The neural and computational systems of social learning

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Abstract | Learning the value of stimuli and actions from others — social learning — adaptively contributes to individual survival and plays a key role in cultural evolution. We review research across species targeting the neural and computational systems of social learning in both the aversive and appetitive domains. Social learning generally follows the same principles as self-experienced value-based learning, including computations of prediction errors and is implemented in brain circuits activated across task domains together with regions processing social information. We integrate neural and computational perspectives of social learning with an understanding of behaviour of varying complexity, from basic threat avoidance to complex social learning strategies and cultural phenomena.

Social learning
Learning from others, for example through observation and instruction, which may or may not involve directly experienced or vicarious reinforcement.

Value-based learning
Learning about rewards and punishers, which promote the organization of behaviour for maximizing rewards and minimizing punishments

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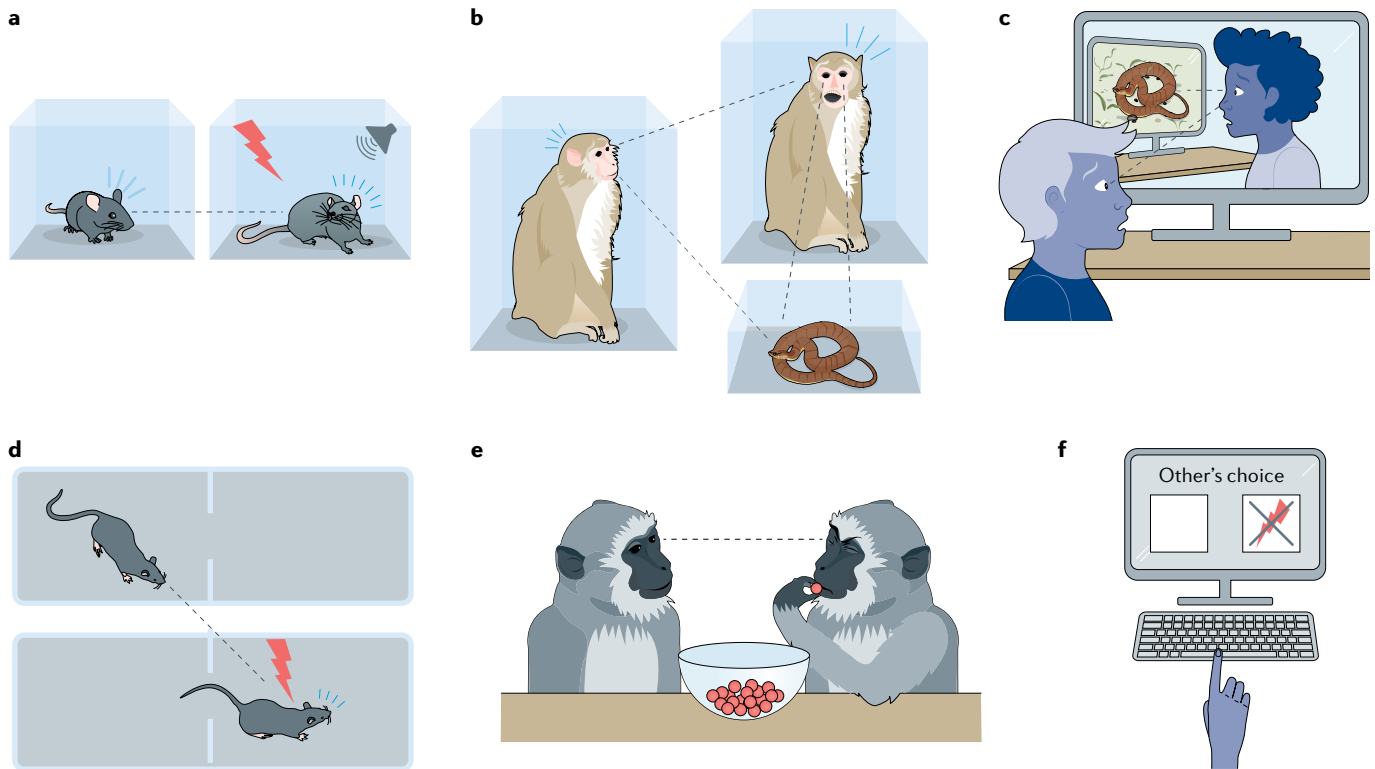
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https://doi.org/10.1038/s41583-020-0276-4

To survive, an animal must continuously learn to predict and respond to challenges and opportunities in its environment. In many species, the social environment contains both the greatest challenges and opportunities to the individual. Conspecifics pose a deadly threat, but are at the same time key to survival, affording protection, nurturance and reproductive opportunities. Importantly, others also serve as vehicles for transmitting value information, helping the individual to avoid harm and maximize rewards. Indeed, from early on in development, social animals learn the value of stimuli and of their own and others' behaviours from each other[1]. The spread of such value information among peers and across generations gives social learning a unique role in the evolution of culture[2]. Here, we review cross-species research on the neural and computational nature of social, value-based learning. Related processes, such as motor-based imitation and social learning of skill and symbolic meaning, which are not directly linked to value information, are beyond the scope of the current review.

Most research on learning has occurred in a social vacuum[3], and little is known about the neural and computational mechanisms supporting the social transmission of value information. The chief reason for this lack of progress is the immense complexity added when several individuals are dynamically interconnected[4]. Addressing this composite issue requires systematic integration across fields, including learning science, computational science, social and affective neuroscience, and behavioural ecology. Research related to social learning within these fields has largely progressed in parallel with little collaboration between them[5], with some notable exceptions[6–8].

In light of this, the current review draws on new knowledge from these diverse fields to provide a synthesized view of social learning. We begin by surveying recent research on domain-general principles of learning, highlighting core computational and neural mechanisms of Pavlovian and instrumental learning that apply across task domains. Next, we survey a selection of findings in the neuroscience of social cognition, which is the study of how individuals connect with the minds of others. This leads us into the central discussion of how domain-general principles of learning and social cognition jointly contribute to social learning. We highlight recent research on social learning in both the aversive and appetitive domains: experimental work in human and non-human animals, describing the mechanisms underlying learning about the value of stimuli (FIG. 1a–c) and actions (FIG. 1d,e), and theoretical models from learning theory and behavioural ecology, outlining the functions of learning in terms of its underlying computations and social learning strategies (SLS), respectively. Throughout, we discuss how social learning provides the basis for a range of more complex social phenomena, such as conformity and cultural traditions. We conclude that research on social learning provides an exceptionally well-suited paradigm to bridge the study of neural mechanisms with behavioural functions and social phenomena on a larger scale.

Computational basis of value learning
Over the past decades, ideas from learning theory, cognitive science and artificial intelligence have converged on a unified framework — reinforcement learning — to describe how agents learn to maximize rewards and minimize punishments[9,10]. A core idea of reinforcement learning is that agents form expectations about the value of possible actions and environmental states (for example, stimuli) through error-minimizing

Fig. 1 | **Schematic illustration of experimental procedures examining social threat and avoidance learning in rodents, monkeys and humans. a–c** | Pavlovian social threat learning through presentation of a conditioned stimulus (CS) paired with defensive responses of a conspecific, the 'demonstrator'. **a** | A naïve rodent, the 'observer', perceives a demonstrator displaying defensive behaviours when it receives a shock combined with a tone (CS) and, subsequently, the observer expresses learned threat responses when the tone is sounded, despite not having experienced a shock[118–121]. **b** | A monkey observer watches a demonstrator's defensive behaviour towards a snake, which serves as the CS, and, subsequently, the observer expresses learned threat responses to the CS[232]. **c** | A human observer watches a demonstrator displaying behavioural signs of discomfort and pain when receiving a shock paired with a picture of a snake or a neutral stimulus, which serves as the CS, and, subsequently, the observer expresses learned threat responses and fear when presented with the CS[28,126,127,133,137]. **d–f** | Instrumental learning of avoidance behaviour occurring through the presentation of a demonstrator's defensive behaviour and avoidance. **d** | A rodent learns to avoid a specific compartment of the cage in a shuttle box after perceiving the demonstrator's defensive responses when receiving a shock in that compartment[172,173]. **e** | A monkey learns to avoid eating certain kinds of food after observing the aversive consequences through a demonstrator's behaviour[213]. **f** | A human learns to avoid selecting cues in a two-alternative forced choice task after watching a co-participant's choice and aversive choice outcome in the same task[175,202].

mechanisms. The most basic of such mechanisms, the Rescorla–Wagner learning rule, specifies that the mismatch — the prediction error — between the experienced value of an unconditioned stimulus (US, such as an electric shock) and the expected value of a discriminative cue (conditioned stimulus (CS), such as a tone) is used to update the value of the CS. Experience with the world hence leads to progressively smaller prediction errors, and stronger association between the CS and the US, during the course of learning[11]. Similar learning rules, modified to allow sequences of actions (for example, in mazes), are also used in more complex action learning algorithms (for example, Q-learning[12]).

Seminal work has established that learning — and, in its extension, the decision-making underlying behaviour — involves multiple neural and computational valuation systems[13,14]. A basic division is drawn between the Pavlovian and instrumental systems[15,16], mirroring the distinction between stimulus and action learning in classical animal learning theory. Both systems share

the common principle of prediction and error correction, which permits modelling both types of learning within a unified formal framework, but differ in how learned predictions relate to behaviour. The Pavlovian system elicits a varied set of reflexes and fixed behaviours, such as freezing, in response to cues with intrinsic or learned (through Pavlovian conditioning) aversive or appetitive value, without evaluating the consequences of these actions. From a computational perspective, such fixed responses can be viewed as evolutionarily preprogrammed, facilitating avoidance of fitness-harming cues and approach towards fitness-promoting cues[15,17].

In contrast to the inflexibility of the Pavlovian system, the instrumental system assigns value to actions based on their reinforcement history, which guides adaptive action selection. Accordingly, animals can learn arbitrary actions to acquire rewards and to avoid punishments. A common view is that there are two parallel instrumental systems, one goal-directed (model-based) and one habitual (model-free)[13]. The goal-directed system

constructs a 'model' of the environment (for example, the probability that action X will lead to specific outcome Y), which can be used to incorporate additional information (for example, changes in motivational state or instructions about changes in the environment) on the fly. In contrast, it is generally thought that the habitual system is model-free and elicits actions in response to the previously learned expected value of cues (see REF.[18] for a recent challenge to this account, arguing that habits can be understood as triggered directly by a stimulus, in a value-free manner). Owing to the lack of an internal model, model-free control is inflexible, as it cannot incorporate new information (for example, hunger) without additional learning (for example, in the hungry state). Importantly, Pavlovian approach and avoidance response tendencies can facilitate, or impede, instrumental actions depending on the alignment between the values of the different systems[16,19–21].

The distinction between Pavlovian and instrumental learning mechanisms discussed above has a direct analogue in Pavlovian and instrumental experimental paradigms, which have been used for a century to examine non-social learning, and, more recently, learning through social means. The use of such behavioural paradigms (FIG. 1) has enabled the study of how the brain implements Pavlovian and instrumental computations, linking behaviour to its neural underpinnings through their common computational basis. Next, we discuss domain-general neural and computational mechanisms underlying such learning.

Learning through direct experiences

Direct Pavlovian threat learning. Until recently, the link between computational mechanisms and their neural implementations has seldom been explicitly addressed in Pavlovian threat or fear-conditioning research, which has been the most common paradigm to examine Pavlovian learning. Research on animals often uses a sound as the CS, a foot shock as the US and freezing as the conditioned response. Decades of research using this simple experimental model in rodents has shown that sensory information from the midbrain, thalamus and cortex converges in the lateral amygdala, where CS–US associations are formed during learning[22] (FIG. 2a). The lateral amygdala and the basal amygdala also receive inputs from other regions, such as the prefrontal cortex (PFC) and the hippocampus. These inputs provide integrated information about the emotional history of a stimulus, the internal state of an organism, context and time[23,24]. The central amygdala, in turn, projects to midbrain regions, such as the periaqueductal gray (PAG), and the brainstem, mediating defensive responses[25]. Interestingly, the PAG has also been linked to the prediction error and modulations of expectations as described by computational learning models[26–28]. Recent studies using optogenetics have added more complexity to this picture and shifted attention from structures to neural circuits within structures. This development has shown that, within a single region, different neuronal populations can have different or even opposite functions. For instance, the basolateral amygdala[29] has been found to contain two separate neuronal populations controlling

responses to negative stimuli and to positive stimuli, respectively. As the same spatial resolution is not possible in research on humans, thus cross-species comparisons are not possible, the current review only discusses research based on optogenetics to a limited degree.

Human studies, using functional MRI, have demonstrated the involvement of a set of regions analogous with those described in rodents, supporting the idea that the basic neural and computational mechanisms for acquiring and expressing threat responses are conserved across species[30,31] (FIG. 2b). The human amygdala is massively interconnected with cortical regions, including the hippocampus and the ventromedial PFC (vmPFC), which are critical for encoding and retrieving contextual information and regulating conditioned threat responses, respectively. A major function of the hippocampal–PFC circuitry is to disambiguate cues with varying meanings in different contexts[23], be they social or non-social in nature. The amygdala has reciprocal connections with the anterior insular cortex and the anterior cingulate cortex (ACC), which have both been implicated in the formation of threat memory across species[32,33]. In humans, these regions are involved in the aversive experiences of receiving, anticipating and controlling painful and otherwise unpleasant stimulation[34–37], as well as learning and regulating defensive responses[38].
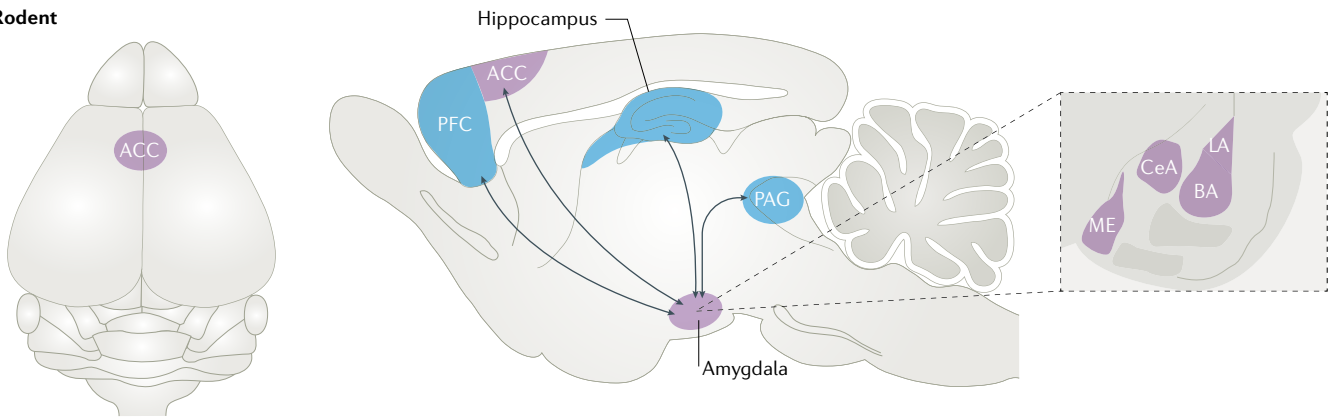
Learning that previously threatening stimuli are safe through repeated exposure without any aversive consequences — extinction learning — draws on the vmPFC and the amygdala[39–41], although the involvement of the vmPFC in humans seems to be specific to certain experimental design features according to a recent meta-analysis[42]. It is currently unclear to what extent the vmPFC contributes to the formation of new memory traces during extinction that inhibit the original CS–US associations and/or permanently alters the original memories, and how other factors, such as approach and avoidance behaviour[39,43], attention[44] and social stimuli[45], contribute to these processes. As discussed below, research has now begun to unveil how the brain regions involved in self-experienced learning take on both similar and distinct roles during social learning about threat and its update to safety, as well as rewards.

Direct Pavlovian reward learning. In both primates and rodents, the processing of reward information and learning of approach behaviours draw on brain circuits involving dopaminergic projections, such as the nucleus accumbens (NAcc) — a forebrain region located in the ventral striatum — receiving dopaminergic input from the ventral tegmental area (VTA) and substantia nigra pars compacta[46–49] (FIG. 3a,b). Recently, optogenetic studies in rodents, allowing for fine-grained parsing of functional units brain regions, have nuanced our understanding of these processes. For example, it has been shown that dopamine neurons in the VTA are phasically excited by both reward and reward-predicting cues, whereas GABAergic neurons signal expected reward[50]. The VTA does not, however, only contain reward-related circuits but also anatomically and functionally heterogeneous neuronal subpopulations with different axonal projections, which separately mediate reward and aversion[50,51].

Optogenetics
The use of genetically encoded, light-activated proteins to modulate activity of specific neural circuits. Optogenetics allows for targeting specific cell types or projections to learn the causal relationship between their activity and behaviour.

a Rodent

b Human

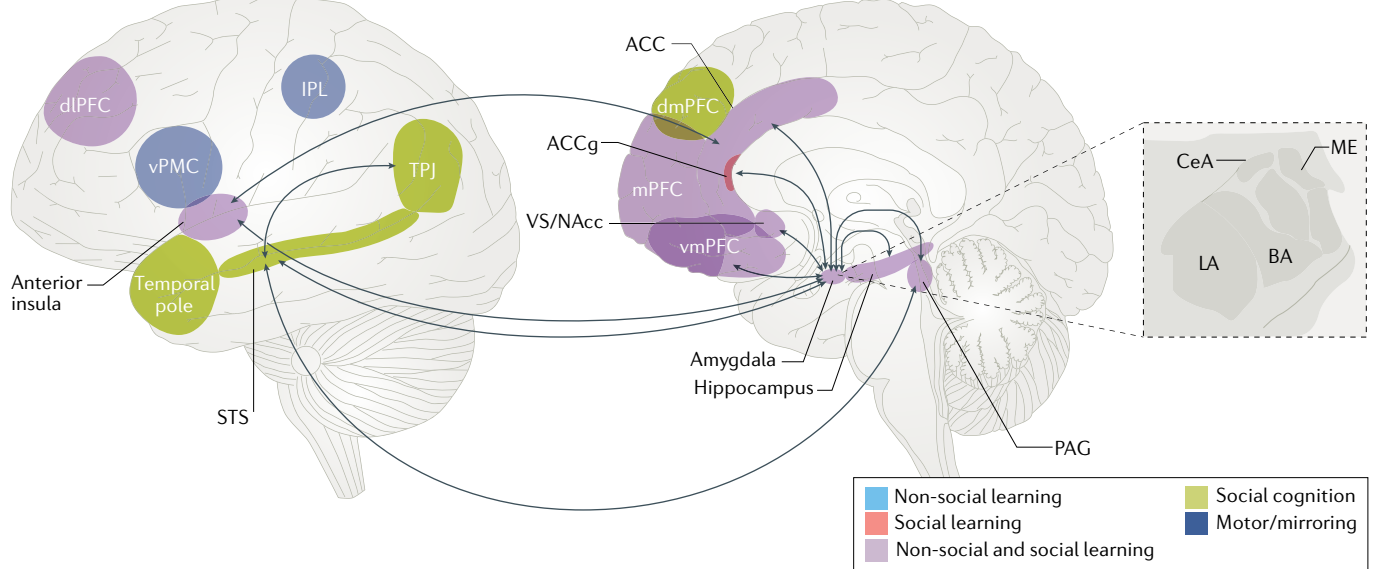| Non-social learning | Social cognition |
| Social learning | Motor/mirroring |
| Non-social and social learning | |

Fig. 2 | **Social threat learning partially shares neural mechanisms with self-experienced learning in both rodents and humans. a–b** | Light blue-shaded areas refer to regions involved in non-social threat learning and red-shaded areas refer to regions involved in social threat learning; regions involved in both social and non-social threat learning are marked by purple shading. Green-shaded areas refer to regions implicated in processing of social information (social cognition), such as the attribution of mental states. The bidirectional arrows illustrate the flow of information between brain regions. Studies on non-social threat learning in rodents reveal a neural circuit centred on the amygdala, in particular its lateral, basal and central nuclei, prefrontal cortex (PFC), hippocampus and periaqueductal gray (PAG), as critical for the acquisition and expression of conditioned threat responses. Brain imaging studies in humans have identified a similar network of structures involved in non-social threat learning, suggesting that the basic mechanisms are conserved across species. Moreover, the anterior cingulate cortex (ACC) and anterior insula, both reciprocally connected to the amygdala, have been implicated in the formation of threat memory across species. Studies of social threat learning in rodents and humans converge on the conclusion that, similarly to direct threat learning, the amygdala, anterior insula and ACC are involved in social learning. Although several neural mechanisms for social and non-social learning are similar, social learning is also distinguished from learning based on self-experience in several ways. For example, the use of optogenetic techniques in rodents has isolated different ACC projections involved in vicarious learning (to the basal amygdala (BA)) and direct learning (to the hippocampus), illustrating the presence of functional heterogeneity within this region. Similar heterogeneity is likely to exist in many brain regions. In humans, learning by watching a demonstrator's reactions to a shock, relative to directly experienced shocks, provokes greater functional connectivity between the threat learning circuit and the mentalizing network (marked here in green, part **b**). ACCg, gyrus of the ACC; CeA, central amygdala; dlPFC, dorsolateral PFC; dmPFC, dorsomedial PFC; IPL, inferior parietal lobule; LA, lateral amygdala; ME, medial nucleus of the amygdala; mPFC, medial PFC; NAcc, nucleus accumbens; STS, superior temporal sulcus; TPJ, temporoparietal junction; vPMC, ventral premotor cortex; vmPFC, ventromedial PFC; VS, ventral striatum.

The NAcc receives projections from the amygdala, hippocampus and prefrontal cortex, which are involved in reward processing[52–55]. Similarly to the VTA, functionally distinct valence-selective neuronal circuits have been identified in the amygdala and tied to behavioural functions[56]. Importantly, the flexible regulation and coordination of behavioural appetitive responses depends on the orbitofrontal cortex[57,58] and other regions of the PFC[59,60]. This network is highly conserved across species[61] and plays an analogous role in human reward learning[62].

Direct instrumental learning. The ability to learn new behaviours to avoid threats and approach rewards in the environment is a key survival function across the animal

kingdom. The acquisition of such adaptive behaviours is typically studied with instrumental learning paradigms, in which the individual learns to avoid shocks or obtain food and safety through first-hand, direct experiences of the action–outcome relationship. Research in rodents shows that the instrumental control of avoidance

behaviour includes the lateral amygdala and the basal amygdala (together forming the basolateral nucleus of the amygdala)[63,64], the hippocampus[65] and PFC–striatal circuits[66], whereas learning a relationship between an action and a reward relies on the striatum[67] and the PFC[68] (FIG. 3a). Encoding the value of a reward has been shown
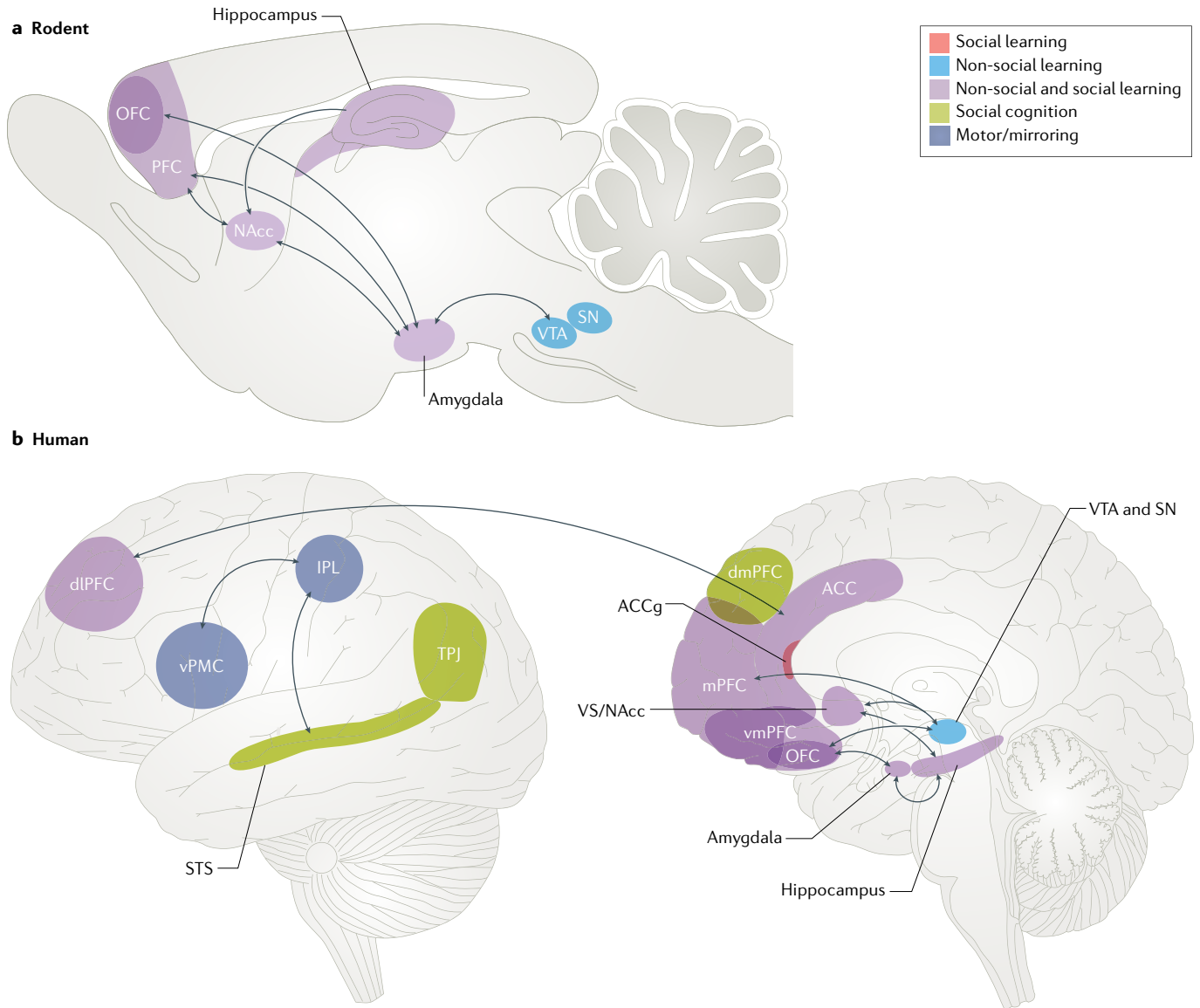

Fig. 3 | **Social reward learning partially shares neural mechanisms with self-experienced learning in both rodents and humans. a–b** | Light blue-shaded areas refer to regions involved in non-social reward learning and red-shaded areas refer to regions involved in vicarious reward learning; regions involved in both social and non-social reward learning are marked by purple shading. Green-shaded areas refer to regions implicated in processing of social information, such as the understanding and attribution of mental states, and dark blue-shaded areas refer to core regions of the action/mirror network. Animal studies on non-social reward learning suggest a network of structures mediating stimulus-reward associations. This network includes dopaminergic neurotransmission from the ventral tegmental area (VTA) and substantia nigra pars compacta (SN) to the nucleus accumbens (NAcc) and the prefrontal cortex (PFC). Importantly, this network also includes the amygdala, hippocampus and prefrontal and orbitofrontal cortices. The network involved in non-social reward learning network is conserved across species and partially overlaps with that

enabling social reward learning. Research across primates highlights the importance of the gyrus of the anterior cingulate cortex (ACCg) for various aspects of vicarious reinforcement learning. In humans, brain imaging research shows that, similarly to non-social reward learning, vicarious reward learning engages the ventral striatum. Imaging studies show that whereas both vicarious and direct rewards activate the ventromedial PFC (vmPFC), the NAcc is preferentially engaged in personal reward, highlighting both common and unique components of vicarious reward processing. Similarly to social learning in the aversive domain, social reward learning engages brain regions supporting the processing of social information, including the mentalizing (marked in green) and the action/mirror (marked in dark blue) networks (part **b**). ACC, anterior cingulate cortex; dlPFC, dorsolateral PFC; dmPFC, dorsomedial PFC; IPL, inferior parietal lobule; mPFC, medial PFC; OFC, orbitofrontal cortex; STS, superior temporal sulcus; TPJ, temporoparietal junction; vPMC, ventral premotor cortex; VS, ventral striatum.

to involve the dorsomedial striatum[69] and the basolateral amygdala[70]. Whereas the dorsomedial striatum and the prelimbic part of the PFC (the rodent homologue of the human dorsal ACC) are implicated in goal-directed, or model-based, actions, the dorsolateral striatum and infralimbic part of the PFC (the rodent homologue of human vmPFC) are involved in the control of habitual, or model-free, actions[71,72], suggesting different neural substrates of model-based and model-free learning.

In humans, the vmPFC, highlighted above for its role in the regulation of threat responses during extinction, has also been implicated in the subjective valuation of chosen actions as formalized in computational models[73]. Interestingly, recent findings in rodents suggest, contrary to mainstream theories claiming that the expected value represented in the PFC drives choices, that the expected value might instead primarily drive learning processes that, in turn, update choice mechanisms in other regions of the brain[74]. Adding further to the interest of the vmPFC in social learning is the role of this region in distinctly social functions, such as perspective taking[75], which is discussed below. Taken together, the implication of the vmPFC in regulation, valuation and social cognition has provoked a discussion of to what extent these functions are separable within this brain region or express common mechanisms[76]. As in rodents, the dopaminergic cortico-striatal circuits are critical to reward learning in humans (FIG. 3b). In particular, the dorsal striatum has been linked to prediction-error signals consistent with formal models of reinforcement learning[77,78].

In sum, learning about instrumental action–outcome contingencies has been linked to cortico-striatal circuits and dopaminergic prediction-error signals across species. Next, we discuss how social learning draws on the domain-general Pavlovian and instrumental learning mechanisms so far described, in conjunction with brain systems responsible for social cognition.

Learning in a social world

Social learning can be broadly defined as learning from, or in interaction with, other individuals. This form of learning is often adaptive, because it allows learning about the world while minimizing exposure to predation and other threats and offers access to others' innovations[2,79]. For instance, a mouse pup might acquire avoidance behaviours through the pairing between arbitrary odour cues and the smell of its frightened mother acting as a 'demonstrator'[80], and a social media user might learn to avoid certain locations through posted warnings during a terror strike or learn about a hidden place to forage wild strawberries. Social learning can, however, also be maladaptive, and clinical research demonstrates that irrational fears and anxieties are commonly transmitted between caretakers and offsprings[81]. In today's social media landscape, threat information quickly spreads globally with large potential impact. For example, a recent meta-analysis demonstrates an increased prevalence of psychological disorders following exposure to media coverage of disasters and large-scale violence in other parts of the world[82].

Formal models in theoretical biology also predict that social learning can sometimes be maladaptive, as information gleaned from demonstrators risks being outdated or error prone. The theoretical solution to this apparent paradox is to only learn from, or copy, the right demonstrators, at the right time[83,84]. SLS, which specify from whom and when to learn to optimize behaviour, offer such solutions. SLS that are determined by the context of learning come in three basic types: 'when', or state-based, SLS (for example, 'copy when non-social learning is costly', 'copy when uncertain'); 'who', or demonstrator-based, SLS (for example, 'copy based on demonstrator's knowledge', 'copy based on familiarity'); and frequency-dependent or conformity-biased SLS ('copy the majority')[79]. It remains a contentious issue whether such SLS actually are distinctly social, or rather emerge from basic, non-social learning mechanisms (BOX 1).

In parallel to research on SLS, a growing number of studies have investigated the neural and computational mechanisms of social learning across species[3,85]. Much of this research has directly or indirectly examined key computations implicated in SLS, providing evidence for the involvement of both domain-general associative/reinforcement learning mechanisms and uniquely social neural mechanisms. Below, we review research on social cognitive processes and how these are related to the computational and neural mechanisms underlying different types of social learning, and, where possible, highlight how these can be linked to different classes of SLS.

Neuro-computations of social cognition. In contrast to non-social learning, which is reinforced by direct aversive or rewarding experiences, social learning relies on the processing of social information. Such processes can be described at different levels of complexity, from the

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Box 1 | What is social about social learning?

Social learning strategies (SLS) — the set of strategies proposed by behavioural ecologists for when to learn socially — are by definition agnostic about the mechanisms that underlie social learning[5]. However, learning theorists have recently proposed that the very notion of SLS as distinctly social, and subject to natural selection, is misleading because many purported experimental demonstrations of SLS can be explained by the same domain-general learning mechanisms as non-social learning[221–223]. According to this account, there are no mechanisms uniquely dedicated to, or adapted for, social learning. Differences between species in social learning abilities depend on 'input mechanisms', such as variability in attention to social stimuli, rather than on different learning mechanisms. Through associative learning and acculturation, social learning might falsely come to appear as uniquely social. Critics of this view argue that the large variability between species in social learning capabilities (for example, humans vs chimpanzees) suggests selection for social learning capacitates[5]. Recently, a computational theory was described that shows that a large part of social learning phenomena among non-human animals can be accounted for by associative, model-free reinforcement learning mechanisms[224]. In contrast, adaptive social learning among humans might require explicit metacognitive knowledge about whom to learn from[206]. As described throughout the main text, the neuroscientific study of social learning has revealed strong similarities in the neural mechanisms that underlie social and non-social forms of learning. However, it is also clear that some forms of social learning recruit a distinct set of neural regions, such as those involved in representing others' motivational states (for example, the temporoparietal junction in humans[114], and the medial prefrontal cortex, superior temporal sulcus and gyrus of the anterior cingulate cortex across primates[3,6,85,111]). Future research across species, combining computational approaches with neural exploration[8], will be required to fully understand what is 'social' about social learning.

Box 2 | The origin of vicarious reinforcement

Why would others' experiences of reward or discomfort motivate behaviour? One hypothesis is that such vicarious reinforcements are in fact learned by past, direct experiences. Accordingly, if rewards and punishments received by others often co-occur with self-experienced outcomes, this would support second-order learning, a process which imbues cues with reinforcement value of their own[224]. For example, if one's siblings often are rewarded at the same time as oneself, this could support an association between others' reward responses and directly experienced rewards. Other social cues, such as faces, can also readily derive their value from direct learning experiences. For example, others' smiling faces might often be paired with direct rewards (an unconditioned stimulus (US), such as food and sexual rewards). So far, the ontogeny of social reward processing is not well mapped, but this account would predict that differences pertaining to the social environment during childhood and adolescence would be predictive of individual differences in adult social learning. This conjecture is supported by research showing that rodents reared in social isolation display impaired social learning[91,92], despite showing similar levels of direct conditioning[92]. Studies on the impact of impoverished environments on the human brain[225] suggest that such experiences might cause similar deficits in social learning in humans, possibly through negative implications of prematurely developed amygdala–medial prefrontal cortex connections[90]. It should be noted, however, that the early social learning might occur without the involvement of the anterior cingulate cortex, which is not developed in the infant[226].

An alternative hypothesis is that vicarious reinforcements carry intrinsic value, serving as USs in their own right. Studies of empathic pain responses in non-human animals provide evidence for the role of both conditioning and intrinsic factors[100]. For example, rhesus macaques avoid pulling a rope that simultaneously delivers food reward to themselves and painful electric shocks to a co-specific[101]. This tendency was amplified by previous shock experience, suggesting the contribution of previous individual learning to vicarious reinforcement, but, importantly, did not require it. The same effect has been observed in rodents where an animal's own experiences of shocks enhance[119,128,130], but are not necessary for[118,131,227], vicarious threat conditioning to occur.

A third approach that integrates the two previous ones is that certain social stimuli, such as emotional faces, and other species-specific social cues are innately predisposed to be associated with congruent USs. Similar to prepared avoidance learning[228] about potentially dangerous stimuli, this view argues that learning is necessary, but not sufficient.

Fear contagion
An individual's fear and related behaviours directly trigger similar emotions and behaviours in others.

Vicarious reinforcement learning
Use of vicarious reinforcement as a stand in for directly experienced reinforcement in reinforcement learning algorithms.

Vicarious reinforcement
A motivating outcome, such as a reward or punishment, observed or otherwise known to be incurred by another individual.

Empathy
The sharing and understanding of the affective state of another individual.

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perception of simple social stimuli to the simulation/sharing and understanding of others' emotions[86–89]. For the sake of simplicity, here we refer to these processes jointly as 'social cognition'. An important goal for the neuroscience of social learning is to investigate the role of such information processing in learning. Studies in both humans and other animals suggest that the ability for social learning is conditional on undisrupted development of social skills[90–92] (see BOX 2), underscoring that social cognition is a key constituent in social learning.

Research in non-human animals has shown that exposure to the sight, sound or smell of a threatened demonstrator animal triggers or potentiates threat responses in the observer[93–95]. This phenomenon, commonly referred to as fear contagion[96,97], enhances subsequent direct aversive learning[98]. Fear contagion typically involves the ACC, lateral amygdala, basal amygdala, central amygdala and medial amygdala[95]. Similar responses in humans[99] and other primates[100,101] suggest that a demonstrator's emotional expression of, for example, pain might itself serve as a vicarious reinforcer, a US, imbuing neutral stimuli with value and motivating actions. In such cases, when the outcomes (for example, rewards or punishments) incurred by others can be observed, the same general computational mechanisms that underlie non-social learning can support social

learning. We refer to this as vicarious reinforcement learning (VRL). In VRL, the vicarious US stands in for the self-experienced US to provide the basis for the same type of prediction-error computations as in non-social learning (BOXES 2,3). VRL can involve either model-free or model-based learning mechanisms, and can be used by both the Pavlovian and instrumental systems, thereby driving Pavlovian responses as well as actions (associating cues and vicarious USs, and others' actions and their outcomes, respectively; BOX 3). Pavlovian VRL might, however, be computationally simpler than instrumental VRL, as the former does not require the transformation between observed and own actions, which is known as the 'correspondence problem'[102].

The value of vicarious reinforcement is influenced by higher social cognition, including a representation of the situation and the motivation of the observer[7,89], thereby implicating internal models of others' mental states in the value computation. For example, expressions of pain in a competitor[103] or an out-group member might instigate learning[104], but not empathy and helping behaviour. Interestingly, the formation of stronger and weaker empathic responses and prosocial behaviours can itself be described in terms of reinforcement learning[105,106]. Furthermore, because VRL is a general process, it might provide the computational foundation for many different SLS, because it enables learning about the environment without direct experience, which can be costly in terms of energy expenditure and exposure to predators.

Brain regions implicated in the perception of others' actions might also contribute to social learning via the computation of action prediction errors, which reflect the mismatch between the observed and expected action performed by another individual, as well as between the expected and actual outcome of the action (BOX 3). Such signals have been reported in the dorsolateral PFC and the vmPFC[107,108], respectively, as well as in mirror neuron regions in the premotor and parietal cortex implicated during action perception and simulation[109] (FIGS 2b,3b). These perceptual and motor regions are closely connected with a network of brain regions processing more abstract social cognition, for example, inferences about others' beliefs and intentions. Such mentalizing or 'theory of mind' builds an internal model of the social world and can thus be seen as contributing model-based processes to learning[110]. The superior temporal sulcus (STS) is a multimodal region integrating mentalizing processes carried out in the dorsomedial PFC (dmPFC)[86,111] with information from the action perception stream, importantly including others' gaze direction[112]. In addition, the temporoparietal junction (TPJ)[113], which directs attention to salient information, represents others' beliefs and has been causally linked to strategic mentalizing processes[114] (FIGS 2b,3b). Taken together, these interconnected regions enable humans to attend, monitor and interpret others' emotional expressions, motivations and actions. These processes not only prepare the perceiver for the appropriate action but also provide the foundation for learning from others, which is supported by studies showing that engaging these regions contributes to learning. For example, activity in the dmPFC, the STS and the TPJ while viewing persuasive messages is associated

Box 3 | Computational modelling of social learning

The neuroscience of social learning has capitalized on the success of formal modelling in cognitive neuroscience[229]. Formal models provide testable accounts of the computational mechanisms assumed to underlie behaviour and allow relating latent signals (for example, prediction errors) to neural activity, using model-based functional MRI[230]. The basis of most learning models in cognitive neuroscience is the simple Rescorla–Wagner updating rule that describes how the expected value of cue i (EV_i) changes after prediction errors from trial t to trial $t + 1$:

$$EV_i^{t+1} = EV_i^t + \alpha \times \delta^t \tag{1}$$

$$\delta^t = R^t - EV_i^t \tag{2}$$

where R^t is the outcome value of the reinforcement (the unconditioned stimulus) and α is a learning rate parameter that determines the impact of the prediction error (δ^t, Eq. 2) on the updated expected value. When agents are selecting between different actions, a choice rule (termed Softmax) transforms the current expected values to action probabilities (example with two alternative choices, A and B) at trial t:

$$P_A^t = \exp\left(EV_A^t \times \beta\right) \big/ \left(\exp\left(EV_A^t \times \beta\right) + \exp\left(EV_B^t \times \beta\right)\right) \tag{3}$$

where β, the inverse temperature, determines the sensitivity of the choice to the difference in value between the alternatives. Studies of social learning have extended this basic model to incorporate learning from others. In a seminal study[107], participants observed a demonstrator making choices between two cues and observed the outcomes of these choices, before making choices themselves between the same cues. In their model[107], the observer's own expected values (transformed into probabilities using Eq. 3) served to generate action prediction errors upon seeing the demonstrator's choice:

$$\delta_{ACTION}^t = 1 - P(\text{Observed action})^t \tag{4}$$

δ_{ACTION}^t, the action prediction error, directly biased the observer's subsequent probability of making the same choice (for example, if choice A was observed):

$$P_A^t = P_A^t + \kappa \times \delta_{ACTION}^t \tag{5}$$

where κ determines the strength of 'imitation'. When the outcomes of the demonstrator's actions were revealed, the observer used vicarious reinforcement learning to update expected values according to Eqs. 1,2, substituting α with a_{Social}. The updated expected values were entered into Eq. 3 to determine the choice. By regressing the latent variables of this model on the functional MRI data, it was shown that the dorsolateral prefrontal cortex encoded action prediction errors, whereas the ventral striatum and the ventromedial prefrontal cortex encoded vicarious prediction errors. These findings demonstrate both the utility of computational models for disentangling the processes underlying social learning and the involvement of similar mechanisms as in non-social learning. Recent studies have extended the computational approach by showing how prediction errors encoding different forms of uncertainty modulate social learning[231].

with feeling persuaded afterwards[115]. The increased efforts to apply formal models to the processing of others' emotional[116] and cognitive[117] states will facilitate the integration of social cognition with learning theory through a common computational basis, and thereby further our mechanistic understanding of these and similar findings. Next, we explore how the computational and neural links between domain-general learning and social cognition provide an account of social learning, beginning with its most fundamental forms.

Learning through social means

Social Pavlovian threat learning. When a rat or a mouse is confronted with a conspecific demonstrator receiving foot shocks (a vicarious reinforcement) in the neighbouring compartment of the experimental cage, the observing animal responds with defensive responses, such as freezing[118–120], heart rate deceleration and distress vocalizations[121], and forms threat memories[118] (FIG. 1a). Social threat learning can also emerge from interaction with a conspecific expressing a learned defensive response (fear conditioning by proxy[122] or a mother-to-infant transfer of fear[80]). For example, seminal studies in monkeys[123] showed that cage-reared monkeys quickly acquired long-lasting defensive responses towards snakes after only one exposure to a conspecific's expressions of distress, including withdrawal, grimacing and vocalization (serving as vicarious reinforcements) (FIG. 1b). Importantly, the strength of the relationship between the demonstrator's distress, the observer's immediate response to the demonstrator and the subsequent expression of learning by the observer was comparable to the relationship between the US, the unconditioned response and the conditioned response in non-social threat learning. These findings support the view that socially transmitted Pavlovian learning and self-experienced Pavlovian conditioning rely on similar learning mechanisms. Recently, these findings have been confirmed and extended in human children[81,124] and adults[125–127] using measures of behaviour and peripheral and central neurophysiology (FIG. 1c). The studies surveyed in this section illustrate how SLS, such as 'copy when non-social learning is costly' (for example, under risk of predation), might be underpinned by basic learning mechanisms.

As discussed previously, the basolateral amygdala, in particular its lateral part and the ACC — key structures involved in direct Pavlovian threat conditioning (FIG. 2) — are similarly critical for vicariously learned freezing responses and subsequent recall of context-specific threat memory[118]. Recently, specific neurons in the ACC that respond both when rats experience pain and while they witness another rat receiving foot shocks have been identified, suggesting that some neuronal mechanisms are shared by direct and social experience[128]. Interestingly, whereas some researchers have suggested that parts of the rodent ACC are specifically involved in vicarious learning[118], other research shows the involvement of the ACC in direct fear learning[129]. This and similar discrepancies may stem from different behavioural protocols of threat conditioning used in these studies, as well as from functional heterogeneity within the ACC, with some projections being involved specifically in social, and others in direct, Pavlovian learning. In line with this, recent studies have shown that the ACC–basolateral amygdala projection[130], as well as the lateral amygdala–medial amygdala projection[131], are critical for social, but not direct Pavlovian, threat learning[130], whereas the ACC–hippocampus projection is involved in retrieval of fear memory originating in self-experienced events[32]. Verifying the potential double functional dissociation of these pathways in direct and social threat learning, respectively, requires further studies that examine the fine-grained functional heterogeneity of the ACC.

Findings from brain imaging studies in humans resonate nicely with the animal work described above by showing that social learning recruits networks involved in direct learning, including the amygdala, anterior insular cortex and ACC[28,126,132,133] (FIG. 2a,b).

Domain-general learning
Mechanisms contributing to many cognitive functions, across situations and tasks.

Vicarious learning
Learning from others without any directly experienced reinforcement. Sometimes used synonymously with 'observational learning'.

As yet, methodological constraints in current human brain imaging techniques preclude the identification of activity in separate amygdala nuclei, but individuals with lesions constrained to the basolateral amygdala[134] and neurosurgical patients with implanted depth electrodes[135] offer future opportunities to test analogous processes in humans.

Combined imaging and computational modelling has shown that the amygdala gates prediction-error signals to the ACC in Pavlovian learning via both direct and social experiences[126]. Learning by observing others' pain expressions also engages activity in the PAG[28], which is known to be central to defensive responses and self-experienced pain via the regulation of the endogenous opioid neuropeptide system and its prediction error-like qualities[27,136].

The studies in both rodents and humans so far reviewed suggest a shared processing of social and non-social information, concurrent with the view that these sources of information are processed in a general value-representation circuit[8]. Importantly, however, recent studies in humans have also shown that social threat learning is distinguished from direct learning in several ways. For example, as compared with learning through direct experiences of an aversive shock, learning by watching a demonstrator's reactions to a shock provokes greater functional connectivity between the PAG and the STS[28], between the amygdala, the STS and the TPJ, as well as between the anterior insular cortex and the TPJ[126]. Moreover, a recent experiment[126] used dynamic causal modelling of connectivity to characterize the flow of information in the amygdala–anterior insular cortex–ACC network during both direct and vicarious threat learning, and showed that information about the US (self and other experienced shock) was most likely to enter the network through the amygdala during direct conditioning and through the anterior insular cortex during vicarious learning. The latter finding resonates well with the role of the anterior insular cortex and the ACC in emotional sharing and empathy[99] and helps to explain why activity in these regions[133], as well as empathic appraisals[137], predicts the strength of vicarious threat learning. Although these findings are compatible with the general value-representation view of social learning, they suggest that learning also occurs outside a common learning circuit.

Recent research has extended the study of social learning in humans to the transmission of safety information. In these experiments, the participants (observers) watch a calm-looking demonstrator modelling safety when confronted with a CS previously paired with shocks to the observers. The results have revealed that social safety learning leads to superior attenuation of the conditioned threat response as compared with traditional self-experienced extinction training[138,139] and recruits the vmPFC[45].

Taken together, research has demonstrated that the neuro-computational mechanisms underlying socially mediated Pavlovian threat and safety learning are both common and distinct to those underlying direct Pavlovian learning. Many of these distinctive features can be examined by systematically manipulating the social characteristics of the demonstrator. This is discussed next.

Social biases. As social learning is dependent on social cognition, it is affected by various biased inferences and decisions that have been described across species in behavioural ecology, and in humans in social psychology. For example, primates are more likely to imitate and learn behaviours demonstrated by a dominant group member[140,141], and greater perceived similarity with a demonstrator produces stronger threat learning in both mice[142] and humans[104,143]. Although not directly tested, these effects are likely to rely partly on both cognitive and affective social processes. For example, observing and thinking about dissimilar versus similar others is related to both less mentalizing[144] and affect sharing/empathic responses[105] in prefrontal regions. In accordance with 'demonstrator-based' SLS, research has shown that social threat learning is less dependent on perceived similarity with the demonstrator, as compared with social safety learning, which depends on observer–demonstrator similarity[138]. The neural substrates of this group bias in safety learning remain unknown. In humans, the perceiver's belief that the demonstrator is truly experiencing pain, in contrast to just pretending, causes stronger learning in an observational learning situation[137,145], suggesting the involvement of model-based computations. Such beliefs about the demonstrator's internal state can be induced by, for example, verbal instructions, comprising another common, and uniquely human, means of socially transmitting information about value.

Verbal learning. Verbal information can elicit Pavlovian responses without observed or self-experienced US and CS–US pairings. Studies in which participants are merely informed about the risk of receiving shocks in a specific context or that specific cues might be paired with shock have consistently demonstrated increased physiological arousal[146,147] and activation in many regions of the brain's threat learning network[146,148]. This on-the-fly linking of social information to Pavlovian responses suggests the involvement of model-based, as opposed to model-free, Pavlovian control in certain types of social learning[15,149]. Social information can also powerfully modify individual learning experiences. For example, verbal instructions about changed threat contingencies can cause rapid updating of conditioned physiological responses[150–152], which is reflected by dorsolateral PFC activity, in contrast to the amygdala, which tracks direct experience[150].

Social Pavlovian reward learning. Studies with insects have provided compelling evidence for the view that social reward learning can originate from basic, domain-general learning processes[153]. For example, a recent study showed that bumblebees' tendency to approach flowers where conspecifics forage directly depends on previous pairings of conspecifics and reward[154]. Conspecifics thereby come to serve as a second-order reward predicting conditioned stimuli through pairing with the direct US (reward), which elicits approach behaviour.

Observational learning
Learning through observing the responses and behaviour of others, which may or may not involve reinforcement.

Notably, when conspecifics were instead paired with an aversive liquid, the bumblebees avoided flowers with other bumblebees. In other words, social reward learning could in these cases be explained by Pavlovian system responses, acquired through basic, model-free non-social learning mechanisms[153]. Social learning that facilitates finding potential food sources has also been described in rodents[155]. For example, the association between a smell of novel food and carbon disulfide, a component of rodent breath, with intrinsic, rewarding value[156,157] expedites the formation of food preference. Such socially acquired food preference memories require the orbitofrontal cortex (but see REF.[158]), PFC, hippocampus and basolateral amygdala[159–161] (FIG. 3a); that is, the same brain regions involved in direct Pavlovian appetitive learning. Similarly, overlapping mechanisms of social and non-social learning might exist in the NAcc, based on the demonstration of dopamine release in the NAcc of rats witnessing a conspecific receiving a sucrose reward[162].

Non-social and social learning also seem to share specific mechanisms underlying the coding of value along the positive–negative continuum. Accordingly, valence-sensitive neurons that process positive and negative non-social stimuli, either innate or learned, have been found in many brain structures, including the NAcc, VTA and amygdala[51,163,164]. These neurons form neuronal circuits that compete with each other through reciprocal inhibition[165]. Interestingly, a class of positive-valence neurons sensitive to both food and a social reward has recently been identified in the mouse basolateral amygdala[165], suggesting that valence-coding neurons are also involved in vicarious learning.

In humans, the learning mechanisms underlying social Pavlovian reward learning are less clear. A meta-analysis[166] of imaging studies examining responses (but not explicitly learning) to personal and vicarious reward found that these engaged overlapping regions of the vmPFC, consistent with the role of this region in the computation of value. However, whereas the NAcc was identified as being preferentially engaged in directly, as compared with vicariously, experienced reward, brain regions related to mentalizing were more engaged in vicariously, as compared with directly, experienced rewards (FIG. 3b). Analogous to the previously discussed findings in the aversive (threat) domain, these results highlight both common and unique components of directly and vicariously experienced reward processing.

Similar to aversive learning, vicarious reward learning is biased by the observer's evaluations of the demonstrator, underscoring the relevance of demonstrator-based or 'who' SLS. For example, an imaging study in humans[167] showed that observers watching a confederate winning at a lottery rated the experience more enjoyable when the demonstrator was seen as socially desirable and similar to the self, as compared with socially undesirable and dissimilar to the self. Perceived liking increased activity in the ventral striatum — a region also activated when the observer won themselves — and similarity in the ACC, underscoring the role of this region in processing of social information. The impact of similarity in vicarious reward and its impact on the ventral striatum

can, however, be modified by motivational factors, such as costs to the self and competitive goals[89,162,168]. For example, watching an unfair competitor receiving painful stimulation can generate reward responses in the observer, including activity in the ventral striatum[169].

Although most studies have assumed that vicarious reward learning is based on model-free learning rules (for example, the Rescorla–Wagner learning rule), a recent study, in which participants observed a demonstrator receiving rewards in association with specific cues, has questioned this view[110]. By contrasting neural activity related to a model-based algorithm (which learned the transition probability, or statistical association, between cues and outcomes) with a model-free reinforcement learning algorithm, the study revealed model-based, but not model-free, neural signals. Specifically, the intraparietal sulcus represented model-based state prediction errors during both vicarious and experiential learning, whereas no regions exhibited model-free vicarious prediction errors. Because a basic model-free algorithm should be able to form such associations easily, when and why such model-based computations are used during simple forms of social learning remains an important open question.

Social instrumental threat learning. One of the theoretical key benefits of social learning is that it allows avoiding predation and other threats without costly first-hand experience[170], as expressed in the 'copy when non-social learning is costly' SLS. Behavioural studies support this prediction. For example, a classic study showed that minnows (a species of fish) are more likely to select a socially demonstrated patch during high, in contrast to low, predation risk, even if previous individual learning conflicted with social demonstration[79]. Similarly, rats can socially learn to avoid harmful stimuli, such as a candle flame[171] or a foot shock[172,173] (FIG. 1d). Lesions of the medial PFC enhance the social learning of such avoidance responses, which suggests a suppressive role of this region in social modulation of avoidance[174].

A recent study in humans demonstrated how Pavlovian threat learning through observation of a demonstrator receiving shocks biases later instrumental self-experienced learning involving the same stimuli, in a manner similar to Pavlovian–instrumental transfer[21]. This study shows how the basic computational principles of non-social learning apply also to the social domain. It remains an open question whether this social bias of decision-making is underpinned by brain regions critical for aversive learning, such as the amygdala and the PAG, or regions involved in higher-level mentalizing processes.

One of the most direct tests of the 'copy when non-social learning is costly' SLS in humans investigated how people behave when they know the environment is dangerous (actions risked incurring electric shocks) and another agent's behaviour can be observed[175] (FIG. 1f). This situation produced an almost deterministic copying of the observed behaviour, much higher than when people thought their actions might be rewarded. Computational modelling showed that people assigned value to the demonstrated action and used this value

to guide their own decisions. This shows how basic domain-general learning mechanisms, combined with a tendency to copy others, can generate behaviour consistent with distinct SLS. It is likely that brain regions involved in non-social reinforcement learning, such as the ventral striatum, contribute to these computational mechanisms.

Social instrumental reward learning. Social learning also allows us to optimize behaviour to maximize reward. For example, rats learn actions to obtain a food reward more quickly if they first observed this response performed by another, well-trained rat, compared with rats that did not observe conspecifics and rats that observed conspecifics not emitting response-relevant cues[176–178]. Similarly, spatial discrimination learning is facilitated by observation of a demonstrator first performing the task[179,180].

The neural structures involved in the vicarious learning of reward-guided instrumental behaviour in non-human animals remain largely unknown. The scarce literature on this topic suggests the involvement of the medial PFC and the ventral striatum, in particular the NAcc, in observational learning of pressing a lever for food reinforcement[181] (FIG. 3a), similar to direct learning of the task. Research in humans has also implicated the striatum in observational instrumental reward learning. For example, in an early study, participants both observed others making decisions and made decisions themselves for juice rewards[182]. Crucially, the dorsal striatum exhibited (model-free) prediction-error signals during both social and non-social learning. This, and other similar studies[107,108], suggest that at least this type of VRL utilizes computational and neural mechanisms highly similar to those involved in non-social learning. A recent study provided converging support for this conclusion by directly recording human single-neuron activity[183]. Neurons in the amygdala, rostral ACC (rACC) and medial PFC were recorded during a probabilistic card-selection task, in which the participants learned about the best options in their environment by both direct experience and observing demonstrators. A subset of neurons in all three recorded regions tracked the expected value of the card chosen by the demonstrator, but, crucially, only the rACC encoded both the demonstrators' outcomes and prediction errors in the way posited by reinforcement learning theory. Notably, the same rACC neurons did not encode direct, self-experienced prediction errors. However, as no neurons were recorded in the ventral striatum or the dopaminergic midbrain, the relationship between the rACC and those more classic reinforcement learning-related regions in social learning remains unclear.

The results reviewed in the previous paragraph dovetail with the recent view that the gyrus of the ACC (ACCg) plays a central role in tracking the motivation of others[184]. Indeed, recent studies in both humans and non-human primates provide direct evidence for the importance of the ACCg for multiple aspects of vicarious reinforcement processing and social learning. For example, a single unit study on the rewarding outcomes of social decisions in monkeys showed that neurons in the ACCg preferentially encoded the allocation of reward to

a peer monkey[185]. Similarly, research in humans using functional MRI has revealed that the ACCg responds to both vicarious punishment and reward, for example cues that are predictive of others' monetary gain[186,187] and experience of pain[99,188]. Interestingly, the ACCg may also conform to the principles of reinforcement learning by coding expectations and prediction errors about other people's decisions[6]. Taken together, these findings have led to the suggestion that the ACCg allocentrically represents information about the consequences of actions of — or for — others, in contrast to egocentric representations about the outcomes of one's own actions[184]. Functionally, such allocentric coding might be especially useful in competitive interaction, as it would enable a representation of an updated prediction of others' behaviour, as well as learning from those predictions. Interestingly, research tracing the white tract fibres of the primate brain shows that the ACCg is uniquely positioned to do so, because it is connected with the neural circuitry implicated in mentalizing and simulation of others' actions, involving the dmPFC, the TPJ and the action/mirror system, respectively[6,108,189].

In sum, the functional, neural and computational properties of the ACCg highlight this as one of the core regions guiding social reward learning. It remains to be seen, however, whether connections between the ACC and social cognitive and reward processing regions are necessary for social instrumental reward learning to occur, similarly to how, for example, connections between the ACC and the amygdala are necessary for social threat learning[118,130].

Although most studies assume that social reward learning of instrumental actions is model-free, a recent study suggests that it can take either model-based or model-free forms, depending on whether observed actions map to specific outcomes or have a simpler stimulus–response form[190]. By devaluating the value of the outcome[13], it was found that observationally learned action–outcome associations were more model-based, because responding ceased for the stimulus linked to the devalued outcome, whereas stimulus–response associations were more model-free, because responding was preserved for the stimulus linked to the devalued outcome. This pattern directly mirrors the distinction between model-based and model-free reinforcement learning in the non-social learning domain. Moreover, the difference between the model-based and model-free modes of social learning was reflected in distinct patterns of striatal activity, where model-based more strongly recruited the dorsomedial parts. Notably, model-based, or goal-directed, social learning has also been established in children[191] and several non-human species[192,193].

For humans, not all types of social instrumental reward learning require direct observation of others' behaviours or their consequences (that is, VRL). The capacity for symbolic representation of information, such as in language, provides a unique means for social learning. In contrast to research on instructed threat described above, studies of verbal information transmission in the reward domain have primarily focused on how verbal or written 'advice' about what action to choose influences subsequent non-social learning.

This experimental setting is intended to capture a wide class of important real-world situations, such as consumer behaviour[194], where others' opinions might be central. Indeed, advice exerts a strong influence on individual behaviour[195–197], which can be directly detrimental in cases when the advice is misleading[198,199]. Computationally, advice appears to influence individual behaviour by adding a 'bonus' to outcomes that are consistent with the advice, which neurally is mirrored by dorsal striatum activity[195]. This might indicate that advice modulates the computations of the model-free instrumental system, although a model-based system could in principle incorporate advice and other types of symbolic information during instrumental learning.

The detrimental effects of misleading advice highlight one of the major theoretical problems with social learning: it can be error prone[200]. This is addressed by the important class of 'who' SLS described above, which prescribes heuristics that increase the chances of learning from individuals with superior skill and knowledge. For example, both human children[201] and chimpanzees use a 'copy knowledgeable others' strategy[141], and adult humans rely more on social learning if demonstrators are described as highly skilled[202] or as having high intelligence[203]. Recent studies provide a window into the neuro-computational mechanisms that might underlie such inferences. For example, learning about others' preferences, which can be used to infer their value as demonstrators, might be underpinned by a type of reinforcement learning that is unique (or most relevant) to social behaviour. In a recent study[204], participants observed the choices, but never the outcomes, of other agents who they knew (from a previous learning session) had either similar or dissimilar preferences for types of food as themselves. Crucially, this setting did not provide the opportunity for VRL, as only others' actions were visible. Notably, rather than simply imitating these actions, participant behaviour was best explained by so-called inverse reinforcement learning[205], a class of algorithms that allows learning from agents that have diametrically opposing preferences from the observer, which is not typically the case for imitation-based methods. Furthermore, inverse reinforcement learning updating signals were represented by the TPJ and the STS, both key regions of the mentalizing network. Taken together, these studies show how 'who' SLS can be implemented in the human brain and underline the unique role of mentalizing regions in this type of learning. It is possible that inferences related to mentalizing are crucial for certain SLS[206], for example, 'copy based on demonstrator's knowledge' that is thought to be important for human culture. Moreover, algorithms based on inverse reinforcement learning might serve as the computational basis for such inferences about others' preferences and intentions[207].

Social learning in groups
The majority of neuroscientific social learning research has focused on situations with only one demonstrator, or a few. One of the most important classes of SLS, however, is 'copy the majority'[79], implicating multiple individuals. Theoretical models have shown that such conformity in social learning is an important ingredient for cultural evolution through its contribution to the rapid transmission of cultural traits[208,209], and field experiments have documented that humans tend to conform to the majority[209,210]. An increasing number of animal studies have shown similar tendencies to follow the majority[211–213], although the question of to what extent non-humans display conformity remains a contentious issue[214].

Although most neuroscience studies of conformity do not focus on learning[215], they do provide important clues about the possible mechanisms underlying 'copy the majority' SLS. For example, studies show that reward-sensitive areas (for example, the ventral striatum and the vmPFC) that are important in learning, signal the individuals' conformity to the majority[216]. In a popular task, participants rate stimuli, such as faces, first alone and then again after seeing the average rating from an anonymous group of people[216,217]. Using this task, it has been shown that initial agreement between the participant and the majority group was related to ventral striatum activity[218], whereas disagreement with the majority resulted in deactivation of the ventral striatum. Both signals predicted subsequent conformity[217,218]. This pattern of activations is consistent with an unsigned prediction error, signalling that one differs from the group but not the direction of the difference, and this signal might be used for adjusting to group norms. Related work on persuasion highlights the role of the mentalizing network in conformity. This research has shown that, across western and Asian cultures, increased activity in the dmPFC, the STS and the TPJ while viewing persuasive messages was associated with subjectively feeling persuaded afterwards[115]. Taken together, the findings discussed here show that, similar to many other forms of social learning, conformity involves both basic learning and valuation brain regions and regions key to social cognition.

The study of social learning is important for our understanding of how interactions in groups emerge and change. To capture the nature of group interaction, studies should ideally allow for real-time interaction between experimental participants[88]. For practical reasons, however, this is rarely done. In a landmark study of the neural mechanisms that allow consensus to be reached in social groups, a focal participant in the MRI scanner interacted in real time with a group of participants in a behavioural laboratory[219]. Using this dynamic set-up, it was found that people reached consensus by integrating their own preferences with the prior majority preference in the group, modulated by an estimate of the perceived 'stickiness' of the majority preferences (that is, how much the other participants tended to stick to their choices). Using computational modelling, it was found that these key social decision-making variables were encoded in distinct brain regions. Whereas the individual's own preferences were encoded in the vmPFC, the prior preferences of the group majority were reflected in the STS and TPJ, suggesting the involvement of mentalizing-related processes. Finally, the perceived stickiness of the majority preferences correlated with activity in the intraparietal sulcus. This study exemplifies how the neural mechanisms involved in social

learning can be studied in a dynamic social environment. Similar experimental paradigms might prove valuable for understanding how neural processes on the individual level can 'scale up', and thereby contribute to social norms, cultural evolution and other large-scale social phenomena.

Conclusions and future directions

Similar to how social cognition repurposes existing domain-general cognitive processes[8,220], social learning co-opts basic Pavlovian and instrumental learning systems, sharing both neural and computational mechanisms with self-experienced learning. This conclusion supports the view that social and non-social sources of information are processed in a general value-representation circuit[8]. Social learning, however, is also distinguished from learning based on self-experiences in several important ways. For example, in close interaction with the surrounding environment, social learning aligns with certain kinds of strategies (SLS) that are shared across species and uses a unique set of brain regions associated with the processes of social information. In our species, social learning also draws extensively on model-based mentalizing and meta-cognitive processes.

The realization that social learning is fundamental to social behaviour across many species and is key to the emergence and transmission of all aspects of human culture has spurred a surge of interest in this topic. New theoretical models in behavioural ecology and computational learning theory propel this development together with new experimental methods to measure dynamic social interactions and neurobiological techniques, such as optogenetics, used to isolate the underlying brain correlates. These exciting developments have also opened up new questions to address in future research.

Important future directions can be summarized in four categories. First, in light of the developments outlined here, we must continue to bring together understanding of the function (computationally and phylogenetically) of social learning with its neural architecture. This pursuit will help to answer important open questions about social learning, such as in what ways this is distinct from non-social learning (BOX 1). Second, and related to the first direction, extended work on non-human animals is needed to better map the molecular and cellular levels of social learning[87]. Optogenetic and functional imaging techniques will enable researchers to control and monitor individual neurons in social situations, which is crucial for uncovering the underlying neural implementation of learning computations. This development should benefit not only from new animal models but from translating human paradigms to animals. Moreover, the use of modern imaging tools together with improved computational and statistical analysis methods will provide more efficient data extraction, thus reducing the number of animals used. Third, social learning plays an important role in the transmission of maladaptive fears and anxiety, such as those occurring in anxiety disorders and post-traumatic stress. More knowledge is needed about both the social aetiology of such disorder and the ways social learning, such as vicarious safety learning[139], can help the development of new treatments. Our understanding of the neural and computational mechanisms of social learning lays the ground for several new promising avenues for research alliances between developmental and clinical scientists. Fourth, research on social learning needs to bridge between the levels of analyses to understand how learning scales up from the individual to social networks and larger group constellations that are of societal importance. For example, social learning mechanisms described in the individual based on laboratory experiments in constrained group settings can be modelled in virtual agents, which are allowed to interact in social environments that simulate natural social situations[170,175]. The emergent social behaviours of these agents can then be verified against observations of large-scale real-world behaviour. We conclude that the study of social learning enables several unique ways to bridge neural and computational models with an understanding of behavioural change of varying complexity, from the learning of basic threat avoidance to complex SLS and cultural phenomena.

Published online 12 March 2020

1. Bandura A. Social Learning Theory (Prentice-Hall, 1977).
2. Boyd, R., Richerson, P. J. & Henrich, J. The cultural niche: why social learning is essential for human adaptation. *Proc. Natl. Acad. Sci. U.S.A.* **108**, 10918–10925 (2011).
   This review describes the central and adaptive role of social learning in human culture.
3. Debiec, J. & Olsson, A. Social fear learning: from animal models to human function. *Trends Cogn. Sci.* **21**, 546–555 (2017).
4. Schilbach, L. et al. Toward a second-person neuroscience. *Behav. Brain Sci.* **36**, 393–414 (2013).
5. Kendal, R. L. et al. Social learning strategies: bridge-building between fields. *Trends Cogn. Sci.* **22**, 651–665 (2018).
6. Apps, M. A. J., Rushworth, M. F. S. & Chang, S. W. C. The anterior cingulate gyrus and social cognition: tracking the motivation of others. *Neuron* **90**, 692–707 (2016).
7. Falk, E. & Scholz, C. Persuasion, influence, and value: perspectives from communication and social neuroscience. *Annu. Rev. Psychol.* **69**, 329–356 (2018).
8. Ruff, C. C. & Fehr, E. The neurobiology of rewards and values in social decision making. *Nat. Rev. Neurosci.* **15**, 549–562 (2014).

9. Dayan, P. & Balleine, B. W. Reward, motivation, and reinforcement learning. *Neuron* **36**, 285–298 (2002).
10. Sutton, R. S. & Barto, A. G. *Reinforcement Learning: An Introduction* (MIT Press, 1998).
11. Rescorla, R. A. & Wagner, A. R. in *Classical Conditioning II: Current Research and Theory* (eds Black, A. H. & Prokasy, W. F.) 64–99 (Appleton-Century-Crofts, 1972).
12. Watkins, C. J. C. H. & Dayan, P. Q-learning. *Mach. Learn.* **8**, 279–292 (1992).
13. Dickinson, A. Actions and habits: the development of behavioural autonomy. *Philos. Trans. R. Soc. B Biol. Sci.* **308**, 67–78 (1985).
14. Dolan, R. J. & Dayan, P. Goals and habits in the brain. *Neuron* **80**, 312–325 (2013).
15. Bach, D. R. & Dayan, P. Algorithms for survival: a comparative perspective on emotions. *Nat. Rev. Neurosci.* **18**, 311–319 (2017).
16. Dayan, P., Niv, Y., Seymour, B. & Daw, D. N. The misbehavior of value and the discipline of the will. *Neural Netw.* **19**, 1153–1160 (2006).
17. Bouton, M. E. *Learning and Behavior: A Contemporary Synthesis* (Sinauer Associates, 2007).
18. Miller, K., Shenhav, A. & Ludvig, E. Habits without values. *Psychol. Rev.* **126**, 292–311 (2019).

19. Rangel, A., Camerer, C. & Montague, P. R. A framework for studying the neurobiology of value-based decision making. *Nat. Rev. Neurosci.* **9**, 545–556 (2008).
20. Guitart-Masip, M., Duzel, E., Dolan, R. & Dayan, P. Action versus valence in decision making. *Trends Cogn. Sci.* **18**, 194–202 (2014).
21. Lindström, B., Golkar, A., Jangard, S., Tobler, P. N. & Olsson, A. Social to instrumental transfer of fear in human decision-making. *Proc. Natl Acad. Sci. USA* **116**, 4732–4737 (2019).
    This study demonstrates how the interaction between Pavlovian and instrumental value systems in social learning can result in maladaptive decision making.
22. LeDoux, J. E., Cicchetti, P., Xagoraris, A. & Romanski, L. M. The lateral amygdaloid nucleus: sensory interface of the amygdala in fear conditioning. *J. Neurosci.* **10**, 1062–1069 (1990).
23. Maren, S., Phan, K. L. & Liberzon, I. The contextual brain: implications for fear conditioning, extinction and psychopathology. *Nat. Rev. Neurosci.* **14**, 417–428 (2013).
24. Sotres-Bayon, F. & Quirk, G. J. Prefrontal control of fear: more than just extinction. *Curr. Opin. Neurobiol.* **20**, 231–235 (2010).

25. LeDoux, J. E., Iwata, J., Cicchetti, P. & Reis, D. J. Different projections of the central amygdaloid nucleus mediate autonomic and behavioral correlates of conditioned fear. *J. Neurosci.* **8**, 2517–2529 (1988).

26. Grahl, A., Onat, S. & Büchel, C. The periaqueductal gray and Bayesian integration in placebo analgesia. *eLife* **7**, e32930 (2018).

27. McNally, G. P., Johansen, J. P. & Blair, H. T. Placing prediction into the fear circuit. *Trends Neurosci.* **34**, 283–292 (2011).

28. Haaker, J., Yi, J., Petrovic, P. & Olsson, A. Endogenous opioids regulate social threat learning in humans. *Nat. Commun.* **8**, 15495 (2017).
Using a pharmacological manipulation, this paper describes the role of the opioidergic system in social Pavlovian threat learning.

29. Gore, F. et al. Neural representations of unconditioned stimuli in basolateral amygdala mediate innate and learned responses. *Cell* **162**, 134–145 (2015).

30. LaBar, K. S., Gatenby, J. C., Gore, J. C., LeDoux, J. E. & Phelps, E. A. Human amygdala activation during conditioned fear acquisition and extinction: a mixed-trial fMRI study. *Neuron* **20**, 937–945 (1998).

31. Phelps, E. A. & LeDoux, J. E. Contributions of the amygdala to emotion processing: from animal models to human behavior. *Neuron* **48**, 175–187 (2005).

32. Rajasethupathy, P. et al. Projections from neocortex mediate top-down control of memory retrieval. *Nature* **526**, 653–659 (2015).

33. Alves, F. H. F. et al. Involvement of the insular cortex in the consolidation and expression of contextual fear conditioning. *Eur. J. Neurosci.* **38**, 2300–2307 (2013).

34. Craig, A. D. How do you feel — now? The anterior insula and human awareness. *Nat. Rev. Neurosci.* **10**, 59–70 (2009).

35. de la Vega, A., Chang, L. J., Banich, M. T., Wager, T. D. & Yarkoni, T. Large-scale meta-analysis of human medial frontal cortex reveals tripartite functional organization. *J. Neurosci.* **36**, 6553–6562 (2016).

36. Lieberman, M. D. & Eisenberger, N. I. The dorsal anterior cingulate cortex is selective for pain: results from large-scale reverse inference. *Proc. Natl Acad. Sci. USA* **112**, 15250–15255 (2015).

37. Shackman, A. J. et al. The integration of negative affect, pain and cognitive control in the cingulate cortex. *Nat. Rev. Neurosci.* **12**, 154–167 (2011).

38. Fullana, M. A. et al. Neural signatures of human fear conditioning: an updated and extended meta-analysis of fMRI studies. *Mol. Psychiatry* **21**, 500–508 (2016).

39. Dunsmoor, J. E., Niv, Y., Daw, N. & Phelps, E. A. Rethinking extinction. *Neuron* **88**, 47–63 (2015).

40. Milad, M. R. & Quirk, G. J. Fear extinction as a model for translational neuroscience: ten years of progress. *Annu. Rev. Psychol.* **63**, 129–151 (2012).

41. Phelps, E. A., Delgado, M. R., Nearing, K. I. & LeDoux, J. E. Extinction learning in humans: role of the amygdala and vmPFC. *Neuron* **43**, 897–905 (2004).

42. Fullana, M. A. et al. Fear extinction in the human brain: a meta-analysis of fMRI studies in healthy participants. *Neurosci. Biobehav. Rev.* **88**, 16–25 (2018).

43. Gentry, R. N. & Roesch, M. R. Neural activity in ventral medial prefrontal cortex is modulated more before approach than avoidance during reinforced and extinction trial blocks. *J. Neurosci.* **38**, 4584–4597 (2018).

44. Dunsmoor, J. E. et al. Role of human ventromedial prefrontal cortex in learning and recall of enhanced extinction. *J. Neurosci.* **39**, 3264–3276 (2019).

45. Golkar, A., Haaker, J., Selbing, I. & Olsson, A. Neural signals of vicarious extinction learning. *Soc. Cogn. Affect. Neurosci.* **11**, 1541–1549 (2016).
The first imaging study on vicarious extinction learning, describing the role of the vmPFC in this social form of safety learning.

46. Roitman, M. F., Wheeler, R. A. & Carelli, R. M. Nucleus accumbens neurons are innately tuned for rewarding and aversive taste stimuli, encode their predictors, and are linked to motor output. *Neuron* **45**, 587–597 (2005).

47. Schultz, W., Apicella, P., Scarnati, E. & Ljungberg, T. Neuronal activity in monkey ventral striatum related to the expectation of reward. *J. Neurosci.* **12**, 4595–4610 (1992).

48. Waelti, P., Dickinson, A. & Schultz, W. Dopamine responses comply with basic assumptions of formal learning theory. *Nature* **412**, 43–48 (2001).

49. Hu, H. Reward and aversion. *Annu. Rev. Neurosci.* **39**, 297–324 (2016).

50. Cohen, J. Y., Haesler, S., Vong, L., Lowell, B. B. & Uchida, N. Neuron-type-specific signals for reward and punishment in the ventral tegmental area. *Nature* **482**, 85–88 (2012).

51. Lammel, S. et al. Input-specific control of reward and aversion in the ventral tegmental area. *Nature* **491**, 212–217 (2012).
Study showing that distinct circuits within this structure can generate reward and aversion.

52. Everitt, B. J. et al. Associative processes in addiction and reward. The role of amygdala–ventral striatal subsystems. *Ann. N. Y. Acad. Sci.* **877**, 412–438 (1999).

53. Stuber, G. D. et al. Excitatory transmission from the amygdala to nucleus accumbens facilitates reward seeking. *Nature* **475**, 377–380 (2011).

54. Ramirez, S. et al. Activating positive memory engrams suppresses depression-like behaviour. *Nature* **522**, 335–339 (2015).
Study showing that optogenetic dissecting of a memory engram is possible.

55. Namburi, P. et al. A circuit mechanism for differentiating positive and negative associations. *Nature* **520**, 675–678 (2015).

56. Janak, P. H. & Tye, K. M. From circuits to behaviour in the amygdala. *Nature* **517**, 284–292 (2015).

57. Reekie, Y. L., Braesicke, K., Man, M. S. & Roberts, A. C. Uncoupling of behavioral and autonomic responses after lesions of the primate orbitofrontal cortex. *Proc. Natl Acad. Sci. USA* **105**, 9787–9792 (2008).

58. Schoenbaum, G., Setlow, B., Saddoris, M. P. & Gallagher, M. Encoding predicted outcome and acquired value in orbitofrontal cortex during cue sampling depends upon input from basolateral amygdala. *Neuron* **39**, 855–867 (2003).

59. Ferenczi, E. A. et al. Prefrontal cortical regulation of brainwide circuit dynamics and reward-related behavior. *Science* **351**, aac9698 (2016).

60. Otis, J. M. et al. Prefrontal cortex output circuits guide reward seeking through divergent cue encoding. *Nature* **543**, 103–107 (2017).

61. Haber, S. N. & Knutson, B. The reward circuit: linking primate anatomy and human imaging. *Neuropsychopharmacology* **35**, 4–26 (2009).

62. Delgado, M. R. Reward-related responses in the human striatum. *Ann. N. Y. Acad. Sci.* **1104**, 70–88 (2007).

63. Amorapanth, P., LeDoux, J. E. & Nader, K. Different lateral amygdala outputs mediate reactions and actions elicited by a fear-arousing stimulus. *Nat. Neurosci.* **3**, 74–79 (2000).

64. Killcross, S., Robbins, T. W. & Everitt, B. J. Different types of fear-conditioned behaviour mediated by separate nuclei within amygdala. *Nature* **388**, 377–380 (1997).

65. Wang, J., Bast, T., Wang, Y.-C. & Zhang, W.-N. Hippocampus and two-way active avoidance conditioning: contrasting effects of cytotoxic lesion and temporary inactivation. *Hippocampus* **25**, 1517–1531 (2015).

66. Bravo-Rivera, C., Roman-Ortiz, C., Brignoni-Perez, E., Sotres-Bayon, F. & Quirk, G. J. Neural structures mediating expression and extinction of platform-mediated avoidance. *J. Neurosci.* **34**, 9736–9742 (2014).

67. Parker, N. F. et al. Reward and choice encoding in terminals of midbrain dopamine neurons depends on striatal target. *Nat. Neurosci.* **19**, 845–854 (2016).

68. Moorman, D. E. & Aston-Jones, G. Prefrontal neurons encode context-based response execution and inhibition in reward seeking and extinction. *Proc. Natl Acad. Sci. USA* **112**, 9472–9477 (2015).

69. Yin, H. H., Ostlund, S. B., Knowlton, B. J. & Balleine, B. W. The role of the dorsomedial striatum in instrumental conditioning. *Eur. J. Neurosci.* **22**, 513–523 (2005).

70. Balleine, B. W., Killcross, A. S. & Dickinson, A. The effect of lesions of the basolateral amygdala on instrumental conditioning. *J. Neurosci.* **23**, 666–675 (2003).

71. Yin, H. H., Knowlton, B. J. & Balleine, B. W. Lesions of dorsolateral striatum preserve outcome expectancy but disrupt habit formation in instrumental learning. *Eur. J. Neurosci.* **19**, 181–189 (2004).

72. Killcross, S. & Coutureau, E. Coordination of actions and habits in the medial prefrontal cortex of rats. *Cereb. Cortex* **13**, 400–408 (2003).

73. Gläscher, J., Hampton, A. N. & O'Doherty, J. P. Determining a role for ventromedial prefrontal cortex in encoding action-based value signals during reward-related decision making. *Cereb. Cortex* **19**, 483–495 (2009).

74. Miller, K. J., Botvinick, M. M. & Brody, C. D. Value representations in orbitofrontal cortex drive learning, not choice. *bioRxiv* https://doi.org/10.1101/245720 (2018).

75. Van Overwalle, F. A dissociation between social mentalizing and general reasoning. *NeuroImage* **54**, 1589–1599 (2011).

76. Delgado, M. R. et al. Viewpoints: dialogues on the functional role of the ventromedial prefrontal cortex. *Nat. Neurosci.* **19**, 1545–1552 (2016).

77. de Boer, L. et al. Dorsal striatal dopamine D1 receptor availability predicts an instrumental bias in action learning. *Proc. Natl Acad. Sci. USA* **116**, 261–270 (2019).

78. O'Doherty, J. et al. Dissociable roles of ventral and dorsal striatum in instrumental conditioning. *Science* **304**, 452–454 (2004).

79. Laland, K. N. Social learning strategies. *Anim. Learn. Behav.* **32**, 4–14 (2004).
This review provides an integrated analysis of social learning strategies from the perspective of behavioural ecology.

80. Debiec, J. & Sullivan, R. M. Intergenerational transmission of emotional trauma through amygdala-dependent mother-to-infant transfer of specific fear. *Proc. Natl. Acad. Sci. U.S.A.* **111**, 12222–12227 (2014).
Study demonstrating intergenerational transmission of specific threat information in rodents.

81. Askew, C. & Field, A. P. Vicarious learning and the development of fears in childhood. *Behav. Res. Ther.* **45**, 2616–2627 (2007).

82. Hopwood, T. L. & Schutte, N. S. Psychological outcomes in reaction to media exposure to disasters and large-scale violence: a meta-analysis. *Psychol. Violence* **7**, 316–327 (2017).

83. Enquist, M., Eriksson, K. & Ghirlanda, S. Critical social learning: a solution to Rogers's paradox of nonadaptive culture. *Am. Anthropol.* **109**, 727–734 (2007).

84. Rendell, L. et al. Why copy others? Insights from the social learning strategies tournament. *Science* **328**, 208–213 (2010).

85. Joiner, J., Piva, M., Turrin, C. & Chang, S. W. C. Social learning through prediction error in the brain. *NPJ Sci. Learn* **2**, 8 (2017).

86. Amodio, D. M. & Frith, C. D. Meeting of minds: the medial frontal cortex and social cognition. *Nat. Rev. Neurosci.* **7**, 268–277 (2006).

87. Meyza, K. Z., Bartal, I. B.-A., Monfils, M. H., Panksepp, J. B. & Knapska, E. The roots of empathy: through the lens of rodent models. *Neurosci. Biobehav. Rev.* **76**, 216–234 (2017).

88. Stanley, D. A. & Adolphs, R. Toward a neural basis for social behavior. *Neuron* **80**, 816–826 (2013).

89. Zaki, J. Empathy: a motivated account. *Psychol. Bull.* **140**, 1608–1647 (2014).

90. Callaghan, B. L. & Tottenham, N. The neuro-environmental loop of plasticity: a cross-species analysis of parental effects on emotion circuitry development following typical and adverse caregiving. *Neuropsychopharmacology* **41**, 163–176 (2016).

91. Panksepp, J. B. & Lahvis, G. P. Differential influence of social versus isolate housing on vicarious fear learning in adolescent mice. *Behav. Neurosci.* **130**, 206–211 (2016).

92. Yusufishaq, S. & Rosenkranz, J. A. Post-weaning social isolation impairs observational fear conditioning. *Behav. Brain Res.* **242**, 142–149 (2013).

93. Inagaki, H. et al. Identification of a pheromone that increases anxiety in rats. *Proc. Natl Acad. Sci. USA* **111**, 18751–18756 (2014).

94. Kim, E. J., Kim, E. S., Covey, E. & Kim, J. J. Social transmission of fear in rats: the role of 22-kHz ultrasonic distress vocalization. *PLoS ONE* **5**, e15077 (2010).

95. Knapska, E. et al. Between-subject transfer of emotional information evokes specific pattern of amygdala activation. *Proc. Natl. Acad. Sci. U.S.A.* **103**, 3858–3862 (2006).
The first rodent model of fear contagion, showing involvement of the amygdala.

96. Dezecache, G., Jacob, P. & Grèzes, J. Emotional contagion: its scope and limits. *Trends Cogn. Sci.* **19**, 297–299 (2015).

97. Keum, S. & Shin, H.-S. Rodent models for studying empathy. *Neurobiol. Learn. Mem.* **135**, 22–26 (2016).

98. Knapska, E., Mikosz, M., Werka, T. & Maren, S. Social modulation of learning in rats. *Learn. Mem.* **17**, 35–42 (2010).

99. Lamm, C., Decety, J. & Singer, T. Meta-analytic evidence for common and distinct neural networks associated with directly experienced pain and empathy for pain. *Neuroimage* **54**, 2492–2502 (2011).

100. de Waal, F. B. M. & Preston, S. D. Mammalian empathy: behavioural manifestations and neural basis. *Nat. Rev. Neurosci.* **18**, 489–509 (2017).

101. Masserman, J. H., Wechkin, S. & Terris, W. 'Altruistic' behavior in rhesus monkeys. *Am. J. Psychiatry* **121**, 584–585 (1964).

102. Brass, M. & Heyes, C. Imitation: is cognitive neuroscience solving the correspondence problem? *Trends Cogn. Sci.* **9**, 489–495 (2005).

103. Lanzetta, J. T. & Englis, B. G. Expectations of cooperation and competition and their effects on observers' vicarious emotional responses. *J. Pers. Soc. Psychol.* **56**, 543–554 (1989).

104. Golkar, A. & Olsson, A. The interplay of social group biases in social threat learning. *Sci. Rep.* **7**, 7685 (2017).

105. Hein, G., Engelmann, J. B., Vollberg, M. C. & Tobler, P. N. How learning shapes the empathic brain. *Proc. Natl Acad. Sci. USA* **113**, 80–85 (2016).

106. Lockwood, P. L., Apps, M. A. J., Valton, V., Viding, E. & Roiser, J. P. Neurocomputational mechanisms of prosocial learning and links to empathy. *Proc. Natl Acad. Sci. USA* **113**, 9763–9768 (2016).

107. Burke, C. J., Tobler, P. N., Baddeley, M. & Schultz, W. Neural mechanisms of observational learning. *Proc. Natl. Acad. Sci. U.S.A.* **107**, 14431–14436 (2010).
    This paper showed for the first time the neural correlates of observational learning during decision making, and pioneered the use of computational modelling in social learning research.

108. Suzuki, S. et al. Learning to simulate others' decisions. *Neuron* **74**, 1125–1137 (2012).

109. Kilner, J. M., Friston, K. J. & Frith, C. D. Predictive coding: an account of the mirror neuron system. *Cogn. Process.* **8**, 159–166 (2007).

110. Dunne, S., D'Souza, A. & O'Doherty, J. P. The involvement of model-based but not model-free learning signals during observational reward learning in the absence of choice. *J. Neurophysiol.* **115**, 3195–3203 (2016).

111. Denny, B. T., Kober, H., Wager, T. D. & Ochsner, K. N. A meta-analysis of functional neuroimaging studies of self- and other judgments reveals a spatial gradient for mentalizing in medial prefrontal cortex. *J. Cogn. Neurosci.* **24**, 1742–1752 (2012).

112. Carlin, J. D. & Calder, A. J. The neural basis of eye gaze processing. *Curr. Opin. Neurobiol.* **23**, 450–455 (2013).

113. Young, L., Camprodon, J. A., Hauser, M., Pascual-Leone, A. & Saxe, R. Disruption of the right temporoparietal junction with transcranial magnetic stimulation reduces the role of beliefs in moral judgments. *Proc. Natl Acad. Sci. USA* **107**, 6753–6758 (2010).

114. Hill, C. A. et al. A causal account of the brain network computations underlying strategic social behavior. *Nat. Neurosci.* **20**, 1142–1149 (2017).

115. Falk, E. B. et al. The neural correlates of persuasion: a common network across cultures and media. *J. Cogn. Neurosci.* **22**, 2447–2459 (2010).

116. Ong, D. C., Zaki, J. & Goodman, N. D. Computational models of emotion inference in theory of mind: a review and roadmap. *Top. Cogn. Sci.* **11**, 338–357 (2018).

117. Saxe, R. & Houlihan, S. D. Formalizing emotion concepts within a Bayesian model of theory of mind. *Curr. Opin. Psychol.* **17**, 15–21 (2017).

118. Jeon, D. et al. Observational fear learning involves affective pain system and Cav1.2 Ca²⁺ channels in ACC. *Nat. Neurosci.* **13**, 482–488 (2010).
    The first model of observational fear learning in rodents, shows the critical role of the ACC.

119. Atsak, P. et al. Experience modulates vicarious freezing in rats: a model for empathy. *PLoS ONE* **6**, e21855 (2011).

120. Pereira, A. G., Cruz, A., Lima, S. Q. & Moita, M. A. Silence resulting from the cessation of movement signals danger. *Curr. Biol.* **22**, R627–628 (2012).

121. Chen, Q., Panksepp, J. B. & Lahvis, G. P. Empathy is moderated by genetic background in mice. *PLoS ONE* **4**, e4387 (2009).

122. Bruchey, A. K., Jones, C. E. & Monfils, M.-H. Fear conditioning by-proxy: social transmission of fear during memory retrieval. *Behav. Brain Res.* **214**, 80–84 (2010).

123. Mineka, S., Davidson, M., Cook, M. & Keir, R. Observational conditioning of snake fear in rhesus monkeys. *J. Abnorm. Psychol.* **93**, 355–372 (1984).

124. Reynolds, G., Field, A. P. & Askew, C. Learning to fear a second-order stimulus following vicarious learning. *Cogn. Emot.* **31**, 572–579 (2017).

125. Hooker, C. I., Germine, L. T., Knight, R. T. & D'Esposito, M. Amygdala response to facial expressions reflects emotional learning. *J. Neurosci.* **26**, 8915–8922 (2006).

126. Lindström, B., Haaker, J. & Olsson, A. A common neural network differentially mediates direct and social fear learning. *NeuroImage* **167**, 121–129 (2018).
    This paper demonstrates that social and direct forms of Pavlovian threat learning are underpinned by a common neural network.

127. Olsson, A. & Phelps, E. A. Learned fear of "unseen" faces after Pavlovian, observational, and instructed fear. *Psychol. Sci.* **15**, 822–828 (2004).

128. Carrillo, M. et al. Emotional mirror neurons in the rat's anterior cingulate cortex. *Curr. Biol.* **29**, 1301–1312.e6 (2019).

129. Bissière, S. et al. The rostral anterior cingulate cortex modulates the efficiency of amygdala-dependent fear learning. *Biol. Psychiatry* **63**, 821–831 (2008).

130. Allsop, S. A. et al. Corticoamygdala transfer of socially derived information gates observational learning. *Cell* **173**, 1329–1342 (2018).
    This paper shows the role of a specific pathway (the ACC→BLA projection) in observational threat learning.

131. Twining, R. C., Vantrease, J. E., Love, S., Padival, M. & Rosenkranz, J. A. An intra-amygdala circuit specifically regulates social fear learning. *Nat. Neurosci.* **20**, 459–469 (2017).

132. Meffert, H., Brislin, S. J., White, S. F. & Blair, J. R. Prediction errors to emotional expressions: the roles of the amygdala in social referencing. *Soc. Cogn. Affect. Neurosci.* **10**, 537–544 (2015).

133. Olsson, A., Nearing, K. I. & Phelps, E. A. Learning fears by observing others: the neural systems of social fear transmission. *Soc. Cogn. Affect. Neurosci.* **2**, 3–11 (2007).

134. Terburg, D. et al. The basolateral amygdala is essential for rapid escape: a human and rodent study. *Cell* **175**, 723–735.e16 (2018).

135. Wang, S. et al. The human amygdala parametrically encodes the intensity of specific facial emotions and their categorical ambiguity. *Nat. Commun.* **8**, 14821 (2017).

136. Eippert, F., Bingel, U., Schoell, E., Yacubian, J. & Buchel, C. Blockade of endogenous opioid neuro-transmission enhances acquisition of conditioned fear in humans. *J. Neurosci.* **28**, 5465–5472 (2008).

137. Olsson, A. et al. Vicarious fear learning depends on empathic appraisals and trait empathy. *Psychol. Sci.* **27**, 25–33 (2016).

138. Golkar, A., Castro, V. & Olsson, A. Social learning of fear and safety is determined by the demonstrator's racial group. *Biol. Lett.* **11**, 20140817 (2015).

139. Golkar, A., Selbing, I., Flygare, O., Öhman, A. & Olsson, A. Other people as means to a safe end. *Psychol. Sci.* **24**, 2182–2190 (2013).

140. Chang, S. W. C., Winecoff, A. A. & Platt, M. L. Vicarious reinforcement in rhesus macaques (*Macaca mulatta*). *Front. Neurosci.* **5**, 27 (2011).

141. Kendal, R. et al. Chimpanzees copy dominant and knowledgeable individuals: implications for cultural diversity. *Evol. Hum. Behav.* **36**, 65–72 (2015).

142. Kavaliers, M., Colwell, D. D. & Choleris, E. Kinship, familiarity and social status modulate social learning about "micropredators" (biting flies) in deer mice. *Behav. Ecol. Sociobiol.* **58**, 60–71 (2005).

143. Goubert, L., Vlaeyen, J. W. S., Crombez, G. & Craig, K. D. Learning about pain from others: an observational learning account. *J. Pain.* **12**, 167–174 (2011).

144. Fiske, S. T., Harris, L. T., Lee, T. L. & Russell, A. M. in *Handbook of Prejudice, Stereotyping, and Discrimination* (ed. Nelson, T. D.) 525–534 (Taylor & Francis, 2009).

145. Berger, S. M. Conditioning through vicarious instigation. *Psychol. Rev.* **69**, 450–466 (1962).

146. Phelps, E. A. et al. Activation of the left amygdala to a cognitive representation of fear. *Nat. Neurosci.* **4**, 437–441 (2001).
    The first study examining the brain responses to verbally instructed threat, highlighting the involvement of the amygdala in support of its conserved function across modes of learning.

147. Schmitz, A. & Grillon, C. Assessing fear and anxiety in humans using the threat of predictable and unpredictable aversive events (the NPU-threat test). *Nat. Protoc.* **7**, 527–532 (2012).

148. Mechias, M.-L., Etkin, A. & Kalisch, R. A meta-analysis of instructed fear studies: implications for conscious appraisal of threat. *NeuroImage* **49**, 1760–1768 (2010).

149. Dayan, P. & Berridge, K. C. Model-based and model-free Pavlovian reward learning: revaluation, revision, and revelation. *Cogn. Affect. Behav. Neurosci.* **14**, 473–492 (2014).

150. Atlas, L. Y., Doll, B. B., Li, J., Daw, N. D. & Phelps, E. A. Instructed knowledge shapes feedback-driven aversive learning in striatum and orbitofrontal cortex, but not the amygdala. *eLife* **5**, e15192 (2016).

151. Atlas, L. Y. & Phelps, E. A. Prepared stimuli enhance aversive learning without weakening the impact of verbal instructions. *Learn. Mem.* **25**, 100–104 (2018).

152. Costa, V. D., Bradley, M. M. & Lang, P. J. From threat to safety: instructed reversal of defensive reactions. *Psychophysiology* **52**, 325–332 (2015).

153. Leadbeater, E. & Dawson, E. H. A social insect perspective on the evolution of social learning mechanisms. *Proc. Natl Acad. Sci. USA* **114**, 7838–7845 (2017).

154. Dawson, E. H., Avarguès-Weber, A., Chittka, L. & Leadbeater, E. Learning by observation emerges from simple associations in an insect model. *Curr. Biol.* **23**, 727–730 (2013).

155. Posadas-Andrews, A. & Roper, T. J. Social transmission of food-preferences in adult rats. *Anim. Behav.* **31**, 265–271 (1983).

156. Galef, B. G., Mason, J. R., Preti, G. & Bean, N. J. Carbon disulfide: a semiochemical mediating socially-induced diet choice in rats. *Physiol. Behav.* **42**, 119–124 (1988).

157. Munger, S. D. et al. An olfactory subsystem that detects carbon disulfide and mediates food-related social learning. *Curr. Biol.* **20**, 1438–1444 (2010).

158. Smith, C. A., East, B. S. & Colombo, P. J. The orbitofrontal cortex is not necessary for acquisition or remote recall of socially transmitted food preferences. *Behav. Brain Res.* **208**, 243–249 (2010).

159. Carballo-Márquez, A., Vale-Martínez, A., Guillazo-Blanch, G. & Martí-Nicolovius, M. Muscarinic transmission in the basolateral amygdala is necessary for the acquisition of socially transmitted food preferences in rats. *Neurobiol. Learn. Mem.* **91**, 98–101 (2009).

160. Ross, R. S., McGaughy, J. & Eichenbaum, H. Acetylcholine in the orbitofrontal cortex is necessary for the acquisition of a socially transmitted food preference. *Learn. Mem.* **12**, 302–306 (2005).

161. Wang, Y., Fontanini, A. & Katz, D. B. Temporary basolateral amygdala lesions disrupt acquisition of socially transmitted food preferences in rats. *Learn. Mem.* **13**, 794–800 (2006).

162. Kashtelyan, V., Lichtenberg, N. T., Chen, M. L., Cheer, J. F. & Roesch, M. R. Observation of reward delivery to a conspecific modulates dopamine release in ventral striatum. *Curr. Biol.* **24**, 2564–2568 (2014).

163. Paton, J. J., Belova, M. A., Morrison, S. E. & Salzman, C. D. The primate amygdala represents the positive and negative value of visual stimuli during learning. *Nature* **439**, 865–870 (2006).

164. Reynolds, S. M. & Berridge, K. C. Positive and negative motivation in nucleus accumbens shell: bivalent rostrocaudal gradients for GABA-elicited eating, taste 'liking'/'disliking' reactions, place preference/avoidance, and fear. *J. Neurosci.* **22**, 7308–7320 (2002).

165. Kim, J., Pignatelli, M., Xu, S., Itohara, S. & Tonegawa, S. Antagonistic negative and positive neurons of the basolateral amygdala. *Nat. Neurosci.* **19**, 1636–1646 (2016).

166. Morelli, S. A., Sacchet, M. D. & Zaki, J. Common and distinct neural correlates of personal and vicarious reward: a quantitative meta-analysis. *NeuroImage* **112**, 244–253 (2015).

167. Mobbs, D. et al. A key role for similarity in vicarious reward. *Science* **324**, 900 (2009).

168. Morelli, S. A., Knutson, B. & Zaki, J. Neural sensitivity to personal and vicarious reward differentially relate to prosociality and well-being. *Soc. Cogn. Affect. Neurosci.* **13**, 831–839 (2018).

169. Singer, T. et al. Empathic neural responses are modulated by the perceived fairness of others. *Nature* **439**, 466–469 (2006).

170. Lindström, B., Selbing, I. & Olsson, A. Co-evolution of social learning and evolutionary preparedness in dangerous environments. *PLoS ONE* **11**, e0160245 (2016).

REVIEWS

171. Bunch, G. B. & Zentall, T. R. Imitation of a passive avoidance response in the rat. *Bull. Psychon. Soc.* **15**, 73–75 (1980).
172. Del Russo, J. E. Observational learning of discriminative avoidance in hooded rats. *Anim. Learn. Behav.* **3**, 76–80 (1975).
173. Masuda, A. & Aou, S. Social transmission of avoidance behavior under situational change in learned and unlearned rats. *PLoS ONE* **4**, e6794 (2009).
174. Masuda, A. & Aou, S. Lesions of the medial prefrontal cortex enhance social modulation of avoidance. *Behav. Brain Res.* **217**, 309–314 (2011).
175. Lindström, B. & Olsson, A. Mechanisms of social avoidance learning can explain the emergence of adaptive and arbitrary behavioral traditions in humans. *J. Exp. Psychol. Gen.* **144**, 688–703 (2015).
176. Heyes, C. M. & Dawson, G. R. A demonstration of observational learning in rats using a bidirectional control. *Q. J. Exp. Psychol. Sect. B Comp. Physiol. Psychol.* **42**, 59–71 (1990).
177. Huang, I.-N., Koski, C. A. & DeQuardo, J. R. Observational learning of a bar-press by rats. *J. Gen. Psychol.* **108**, 103–111 (1983).
178. Zentall, T. R. & Levine, J. M. Observational learning and social facilitation in the rat. *Science* **178**, 1220–1221 (1972).
179. Bem, T., Jura, B., Bontempi, B. & Meyrand, P. Observational learning of a spatial discrimination task by rats: learning from the mistakes of others? *Anim. Behav.* **135**, 85–96 (2018).
180. Groesbeck, R. W. & Duerfeldt, P. H. Some relevant variables in observational learning of the rat. *Psychon. Sci.* **22**, 41–43 (1971).
181. Jurado-Parras, M. T., Gruart, A. & Delgado-García, J. M. Observational learning in mice can be prevented by medial prefrontal cortex stimulation and enhanced by nucleus accumbens stimulation. *Learn. Mem.* **19**, 99–106 (2012).
182. Cooper, J. C., Dunne, S., Furey, T. & O'Doherty, J. P. Human dorsal striatum encodes prediction errors during observational learning of instrumental actions. *J. Cogn. Neurosci.* **24**, 106–118 (2012).
183. Hill, M. R., Boorman, E. D. & Fried, I. Observational learning computations in neurons of the human anterior cingulate cortex. *Nat. Commun.* **7**, 12722 (2016).
184. Apps, M. A. J. & Sallet, J. Social learning in the medial prefrontal cortex. *Trends Cogn. Sci.* **21**, 151–152 (2017).
185. Chang, S. W. C., Gariépy, J.-F. & Platt, M. L. Neuronal reference frames for social decisions in primate frontal cortex. *Nat. Neurosci.* **16**, 243–250 (2013). **This study demonstrates the differentiated roles of sub-regions of the ACC in vicarious reward in primates.**
186. Apps, M. A. J. & Ramnani, N. The anterior cingulate gyrus signals the net value of others' rewards. *J. Neurosci.* **34**, 6190–6200 (2014).
187. Lockwood, P. L., Apps, M. A. J., Roiser, J. P. & Viding, E. Encoding of vicarious reward prediction in anterior cingulate cortex and relationship with trait empathy. *J. Neurosci.* **35**, 13720–13727 (2015). **This study demonstrates that the gyrus of the anterior cingulate cortex (ACCg) is more responsive to vicarious than self-experienced reward, and related to trait empathy.**
188. Lockwood, P. L. et al. Association of callous traits with reduced neural response to others' pain in children with conduct problems. *Curr. Biol.* **23**, 901–905 (2013).
189. Hesslow, G. The current status of the simulation theory of cognition. *Brain Res.* **1428**, 71–79 (2012).
190. Liljeholm, M., Molloy, C. J. & O'Doherty, J. P. Dissociable brain systems mediate vicarious learning of stimulus–response and action–outcome contingencies. *J. Neurosci.* **32**, 9878–9886 (2012).
191. Williamson, R. A., Meltzoff, A. N. & Markman, E. M. Prior experiences and perceived efficacy influence 3-year-olds' imitation. *Dev. Psychol.* **44**, 275–285 (2008).
192. Dorrance, B. R. & Zentall, T. R. Imitation of conditional discriminations in pigeons (*Columba livia*). *J. Comp. Psychol.* **116**, 277–285 (2002).

193. Saggerson, A. L., George, D. N. & Honey, R. C. Imitative learning of stimulus–response and response–outcome associations in pigeons. *J. Exp. Psychol. Anim. Behav. Process.* **31**, 289–300 (2005).
194. Duhan, D. F., Johnson, S. D., Wilcox, J. B. & Harrell, G. D. Influences on consumer use of word-of-mouth recommendation sources. *J. Acad. Mark. Sci.* **25**, 283–295 (1997).
195. Biele, G., Rieskamp, J., Krugel, L. K. & Heekeren, H. R. The neural basis of following advice. *PLoS Biol.* **9**, e1001089 (2011).
196. Doll, B. B., Jacobs, W. J., Sanfey, A. G. & Frank, M. J. Instructional control of reinforcement learning: a behavioral and neurocomputational investigation. *Brain Res.* **1299**, 74–94 (2009).
197. Li, J., Delgado, M. R. & Phelps, E. A. How instructed knowledge modulates the neural systems of reward learning. *Proc. Natl Acad. Sci. USA* **108**, 55–60 (2011).
198. Fazio, R. H., Eiser, J. R. & Shook, N. J. Attitude formation through exploration: valence asymmetries. *J. Pers. Soc. Psychol.* **87**, 293–311 (2004).
199. Staudinger, M. R. & Büchel, C. How initial confirmatory experience potentiates the detrimental influence of bad advice. *NeuroImage* **76**, 125–133 (2013).
200. Bikhchandani, S., Hirshleifer, D. & Welch, I. Learning from the behavior of others: conformity, fads, and informational cascades. *J. Econ. Perspect.* **12**, 151–170 (1998).
201. Wood, L. A., Kendal, R. L. & Flynn, E. G. Whom do children copy? Model-based biases in social learning. *Dev. Rev.* **33**, 341–356 (2013).
202. Selbing, I. & Olsson, A. Beliefs about others' abilities alter learning from observation. *Sci. Rep.* **7**, 16173 (2017).
203. Vostroknutov, A., Polonio, L. & Coricelli, G. The role of intelligence in social learning. *Sci. Rep.* **8**, 6896 (2018).
204. Collette, S., Pauli, W. M., Bossaerts, P. & O'Doherty, J. Neural computations underlying inverse reinforcement learning in the human brain. *eLife* **6**, e29718 (2017).
205. Ng, A. Y. & Russell, S. in *Proceedings of the Seventeenth International Conference on Machine Learning* (ed. Langley, P.) 663–670 (Morgan Kaufmann, 2000).
206. Heyes, C. Who knows? Metacognitive social learning strategies. *Trends Cogn. Sci.* **20**, 204–213 (2016).
207. Jara-Ettinger, J. Theory of mind as inverse reinforcement learning. *Curr. Opin. Behav. Sci.* **29**, 105–110 (2019).
208. Efferson, C., Lalive, R., Richerson, P., McElreath, R. & Lubell, M. Conformists and mavericks: the empirics of frequency-dependent cultural transmission. *Evol. Hum. Behav.* **29**, 56–64 (2008).
209. Morgan, T. J. H., Rendell, L. E., Ehn, M., Hoppitt, W. & Laland, K. N. The evolutionary basis of human social learning. *Proc. R. Soc. B Biol. Sci.* **279**, 653–662 (2012).
210. Rendell, L. et al. Cognitive culture: theoretical and empirical insights into social learning strategies. *Trends Cogn. Sci.* **15**, 68–76 (2011).
211. Kendal, R. L., Coolen, I. & Laland, K. N. The role of conformity in foraging when personal and social information conflict. *Behav. Ecol.* **15**, 269–277 (2004).
212. Pike, T. W. & Laland, K. N. Conformist learning in nine-spined sticklebacks' foraging decisions. *Biol. Lett.* **6**, 466–468 (2010).
213. van de Waal, E., Borgeaud, C. & Whiten, A. Potent social learning and conformity shape a wild primate's foraging decisions. *Science* **340**, 483–485 (2013). **This field study with vervet monkeys demonstrates social transmission of food preferences induced by aversive learning, which results in strong local food choice traditions.**
214. van Leeuwen, E. J. C. & Haun, D. B. M. Conformity in nonhuman primates: fad or fact? *Evol. Hum. Behav.* **34**, 1–7 (2013).
215. Huang, T.-R., Hazy, T. E., Herd, S. A. & O'Reilly, R. C. Assembling old tricks for new tasks: a neural model of instructional learning and control. *J. Cogn. Neurosci.* **25**, 843–851 (2013).
216. Wu, H., Luo, Y. & Feng, C. Neural signatures of social conformity: a coordinate-based activation likelihood estimation meta-analysis of functional brain imaging studies. *Neurosci. Biobehav. Rev.* **71**, 101–111 (2016).

217. Klucharev, V., Hytönen, K., Rijpkema, M., Smidts, A. & Fernández, G. Reinforcement learning signal predicts social conformity. *Neuron* **61**, 140–151 (2009).
218. Nook, E. C. & Zaki, J. Social norms shift behavioral and neural responses to foods. *J. Cogn. Neurosci.* **27**, 1412–1426 (2015).
219. Suzuki, S., Adachi, R., Dunne, S., Bossaerts, P. & O'Doherty, J. P. Neural mechanisms underlying human consensus decision-making. *Neuron* **86**, 591–602 (2015).
220. Parkinson, C. & Wheatley, T. The repurposed social brain. *Trends Cogn. Sci.* **19**, 133–141 (2015).
221. Heyes, C. What's social about social learning? *J. Comp. Psychol.* **126**, 193–202 (2012). **This review questions whether social learning involves any distinctly social processes, and argues that social learning is governed by domain-general learning processes.**
222. Heyes, C. Enquire within: cultural evolution and cognitive science. *Philos. Trans. R. Soc. B Biol. Sci.* **373**, 20170051 (2018).
223. Heyes, C. & Pearce, J. M. Not-so-social learning strategies. *Proc. R. Soc. B Biol. Sci.* **282**, 20141709 (2015).
224. Lind, J., Ghirlanda, S. & Enquist, M. Social learning through associative processes: a computational theory. *R. Soc. Open. Sci.* **6**, 181777 (2019). **In this paper, the authors provide a theoretical reinforcement learning model that can account for many types of social learning in animals.**
225. Gee, D. G. et al. Early developmental emergence of human amygdala–prefrontal connectivity after maternal deprivation. *Proc. Natl Acad. Sci. USA* **110**, 15638–15643 (2013).
226. Chang, D.-J. & Debiec, J. Neural correlates of the mother-to-infant social transmission of fear. *J. Neurosci. Res.* **94**, 526–534 (2016).
227. Sanders, J., Mayford, M. & Jeste, D. Empathic fear responses in mice are triggered by recognition of a shared experience. *PLoS ONE* **8**, e74609 (2013).
228. Öhman, A. & Mineka, S. Fears, phobias, and preparedness: toward an evolved module of fear and fear learning. *Psychol. Rev.* **108**, 483–522 (2001).
229. Cohen, J. D. et al. Computational approaches to fMRI analysis. *Nat. Neurosci.* **20**, 304–313 (2017).
230. O'Doherty, J. P., Hampton, A. & Kim, H. Model-based fMRI and its application to reward learning and decision making. *Ann. N. Y. Acad. Sci.* **1104**, 35–53 (2007).
231. Diaconescu, A. O. et al. Hierarchical prediction errors in midbrain and septum during social learning. *Soc. Cogn. Affect. Neurosci.* **12**, 618–634 (2017).
232. Mineka, S. & Cook, M. Mechanisms involved in the observational conditioning of fear. *J. Exp. Psychol. Gen.* **122**, 23–38 (1993).

Author contributions
All authors contributed equally to all aspects of the manuscript.

Competing interests
The authors declare no competing interest.

Peer review information
Nature Reviews Neuroscience thanks R. Kendal, R. Adolphs and K. Tye for their contribution to the peer review of this work.

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