. Three target letters were presented randomly among digits and keyboard symbols, at a rate of 267 ms per item. Each scan started and ended with a 16–28 s presentation block comprising a fixation point on a blank screen.

Subject training and eye movement control. Before scanning, subjects were extensively trained on all tasks. Eye movement control experiments were run outside the scanner while subjects performed the same attention tasks as in the scanner, under matched display and viewing conditions. Subjects' eye positions were monitored at a sampling rate of 60 Hz by a head-mounted infrared eyetracking system (ISCAN ETL-500, Iscan Inc., Burlington, Massachusetts). The foveated location was superimposed on a scene-camera image of the visual display and recorded on videotape. Eye position data were output as raw scene camera pixel locations and converted from pixel values to degrees of visual angle. Frequency histograms of eye position were derived for each subject and each condition; they are shown here as group data (**Fig. 5**). For the covert attention tasks of experiments 1, 3 and 4, eye positions while attending to the right and to the left were analyzed separately. For the rare samples of eye movements that deviated from fixation by more than 1.5° (<1% of horizontal samples), *t*-tests were used to determine whether there was any relation between condition and eye position.

Data acquisition and analysis. General scanning and data analysis procedures were identical in all experiments. Data were acquired in 20 scan sessions with a 3-tesla head scanner (Allegra, Siemens, Erlangen, Germany) using a standard head coil. Functional images were taken with a gradient echo, echoplanar sequence (TR, 2 s; TE, 30 ms; flip angle, 90°; matrix 64 × 64 voxels). Twenty-two contiguous, axial slices (thickness, 3 mm; gap, 1 mm; in-plane resolution, 3.17 × 3.17 mm) were acquired in 6–12 series of 96–129 images each, covering the thalamus and visual cortex. Echoplanar images were compared with a co-aligned, high-resolution anatomical scan of the same subject's brain taken in the same session (FLASH; TR, 150 ms; TE, 4.6 ms; flip angle, 90°; 256 × 256 matrix). Another high-resolution anatomical scan of the whole brain (MPRAGE sequence; TR, 11.1 ms; TE, 4.3 ms; flip angle, 8°; 256 × 256 matrix; three-dimensional resolution, 1 mm^3) was taken to perform spatial normalization and three-dimensional surface reconstructions in BrainVoyager (Brain Innovation, Maastricht, Netherlands).

Functional images were motion-corrected[38]; statistical analyses were restricted to brain voxels with adequate signal intensity (average intensity of >20% of the maximum value across voxels). The first five images of each scan were excluded from analysis. Statistical analyses were performed using multiple regression in the framework of the general linear model[39] with National Institutes of Health functional imaging data analysis program (FIDAP) software. In experiments 1–3, a square wave function reflecting the contrast of left versus right visual hemifield stimulation was convolved with a Gaussian model of the hemodynamic response (lag 4.8 s, dispersion 1.8 s) to generate an idealized response function, which was used in the regression model. In experiment 4, checkerboard presentations were contrasted with blank presentations. Additional regressors were used to factor out variance due to between-run changes in mean

intensity and within-run linear changes. With this statistical model, voxels corresponding to foveal stimulus representations were excluded from further analysis; in all experiments, fMRI activity reflects only activity evoked by the peripheral checkerboard stimuli. Statistical maps were thresholded at a Z-score of 2.33 ($P < 0.05$). LGN activations were identified based on contiguous voxels in its anatomical location (**Fig. 1c**; **Table 1**). Activity in visual cortex was assigned to retinotopically organized areas. FMRI time series analyses were performed on all activated voxels within a given region and are presented as group data. Mean signals were computed by averaging across all intensity values obtained in a given condition and are given as percent signal change. Percent signal change was computed relative to the mean of the six intensity values preceding a visual stimulation period in experiments 1, 2 and 4 or preceding an expectation period in experiment 3. Because we did not find any differences between activity in the right and left LGN or visual cortex, data were combined across hemispheres. Statistical significance of time series data was determined by a random effects analysis using one-sample, one-tailed t-tests. Statistical significance of index values was determined by ANOVAs with three factors: subject, experiment and anatomical region. For each subject, statistical maps and structural images were transformed into Talairach space[40] using BrainVoyager software.

To quantify the attention effects and to compare them across areas, an attentional enhancement index (AEI; experiment 1), an attentional suppression index (ASI; experiment 2) and a baseline modulation index (BMI; experiment 3) were computed using the mean fMRI signals obtained in a given condition. $AEI = (R_{ATT} - R_{UNATT})/(R_{ATT} + R_{UNATT})$; $ASI = (R_{EASY} - R_{HARD})/(R_{EASY} + R_{HARD})$; $BMI = R_{EXP}/R_{ATT}$); R, response; ATT, attended visual presentations; UNATT, unattended visual presentations; EASY, easy attention task; HARD, hard attention task; EXP, expectation period. Indexes were computed for each subject and were then averaged across the group of subjects.

Mapping visual areas. Retinotopic mapping was performed for each subject in a separate scanning session using established procedures[24] described in detail elsewhere[25]. Areas V1, V2, V3/VP and V3A were identified by the alternating representations of the vertical and horizontal meridians, which form the borders of these areas. Areas V4 and TEO were identified by their characteristic upper (UVF) and lower (LVF) visual field retinotopy. The UVF and LVF are separated in V4 and located medially and laterally on the fusiform gyrus[41], whereas this separation is not seen in the region anterior to V4, which we term TEO. Activations in area MT/MST were identified on the basis of this area's characteristic anatomical location[42].

Acknowledgments

We thank J. D. Cohen, G. M. Doniger, M. S. A. Graziano, C. G. Gross, J. V. Haxby, F. Tong and A. Treisman for valuable discussions, and M. Gilzenrat for help with eye movement measurements. Supported by National Science Foundation Graduate Research Fellowships to D.H.O. and M.A.P. and by grants from the National Institute of Mental Health and the Whitehall Foundation to S.K.

Competing interests statement

The authors declare that they have no competing financial interests.

RECEIVED 12 AUGUST; ACCEPTED 17 SEPTEMBER 2002

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