

Social learning of fear

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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.

Learning about potentially harmful stimuli and events is critical in shaping adaptive behavior in a rapidly changing environment. It allows animals to establish and update associations between external events and motivational states such as fear. Fear can be expressed, transmitted and acquired in various ways. For example, you might fear a particular neighborhood because you were assaulted there, because you saw someone being assaulted there, or because someone told you an intimidating anecdote about a similar crime there. Thus, fears can be acquired through direct experiences or indirectly through social transmission (Fig. 1a–c). In all cases, your fear of the locality might express itself similarly, such as by avoidance of the locality and increased autonomic arousal when approaching it. These responses might serve you well. However, if the experienced assault was a one-time event, the observed event a scene in a movie, or the anecdote a distortion of reality, your responses might disrupt normal functioning, especially if the neighborhood was your home.

Whereas the neural circuitry of fear learning through classical conditioning is understood in considerable detail, researchers have just begun to study the neural mechanisms underlying social fear learning. Although similar neural processes may support direct and indirect fear learning, a distributed network of regions is involved in social perception and evaluation. Our aim is to survey cross-species work on social fear learning in light of our current understanding of the social brain and to outline a model describing how social interaction can guide affective processes underlying acquisition and expression of fear learning.

We begin by summarizing the neural substrates of direct fear learning through classical (pavlovian) fear conditioning with an emphasis on the role of the amygdala. Next, we selectively review behavioral findings of observational fear learning in nonhuman animals, followed by research on social fear learning in humans.

Taken together, this work implies that the basic associative learning processes that are responsible for acquisition and expression of learned fear are similar across species and across different learning procedures, such as social observation and verbal instruction. However, social, affective and cognitive processes are likely to contribute to fear learning in a social context. Based on this literature, we propose a neural model for how social-affective processes contribute to acquisition and expression of fears acquired through social means (Fig. 2a–c).

Fear conditioning

Most of our knowledge about basic neurobiological mechanisms of fear learning stems from classical conditioning. In a typical fear conditioning protocol, a neutral conditioned stimulus (CS) is paired with a naturally aversive stimulus (unconditioned stimulus, US), leading to a conditioned fear response to the CS. The extensive use of fear conditioning protocols since Pavlov¹ has established this procedure as a model of fear learning². Consistency in the physiological expression of conditioned fear elicited by the basic protocol indicates that mechanisms of emotional learning are analogous across species.

Research on the neurobiology of fear conditioning has focused on the amygdala in the medial temporal lobe, a key structure in the brain's fear circuitry (Fig. 2a). Although the amygdala processes a wide range of emotionally relevant information, much of its anatomical and functional role in fear conditioning is homologous and analogous across species. The amygdala is a conglomerate of subnuclei, some of which have specific roles in fear conditioning. In rodents, sensory information arrives in the lateral nucleus from the thalamus and sensory cortices^{3,4}. The lateral nucleus also receives nociceptive information and is where synaptic plasticity builds an association between representations of the CS and US^{5–7}. The lateral nucleus further projects to the central nucleus and basal nucleus, which mediates the output to other regions that regulate expression of fear and anxiety⁸. For example, projections to the hypothalamus⁹ are important for mediation of autonomic responses, which in humans can be indexed through the skin conductance response¹⁰. Other areas of projection, such as the ventral tegmental area¹¹ and the central gray¹² are important in regulation of behavioral expressions of fear.

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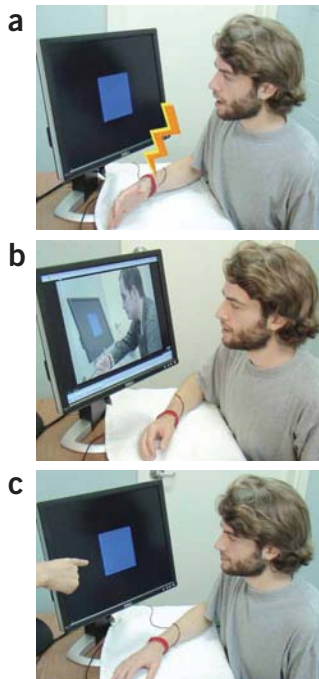


Figure 1 Nonsocial and social fear learning in humans. An individual learns to fear a CS through its pairing with (a) an electric shock to the wrist (fear conditioning), (b) a learning model's expression of distress (observational fear learning), and (c) verbal information about its aversive qualities (instructed fear).

Another behavioral output, avoidance behavior, is mediated by input to the basal ganglia from the basal nucleus¹³.

Under most circumstances, the role of the amygdala in fear conditioning is best understood together with other functional regions within a greater circuitry of fear learning. This circuitry involves sensory input and motor output systems, as well as regions that contribute to explicit and conscious aspects of learning and expression of fear. For example, the hippocampus, another medial temporal lobe structure adjacent to the amygdala, is critical for coding contextual information about the fear learning situation, such as relationships between different features and the timing of events. In other words, whereas the amygdala is responsible for forming associations between somatosensory states and representations of individual stimuli (cue learning), the hippocampus is important for encoding relations between the various cues that comprise the learning context (contextual learning). Patients with bilateral and unilateral amygdala lesions can verbally report the CS-US contingency, although they lack the normally associated autonomic response¹⁴, leading to the suggestion that the amygdala is critically involved only in implicit, nonverbal processes underlying acquisition and expression of conditioned fear. In contrast, the hippocampus is essential for consolidation and retention of explicit or declarative memory of the CS-US contingency¹⁵ and the environmental contexts that regulate conditioned fear responses¹⁶. In addition, across species, the prefrontal cortex (PFC) has a unique role in top-down regulation of affective responses through its regulation of activation in subcortical regions, such as the amygdala^{17,18}. More specifically, the ventral (infralimbic) region of the medial prefrontal cortex (MPFC) is critical to retention of extinction of conditioned fear responses in rats¹⁹, and the human homolog of this region is involved in extinction in humans²⁰.

The demonstration that the amygdala can operate independently from other neural systems critical to explicit expression of learned fear provides a possible explanation for the observation that a conditioned fear response can be elicited without explicit awareness of the CS^{21,22}. A subliminal presentation of CS results in activation of the right amygdala²³. Conditioned responses to subliminally presented CSs are only reported when the CSs are drawn from naturally fear-relevant stimulus categories, such as snakes, spiders and angry faces. Fear responses conditioned to fear-relevant stimuli are more resistant to modification by extinction and verbal instructions than are responses to fear-irrelevant natural categories, such as butterflies, happy faces or fear-irrelevant artifacts, such as broken electrical outlets and guns^{22,24}. These observations, combined with the superior fear conditioning observed in nonhuman animals to certain types of ecologically relevant stimuli, has led researchers to posit that these particular stimuli may be prepared by evolution to engage in aversive associations. Socially and culturally defined categories can also act as prepared stimuli in a fear conditioning protocol²⁵.

Just as the role of the amygdala in fear learning cannot be fully understood without recognizing the role of other regions in the same fear learning circuit, this kind of learning cannot be completely understood without considering the intricacy of the natural environment in which it occurs. For example, fear conditioning procedures have traditionally examined learning involving direct, individual experience of an aversive stimulus, the US. However, the natural milieu of many species offers both safer and more economical alternative means to attain corresponding information about potentially noxious stimuli. The social environment provides a suitable medium to transfer emotionally significant information between individuals. Verbally communicating with a fellow human or observing a conspecific's expressions of fear are two such means that can produce learning that shares both behavioral and neural qualities with fear acquired through fear conditioning (Figs. 3 and 4).

Observational fear learning across species

Social transmission and detection of fear signals is well documented in a range of species²⁶. The ability to detect and respond appropriately to signs of fear and pain in a conspecific probably has conferred a significant selective advantage during evolution. However, these signs not only alert the receiver about potential imminent danger, they also assign a threat value to the context or cue associated with the threat. For example, a conspecific's fear expression may serve as an US, eliciting an immediate aversive response in the observer that becomes associated with the paired stimuli. Observational learning may also be subserved by social inference, in which the conspecific's fear expression is a CS that was previously associated with a directly experienced aversive event (US) and may act as a secondary reinforcer in future learning.

The study of fear learning through social observation is informed by different lines of research, from emotional contagion and imitation to more complex operant tasks. Here we focus on social learning, as defined by processes contributing to formation of associations between different stimuli and expressed later in the absence of the conspecific serving as the learning model. We do not discuss simpler forms of socially facilitated and contagious fear responses, such as those seen in flocks behaving in unison, schools and herds of animals^{27,28} or imitation²⁹⁻³¹. To provide an appropriate parallel to existing research on social fear learning in humans, we focus our discussion of the animal literature on social learning in the visual domain. However, similar associative mechanisms are likely to be involved in social learning relying on other modalities, such as auditory and olfactory information³².

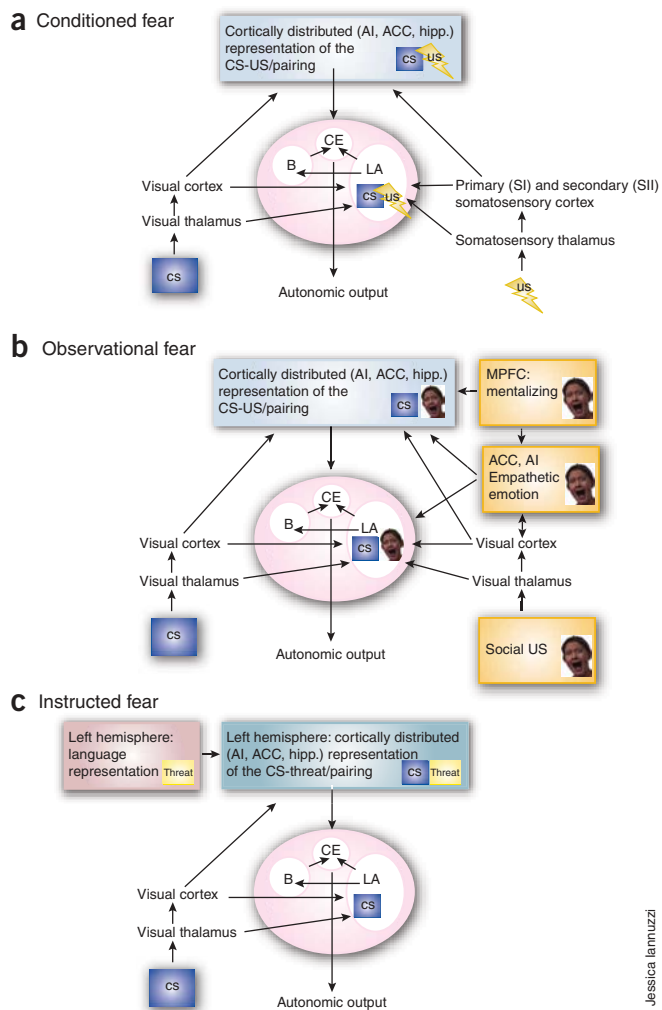


Figure 2 A neural model of nonsocial and social fear learning in humans. The arrows describe the flow of information between different functional brain regions. Although the arrows point only in one direction, the connectivity might be bidirectional. **(a)** Fear conditioning occurs by associating the visual representation of the CS with the somatosensory representation of the aversive US. The lateral nucleus (LA), in which sensory representations of the CS and US converge, is believed to be the site of learning. The amygdala also receives input from the hippocampal memory system (hipp.), anterior insula (AI) and anterior cingulate cortex (ACC) containing secondary representations of the CS and US, information about the learning context and the internal state of the organism. **(b)** In observational fear learning, the visual representation of the CS is modified by its association with a representation of the distressed other, serving as the US. As in fear conditioning, it is hypothesized that representations of the CS and the US converge in the LA. The strength of the US may be modified by MPFC input related to the interpretation of the other's mental state, as well as cortical representations of empathic pain through the ACC and AI. **(c)** Instructed fear learning occurs by modifying the processing of the visual representation of the CS through its association with an abstract representation of threat. Instead of being coded in the amygdala, the CS–'threat' US contingency is likely to be represented in a cortically distributed network, critically depending on the hippocampal memory system.

model's expressed distress, the observer's immediate response to the model's distress, and the resulting fear learning in the observer were comparable to the relationship reported between US, UR, and conditioned response in classical fear conditioning^{39,40}. This one-trial social encounter with a fearful model produces a robust fear response that lasts several months³⁹. Again, these findings strongly indicate that observational fear learning draws on the same processes as fear conditioning. Still, the neural processes remain to be explored in nonhuman animals.

The ultrasocial environment of humans provides ample opportunities to watch others' emotional responses to stimuli^{47,48}. Children with subclinical animal phobias or extreme fears toward certain situations, such as darkness, often report having observed parents fearful in the same or similar situations^{49,50}. Normal children can acquire a strong and persistent aversive response to a fear-relevant object (such as a toy snake) after seeing it paired with their mothers' fear expressions⁴⁶.

In adults, another person's arm movement in response to a shock can act as an US, but only when the observer believes that it was caused by a shock, not when the model's arm moves without a shock or when a shock is delivered without arm movements³⁷. These results support the conclusion that perceptual properties of the learning model interact with the observer's knowledge to instigate an unconditioned response. Similarly, information about another person's spider phobia can induce an aversive response to a spider that is presented to the allegedly phobic model, even without any physical cues of distress³⁸, and the affective response in an observer can be modified by context^{51,52}. In sum, research on observational fear learning consistently establishes that a facial expression can serve as an US, but, as discussed below, social variables can also modulate the response.

Observational fear learning may draw on the same processes as fear conditioning, with the expression of the conspecific learning model serving as the US. However, some studies on social learning in rats do not replicate core features of classical fear conditioning, such as blocking, overshadowing and latent inhibition⁵³, and at least one study did not find evidence for fears acquired through observation⁵⁴. In contrast, humans demonstrate classical conditioning characteristics for observational learning, including overshadowing and blocking⁵⁵, indicating that observational learning may show greater interspecies variability than does classical fear conditioning.

Given the adaptive function of the ability, it is not surprising that many animals, including birds³³, mice³⁴, cats³⁵, cows³⁶ and primates^{37–43}, can learn fears by observing a conspecific. In one ecologically valid study³⁴, model mice were attacked by biting flies while observer mice watched. When exposed 24 hours later to flies, whose biting parts had been removed, the model and observer mice expressed conditioned analgesia and avoidance responses to similar degrees, implying that individual and social fear learning were equally effective. The strength of the model's fear response during its individual learning was not correlated with expressed fear learning in the observer at a later test. In contrast, such a relationship was found during observational fear learning in primates³⁹, indicating that there may be a greater reliance on emotional expressions during the learning process. The rich and flexible musculature of the primate face allows it to produce a wide repertoire of emotional expressions, superior to that of many other species⁴⁴. The cortical areas dedicated to face processing are also relatively enlarged in primates⁴⁵, implying an enhanced ability to rely on facially transmitted emotional information.

In monkeys^{39,40} and humans^{41–43,46}, facial fear expression is a reliable US. Cage-reared monkeys were shown either live presentations or movies of model monkeys reacting fearfully to snakes (toy or real) or to non-fear-relevant objects³⁹. When fear-relevant objects were used, the relationships between the strength of a learning

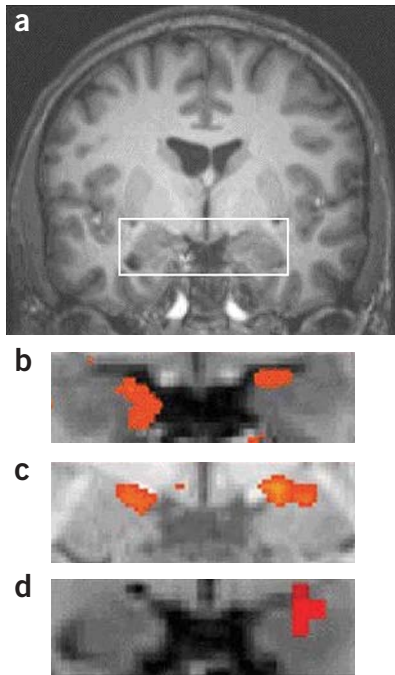


Figure 3 Fear learning in the human amygdala. (a) The outlined box contains the area of the medial temporal lobe that includes the bilateral amygdala. (b–d) Amygdala activation to the CS is seen bilaterally after fear conditioning (b) and observational fear learning (c), and unilaterally (d) in the left amygdala after instructed fear.

Neural systems of observational fear learning

Despite the extensive evidence for observational fear learning across species, there is surprisingly little research in nonhumans investigating the underlying neural mechanisms. Behavioral findings indicating that observational fear learning draws on the same processes as fear conditioning predict a role for the amygdala. Amygdala lesions in monkeys confirm that this region is critical in acquisition and appropriate display of fear in social and novel situations⁵⁶. The hippocampus is important in socially mediated formation of food preferences⁵⁷ and, under certain circumstances, social recognition memory in rodents⁵⁸. In addition, lesions in the MPFC in rodents alter social behavior⁵⁹. Still, the neural processes engaged in observational fear learning remain to be explored in nonhuman animals.

In humans, as in other animals, most of our knowledge about observational fear learning is drawn from behavioral experiments. Only recently have the neural mechanisms underlying this kind of learning been explored^{42,60}. In an imaging study⁴², subjects watched a movie of another person expressing distress when receiving electric shocks paired with a CS. Later, subjects expected to receive shocks along with the same stimulus as that in the movie they just watched. However, no shocks were administered during the test stage to ensure that their representation of the US-CS pairing was based solely on vicarious experiences. As in previous fear conditioning studies, the bilateral amygdala was involved during both learning (observation) and expression (test) of learned fear, strongly supporting the assumption that similar associative mechanisms and their underlying neural processes support both conditioned and observational fear learning (Fig. 3a–c).

In fear conditioning, the amygdala is believed to process and store representations of the CS-US contingency. Although the amygdala has an ancient evolutionary history, its interconnectedness to neocortex has increased substantially in primates. The basolateral complex in the

primate amygdala has strong reciprocal connections to visual cortex, in particular to the inferotemporal region that responds to face identity and to facial expression⁶¹. In addition, the basolateral complex is directly connected to the ventral part of the MPFC and indirectly with more dorsal regions of the MPFC^{62,63}. These observations are compatible with the suggestion that the primate amygdala may be particularly prone to form associations between more complex socio-emotional stimuli, especially when they are visually represented.

The evidence indicates that, at least in primates, representation of fear learning through observation and classical conditioning may be rather similar within the amygdala. However, in spite of the many features shared between conditioned and observational fear, nonsocial and social forms of learning must differ in several fundamental ways, implying involvement of partially dissociable neural networks outside the amygdala. For example, a conspecific's expression of distress may signal an imminent threat that serves as a US and elicits an immediate unconditioned response in the observer that is associated with a CS. However, this response is also mediated by the observer's perception of the model, which can be influenced by more elaborated processes, such as emotional perspective taking and mental attributions. These, in turn, may be dependent on social factors, such as familiarity, relatedness, social status and interpersonal learning history. Indeed, in mice, observing a familiar, but not an unfamiliar, mouse experiencing pain enhances sensitization to pain at a later test time⁶⁴. The intrinsic aversiveness of observing a conspecific in pain is evidenced by the willingness of monkeys to starve themselves if a shock is administered to a fellow monkey every time the observer attempts to eat⁶⁵, but again, this altruistic behavior is influenced by familiarity and past experience of the conspecific^{65,66}.

These studies hint at two interacting pathways mediating fear learning through observation. First, as suggested by work on observational fear learning in primates^{39,41,42}, a conspecific's expression of distress can be intrinsically aversive, indicating that somatosensory representations may be primed by mere observation of another individual's emotional display without necessarily being accompanied by higher order social cognition thought to be unique to humans. This point has been emphasized by proponents of mirror-neuron models of emotion perception and empathy. According to these accounts, shared neural representations of one's own experiences of an emotion and perception of the corresponding emotion in another individual are critical to emotional understanding and to empathizing with others^{67,68}. Second, in spite of a partial independence from higher cognitive functions, factors related to the social context can be involved in the regulation of basic emotional responding during observation and the resulting learning.

Studies in humans support these two interacting mechanisms²². First, stressing the independence of goals, expectations and social context, subliminally presented faces that signal threat, either by appearing angry or fearful⁶⁹ or through previous pairing with an aversive stimulus, can elicit amygdala-mediated fear response in an observer²³. On the other hand, affective responses to emotional faces and their recruitment of the amygdala depend on the context provided⁷⁰ and on cognitive appraisals by means of prefrontal brain systems¹⁷. Basic emotional responses to another's distress are affected by interpersonal learning history and the goals of the observer. For example, an observer's affective response to another's distress depends on whether the other person is expected to cooperate or compete in a future interactive game⁵¹. Imaging studies indicate that a neural network, including the anterior insula and anterior cingulate cortex (ACC), that encodes the affective (as opposed to sensory), motivational and autonomic aspects of pain^{10,71,72} is also involved when people

observe or imagine another individual's pain^{73–75}. This finding has led researchers to propose that these shared emotional representations are involved in empathy^{67,68,73–75}. These regions also track reported fairness of another individual in pain after a competitive game⁵² and empathic concern after receiving instructions to take the other's emotional perspective⁷⁶.

Socially mediated variability in affective response to another's distress is likely to influence ensuing learning. An fMRI (functional magnetic resonance imaging) study on observational fear learning⁴² found activation in the ACC and anterior insula both during observation of another person receiving shocks paired with a CS and in the later test stage when the person being imaged expected to receive shocks accompanying the same stimulus, indicating that regions linked to empathy may be involved in observational fear learning. This assumption was further supported by the finding that activation in both these regions during observation predicted learning as expressed in the subsequent test stage. In addition, another region of interest, the rostral MPFC, was only activated during the observation stage. Responses in this region marginally predicted the magnitude of subsequent learning. The MPFC is implicated in thinking about one's own and others' mental states^{77–79}, indicating that social cognition may be involved in observational learning of fear.

In accordance with research on nonhuman animals, the findings of observational fear learning in humans demonstrate, on the one hand, an independence of conscious awareness and strategic regulation of affective responses and, on the other, a dependence on social and contextual manipulations. However, it seems likely that the amygdala, supporting automatic affective responses, interacts with the orbito-frontal cortex, the temporal lobe and the MPFC that together mediate social and contextually regulated processes, to produce an adaptive affective response (Fig. 2b). It remains to be explored what social factors cause formation of a learned fear response through social observation and what neural systems support these social influences.

Based on this research and the brain's connectivity, it is possible that amygdala-centered observational fear learning in both rodents and primates is supported by automatically activated cortical mechanisms of shared affective representations in the anterior insula, as well as more explicit hippocampal representations about context and relevant social information about the learning model (such as social status and familiarity). Although in rodents the ventral MPFC has a role in some social behaviors⁵⁹, the primate MPFC is likely to be more important in social perception and learning, as shown by deficits in social behavior after prefrontal lesions in both monkeys⁸⁰ and humans⁸¹. However, the more anterior-rostral region of the MPFC is both quantitatively and qualitatively more developed in humans as compared with other primates⁸², implying a neural substrate for the support of more complex mental representations that might be involved in human observational learning. A meta-analysis of imaging studies reports that this region is especially sensitive to experiments involving both social and emotional tasks⁸³. In sum, regardless of the complexity of the underlying neural representations, the research discussed above shows that a conspecific's emotional display can serve as an US, stressing the similarity with conventional conditioning.

Instructed fear learning

Humans possess the unique ability to obtain emotional information through language. Whereas fear learning through observation involves visual representation of emotional properties of a stimulus, language is arbitrarily related to, and thus detached from, its referent in the world. Language forces the receiver to rely on similar past experiences and internally generated imagery to establish an emotional memory.

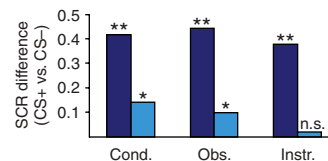


Figure 4 Mean expression of learned fear as assessed with difference in skin conductance response (SCR) in three groups of subjects after conditioned, observational and instructed fear learning. CS+, CS paired with US; CS–, CS unpaired with US. Asterisks, learned fear response significantly greater than zero ($*P < 0.05$; $**P < 0.01$; NS, not significant). Dark blue bars, responses to supraliminal (perceived) CSs; light blue bars, responses to subliminal (unperceived) CSs. For all three learning groups, unmasked as compared with masked CSs elicited a stronger learning response that was equivalent across groups. In the conditioned and observational, but not in the instructed, groups, a fear response was also elicited to unseen (subliminal) CSs.

Imagery and self-projection into the future are thought to rely on neural systems similar to those involved in perception⁸⁴ and episodic memory formation⁸⁵. Like recollection of the past, projection into the future is impaired after hippocampal lesions⁸⁶. In addition, regions of MPFC implicated in simulation of future events^{87,88} overlap with those involved in thinking about others' minds.

Both clinical accounts that retrospectively target the etiology of phobic fears⁸⁹ and experimental studies on children involving fear provoked through storytelling⁹⁰ reveal that verbal instructions can be a strong stimulus for fear learning. Along the same lines, adults instructed to expect a shock paired with a specific CS and later exposed to the same CS show learned responses similar to those seen after classical fear conditioning^{41,91–93}.

To directly compare fears acquired through conditioning, observation and verbal instruction, we⁴¹ manipulated the learning procedure, keeping other factors constant. Conditioned stimuli acquired their threat value through being paired with a shock, with observed fear expression in another person or with the experimenter's verbal instructions (Fig. 1a–c). Fear responses to the CS were of comparable magnitude after the three kinds of learning (Fig. 4). In addition, replicating previous findings²¹, a subliminally presented (unperceived) CS triggered a response in the fear conditioning group. The observational, but not the instructed, group also showed a learning response to subliminal presentations of the CS, indicating that common learning mechanisms may underlie fear learning through conditioning and observation, but a different mechanism may support learning through language. These results support the notion that there are partially dissociable systems involved in different modes of social, emotional learning. Classical conditioning and observational learning, which humans share with many other species, might be supported by an evolutionarily old system that predates the emergence of language. In contrast, learning based on language is unique to humans and is likely to be, at least initially, dependent on representations in higher cortical areas that also support conscious processes. Indeed, these findings indicate that such cortically represented fear associations might depend on conscious awareness, in accordance with the observation that conscious awareness can be used to distinguish subdivisions of conditioning (such as context versus cue or trace versus delayed).

To examine the mechanisms underlying expression of fears acquired through verbal instruction, Phelps and colleagues⁹³ told subjects they might receive a shock when shown a square of a particular color ('threat' stimulus), but not another color ('safe' stimulus). Supporting

extension of the fear conditioning model to instructed fear, there was robust activation of the left amygdala, which correlated with the physiological expression of fear learning (Fig. 3d). Activation of the left insular cortex also correlated with expression of learning. The insular cortex is a critical component for conveying a cortical representation of pain to the amygdala⁹⁴ and for subjective awareness of physiological states⁷². The verbally mediated learning is likely to have resulted in an abstract cortical representation of the potentially painful shock, which may have been communicated to the amygdala through projections from the insular cortex (Fig. 2c). The left lateralization of the activation is consistent with the common view that the left hemisphere is more involved in language processing⁹⁵. However, brain imaging results cannot rule out involvement of the right amygdala, or indicate a critical role for the left amygdala in expression of fears learned through verbal instruction. Further support that the left amygdala mediates physiological expression of instructed fear learning was demonstrated in subjects with unilateral amygdala damage after a similar learning protocol. Those with damage to the left, but not right, amygdala showed an impaired expression of instructed fear. Instructed fear is dependent on awareness⁴¹, further indicating that learning based on abstract representations of contingencies may involve neural networks partially different from those involved in fears acquired through classical conditioning and observation.

A model of social fear learning

Social fear learning offers the opportunity to study transmission of biologically relevant information between individuals. Indeed, social learning at large may lie at the core of the forces that create and maintain culture^{31,96}, which might then affect biological evolution^{96,97}. Fear learning also provides insights into neurobiological mechanisms of social learning and thus may serve as a model for the intricate links between biological principles of learning and cultural evolution. Here we provide a framework for the relationship between neural mechanisms underlying fear conditioning and two forms of social learning: observational and instructed fear. The model is centered on the amygdala, which is critical to physiological expression of learned fear, regardless of how learning is acquired.

As outlined earlier, in classical fear conditioning (Fig. 2a), information about the CS is communicated to the lateral nucleus of the amygdala by way of the sensory cortices and thalamus; this information converges with US input from the somatosensory cortex and thalamus. Through synaptic plasticity in the lateral nucleus, the CS-US association is formed. An additional, distributed cortical representation of the CS-US contingency is also acquired through the hippocampal memory system and may be expressed in regions associated with pain processing, such as the ACC and insular cortex. In the presence of the CS, learned fear is expressed through projections from the lateral nucleus to the central nucleus, which in turn mediates autonomic expression. (Other means of expression may depend on other pathways⁸.) In addition, projections from the cortical representation of the CS-US contingency to the amygdala may contribute to autonomic expression of fear learning when there is subjective awareness of the CS-US contingency.

We propose that the mechanisms underlying learning through social observation (Fig. 2b) may be similar, with a few exceptions. First, the US in observational fear learning is the perceived fear expression of a conspecific and, as such, is conveyed to the lateral nucleus through the sensory cortices and perhaps the sensory thalamus. The representation of the strength of the US in the lateral nucleus may be modified by MPFC input related to perception and interpretation of the learning model's mental state during the observed painful experience, as well as

a cortical representation of empathic pain through input from the ACC and insular cortex. We propose that, as in classical fear conditioning, the lateral nucleus is a site of plasticity underlying memory for the CS-US association, in addition to a distributed cortical representation of the CS-US association acquired through the hippocampal memory system. The output mechanism for observational fear learning does not differ from that for fear conditioning.

Fears that are acquired through verbal communication (Fig. 2c), we suggest, rely on a slightly different representation, given the symbolic nature of the learning. It is unlikely that abstract representations of verbal threat are represented in subcortical structures, such as the amygdala. Although sensory information about the CS is conveyed to the lateral nucleus, we hypothesize that the association between the CS-US is only represented in a distributed cortical network. Furthermore, this cortical representation is left-lateralized, reflecting the verbal nature of the US. We propose that memory for this cortical association depends on the hippocampal complex for acquisition, and that plasticity in the amygdala is not necessary. Nevertheless, autonomic expressions of instructed fears occur through communication of the cortical representation of the CS-US association and the potential for pain to the amygdala, perhaps by way of the insular cortex. As with other means of fear learning, we propose that the central nucleus mediates autonomic expression of instructed fear.

This proposed framework is simply our best guess of the processes underlying social learning of fear based on a limited literature, so a few caveats are appropriate. First, another brain region that may be involved is the striatum. Human brain imaging studies on fear learning, including those examining social fear learning^{42,93}, report activation of the striatum^{98,99}. Animal models of fear conditioning have not emphasized the striatum beyond its role in avoidance learning and active coping⁸, but this region, which is important in reinforcement learning¹⁰⁰, may represent the CS-US association. Second, we have emphasized unidirectional projections in our model, but most of the regions we discuss have bidirectional connections with the amygdala. Third, this framework outlines how fear learning is first expressed after social and nonsocial means of acquisition. Once a CS is experienced and a fear reaction occurs, further learning may result, which could change the nature of the representation further. For instance, in instructed fear, co-occurrence of the CS and autonomic arousal may cause the CS to act as a secondary reinforcer, which projects its emotional salience to the lateral nucleus to facilitate an amygdala-dependent representation of the CS-threat association that was not present after initial verbal instruction. In this way, representation of verbally communicated fears may change over time and be experienced to be more similar to conditioned fears.

In spite of these caveats, the proposed framework represents a neural model that can begin to help us understand the complexity and subtlety of human fear learning in a social and cultural environment. This understanding may provide important knowledge about the underlying socio-emotional impairments that are hallmarks of many psychological disorders, such as phobias and anxiety disorders, which are characterized by dysfunctional assignment of emotional value to certain stimuli and situations. Finally, a better understanding of the neural mechanisms supporting socially transmitted fears is essential to integrate our knowledge about the biological foundations of learning and cultural change to evolution at large.

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COMPETING INTERESTS STATEMENT

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