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Evolutionary themes in the neurobiology of social cognition

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Remarkable examples of social cognition have been described across a diverse range of species, yet surprisingly little is known about the neurobiological underpinnings of these behaviors. Recent studies suggest that the molecular pathways and neural networks that mediate social behavior have been relatively conserved across vertebrate evolution, suggesting that shared mechanisms may drive adaptive behavioral responses to social stimuli. Here, we review recent advances in the neurobiology of flexible and context-dependent social behaviors across vertebrate taxa, focusing on female mate choice, pair-bonding, and aggressive behavior. Furthermore, we highlight the outstanding opportunities for uncovering the mechanisms mediating cooperative behavior, an exemplar of social cognition. We suggest a framework for investigating context-dependent neural organization and the evoked neural response to social stimuli.

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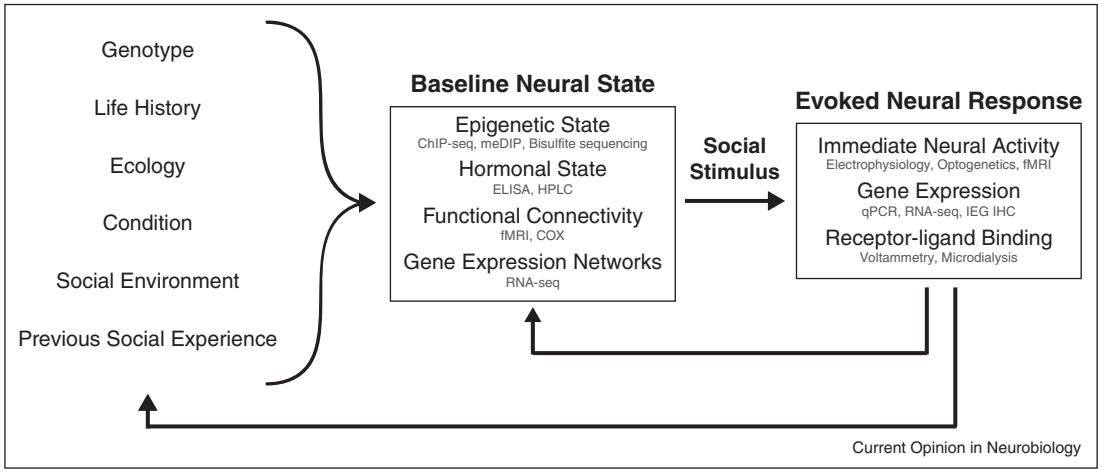
Introduction

Members of social groups integrate in real-time the behavior of their social partners with memory of past interactions and predictions of future behavior in order to respond in a context-appropriate manner. It is thus not surprising that social behavior (such as aggressive, sexual, and parental behavior) is influenced by a variety of factors, including previous experience and the current social environment [1]. The expression of such behavioral flexibility has important fitness consequences for an individual [2]. In fact, evidence for sophisticated social cognitive abilities has been accumulating across diverse taxa and behavioral contexts. Complex social cognitive abilities are thus no longer considered to be limited to human and non-human primates. Rather, we are beginning to appreciate that most social animals have evolved cognitive mechanisms for assessing, evaluating, and responding flexibly to a wide variety of often subtle yet vital social cues [3–5]. For example, in many species females prefer males that are in the presence of other females, suggesting that such mate choice copying relies on the assessment of a male's quality by other females [6]. An audience can strongly affect an individual's response to social information more generally (e.g., [7]). For example, subordinate males of the cichlid fish *Astatotilapia burtoni* increase aggressive displays when the dominant community members are not paying attention [8]. Even more remarkably, these fish can use known relationships to deduce unknown ones to infer the social rank of other individuals transitively (e.g., using $A > B$ and $B > C$ to infer $A > C$; [9]). While great strides have been made in identifying a diversity of behaviors involving social cognition, little remains known about the neuromolecular basis of these behaviors. Given the recent advances in high-throughput approaches and our increased understanding of neural circuit evolution, there are now unprecedented opportunities to identify the neural and molecular mechanisms mediating social decision-making and cognition [10]).

Spontaneous activity and recurrent connections between brain areas give rise to coordinated global network states, which affect neural processing as stimuli are incorporated into existing neural representations [11]. These patterns represent a baseline neural state of activity, which can be shifted at the level of single neurons as well as larger neural units by social experience and learning, as well as by genotype and life history factors [12]. Changes to this baseline can be encoded by modifications to the epigenome, hormone levels, functional connectivity between brain regions, as well as to gene expression networks. Plasticity in the perception of and response to a social stimulus is largely dependent on the structure of this baseline state. The stimulus-evoked neural response, as measured in terms of immediate neural activity, gene expression, and receptor–ligand binding, can then differ even in response to identical social stimuli. In Figure 1, we offer a framework integrating these concepts.

Here, we review recent research on three forms of social interactions that best highlight the remarkable advances that have been made in the neurobiology of social cognition, particularly in non-traditional, non-primate model

Figure 1



Context-dependent neural response to social stimuli. An individual's baseline neural state is shaped by a variety of factors throughout the lifetime, including genotype, life history, ecology, condition, social environment, and previous social experiences. The baseline neural state may be temporally stable, but is continuously updated with social information. The evoked neural response to a social stimulus is dependent on this baseline neurogenomic state of the brain. Divergent neural responses to identical social stimuli can occur depending on the baseline state. Measuring both the baseline neural state (prior to stimulus) and the evoked neural response (post-stimulus) offer insights into the neural mechanisms mediating flexible and adaptive behavioral responses to social information. Common techniques for measuring each level of analysis are provided (ChIP-seq, chromatin immunoprecipitation sequencing; meDIP, methylated DNA immunoprecipitation; ELISA, enzyme-linked immunosorbent assay; HPLC, high-performance liquid chromatography; fMRI, functional magnetic resonance imaging; COX, cytochrome oxidase staining; RNA-seq, whole transcriptome shotgun sequencing; qPCR, quantitative polymerase chain reaction; IEG IHC, immediate-early gene immunohistochemistry).

systems (the neurobiology of primate social cognition has recently been reviewed elsewhere [13,14]). Mate preference behavior, pair-bonding, and aggressive behavior are each composed of a variety of social cognitive core elements, such as individual and/or social recognition, partner preference and/or avoidance, and advanced learning and memory. Importantly, each behavior has the potential to be highly plastic and can be shaped by social context and experience. In addition to the advances made in studying these behaviors, we also outline, in Box 1, the outstanding opportunities for gaining functional insight into the mechanisms underlying cooperative behavior. We consider strategic cooperative behavior, and alternatively cheating or deception, to epitomize the elements of social cognition. Cooperation between members of a social group and even between heterospecific individuals has evolved repeatedly in numerous lineages of vertebrates and invertebrates [15]. Even though the factors that favor the evolution of cooperative behavior and its fitness benefits are well understood, the underlying neuromolecular mechanisms are largely unknown.

Conserved neural pathways

Although specific behavioral outputs vary widely among species, the biological functions and metabolic needs that drive these behaviors are deeply shared [10]. Also, the principles of brain development and organization are highly conserved across vertebrates [16,17]. Moreover, all systems subserving social behavior are keenly sensitive and responsive to social inputs, which are perceived and transduced by one or more sensory pathways. These neural signals, in turn, are processed and integrated in specific regions of the brain through various neuromodulatory systems such as steroid hormones, neuropeptides, and monoamines [10,18,19], which ultimately lead to adaptive behavior. Insights from mammals suggest two neural circuits of crucial importance in this context: the Social Behavior Network (SBN) [20,21] and the mesolimbic Reward System [22], which together form a larger Social Decision-Making (SDM) Network [23••,24••]. The nodes of this circuitry interact to integrate environmental and physiological cues and encode stimulus salience and valence to generate adaptive behavioral responses.

The Social Behavior Network and the Reward System were first described in mammals. Thus, in order to apply this framework to non-mammalian model systems we need to resolve homology for the relevant brain regions across a wide range of taxa (for the SBN, see [21]). O'Connell and Hofmann [23••] recently inferred homology relationships across taxa for the entire SDM Network. Although some of these homologies remain tentative [25], this work provided for the first time a comprehensive comparative synthesis of this circuitry, suggesting that it was already present in early vertebrates [23••]. In fact, the SDM Network is remarkably conserved across vertebrates not only in terms of neuroanatomy but also with regards to candidate gene expression patterns. In an additional study, O'Connell and Hofmann [24••] analyzed expression profiles for 10 neurochemical

Box 1 Toward a neurobiology of cooperative behavior.

Cooperative behavior involves some of the most remarkable expressions of social cognition. A strong theoretical literature posits how and why cooperation has evolved, explaining, for example, why vampire bats regurgitate food to feed hungry roost-mates, or why marmots risk survival to warn neighbors about predators [15]. Nevertheless, despite innovative behavioral assays and mathematical theory predicting when cooperation should occur and to what extent cognition must be involved, mechanistic studies that identify the link between cooperation, social cognition, and its neural basis are severely lacking [46,47]. While some forms of cooperation appear as emergent properties [46], there are distinct and explicit cognitive components of cooperative behavior that are easily amenable to mechanistic studies.

Two recent studies have begun to examine the hormonal mechanisms mediating cooperative behavior in the cleaner–client mutualism in fishes. Using the Indo-Pacific bluestreak cleaner wrasse, *Labroides dimidiatus*, Soares *et al.* [48] showed that an arginine vasotocin (AVT; homologue of mammalian AVP) receptor agonist decreased cleaning behavior, while the antagonist increased the number of cleaning interactions and cheating toward clients. Manipulating the isotocin (homologue of mammalian OT) pathway had no measurable effect. An additional study found a reduced size and number of AVT gigantocellular neurons in the preoptic area of an obligate cleaner species relative to a non-cleaner species, providing further support for the potential role of AVT in cooperative cleaning behavior [49]. Additionally, recent peripheral pharmacological manipulations in cooperatively breeding meerkats, *Suricata suricatta*, provide support for the role of oxytocin (though not glucocorticoids [50]) in modulating a suite of cooperative behaviors [51].

These studies are clearly important, though the rich individual and species variation in cooperative behavior has yet to be examined in detail, and well-established model systems for behavioral studies of cooperation offer great opportunity to identify neural substrates. Conversely, there is also a great, unexplored potential for neurobiological studies to illuminate aspects of cooperative behavior. For example, while the evolution of cooperation through reciprocity remains contentious, reciprocal interactions between partners may be more common than is currently acknowledged, possibly because recognizing such behaviors is difficult [52]. Identifying neural correlates of decision-making during potentially reciprocal interactions can shed light on both the existence and the evolution of these behaviors. Future studies can also contribute to addressing the fascinating question of the degree to which the neural and molecular pathways mediating cooperation are conserved across both taxa and social contexts.

genes across the 12 SDM Network nodes in 88 vertebrate species and found that gene expression patterns are highly conserved in this network over 450 million years of evolution, suggesting that the diversity of social behavior in vertebrates can be explained, at least in part, by variations on a theme of conserved neural and gene expression networks. Thus, social stimuli may trigger shared common molecular pathways and neural networks that drive adaptive behavioral responses, even if the species-specific motor programs they orchestrate differ greatly and have evolved independently.

Mate preference

Female mate preference is critical not only for maximizing fitness, but can also shape the evolution of male characters and can serve as a mechanism for species divergence [26,27]. In most mating systems, females must perceive, integrate, and evaluate signals from multiple males before choosing whether or not to mate with a given male. Furthermore, learning and plasticity can play important roles in mate choice [27]. Recent studies have shown teleost fishes, in particular, to be a powerful system in which to identify the neural mechanisms underlying female choice [28]. Wong *et al.* [29] examined expression of *egr-1* and *neuroserpin*, genes previously implicated in mate preference behavior [30], across SDM Network nodes in female *Xiphophorus nigrensis*, the Northern swordtail. Using a dichotomous choice paradigm, they identified relationships between the degree of preference for the larger of two males and gene expression in regions associated with reward, sensory processing, and sexual behavior, providing further evidence that female mate preference involves complex, coordinated neural activity. A related study that included additional social conditions in the dichotomous paradigm examined the relationship between whole brain gene expression and preference behavior in *X. nigrensis*. The mate choice environment influenced an assemblage of genes associated with preference (e.g., *neuroserpin*, *neuroligin-3*) whereas variation in affiliative behaviors was associated with genes that mediate social bonding (e.g. *isotocin* and *vasotocin*) [31]. Interestingly, while *neuroserpin* and *neuroligin-3* expression was positively associated with female preference behavior in *X. nigrensis*, expression levels of the same genes tended to be negatively associated with preference in a species in which males exhibit coercive mating tactics, *Gambusia affinis*, the Western mosquitofish [32].

Familiarity with males affects female choice in a number of species [27]. Okuyama *et al.* [33••] examined this phenomenon in medaka fish and found that visual familiarity with males enhances female preference. They identified mutant strains in which females did not exhibit preference behavior and found that preference was inhibited by abnormal development of terminal-nerve (TN)-gonadotropin releasing hormone 3 (GnRH3) neurons, which function to suppress female receptivity. Using additional mutant lines, ablation, and single neuron electrophysiology, the authors demonstrated that GnRH3 peptide released from TN neurons is necessary for the switch from suppressed receptivity to preference behavior. They also showed that GnRH3 peptides facilitate the pacemaker frequencies in TN-GnRH neurons, which may be involved in mediating preference for familiar males. This study represents one of the most compelling advances toward a complete understanding of the neural mechanisms underlying various forms of social cognition. There is ample opportunity in other model systems of mate preference for studies of similar depth and precision (reviewed in birds: [34]).

Pair-bonding

The neurobiology of pair-bonding has been studied most extensively in the monogamous prairie vole, *Microtus ochrogaster* [35]. The dynamics of the pair-bond can be surprisingly plastic, and are affected, for example, by the early-life social environment and can also differ between populations and mating strategies [36]. Pair-bond formation involves a variety of neurotransmitter pathways, most notably oxytocin (OT), vasopressin (AVP), and dopamine (DA). In a recent study, Wang *et al.* [37••] demonstrated epigenetic regulation of partner preference formation in female *M. ochrogaster*. Treatment with histone deacetylase inhibitors facilitated partner preference formation in the absence of mating by up-regulating OT and AVP receptors (OTR, V1aR) in the nucleus accumbens. Additionally, females exhibiting natural mating-induced preference had higher acetylation at the promoters of the OT and V1a receptors in the nucleus accumbens, likely leading to the observed increase in mRNA and protein levels of the receptors.

Pair-bonding has evolved independently numerous times in both vertebrates and invertebrates [4]. As such, there is a great opportunity to examine the extent to which the neurobiological mechanisms regulating pair-bond formation and maintenance are conserved across species. Pair-bond formation in the monogamous zebra finch, *Taeniopygia guttata*, was inhibited by intracerebroventricular administration of an OTR antagonist in females, with males following a similar trend [38]. Interestingly, a previous study found that systemic injections of OTR antagonist also reduced courtship behavior [39], suggesting the presence of sub-systems underlying different functions. Dopamine (DA) was also recently implicated in pair-bonding in zebra finches. Banerjee *et al.* [40•] report higher levels of DA and its metabolite in a portion of the brain encompassing the nucleus accumbens in newly pair-bonded zebra finches. Similarly, using c-Fos immunohistochemistry, they show that the proportion of active dopaminergic neurons is higher in the ventral tegmental area of pair-bonded birds. These results in zebra finches are similar to the mechanisms identified in prairie voles and are important first steps toward identifying conserved neural circuits.

Aggressive behavior

Aggressive behavior is often modulated by a variety of factors, including physical state, social status, previous fighting experience, resource quality, and the type of audience present, among others [5]. The neural pathways through which each of these factors influence the expression of aggressive behavior is an active area of research. An impressive array of signaling molecules have been implicated in aggression [41]. For example, a recent study by Coura *et al.* (2013) examined how the cholinergic, dopaminergic, and norepinephrine systems interact in regulating aggression and flexible social cognition [42]. Specifically, depletion of norepinephrine in the prefrontal cortex (PFC) of mice reduced behavioral flexibility and increased aggression in a social interaction task, an effect that was absent in mice lacking a specific subunit (B2) of the nicotinic acetylcholine receptor [42]. In the PFC, basal levels of monoamines and acetylcholine were also higher in the mutant strain [42].

Measures of circulating levels of hormones are not always consistent predictors of levels of aggression. Using free-living male and female dark-eyed juncos, *Junco hyemalis*, Rosvall *et al.* [43] examined variation in levels of circulating testosterone and gene expression of androgen receptor, estrogen receptor alpha, and aromatase in response to a simulated territorial intrusion. They found that gene expression levels in behavior-relevant brain regions relate to individual measures of aggressive behavior in both males and females, whereas testosterone levels related to aggression only in males. These results provide support for the hypothesis that sensitivity to sex steroids is an important mechanism by which selection may act to influence aggression, and likely plays a similar role in other forms of social cognition. Further support for the specific role of aromatase in the regulation of aggressive behavior comes from a recent study in the African cichlid fish, *Astatotilapia burtoni*. Socially subordinate males had higher levels of aromatase expression than dominant males in the preoptic area, a neuroendocrine relay station in the vertebrate brain [44]. Interestingly, pharmacologically blocking aromatase in dominant males decreased aggressive behavior and circulating estradiol, but increased circulating testosterone levels [44].

Future directions

As more high-throughput technologies and sophisticated modeling approaches become available, we expect the distinction between model and non-model systems to dissolve. As such, species can begin to be selected for neurobiological studies based on their unique social cognitive abilities and studied in interacting individuals within both free-living and captive populations. Bobroy *et al.* [45••] provides a compelling example, applying novel technologies to social cognition by demonstrating the neural correlates of social facial touch in interacting rats, using extracellular recordings in the barrel cortex. Applying similar techniques to investigate flexibility in social behavior can greatly advance our understanding of the mechanisms regulating social cognition.

We outlined a framework for studying the neurobiology of social cognition, in Figure 1, with hopes that future research will continue to dissociate the contributions of each of these mechanistic levels to the factors mediating social cognition. Within this framework, a thorough understanding of the neurobiology of social cognition will require an integrative approach that tests (1) how animals behave in response to subtle social cues; (2) how

assessment of and response to social stimuli affect the activity of the underlying neural networks; (3) how the neuromolecular states of these networks regulate motor and hormonal outputs; and (4) how candidate neurochemical pathways mediate genomic, neural, and behavioral responses.

Conflict of interest statement

Nothing declared.

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