

Emotional vision

Ralph Adolphs

The perception of a stimulus can result in an emotional response, as we all know, but modulation of perception by emotion has been more difficult to demonstrate. A new study combines imaging and patient data to point to an anatomical substrate for such an effect, raising important implications regarding how sensory-processing impairments might arise in affective disorders.

Optimists see the world through rose-colored glasses; both rage and love can blind us. We usually take such metaphors to refer to our judgments, rather than to the perceptions on which they are based. A new study in this issue¹ uses an elegant combination of lesion and imaging techniques to add to existing evidence^{2,3} that this assumption may be mistaken: emotion can directly influence sensory processing, and can do so at surprisingly early stages.

Cells within temporal visual cortices have long been known to show robust responses to faces, which are modulated by two factors: attention and emotion. While the effects of attention are likely to arise from a number of different neural regions, those due to emotion have been hypothesized to arise from the amygdala⁴, a proposal that is consistent with the amygdala's role in emotion processing⁵ and with its projections to all temporal visual cortices⁶. To test this idea, the new study¹ compared emotional modulation of brain activity in the visual cortex of people with or without amygdala damage. As predicted, amygdala damage abolished emotional modulation; moreover, it did so selectively, leaving modulation due to attention unaffected.

Subjects were shown faces that either did or did not demand attention, and that either did (fearful expressions) or did not (neutral expressions) show strong emotion. The authors measured regional activation in visual cortical areas, and statistically assessed the degree of enhancement of that activity caused by these factors. Two groups

of subjects were tested: healthy individuals, and patients suffering from medial temporal sclerosis, a condition that results in cell loss in the medial temporal lobe due to long-standing epilepsy. The disease had left the patients with varying degrees of damage to the amygdala and/or the hippocampus, and the authors capitalized on this variance by investigating its relationship with the variance in visual-cortex activation during the task.

In strong support of a role for the amygdala in emotional enhancement of visual cortex activation, amygdala damage not only resulted in lower emotional modulation of the visual cortex compared to normal individuals, but also correlated significantly with the degree of modulation (unlike damage to the hippocampus). The combined use of functional-imaging and lesion methods is a particularly powerful approach to understanding structure-function relationships in the brain, going beyond some of the shortcomings of each technique used in isolation. A prior study, for instance, found correlated activation of the amygdala and visual cortices when healthy individuals viewed emotional stimuli⁷, but the present study's inclusion of lesion subjects provides evidence that the correlation is causal.

Several questions remain open, including what mechanism underlies this modulation. Anatomical studies in monkeys have shown direct and indirect feedback connections from the amygdala to visual cortices⁶ (Fig. 1). These anatomical connections could be responsible for the close correspondence between amygdala damage and impaired emotional modulation in visual cortices. Neurons from the basal amygdala project directly to the border of layers 1 and 2 in the visual cortex, as well as to deeper layers in more anterior temporal cortices, where they make diffuse connec-

tions (J. Freese and D. G. Amaral, personal communication; K. Rockland, personal communication). They probably synapse with apical dendrites of pyramidal neurons, using glutamate as a neurotransmitter, but the functional details of these projections are still unknown. BOLD signals measured with fMRI are thought to correlate better with the synaptic inputs to a region than with spiking outputs⁸, so it is conceivable that the signals measured in the visual cortex in this study¹ largely or even solely reflect changes in synaptic input originating from the amygdala, rather than changes in the firing rates of visual cortical neurons themselves.

There are potentially other, less direct, ways in which the amygdala could influence visual cortical activity. One possibility could be that subjects make different eye movements depending on whether they fixate fearful or neutral faces, and that the resultant difference in visual information at the retina could account for the observed differences in visual-cortex activation. Amygdala damage then could influence visual cortical activity indirectly through its influence on eye movements related to fearful versus neutral stimuli. This is unlikely to account for the present findings¹, as the stimuli were presented too briefly (200 ms) to permit such eye movements, and fixations were monitored and did not noticeably differ between subject groups. Another possibility is that the amygdala influences visual processing indirectly through a modulating influence on attention, which in turn modulates vision. Potential anatomical candidates for such a mechanism might include amygdala projections to the basal forebrain or to prefrontal cortices, but there are so far no data to implicate these pathways in the effect studied in this paper.

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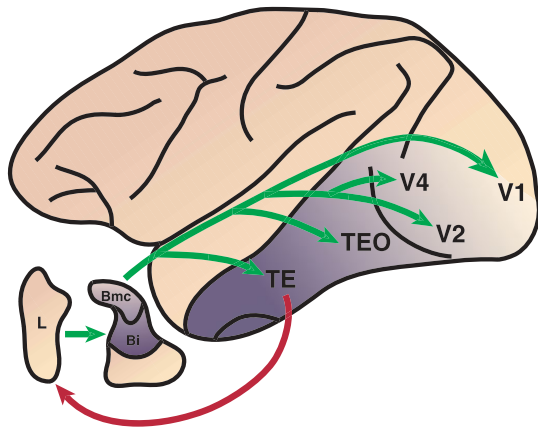


Figure 1 Anatomical connections of the amygdala with visual cortices in the monkey. Nuclei of the amygdala are shown in the lower left: the lateral nucleus (L) receives input from high-level visual cortices in the temporal lobe, whereas sectors of the basal nucleus (Bmc, Bi) in turn project to all temporal visual cortices as well as V1. Darker shading indicates denser projections. Courtesy of David G. Amaral.

Furthermore, it is still not known how visual information from the retina reaches the amygdala in the first place. At least two non-exclusive possibilities exist. Visual information could reach the amygdala via the same visual cortices that it subsequently modulates (Fig. 1). Alternatively, coarse visual information might reach the amygdala rapidly via subcortical pathways involving the superior colliculus. Although direct neuroanatomical evidence for such a route remains unclear, the idea receives support from studies of a patient with blindsight⁹: despite the destruction of primary visual cortex (resulting in a lack of conscious vision in the blind visual field), the patient can still discriminate emotional facial expressions in forced-choice tasks, and shows differential activation of the amygdala when presented with these faces. This presumptive subcortical pathway would allow the amygdala to modulate visual cortices at the earliest response latencies and without requiring any awareness of the stimuli. These two consequences would also account for findings, respectively, of very short (60 to 80 ms) latencies to emotional faces obtained in ERP studies of the occipital cortex, and of amygdala responses to faces without visual awareness achieved with binocular rivalry^{10,11}.

It remains to be confirmed whether the effects of chronic amygdala damage are the same as those caused by acute lesions, a type of experiment that is only feasible in animals. The new findings¹ support the hypothesis that the amygdala normally provides the 'boost' to visual cortices that accounts for their increased activity in response to fearful faces. This hypothesis suggests that acutely shutting down the amygdala should reduce

or abolish that modulation. In the case of the patients in this study, the amygdala had slowly atrophied over the course of years; it is therefore possible that the anatomical substrate would not be entirely comparable to an acute case, due to neural reorganization in the degenerating pathways and reorganization of processing elsewhere. It would therefore be interesting to see a replication of this finding in monkeys with experimentally induced lesions of the amygdala, ideally using single-unit recordings in the visual cortices. Monkey temporal-lobe neurons show a modulation by emotion in faces¹², but whether this is attributable to projections from the amygdala remains unknown.

All these data support the idea that emotional significance indeed modulates visual regions of the brain via the amygdala, but they still leave questions about the psychological and behavioral consequences of this modulation unresolved. Do fearful faces literally appear different to subjects with amygdala damage than they do to healthy individuals? And is the impaired ability of such patients to recognize fear in faces¹³ a result (in part) of a failure to modulate the visual cortex? Although the former question may be difficult to answer, the authors of the study probed the latter one by asking the subjects to identify the emotions shown in the faces, and found no impairment in the group with amygdala damage. This is in contrast to prior work³ demonstrating that amygdala damage does result in impairments in perceptual processing of emotional stimuli, although no correlation with visual-cortex activation was examined in that study. Perhaps more sensitive behavioral tasks, with implicit or psychophysiological measures, might reveal an impairment—it would

be deeply puzzling to find absolutely no behavioral consequence of the impaired neural activation.

The implications of the new findings are broad. Amygdala dysfunction has been posited in an array of psychiatric disorders, and so it is possible that perceptual processing of emotional stimuli is impaired in such populations. When depressed, anxious or manic people give abnormal judgments of facial expressions, they may not only be interpreting what they see differently, they may literally perceive the stimuli themselves abnormally. The abnormal activation of the fusiform gyrus and amygdala observed in people with autism when they view faces may result from impairments in the same mechanism¹⁴. One might even speculate that dysfunctional amygdala modulation of visual cortices early in life might contribute to the development of abnormal social perception in that disease. Sensory-processing impairments could thus conceivably contribute to neuropsychiatric diseases as a result of abnormal modulation by the amygdala.

The amygdala is also influenced by how we regulate and control our affective responses. When subjects are instructed to purposefully maintain a negative emotional state in response to aversive pictures, for example, amygdala activation is increased¹⁵. Does this mean that we have some control over how we see things in deciding how we feel about them? If so, self-regulation of emotions, and its dysregulation in disease, could influence nearly all information processing in its earliest stages.

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