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Review

Understanding Amygdala Responsiveness to Fearful Expressions Through the Lens of Psychopathy and Altruism

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Because the face is the central focus of human social interactions, emotional facial expressions provide a unique window into the emotional lives of others. They play a particularly important role in fostering empathy, which entails understanding and responding to others' emotions, especially distress-related emotions such as fear. This Review considers how fearful facial as well as vocal and postural expressions are interpreted, with an emphasis on the role of the amygdala. The amygdala may be best known for its role in the acquisition and expression of conditioned fear, but it also supports the perception and recognition of others' fear. Various explanations have been supplied for the amygdala's role in interpreting and responding to fearful expressions. They include theories that amygdala responses to fearful expressions 1) reflect heightened vigilance in response to uncertain danger, 2) promote heightened attention to the eye region of faces, 3) represent a response to an unconditioned aversive stimulus, or 4) reflect the generation of an empathic fear response. Among these, only empathic fear explains why amygdala lesions would impair fear recognition across modalities. Supporting the possibility of a link between fundamental empathic processes and amygdala responses to fear is evidence that impaired fear recognition in psychopathic individuals results from amygdala dysfunction, whereas enhanced fear recognition in altruistic individuals results from enhanced amygdala function. Empathic concern and caring behaviors may be fostered by sensitivity to signs of acute distress in others, which relies on intact functioning of the amygdala.

Key words: fear; facial expressions; empathy; psychopathy

The discovery that facial expressions of emotions such as happiness, anger, and fear are recognized and expressed by humans across cultures was an important advance in the study of human emotions (Elfenbein and Ambady, 2002). In demonstrating that inhabitants of a remote village in New Guinea could make sense of emotional facial expressions displayed by Americans, Ekman and colleagues showed that some emotional behaviors are not purely products of socialization but are to some extent universal and innate (Ekman et al., 1969; Ekman and Friesen, 1971). This makes the study of facial expressions (and related vocal and postural expressions) an invaluable window into the biology of human emotional processes. In the ensuing decades, neuroscience and psychology research investigating when and how humans recognize and respond to emotional facial expressions has continued to provide important clinical, cultural, developmental, and social insights (Calder et al., 2011; Somerville et al., 2011; Gilboa-Schechtman and Shachar-Lavie, 2013; Whalen et al., 2013; Tracy et al., 2015).

The study of facial expressions has provided particular traction to research on empathy. Broadly speaking, empathy involves recognizing, understanding, or responding appropriately to another person's cognitive or emotional state (de Waal, 2009). Facial expressions are among

SIGNIFICANCE:

Fearful facial expressions are commonly used as stimuli in developmental, clinical, social, and neuroscience research seeking to understand features of human social and emotional functioning. It is therefore essential to understand the significance of these stimuli to perceivers and the mechanisms underlying the identification of and responses to these expressions. This Review considers various theories concerning the role of the amygdala in responding to fear communicated by the face as well as by the voice, the body, and music, with a focus on the relationship between the perception of fear and empathic processes, including emotional empathy, empathic concern, aggression, and altruism.

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the cues upon which human perceivers most rely to learn about others' emotional states and so are critical to fostering empathy (Elfenbein et al., 2002). Not all expressions are equally relevant to empathy, however. The form of empathy sometimes termed *empathic concern* or *compassion* is predominantly a response to distress, and its occurrence is most closely linked with the perception of expressions that communicate distress (Nichols, 2001). The emotional expression that conveys the most acute and severe form of distress is fear, and the perception of this expression is particularly strongly linked to empathic concern (Marsh, 2013). Understanding how fearful expressions are perceived and interpreted at a neurological level may be essential for understanding the basis of empathy, particularly of empathic concern, as well as social behaviors such as altruism and aggression that empathic concern may promote or inhibit (Marsh and Blair, 2008; Dolan and Fullam, 2009; Jones et al., 2009; Marsh et al., 2014b). This Review considers the question of how fearful expressions, including facial, vocal, and postural expressions, are understood, with an emphasis on the role of the amygdala, which has been consistently linked with the ability to perceive and respond to others' expressions of fear (Adolphs et al., 1999; Sprengelmeyer et al., 1999; Fusar-Poli et al., 2009).

THE AMYGDALA AND THE EXPERIENCE OF FEAR

The amygdala is a subcortical temporal lobe structure said to be the most densely interconnected structure in the forebrain (Young et al., 1994). White matter connections with the amygdala extend to, among other regions, the brainstem, the hypothalamus, the striatum, and regions of the cortex that include orbitofrontal cortex, superior temporal cortex, insula, and anterior cingulate cortex (Davis and Whalen, 2001; Rosen and Donley, 2006; Robinson et al., 2010). This dense connectivity suggests that the amygdala plays a modulatory role in the many functions subserved by these regions (LaBar and Warren, 2009).

The amygdala may be best known for the essential role it plays in processes related to fear, including fear learning and the generation of the subjective experience of fear (Davis and Whalen, 2001; LeDoux, 2003; Wilensky et al., 2006). Research in rodents has established that an intact amygdala is essential for fear learning and, in particular, learning to associate a stimulus or behavior with a subsequent aversive outcome (LeDoux, 2003). In a standard fear-learning paradigm, a mouse learns that when a particular tone plays, an electric current may flood the metal grating on the floor of the mouse's cage shortly afterward, causing the mouse to experience an uncomfortable shock to its feet. In this paradigm, the tone starts out as a neutral stimulus. After repeated pairing of the tone with the shock, mice come to respond to the tone with characteristic physiological and behavioral responses, including freezing, increased heart rate, respiration changes, startle, and ultrasonic distress vocalizations (Davis and Whalen, 2001; Kellett and Kokkinidis, 2004); they have come to fear the tone. The components of the fear response emerge because they are useful adaptations for avoiding or escaping anticipated danger (Lang et al., 2000).

Relevant to this is that fear-related responses are more likely to emerge when the anticipated aversive outcome is uncertain (Whalen, 1998; Rosen and Donley, 2006; Sarinopoulos et al., 2010). For example, animals exhibit stronger fear responding when the tone has usually predicted a shock in the past than when it has always predicted a shock (Powell and Milligan, 1975). This is consistent with the idea that fear-related behaviors exist to allow the organism to avoid or escape unpleasant or harmful outcomes. If an outcome is inevitable there is no possible escape, so the animal is better off conserving its energy rather than marshaling a futile response. Indeed, learned cues that signal unavoidable aversive outcomes elicit low-energy resource-conservation responses instead of high-energy fear behaviors (Maier and Seligman, 1976).

How does activation in the amygdala contribute to the state that we call fear? The amygdala is an internally complex structure consisting of multiple interconnected nuclei that are defined based on their histological features as well as their projections. They include the basolateral nuclei (including the basal, lateral, and accessory basal nuclei) and the corticomedial nuclei (including the medial and central nuclei) (LeDoux, 2007). The lateral nucleus is the primary input nucleus of the amygdala and is the target of projections from sensory cortex and the thalamus. When these external regions project information about a fear-relevant stimulus in the environment to the lateral nucleus, it routes this information to the central nucleus, which is the primary output nucleus of the amygdala, particularly with regard to physiological and behavioral emotional responses (LeDoux, 2007). The central nucleus projects to the hypothalamus and periaqueductal gray, which coordinate autonomic, hormonal, and behavioral fear responses.

The lateral-nucleus-potentiated increase in central nucleus output to the hypothalamus and periaqueductal gray seems to represent the core of the conditioned fear response system in mammals. Plasticity in this circuit underlies the development of a conditioned fear response to a previously neutral stimulus (LeDoux, 2007). Electrical stimulation of components of this circuit can generate the complete suite of fear responses, including, in humans, subjective reports of feelings of fear and anxiety (Chapman et al., 1954; Davis and Whalen, 2001). Ablation of these regions of the amygdala results in marked fearlessness both in nonhuman animals in the laboratory and in humans with rare complete and selective bilateral lesions to the amygdala (Aggleton and Passingham, 1981; LaBar and LeDoux, 1996; Feinstein et al., 2011). Particularly useful information with regard to the fear–amygdala link has been acquired through extensive testing of amygdala lesion patients such as SM, who suffered focal, complete destruction of her bilateral amygdala during adolescence. This resulted from lipoid proteinosis caused by the rare genetic disorder Urbach-Wiethe (which only in rare cases produces total bilateral amygdala lesions as

seen in SM). SM reports subjective experiences of multiple emotions, including excitement, anger, and sadness but reports essentially no subjective experiences of fear in response to snakes, spiders, and other stimuli that commonly induce intense fear (Bechara et al., 1995; Feinstein et al., 2011).

The amygdala appears to be central to fear in a way that it is not central to other emotions. Its role in emotional responding should not be oversimplified, however. The essential function for which the amygdala appears to be necessary is the acquisition and expression of conditioned fear (Duvarci and Pare, 2014). Related processes, such as panic in response to air hunger (Feinstein et al., 2013) or second-order conditioning (Hatfield et al., 1996), may involve the amygdala but are reliant on distinct processes and neural structures. The amygdala can also be responsive to cues that signal reward (Murray, 2007). In some such cases, amygdala responses may represent a signal that there is *not* currently anything fearful in the environment requiring attention. This reflects the mutually inhibitory interactions observed between the striatum and the central nucleus of the amygdala such that, for example, inhibiting activity in the basolateral neurons that project to the central nucleus inhibits fear conditioning and simultaneously potentiates reward conditioning (Namburi et al., 2015). It is clear, however, that the amygdala is not required for responding to and learning about rewards in the same way that it is required for responding to and learning about threat (Baxter and Murray, 2002). Reward learning that parallels the fear learning described above is unaffected by amygdala lesions (Hatfield et al., 1996). If amygdala lesions impaired all forms of emotional responding equally, amygdala-lesioned animals and humans would appear anhedonic, exhibiting neither approach nor avoidance, neither pleasure nor displeasure, and this is plainly not the case (Davis and Whalen, 2001; Feinstein et al., 2011).

RESPONDING TO FEAR IN OTHERS

The amygdala plays a critical role not only in generating internal experiences of fear but also in responding to others' fear. This has been demonstrated through experiments in which participants are presented with facial expressions (or sometimes vocal expressions or body postures) and then select the emotion displayed by each from multiple options. The expressions usually include the "basic" emotions anger, disgust, fear, happiness, sadness, and surprise. Individuals with diffuse temporal lobe damage may have trouble identifying multiple emotions (Adolphs et al., 1999), but when lesions are confined to the bilateral amygdala the recognition deficits tend to be most pronounced for fear (Adolphs et al., 1994; Sprengelmeyer et al., 1999; Anderson and Phelps, 2000).

Seminal studies on this topic have been conducted with SM. These have shown that her responses to facial expressions of anger, disgust, happiness, sadness, and surprise are strongly correlated with responses of healthy controls (all $r > 0.65$), but her responses to fear are dramatically impaired, with r ranging from <0.50 to <0.10 across testing sessions (Adolphs et al., 1994). Controls with damage in other regions of the brain do not show comparable patterns. This finding holds up in repeated testing of SM and other amygdala-lesion patients (Sprengelmeyer et al., 1999; Anderson and Phelps, 2000; Adolphs, 2008) and extends to other domains of emotion recognition. For example, SM also cannot draw fearful expressions, although she capably draws faces expressing other emotions. When asked to draw a fearful face, she stated that "she did not know what an afraid face would look like" (Adolphs et al., 1995). Her final drawing showed not a face at all, but a human figure in profile described by the authors as a person cowering with his hair standing on end.

The effects of bilateral amygdala lesions on recognition of fear from other channels are not as uniform, but impairments in recognizing fear from vocal cues and body postures (Sprengelmeyer et al., 1999) and even music (Gosselin et al., 2007) have been observed (but see Anderson and Phelps, 1998; Atkinson et al., 2007). Subsequent evidence from other Urbach-Wiethe patients indicates that the central amygdala is the essential locus of these effects, given that patients in whom only the basolateral region is destroyed may show, if anything, heightened sensitivity to fearful expressions (Terburg et al., 2012). In addition, individuals with extensive additional damage to regions outside the amygdala tend to show divergent patterns of effects (Adolphs et al., 1999).

Neuroimaging research in healthy adults has also shown that the amygdala responds preferentially to fearful expressions. The most comprehensive evaluation of this topic is a meta-analysis of neural responses to facial expressions using quantitative data from 105 functional magnetic resonance imaging (fMRI) studies (Fusar-Poli et al., 2009) that were analyzed using activation-likelihood estimation (Turkeltaub et al., 2002). Results of this meta-analysis showed that responses in bilateral amygdala to fearful expression dwarf responses to other expressions. Relative to baseline, activity in bilateral amygdala increases in response to fear; activity in left amygdala also increases in response to neutral, disgusted, and happy expressions. The authors compared amygdala activation in response to each emotional expression with responses to neutral expressions and found increases only in response to fear (bilaterally), happiness (bilaterally), and sadness (right only). Finally, directly comparing amygdala responses to fear with both happiness and sadness shows that, for both comparisons, the amygdala is significantly more active in response to fear (Fig. 1).

Amygdala responses to fearful expressions emerge independently of higher level cognitive processes. Intracranial recordings and magnetoencephalography show that the amygdala's preferential response to fearful expressions occurs preattentively, within a few hundred milliseconds (Luo et al., 2007; Pourtois et al., 2010). The amygdala remains preferentially responsive to fearful expressions when they are presented for less than 30 msec, too fast to be consciously detected (Whalen et al.,

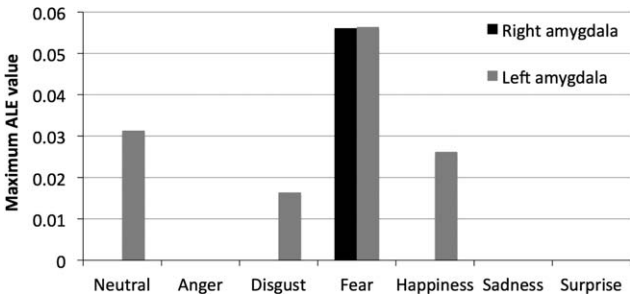

Fig. 1. Activation likelihood estimate (ALE) values reported by Fusar-Poli and colleagues (2009) that represent the results of 125 fMRI studies investigating neural responses to facial expressions. ALE is a method for meta-analyzing neuroimaging data that provides a quantitative estimate of the probability of activation in a given region. Activation in bilateral amygdala in response to fearful expressions is greater than activation in response to any other expression.

1998; Viding et al., 2012), even when only the whites of fearful eyes are presented at these latencies (Whalen et al., 2004). The amygdala is also preferentially responsive to fearful expressions shown to blindsight patients who lack a functioning visual cortex or any conscious visual perception (Pegna et al., 2004).

Although less fMRI research has examined responses to emotional expressions other than facial expressions, the amygdala also appears to respond preferentially to vocal expressions of fear (Phillips et al., 1998; Aube et al., 2015) and to written statements that evoke fear relative to other emotions (Marsh and Cardinale, 2014). One investigation of emotional responding across three modalities (faces, voices, and music) showed that, within a given subject, the same voxels within the amygdala responded preferentially to fear stimuli across modalities but not to happy or sad stimuli (Aube et al., 2015). That the amygdala responds in similar ways to expressions of fear across modalities (visual, auditory, semantic) suggests that these responses are not dependent on low-level stimulus features. That it responds in the absence of awareness suggests that these responses are not reliant on higher level conscious cognitive processes. That amygdala lesions impair recognition of fear suggests that the activity taking place in the amygdala during the perception of others' fear is important for correctly interpreting it, not just for attending or responding to it.

WHY DOES THE AMYGDALA RESPOND PREFERENTIALLY TO FEAR?

So what *is* the amygdala doing during the perception of others' fear? Several possible explanations have been proposed since the first fMRI and positron emission tomography studies demonstrating this effect were published in the 1990s (Morris et al., 1996; Phillips et al., 1997; Whalen et al., 1998). Morris and colleagues (1996) were critical in shaping how the effect has been interpreted. They wrote that, because "integrated responses to threat or danger . . . can be mediated by the amygdala," it can be inferred that "perceiving an expression of fear in a conspecific may trigger an automatic response to potential danger that accounts for the observed amygdala activation in response to fearful faces." In other words, if the amygdala responds to cues that signal threat and if the amygdala responds to fearful facial expressions, then fearful expressions must signal threat. Problems with this interpretation arose when subsequent studies confirmed that the amygdala is not strongly responsive to angry facial expressions (Sprengelmeyer et al., 1998; Blair et al., 1999; Whalen et al., 2001; Fusar-Poli et al., 2009) or necessary for interpreting them (Adolphs et al., 1994; Sprengelmeyer et al., 1999). This is problematic because angry expressions clearly signal threat, with anger being the emotion that typically accompanies aggressive attack (Blair, 2012).

Vigilance in Response to Ambiguity

Addressing this discrepancy, Whalen and colleagues (1998) formulated a theory that considered the different types of information conveyed by fearful vs. angry expressions. Recall that the amygdala is preferentially engaged when an anticipated aversive outcome is uncertain rather than certain. This suggests that the amygdala may be particularly responsive to fearful expressions because they represent a more uncertain or ambiguous source of threat than angry expressions. In the case of an angry expression, the source of the threat is the expresser. In the case of a fearful expression, the source of the threat is *not* the expresser but something else in the environment to which the expresser is responding. Whalen and colleagues surmised that fearful facial expressions convey an inherent ambiguity about the nature of the threat that serves to heighten vigilance in the perceiver. Supporting this theory, viewing fearful but not angry expressions increases perceivers' sensitivity to stimuli in the visual periphery, indicative of heightened vigilance (Becker, 2009; Taylor and Whalen, 2014).

The vigilance in response to ambiguity theory remains perhaps the dominant explanation for heightened amygdala responses during the perception of fearful facial expressions. There are two potential concerns with the theory, however. The first is that it represents a reverse inference, in that the cognitive response to a stimulus is inferred from the observed neural responses to it (Poldrack, 2006). Reverse inferences may represent a useful starting point but are not conclusive. A second concern is that the theory explains why patients with amygdala lesions show aberrant autonomical or behavioral responses to fearful expressions, but it does not obviously explain their impairments in *identifying* these expressions. Why would an intact amygdala be required to explicitly link the appearance of raised brows, widened eyes, and a grimace with the concept of fear?

Attention to the Eyes

A variation on this theory that addresses this second concern focuses on the importance of the eye region in recognizing fearful expression. The attention to eyes theory shares with the vigilance in response to ambiguity

theory an emphasis on the amygdala's known role in helping to direct attention to salient stimuli (Anderson and Phelps, 2001; Adolphs, 2008; Pessoa and Adolphs, 2010) but focuses on the amygdala's role in directing attention to the features of the face itself. The eyes are the facial feature most critical for making many social judgments and are the features to which people naturally direct their attention (Guo et al., 2010). The eye region conveys particularly important information for recognizing fearful expressions, the most prominent features of which are wide eyes and raised and drawn-together brows (Bassili, 1979; Smith et al., 2005). An elegant series of studies with SM found that, in contrast to controls, she does not naturally direct her gaze to the eye region of any facial expressions. However, she can direct her attention to the eyes when instructed to do so, and this instruction normalizes her ability to recognize fearful expressions (Adolphs et al., 2005). The instructions did not permanently rehabilitate her ability to recognize fear, however; in subsequent testing sessions her gaze pattern and recognition abilities reverted to baseline. The authors concluded that amygdala lesions impair fear recognition because they impede normal gaze patterns required to extract information relevant to recognizing fear.

This theory potentially explains the deficits in recognizing fearful facial expressions observed in SM and other amygdala lesion patients. It does not, however, explain the role of the amygdala in recognizing and responding to fear conveyed by other modalities, such as vocal expressions, body postures, or music. If patients such as SM simply failed to show preferential attention toward fearful vocal or body expressions or music, this could be attributed to the amygdala's role in directing attention to salient stimuli, but the failure of these patients to *identify* these stimuli as fearful cannot as easily be explained in this way. Body movements, vocal expressions, and music do not convey fear via one particular low-level sensory feature, such as the eyes of fearful faces, to which attention can be preferentially directed. Rather, they convey fear through combinations of elements that must be interpreted in relation to one another. For example, the emotional tone of music depends on combinations of features, such as consonance and changes in pitch and rate (Sievers et al., 2013). It is unclear how directed attention to any one of these features would improve identification of this kind of fearful stimulus.

Unconditioned Aversive Stimuli

A theory that better captures the cross-modal nature of amygdala responses to fearful expressions, Blair's (2005) integrated emotion systems model, focuses on affective processes rather than low-level perceptual processes. According to this theory, fearful facial expressions represent unconditioned aversive stimuli. As a result, fearful expressions serve as punishment cues that help socialize developing children to avoid aggressive behaviors that cause others fear. In support of the idea that fearful expressions serve as unconditioned reinforcers, the amygdala is more active when fearful (and happy) expressions are presented during a learning task requiring participants to learn emotion–object pairings than when the expressions are presented in isolation (Hooker et al., 2006). This theory also captures important aspects of how nonverbal displays of fear are actually used by humans and other social species. Ethologists agree that, across multiple species, nonverbal fear displays are typically exhibited during agonistic interactions and convey submission and appeasement with the goal of preventing or ending an aggressive encounter (Eibl-Eibesfeldt, 1996; Schenkel, 1967).

As is the case for the vigilance in response to ambiguity theory, the integrated emotion systems model generates a clear explanation for why amygdala lesions impair normal behavioral and autonomic responses to fearful expressions. However, it does not explain as clearly why amygdala lesions prevent fearful expressions from being identified. In addition, and as is also the case for the vigilance in response to ambiguity theory, this theory assumes that fearful facial expressions represent aversive stimuli. However, the results of approach–avoidance tasks show the opposite; fearful expressions elicit approach, not avoidance (Marsh et al., 2005b; Hammer and Marsh, 2015), indicating that they are primarily appetitive. These theories, then, appear not to capture important features of the perceptual significance of fearful expressions.

Empathic Simulation

How then to best understand the relationship between the amygdala and the recognition of others' fear? None of these theories clearly accounts for all the observed data. Even combined (they are not mutually incompatible), they do not account for why amygdala lesions can impair the recognition of fear across modalities, including the face, the voice, the body, and music. One early hypothesis put forth by Adolphs and colleagues (1995) does account for this pattern of findings, however. This is the hypothesis that the amygdala is essential for linking perceptual representations of fear, such as facial or vocal expressions, to internal representations of fear. In other words, amygdala responses to fearful expressions reflect the generation of an internal representation of fear that can then be linked to visual, auditory, or semantic cues associated with fear. This hypothesized process is alternately called *emotional empathy*, *emotional contagion*, or *simulation* (Preston and de Waal, 2002; Goldman and Sripada, 2005; Bird and Viding, 2014; Dvash and Shamay-Tsoory, 2014). It clearly explains why amygdala damage impairs the ability to label fearful expressions and not only respond behaviorally or autonomically to them: because accurate labeling requires linking the external perceptual cue to an internally generated representation of fear. The amygdala is not required for identifying other emotions, such as disgust, anger, or happiness, because activity in the amygdala is not essential for generating internal representations of these emotions.

This theory most cleanly explains why fear is the emotion in which SM and other patients with complete

bilateral amygdala damage are impaired, both in personal experience and in recognition of it in others. It is *because* they are impaired in experiencing fear—because they cannot generate an adequate internal representation of this state—that they have difficulty labeling it in others. A recent study of identical twins affected by Urbach-Wiethe who both have near-complete bilateral amygdala damage supports this possibility. One twin exhibits no fear-potentiated startle response and is also highly impaired in recognizing fearful expressions; the other twin retains an intact fear-potentiated startle response and some ability to recognize fearful expressions (Becker et al., 2012). This study directly links impairments in experiencing fear to impairments in recognizing fear. Along the same lines, a recent study in healthy adults found that the self-reported intensity of personal experiences of fear predicts individual variation in the ability to recognize fearful expressions (Buchanan et al., 2010). Together, these lines of evidence lead to the conclusion that the amygdala is essential to a basic form of empathy, which is the ability to link another person's experience of fear to one's own experiences of fear. That the perceiver is able to label the other person's internal state correctly is evidence that this empathic process has occurred.

Much remains unknown about how the internal representations are generated that allow this empathic process to occur, although a few features seem clear. The representations likely share associative links with semantic representations of fear but are not themselves semantic (Bird and Viding, 2014). Evidence for this includes the fact that amygdala lesions impair physiological responses to threat but not semantic knowledge about threats, whereas the reverse is true for hippocampal lesions (Bechara et al., 1995). Representations of fear seem unlikely to originate in the cortex, given evidence of intact fear conditioning in the absence of a cortex (Bromiley, 1948; Oakley and Russell, 1972), but full-fledged, coherent representations of fear likely incorporate information generated by somatosensory and premotor cortex, which contribute to sensory and motor features of fear (Adolphs et al., 2000; Hussey and Safford, 2009; Braadbaart et al., 2013), and midline parietal regions such as the precuneus, which may generate multimodal autobiographical representations related to fear (Cavanna and Trimble, 2006; Marsh and Cardinale, 2014). Perhaps the amygdala plays a role in integrating information from these regions to form a coherent representation of fear (LeDoux et al., 1990). Whether lower level neural regions, such as the hypothalamus and brainstem, or peripheral systems, such as the autonomic nervous system or adrenal system, play a role in internal representations of fear is less clear. Clearly the hypothalamic–pituitary–adrenal axis becomes active during the experience or perception of fear, but this could be a downstream effect rather than an essential feature (Dunn et al., 2006).

Several points should be made about the hypothesized empathic fear response. First, emotion recognition is only a proxy for an empathic response. One weakness in relying on emotion identification is that semantic knowledge-based workarounds could enable even highly impaired patients to identify some expressions accurately. For example, the knowledge that fearful vocalizations are high pitched or that frightened people crouch (information that SM, for example, is known to possess; see Adolphs et al., 1995) might improve labeling of fear vocalizations or postures even in the absence of an empathic fear response if the paradigm does not include well-matched stimuli and response options. Semantic-knowledge-based judgments may help to explain inconsistencies in the emotion recognition literature (Anderson and Phelps, 1998; Adolphs and Tranel, 1999; Atkinson et al., 2007). It would be useful to determine whether amygdala lesions impair labeling of emotional stimuli for which no low-level perceptual features can be used as clues. Features such as wide eyes (for faces) or high pitch (for voices) are relatively easy markers for identifying fearful stimuli using semantic knowledge. In contrast, the emotionally evocative statements developed by Marsh and Cardinale (2012, 2014) cannot be identified via low-level features. In the emotional statements paradigm, participants must identify the emotion that a statement such as "I could easily hurt you" would cause the listener to experience. Identifying emotion from a written verbal stimulus such as this is a conceptual, not perceptual, process. There is no single piece of semantic knowledge that could be used to identify the 20 different fear-evoking stimuli in this paradigm. If amygdala lesions preferentially impaired recognition of fear-eliciting statements such as these, it would provide additional support for the theory of empathic simulation.

Second, it is not suggested that an empathic fear response means that the perceiver necessarily experiences full-fledged fear following the perception of others' fear (Bird and Viding, 2014). Given that amygdala responses to fearful expressions occur even in the absence of conscious awareness that a face has been detected, empathic fear responses may presumably be quite subtle. This is consistent with other types of empathic responses, such as empathic pain responses in anterior insula and anterior cingulate cortex in response to others' pain. It is not thought that activity in these regions literally causes the perceiver to feel somatosensory pain; rather, internal simulation permits the perceiver to gain an understanding of the other's experiences (Decety, 2010; Bernhardt and Singer, 2012). Of course, under certain circumstances, such as a perceiver being highly anxious or a fearful expression being seen in a dangerous context, the sight of someone else's fear could cause genuine emotional contagion, but this would not be typical or necessary.

Third, the findings discussed above demonstrate that empathy is not a monolithic phenomenon (Blair, 2008; Olderbak et al., 2014). It is commonly understood that empathy should at the very least be divided into "cognitive empathy" and "affective empathy" (Dvash and Shamay-Tsoory, 2014). Cognitive empathy is sometimes referred to as *theory of mind* or *perspective taking* and refers to the ability to infer others' goals, beliefs, and intentions (Brune and Brune-Cohrs, 2006). This is the kind of

empathy that is impaired in autism spectrum disorders, and it is not strongly related to affective empathy, which is the ability to understand others' emotional states (Blair, 2008). However, as data from lesion patients show, affective empathy can itself be decomposed into empathy for multiple different emotional states that result from dissociable neural processes (Olderbak et al., 2014). Low-level empathic responses to fear are dissociable from empathic responses to pain (Decety et al., 2015) and from responses to emotions such as disgust or anger (Goldman and Sripada, 2005). Finally, none of these forms of empathy should be confused with empathic concern (i.e., sympathy or compassion), which entails the desire to alleviate someone else's distressed emotional state (de Waal, 2009; Batson, 2010). Empathic concern often *follows* affective empathy, but it is not *synonymous* with affective empathy (Marsh, 2013; Decety et al., 2015). It is perfectly possible, in theory, to apprehend correctly that someone is experiencing pain or fear without experiencing concern for the person or wishing to alleviate that state.

EMPATHIC RESPONSES TO FEAR

The hypothesis that the amygdala response to facial, postural, or vocal expressions of fear reflects empathic fear is not incompatible with other theories about what amygdala responses to fearful expressions signify. However, this theory provides explanatory value that these models do not provide. It is the model that best explains why damage to the amygdala could impair both the subjective experience of fear and the ability to recognize others' fear across multiple modalities. Second, it has the useful feature of reframing fearful facial expressions as something other than "threat cues," aversive stimuli that serve only to signal perceivers to be on alert for danger or that serve to punish perceivers' misbehavior. These conceptualizations of fearful expressions would require these expressions to elicit behavioral avoidance, whereas they in fact elicit predominantly behavioral approach (Marsh et al., 2005b; Hammer and Marsh, 2015).

What could explain the seemingly paradoxical finding that an expression that conveys the negative emotion of fear is viewed as appetitive? A consideration of the function of expressive fear behaviors in other group-living social species may be useful. In species such as primates, wolves, and dogs, expressive fear behaviors tend to consist of similar types of nonverbal behaviors, such as crouching, rolling over, and (in canines) holding the ears and tail close to the body, all of which make the expresser appear smaller and more vulnerable (Schenkel, 1967; Preuschoft and van Hooff, 1995; Eibl-Eibesfeldt, 1996). These cues create a striking visual contrast with cues that signal dominance and readiness to attack, which include erect posture, piloerection, and holding the ears and tail high making the expresser appear larger and fiercer (Schenkel, 1967; Marsh et al., 2009). The purpose of expressive fear displays is appeasement, to signal that the expresser means no harm and to inhibit others' aggression. Fear displays may accomplish this goal in part because cues such as small size, vulnerable posture, and flattened ears and tail heighten the expresser's resemblance to an infant or juvenile of the species (Schenkel, 1967). Infantile features naturally inhibit aggression and elicit caring behavior among group-living social species (Eibl-Eibesfeldt, 1996).

Human fearful expressions may serve a function similar to that of fear displays in other species. Fearful facial expressions are characterized by wide eyes, raised brows, a flattened brow ridge, and a grimace that rounds the appearance of the mouth and jaw. These movements alter the proportions of a face to appear less aggressive (Marsh et al., 2014a) and increase resemblance to an infant's face, which features large eyes, high eyebrows, a flat brow, and a rounded appearance (Marsh et al., 2005a; Sacco and Hugenberg, 2009). People expressing fear are judged to be more babyish and less aggressive, even when the expression is distorted so that it cannot be recognized as conveying fear (Marsh et al., 2005a). Together these data suggest that fearful facial expressions may derive their appeasing, appetitive properties from their perceptual and conceptual associations with infantile features (Hammer and Marsh, 2015).

PREDICTING ANTISOCIALITY AND ALTRUISM FROM RESPONSES TO FEAR

In assuming that fearful facial expressions serve an appeasement function in social encounters, the accurate perception of fear should be inversely associated with aggression and positively associated with compassionate and altruistic behaviors. Recent evidence suggests that this is the case. Studying aggression and altruism in the laboratory is difficult both for ethical reasons and because these behaviors are subject to strong demand characteristics, but these difficulties can be avoided by identifying special populations of individuals who engage in real-world aggression or altruism and assessing how they differ neurocognitively from controls. Evidence from both highly antisocial populations and highly altruistic populations suggests that sensitivity to others' fear may be a strong predictor of compassionate, caring behavior, or its absence (Marsh et al., 2007, 2014b; Marsh and Blair, 2008; Lozier et al., 2014a).

Psychopathy

Psychopathy is a useful phenomenon for understanding the neurocognitive basis of aggression and antisocial behaviors. Psychopathy is a personality variable that represents the co-occurrence of affective and interpersonal deficits, such as reduced empathy, reduced remorse, and manipulativeness, and antisocial behaviors, such as impulsiveness and poor anger control (Frick and Ray, 2014; Seara-Cardoso and Viding, 2014; Venables et al., 2015). The callousness and remorselessness captured by the affective and interpersonal component (commonly referred to as *callous–unemotional traits*) most clearly distinguish psychopathic from other antisocial populations (Frick and White, 2008). Psychopathic traits typically

emerge early in development and are associated with a severe, persistent, and treatment-resistant course of antisocial and aggressive behavior (Pardini and Frick, 2013). Roughly 1–2% of the adult population is thought to exhibit high levels of psychopathy, but the percentage may be as high as 50% among violent offenders (Hare et al., 1993). Psychopathy particularly elevates the incidence of proactive aggression, which is directed at achieving an instrumental goal (Blair, 2001).

The results of three meta-analyses confirm that psychopathy impairs the ability to recognize others' fear (Marsh and Blair, 2008; Wilson et al., 2011; Dawel et al., 2012). Although the methods and the results of these analyses were not identical, they converged in their central findings. Wilson and colleagues (2011) found relatively weak facial expression recognition deficits in psychopathic populations, but the strongest deficits were observed for the recognition of fear and sadness. Marsh and Blair (2008) found that fear recognition was significantly more impaired than recognition of any other emotion in a variety of antisocial populations, including those high in psychopathy; recognition of most emotions, including disgust, anger, and happiness, was not affected. Dawel and colleagues (2012) conducted the most comprehensive of the three meta-analyses, examining emotion recognition across channels in adult and juvenile psychopathic populations. They found that the affective and interpersonal factor of psychopathy was associated with deficits only in recognizing others' fear. Together, these meta-analyses paint a relatively consistent picture in which psychopathy impairs recognition of others' fear more than other emotions.

Physiological and neuroimaging evidence suggests that this deficit may result from dysfunction in the amygdala. Peripherally, both adults and children with psychopathic or callous–unemotional traits exhibit reduced electrodermal responses to images of fear and related distress expressions (Blair, 1999; Blair and Cipolotti, 2000). This deficit implicates amygdala dysfunction because the electrodermal response to fear-relevant stimuli is coordinated by the amygdala via its efferent projections to the hypothalamus (Davis and Whalen, 2001). Neuroimaging evidence demonstrates that the amygdala is hypoactive in psychopathic individuals as they view fearful facial expressions. Multiple studies have found reduced amygdala responses to fearful expressions (but not other expressions) in psychopathic and callous–unemotional youths (Marsh et al., 2008; Jones et al., 2009; Viding et al., 2012; Lozier et al., 2014a; Sebastian et al., 2014) and adults (Dolan et al., 2009) relative to controls. Although these studies were not designed to identify the particular nuclei associated with these effects, deficits almost certainly involve the central nucleus (Moul et al., 2012). Structural imaging has produced evidence of significant amygdala malformations in psychopathy, including structural deformities to multiple nuclei, including the central nucleus, and decreased overall volume (Yang et al., 2009; Pardini et al., 2014; Vieira et al., 2015).

Some research supports the possibility that amygdala-dysfunction-based fear recognition deficits in psychopathy result from aberrant gaze patterns, in keeping with the theory that eye gaze links amygdala dysfunction to fear recognition deficits (Dadds et al., 2006, 2008). However, this approach faces the same difficulty as in the case of lesion patients in that psychopathy also consistently and specifically impairs recognition of fear from the voice, body postures, and semantic statements that evoke fear (Munoz, 2009; Dawel et al., 2012; Marsh and Cardinale, 2012). The attention to eyes hypothesis also struggles somewhat to account for the facts that psychopathic deficits in responding to fear persist even after controlling for attention (White et al., 2012) or when stimuli are presented preattentively (Sylvers et al., 2011). Moreover, it is unclear why, if aberrant gaze is the driving factor, specific fear recognition deficits are *not* observed in autism spectrum disorders, which profoundly affect social gaze (Lozier et al., 2014b).

An alternate explanation—that fear recognition deficits in psychopathy result from empathic fear deficits—is supported by the fact that fearlessness is a hallmark of this disorder. Low fear responding has long been recognized as characteristic of psychopaths relative both to other violent offenders and to the normal population (Cleckley, 1988; Lykken, 1995). Psychopathy is associated with multiple indices of reduced fear responding, including reduced fear-potentiated startle (Patrick, 1994; Rothemund et al., 2012), electrodermal responding (Herpertz et al., 2001), Pavlovian fear conditioning (Flor et al., 2002; Birbaumer et al., 2005), and subjectively experienced fear (Jones et al., 2010; Marsh et al., 2011). Together, these lines of evidence suggest that amygdala dysfunction impairs the ability to recognize fearful expressions in psychopathy by precluding the generation of an empathic fear response in this population. The results of two recent studies support the possibility that the psychopathic phenotype results from empathic fear deficits. In a neuroimaging study of adolescents, reduced amygdala responses to fearful expressions directly mediated the relationship between the affective and interpersonal dimension of psychopathy and proactive aggression (Lozier et al., 2014a). Also, a behavioral study of psychopathy in a community sample showed a direct relationship between (1) the inability to recognize what causes others to feel fear and (2) the belief that it is acceptable to engage in behaviors that cause others to feel fear (Marsh and Cardinale, 2012).

An obvious question suggested by these findings is why amygdala dysfunction and fear insensitivity are not also associated with antisociality or aggression in amygdala lesion patients. Amygdala lesions are associated with unusual social behaviors but not antisociality (Kennedy et al., 2009). The answer may be that only in the case of psychopathy are amygdala deficits developmental. Psychopathic traits are highly heritable (Viding et al., 2005; Tuvblad et al., 2014), and signs of fear insensitivity predictive of later aggression and criminality can be detected even in infancy and early childhood (Gao et al., 2010a,b;

Baker et al., 2013). In contrast, specific amygdala lesions in adults usually result either from surgery or from a progressive genetic condition that does not result in complete loss of amygdala tissue until adolescence or adulthood. In monkeys, the timing of amygdala lesions has been shown to be particularly important for shaping social behaviors (Amaral, 2003). In humans, the effects of moral socialization during childhood may withstand later loss of the amygdala.

Altruism

Psychopathy is now commonly understood to be not a discrete taxon but a continuum that varies across the population, with highly psychopathic individuals at one extreme end of the continuum (Edens et al., 2006; Guay et al., 2007). Of course, this conceptualization raises the question of "what lies at the other end?" If insensitivity to fear resulting from reduced amygdala functioning is associated with unusually antisocial behavior, could heightened sensitivity to fear be associated with enhanced amygdala functioning and prosocial behavior? Several recent studies suggest that this may be the case.

In the laboratory, improved recognition of fearful expressions is linked to prosocial behaviors, including enhanced mental state attribution and charitable giving (Corden et al., 2006; Marsh et al., 2007). Outside the laboratory, sensitivity to others' fear is even associated with rare and extraordinary altruistic behaviors, such as altruistic kidney donation. Altruistic kidney donation is a voluntary, costly behavior aimed at improving the welfare of an anonymous, unrelated other, thereby meeting the most stringent definitions of altruism (Batson, 2010). A recent brain imaging study of altruistic kidney donors linked this extreme form of altruism to sensitivity to others' fear (Marsh et al., 2014b). Structural imaging results indicated that, in contrast to psychopathy, the right amygdala of altruists was larger than that of controls. Functional neuroimaging results showed that altruists exhibited enhanced amygdala responses to fearful, but not angry or neutral, expressions and that this enhanced amygdala response corresponded to improved recognition of fearful expressions during neurocognitive testing.

These results are compatible with the conclusion that the ability to generate a strong empathic response to others' fear is an important prerequisite for experiencing empathic concern, which in turn promotes prosocial behaviors and altruism and inhibits aggression. Recent evidence suggests that this process might not be unique to humans (de Waal, 2012). The perception of distress in a conspecific promotes empathic responses and altruism even in rodents (Ben-Ami Bartal et al., 2011; Sato et al., 2015). Whether these responses are similarly reliant on an intact amygdala remains to be seen.

CONCLUSIONS

A psychopathic offender being tested for his recognition of emotional facial expressions by Viding (personal communication) knew he was doing badly. Toward the end of the testing session he found himself stumped by a fearful expression and eventually admitted, "I don't know what that expression is called, but I know that's what people look like right before I stab them." This remarkable statement underscores the nature of the relationship between empathy and the perception of others' fear.

In most individuals, the perception of another's fear from the face, voice, or body results in increased activity in the amygdala, a response that appears to be necessary for the ability to recognize the expression. The precise nature of this amygdala response has several possible interpretations, among them the theory that it reflects empathic fear, the generation of an internal representation of fear that can then be linked to another person's outward display of fear to aid in interpreting and responding appropriately to it. Individuals with damaged or dysfunctional amygdalae tend not to experience fear strongly themselves, particularly when the damage is located in the central nucleus of the amygdala. These individuals find themselves impaired in interpreting others' fear, perhaps resulting from their inability to generate an empathic fear response. When this dysfunction is developmental rather than incurred in adolescence or adulthood, it appears to have significant downstream effects on other aspects of empathy. Individuals such as the psychopath quoted above not only fail to recognize others' fear but seem to be unable to appreciate the moral seriousness of behaviors that cause others fear. They are also at risk for engaging in aggressive, antisocial, and criminal behaviors. In contrast, individuals with enhanced amygdala responses to others' fear may be not only better at recognizing fearful expressions but also unusually likely to engage in altruistic behavior. Together, these findings indicate the continuing importance of understanding the nature of the amygdala's role in critical social and affective processes.

CONFLICT OF INTEREST STATEMENT

The author has no conflicts of interest.

ROLE OF AUTHORS

The author takes full responsibility for the conceptualization and drafting of this Review.

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