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Feedback effects from cortical area MT modulate response properties of lateral geniculate nucleus cells

H.E. Jones¹, W. Wang¹, I.M. Andolina¹, T.E. Salt¹, K.L. Grieve², J. Cudeiro³ and A.M. Sillito¹

¹Institute of Ophthalmology, Dept of Visual Science, UCL, London, UK, ²Faculty of Life Sciences, University of Manchester, Manchester, UK and ³Dept. Med-Neurocom, University of A Coruna, Coruna, Spain

The visual cortical motion area, MT/V5, provides a feedback projection to layers 1, 4B and 6 of the primary visual cortex (V1). It has been recognized for some time that the projection to layer 6 of V1 provides the potential for MT to influence the cells providing corticofugal feedback to both the magno and parvocellular streams (Shipp & Zeki, 1989). Here we report data showing that very focal pharmacological manipulation of response magnitude in area MT of the macaque, by iontophoretic application of the GABA_B receptor antagonist CGP 55845, produces clear changes in the response properties of cells in the lateral geniculate nucleus (LGN) in magno, parvo and koniocellular streams.

Animals were premedicated with atropine sulphate (0.04mg/kg i.m.) and acepromazine maleate (0.05mg/kg i.m.). Anaesthesia was induced by injection of ketamine (10-15mg/kg i.m.). Surgical procedures were carried out under ketamine anaesthesia (10-15mg/kg i.m.). Bupivicaine hydrochloride (0.75% w/v) was applied to all wound margins. Throughout the course of the experiment, anaesthesia was maintained with sufentanil (4-8µg/kg/h i.v.) and a mixture of 70% $\rm N_2O$ and 30% $\rm O_2$. Recordings were carried out in the presence of neuromuscular blockade (0.1mg/kg/h vecuronium bromide i.v.). See Jones $\it et\,al.$ (2001) for full details of the precautions taken to ensure adequacy of anaesthesia.

We took data from 55 LGN cells (30 parvocellular, 15 koniocellular and 10 magnocellular layer cells). In the presence of enhanced visual responses in MT (monitored by simultaneous recording at the site of drug application) we observed significant and reversible changes in visual response magnitude for 75% of these (41/55 cells). Changes in response magnitude were seen in cells in parvo-, konio- and magno-cellular layers (23 parvo, 10 konio and 8 magno). Of these, approximately half (21) showed response increases (mean increase 116±33% (SEM)) and half response decreases (mean decrease 40±5% (SEM)).

Increases and decreases were seen in magno, parvo and koniocellular cells. In a further experiment we compared the responses of LGN cells to a grating drifting in the preferred direction of cells at the MT drug application site and in the opposite direction. In the presence of drug application (in this case iontophoretic application of GABA to reduce MT responsiveness) we observed directionally specific effects (see Fig. 1) on 4/8 of the LGN cells tested.

These preliminary findings suggest that feedback from MT via V1 influences the responses of LGN cells to moving stimuli and has the capacity to modulate their responses in a directionally specific manner.



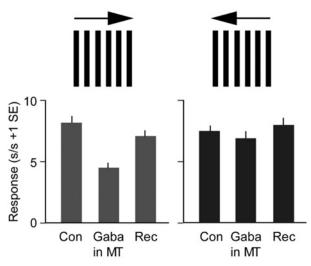


Figure 1. Top, location of MT and LGN cell receptive fields. Middle, iconic representation of stimuli. Bottom, LGN cell responses before (Con), during (GABA in MT) and after (Rec) focal decrease of MT visual responses to a grating drifting in the preferred (left) and non-preferred (right) directions of the MT cell.

Shipp S & Zeki S (1989). Eur J Neurosci 1, 309-332.

Jones HE et al. (2001). J Neurophysiol 88, 2796-2808.

Where applicable, the authors confirm that the experiments described here conform with the Physiological Society ethical requirements.

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Distributed encoding of face identity and face emotion information by ensembles of neurons in sheep temporal cortex

H. Fischer, A.J. Tate and K.M. Kendrick

Cognitive Neuroscience, The Babraham Institute, Cambridge, UK

The representation of face-based identity and emotion displays in the temporal cortex of sheep was investigated using behavioural test protocols in combination with bilateral 128 channel multi-electrode array (MEA) electrophysiology. Sheep were trained to discriminate between neutral and emotional facial displays of familiar vs. unfamiliar sheep. MEA arrays were inserted under halothane anaesthesia. ITC activity and response latency maps recorded during face emotion recognition (FER) tasks were analysed. During the tests, sheep showed a 92±2.8% (mean±SD) preference for sheep faces displaying a calm expression over faces displaying signs of stress/anxiety (i.e. enlarged protruding eyes, pupils showing the whites, flattened ears and flared nostrils) (N=5 animals, n=40 trials, t test, P<0.05). When the calm face in the pairs of familiar sheep was replaced by a calm face of an

unfamiliar sheep, the animals maintained a significant preference for the calm face (N=5, n=40, P<0.05). These behavioural findings suggest that face emotion information can override face identity information in determining preference. MEA data comprised on average 236±15 neurons per hemisphere (N=3 animals), of which 12.3±4.2% responded significantly to face stimuli with either an increase or a decrease in firing rate (t test, P<0.05). The array response was calculated as the percentage of neurons changing their activity in response to a given face. The difference in array response across trials was 5.8±1.8% (N=3 animals, 10 trials/hemisphere) for a particular face or shape. However, the difference in array response between different faces or expressions was 16.8±2.8% (3 different faces, ANOVA, F=4.64, df=2, P<0.05). This suggests that population-encoding of a particular face identity/expression requires changes in a small proportion of neurons. In addition, our data show a 34.4±5.2% reduction in the number of ITC cells responding with an increased firing rate to familiar faces independent of the facial expression (sparsening), compared to unfamiliar faces (data from 60 trials in 3 individuals). Furthermore, the number of neurons with a reduced firing rate increased by $32.3\pm4.9\%$ (N=3, n=60). For each hemisphere, the overall response latencies of the investigated populations of neurons were not significantly different in response to face identity (283±102ms, n=275) or facial expression (298±123ms, n=289, P<0.05, data pooled from 3 individuals). Between both hemispheres, the response latencies of neurons during familiarity tasks were compared to FaBER tasks. During tasks addressing familiarity alone, the average latency was 68.7±36.1ms shorter in the right hemisphere whereas during FER tasks, this latency was 35.4 ± 43.2 ms (N=3, n=70, P<0.05). This suggests that right hemisphere dominance may be less pronounced during FER than during face identity recognition. In summary, our data point towards functionally lateralised ensembles for encoding face identity and expression using sparse codes in combination with distributed representation.

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Developmental changes in synaptic plasticity in the retinocollicular pathway of the mouse

A.L. Georgiou¹, S. Maione² and T.E. Salt¹

¹Institute of Ophthalmology, UCL, London, UK and ²University of Naples, Naples, Italy

The rodent superior colliculus (SC) is a major target of retinal axons. Synaptic plasticity in the form of long-term depression

(LTD) and long-term potentiation (LTP) is known to occur in the rat retino-collicular pathway, and this is known to change with age during early postnatal development (Lo & Mize, 2002; Mize & Salt, 2004), with a peak in LTD expression occurring at about postnatal day 9 (P9). The aim of the present study was to investigate these processes in the pigmented mouse. Parasagital slices (350µm) of the superior colliculus were prepared from C57BL/6 mice (age P8 to 10weeks) as previously described for the rat (Mize & Salt, 2004). Recordings of field excitatory postsynaptic potential (fEPSP) responses to submaximal stimulation of the optic tract (OT) were made in the superficial gray layer of the SC. After a stable control period (at least 15 min) of responses to pairs of test stimuli (0.1ms pulses, 20ms separation) repeated at 30s intervals, a 50Hz 20s tetanus was applied, following which test stimulation was resumed and responses were recorded for a further 60 min (Mize & Salt, 2004).

In all experiments, the tetanus resulted in a period of shortterm depression. In slices from young mice this was followed by a period of LTD (reduction of responses to less than 95% of control values). In P8-P13 mice (n=5), fEPSPs were reduced to 76 \pm 8.1% (mean \pm standard error of mean, n=6 slices) of control amplitude 50-60 minutes after tetanus, and in P14-P17 mice (n=3) they were reduced to 82±5.7% of control (n=5 slices). During LTD, there was also a reduction in paired-pulse depression or increase in paired-pulse potentiation in slices from young mice. In contrast, in SC slices from adults (age 5-10 weeks, 7 mice) the effects of tetanus were less uniform, and either LTD (78±8.7% of control, n=3 slices), LTP (increase of responses to more than 105% of control values) (125±6% of control, n=5 slices), or no effect (102 \pm 1.7% of control, n=3slices) was observed. Overall, in adult mice fEPSPs were 106±7.1% (n=11 slices) of control amplitude 50-60 minutes after tetanus.

These findings indicate that there is a period of synaptic plasticity in young mice that manifests itself as LTD. This has similar characteristics to the LTD described previously in the young rat (Lo & Mize, 2002; Mize & Salt, 2004), and this coincides with a period of refinement of the retino-collicular pathway. In contrast, in adult mice a consistent LTD was not seen. This suggests that mechanisms of synaptic plasticity in adult mouse SC differ from those in developing mice.

Lo FS & Mize RR (2002). Eur J Neurosci 15, 1421-1432.

Mize RR & Salt TE (2004). Eur J Neurosci 20, 1331-1340.

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