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Investigating the neurochemistry of human binocular vision

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The visual brain needs balanced input from the two eyes to see in 3D. This balance is likely established by mutual inhibition, whereby activation of one eye inhibits input from the other eye. Thus, when input from the two eyes is comparable, both will contribute equally to binocular vision to promote 3D vision. However, if input from one eye is consistently weaker, the other eye may dominate. Imbalanced vision between the eyes is a hallmark of pathological eye dominance in amblyopia, also known as lazy eye.

In this talk I will describe a sequence of experiments designed to understand the neurochemical interactions involved in binocular vision. The first experiment used magnetic resonance spectroscopy (MRS) to investigate whether temporarily disturbing the balance between the eyes using short term monocular patching (2.5 hours) changes the level of inhibitory neurotransmitter GABA in the primary visual cortex (V1). This type of patching has previously been shown to increase the relative strength of vision in the patched eye. We found that the perceptual boost in the deprived eye correlated with the change in cortical inhibition, measured by GABA concentration. Thus, temporary disruption of ocular balance appears to be associated with changes in neurochemistry.

To relate neurotransmitter levels during visual stimulation to the more commonly used fMRI-BOLD signal, we developed a combined fMRI-MRS sequence. To establish the feasibility of this approach, we showed (i) stimulating the visual system with flickering checkerboards caused an increase in excitatory neurotransmitter glutamate that correlated with the change in BOLD signal and (ii) this type of concentration change only occurred when using high contrast stimuli.

Finally, we applied this technique to test whether GABA concentration in normally sighted participants was related to subtle, but reliable, imbalance in vision between eyes, when stimulated with flickering checkerboards. We found that eye dominance correlated with the difference in GABA concentration when stimulating the dominant, compared to the non-dominant eye. However, the difference in BOLD signal when stimulating the two eyes did not show any relationship with eye dominance.

Thus, we provide evidence that subtle imbalances in vision between the eyes of people with normal vision is related to GABAergic inhibition. These findings support the idea of mutual inhibition and may be useful when investigating binocular vision disorders such as amblyopia.