amygdala-fear.ml

Description

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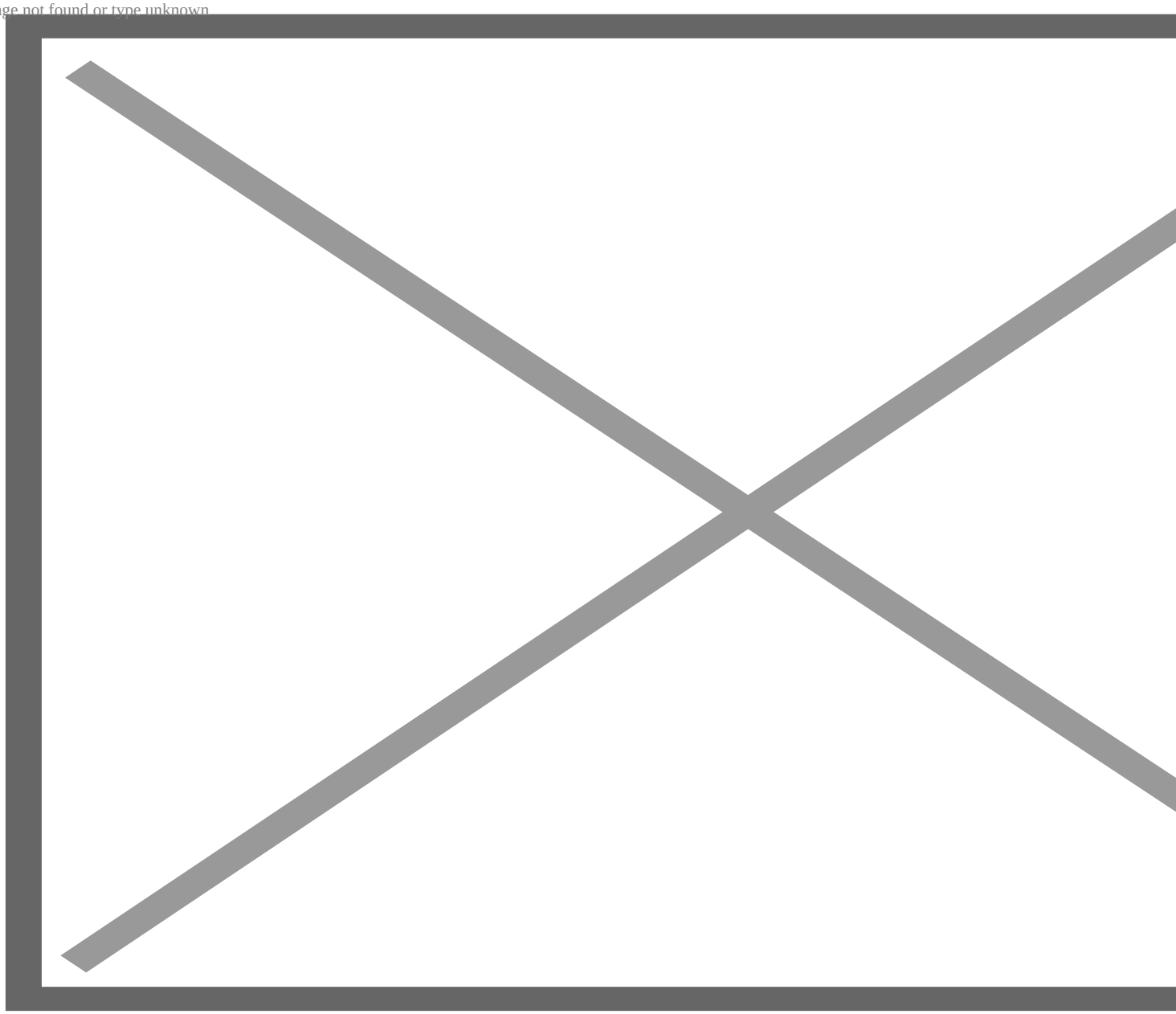

The phylogenetically ancient Amygdala : The neuroanatomical correlate of Fear, Anxiety, and Aggression

The **amygdala** (also *corpus amygdaloideum*; Greek, ????????, *amygdal?*, 'Almond', 'tonsil') is one of two almond-shaped clusters of nuclei located deep and medially within the temporal lobes of the brain in complex vertebrates, including humans. Shown in research to perform a primary role in the processing of memory, decision-making and emotional responses (including fear, anxiety, and aggression), the amygdalae are considered part of the limbic system.

Each side holds a specific function in how we perceive and process emotion. The right and left portions of the amygdala have independent memory systems, but work together to store, encode, and interpret emotion.

The right hemisphere is associated with negative emotion. t plays a role in the expression of fear and in the processing of fear-inducing stimuli. Fear conditioning, which occurs when a neutral stimulus acquires aversive properties, occurs within the right hemisphere. When an individual is presented with a conditioned, aversive stimulus, it is processed within the right amygdala, producing an unpleasant or fearful response. This emotional response conditions the individual to avoid fear-inducing stimuli and more importantly, to assess threats in the environment.

[su_youtube url="https://www.youtube.com/watch?v=YB9rs4tEAaE" width="400? responsive="no"]
www.brainfacts.org/3d-brain#intro=false&focus=Brain-limbic_system-amygdala

Structure

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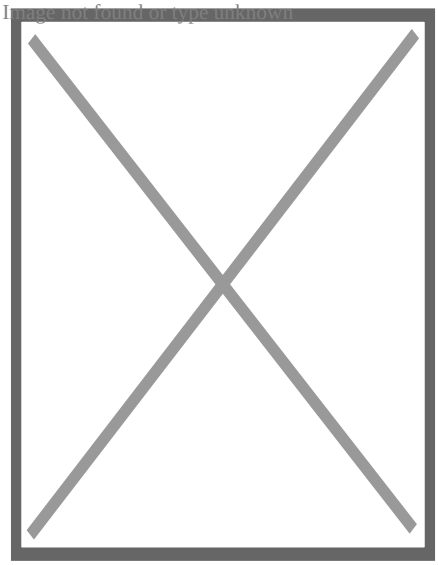

Subdivisions of the mouse amygdala

The regions described as amygdala nuclei encompass several structures with distinct connectional and functional characteristics in humans and other animals. Among these nuclei are the basolateral complex, the cortical nucleus, the medial nucleus, the central nucleus, and the intercalated cell clusters. The basolateral complex can be further subdivided into the lateral, the basal, and the accessory basal nuclei.

MRI coronal view of the amygdala

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MRI coronal view of the right amygdala

Anatomically, the amygdala, and more particularly its central and medial nuclei have sometimes been classified as a part of the basal ganglia.

Hemispheric specializations

There are functional differences between the right and left amygdala. In one study, electrical

stimulations of the right amygdala induced negative emotions, especially fear and sadness. In contrast, stimulation of the left amygdala was able to induce either pleasant (happiness) or unpleasant (fear, anxiety, sadness) emotions. Other evidence suggests that the left amygdala plays a role in the brain's reward system.

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Further References

Baxter, M. G., & Murray, E. A.. (2002). The amygdala and reward. Nature Reviews Neuroscience

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"The amygdala — an almond-shaped group of nuclei at the heart of the telencephalon — has been associated with a range of cognitive functions, including emotion, learning, memory, attention and perception. most current views of amygdala function emphasize its role in negative emotions, such as fear, and in linking negative emotions with other aspects of cognition, such as learning and memory. however, recent evidence supports a role for the amygdala in processing positive emotions as well as negative ones, including learning about the beneficial biological value of stimuli. indeed, the amygdala's role in stimulus-reward learning might be just as important as its role in processing negative affect and fear conditioning."
Li, H., Penzo, M. A., Taniguchi, H., Kopec, C. D., Huang, Z. J., & Li, B.. (2013). Experience-dependent modification of a central amygdala fear circuit. Nature Neuroscience, 16(3), 332–339.

Plain numerical DOI: 10.1038/nn.3322
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"The amygdala is essential for fear learning and expression. the central amygdala (cea), once viewed as a passive relay between the amygdala complex and downstream fear effectors, has emerged as an active participant in fear conditioning. however, the mechanism by which cea contributes to the learning and expression of fear is unclear. we found that fear conditioning in mice induced robust plasticity of excitatory synapses onto inhibitory neurons in the lateral subdivision of the cea (cel). this experience-dependent plasticity was cell specific, bidirectional and expressed presynaptically by inputs from the lateral amygdala. in particular, preventing synaptic potentiation onto somatostatin-positive neurons impaired fear memory formation. furthermore, activation of these neurons was necessary for

fear memory recall and was sufficient to drive fear responses. our findings support a model in which fear conditioning-induced synaptic modifications in cel favor the activation of somatostatin-positive neurons, which inhibit cel output, thereby disinhibiting the medial subdivision of cea and releasing fear expression."
Morris, J. S., Ohman, A., & Dolan, R. J.. (1999). A subcortical pathway to the right amygdala mediating "unseen" fear. Proceedings of the National Academy of Sciences, 96(4), 1680–1685.

Plain numerical DOI: 10.1073/pnas.96.4.1680
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"Neuroimaging studies have shown differential amygdala responses to masked ('unseen') emotional stimuli. how visual signals related to such unseen stimuli access the amygdala is unknown. a possible pathway, involving the superior colliculus and pulvinar, is suggested by observations of patients with striate cortex lesions who show preserved abilities to localize and discriminate visual stimuli that are not consciously perceived ('blindsight'). we used measures of right amygdala neural activity acquired from volunteer subjects viewing masked fear-conditioned faces to determine whether a colliculo-pulvinar pathway was engaged during processing of these unseen target stimuli. increased connectivity between right amygdala, pulvinar, and superior colliculus was evident when fear-conditioned faces were unseen rather than seen. right amygdala connectivity with fusiform and orbitofrontal cortices decreased in the same condition. by contrast, the left amygdala, whose activity did not discriminate seen and unseen fear-conditioned targets, showed no masking-dependent changes in connectivity with superior colliculus or pulvinar. these results suggest that a subcortical pathway to the right amygdala, via midbrain and thalamus, provides a route for processing behaviorally relevant unseen visual events in parallel to a cortical route necessary for conscious identification."
Phillips, R. G., & LeDoux, J. E.. (1992). Differential Contribution of Amygdala and Hippocampus to Cued and Contextual Fear Conditioning. Behavioral Neuroscience, 106(2), 274–285.

Plain numerical DOI: 10.1037/0735-7044.106.2.274
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"The contribution of the amygdala and hippocampus to the acquisition of conditioned fear responses to a cue (a tone paired with footshock) and to context (background stimuli continuously present in the apparatus in which tone-shock pairings occurred) was examined in rats. in unoperated controls, responses to the cue conditioned faster and were more resistant to extinction than were responses to contextual stimuli. lesions of the amygdala interfered with the conditioning of fear responses to both the cue and the context, whereas lesions of the hippocampus interfered with conditioning to the context but not to the cue. the amygdala is thus involved in the conditioning of fear responses to simple, modality-specific conditioned stimuli as well as to complex, polymodal stimuli, whereas the hippocampus is only involved in fear conditioning situations involving complex, polymodal events. these findings suggest an associative role for the amygdala and a sensory relay role for the

hippocampus in fear conditioning."
Phelps, E. A., Delgado, M. R., Nearing, K. I., & Ledoux, J. E.. (2004). Extinction learning in humans: Role of the amygdala and vmPFC. Neuron, 43(6), 897–905.

Plain numerical DOI: 10.1016/j.neuron.2004.08.042
DOI URL
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"Understanding how fears are acquired is an important step in translating basic research to the treatment of fear-related disorders. however, understanding how learned fears are diminished may be even more valuable. we explored the neural mechanisms of fear extinction in humans. studies of extinction in nonhuman animals have focused on two interconnected brain regions: the amygdala and the ventral medial prefrontal cortex (vmpfc). consistent with animal models suggesting that the amygdala is important for both the acquisition and extinction of conditioned fear, amygdala activation was correlated across subjects with the conditioned response in both acquisition and early extinction. activation in the vmpfc (subgenual anterior cingulate) was primarily linked to the expression of fear learning during a delayed test of extinction, as might have been expected from studies demonstrating this region is critical for the retention of extinction. these results provide evidence that the mechanisms of extinction learning may be preserved across species."
Duvarci, S., & Pare, D.. (2014). Amygdala microcircuits controlling learned fear. Neuron

Plain numerical DOI: 10.1016/j.neuron.2014.04.042
DOI URL
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"We review recent work on the role of intrinsic amygdala networks in the regulation of classically conditioned defensive behaviors, commonly known as conditioned fear. these new developments highlight how conditioned fear depends on far more complex networks than initially envisioned. indeed, multiple parallel inhibitory and excitatory circuits are differentially recruited during the expression versus extinction of conditioned fear. moreover, shifts between expression and extinction circuits involve coordinated interactions with different regions of the medial prefrontal cortex. however, key areas of uncertainty remain, particularly with respect to the connectivity of the different cell types. filling these gaps in our knowledge is important because much evidence indicates that human anxiety disorders results from an abnormal regulation of the networks supporting fear learning. duvarci and pare review recent work on the role of intrinsic amygdala networks in regulating conditioned fear, revealing that it depends on multiple parallel inhibitory and excitatory circuits that are differentially recruited during the expression versus extinction of conditioned fear. © 2014 elsevier inc."
Ehrlich, I., Humeau, Y., Grenier, F., Ciocchi, S., Herry, C., & Lüthi, A.. (2009). Amygdala Inhibitory Circuits and the Control of Fear Memory. Neuron

Plain numerical DOI: 10.1016/j.neuron.2009.05.026

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“Classical fear conditioning is a powerful behavioral paradigm that is widely used to study the neuronal substrates of learning and memory. previous studies have clearly identified the amygdala as a key brain structure for acquisition and storage of fear memory traces. whereas the majority of this work has focused on principal cells and glutamatergic transmission and its plasticity, recent studies have started to shed light on the intricate roles of local inhibitory circuits. here, we review current understanding and emerging concepts of how local inhibitory circuits in the amygdala control the acquisition, expression, and extinction of conditioned fear at different levels. © 2009 elsevier inc. all rights reserved.”
LeDoux, J. E.. (2009). Emotion Circuits in the Brain. Focus, 7(2), 274–274.

Plain numerical DOI: 10.1176/foc.7.2.foc274
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“The field of neuroscience has, after a long period of looking the other way, again embraced emotion as an important research area. much of the progress has come from studies of fear, and especially fear conditioning. this work has pin-pointed the amygdala as an important component of the system involved in the acqui-sition, storage, and expression of fear memory and has elucidated in detail how stimuli enter, travel through, and exit the amygdala. some progress has also been made in understanding the cellular and molecular mechanisms that underlie fear conditioning, and recent studies have also shown that the findings from experimental animals apply to the human brain. it is important to remember why this work on emotion succeeded where past efforts failed. it focused on a psychologically well-defined aspect of emo-tion, avoided vague and poorly defined concepts such as ” affect, ” ” hedonic tone, ” or ” emotional feelings, ” and used a simple and straightforward experimental approach.”
Maren, S., & Quirk, G. J.. (2004). Neuronal signalling of fear memory. Nature Reviews Neuroscience

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“The learning and remembering of fearful events depends on the integrity of the amygdala, but how are fear memories represented in the activity of amygdala neurons? here, we review recent electrophysiological studies indicating that neurons in the lateral amygdala encode aversive memories during the acquisition and extinction of pavlovian fear conditioning. studies that combine unit recording with brain lesions and pharmacological inactivation provide evidence that the lateral amygdala is a crucial locus of fear memory. extinction of fear memory reduces associative plasticity in the lateral amygdala and involves the hippocampus and prefrontal cortex. understanding the signalling of

aversive memory by amygdala neurons opens new avenues for research into the neural systems that support fear behaviour."
Gross, C. T., & Canteras, N. S.. (2012). The many paths to fear. Nature Reviews Neuroscience

Plain numerical DOI: 10.1038/nrn3301
DOI URL
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"Fear is an emotion that has powerful effects on behaviour and physiology across animal species. it is accepted that the amygdala has a central role in processing fear. however, it is less widely appreciated that distinct amygdala outputs and downstream circuits are involved in different types of fear. data show that fear of painful stimuli, predators and aggressive members of the same species are processed in independent neural circuits that involve the amygdala and downstream hypothalamic and brainstem circuits. here, we discuss data supporting multiple fear pathways and the implications of this distributed system for understanding and treating fear."
Karalis, N., Dejean, C., Chaudun, F., Khoder, S., R Rozeske, R., Wurtz, H., … Herry, C.. (2016). 4-Hz oscillations synchronize prefrontal-amygdala circuits during fear behavior. Nature Neuroscience, 19(4), 605–612.

Plain numerical DOI: 10.1038/nn.4251
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"Fear expression relies on the coordinated activity of prefrontal and amygdala circuits, yet the mechanisms allowing long-range network synchronization during fear remain unknown. using a combination of extracellular recordings, pharmacological and optogenetic manipulations, we found that freezing, a behavioral expression of fear, temporally coincided with the development of sustained, internally generated 4-hz oscillations in prefrontal-amygdala circuits. 4-hz oscillations predict freezing onset and offset and synchronize prefrontal-amygdala circuits. optogenetic induction of prefrontal 4-hz oscillations coordinates prefrontal-amygdala activity and elicits fear behavior. these results unravel a sustained oscillatory mechanism mediating prefrontal-amygdala coupling during fear behavior."
Haubensak, W., Kunwar, P. S., Cai, H., Ciocchi, S., Wall, N. R., Ponnusamy, R., … Anderson, D. J.. (2010). Genetic dissection of an amygdala microcircuit that gates conditioned fear. Nature, 468(7321), 270–276.

Plain numerical DOI: 10.1038/nature09553
DOI URL
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"The role of different amygdala nuclei (neuroanatomical subdivisions) in processing pavlovian

conditioned fear has been studied extensively, but the function of the heterogeneous neuronal subtypes within these nuclei remains poorly understood. here we use molecular genetic approaches to map the functional connectivity of a subpopulation of gaba-containing neurons, located in the lateral subdivision of the central amygdala (cel), which express protein kinase c-? (pkc-?). channelrhodopsin-2-assisted circuit mapping in amygdala slices and cell-specific viral tracing indicate that pkc-?(+) neurons inhibit output neurons in the medial central amygdala (cem), and also make reciprocal inhibitory synapses with pkc-?(-) neurons in cel. electrical silencing of pkc-?(+) neurons in vivo suggests that they correspond to physiologically identified units that are inhibited by the conditioned stimulus, called cel(off) units. this correspondence, together with behavioural data, defines an inhibitory microcircuit in cel that gates cem output to control the level of conditioned freezing."
Adolphs, R.. (2008). Fear, faces, and the human amygdala. Current Opinion in Neurobiology

Plain numerical DOI: 10.1016/j.conb.2008.06.006
DOI URL
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"The amygdala's historical role in processing stimuli related to threat and fear is being modified to suggest a role that is broader and more abstract. amygdala lesions impair the ability to seek out and make use of the eye region of faces, resulting in impaired fear perception. other studies in rats and humans revive earlier proposals that the amygdala is important not only for fear perception as such, but also for detecting saliency and biological relevance. debates about some features of this processing now suggest that while the amygdala can process fearful facial expressions in the absence of conscious perception, and while there is some degree of preattentive processing, this depends on the context and is not necessarily more rapid than cortical processing routes. a large current research effort extends the amygdala's putative role to a number of psychiatric illnesses. © 2008 elsevier ltd. all rights reserved."
Kirsch, P.. (2005). Oxytocin Modulates Neural Circuitry for Social Cognition and Fear in Humans. Journal of Neuroscience, 25(49), 11489–11493.

Plain numerical DOI: 10.1523/JNEUROSCI.3984-05.2005
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"In non-human mammals, the neuropeptide oxytocin is a key mediator of complex emotional and social behaviors, including attachment, social recognition, and aggression. oxytocin reduces anxiety and impacts on fear conditioning and extinction. recently, oxytocin administration in humans was shown to increase trust, suggesting involvement of the amygdala, a central component of the neurocircuitry of fear and social cognition that has been linked to trust and highly expresses oxytocin receptors in many mammals. however, no human data on the effects of this peptide on brain function were available. here, we show that human amygdala function is strongly modulated by oxytocin. we used functional magnetic resonance imaging to image amygdala activation by fear-inducing visual stimuli in 15 healthy males after double-blind crossover intranasal application of placebo or oxytocin. compared with placebo, oxytocin potently reduced activation of the amygdala and reduced coupling of the amygdala

to brainstem regions implicated in autonomic and behavioral manifestations of fear. our results indicate a neural mechanism for the effects of oxytocin in social cognition in the human brain and provide a methodology and rationale for exploring therapeutic strategies in disorders in which abnormal amygdala function has been implicated, such as social phobia or autism."
Davis, M., Walker, D. L., Miles, L., & Grillon, C.. (2010). Phasic vs sustained fear in rats and humans: Role of the extended amygdala in fear vs anxiety. Neuropsychopharmacology

Plain numerical DOI: 10.1038/npp.2009.109
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"Data will be reviewed using the acoustic startle reflex in rats and humans based on our attempts to operationally define fear vs anxiety. although the symptoms of fear and anxiety are very similar, they also differ. fear is a generally adaptive state of apprehension that begins rapidly and dissipates quickly once the threat is removed (phasic fear). anxiety is elicited by less specific and less predictable threats, or by those that are physically or psychologically more distant. thus, anxiety is a more long-lasting state of apprehension (sustained fear). rodent studies suggest that phasic fear is mediated by the amygdala, which sends outputs to the hypothalamus and brainstem to produce symptoms of fear. sustained fear is also mediated by the amygdala, which releases corticotropin-releasing factor, a stress hormone that acts on receptors in the bed nucleus of the stria terminalis (bnst), a part of the so-called 'extended amygdala.' the amygdala and bnst send outputs to the same hypothalamic and brainstem targets to produce phasic and sustained fear, respectively. in rats, sustained fear is more sensitive to anxiolytic drugs. in humans, symptoms of clinical anxiety are better detected in sustained rather than phasic fear paradigms."
SAH, P., FABER, E. S. L., LOPEZ DE ARMENTIA, M., & POWER, J.. (2003). The Amygdaloid Complex: Anatomy and Physiology. Physiological Reviews, 83(3), 803–834.

Plain numerical DOI: 10.1152/physrev.00002.2003
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"A converging body of literature over the last 50 years has implicated the amygdala in assigning emotional significance or value to sensory information. in particular, the amygdala has been shown to be an essential component of the circuitry underlying fear-related responses. disorders in the processing of fear-related information are likely to be the underlying cause of some anxiety disorders in humans such as posttraumatic stress. the amygdaloid complex is a group of more than 10 nuclei that are located in the midtemporal lobe. these nuclei can be distinguished both on cytoarchitectonic and connectional grounds. anatomical tract tracing studies have shown that these nuclei have extensive intranuclear and internuclear connections. the afferent and efferent connections of the amygdala have also been mapped in detail, showing that the amygdaloid complex has extensive connections with cortical and subcortical regions. analysis of fear conditioning in rats has suggested that long-term synaptic plasticity of inputs to the amygdala underlies the acquisition and perhaps storage of the fear memory. in agreement with this proposal, synaptic plasticity has been demonstrated at synapses in the

amygdala in both in vitro and in vivo studies. in this review, we examine the anatomical and physiological substrates proposed to underlie amygdala function."
Davis, M., & Whalen, P. J.. (2001). The amygdala: Vigilance and emotion. Molecular Psychiatry

Plain numerical DOI: 10.1038/sj.mp.4000812
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"Here we provide a review of the animal and human literature concerning the role of the amygdala in fear conditioning, considering its potential influence over autonomic and hormonal changes, motor behavior and attentional processes. a stimulus that predicts an aversive outcome will change neural transmission in the amygdala to produce the somatic, autonomic and endocrine signs of fear, as well as increased attention to that stimulus. it is now clear that the amygdala is also involved in learning about positively valenced stimuli as well as spatial and motor learning and this review strives to integrate this additional information. a review of available studies examining the human amygdala covers both lesion and electrical stimulation studies as well as the most recent functional neuroimaging studies. where appropriate, we attempt to integrate basic information on normal amygdala function with our current understanding of psychiatric disorders, including pathological anxiety."
LeDoux, J.. (2003). The emotional brain, fear, and the amygdala. Cellular and Molecular Neurobiology

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DOI URL
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"1. considerable progress has been made over the past 20 years in relating specific circuits of the brain to emotional functions. much of this work has involved studies of pavlovian or classical fear conditioning, a behavioral procedure that is used to couple meaningless environmental stimuli to emotional (defense) response networks. 2. the major conclusion from studies of fear conditioning is that the amygdala plays critical role in linking external stimuli to defense responses. 3. before describing research on the role of the amygdala in fear conditioning, though, it will be helpful to briefly examine the historical events that preceded modern research on conditioned fear."
Senn, V., Wolff, S. B. E., Herry, C., Grenier, F., Ehrlich, I., Gründemann, J., … Lüthi, A.. (2014). Long-range connectivity defines behavioral specificity of amygdala neurons. Neuron, 81(2), 428–437.

Plain numerical DOI: 10.1016/j.neuron.2013.11.006
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"Memories are acquired and encoded within large-scale neuronal networks spanning different brain areas. the anatomical and functional specificity of such long-range interactions and their role in

learning is poorly understood. the amygdala and the medial prefrontal cortex (mpfc) are interconnected brain structures involved in the extinction of conditioned fear. here, we show that a defined subpopulation of basal amygdala (ba) projection neurons targeting the prelimbic (pl) subdivision of mpfc is active during states of high fear, whereas ba neurons targeting the infralimbic (il) subdivision are recruited, and exhibit cell-type-specific plasticity, during fear extinction. pathway-specific optogenetic manipulations demonstrate that the activity balance between pathways is causally involved in fear extinction. together, our findings demonstrate that, although intermingled locally, long-range connectivity defines distinct subpopulations of amygdala projection neurons and indicate that the formation of long-term extinction memories depends on the balance of activity between two defined amygdala-prefrontal pathways. © 2014 elsevier inc."
Ciocchi, S., Herry, C., Grenier, F., Wolff, S. B. E., Letzkus, J. J., Vlachos, I., … Lüthi, A.. (2010). Encoding of conditioned fear in central amygdala inhibitory circuits. Nature, 468(7321), 277–282.

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"Nature 468, 277 (2010). doi:10.1038/nature09559"
Wolff, S. B. E., Gründemann, J., Tovote, P., Krabbe, S., Jacobson, G. A., Müller, C., … Lüthi, A.. (2014). Amygdala interneuron subtypes control fear learning through disinhibition. Nature, 509(7501), 453–458.

Plain numerical DOI: 10.1038/nature13258
DOI URL
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"Learning is mediated by experience-dependent plasticity in neuronal circuits. activity in neuronal circuits is tightly regulated by different subtypes of inhibitory interneurons, yet their role in learning is poorly understood. using a combination of in vivo single-unit recordings and optogenetic manipulations, we show that in the mouse basolateral amygdala, interneurons expressing parvalbumin (pv) and somatostatin (som) bidirectionally control the acquisition of fear conditioning–a simple form of associative learning–through two distinct disinhibitory mechanisms. during an auditory cue, pv(+) interneurons are excited and indirectly disinhibit the dendrites of basolateral amygdala principal neurons via som(+) interneurons, thereby enhancing auditory responses and promoting cue-shock associations. during an aversive footshock, however, both pv(+) and som(+) interneurons are inhibited, which boosts postsynaptic footshock responses and gates learning. these results demonstrate that associative learning is dynamically regulated by the stimulus-specific activation of distinct disinhibitory microcircuits through precise interactions between different subtypes of local interneurons."
Feinstein, J. S., Adolphs, R., Damasio, A., & Tranel, D.. (2011). The human amygdala and the induction and experience of fear. Current Biology, 21(1), 34–38.

Plain numerical DOI: 10.1016/j.cub.2010.11.042
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“Although clinical observations suggest that humans with amygdala damage have abnormal fear reactions and a reduced experience of fear [1-3], these impressions have not been systematically investigated. to address this gap, we conducted a new study in a rare human patient, sm, who has focal bilateral amygdala lesions [4]. to provoke fear in sm, we exposed her to live snakes and spiders, took her on a tour of a haunted house, and showed her emotionally evocative films. on no occasion did sm exhibit fear, and she never endorsed feeling more than minimal levels of fear. likewise, across a large battery of self-report questionnaires, 3 months of real-life experience sampling, and a life history replete with traumatic events, sm repeatedly demonstrated an absence of overt fear manifestations and an overall impoverished experience of fear. despite her lack of fear, sm is able to exhibit other basic emotions and experience the respective feelings. the findings support the conclusion that the human amygdala plays a pivotal role in triggering a state of fear and that the absence of such a state precludes the experience of fear itself. © 2011 elsevier ltd.”
Phelps, E. A., & LeDoux, J. E.. (2005). Contributions of the amygdala to emotion processing: From animal models to human behavior. Neuron

Plain numerical DOI: 10.1016/j.neuron.2005.09.025
DOI URL
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“Research on the neural systems underlying emotion in animal models over the past two decades has implicated the amygdala in fear and other emotional processes. this work stimulated interest in pursuing the brain mechanisms of emotion in humans. here, we review research on the role of the amygdala in emotional processes in both animal models and humans. the review is not exhaustive, but it highlights five major research topics that illustrate parallel roles for the amygdala in humans and other animals, including implicit emotional learning and memory, emotional modulation of memory, emotional influences on attention and perception, emotion and social behavior, and emotion inhibition and regulation. copyright ©2005 by elsevier inc.”
Olsson, A., & Phelps, E. A.. (2007). Social learning of fear. Nature Neuroscience

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“Research across species highlights the critical role of the amygdala in fear conditioning. however, fear conditioning, involving direct aversive experience, is only one means by which fears can be acquired. exploiting aversive experiences of other individuals through social fear learning is less risky. behavioral

research provides important insights into the workings of social fear learning, and the neural mechanisms are beginning to be understood. we review research suggesting that an amygdala-centered model of fear conditioning can help to explain social learning of fear through observation and instruction. we also describe how observational and instructed fear is distinguished by involvement of additional neural systems implicated in social-emotional behavior, language and explicit memory, and propose a modified conditioning model to account for social fear learning. a better understanding of social fear learning promotes integration of biological principles of learning with cultural evolution." Adolphs, R.. (1997). Fear and the human amygdala. Neurocase, 3(4), 267–274.

Plain numerical DOI: 10.1093/neucas/3.4.267
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"We have previously reported that bilateral amygdala damage in humans compromises the recognition of fear in facial expressions while leaving intact recognition of face identity (adolphs et al., 1994). the present study aims at examining questions motivated by this finding. we addressed the possibility that unilateral amygdala damage might be sufficient to impair recognition of emotional expressions. we also obtained further data on our subject with bilateral amygdala damage, in order to elucidate possible mechanisms that could account for the impaired recognition of expressions of fear. the results show that bilateral, but not unilateral, damage to the human amygdala impairs the processing of fearful facial expressions. this impairment appears to result from an insensitivity to the intensity of fear expressed by faces. we also confirmed a double dissociation between the recognition of facial expressions of fear, and the recognition of identity of a face: these two processes can be impaired independently, lending support to the idea that they are subserved in part by anatomically separate neural systems. based on our data, and on what is known about the amygdala's connectivity, we propose that the amygdala is required to link visual representations of facial expressions, on the one hand, with representations that constitute the concept of fear, on the other. preliminary data suggest the amygdala's role extends to both recognition and recall of fearful facial expressions."

Category

1. External Domains

Tags

1. anxiety
2. fear
3. neuroscience

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