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Research report

Spatial frequency-dependent feedback of visual cortical area 21a modulating functional orientation column maps in areas 17 and 18 of the cat

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Abstract

The feedback effect of activity of area 21a on orientation maps of areas 17 and 18 was investigated in cats using intrinsic signal optical imaging. A spatial frequency-dependent decrease in response amplitude of orientation maps to grating stimuli was observed in areas 17 and 18 when area 21a was inactivated by local injection of GABA, or by a lesion induced by liquid nitrogen freezing. The decrease in response amplitude of orientation maps of areas 17 and 18 after the area 21a inactivation paralleled the normal response without the inactivation. Application in area 21a of bicuculline, a GABAa receptor antagonist caused an increase in response amplitude of orientation maps of area 17. The results indicate a positive feedback from high-order visual cortical area 21a to lower-order areas underlying a spatial frequency-dependent mechanism.

Theme: Sensory systems
Topic: Neurophysiology

Keywords: Visual cortex; Area 21; Area 17; Orientation column; Feedback; Area 18; Optical imaging

1. Introduction

It is generally assumed that in the mammalian visual system, the information about the outside visual world is processed in the excitatory 'feedforward' pathway from the retina, via the dorsal lateral geniculate nucleus in the dorsal thalamus to the primary visual cortex and hence to the 'higher-order' visual cortical areas. A substantial amount of data has also been gathered concerning the role of the feedforward cortico-cortical projections from the 'lower-order' cortical areas to 'higher-order' cortical areas [6,21,23]. However, much less is known about the functional role of the so-called feedback cortico-cortical projections from the 'high-order' cortical areas to 'lower-order' cortical areas [3,6,19,20]. Several lines of evidence indicate that in highly visual mammals such as cats and macaque monkeys the feedback projections from the "higher-order" visual cortical areas (V5, V2(18), 21a) modulate many response properties of neurons in the 'lower-order' visual cortical areas [8,10,11,21,30]. Area 21a of the cat is strongly and reciprocally connected to cytoarchitectonic area 17 (striate cortex, area V1) [3,5,14,15,16]. Indeed, in many respects, receptive field properties of the area 21a neurons are very similar to those of area 17 neurons [3,4,15,29–31]. In particular, cells in area 21a, like those in area 17, exhibit sharp orientation-tuning, preference to low temporal frequency stimulation and strong binocular interactions. On the other hand, at any eccentricity, area 21a neurons exhibit much larger receptive fields and broader spatial frequency-tuning. Furthermore, unlike area 17, only the upper contralateral visual field and the strip of the retina 3–7° below the zero horizontal meridian are represented in area 21a [4,25,26,31]. In the present study using intrinsic signal

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optical imaging, we attempted to assess the effect of reversible inactivation or lesion of area 21a on the magnitude of responses to sinusoidal gratings and global 'orientation maps' in the parts of areas 17 and 18 visuotopically corresponding to area 21a.

2. Materials and methods

All experiments conformed to the policy of the Society for Neuroscience on the Use of Animals in Neuroscience Research. Animals were originally anaesthetized with ketamine (20 mg/kg). To maintain anesthesia and block the neuromuscular transmission, a mixture of sodium pentobarbital (3 mg/kg h) and gallamine triethiodide (10 mg/kg h) in saline was injected intravenously in 17 cats. The animals were artificially ventilated with a pulmonary pump. The end-tidal volume of carbon dioxide (CO_2) was monitored and kept at about 4% by adjusting the rate and/or stroke volume of the respirator. Electroencephalogram (EEG) and electrocardiogram (ECG) were continuously monitored with the EEG always showing a slow wave record throughout. The body temperature was continuously monitored and maintained at 38 °C throughout the experiment. The pupils were dilated with atropine (0.5%) and nictitating membranes were retracted with neosynephrine (2%). The eyes were carefully refracted and corrected with contact lenses of appropriate refractive power. To reduce the amount of spherical aberration, artificial pupils (3 mm in diameter) were placed in front of each eye. Cortical area 21a was exposed at Horsley–Clarke coordinates L7–12, P1–6 while areas 17 and 18 were exposed at Horsley–Clarke coordinates L0–8, P0–10. The strip of area 17 exposed contains the representation of most of the upper part of the contralateral visual field and about 10° below the horizontal meridian both in close proximity to the zero vertical meridian [26]. Similarly the exposed part of area 18 contained the representation of most the upper contralateral visual field and the region down to about 10° below the horizontal meridian [27]. Thus, the exposed parts of areas 17 and 18 contained the representation of most of the visual field found in area 21a [4,25,31].

Area 21a was reversibly inactivated by microinjection of 1.5 μl of γ-amino-butyric acid (10–500 mM GABA, Sigma, USA) and activated by micro-injections of 1.5 μl $GABA_A$ receptor antagonist bicuculline (50 μM, Sigma). As a control, we have injected 1.5 μl of phosphate-buffered saline (PBS, pH 7.4) at the same site. Solutions were injected slowly (over the period of 4 min) and the needle of the micro-syringe was withdrawn 10 min after the termination of injection. The injection sites were centered 0.8–1.2 mm beneath the pial surface (about layer 4 or 5). We do not have direct assessment of the area of diffusion of our injections, however, previous study [11,32] indicates that 1.5 μl of GABA tends to diffuse over a region of 1.5 mm in diameter in the mammalian cortex. Finally, in some cases, area 21a was inactivated irreversibly by touching it (four to five times within a minute) with a Q-tip soaked in liquid nitrogen [22]. To avoid direct effect of liquid nitrogen on the temperature of the cortex, the optical imaging recording was carried out 80 min after completion of the irreversible inactivation. The location of the centers of the injection sites and the extent of the liquid nitrogen lesions were assessed histologically in 0.2-mm sections with Nissl staining. Only data from animals with the correct lesion locations within area 21a were analyzed.

A slow-scan CCD camera (DALSA, Canada) was used to record the optical images of intrinsic signals from the exposed part of area 17 [2]. With red light (640 nm) the vessel map of the cortical surface (Figs. 1A and 4A) was obtained with green light (546 nm) shining on the cortex. The functional orientation maps of the cortical cells responding to drifting sinusoidal grating stimuli were obtained with red light (640 nm) when the camera was focused on the cortical depth of 500 μm.

The cats were stimulated binocularly with the computer-generated gratings oriented horizontally and vertically respectively, and then the horizontal vs. vertical subtracted maps (differential maps) were obtained for quantitative analysis. For better map display, with red light (640 nm), the high-pass and low-pass filtering as well as histogram equalization techniques were employed as described elsewhere [2]. The gratings used varied in 4 spatial frequencies from 0.18 to 2.0 cycles/degree (c/d) with a contrast of 0.9 and a temporal frequency of 2 Hz. The mean luminance of gratings or a blank screen was 15.1 cd/m^2. Usually, 16 or 32 trials, in which a grating stimulus of 2 s and a blank of 10 s were repeatedly presented on the screen of the monitor, were enough to produce a clear orientation map of area 17. There are substantial differences in the spatial frequency tuning between cat's areas 17 and 18 [1,2,7,9,12]. The location of the boundary between areas 17 and 18 was determined by subtracting the orientation maps elicited by gratings of spatial frequency 0.58 c/d from the maps elicited by 0.14 c/d.

To quantify the degree of a map's orientation selectivity, the response amplitude of an orientation map was defined as the averaged contrast of original orientation maps, as described previously [2]. The contrast was defined as $(L_{white} - L_{black})/(L_{white} + L_{black})$, where L is the mean luminance of a black or white area on a map. The contrast was calculated for each of six to nine pairs of black and white circular areas which were always located at the centers of black and white zones in a map. The paired areas, whose diameter (250 μm) was about half of the width of a zone were randomly chosen from orientation maps of areas 17 or 18. Then, the six to nine values of contrast were averaged as the response amplitude of a map either in normal or in treated conditions. Two-dimensional cross-correlation coefficient (CCC) analysis as described before [2] was used to quantify the similarity of two maps of the same cortical area obtained under different conditions. Student's t-tests were

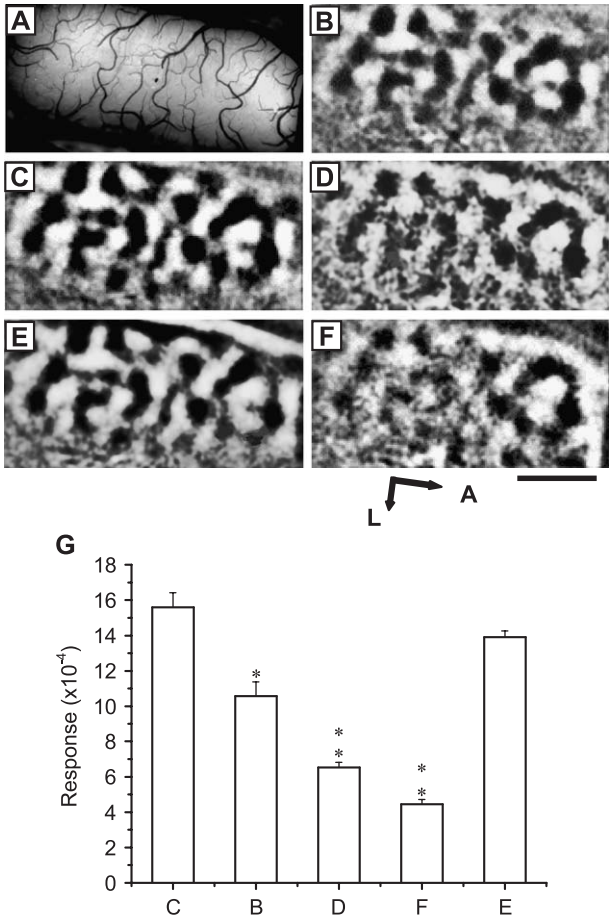

Fig. 1. Modulatory effect of GABA in area 21a on the orientation maps of area 17 elicited by a 0.5-c/d grating stimulus. (A) Blood vessel map of the surface of cortical area 17 studied. (B, D and F) Orientation maps of area 17 revealed by optical imaging when area 21a was injected with 10, 100 and 500 mM GABA, respectively. (C) Normal orientation map of area 17 as a control. (E) Recovered orientation map obtained one hour after GABA in area 21a was washed up with saline. (G) Histograms of response amplitude (contrast) of the above maps shown in (B–F). Statistical significance for the responses obtained in GABA-treated vs. control conditions: * and ** represent $P<0.05$ and $P<0.01$, respectively. Note that the patterns of orientation maps of area 17 become progressively more blurred as the concentration of GABA increase from 10 to 500 mM indicating a clear dose-dependent effect. However, the shape and distribution of the visible dark areas in the maps remain unchanged. All the short vertical bars indicate standard errors. The center of all maps was located at Horsley–Clarke coordinates P5.2, L2.3. A, anterior; L, lateral. Scale bar: 2 mm.

also used to statistically compare the significance of changes in response amplitude.

3. Results

3.1. Effect of GABA injection in area 21a on area 17

In all 13 cats without exception, local injection of GABA into ipsilateral area 21a resulted in reversible decrease in magnitude of responses of area 17 cells as well as in the sharpness of the orientation maps in area 17. This decrease was significantly dose-dependent on GABA applied. A typical response before injection of GABA into area 21a is shown in Fig. 1C. For this animal, it is apparent that in Fig. 1B, D, F and G injections of 1.5 μl of GABA of gradually increasing concentration (10, 100 and 500 mM) into area 21a result in progressively increasing response amplitude of area 17 cells (33.1%, 58.6% and 71.8%, respectively). Although the reduction in the response magnitude resulted in gradually more blurred orientation maps in area 17, the basic pattern of visible orientation maps remained and the CCCs in all cases were around 0.85–0.92. An hour after injections of GABA into area 21a, there was virtually complete recovery of the response magnitude in area 17 (Fig. 1E). Hereafter, the concentration of GABA used for 21a inactivation was always chosen at 100 mM for ease of recovery in repeated experiments. The mean decrease by 100 mM GABA applied in area 21a was significant in the area 17 maps

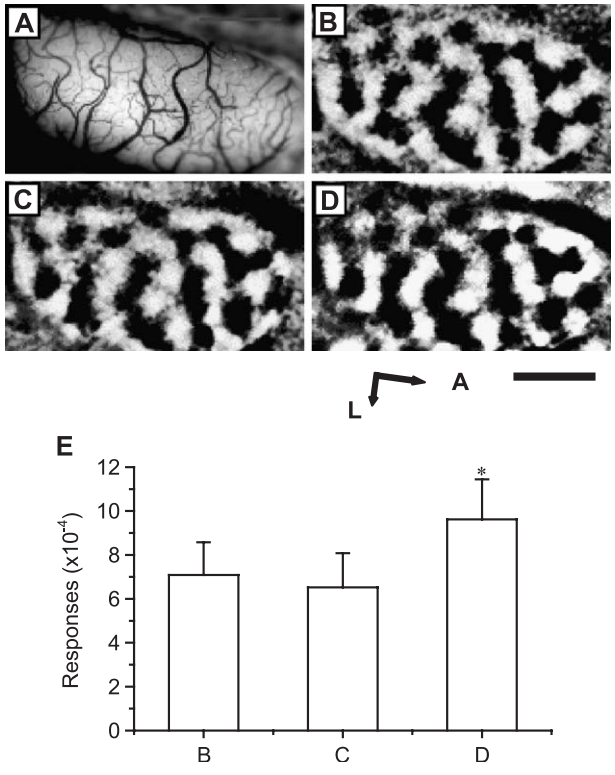

Fig. 2. Orientation maps of area 17 before and after microinjection of bicuculline in area 21a of a cat. (A) Vessel map of area 17 studied. (B) Orientation map of area 17 before bicuculline injection. (C) Orientation map after 1.5 μl phosphate-buffered saline was injected into area 21a. (D) Orientation map after injecting 1.5 μl of 50 μM bicuculline. (E) Comparison in response amplitudes of the maps of (B), (C) and (D) showing a significant excitatory effect of area 21a on area 17 (*t*-test, $P<0.05$). The center of all maps was located at Horsley–Clarke coordinates P4.7, L2.2. All maps were elicited by a grating stimulus of 0.5 c/d. All the short vertical bars indicate standard errors. A, anterior; L, lateral. Scale bar: 2 mm.

(31.9 ± 3.7%, hereafter, referred as the mean ± standard deviation, *t*-test, $P < 0.01$).

Freezing area 21a with liquid nitrogen in seven cats, some of which was done after complete recovery from GABA treatments, resulted in a mean decrease of 49.8 ± 4.0% in the response amplitude of area 17 cells (Fig. 4A–I). The decrease was significantly greater (*t*-test, $P < 0.01$) than that (31.9 ± 3.7%) induced by 100 mM GABA injections. Thus, both GABA injections and liquid nitrogen lesions indicate that area 21a exerts an excitatory modulatory effect on visually evoked neuronal activity in area 17.

3.2. Effect of activation of area 21a by bicuculline on area 17

The response amplitude of orientation maps of area 17 increased when 1.5 μl of 50 μM bicuculline, a $GABA_A$ receptor antagonist, was ipsilaterally injected into area 21a in 7 of 11 cats (63.6%) studied. A significant excitatory influence of area 21 on area 17 (58% of increase in response amplitude) is shown in Fig. 2. The mean increase in response amplitude (35.6 ± 5.8%, ranged from 9.7% to 39.7%) of area 17 seen in 7 of the 11 cats, following the bicuculline-induced disinhibition in area 21 neurons was statistically significant (*t*-test, $P < 0.01$) and indicated a significant excitatory influence of area 21a on area 17. However, in the remaining four cats (36.4%), a weak reduction in mean response (13.7 ± 3.7%, ranged from 11.2% to 16.8%) was observed in area 17.

In one cat, a pre-injection of bicuculline (1.5 μl of 50 μM) in area 21a prior to the GABA application completely inhibited the GABA (1.5 μl of 50 mM) effect on area 17. Thus, there was no change found on the orientation map of area 17.

3.3. Spatial frequency dependent effect of inactivation of area 21a on areas 17 and 18

Interestingly, the effect of GABA or cold-induced lesions in area 21a on neuronal activity in area 17 appeared most significantly when the spatial frequency of the stimulating grating used was about 0.5–0.6 c/d, which appears to be optimal for most cells in area 17 under normal condition. By contrast, the effects on response decrease in area 17 were relatively weaker when spatial frequencies either higher or lower than 0.5–0.6 c/d were used (Figs. 3J and 4J).

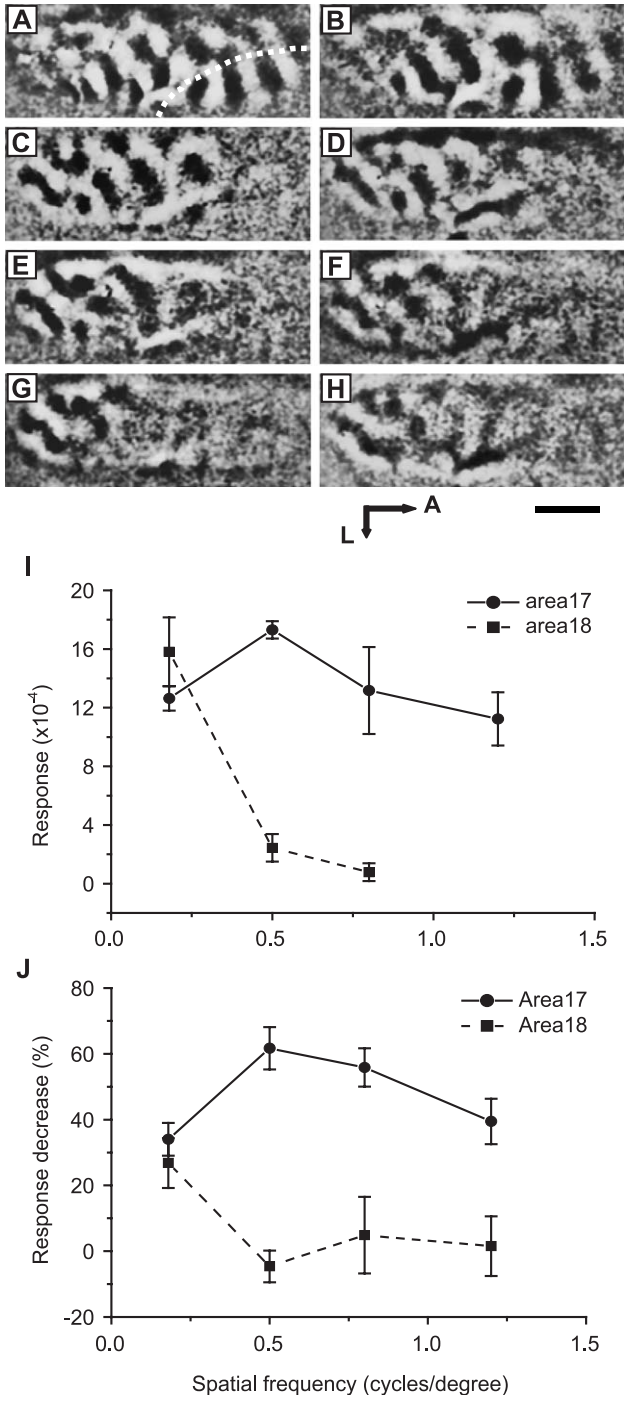

Fig. 3. Spatial frequency-dependent effect of inactivation of area 21a caused by GABA microinjection on the orientation maps of areas 17 and 18 of a cat. (A) and (B), (C) and (D), (E) and (F), (G) and (H) show the orientation maps elicited by grating stimuli of 0.18, 0.5, 0.8 and 1.2 c/d, respectively. Maps of area of (A), (C), (E) and (G) were obtained before GABA injection; maps of (B), (D), (F) and (H) were obtained under 100 mM GABA-treated conditions. The vertical axis in (I) represents the contrast measure defined in the text and that in (J) indicates the relative decrease in the contrast. A white dotted line in (A) indicates the boundary between areas 17 and 18 which was determined by subtracting the orientation map elicited by a spatial frequency 0.58 c/d grating from the one by 0.14 c/d [2,9,12]. (I) Response amplitude of orientation maps of areas 17 and 18 as a function of spatial frequencies before the GABA inactivation of area 21a. (J) Response decrease in relative amplitude of orientation maps of areas 17 and 18 elicited by gratings as a function of spatial frequencies after area 21a was injected with 100 mM GABA. Note that the maps of 0.5 and 0.18 c/d show the most significant effect of GABA in area 21a on areas 17 and 18, respectively, though the visible patterns of orientation columns shrank and went towards to the posterior part of the cortex gradually with increasing of spatial frequency used. Note that there is a peak vs. peak correlation between the two curves either for area 17 or for area 18 in (I) and (J). All the short vertical bars indicate standard errors. The center of all maps was located at Horsley–Clarke coordinates P4.8, L2.0. A, anterior; L, lateral. Scale bar: 2 mm.

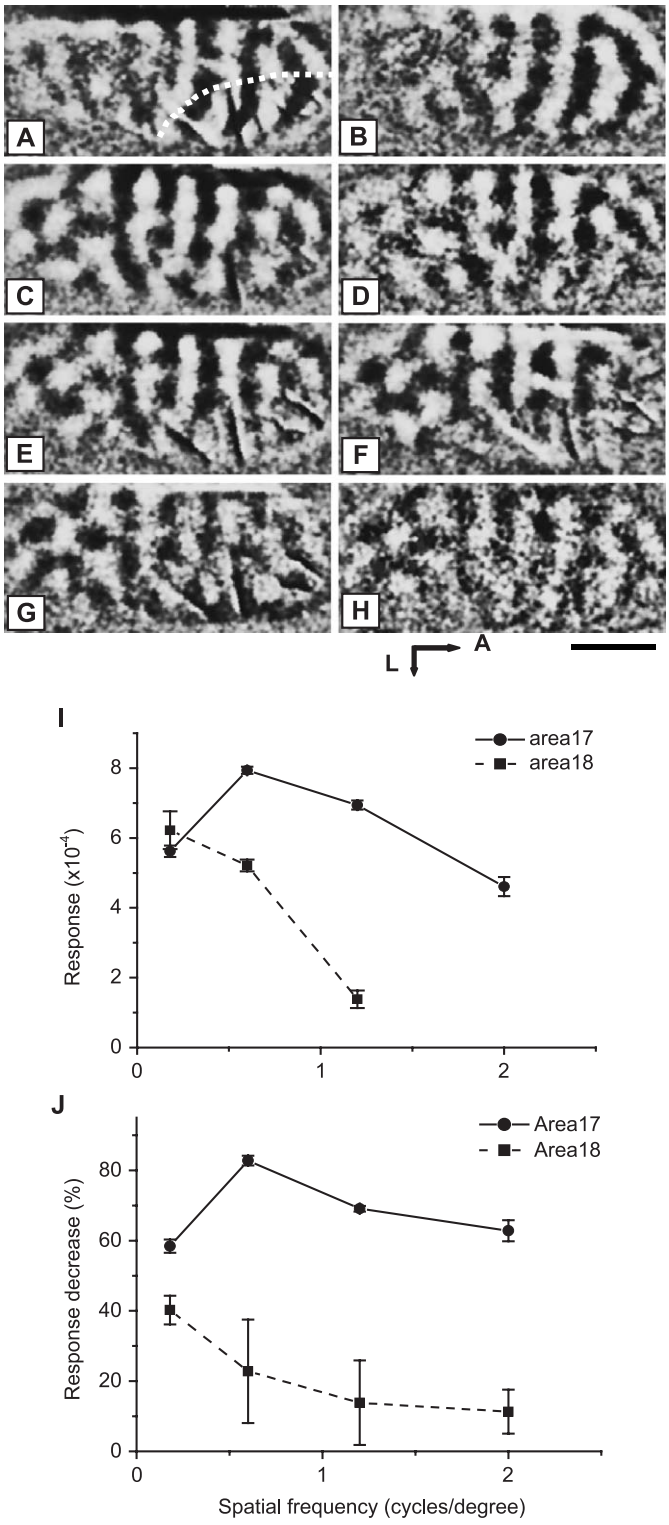

Fig. 4. Spatial frequency-related effect of the area 21a freezing lesion on the orientation maps of areas 17 and 18 of a cat. (A) and (B), (C) and (D), (E) and (F), (G) and (H) showing the orientation maps elicited by grating stimuli of 0.18, 0.6, 1.2 and 2.0 c/d, respectively. Maps of area of (A), (C), (E) and (G) were obtained before lesion; maps of (B), (D), (F) and (H) were obtained after lesion. A white dotted line in (A) indicates the boundary between areas 17 and 18. (I) Response amplitude of orientation maps of areas 17 and 18 as a function of spatial frequencies before the lesion of area 21a. (J) Response decrease in relative amplitude of orientation maps of areas 17 and 18 elicited by gratings as a function of spatial frequencies after area 21a was frozen. Note that, again, the maps of 0.6 and 0.18 c/d show the most significant effect of the area 21a lesion on areas 17 and 18, respectively. Note that there is a peak vs. peak correlation between the two curves either for area 17 or for area 18 in (I) and (J). All the short vertical bars indicate standard errors. The center of all maps was located at Horsley–Clarke coordinates P5.0, L1.9. A, anterior; L, lateral. Scale bar: 2 mm.

Similarly, we found that inactivation of area 21a also decreased the response amplitude of orientation maps in area 18. The effect was also spatial frequency dependent showing a peak decrease at about 0.18 c/d (Figs. 3J and 4J). These phenomena were repeatedly observed in two GABA-treated and three freeze lesioned cats. This demonstrated that the degree of excitatory modulation of 21a is not uniform for all the cortical cells in areas 17 and 18, but somehow spatial frequency-dependent. However, the effect was usually less significant in area 18 than in area 17 suggesting that the modulation of area 21a is stronger in area 17 than in area 18.

The normal spatial frequency tuning curves of orientation maps of areas 17 and 18 (Figs. 3I and 4I) obtained before inactivation of area 21a were compared with the two curves after the inactivation (Figs. 3J and 4J). It is evident that the two curves are parallel to each other and that both the greatest decreases in responses amplitude of area 17 and 18 after the inactivation of area 21a and the largest responses in the normal condition occur at a spatial frequency of 0.5–0.6 c/d for area 17 and of 0.18 c/d for area 18, respectively. Thus, the positive feedback signals from area 21a appear to contribute to the spatial frequency dependence of areas 17 and 18. If there was no such a positive feedback, the spatial frequency tuning curves in areas 17 and 18 would be expected to be flatter.

4. Discussion

Using intrinsic signal optical imaging and reversible pharmacological inactivation methods, we have observed for the first time a spatial frequency-dependent modulatory effect of area 21a on the overall neuronal activity in a large area of areas 17 and 18. Either application of GABA or freeze lesioning of area 21a affected the magnitude of the responses of neurons in both areas 17 and 18, though the effect was more significant in area 17 than in area 18. In general, the modulatory effect of area 21a is mainly excitatory because inactivation or disinhibition of area 21a results in a decrease in responses of orientation maps of areas 17 and 18 or an increase of area 17 with few exceptions. This is in agreement with the results of a recent electrophysiological study by Wang et al. [30].

The activities of the most neurons in areas 17 and 18 reported here, that send the synchronized signals to area 21a, were preferentially enhanced by the excitatory modulation of area 21a at 0.5–0.6 and 0.14–0.18 c/d respectively. The correlation of the feedforward and feedback signals between these areas indicates that the spatial frequency-dependent modulation of area 21a we observed may contribute in a great deal to the change in response amplitude at different spatial frequencies in areas 17 and 18, i.e. shaping spatial frequency tuning.

Both areas 17 and 18 receive strong feedback projections from the ipsilateral area 21a, however, here the magnitude of responses of area 18 cells appears to be less affected by inactivation of ipsilateral area 21a than that of area 17 cells. This might be due to the difference in spatio-temporal property between cells in areas 17 and 18. Overall, cells at various locations in areas 17 and 18 responded differentially according to retinotopic projection, as shown in optical imaging studies [2,9,12] with most cells in area 17 responding optimally to gratings of spatial frequency 0.5–0.6 c/d and those in area 18 responding optimally to gratins of about 0.2 c/d in normal condition [17]. Furthermore, the temporal frequency used here was low (2 Hz) and the spatial frequencies used were 0.14–2.0 c/d generating these gratings of relative low velocities from 1.0 to 14.3 deg/s in the experiments. The neurons in area 21a and 17 respond optimally to the lower temporal frequencies and velocities [12,16,17], while neurons in area 18 respond optimally to the higher temporal frequencies and velocities [1,7,17]. This is consistent with the report that reversible inactivation (by cooling) of the areas in the PMLS (posteriomedial lateral suprasylvian area) of the cat, where cells frequently respond well to the higher temporal frequencies [28,33], results in strong decrease in signal strength in both orientation and direction domains in the ipsilateral area 18. Furthermore, inactivation of higher-order areas in the visual parietal cortex results in virtual abolition of global layout of direction maps in area 18 [8].

The applications of GABA in area 21 caused a spatial frequency-dependent effect similar to that of freezing lesions on areas 17 and 18. This demonstrates that the liquid nitrogen lesion we used is a reliable method like previous experiments [22]. The more severe effect of the lesion than that of 100 mM GABA treatment may result from the more complete inactivation of area 21a by lesions (mean decrease $49.8 \pm 4.0\%$ for freezing lesions; $31.9 \pm 3.7\%$ for 100 mM GABA injection). However, if 500 mM GABA had been used, it may have resulted in a comparable effect to that of lesions (decrease 71.8% for 500 mM GABA injection; 82.0% for lesion, as shown in 1G and 4J). The effect of area 21a inactivation on area 17 revealed by intrinsic signal optical imaging is more significant than that observed with single-unit electrophysiological recording with reversible cooling of area 21a (mean decrease $21.8 \pm 4.7\%$) by Wang et al. [30]. It might be due to the much higher sensitivity of the optical imaging to overall neuronal activity than that of the single-unit recording. Optical imaging reflects not only summation of neuronal spiking responses but also their subthreshold activity that the later technique cannot [24].

In addition to the direct feedback projection, the influence of area 21a on area 17 might also result from indirect projections via areas 18, 19, PMLS and even via subcortical structures [3]. Although we cannot differentiate the direct from indirect effects here, the direct projection might be expected to play a more important role in visual information processing because the direct

projection from areas 21a to 17 employs fewer synapses and the observed area 21a effect is weaker on area 18 than on area 17.

In four cats, injection of bicuculline in area 21a resulted in a decrease instead of increase in response amplitude of area 17. This might be induced by an overall over-elevation of the level of feedback activity from areas 21a to 17 caused by the critical concentration of 50 μM bicuculline because we have observed in another experiments that in vivo infusion of 20–40 μM bicuculline on the cortical surface could evoked significant epileptic EEG waves in the cat's visual cortex. All neurons in areas 17 and 18, no matter whether they preferred or do not preferred to respond to the orientation of stimulating gratings, might be elicited unselectively by the over-excitatory influence of area 21a. As a result, the response amplitude or contrast of resultant orientation maps decrease.

As previously shown with electrophysiology, the modulatory feedback of area 21a caused no change in global orientation preference of areas 17 cells [30], however, it did cause change in the overall responses of cells in areas 17 and 18 to grating stimuli of different spatial frequencies even when the animal was anesthetized. It is argued that effects of higher-order areas may only impose their influence through mechanisms such as attention or "top-down" perceptual states, which would require an awake animal. An attention-induced enhancement in the responses of neurons has previously described in monkey areas V1 and V4 that is the putative homologue of area 21a in cats [13,18]. Therefore, it is likely that the mechanism involving the attention-free spatial frequency-dependent influence from area 21a to areas 17 and 18 in anesthetized animals may also play an essential role in processing visual attention and perception in alert animals.

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References

[1] S. Bisti, G. Carmignoto, L. Galli, L. Maffei, Spatial-frequency characteristics of neurones of area 18 in the cat: dependence on the velocity of the visual stimulus, J. Physiol. 359 (1985) 259–268.
[2] X. Chen, C. Sun, L. Huang, T. Shou, Selective loss of orientation column maps in cat.s area 17 during brief intraocular pressure elevation, Invest. Ophthalmol. Visual Sci. 44 (2003) 435–441.
[3] B. Dreher, Thalamocortical and corticocortical interconnections in the cat visual system: relation to the mechanisms of information processing, in: J.D. Pettigrew, K.J. Sanderson, W.R. Levick (Eds.), Visual Neuroscience, Cambridge Univ. Press, Cambridge, 1986, pp. 290–314.
[4] B. Dreher, A. Michalski, R.H.T. Ho, C.W.F. Lee, W. Burke, Processing of form and motion in area 21a of cat visual cortex, Vis. Neurosci. 10 (1993) 93–115.
[5] B. Dreher, R.L. Djavadian, K.J. Turlejski, C. Wang, Areas PMLS and 21a of cat visual cortex are not only functionally but also hodologically distinct, Prog. Brain Res. 112 (1996) 251–276.
[6] D.J. Felleman, D.C. Van Essen, Distributed hierarchical processing in the primate cerebral cortex, Cereb. Cortex 1 (1991) 1–47.
[7] L. Galli, L. Chalupa, L. Maffei, S. Bisti, The organization of receptive fields in area 18 neurones of the cat varies with the spatio-temporal characteristics of the visual stimulus, Exp. Brain Res. 71 (1988) 1–7.
[8] R.A. Galuske, K.E. Schmidt, R. Goebel, S.G. Lomber, B.R. Payne, The role of feedback in shaping neural representations in cat visual cortex, Proc. Natl. Acad. Sci. U. S. A. 99 (2002) 17083–17088.
[9] C.P. Hung, B.M. Ramsden, L.M. Chen, A.W. Roe, Building surfaces from borders in areas 17 and 18 of the cat, Vis. Res. 41 (2001) 1389–1407.
[10] J.M. Hupe, A.C. James, B.R. Payne, S.G. Lomber, P. Girard, J. Bullier, Cortical feedback improves discrimination between figure and background by V1 V2 and V3 neurons, Nature 394 (1998) 784–787.
[11] J.M. Hupe, G. Choulet, J. Bullier, Spatial and temporal parameters of cortical inactivity by GABA, J. Neurosci. Methods 86 (1999) 129–143.
[12] N.P. Issa, C. Trepel, M.P. Stryker, Spatial frequency maps in cat visual cortex, J. Neurosci. 20 (2000) 8504–8514.
[13] C.J. McAdams, J.H.R. Maunsell, Effects of attention on orientation-tuning functions of single neurons in macaque cortical area V4, J. Neurosci. 19 (1999) 431–441.
[14] A. Michalski, B.M. Wimborne, G.H. Henry, The role of ipsilateral and contralateral inputs from primary cortex in responses of area 21a neurons in cats, Vis. Neurosci. 11 (1994) 839–849.
[15] J.W. Morley, R.M. Vickery, Spatial and temporal frequency selectivity of cells in area 21a of the cat, J. Physiol. (Lond.) 501 (1997) 405–413.
[16] J.W. Morley, L. Yuan, R.M. Vickery, Corticocortical connections between area 21a and primary visual cortex in the cat, NeuroReport 8 (1997) 1263–1266.
[17] J.A. Movshon, I.D. Thompson, D.J. Tohurst, Spatial and temporal contrast sensitivity of neurons in areas 17 and 18 of the cat's visual cortex, J. Physiol. (Lond.) 283 (1978) 101–120.
[18] B.G. Payne, Evidence for visual cortical area homologs in cat and macaque monkey, Cereb. Cortex 3 (1993) 1–25.
[19] K.S. Rockland, The organization of feedback connections from area V2 (18) to V1 (17), in: A. Peters, K.S. Rockland (Eds.), Primary Visual Cortex in Primates, Cerebral Cortex, vol. 10, Plenum, New York, 1994, pp. 261–299.
[20] J.H. Salin, J. Bullier, Corticocortical connections in the visual system structure and function, Physiol. Rev. 71 (1995) 107–154.
[21] J.H. Sandell, P.H. Schiller, Effect of cooling area 18 on striate cortex cell in the squirrel monkey, J. Neurophysiol. 48 (1982) 38–48.
[22] T. Shou, X. Li, Y. Zhou, B. Hu, Adaptation of visually evoked responses of relay cells in the dorsal lateral geniculate nucleus of the cat following prolonged exposure to drifting gratings, Vis. Neurosci. 13 (1996) 607–614.
[23] P.D. Spear, Functions of extrastriate visual cortex in non-primate species, in: A.G. Leventhal (Ed.), The Neural Basis of Visual Function. Vision and Visual Dysfunction, vol. 4, MacMillan, London, 1991, pp. 339–370.
[24] L.J. Toth, D.S. Kim, D.S. Rao, M. Sur, Integration of local inputs in visual cortex, Cereb. Cortex 7 (1997) 703–710.
[25] R.J. Tusa, L.A. Palmer, Retinotopic organization of area 20 and 21a in the cat, J. Comp. Neurol. 193 (1980) 147–164.
[26] R.J. Tusa, L.A. Palmer, A.C. Rosenquist, The retinotopic organization of area 17 striate cortex in the cat, J. Comp. Neurol. 177 (1978) 213–236.

[27] R.J. Tusa, A.C. Rosenquist, L.A. Palman, Retinotopic organization of areas 18 and 19 in the cat, J. Comp. Neurol. 185 (1979) 657–678.
[28] M.W. von Grünau, T.J. Zumbroich, C. Poulin, Visual receptive field properties in the posterior suprasylvian cortex of the cat: a comparison between the areas PMLS and PLLS, Vis. Res. 27 (1987) 343–356.
[29] C. Wang, B. Dreher, Binocular interactions and disparity coding in area 21a of cat extrastriate visual cortex, Exp. Brain Res. 108 (1996) 257–272.
[30] C. Wang, J. Waleszczyk, J.W. Burke, B. Dreher, Modulatory influence of feedback projections from area 21a on neuronal activities in striate cortex of the cat, Cereb. Cortex 10 (2000) 1217–1232.
[31] B.M. Wimborne, G.H. Henry, Response characteristics of the cells of cortical area 21a of the cat with special reference to orientation specificity, J. Physiol. (Lond.) 449 (1992) 457–478.
[32] M. Yashida, Y. Nagatsuka, S. Muramatsu, K. Niijima, Differential roles of the caudate nucleus and putamen in motor behavior of the cat as investigated by local injection of GABA antagonists, Neurosci. Res. 10 (1991) 34–51.
[33] T.J. Zumbroich, C. Blakemore, Spatial and temporal selectivity in suprasylvian visual cortex of the cat, J. Neurosci. 7 (1987) 482–500.