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Cognitive neuroscience of social emotions and implications for psychopathology: Examining embarrassment, guilt, envy, and schadenfreude

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Social emotions are affective states elicited during social interactions and integral for promoting socially appropriate behaviors and discouraging socially inappropriate ones. Social emotion-processing deficits significantly impair interpersonal relationships, and play distinct roles in the manifestation and maintenance of clinical symptomatology. Elucidating the neural correlates of discrete social emotions can serve as a window to better understanding and treating neuropsychiatric disorders. Moral cognition and social emotion-processing broadly recruit a fronto–temporo–subcortical network, supporting empathy, perspective-taking, self-processing, and reward-processing. The present review specifically examines the neural correlates of embarrassment, guilt, envy, and schadenfreude. Embarrassment and guilt are self-conscious emotions, evoked during negative evaluation following norm violations and supported by a fronto–temporo–posterior network. Embarrassment is evoked by social transgressions and recruits greater anterior temporal regions, representing conceptual social knowledge. Guilt is evoked by moral transgressions and recruits greater prefrontal regions, representing perspective-taking and behavioral change demands. Envy and schadenfreude are fortune-of-other emotions, evoked during social comparison and supported by a prefronto–striatal network. Envy represents displeasure in others' fortunes, and recruits increased dorsal anterior cingulate cortex, representing cognitive dissonance, and decreased reward-related striatal regions. Schadenfreude represents pleasure in others' misfortunes, and recruits reduced empathy-related insular regions and increased reward-related striatal regions. Implications for psychopathology and treatment design are discussed.

Key words: embarrassment, envy, functional magnetic resonance imaging, guilt, schadenfreude.

SO FAR, ABOUT morals, I know only that what is moral is what you feel good after and what is immoral is what you feel bad after.

Ernest Hemingway, *Death in the Afternoon*

INTRODUCTION TO SOCIAL EMOTIONS

Social emotions are the driving force for maintaining interpersonal relationships, integral for motivating

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socially appropriate behaviors and seeking reparations for inappropriate ones. A large body of research has explored the neural correlates of social emotions, broadly examining moral cognition, empathy, and social decision-making, as well as investigating distinct emotions, such as guilt and envy. This literature not only informs the neural bases of prosocial and antisocial behaviors, such as altruistic giving, reciprocal cooperation, and social withdrawal, but also sheds light onto atypical cognitive processes underlying neuropsychiatric disorders, ranging from depression and anxiety to autism and frontotemporal dementia. This research is not only capable of elucidating the antecedents of clinical symptomatology, but also offers insight into future treatment avenues. Although a complete review of the neural bases of social emotions is beyond the scope of this paper, we summarize recent neuroimaging research – specifically examining embarrassment, guilt, envy, and schadenfreude – and discuss clinical implications.

Moral emotions

Social emotions are broadly defined as context-dependent affective states, evoked during social interactions.[1] These include moral and self-conscious emotions (SCE), associated with obedience or transgression of societal norms, and fortune-of-other emotions (FOE), evoked via social comparison. Moral emotions are related to the interests and welfare of specific individuals or society at large,[2] and are integral for promoting socially acceptable behaviors and inhibiting socially unacceptable actions.[3,4] This class of emotions represents the recognition and adoption of universally accepted rules and culturally defined conventions.[5] Moll *et al*. characterized moral emotions as the product of 'contextual social knowledge', integrating event knowledge, social semantic knowledge, and emotional/motivational states.[6]

Self-conscious emotions

SCE are a subset of moral emotions and the product of personal reflection and inferred evaluation. SCE are evoked by direct experience or in anticipation of others' evaluations. SCE are primarily supported by three cognitive processes: (i) self-awareness, underlying self-referential processing; (ii) other-awareness, underlying mental state attribution; and (iii) social norm-awareness, underlying the identification and adoption of societal standards.[7,8] Unlike basic emotions (e.g., happiness, sadness), which emerge within the first 9 months, SCE are more cognitively demanding and emerge after 36 months, coinciding with the acquisition and internalization of social rules, expectations, and values.[9] Similar to moral emotions, SCE promote social goals, such as social regulation.[7,10,11] Tangney *et al*. suggested that SCE serve as an 'emotional moral barometer,' providing feedback on the acceptability of one's behavior.[5] Consequently, these predicted or inferred evaluations guide moral behavior and social decision-making. Prototypical SCE include embarrassment, guilt, shame, and pride.

Fortune-of-other emotions

FOE are the product of social comparison and represent affective responses to others' attributes, possessions, or outcomes.[12–14] Similar to SCE, which arise from a discrepancy between one's ideal self and current self, FOE represent a discrepancy between one's current status and the status of another person. FOE are divided into four categories: 'happy for', 'sorry for', 'resentment', and 'gloating', and are modulated by four factors: (i) personal desirability of an outcome; (ii) inferred desirability of an outcome for a target; (iii) inferred target deservedness; and (iv) target likeability.[12]

A CLOSER LOOK AT EMBARRASSMENT, GUILT, ENVY, AND SCHADENFREUDE

Embarrassment

Embarrassment and guilt are SCE, representing inferred negative self-evaluations in response to moral or social norm violations. Embarrassment is generally evoked by less severe, but more personal, social transgressions.[5,15,16] Embarrassment has a strong public focus and commonly :represents 'normative public deficiencies,' such as acting clumsy or committing a faux pas. Embarrassment is typically short-lived and more likely to be found amusing and discussed with others.[5,15] Embarrassment is characterized by heightened physiological responding (e.g., blushing, increased heart rate) and universally recognized appeasement gestures, such as downward head tilt and eye gaze.[17–20] Accordingly, embarrassment serves self-regulatory functions, signaling the need to monitor, hide, or change one's behavior,[5,11] and promoting prosocial or reparative actions.[21,22]

Guilt

Guilt and shame are similar SCE, typically elicited by severe, moral violations, and are more painful and less fleeting than embarrassment. Guilt and shame are evoked by comparable situations that do not differ in severity, morality, or personal responsibility; however, they represent distinct attributions and promote different behaviors.[23] Guilt represents internal attributions of unstable, discrete behaviors, while shame represents internal attributions of stable, global self-evaluations.[24] Guilt is evoked by specific transgressions and motivates prosociality, other-oriented empathy, and reparation, while shame is evoked by perceived self-deficiencies and promotes self-defense, denial, and avoidance.[11,15] A person may feel guilt after offending a friend (action-focused), but feel shame when perceiving oneself as disloyal (global self-focused). While guilt encourages accountability and atonement, intended to restore relationships, shame motivates escape and withdrawal, intended to prevent further self-denigration. Thus, both embarrassment and guilt (but not shame) are psychologically adaptive emotions that serve social regulatory functions, including inhibiting transgressions and encouraging reparation.[11]

Envy

Envy is a 'resentment' FOE, representing displeasure evoked by another person's fortune, or negative affect resulting from upward social comparison.[12] A person may feel envy when a colleague receives a sought-after promotion or a coveted role in a play. Dispositional envy is associated with feelings of inferiority,[25] while episodic envy is modulated by domain self-relevance, perceived target likeability, and perceived target deservedness, such that greater envy is evoked during social comparison of self-relevant traits, particularly when a comparison target is disliked or perceived as undeserving.[12,26–28]

Schadenfreude

Schadenfreude is a 'gloating' FOE, representing pleasure evoked by another person's misfortune, or positive affect resulting from downward social comparison.[12] A person may feel schadenfreude when a rival athlete loses a competition or a bully experiences pain. A related concept, 'counterempathy', or anti-empathy, represents incongruent affective responding to others' fortunes and misfortunes, and strongly resembles envy and schadenfreude. Schadenfreude is modulated by target likeability and target deservedness.[28–31] Schadenfreude is strongly related to envy: episodic envy is a significant predictor of episodic schadenfreude and mediates the relationship between dispositional envy and episodic schadenfreude.[28,32]

WHAT CAN WE LEARN FROM SOCIAL COGNITIVE NEUROSCIENCE?

Clinical studies

Two broad bodies of literature, clinical behavioral studies and neuroimaging studies, have investigated the neural bases of social emotions. Clinical studies allow researchers to examine naturally occurring social cognitive impairments, resulting from atypical neural development or acquired neural damage. Studying clinical populations can help elucidate the neural mechanisms underlying distinct social cognitive processes and can offer causal explanations for the manifestation of specific deficits.

A primary limitation of clinical research is a reduced level of control and specificity, more readily afforded by experimental manipulations. Clinical populations demonstrate a wide range of social and cognitive deficits, making it difficult to determine, from behavioral studies alone, how neural impairments influence distinct abilities. In addition, clinical populations often reveal impairments extending across multiple neural regions or complex neural networks, making it difficult to ascertain which regions are distinctly responsible for diverse deficits.

Neuroimaging studies

Neuroimaging research complements and extends clinical studies by investigating the neural correlates of similar, but distinct, processes using fine-grain manipulations. Neuroimaging can shed light onto the unique functions of neural regions and the integrative roles of large-scale neural networks, while avoiding the confounds of comorbidity and extensive neural damage. Neuroimaging can also elucidate the separable cognitive and affective components underlying varying processes. For example, neuroimaging can help clarify if attenuated empathy is due to poor perspective-taking, emotion recognition, or interoception, by revealing activation patterns of dis-

tinct brain regions. This knowledge, in turn, can offer important insights into intervention design. Neuroimaging can further provide information typically inaccessible via behavioral methods. Self-report may yield inaccurate responses, due to poor self-awareness (alexithymia) or biased responding, particularly when measuring socially undesirable traits and events. Participants may be reluctant to share embarrassing or guilt-evoking experiences or admit feelings of envy or schadenfreude. Some studies even suggest that the objective measurement of neural activity may better predict social behavior than subjective self-report.[33] Neuroimaging may also shed light onto modulators of social cognitive phenomena, such as reward-processing or in-group/out-group membership, which may lie outside conscious awareness.

ASSOCIATED COGNITIVE PROCESSES AND THEIR NEURAL CORRELATES

Moral cognition

The neural substrates of moral cognition have been studied in great detail.[6,34] Broadly, research converges on a fronto–temporo–subcortical network, including medial, lateral, and dorsolateral prefrontal cortex (mPFC, lPFC, dlPFC); anterior cingulate cortex (ACC); insula; anterior temporal lobe (aTL); posterior superior temporal sulcus (pSTS); precuneus (PC); amygdala; hypothalamus; and striatum.[6] This network represents context-dependent structured event knowledge, context-independent social perceptual and functional features, and motivational and affective states, respectively represented by prefrontal regions, anterior/posterior temporal regions, and a cortico–limbic network.[6,35,36]

Empathy

An important process underlying both moral cognition and social emotion-processing is empathy. Decety described empathy as a functional bridge between first- and third-person information, enabling individuals to resonate with other people's affective states while distinguishing between self and other.[37] Empathy is supported by affect-sharing, perspective-taking, self-processing, and emotion regulation.[37–39] Recent meta-analyses highlight the primary roles of anterior insula and adjacent fronto-operculum (AI/FO; extending into inferior frontal gyrus [IFG]) and ACC (particularly dorsal and middle regions [dACC/aMCC]).[40,41] AI supports interoceptive awareness,[42] uncertainty monitoring,[1] and salience-processing,[43] while ACC supports motivation-processing[42] and the integration of interoception and salience.[44] Some researchers advocate that AI and dACC/aMCC work together to represent and predict affective states, compute prediction errors, and guide social decision-making,[38] which support their roles in emotional inference and affective responding.

Self-processing

Self-processing plays a significant role in moral cognition and social emotion-processing. Self-awareness and self-reflection are necessary for representing personal agency and for monitoring, evaluating, and regulating social behavior. Self–other decoupling is critical for distinguishing between one's own cognitive and affective states and those of another person, in order to empathize with others while avoiding personal distress. Furthermore, self–other decoupling is necessary for social comparison, and self-relevance modulates feelings of envy and schadenfreude.[26] Self-processing primarily recruits cortical midline structures, including mPFC (dorsal, anterior rostral, and ventral regions [dmPFC, armPFC, vmPFC], and ACC) and medial posterior parietal cortex (mPPC; PC, posterior cingulate cortex [PCC], and retrosplenial cortex [RSC]).[45–48]

Theory of mind

Perspective-taking, or theory of mind (ToM) is another important component. During SCE-processing, a person infers how his/her social behaviors are evaluated by others, and during FOE-processing, a person attributes others' affective reactions to their fortunes/misfortunes. ToM is supported by a distinct fronto–temporo–parietal network, consisting of mPFC, pSTS, temporo–parietal junction (TPJ), temporal poles (TP), and PC/PCC.[48,49] Broadly, social emotion-processing recruits superior frontal gyrus (SFG), TP, and PC/PCC, underscoring the importance of mental state attribution.[48] Research suggests that TP represents social emotional, third-person perspective-taking;[48] right TPJ tracks intention attribution during moral cognition;[50] and mPFC, PCC, and inferior parietal lobe (IPL) support emotional salience-processing during moral decision-making.[51]

Reward-processing

Reward-processing is highly relevant to envy and schadenfreude. Both primary and secondary reward-processing recruits mesolimbic regions, in particular dorsal striatum (DS; caudate, putamen) and ventral striatum (VS, including nucleus accumbens [NAcc]). In addition, a prefronto–striatal network is often recruited when processing losses and gains.[52–55] Social preferences and rewarding social behaviors, such as cooperation, punishment, and charitable giving, are also affiliated with striatal activity.[56,57] This suggests that envy may represent diminished reward-processing and reduced striatal recruitment, while schadenfreude may represent heightened reward-processing and increased striatal recruitment.

While the general neural circuitry underlying social emotions and associated cognitive processes has been examined extensively, the investigation of *distinct* social emotions is currently in its nascence. Moll *et al*. suggested that social emotions are supported by a partially dissociable prefronto–temporo–limbic system.[6] Below, we review recent neuroimaging research exploring embarrassment, guilt, envy, and schadenfreude, and discuss important clinical implications.

NEURAL BASIS OF EMBARRASSMENT

Elucidating the neural correlates of embarrassment is highly relevant towards better understanding clinical symptomatology. A host of psychological disorders are characterized by socially inappropriate behaviors, which can negatively affect interpersonal relationships and impair adaptive functioning. In particular, patients with mPFC damage (e.g., vmPFC lesions/orbital frontal cortex [OFC] lesions, frontal temporal lobe degeneration [FTLD]) or mPFC dysfunction (e.g., autism spectrum disorders [ASD], schizophrenia) demonstrate a diminished understanding of embarrassment, commonly attributed to impaired ToM.[58–61]

Faux pas tasks shed light onto atypical processes underlying impaired embarrassment-processing within clinical populations. Patients with OFC lesions, relative to lateral frontal, dorsal frontal, or non-frontal lesions, or typically developing controls, demonstrate poor faux pas recognition, particularly when making inferences about transgressors' intentions, yet intact first-order ToM, second-order ToM, and empathic understanding.[60,62] These results demonstrate that OFC damage may not impair cognitive perspective-taking or empathy, per say, but the *integration* of mental state attribution with empathic understanding and emotion-processing. The authors posited that similar social cognitive deficits observed in mPFC damage and ASD may represent common vmPFC/OFC impairment.[62,63]

Beer *et al*. were particularly interested in the impact of mPFC damage on social regulation and SCE recognition.[64,65] OFC patients, lPFC patients, and typically developing controls completed tasks assessing self-disclosure, teasing, over-praise, and emotion recognition. OFC patients provided appropriate definitions of basic and self-conscious emotions, yet revealed impaired recognition of SCE facial expressions and displayed inappropriately intimate self-disclosures and teasing. OFC patients failed to demonstrate socially appropriate embarrassment and reparative actions following social transgressions, yet displayed socially inappropriate embarrassment when it was unwarranted. In general, OFC patients underestimated the inappropriateness of their own behaviors, but demonstrated heightened embarrassment following increased self-monitoring. These findings suggest that OFC patients have intact conceptual social knowledge but fail to apply this information to infer others' emotions or guide social decisions. Consequently, OFC patients display socially inappropriate behaviors, including atypical embarrassment, which may represent altered self- and other-awareness.

FTLD is similarly associated with atypical embarrassment-processing. In response to an acoustic startle response task, FTLD patients demonstrated intact physiological and basic emotional responding, but impaired SCE responding (diminished embarrassment facial expressions).[66] Prefrontal, temporal, and subcortical gray matter volume was negatively correlated with physiological and emotional responding to embarrassing situations, and right perigenual ACC volume was a significant predictor of physiological and emotional embarrassment responding.[67] These findings highlight the importance of PFC/vmPFC in self-monitoring and emotional perspective-taking, which are necessary for processing embarrassment. The authors suggested that socially inappropriate behaviors observed in mPFC damage, including diminished embarrassment expression, reduced embarrassment recognition, increased social norm violations, and decreased

reparative actions, may represent impaired self-awareness and poor affective ToM.

Atypical embarrassment is also observed in ASD, neurodevelopmental disorders characterized by social and communicative deficits, restricted interests, and repetitive behaviors.[68] People with ASD demonstrate impaired social cognition, including atypical self-processing, complex cognitive and affective ToM, and empathy,[69–72] as well as altered underlying neural circuitry, including mPFC, ACC, AI, IFG, TPJ/pSTS, PC/PCC, and amygdala.[59,73–78] Performance on moral reasoning tasks demonstrates the impact of impaired affective perspective-taking on social decision-making. When evaluating moral behavior, people with ASD often give more weight to outcomes than intentions, rating accidental and attempted harms as equally unacceptable,[79] and rating agents of accidental harms as more responsible and more deserving of punishment than typically developing controls.[80] Buon *et al*. suggested that atypical moral reasoning in ASD may represent misattribution of intentions and elevated severity of moral evaluation.[80]

Similar to mPFC patients, people with ASD demonstrate impaired performance on faux pas tasks, including poor differentiation between sad and embarrassing situations, less frequent and explicit reference to an audience, impoverished justifications, and atypical severity ratings.[63,81–84] When asked to identify the content of transgressions, people with ASD more frequently referenced rule violations or poor manners, but rarely referenced another person's welfare.[79] Furthermore, atypical severity ratings of norm violations were associated with impaired cognitive ToM (but not empathy).[83,84] These findings offer additional evidence that people with ASD overemphasize external outcomes and deemphasize mental states (intentions) during moral reasoning and embarrassment-processing. Thus, ASD may be associated with intact conceptual social knowledge and false belief-processing, but a diminished ability to integrate social rules and mental states in everyday social interactions, particularly when mental state information conflicts with outcome information.[79,85] Overall, this research suggests that ASD are associated with atypical embarrassment-processing, due to impaired mental state representation and evaluation, possibly indicating altered ToM neural circuitry.

Findings from neuroimaging research converge with those from clinical studies, underscoring the importance of prefrontal and temporal regions in mental state attribution. Berthoz *et al*. investigated the neural correlates of unintentional (embarrassing) and intentional social norm violations in typically developing adults.[86,87] Unintentional transgressions recruited bilateral prefrontal (right medial and superior PFC, left middle and inferior PFC, left OFC), bilateral temporal (bilateral anterior and middle TP, left TPJ), and occipital regions. While unintentional and intentional violations similarly recruited bilateral prefrontal (left medial, middle, and inferior PFC, left OFC, bilateral SFG), bilateral temporal (bilateral anterior and middle TP, left TPJ), and occipital regions, unintentional violations recruited greater left TP. Thus, processing social norm violations (regardless of intentionality) requires ToM (mPFC, temporal regions) and negative emotion-processing (lPFC, mPFC), which likely aids in inferring transgressors' intentions, observers' evaluations, and observers' emotional reactions. Furthermore, greater TP recruitment during unintentional, relative to intentional, violations likely represents enhanced emotional perspective-taking and affective empathy.[48,88,89]

Takahashi *et al*. similarly highlighted the roles of mPFC and temporal regions in embarrassment-processing.[90] Participants read sentences representing first-person embarrassment (e.g., 'I noticed that the zipper on my pants was open'), guilt (e.g., 'I shoplifted a dress from the store'), and neutral emotion, and rated emotional intensity. Embarrassment recruited bilateral prefrontal (bilateral mPFC, left OFC), bilateral temporal (left pSTS, bilateral ATL, left middle temporal, left hippocampal), and occipital regions, and left pSTS and right occipital activity was positively correlated with embarrassment intensity. These neural patterns underscore the importance of perspective-taking (mPFC), social perception (pSTS), memory (hippocampus), and attention/salience (occipital cortex) in embarrassment-processing. They also converge with reports of greater frontal (MFG, ACC), insula/IFG, and superior temporal/inferior parietal (STS/TPJ, IPL/TPJ) recruitment during embarrassment-processing, relative to neural agency.[91] Furthermore, compared to guilt, embarrassment recruited greater right ATL, bilateral hippocampus, and bilateral occipital regions. The authors posited that greater temporal and occipital recruitment during embarrassment represented greater complexity (requiring greater discernment of social conventions).

A German replication study reported similar results.[92] Embarrassment recruited bilateral frontal

(SFG, IFG), bilateral temporal (left superior temporal gyrus [STG], bilateral parahippocampus), and occipital regions. Embarrassment and guilt commonly recruited prefrontal, temporal, and occipital regions, but embarrassment, relative to guilt, recruited greater right frontal (MFG, ACC) and bilateral temporal (parahippocampus) regions.

Overall, these studies suggest that embarrassment recruits a network of frontal (mPFC/OFC), temporal (pSTS, anterior and middle temporal cortex) and occipital regions, representing self-processing, mental state attribution, social semantic-processing, and emotional salience. Relative to guilt and intentional norm violations, embarrassment recruits greater ATL, representing greater conceptual social knowledge and richness of social detail.[35,36] This highlights the importance of identifying and understanding culturally dependent social norms during embarrassment-processing and distinguishing between socially appropriate and inappropriate behaviors.

NEURAL BASIS OF GUILT

Guilt-processing plays a significant role in the development and maintenance of psychopathology, including depression, anxiety, obsessive–compulsive disorder (OCD), and somatization.[93] In particular, impaired moral cognition and guilt are characteristic of antisocial personality disorder (APD) and psychopathy, likely representing intact ToM but impaired affective-processing.[94] Gray matter reductions within prefrontal (mPFC, medial/lateral OFC), AI, and temporal (STS, ATL) regions are associated with conduct disorder,[95] APD,[96] interpersonal/affective psychopathic traits,[97] and callous and unemotional traits.[95] Psychopathy is associated with a range of social cognitive and emotional deficits, representing underlying cortico–subcortical dysfunction. Violent psychopaths demonstrate decreased metabolism within prefrontal, parietal, and limbic regions,[98] and prisoners high in psychopathy show reduced AI and ACC activity during empathy-processing.[99]

A related set of studies has investigated the impact of adult-onset vmPFC/OFC damage on 'acquired psychopathy'.[100–102] A famous case study of Patient JS demonstrated that OFC damage is associated with intact ToM and social norm knowledge, but impaired emotion recognition, affective responding, emotional reaction prediction, and moral judgment.[101] A larger sample of patients with adult-onset vmPFC damage revealed typical responding to *impersonal* moral dilemmas but enhanced utilitarian responding to highly *emotional, personal* moral dilemmas.[103] Blair *et al*. posited that diminished guilt, reduced empathy, atypical emotional responding and antisocial behavior may represent underlying OFC–amygdala dysfunction, which impairs the prediction of negative emotional reactions.[101,104] These studies shed light onto the possible neural mechanisms of guilt-processing by highlighting the importance of prefrontal regions, particularly OFC, in the generation and application of emotional expectations, which guide social decision-making.

A large body of neuroimaging research has explored guilt-processing within typical development. One of the earliest studies used positron emission tomography to examine guilt recollection.[105] Participants listened to audio-recordings of guilt-evoking or neutral autobiographical events and used script-driven imagery to recall them. Guilt recruited empathy regions highly connected with amygdala,[106] including left AI/IFG, ACC, and bilateral ATL. Despite using stimuli that evoked multiple negative emotions, this study offers preliminary evidence for anterior paralimbic recruitment during guilt-processing, possibly representing negative emotional responding.

A related autobiographical memory study extended these findings by comparing the neural correlates of multiple negative emotions.[107] Guilt, shame, and sadness commonly recruited regions supporting ToM, empathy, self- and other-processing, and emotional memory retrieval, including bilateral medial frontal pole, left AI, bilateral RSC (extending into PC/PCC), bilateral TP, left TPJ, left aSTS, bilateral lingual gyrus, and cerebellum. Guilt, relative to shame, recruited greater right dlPFC/anterior SFG and right amygdala (also recruited during other-processing). Guilt, relative to shame and sadness, recruited greater right lateral OFC (lOFC) and left paracingulate/dmPFC (also recruited during self-processing), and lOFC activity was positively correlated with dispositional guilt. These findings support the role of self-reference and ToM in guilt-processing, and suggest that perspective-taking demands are greater for guilt-processing than shame or sadness. Furthermore, the authors posited that lOFC activity represented inhibition of social norm violations or anticipation of negative outcomes, and proposed a dissociation within PFC, such that vmPFC represented general social emotion-processing, while OFC

uniquely represented guilt-processing. Thus, lOFC activity may not simply represent negative valence or SCE, but instead, serve as a distinct marker for transient guilt-processing and guilt sensitivity.

Multiple studies have highlighted the role of prefrontal, temporal, and occipital regions during imagined guilt-processing. Takahashi *et al*. reported bilateral mPFC, left pSTS, and bilateral occipital recruitment during imagined guilt-evoking situations, and greater left mPFC recruitment, relative to embarrassment.[90] A German replication reported similar frontal, temporal, and limbic recruitment, including bilateral PFC, left OFC, left precentral gyrus, left insula, bilateral temporal (middle temporal gyrus [MTG], STG), and bilateral occipital regions.[92] Guilt recruited greater right amygdala/insula, left MTG, and right fusiform gyrus, relative to embarrassment, possibly underscoring the greater role of negative emotion-processing.[108,109] Berthoz *et al*. similarly reported bilateral PFC, left TPJ, bilateral ATL, and occipital recruitment during intentional violations (guilt). Intentional, relative to unintentional, violations recruited greater left PFC (mPFC, superior PFC, ACC), precentral/postcentral gyri, right TP, left IPL, PC, and occipital regions,[86] which the authors suggested represented greater mentalizing demands required to infer the motivations, and thus liability, of intentional transgressors. Personal, intentional violations recruited greatest bilateral amygdala.[87]

Recent neuroimaging studies have begun to investigate cognitive and motivational modulators of guilt-processing, including agency, social consequence, behavioral change demands, and guilt-sensitivity/aversion. Moll *et al*. investigated the neural correlates of emotionally neutral agency and moral emotions.[91] Neutral agency recruited prefrontal, insular, and anterior/superior temporal regions, associated with ToM, empathy, self-reference, and conceptual social knowledge, and guilt recruited greater right anterior PFC (MFG, lOFC), left insula, right midbrain (ventral tegmental area [VTA], thalamus), and left anterior/superior temporal regions (STS/TPJ). Conjunction analyses revealed unique neural patterns for distinct emotion categories: prosocial emotions (guilt, embarrassment, compassion) recruited anterior mPFC and STS/TPJ; empathic emotions (guilt, compassion) recruited VTA and VS; and other-critical emotions (self-indignation, other-indignation, disgust) recruited dACC, lOFC, and parahippocampus/amygdala. Relative to other-critical emotions, prosocial emotions recruited greater medial OFC (mOFC) and TP, and empathic emotions recruited greater anterior PFC, VTA, and VS. These findings emphasize the importance of anterior PFC, midbrain, and temporal regions in guilt-processing, supporting prosociality and empathy.

Morey *et al*. examined the impact of social consequences in guilt-processing.[110] Imagining harming others, compared to harming oneself, was associated with greater right mPFC (vmPFC, dorsomedial PFC [dmPFC] frontal pole [FP]), bilateral pSTS, bilateral PC/PCC, and right occipital recruitment. Guilt-sensitivity was positively correlated with left PFC (dmPFC, SFG, OFC, ventrolateral PFC [vlPFC], paracingulate gyrus; subgenual ACC/septal area [SCSR] at a reduced threshold) and supramarginal gyrus activity. Furthermore, an interaction between guilt intensity and social consequence within left vmPFC/FP, OFC, IFG, and anterior IPL suggested that anterior frontal and parietal activity tracks guilt-sensitivity associated with harming others.

A strong motivator for behavior modification and reparation is fear of negative evaluation. Finger *et al*. examined how the presence of an audience impacted guilt-processing.[111] Moral violations, which elicited highest guilt ratings, recruited greater left dmPFC and bilateral temporal regions (TP, middle temporal gyrus [MTG]) than social violations. Regardless of an audience presence, moral and social violations recruited left TPJ, underscoring shared perspective-taking demands; however, moral and *witnessed* social violations commonly recruited left vlPFC and dmPFC, which the authors interpreted as representing behavioral change demands.

A final set of studies has examined guilt-proneness and associated neural modulators. Using a donation task, Chang *et al*. found that guilt-aversion and guilt-sensitivity were associated with dlPFC, as well as dACC, insula, supplementary motor area (SMA), and TPJ activity, which was positively correlated with counterfactual guilt.[112] In a study by Zahn *et al*., participants read sentences evoking guilt, indignation, pride, and gratitude.[113] Guilt-proneness was positively correlated with bilateral prefrontal (left anterior vmPFC, left subgenual ACC [sgACC], bilateral ventral ACC) activity. A subsequent psychophysiological interaction (PPI) analysis revealed functional connectivity between right superior ATL (sATL) and bilateral sgACC during guilt-processing, which was positively correlated with guilt-proneness.[114] Other research suggested that sgACC activity during guilt-processing was positively correlated with indi-

vidual differences in empathic concern.[115] These neural patterns highlight functional integration between right sATL, representing conceptual social knowledge, and context-dependent fronto–limbic regions, representing distinct moral emotions. In particular, sgACC activity may serve as a distinct neural marker for guilt-proneness, supported by dispositional empathy.

These findings have important implications for understanding and treating major depressive disorder (MDD). Patients with MDD demonstrate excessive or inappropriate guilt associated with heightened self-blame.[116] Research investigating guilt-proneness in MDD revealed intact ATL and SCSR recruitment, but reduced functional coupling between right sATL and left SCSR, as well as between bilateral medial frontal pole, right hippocampus, and right lateral hypothalamus.[117] Furthermore, sATL–SCSR decoupling was positively correlated with self-hate ratings. The authors suggested that this decoupling represented reduced integration of conceptual social knowledge,[35] specific memories, and guilt-processing,[114] resulting in overgeneralized self-blame, and thus, elevated guilt-proneness. Preliminary research suggests that SCSR deep brain stimulation may effectively treat abnormal guilt-processing in MDD.[118]

Neuroimaging research investigating guilt also sheds light onto OCD etiology. Clinical studies suggest that OCD is associated with atypical guilt-processing, driven by inner moral rule violations.[119,120] Research by Basile *et al*. found that OCD patients recruited a typical prefronto–temporo–occipital network when processing anger and sadness, but reduced ACC, bilateral AI and left PC recruitment during guilt-processing, which was negatively correlated with OCD symptoms.[121] These findings demonstrate intact basic negative emotion-processing in OCD, but impaired (possibly overly efficient[122]) guilt-processing, elicited by personal moral transgressions.

These studies suggest that guilt-processing recruits a network of anterior prefrontal (vmPFC, mOFC/lOFC, sgACC), fronto–opercular (AI/IFG), temporal (ATL, TP, STS, TPJ), medial posterior parietal (PC, RSC), limbic (amygdala), and occipital (including fusiform gyrus) regions supporting perspective-taking, empathy, self-processing, other-processing, and negative emotion-processing. In particular, lOFC–amygdala and sATL–SCSR coupling may support guilt-proneness. Relative to other negative emotions, such as embarrassment, shame, and sadness, guilt recruits greater PFC (mPFC, dmPFC, dlPFC, OFC, ACC), amygdala, and ToM regions (TP, pSTS, IPL, PC). Furthermore, neuroimaging findings help dissociate the cognitive and emotional modulators of guilt-processing: social agency recruits a prefronto–insular–superior temporal network, associated with moral cognition; empathic motivation recruits VTA and VS, associated with social attachment; negative emotional prediction and responding recruits lOFC and parahippocampus/amygdala; prosocial motivation recruits anterior mPFC and STS; and behavioral change demands/compensatory behavior recruits vlPFC and dmPFC.

NEURAL BASIS OF ENVY AND SCHADENFREUDE

A large body of research has examined the neural correlates of social comparison and its relation to prosocial and antisocial behaviors. Typically, these studies have either adopted a neuroeconomics approach, where participants made monetary allocation decisions or competed against a confederate for monetary rewards, or a social psychological approach, where participants made costly helping decisions or compared themselves with others along highly self-relevant domains. In general, these studies underscore the importance of a prefronto–striatal network, particularly VS, for processing envy and schadenfreude. Critical work by Fleissbach *et al*. investigated the neural correlates of relative reward-processing.[123] Participants competed against a partner in a dot estimation task and received monetary rewards for correct answers. A main effect of inequality was found within OFC and posterior regions (angular gyrus, PC/PCC, occipital), such that larger reward discrepancies between the partners were associated with greater activity. A main effect of relative reward was found within bilateral VS, such that relatively larger rewards recruited greater VS activity, equal rewards recruited intermediate activity, and relatively smaller rewards recruited reduced activity, independent of absolute rewards. This VS response was positively correlated with willingness to reciprocate, such that greater concern for relative outcomes was associated with a greater tendency to reciprocate prosocial and antisocial behaviors. Related electroencephalography (EEG) studies revealed similar neural patterns; relative outcomes recruited prefrontal (mPFC/ACC),

striatal, and parahippocampal activity, possibly supporting perspective-taking, monitoring, and reward-processing, and unequal gains were rated more positively and recruited greater mPFC and caudate activity than equal gains.[124,125]

Research exploring inequality aversion extended these findings.[126] Pairs of participants played a monetary allocation task after economic inequality was manipulated (one participant received a large monetary endowment ['rich'], the other did not ['poor']). 'Rich' participants rated transfers to others (advantageous inequality aversion) positively and recruited increased frontal and striatal activity. 'Poor' participants rated transfers to others negatively and recruited reduced frontal and striatal activity, even though these transfers did not influence their own financial status (envy); however, they rated transfers to themselves (disadvantageous inequality aversion) positively and recruited increased activity. These findings offer further support that inequality-averse social preferences modulate vmPFC and VS recruitment during relative losses and gains.

Research comparing the neural correlates of absolute and relative losses and gains offer additional evidence for a prefronto–striatal network underlying envy and schadenfreude. Using a game of chance, Dvash *et al*. found that relative losses evoked greater envy but recruited similar VS activity as absolute losses, while relative gains evoked greater schadenfreude but recruited similar VS activity as absolute gains.[127] Thus, relative losses and gains were processed similarly to absolute losses and gains on the neural level. Research exploring private (absolute) and public (relative) lottery outcomes revealed similar neural patterns.[128] Results revealed a main effect of outcome valence within OFC and DS; a main effect of outcome context within PFC (mPFC, dlPFC), VS, and TPJ; and an interaction effect between outcome valence and context within DS. Participants recruited greater caudate during relative gains compared to absolute gains, and reduced caudate and NAcc during relative losses compared to absolute losses. These results highlight the impact of social context on the subjective value, affective experience, and neural basis of reward-processing.

Research examining the modulatory role of learned preferences in empathy and counterempathy offer additional support for an underlying fronto–striatal network. In a seminal study by Singer *et al*., participants observed confederates play fairly or unfairly during a neuroeconomic game and receive painful stimulation. Both male and female participants recruited empathy-related AI/FO and ACC activity when they observed fair players in pain, which was positively correlated with self-reported empathy.[129] However, male participants recruited reduced empathy-related AI/FO activity and increased reward-related VS activity when they observed unfair players in pain, which was respectively correlated with diminished empathy and an enhanced desire for revenge.

Similar neural patterns were found in studies where participants observed out-group members in pain. In a study capitalizing on a real-word sports rivalry, Yankees and Red Socks fans watched clips of a computer-generated baseball game.[33] During absolute and relative losses (favorite team lost or rival team won), participants recruited right sensorimotor (insula, ACC, SMA) regions. During absolute and relative gains (favorite team won or rival team lost), participants recruited left frontal (MFG, SFG), bilateral striatal (right VS, bilateral DS), and bilateral sensorimotor (left insula, right SMA) regions, and this VS response was positively correlated with self-reported pleasure and likelihood of harming a rival fan. This suggests that absolute and relative gains are similarly rewarding on a subjective and neural level, and VS activity associated with schadenfreude predicts antisocial behavior. In a related study, local soccer players recruited reduced empathy-related left AI and increased reward-related right NAcc activity when they observed rival players in pain.[130] Furthermore, negative impressions of rivals predicted greater AI reduction and NAcc recruitment, and both negative impressions and NAcc recruitment predicted costly helping behavior (volunteering to experience pain on behalf of another player).

Research has also examined neural modulators of social cooperation and competition. When observing racial out-group members in pain, participants demonstrated diminished empathic responding, including reduced ACC, SMA, and insula recruitment.[131] Research also suggests that stereotypes of warmth and competence influence empathy/counterempathy, beyond the effect of out-group membership.[132,133] When envied targets experienced negative events, participants felt greatest pleasure, yet when envied targets experienced positive events, participants felt greatest displeasure, which was associated with increased right insula/MFG and superior parietal recruitment. Furthermore, bilateral insula activity during positive events was positively correlated with

willingness to harm envied targets, but negatively correlated with willingness to harm pride targets. Overall, these findings suggest that a fronto–sensorimotor–striatal network supports empathy and counterempathy for pain and predicts prosocial and antisocial behaviors.

Several studies have investigated envy and schadenfreude via social comparison of self-relevant traits. Takahashi *et al*. reported fronto–striatal recruitment, including mOFC, dACC, VS, and DS, when participants compared themselves to superior targets.[134] dACC activity was positively correlated with envy, striatal activity was positively correlated with schadenfreude, and envy-related dACC activity predicted schadenfreude-related VS activity. The authors suggested that dACC activity represented distress resulting from self-concept threat (similar to cognitive dissonance[135]) or social pain resulting from salient upward social comparison (similar to social exclusion[136]), which motivates cognitive dissonance reduction via attitudinal change or schadenfreude. The authors interpreted the results to suggest that greater pain during self-concept threat (greater envy-related dACC activity during upward social comparison) was associated with greater reward during cognitive dissonance reduction (greater schadenfreude-related VS activity during others' misfortunes).

Friederich *et al*. investigated a highly salient domain of social comparison, body shape, in female adults.[137] Participants recruited bilateral frontal (left ACC, right dlPFC/precentral gyrus), right inferior parietal, and bilateral occipitotemporal regions when comparing their own bodies to images of slim, idealized figures, converging with ACC recruitment reported during envy-processing. Anxiety during body shape comparison was positively correlated with body concerns, disordered eating, and ventral limbic recruitment, including dACC, vlPFC, basal ganglia, amygdala, and occipital regions. The authors suggested that these neural patterns supported fear-processing, motivation, and emotion-processing, and represented body dissatisfaction and risk of eating disorders.

A follow-up study explored social comparison in anorexia as a means of understanding body dissatisfaction.[138] During body shape comparison, patients with anorexia recruited right lPFC, right PC, left superior parietal, and bilateral occipitotemporal regions. Relative to controls, patients reported greater body dissatisfaction and recruited reduced left rostral ACC and increased right sensorimotor (AI, including putamen and premotor) activity, which was negatively correlated with anorexia symptoms. These results suggest that body dissatisfaction in anorexia does not represent atypical envy-processing, as demonstrated by intact ventral limbic activity, but instead, likely represents altered self-processing/interoception and motivation/emotion-processing, as demonstrated by atypical sensorimotor activity. Interventions designed to improve body satisfaction in anorexia should address distorted self-perceptions and unhealthy motivations.

A final set of studies has investigated atypical envy- and schadenfreude-processing associated with prefrontal dysfunction. Shamay-Tsoory *et al*. examined FOE recognition and ToM abilities in lesion patients using an eye gaze task.[139] Patients with both vmPFC and dlPFC lesions demonstrated impaired cognitive and affective ToM; patients with vmpFC lesions or both vmPFC and dlPFC lesions demonstrated impaired envy recognition; and patients with vmPFC lesions, both vmPFC and dlPFC lesions, or inferior parietal/temporo-parietal lesions demonstrated impaired schadenfreude recognition. Furthermore, envy and schadenfreude recognition were associated with real-world perspective-taking. These findings underscore the importance of complex mental state attribution in FOE-processing. In particular, vmPFC is important for processing envy, while both vmPFC and inferior parietal/temporo-parietal regions are important for processing schadenfreude. A follow-up study examined FOE-processing in adults with ASD.[140] Participants with ASD demonstrated intact 1st- and 2nd-order ToM, but impaired real-world perspective-taking and diminished envy and schadenfreude recognition. These findings lend further support for the role of complex mental state attribution in FOE, and suggest that impaired envy- and schadenfreude-processing in ASD may represent underlying ToM deficits.

Overall, these studies offer evidence for a prefronto–striatal network underlying envy and schadenfreude and highlight a neural basis for the modulatory roles of target superiority and trait self-relevance. In particular, research underscores the roles of vmPFC and inferior parietal/temporo-parietal regions in perspective-taking and self-relevance; striatal regions (especially VS) in reward-processing; and a ventral limbic network in fear-processing, emotion-processing, and motivation. Research further highlights the influence of

social context on reward-processing. Similar to absolute losses, relative losses (envy) are associated with increased dACC activity, representing cognitive dissonance, and decreased striatal activity, representing diminished subjective value and reward. Similar to absolute gains, relative gains (schadenfreude) are associated with reduced insular activity, representing diminished empathy, and increased striatal activity, representing enhanced subjective value and reward. Finally, neural recruitment during social comparison significantly predicts prosocial (costly helping) and antisocial (physically harming) behaviors.

FUTURE DIRECTIONS

The current literature provides a strong foundation for elucidating the neural patterns of distinct social emotions, as well as for shedding light onto neural dysfunction underlying psychopathology. The neurobiology of social emotions is a bourgeoning field with several lines of research ripe for investigation. In the following sections, we discuss ongoing questions, suggest topics for future research, and describe potential treatment avenues.

Ecologically valid paradigms

This review has summarized popular paradigms for investigating social emotions, while highlighting prime areas for further development. Since this research is still in its nascence, previous studies have generally implemented strictly controlled designs with straightforward manipulations. Future studies should adopt more ecologically valid paradigms, using stimuli that more closely resemble variables of interest.

Research investigating SCE has commonly used vignettes, encouraging participants to imagine emotion-eliciting situations or rate the severity of hypothetical transgressions. While these tasks use prototypical examples of social emotions, they often lack personal relevance and emotional salience, characteristics that drive the experience of these emotions in the real world. Future studies should use more realistic, interactive stimuli, such as dynamic videos, or elicit emotions online. Research would benefit from adopting a combined approach of evoking emotions in real-time, tracking subjective ratings of emotional intensity, and measuring self-reported emotional tendencies and real-world behaviors.

Research examining FOE has typically adopted neuroeconomics paradigms, tracking donation behavior or measuring responses to monetary losses and gains. These tasks afford the development of computational models that further the understanding of social cooperation and competition. However, these tasks serve as abstract proxies for FOE, and may not accurately reflect real-world decision-making. Future research should elicit social comparisons online, along highly self-relevant domains, relative to salient, familiar social targets. A popular paradigm for studying schadenfreude has assessed empathic/counterempathic reactions when participants observe rivals in physical pain. Future studies should extend this research to examine empathic/counterempathic responses to more realistic, non-physical misfortunes.

Developmental and clinical samples

Previous research has primarily examined typically developing adults, yet few studies have explored developmental trends. Adolescence is a stage of significant growth, characterized by enhanced self-awareness, perspective-taking, and social comparison, as well as neural maturation of the 'social brain'.[141–144] Adolescence is an ideal developmental period for investigating changes in social emotion-processing. In addition, preliminary research suggests that SCE-processing may continue to develop into late adulthood.[145] Future studies should investigate the neurodevelopmental trajectories of social emotion-processing from pre-pubescence, through adolescence, and into adulthood.

Research should also more extensively explore differences in social emotion-processing within psychopathology. Embarrassment has largely been studied within the context of prefrontal damage, yet minimally within neuropsychiatric disorders, and envy and schadenfreude have rarely been investigated outside of typical development. Future studies should examine a wider range of clinical populations, particularly patients characterized by diminished perspective-taking abilities, atypical self-awareness, and altered reward-processing. Additionally, findings from typical development should be applied towards clarifying the etiology of clinical symptoms, as demonstrated by Friederich *et al.*,[137,138] as well as towards exploring compensatory mechanisms adopted in response to social cognitive impairments.

Avenues for treatment/intervention

Investigating the neural correlates of social emotions has significant implications for designing clinical interventions. First, researchers can determine the impact of specific cognitive deficits on social emotion-processing and design interventions to increase these abilities. For example, Beer *et al*.[64] demonstrated that atypical embarrassment in patients with PFC damage might reflect diminished self-awareness, and increased self-monitoring via video observation may improve embarrassment recognition. Future cognitive interventions may aim to increase self-monitoring, perspective-taking, or interoception.

Second, neuroendocrine interventions may enhance social emotion-processing and reduce associated clinical impairments. In particular, a subcortical neuropeptide called oxytocin significantly influences broad social behavior, including social bonding,[146,147] empathy,[148,149] and social salience,[150–152] as well as FOE.[153,154] Oxytocin administration modulates cooperation and competition among in-group and out-group members, empathic responding to others' pain, and associated neural recruitment, including prefrontal, inferior parietal/temporo-parietal, and striatal activity.[153] Of particular relevance, oxytocin administration increases envy and schadenfreude when participants process relative losses and gains.[154] These findings suggest that oxytocin may effectively increase the saliency of social emotion-processing. Future research should examine the impact of oxytocin on a range of social emotions.

Research has also highlighted the role of oxytocin in the manifestation of clinical symptoms. A popular line of research has explored the impact of oxytocin administration on reducing ASD symptoms, revealing decreased repetitive behaviors,[155] enhanced emotion recognition,[156] and increased social salience.[157] Future research should examine the impact of oxytocin on social emotion-processing across multiple clinical disorders, such as schizophrenia or MDD.

A third line of research has examined the efficacy of real-time functional magnetic resonance imaging (fMRI) to measure and modulate brain activity.[158] EEG neurofeedback and transcranial magnetic stimulation have reduced spatial resolution, which limits these methods to only measuring cortical activity; however, real-time fMRI affords the measurement of subcortical activity, such as striatal and limbic regions, which is integral for investigating social emotion-processing. During real-time fMRI, participants complete tasks in the MRI scanner while neural activity from regions of interest are simultaneously collected and preprocessed. Participants receive feedback of their neural activity from an image of a thermometer with a dynamic bar level. Participants use this information to train themselves to self-regulate activity in specific brain regions, while associated behaviors are measured. Previous studies have demonstrated that real-time fMRI is effective in modulating physical pain-processing and associated rostral ACC activity,[159] emotional prosody-processing and associated IFG activity,[160] and negative emotion-processing and associated AI activity.[161] Future research should examine the efficacy of real-time fMRI on modulating neural patterns supporting mental state attribution, self-awareness, and empathy for non-physical pain, as well as enhancing social emotion-processing across typical development and clinical disorders.

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REFERENCES

1. Lamm C, Singer T. The role of anterior insula cortex in social emotions. *Brain Struct Funct.* 2010; **214**: 579–591.
2. Haidt J. The moral emotions. **In**: Davidson RJ, Scherer KR, Goldsmith HH (eds). *Handbook of Affective Sciences*. Oxford University Press, Oxford, 2003; 852–870.

3. Beer JS, Keltner D. What is unique about self-conscious emotions? *Psychol. Inq.* 2004; **15**: 126–170.
4. Lewis M. Self-conscious emotions: Embarrassment, pride, shame, and guilt. **In**: Lewis M, Haviland-Jones JM, Feldman Barrett L (eds). *Handbook of Emotions*. Guilford, New York, 2008; 742–756.
5. Tangney JP, Stuewig J, Mashek DJ. Moral emotions and moral behavior. *Annu. Rev. Psychol.* 2007; **58**: 345–372.
6. Moll J, Zahn R, de Oliveira-Souza R, Krueger F, Grafman J. The neural basis of human moral cognition. *Nat. Rev. Neurosci.* 2005; **6**: 799–809.
7. Beer JS. Neural systems for self-conscious emotions and their underlying appraisals. **In**: Tracy JL, Robins RW, Tangney JP (eds). *The Self-Conscious Emotions: Theory and Research*. Guilford, New York, 2007; 53–67.
8. Lagattuta KH, Thompson RA. The development of self-conscious emotions: Cognitive processes and social influences. **In**: Tracy JL, Robins RW, Tangney JP (eds). *The Self-Conscious Emotions: Theory and Research*. Guilford, New York, 2007; 91–113.
9. Lewis M. Self-conscious emotional development. **In**: Tracy JL, Robins RW, Tangney JP (eds). *The Self-Conscious Emotions: Theory and Research*. Guilford, New York, 2007; 134–149.
10. Tracy JL, Robins RW. The self in self-conscious emotions: A cognitive appraisal approach. **In**: Tracy JL, Robins RW, Tangney JP (eds). *The Self-Conscious Emotions: Theory and Research*. Guilford, New York, 2007; 3–20.
11. Tangney JP, Tracy JL. Self-conscious emotions. **In**: Leary MR, Tangney JP (eds). *Handbook of Self and Identity*. Guilford, New York, 2012; 446–478.
12. Ortony A, Clore GL, Collins A. *The Cognitive Structure of Emotions*. Cambridge University Press, Cambridge, 1988.
13. Festinger LA. Theory of social comparison processes. *Human Relations.* 1954; **7**: 117–140.
14. Heider F. *The Psychology of Interpersonal Relations*. Lawrence Erlbaum Associates, Hillsdale, 1958.
15. Tangney JP, Mashek D, Stuewig J. Shame, guilt, and embarrassment: Will the real emotion please stand up? *Psychol. Inq.* 2005; **16**: 44–48.
16. Babcock MK, Sabini J. On differentiating embarrassment and shame. *Eur. J. Soc. Psychol.* 1990; **20**: 151–169.
17. Drummond PD, Back K, Harrison J *et al*. Blushing during social interactions in people with a fear of blushing. *Behav. Res. Ther.* 2007; **45**: 1601–1608.
18. Edelmann RJ, Hampson SE. Changes in non-verbal behaviour during embarrassment. *Br. J. Soc. Clin. Psychol.* 1979; **18**: 385–390.
19. Keltner D. Signs of appeasement: Evidence for distinct displays of embarrassment, amusement, and shame. *J. Pers. Soc. Psychol.* 1995; **68**: 441–454.
20. Tracy JL, Robins RW, Schriber RA. Development of a FACS-verified set of basic and self-conscious emotion expressions. *Emotion* 2009; **9**: 554–559.
21. Keltner D, Buswell BN. Embarrassment: Its distinct form and appeasement functions. *Psychol. Bull.* 1997; **122**: 250–270.
22. Tangney JP. The self-conscious emotions: Shame, guilt, embarrassment, and pride. **In**: Dalgleish T, Power MJ (eds). *Handbook of Cogition and Emotion*. Wiley & Sons, New York, 1999; 541–568.
23. Tangney JP, Miller RS, Flicker L, Barlow RS. Are shame, guilt, and embarrassment distinct emotions? *J. Pers. Soc. Psychol.* 1996; **70**: 1256–1269.
24. Tracy JL, Robins RW. Putting the self into self-conscious emotions: A theoretical model. *Psychol. Inq.* 2004; **15**: 103–125.
25. Smith RH, Parrott WG, Diener EF, Hoyle RH, Kim SH. Dispositional envy. *Pers. Soc. Psychol. Bull.* 1999; **25**: 1007–1020.
26. Salovey P, Rodin J. Some antecedents and consequences of social-comparison jealousy. *J. Pers. Soc. Psychol.* 1984; **47**: 780–792.
27. Smith RH, Parrott WG, Ozer D, Moniz A. Subjective injustice and inferiority as predictors of hostile and depressive feelings in envy. *Pers. Soc. Psychol. Bull.* 1994; **20**: 705–711.
28. Smith RH, Turner TJ, Garonzik R, Leach CW, Urch-Druskat V, Weston CM. Envy and schadenfreude. *Pers. Soc. Psychol. Bull.* 1996; **22**: 158–168.
29. Hareli S, Weiner B. Dislike and envy as antecedents of pleasure at another's misfortune. *Motiv. Emot.* 2002; **26**: 257–277.
30. Parrott WG, Smith RH. Distinguishing the experiences of envy and jealousy. *J. Pers. Soc. Psychol.* 1993; **64**: 906–920.
31. van Dijk WW, Ouwerkerk JW, Goslinga S, Nieweg M. Deservingness and schadenfreude. *Cogn. Emot.* 2005; **19**: 933–939.
32. van Dijk WW, Ouwerkerk JW, Goslinga S, Nieweg M, Gallucci M. When people fall from grace: Reconsidering the role of envy in schadenfreude. *Emotion* 2006; **6**: 156–160.
33. Cikara M, Botvinick MM, Fiske ST. Us versus them: Social identity shapes neural responses to intergroup competition and harm. *Psychol Sci.* 2011; **22**: 306–313.
34. Moll J, De Oliveira-Souza R, Zahn R. The neural basis of moral cognition: Sentiments, concepts, and values. *Ann. N. Y. Acad. Sci.* 2008; **1124**: 161–180.
35. Zahn R, Moll J, Krueger F, Huey ED, Garrido G, Grafman J. Social concepts are represented in the superior anterior temporal cortex. *Proc. Natl Acad. Sci. U.S.A.* 2007; **104**: 6430–6435.
36. Zahn R, Moll J, Iyengar V *et al*. Social conceptual impairments in frontotemporal lobar degeneration with right anterior temporal hypometabolism. *Brain* 2009; **132**: 604–616.
37. Decety J, Jackson PL. A social-neuroscience perspective on empathy. *Curr. Dir. Psychol. Sci.* 2006; **15**: 54–58.

38. Bernhardt BC, Singer T. The neural basis of empathy. *Annu. Rev. Neurosci.* 2012; **35**: 1–23.
39. Decety J, Jackson PL. The functional architecture of human empathy. *Behav. Cogn. Neurosci. Rev.* 2004; **3**: 71–100.
40. Fan Y, Duncan NW, de Greck M, Northoff G. Is there a core neural network in empathy? An fMRI based quantitative meta-analysis. *Neurosci. Biobehav. Rev.* 2011; **35**: 903–911.
41. 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* 2011; **54**: 2492–2502.
42. Craig AD. How do you feel – now? The anterior insula and human awareness. *Nat. Rev. Neurosci.* 2009; **10**: 57–70.
43. Menon V, Uddin LQ. Saliency, switching, attention and control: A network model of insula function. *Brain Struct. Funct.* 2010; **214**: 655–667.
44. Taylor KS, Seminowicz DA, Davis KD. Two systems of resting state connectivity between the insula and cingulate cortex. *Hum. Brain Mapp.* 2009; **30**: 2731–2745.
45. Northoff G, Bermpohl F. Cortical midline structures and the self. *Trends Cogn. Sci.* 2004; **8**: 102–107.
46. Northoff G, Heinzel A, de Greck M, Bermpohl F, Dobrowolny H, Panksepp J. Self-referential processing in our brain: A meta-analysis of imaging studies on the self. *Neuroimage* 2006; **31**: 440–457.
47. Qin P, Northoff G. How is our self related to midline regions and the default-mode network? *Neuroimage* 2011; **57**: 1221–1233.
48. Ruby P, Decety J. How would you feel vs how do you think she would feel? A neuroimaging study of perspective-taking with social emotions. *J. Cogn. Neurosci.* 2004; **16**: 988–999.
49. Gallagher HL, Frith CD. Functional imaging of 'theory of mind'. *Trends Cogn. Sci.* 2003; **7**: 77–83.
50. Young L, Saxe R. Innocent intentions: A correlation between forgiveness for accidental harm and neural activity. *Neuropsychologia* 2009; **47**: 2065–2072.
51. Greene JD, Sommerville RB, Nystrom LE, Darley JM, Cohen JD. An fMRI investigation of emotional engagement in moral judgment. *Science* 2001; **293**: 2105–2108.
52. Breiter HC, Aharon I, Kahneman D, Dale A, Shizgal P. Functional imaging of neural responses to expectancy and experience of monetary gains and losses. *Neuron* 2001; **30**: 619–639.
53. O'Doherty JP, Deichmann R, Critchley HD, Dolan RJ. Neural response during anticipation of a primary taste reward. *Neuron* 2002; **33**: 815–826.
54. O'Doherty JP. Reward representations and reward-related learning in the human brain: Insights from neuroimaging. *Curr. Opin. Neurobiol.* 2004; **14**: 769–776.
55. Seymour B, Daw N, Dayan P, Singer T, Dolan R. Differential encoding of losses and gains in the human striatum. *J. Neurosci.* 2007; **27**: 4826–4831.
56. Fehr E, Camerer CF. Social neuroeconomics: The neural circuitry of social preferences. *Trends Cogn. Sci.* 2007; **11**: 419–427.
57. Izuma K, Saito DN, Sadato N. Processing of social and monetary rewards in the human striatum. *Neuron* 2008; **58**: 284–294.
58. de Achaval D, Costanzo EY, Villarreal M *et al*. Emotion processing and theory of mind in schizophrenia patients and their unaffected first-degree relatives. *Neuropsychologia* 2010; **48**: 1209–1215.
59. Di Martino A, Ross K, Uddin LQ, Sklar AB, Castellanos FX, Milham MP. Functional brain correlates of social and nonsocial processes in autism spectrum disorders: An activation likelihood estimation meta-analysis. *Biol. Psychiatry* 2009; **65**: 63–74.
60. Lee TM, Ip AK, Wang K *et al*. Faux pas deficits in people with medial frontal lesions as related to impaired understanding of a speaker's mental state. *Neuropsychologia* 2010; **48**: 1670–1676.
61. Torralva T, Kipps CM, Hodges JR *et al*. The relationship between affective decision-making and theory of mind in the frontal variant of fronto-temporal dementia. *Neuropsychologia* 2007; **45**: 342–349.
62. Stone V, Baron-Cohen S, Knight RT. Frontal lobe contributions to theory of mind. *J. Cogn. Neurosci.* 1998; **10**: 640–656.
63. Baron-Cohen S, O'Riordan M, Stone V, Jones R, Plaisted K. Recognition of faux pas by normally developing children and children with Asperger Syndrome or high-functioning autism. *J. Autism Dev. Disord.* 1999; **29**: 407–418.
64. Beer JS, Heerey EA, Keltner D, Scabini D, Knight RT. The regulatory function of self-conscious emotion: Insights from patients with orbitofrontal damage. *J. Pers. Soc. Psychol.* 2003; **85**: 594–604.
65. Beer JS, John OP, Scabini D, Knight RT. Orbitofrontal cortex and social behavior: Integrating self-monitoring and emotion-cognition interactions. *J. Cogn. Neurosci.* 2006; **18**: 871–879.
66. Sturm VE, Rosen HJ, Allison S, Miller BL, Levenson RW. Self-conscious emotion deficits in frontotemporal lobar degeneration. *Brain* 2006; **129**: 2508–2516.
67. Sturm VE, Sollberger M, Seeley WW *et al*. Role of right pregenual anterior cingulate cortex in self-conscious emotional reactivity. *Soc. Cogn. Affect. Neurosci.* 2013; **8**: 468–474.
68. American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders*. APA, Washington, DC, 2000.
69. Baron-Cohen S, Leslie AM, Frith U. Does the autistic child have a 'theory of mind'? *Cognition* 1985; **21**: 37–46.

70. Baron-Cohen S, Jolliffe T, Mortimore C, Robertson M. Another advanced test of theory of mind: Evidence from very high functioning adults with autism or Asperger syndrome. *J. Child Psychiatry* 1997; **38**: 813–822.
71. Lombardo MV, Barnes JL, Wheelwright SJ, Baron-Cohen S. Self-referential cognition and empathy in autism. *PLoS ONE* 2007; **2**: e883.
72. Lombardo MV, Chakrabarti B, Bullmore ET *et al*. Atypical neural self-representation in autism. *Brain* 2010; **133**: 611–624.
73. Castelli F, Frith C, Happe F, Frith U. Autism, Asperger syndrome and brain mechanisms for the attribution of mental states to animated shapes. *Brain* 2002; **125**: 1839–1849.
74. Fletcher PC, Happe F, Frith U *et al*. Other minds in the brain: A functional imaging study of 'theory of mind' in story comprehension. *Cognition* 1995; **57**: 109–128.
75. Gallagher HL, Happe F, Brunswick PC, Fletcher PC, Frith U, Frith CD. Reading the mind in cartoons and stories: An fMRI study of 'theory of mind' in verbal and nonverbal tasks. *Neuropsychologia* 2000; **38**: 11–21.
76. Morita T, Kosaka H, Saito DN *et al*. Emotional responses associated with self-face processing in individuals with autism spectrum disorders: An fMRI study. *Soc Neurosci.* 2011; **7**: 223–239.
77. Pfeifer JH, Merchant JS, Colich NL, Hernandez LM, Rudie JD, Dapretto M. Neural and behavioral responses during self-evaluative processes differ in youth with and without autism. *J. Autism Dev. Disord.* 2013; **43**: 272–285.
78. Schneider K, Pauly KD, Gossen A *et al*. Neural correlates of moral reasoning in autism spectrum disorder. *Soc. Cogn. Affect. Neurosci.* 2013; **8**: 702–710.
79. Moran JM, Young LL, Saxe R *et al*. Impaired theory of mind for moral judgment in high-functioning autism. *Proc. Natl Acad. Sci. U.S.A.* 2011; **108**: 2688–2692.
80. Buon M, Dupoux E, Jacob P, Chaste P, Leboyer M, Zalla T. The role of causal and intentional judgments in moral reasoning in individuals with high functioning autism. *J. Autism Dev. Disord.* 2013; **43**: 458–470.
81. Hillier A, Allinson L. Beyond expectations: Autism, understanding embarrassment, and the relationship with theory of mind. *Autism* 2002; **6**: 299–314.
82. Hillier A, Allinson L. Understanding embarrassment among those with autism: Breaking down the complex emotion of embarrassment among those with autism. *J. Autism Dev. Disord.* 2002; **32**: 583–592.
83. Zalla T, Sav AM, Stopin A, Ahade S, Leboyer M. Faux pas detection and intentional action in Asperger Syndrome. A replication on a French sample. *J. Autism Dev. Disord.* 2009; **39**: 373–382.
84. Zalla T, Barlassina L, Buon M, Leboyer M. Moral judgment in adults with autism spectrum disorders. *Cognition* 2011; **121**: 115–126.
85. Young L, Saxe R. The neural basis of belief encoding and integration in moral judgment. *Neuroimage* 2008; **40**: 1912–1920.
86. Berthoz S, Armony JL, Blair RJR, Dolan RJ. An fMRI study of intentional and unintentional (embarrassing) violations of social norms. *Brain* 2002; **125**: 1696–1708.
87. Berthoz S, Grezes J, Armony JL, Passingham RE, Dolan RJ. Affective response to one's own moral violations. *Neuroimage* 2006; **31**: 945–950.
88. Legrand D, Ruby P. What is self-specific? Theoretical investigation and critical review of neuroimaging results. *Psychol. Rev.* 2009; **116**: 252–282.
89. Rankin KP, Kramer JH, Miller BL. Patterns of cognitive and emotional empathy in frontotemporal lobar dementia. *Cogn. Behav. Neurol.* 2005; **18**: 28–36.
90. Takahashi H, Yahata N, Koeda M, Matsuda T, Asai K, Okubo Y. Brain activation associated with evaluative processes of guilt and embarrassment: An fMRI study. *Neuroimage* 2004; **23**: 967–974.
91. Moll J, de Oliveira-Souza R, Garrido GJ *et al*. The self as a moral agent: Linking the neural bases of social agency and moral sensitivity. *Soc Neurosci.* 2007; **2**: 336–352.
92. Michl P, Meindl T, Meister F *et al*. Neurobiological underpinnings of shame and guilt: A pilot fMRI study. *Soc. Cogn. Affect. Neurosci.* 2014; **9**: 150–157.
93. O'Connor LE, Berry JW. Interpersonal guilt shame and psychological problems. *J. Soc. Clin. Psychol.* 1999; **18**: 181–203.
94. Decety J, Moriguchi Y. The empathic brain and its dysfunction in psychiatric populations: Implications for intervention across different clinical conditions. *Biopsychosoc. Med.* 2007; **1**: 22–65.
95. Wallace GL, White S, Robustelli B *et al*. Cortical and subcortical abnormalities in youths with conduct disorder and elevated callous-unemotional traits. *J. Am. Acad. Child Adolesc. Psychiatry* 2013; **53**: 456–465.
96. Raine A, Lencz T, Bihrle S, LaCasse L, Colletti P. Reduced prefrontal gray matter volume and reduced autonomic activity in antisocial personality disorder. *Arch. Gen. Psychiatry* 2000; **57**: 119–127.
97. de Oliveira-Souza R, Hare RD, Bramati IE *et al*. Psychopathy as a disorder of the moral brain: Fronto-temporolimbic grey matter reductions demonstrated by voxel-based morphometry. *Neuroimage* 2008; **40**: 1202–1213.
98. Raine A, Buchsbaum M, LaCasse L. Brain abnormalities in murderers indicated by positron emission tomography. *Biol. Psychiatry* 1997; **42**: 495–508.
99. Decety J, Chen C, Harenski C, Kiehl KA. An fMRI study of affective perspective taking in individuals with psychopathy: Imagining another in pain does not evoke empathy. *Front Hum. Neurosci.* 2013; **7**: 489.
100. Blair RJR. Neurocognitive models of aggression, the antisocial personality disorders, and psychopathy. *J. Neurol. Neurosurg. Psychiatry* 2001; **71**: 727–731.

101. Blair JR, Cipolotti L. Impaired social response reversal: A case of 'acquired sociopathy'. *Brain* 2000; **123**: 1122–1141.
102. Damasio AR, Tranel D, Damasio H. Individuals with sociopathic behavior caused by frontal damage fail to respond autonomically to social stimuli. *Behav. Brain Res.* 1990; **41**: 81–94.
103. Koenigs M, Young L, Adolphs R *et al*. Damage to the prefrontal cortex increases utilitarian moral judgements. *Nature* 2007; **446**: 908–911.
104. Blair RJ. Dysfunctions of medial and lateral orbitofrontal cortex in psychopathy. *Ann. N. Y. Acad. Sci.* 2007; **1121**: 461–479.
105. Shin LM, Dougherty DD, Orr SP *et al*. Activation of anterior paralimbic structures during guilt-related script-driven imagery. *Biol. Psychiatry* 2000; **48**: 43–50.
106. Aggleton JP, Burton MJ, Passingham RE. Cortical and subcortical afferents to the amygdala of the rhesus monkey (Macaca mulatta). *Brain Res.* 1980; **190**: 347–368.
107. Wagner U, N'Diaye K, Ethofer T, Vuilleumier P. Guilt-specific processing in the prefrontal cortex. *Cereb. Cortex* 2011; **21**: 2461–2470.
108. Goldin PR, McRae K, Ramel W, Gross JJ. The neural bases of emotion regulation: Reappraisal and suppression of negative emotion. *Biol. Psychiatry* 2008; **63**: 577–586.
109. Ochsner KN, Ray RD, Cooper JC *et al*. For better or for worse: Neural systems supporting the cognitive down- and up-regulation of negative emotion. *Neuroimage* 2004; **23**: 483–499.
110. Morey RA, McCarthy G, Selgrade ES, Seth S, Nasser JD, LaBar KS. Neural systems for guilt from actions affecting self versus others. *Neuroimage* 2012; **60**: 683–692.
111. Finger EC, Marsh AA, Kamel N, Mitchell DG, Blair JR. Caught in the act: The impact of audience on the neural response to morally and socially inappropriate behavior. *Neuroimage* 2006; **33**: 414–421.
112. Chang LJ, Smith A, Dufwenberg M, Sanfey AG. Triangulating the neural, psychological, and economic bases of guilt aversion. *Neuron* 2011; **70**: 560–572.
113. Zahn R, Moll J, Paiva M *et al*. The neural basis of human social values: Evidence from functional MRI. *Cereb. Cortex* 2009; **19**: 276–283.
114. Green S, Ralph MA, Moll J, Stamatakis EA, Grafman J, Zahn R. Selective functional integration between anterior temporal and distinct fronto-mesolimbic regions during guilt and indignation. *Neuroimage* 2010; **52**: 1720–1726.
115. Zahn R, de Oliveira-Souza R, Bramati I, Garrido G, Moll J. Subgenual cingulate activity reflects individual differences in empathic concern. *Neurosci. Lett.* 2009; **457**: 107–110.
116. Pulcu E, Zahn R, Elliott R. The role of self-blaming moral emotions in major depression and their impact on social-economical decision making. *Front. Psychol.* 2013; **4**: 310.
117. Green S, Lambon Ralph MA, Moll J, Deakin JF, Zahn R. Guilt-selective functional disconnection of anterior temporal and subgenual cortices in major depressive disorder. *Arch. Gen. Psychiatry* 2012; **69**: 1014–1021.
118. Johansen-Berg H, Gutman DA, Behrens TE *et al*. Anatomical connectivity of the subgenual cingulate region targeted with deep brain stimulation for treatment-resistant depression. *Cereb. Cortex* 2008; **18**: 1374–1383.
119. Mancini F, Gangemi A. Fear of guilt from behaving irresponsibly in obsessive-compulsive disorder. *J. Behav. Ther. Exp. Psychiatry* 2004; **35**: 109–120.
120. Shafran R, Watkins E, Charman T. Guilt in obsessive-compulsive disorder. *J. Anxiety Disord.* 1996; **10**: 509–516.
121. Basile B, Mancini F, Macaluso E, Caltagirone C, Bozzali M. Abnormal processing of deontological guilt in obsessive-compulsive disorder. *Brain Struct. Funct.* 2013. doi 10.1007/s00429-013-0570-2
122. Neubauer AC, Fink A. Intelligence and neural efficiency. *Neurosci. Biobehav. Rev.* 2009; **33**: 1004–1023.
123. Fliessbach K, Weber B, Trautner P *et al*. Social comparison affects reward-related brain activity in the human ventral striatum. *Science* 2007; **318**: 1305–1308.
124. Qiu J, Yu C, Li H *et al*. The impact of social comparison on the neural substrates of reward processing: An event-related potential study. *Neuroimage* 2010; **49**: 956–962.
125. Yamada M, Lamm C, Decety J. Pleasing frowns, disappointing smiles: An ERP investigation of counterempathy. *Emotion* 2011; **11**: 1336–1345.
126. Tricomi E, Rangel A, Camerer CF, O'Doherty JP. Neural evidence for inequality-averse social preferences. *Nature* 2010; **463**: 1089–1091.
127. Dvash J, Gilam G, Ben-Ze'ev A, Hendler T, Shamay-Tsoory SG. The envious brain: The neural basis of social comparison. *Hum. Brain Mapp.* 2010; **31**: 1741–1750.
128. Bault N, Joffily M, Rustichini A, Coricelli G. Medial prefrontal cortex and striatum mediate influence of social comparison on the decision process. *Proc. Natl Acad. Sci. U.S.A.* 2011; **108**: 16044–16049.
129. Singer T, Seymour B, O'Doherty JP, Stephan KE, Dolan RJ, Frith CD. Empathic neural responses are modulated by the perceived fairness of others. *Nature* 2006; **439**: 466–469.
130. Hein G, Silani G, Preuschoff K, Batson CD, Singer T. Neural responses to ingroup and outgroup members' suffering predict individual differences in costly helping. *Neuron* 2010; **68**: 149–160.
131. Xu X, Zuo X, Wang X, Han S. Do you feel my pain? Racial group membership modulates empathic neural responses. *J. Neurosci.* 2009; **29**: 8525–8529.
132. Cikara M, Fiske ST. Bounded empathy: Neural responses to outgroup targets' (mis)fortunes. *J. Cogn. Neurosci.* 2011; **23**: 3719–3803.

133. Cikara M, Fiske ST. Stereotypes and Schadenfreude: Affective and physiological markers of pleasure at outgroup misfortunes. *Soc. Psychol.* 2012; **3**: 63–71.
134. Takahashi H, Kato M, Matsuura M, Mobbs D, Suhara T, Okubo Y. When your gain is my pain and your pain is my gain: Neural correlates of envy and schadenfreude. *Science* 2009; **323**: 937–939.
135. van Veen V, Krug MK, Schooler JW, Carter CS. Neural activity predicts attitude change in cognitive dissonance. *Nat. Neurosci.* 2009; **12**: 1469–1474.
136. Eisenberger NI, Lieberman MD. Why rejection hurts: A common neural alarm system for physical and social pain. *Trends Cogn. Sci.* 2004; **8**: 294–300.
137. Friederich HC, Uher R, Brooks S *et al*. I'm not as slim as that girl: Neural bases of body shape self-comparison to media images. *Neuroimage* 2007; **37**: 674–681.
138. Friederich HC, Brooks S, Uher R *et al*. Neural correlates of body dissatisfaction in anorexia nervosa. *Neuropsychologia* 2010; **48**: 2878–2885.
139. Shamay-Tsoory SG, Tibi-Elhanany Y, Aharon-Peretz J. The green-eyed monster and malicious joy: The neuroanatomical bases of envy and gloating (schadenfreude). *Brain* 2007; **130**: 1663–1678.
140. Shamay-Tsoory SG. Recognition of 'fortune of others' emotions in Asperger syndrome and high functioning autism. *J. Autism Dev. Disord.* 2008; **38**: 1451–1461.
141. Blakemore SJ. The social brain in adolescence. *Nat. Rev. Neurosci.* 2008; **9**: 267–277.
142. Choudhury S, Blakemore SJ, Charman T. Social cognitive development during adolescence. *Soc. Cogn. Affect. Neurosci.* 2006; **1**: 165–174.
143. Pfeifer JH, Peake SJ. Self-development: Integrating cognitive, socioemotional, and neuroimaging perspectives. *Dev. Cogn. Neurosci.* 2012; **2**: 55–69.
144. Somerville LH. The teenage brain: Sensitivity to social evaluation. *Curr. Dir. Psychol. Sci.* 2013; **22**: 121–127.
145. Orth U, Robins RW, Soto CJ. Tracking the trajectory of shame, guilt, and pride across the life span. *J. Pers. Soc. Psychol.* 2010; **99**: 1061–1071.
146. Williams JR, Insel TR, Harbaugh CR, Carter CS. Oxytocin administered centrally facilitates formation of a partner preference in female prairie voles (Microtus ochrogaster). *J. Neuroendocrinol.* 1994; **6**: 247–250.
147. Young LJ, Wang Z. The neurobiology of pair bonding. *Nat. Neurosci.* 2004; **7**: 1048–1054.
148. Barraza JA, Zak PJ. Empathy toward strangers triggers oxytocin release and subsequent generosity. *Ann. N. Y. Acad. Sci.* 2009; **1167**: 182–189.
149. Hurlemann R, Patin A, Onur OA *et al*. Oxytocin enhances amygdala-dependent, socially reinforced learning and emotional empathy in humans. *J. Neurosci.* 2010; **30**: 4999–5007.
150. Averbeck BB. Oxytocin and the salience of social cues. *Proc. Natl Acad. Sci. U.S.A.* 2010; **107**: 9033–9034.
151. Bartz JA, Zaki J, Bolger N, Ochsner KN. Social effects of oxytocin in humans: Context and person matter. *Trends Cogn. Sci.* 2011; **15**: 301–309.
152. Shamay-Tsoory SG. Oxytocin, social salience, and social approach. *Biol. Psychiatry* 2010; **67**: e35.
153. De Dreu CK. Oxytocin modulates cooperation within and competition between groups: An integrative review and research agenda. *Horm. Behav.* 2012; **61**: 419–428.
154. Shamay-Tsoory SG, Fischer M, Dvash J, Harari H, Perach-Bloom N, Levkovitz Y. Intranasal administration of oxytocin increases envy and schadenfreude (gloating). *Biol. Psychiatry* 2009; **66**: 864–870.
155. Hollander E, Novotny S, Hanratty M *et al*. Oxytocin infusion reduces repetitive behaviors in adults with autistic and Asperger's disorders. *Neuropsychopharmacology* 2003; **28**: 193–198.
156. Hollander E, Bartz J, Chaplin W *et al*. Oxytocin increases retention of social cognition in autism. *Biol. Psychiatry* 2007; **61**: 498–503.
157. Domes G, Heinrichs M, Kumbier E, Grossmann A, Hauenstein K, Herpertz SC. Effects of intranasal oxytocin on the neural basis of face processing in autism spectrum disorder. *Biol. Psychiatry* 2013; **74**: 164–171.
158. Caria A, Sitaram R, Birbaumer N. Real-time fMRI: A tool for local brain regulation. *Neuroscientist* 2012; **18**: 487–501.
159. DeCharms RC. Applications of real-time fMRI. *Nat. Rev. Neurosci.* 2008; **9**: 720–729.
160. Rota G, Sitaram R, Veit R *et al*. Self-regulation of regional cortical activity using real-time fMRI: The right inferior frontal gyrus and linguistic processing. *Hum. Brain Mapp.* 2009; **30**: 1605–1614.
161. Caria A, Veit R, Sitaram R *et al*. Regulation of anterior insular cortex activity using real-time fMRI. *Neuroimage* 2007; **35**: 1238–1246.