Emotional memory: **What does the amygdala do?**

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Recent studies of the human amygdala have shed new light on its roles in two distinct, but related processes: emotional memory and the evaluation of emotional stimuli.

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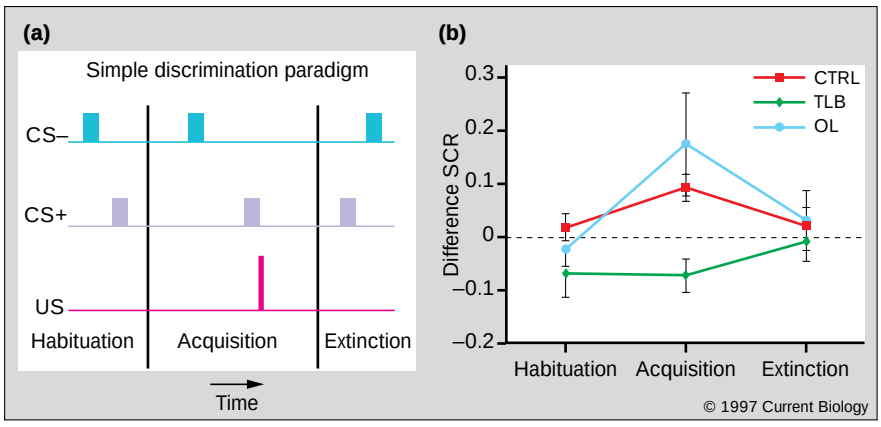
Until recently, our understanding of the neural processes underlying emotion was derived primarily from research with animals other than humans. Although these animal models have been extremely useful in identifying brain systems, few would disagree that the emotional life of humans may not lend itself to easy analogy across species. For this reason, it is especially important that animal systems for studying the neural basis of emotion are tested for their applicability to human experience. One brain structure that has emerged as a focus of emotion research in non-human animals is the amygdala, a small almond-shaped structure located anterior to the hippocampus in the medial temporal lobe. Studies of the role of the amygdala in non-human animals suggest that it may be necessary for learning or assessing the emotional significance of events [1,2].

In humans, the function of the amygdala has been difficult to study for several reasons. Firstly, research into brain–behavior correlates in humans has relied heavily on patients with lesions, and discrete lesions to the amygdala are extremely rare. Patients with amygdala damage always have other brain abnormalities or congenital disorders that must be taken into consideration when interpreting results. Secondly, studies of the amygdala in humans using new imaging techniques have been slow to emerge, because the amygdala is a small structure, close in size to the resolution of positron emission tomography (PET), and in a location that is subject to significant noise in functional magnetic resonance imaging (fMRI). Lastly, emotion is a broad concept that is not well defined in traditional psychology, making the selection of appropriate tasks for studying emotion difficult. In spite of these drawbacks, a body of literature on the role of the amygdala in humans is beginning to emerge. Studies of the behavioral role of the human amygdala generally fall into two categories, emotional memory and the evaluation of emotional stimuli, and we shall consider these in turn.

Emotional memory

There is an extensive literature showing that, in non-human animals, the amygdala is involved in emotional memory tasks [2]. The task most often studied is aversive or 'fear' conditioning, where the subject learns to associate a random stimulus — the conditioned stimulus (CS) — with a fear-inducing stimulus — the unconditioned stimulus (US). A study conducted in our laboratory [3] found a deficit in aversive conditioning in a large group of patients who had undergone unilateral temporal lobectomy that included the amygdala (Fig. 1). Bechara *et al.* [4] found a similar deficit in a patient suffering from Urbach–Weithe

Figure 1



(a) The simple discrimination paradigm used to show a deficit in averse conditioning in patients with unilateral temporal lobe lesions affecting the amygdala [3]. The conditioned stimuli (CS+ and CS–) were two tones of different frequencies; the unconditioned stimulus (US) was a burst of white noise. During the acquisition period, each presentation of the CS+ was immediately followed by the US. (b) The results of the simple discrimination paradigm for epileptic patients with unilateral temporal lobe lesions (TLB), epileptic patients with lesions outside the temporal lobe (OL), and normal control subjects (CTRL). The measure of conditioning is the difference in skin conductance response (SCR) between CS+ trials and CS– trials, grouped across the three phases of conditioning. (Adapted from [3].)

syndrome, a congenital dermatological disorder that can lead to bilateral mineralization of the amygdaloid region. In both studies, patients with amygdala damage demonstrated normal unconditioned responses and were able to report verbally the relationship between the CS and US.

These findings on humans with amygdala damage are consistent with a large body of research on non-human animals suggesting that the amygdala plays a critical role in aversive conditioning. Human emotional memory, however, is more complex than simple conditioning, and it is important to understand how this deficit may extend to tasks more representative of human memory. It has been shown that emotion can lead to enhanced memory performance, particularly when memory is tested after a delay [5]. Cahill and colleagues [6,7] found an impairment in recall for the arousing portion of a story in a patient with Urbach–Wiethe syndrome and in normal subjects who had been given propranolol, a beta-adrenergic blocker that has been shown to have similar behavioral effects as amygdala lesions in rats [8]. These results suggest that the human amygdala may be necessary for our enhanced memory for arousing events.

Two recent studies from our laboratory suggest that arousal may be a key component in an amygdala-based memory system. The first study (unpublished data) used words that were arousing as measured by skin conductance response. Normal subjects show different forgetting curves for arousing and non-arousing stimuli, so that arousing stimuli are remembered better over time. Patients with amygdala damage did not show this differential pattern of forgetting, suggesting that one role of the amygdala in human memory may be to enhance the long-term consolidation of arousing events. In the second study [9], patients with amygdala damage showed intact recall for words that were emotional in meaning, but not arousing as measured by skin conductance response.

Further support for the role of the amygdala in the consolidation of memory for arousing events comes from a PET study conducted by Cahill and colleagues [10]. They reported that activation in the amygdala was correlated with later recall for emotionally arousing, but not neutral, film clips. And another PET study found amygdala activation during retrieval of autobiographical events [11]. These various findings suggest that arousal is a key component of the amygdala memory system; they are consistent with animal studies which have also suggested a role for the amygdala in memory consolidation [8].

Although we are just beginning to examine the role of the amygdala in human memory, some preliminary conclusions can be drawn. First, patients with amygdala damage exhibit intact responses to arousing events, confirming that the effects are specifically on memory and not perception. Second, the results of fear-conditioning tests imply there is a dichotomy between declarative (explicit) knowledge of a CS–US association, thought to be mediated by the hippocampus, and learning as measured by physiological responses, which is mediated by the amygdala. The patients with amygdala damage were able to report the CS–US contingency, even though they did not show skin conductance responses to the CS, indicating these two memory systems can operate independently. Third, the studies examining declarative memory (recall, for example) for arousing events indicate that an amygdala-mediated memory system can modulate hippocampal learning, perhaps by enhancing long-term consolidation.

Evaluation of emotional stimuli

A fairly coherent picture of the role of the amygdala in humans is emerging, but our picture of the role of the amygdala in the evaluation of emotional stimuli is less clear. There is an extensive literature showing that non-human animals suffer deficits in the evaluation of stimuli, particularly social cues, after amygdala damage [12]. However, the types of behavior studied in these other species — such as aggressiveness, sexuality and social rank in monkeys — may not be easily generalizable to humans. In humans, it is difficult to evaluate the overall social functioning of the few reported cases of bilateral amygdala damage, as social abilities vary widely among normal subjects and it is unclear how to define 'normal' social behavior. It is clear from the few reported cases, however, that humans with bilateral amygdala damage do not show markedly impaired social functioning [13,14].

More specific tests of how emotional stimuli are evaluated following amygdala damage in humans have primarily focused on responses to faces. Adolphs *et al.* [15] examined 'facial-affect' processing in a Urbach–Wiethe syndrome patient (referred to as SM046). This patient could recognize faces normally, but was impaired in how she responded to facial expressions, in that her assessment of the intensity of the expression differed from controls for a subset of emotions; her deficit was especially acute for fearful faces. Two similar case studies have been reported by Young and colleagues [16,17]. Imaging studies have also observed amygdala involvement in processing specific facial expressions. A PET study [18] reported activation in the amygdala when subjects viewed fearful faces in comparison to neutral faces. An fMRI study [19] reported similar observations, but also found activation for happy compared to neutral faces. These studies suggest one function of the human amygdala may be the interpretation of facial affect, particularly fear.

There are, however, cases of patients with bilateral amygdala damage who do not show a deficit in facial-affect processing. Hamann *et al.* [20] reported two patients with bilateral amygdala damage following encephalitis. Using

the same task as Adolphs *et al.* [15], these patients gave a normal evaluation of facial expressions. Hamann *et al.* suggest that age at the time of brain injury may be one critical difference between their patients and those in other case studies. Consistent with this hypothesis, Anderson *et al.* [21] tested patients who had undergone unilateral temporal lobectomy, and found a correlation between age-of-onset for epilepsy and deficits in interpreting facial expressions in the patients with right-hemisphere damage — earlier onset is correlated with a greater deficit. It is clear from these results that the amygdala is somehow involved in the interpretation of facial expression, but may not always be necessary for normal facial-affect processing.

There have been only a few studies of the role of the human amygdala in evaluating emotional stimuli other than faces. Scott *et al.* [22] showed that the deficit in evaluating social cues after amygdala damage extends to the auditory domain. They described a patient with bilateral amygdala damage who exhibits a deficit in the ability to detect emotion in spoken language, which is most severe for fear and anger. A PET study has detected amygdala involvement in processing body movement [23], and an fMRI study found that the amygdala is activated when subjects are viewing emotionally arousing scenes [24]. In both of these imaging studies, the authors proposed that amygdala activation might be the result of the affective judgment of the stimuli.

The available evidence thus suggests that the human amygdala does play some role in our responses to social and emotional cues, primarily fearful faces, which is consistent with the animal literature. However, the precise role of the amygdala is difficult to assess. Given the role of the amygdala in arousal-mediated memory, one might expect the amygdala to be activated whenever the stimuli are arousing. Damage to the amygdala in humans does not, however, lead to a global deficit in the evaluation of emotional or social stimuli, and may not lead to any deficit if the amygdala is damaged later in life.

Memory *versus* evaluation: what have they in common?

Studies on non-human animals have indicated that the amygdala is important for both the acquisition and expression of emotional memories [2]. In humans, the primary role of the amygdala may be similar, and any role in the evaluation of the emotional content of a stimulus may be a secondary consequence of this function. Evaluation tasks that are difficult for patients with amygdala damage may rely on responses that were learned in arousing situations. One might expect encounters with fearful facial expressions to be particularly arousing. If, however, the human amygdala is important for both acquisition and expression of learned responses, then why would some patients with amygdala damage have intact responses when evaluating fearful faces?

These variable results may have been obtained because the responses of patients who were relatively old at the time of their lesion were 'over-learned'. A study with rats has suggested that amygdala lesions may not lead to a complete deficit in expression of a learned response if the response was over-learned before lesioning [25]. Extensive practice, or over-learning, may lead to the gradual involvement of other memory systems. If this were true for humans, then older patients, who might have over-learned how to evaluate social cues, may not show a deficit following amygdala damage.

Although the emotional experience of humans may differ from other species, what is known about the amygdala and human emotion is consistent with animal models. We hypothesize that the primary role of the human amygdala is the acquisition and expression of emotional memory. The amygdala may additionally play an important role, particularly during development, in learning how to evaluate social cues.

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References

1. Aggleton JP: *The Amygdala: Neurobiological Aspects of Emotion, Memory, and Mental Dysfunction*. New York: Wiley-Liss; 1992.
2. LeDoux J: *The Emotional Brain*. New York: Simon & Schuster; 1996.
3. LaBar KS, LeDoux JE, Spencer DD, Phelps EA: Impaired fear conditioning following unilateral temporal lobectomy in humans. *J Neurosci* 1995, 15:6846–6855.
4. Bechara A, Tranel D, Damasio H, Adolphs R, Rockland C, Damasio AR: Double dissociation of conditioning and declarative knowledge relative to the amygdala and hippocampus in humans. *Science* 1995, 269:1115–1118.
5. Christianson S-A: *The Handbook of Emotion and Memory: Research and Theory.* New Jersey: Lawrence Erlbaum Associates; 1992.
6. Cahill L, Prins B, Weber M, McGaugh JL: Beta-adrenergic activation and memory for emotional events. *Nature* 1994, 371:702–704.
7. Cahill L, Babinsky R, Markowitsch HJ, McGaugh JL: The amygdala and emotional memory. *Science* 1995, 377:295–296.
8. McGaugh JL: Affect, neuromodulatory systems, and memory storage. In *The handbook of emotion and memory: research and theory.* Edited by Christianson SA. 1992. New Jersey: Lawrence Erlbaum Associates; 1992:245–268.
9. Phelps EA, LaBar KS, Spencer DD: Emotional memory following unilateral temporal lobectomy. *Brain Cogn* 1997, 34:512–521.
10. Cahill L, Haier RJ, Fallon J, Alkire MT, Tang C, Keator D, Wu J, McGaugh JL: Amygdala activity at encoding correlated with long-term, free recall of emotional information. *Proc Natl Acad Sci USA* 1996, 93:8016–8021.
11. Fink GR, Markowitsch HJ, Reinkemeier M, Bruckbauer T, Kessler J, Heiss W-D: Cerebral representation of one's own past: neural networks involved in autobiographical memory. *J Neurosci* 1996, 16:4275–4282.
12. Kling AS, Brothers LA. The amygdala and social behavior. In *The Amygdala*. Edited by Aggleton JP. New York: Wiley; 1992:255–306.
13. Markowitsch HJ, Calabrese P, Wuerker M, Durwen HF, Kessler J, Babinsky R, Brechtelsbauer D, Heuser L, Gehlen W: The amygdala's contribution to memory: a study on two patients with Urbach–Wiethe disease. *Neuroreport* 1994, 5:1349–1352.
14. Tranel D, Damasio H: Intact electrodermal skin conductance responses after bilateral amygdala damage. *Neuropsychologia* 1989, 27:381–390.
15. Adolphs R, Tranel D, Damasio H, Damasio A: Impaired recognition of emotion in facial expressions following bilateral amygdala damage to the human amygdala. *Nature* 1994, 372:669–672.

16. Young AW, Hellawell DJ, Van De Wal C, Johnson M: Facial expression processing after amygdalotomy. *Neuropsychologia* 1996, 34:31–39.
17. Calder AJ, Young AW, Rowland D, Perrett D, Hodges JR, Etcoff NL: Facial emotion recognition after bilateral amygdala damage: differentially severe impairment of fear. *Cogn Neuropsychol* 1996, 13:699–745.
18. Morris JS, Frith CD, Perrett DI, Rowland D, Young AW, Calder AJ, Dolan RJ: A differential neural response in the human amygdala to fearful and happy facial expressions. *Nature* 1996, 383:812–815.
19. Breiter HC, Etcoff NL, Whalen PJ, Kennedy WA, Rauch SL, Buckner RL, Strauss MM, Hyman S, Rosen B: Response and habituation of the human amygdala during visual processing of facial expression. *Neuron* 1996, 17:875–887.
20. Hamann SB, Stefanacci L, Squire LR, Adolphs R, Damasio AR: Recognizing facial emotion. *Nature* 1996, 379:497.
21. Anderson AK, LaBar KS, Phelps EA: Facial affect processing abilities following unilateral temporal lobectomy. *Soc Neurosci Abstr* 1996, 22:1866.
22. Scott SK, Young AW, Calder AJ, Hellawell DJ, Aggleton JP, Johnson M: Impaired auditory recognition of fear and anger following bilateral amygdala lesions. *Nature* 1997, 385:254–257.
23. Bonda E, Petrides M, Ostry D, Evans A: Specific involvement of human parietal systems and the amygdala in the perception of biological motion. *J Neurosci* 1996, 16:3737–3744.
24. Irwin W, Davidson RJ, Lowe MJ, Moch BJ, Sorenson JA, Turski PA: Human amygdala activation detected with echo-planar functional magnetic resonance imaging. *Neuroreport* 1996, 7:1765–1769.
25. Kim M, Davis M: Electrolytic lesions of the amygdala block acquisition and expression of fear-potentiated startle. *Behav Neurosci* 1993, 107:580–595.