11 Empathy and Compassion: A Cognitive Neuroscience Perspective

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A casual viewer might think that the *Star Wars* series focuses more on displays of weaponry than displays of empathy. But aficionados know that the core of the series is a phenomenon called the Force, which is described as binding all living beings together. Barry Benecke II is a *Star Wars* aficionado who exemplifies the Force unusually well. A frequent online contributor to a forum for *Star Wars* collectors, in 2008 Benecke read a post from another young collector named Josh Weisleberg in which he described his rapidly failing kidneys and need for a kidney transplant. Despite having never met Weisleberg, Benecke wrote him back to say he would give him a kidney. Why would the then-forty-five-year-old husband and father volunteer to have one of his internal organs removed and given to a young man he had never met? Benecke had recently lost a number of close friends and family members to cancer and recounted that, "It was because of losing those folks that I was compelled to try to help someone else. The first person who I found that I might be able to help was Josh" (Rosenberg 2010). In other words, Benecke's own distressing experiences influenced his response to another's distress. Although perhaps not *the* Force, an empathic experience like this is clearly a powerful force—one that moved Benecke to risk his own health to save the life of a stranger.

This chapter discusses the neural and psychological forces associated with empathy and the drive to help others in distress. In particular it focuses on recent cognitive neuroscience research that highlights the relation between empathy and the ability to detect and respond to distress in others. Neurocognitive research is discussed that has identified structures involved in empathizing with three distress-related emotions: disgust, pain, and fear. The focus of the chapter then turns to fear, a particularly effective elicitor of concerned or compassionate empathic responses. We conclude by considering how responses to fear cues may be particularly useful in understanding aspects of empathy such as its evolutionary basis and clinical disorders such as psychopathy that are marked by empathy deficits.

The Nature of Empathy

Empathy is a general term for an emotional response to another's emotional state and is sometimes described as "feeling with" another person (Eisenberg and Strayer 1987). Empathic emotional responses include both matching emotional responses—you feel sad, so I feel sad—and compatible responses—you feel pain, so I feel anxiety, or you feel anxiety so I feel compassion (Decety and Meyer 2008; de Waal 2008). Empathy's most basic precursor is the simple ability to detect what another person is feeling (Jackson, Meltzoff, and Decety 2005). Particularly when they are communicated through vivid nonverbal cues like emotional facial expressions or vocalizations, the emotions of others can typically be detected rapidly, perhaps even automatically (Whalen et al. 1998; Öhman 2002; Marsh and Ambady 2007). As such, empathy allows for quick and automatic awareness of others' internal states and may be crucial for regulating social interactions, coordinating behavior, and promoting cooperation among individuals (de Waal 2008).

Neuropsychological evidence indicates that it may be literally true that empathy involves "feeling with" another. A number of theoretical models suggest that when we observe another person's behavior, we make sense of it by activating our own representations of that behavior (Barsalou et al. 2003; Rizzolatti and Craighero 2004; Prinz 2006). This representational mapping appears to occur not only in response to instrumental behaviors such as kicking a ball but in response to emotional behaviors such as facial and body expressions, the observation of which may activate corresponding emotional representations in the observer (Preston and de Waal 2002). For several emotions the brain regions in which this emotional mapping is thought to occur have been identified. Evidence that expressions of disgust, pain, and fear are understood via emotional mapping has been described in detail elsewhere (Adolphs 2002; Goldman and Sripada 2005; Heberlein and Atkinson 2009) and will be summarized here.

Empathy for Disgust, Pain, and Fear

Disgust is an aversive emotional response to distasteful or offensive items (Rozin, Haidt, and McCauley 2000). The neural region most closely associated with the experience of disgust is the insula, a region of cortex underlying the temporal lobes that was identified by Penfield and colleagues. They stimulated the insulae of epileptic patients, who consequently reported experiencing nausea, roiling sensations in their abdomens, or disgusting odors or flavors (Penfield and Faulk 1955). In line with this, weakened experiences of disgust result from damage to the insula, as occurs in neurodegenerative disorders such as Huntington disease (Calder et al. 2000).

If recognizing disgust in others occurs via empathic emotional mapping, common regions should be active both when disgust is experienced and when it is perceived in someone else. This has been confirmed using functional magnetic resonance imaging (fMRI), which shows that regional blood flow to the anterior insula increases when a person experiences, or imagines experiencing, a foul odor or taste (Phillips et al. 1997, 1998). When subjects who experience or imagine tasting something disgusting subsequently witness another person sampling a disgusting fluid and looking disgusted, a striking correspondence in insula activity is observed (Wicker et al. 2003; Jabbi, Bastiaansen, and Keysers 2008). That this emotional mapping is critical to low-level aspects of empathy—like detecting the emotional states of others—is suggested by the fact that insula lesions dampen not only the experience of disgust but the ability to recognize when others are experiencing disgust. Insula lesions cause specific impairments in recognizing disgusted facial expressions (Calder et al. 2000; Adolphs, Tranel, and Damasio 2003; Hennenlotter et al. 2004).

Emotional mapping in the insula together with the anterior cingulate cortex also appears important for empathic experiences of pain. These regions seem primarily involved in the motivational and affective components of painful experiences (Rainville 2002; Jackson, Meltzoff, and Decety 2005). Akin to the case of disgust, both feeling pain and seeing a cue that signals that another person is experiencing pain produce changes in activation in the anterior insula as well as the anterior cingulate cortex (Singer et al. 2004). Work by Decety and colleagues has extended these findings, ascertaining that generating these mental representations of pain allows a person to reconstruct the rich associative network associated with the concept of pain (Jackson, Rainville, and Decety 2006).

That emotional mapping underlies empathy for others' affective experiences remains subject to debate. Discrete regions that subserve empathy for some basic emotions such as sadness and happiness have not been identified. And regions such as the insula are involved in empathic responses to multiple emotional states. This latter concern also applies to empathic fear responses, which appear to rely upon the amygdala. The amygdala is a subcortical structure composed of several nuclei that subserves a wide array of social and emotional functions, not all of which are relevant to fear (LeDoux 2003). However, the amygdala is more active during fear-related events than during other kinds of emotional event (Murphy, Nimmo-Smith, and Lawrence 2003). And accumulating evidence suggests that the amygdala is also important to empathic fear responses.

The amygdala appears to be involved in the generation of fear (Fredrikson and Furmark 2003; Feldman Barrett et al. 2007). Localized damage to the amygdala tends to result in reduced fear responding, as is the case for a patient known as SM. She carries a rare genetic disorder called Urbach-Wiethe that has destroyed her

amygdalae bilaterally. Damasio has reported that this individual "Does not experience fear in the same way you or I would in a situation that would normally induce it" (Damasio 1999, 66). Although she is intellectually aware of what fear is and what causes it, her own daily emotional responses are unusually fearless, and she does not exhibit signs of sympathetic arousal during, for example, fear conditioning trials (Bechara et al. 1995). Experimenters working with SM recently attempted to induce fear in her by taking her to a pet store to handle live snakes and to a purportedly haunted house. SM responded to both experiences was intense curiosity and interest, showing no evidence of fear or avoidance in either situation (Feinstein et al. 2011).

If empathic emotional mapping is required to empathize, we would expect amygdala damage to impair the ability to recognize fear in others. Research with SM has found this to be the case. In keeping with the amygdala's many functions, SM shows a variety of impairments related to processing emotion, but her emotion *recognition* impairments are specific to fear (Adolphs et al. 1994). Another amygdala-damaged patient has also been shown to have difficulty recognizing fear conveyed through body postures and sounds as well (Sprengelmeyer et al. 1999). A large study of nine patients with bilateral amygdala damage also found recognition of fearful facial expressions to be significantly impaired (Adolphs et al. 1999). Some of these patients are also impaired in recognizing other emotions. However, with the exception of SM, these other patients sustained extensive damage in regions other than the amygdala, which may be related to their broader emotional deficits. On the opposite end of the spectrum, a large study recently demonstrated that individuals who report experiencing fear most strongly in their own life are best able to identify fearful expressions in others (Buchanan, Bibas, and Adolphs 2010).

Empathy and Simulation

Evidence from neurocognitive studies of disgust, pain, and fear provide the basis for simulation-based explanations of emotion recognition (Goldman and Sripada 2005; Heberlein and Atkinson 2009). Simulation-based models specify that the ability to recognize certain emotions in nonverbal cues such as facial expressions requires observers to be able to simulate the same emotional state in themselves (Heberlein and Atkinson 2009). Simulation theories propose that observers generate emotional attributions by replicating or reproducing in their own mind the same state as an expresser or by attempting to do so (Goldman and Sripada 2005). When circuits that are involved in the experience of comparable emotions are reactivated, this may then allow the observer to retrieve knowledge that links nonverbal cues like facial expressions to the appropriate emotion (Adolphs et al. 2000). Damage to regions like the insula or the amygdala may impair the ability to generate simulated hedonic

experiences of, respectively, fear or disgust. Such damage may also thereby impair the ability to recognize these emotions in others' emotional facial expressions

It should be noted that simulation theories have been criticized as insufficiently specific (Zahavi 2008). Zahavi argues that the term "simulation" suggests that the entirety of another's emotional experience is recaptured by the perceiver, which is clearly not the case, as this would lead to simple emotional contagion (e.g., witnessing you disgusted would cause me to actually feel disgusted, rather than simply to recognize your emotional state). Although the recognition of others' emotional expressions is likely associated with activation of some the neural structures involved in emotional experience, emotion recognition probably does not involve the entire network of structures involved in emotional experience (Jabbi, Bastiaansen, and Keysers 2008). Activation in a more limited set of structures is unlikely to capture the entirety of the experience captured by words like "disgust" and "fear," so Zahavi has suggested the term *resonance* to describe the neural-level processes that occur during the perception of others' emotions.

Assuming the basic validity of simulation theories, why does empathy matter? What is the functional significance of being able (or not being able) to create representations of others' emotional states in order to recognize them and respond appropriately? As described above, empathy is thought to be integral to the coordination of various adaptive social behaviors. Empathy also seems essential for moral development (Decety and Meyer 2008). Many have argued that empathy is the primary motivational force behind altruistic behavior (Hoffman 1981; Batson 1990). The form of empathy most conceptually related to altruism and other prosocial behaviors is sympathy, generally considered to be a sorrowful or concerned emotional response to another's distress or need (de Waal 2008). Empirically, do the data suggest that empathic emotional responses are associated with sympathetic concern, moral behavior, and altruism? The answer appears to be yes, but only clearly for one emotion: fear.

Fear Simulation and Compassion

Among the basic emotions, fear is the most vivid communicator of distress. Fear is associated with high arousal and an intensely negative hedonic state. It signifies that harm is impending, unlike sadness, which is generally associated with an unpleasant event that has already happened. It has been suggested that the ability to accurately represent others' distress, particularly fear, is essentially the *only* neurocognitive requirement for generating sympathetic concern (Nichols 2001). Although other empathic abilities, such as cognitive perspective-taking, may be important for devising sophisticated responses to others' distress, perspective-taking abilities and theory of mind do not seem crucial for generating sympathetic concern or compassion. This is suggested by the fact that very young children and autistic children who do not

possess sophisticated perspective-taking abilities nevertheless demonstrate basic compassion in response to others' distress (Sigman et al. 1992).

Studies of psychopathy also provide evidence that the ability to generate representations of fear is associated with sympathy. Psychopathy is a disorder characterized by a lack of remorse or compassion, a tendency to use and manipulate other people, and antisocial behaviors (Hare 1991). Psychopaths are also specifically impaired in recognizing fearful facial expressions. A recent meta-analysis concluded that highly antisocial individuals, including psychopaths, are significantly more impaired in recognizing fearful facial expressions than any other expression (Marsh and Blair 2008). This effect is not attributable to the difficulty of recognizing fearful expression and is not associated with gender or age.

Psychopathy also provides evidence that recognizing emotional facial expressions may result from simulation. Psychopathy has long been associated with a fearless temperament (Lykken 1957; Fowles 2000; van Honk and Schutter 2006). Cleckley's original conceptualization of psychopathy suggested that "Within himself [the psychopath] appears almost as incapable of anxiety as of profound remorse" (Cleckley 1988, 340). Laboratory studies measuring physiological fear responses confirm this. In typical study participants, threats like an impending electrical shock unsurprisingly provoke signs of sympathetic arousal that are associated with fear—sweat on the palms, increased heart rate, increased respiration, and increased blood pressure. Impending threats also exaggerate a person's startle responses. But in similar circumstances psychopaths show reduced sympathetic arousal and reduced startle responses (Levenston et al. 2000). Psychopathic individuals also report attenuated subjective experiences of fear in response to frightening real-life events (Marsh et al. 2010).

Simulation theories predict that someone with diminished experiences of fear and trouble identifying fear in others would exhibit defects in the neural structures that underlie fear responding (Goldman and Sripada 2005). Assuming that the amygdala is integral to fear, this appears to be the case for psychopathy. Functional neuroimaging studies show dysfunction in amygdala activation patterns in psychopathic individuals when they view stimuli that normally elicit fear (Birbaumer et al. 2005; Finger et al. 2008). And anatomical studies have identified structural abnormalities in the amygdalae of psychopaths and in the fibers connecting the amygdala and orbitofrontal cortex (Craig et al. 2009; Yang et al. 2009). This latter finding is interesting in light of the purported role of the orbitofrontal cortex in generating emotional experience (Damasio 2000).

Amygdala dysfunction is also observed in psychopathic subjects when they view fearful facial expressions. Whereas most healthy adults exhibit a greater amygdala response to fearful facial expressions than to any other expression, psychopaths fail to show this response pattern (Marsh et al. 2008; Jones et al. 2009; Dolan and Fullam, 2009). And evidence that psychopaths show reduced electrodermal responses when

viewing fearful facial expressions could be interpreted as signifying that psychopaths fail to generate an empathic fear response to others' expressions of fear (Blair et al. 1997). In summary, psychopaths have fearless temperaments, fail to recognize fear in others, and lack sympathy or compassion. A parsimonious explanation for this pattern is that psychopaths do not exhibit the increased amygdala activation and sympathetic nervous system activation in response to fearful expressions that indicate empathic simulation and that would enable emotion recognition.

Empirical evidence also directly links compassionate behavior to the ability to identify fear in others. A recent series of studies assessed the relation between facial expression recognition and altruistic behavior using a classic paradigm developed by Batson and colleagues (Coke, Batson, and McDavis 1978). In this paradigm participants heard a recording featuring an ostensibly real woman named Katie Banks as she described the recent loss of her parents in a car accident and her struggle to raise her young siblings. At the end of the experiment participants were given the option to pledge their time or money anonymously to help Katie if they chose. During the experiment participants also completed a measure of facial expression recognition. The best predictor of participants' pledges of time and money to Katie was their ability to recognize fearful facial expressions. Another experiment in this series found fear recognition to predict prosocial behavior better than gender, mood, responses on a self-reported empathy scale, or the recognition of other emotional facial expressions (Marsh, Kozak, and Ambady 2007). Thus, the evidence is reasonably strong that the ability to detect fear in others is associated with sympathetic concern. People who are particularly good at detecting fear in others seem especially compassionate, and people who are poor at this are more likely to be antisocial, even psychopathic.

Why would empathy for fear in particular be so important for the elicitation of compassionate behavior? In general, accurately perceiving another person to be in distress prompts helping behavior (Clark and Word 1974). And fear expressions are vivid and urgent signifiers of distress, perhaps more so than related expressions like sadness. In addition, perceptual properties of the nonverbal cues associated with fear may be particularly likely to elicit helping behavior. Fearful facial expressions, for example, possess appearance features similar to that of an infantile face, such as high brows, large, round eyes, a flattened brow ridge, and rounded features. Fearful facial expressions and infantile faces also elicit similar attributions, including dependency, warmth, and youth (Marsh, Adams, and Kleck 2005).

In this sense, fearful facial expressions are analogous to distress cues displayed by other social mammals, which often mimic infantile cues (Lorenz 1966). For example, the subordinate wolf in an aggressive encounter will adopt cues that make him appear more puppyish, including folding his ears back, rolling onto his back, whining, and licking the other wolf's jaws. And the high-pitched distress vocalizations that adult humans and other mammals make when distressed appear to be retained from infancy

(de Waal 2008). By mimicking the characteristics of an infant, expressions of fear may thereby generate the nonaggressive, protective responses that actual infants usually elicit in adults. The neural correlates of human responses to infants are not yet well understood, but likely they include subcortical structures involved in parental care, such as the periacqueductal gray and the oxytocin-producing hypothalamus (Lonstein and Stern 1997; Numan 2006).

Would the detection of fear in others always lead to a compassionate response? One could generate an alternative scenario under which expressions of fear, because they signal weakness and helplessness, would lead to attack behavior. This may well be the case among species that are not reliant on the formation of strong social bonds for survival. But among social carnivores, adaptive success is associated with adults forming social groups in which the adults mutually care for the young and helpless, resulting in "fitness interdependent" group members (Brown and Brown 2006). In species like these it may be adaptive for adults to have developed a general set of responses to infantile features (Zebrowitz 1997). Simmons (1991) has suggested empathy developed as a mechanism that prompts caregivers to recognize distressed infants' needs, and that this response generalizes to others in need and results in a caregiver's desire to help them as well. That human adults respond to a variety of stimuli that look vaguely infantile is well established (Zebrowitz 1997). If fearful expressions are also infantile in appearance, this may explain why the ability to process these cues is associated with a tendency to provide care and refrain from aggression.

Unanswered Questions

Thus far, the evidence seems reasonably strong that empathy for others' fear is a good predictor of compassionate behavior and that the ability to empathize with others' fear relies on the intact functioning of neural structures involved in generating a fear response, particularly the amygdala. However, many questions remain unanswered.

In particular a complete understanding of the neural mechanisms that underlie empathy and compassion is still lacking. Although amygdala activation may be necessary to generate sympathetic concern, it is certainly not sufficient. Prior research has highlighted the role of other regions involved in recognizing and responding to emotion in others. Right somatosensory-related cortex may play an important role, given that it is involved in the general recognition of emotion (Adolphs et al. 2000) and that activation in this region has been linked to self-reported altruism (Adolphs et al. 2000; Tankersley, Stowe, and Huettel 2007). This region may be important for generating the somatic sensation associated with emotional states; when this ability is impaired, the recognition of emotion in faces is impaired as well (Adolphs et al.

2000). Another critical region may be the inferior frontal operculum, which has also been linked to the simulation of bodily feeling states during social cognition (Jabbi, Bastiaansen, and Keysers 2008). Better understanding of the role of activation in these structures and functional connectivity among them is vital to a complete understanding of empathic responding (Jabbi, Bastiaansen, and Keysers 2008).

The mechanisms underlying empathy for other distress-related emotional states such as pain and sadness also merit further exploration. Identifying conditions that reduce the experience of these emotions may illuminate their role in fostering compassionate social behavior. Rare congenital syndromes exist that result in insensitivity to pain caused by external stimulation. However, data regarding empathy in this population are ambiguous. Adults with this syndrome underestimate others' pain when emotional cues are lacking, but they can accurately recognize painful facial expressions (Danziger, Prkachin, and Willer 2006). They also show normal insula and anterior cingulate cortex activation in response to images of hands and feet being injured but significantly less anterior cingulate cortex activation than controls in response to pained facial expressions (Danziger, Faillenot, and Peyron 2009). However, individuals with congenital pain insensitivity can still experience pain due to internal causes such as spontaneous electrical discharges or migraine headaches (Danziger, Prkachin, and Willer 2006). Additionally, they experience psychological pain, which activates similar neural circuits as physical pain (Eisenberger, Lieberman, and Williams 2003). It is not known whether individuals with congenital pain insensitivity are unusually likely to inflict pain on other people.

It is not clear whether any population exists in which the experience of other distress-related emotions like that of sadness is impaired. Evidence that this is the case in psychopaths is mixed. Finally, it should be noted that the same large study that found an association between fear experience and recognition found no equivalent association for some other emotions, including surprise (Buchanan, Bibas, and Adolphs 2010). This suggests that simulation may be a more important mechanism for the recognition of some emotions than others.

Another question to be addressed is the role of dynamic social behaviors such as eye gaze in responding to fearful facial expressions. The eye region is the region of the face most important to recognizing fear (Adolphs et al. 2005). Convergent evidence suggests that the amygdala is involved in directing gaze toward the eye region of fearful faces. Patient SM's impaired fear recognition improves when she is directed to focus on the eye region of emotions facial expressions (Adolphs et al. 2005). This technique also improves fear recognition in children with psychopathic traits (Dadds et al. 2006). This accords with other evidence that redirecting attention to distress cues may be an effective means of improving social processing in individuals with psychopathic traits (van Baardewijk et al. 2009). Perhaps the amygdala's role in fear recognition includes responding to the presence of certain facial features, such as the

eyes, and directing attention to further processing of those features (Heberlein and Atkinson 2009).

Conclusions

Psychologists, philosophers, economists, and biologists have argued for centuries whether humans can be moved to genuinely care for the welfare of family members, friends, or even strangers. Convincingly explaining the behavior of individuals like *Star Wars* aficionado and kidney donor Barry Benecke, who risked his health to help a stranger in a display of seemingly genuine altruism, is difficult enough to make even supernatural forces sound plausible. But recent neurocognitive research has begun to reveal the neural mechanisms that underlie empathy. The systems that generate the experience of emotion appear to respond to equivalent emotions in others, which allows emotion recognition, the most fundamental form of empathy, to occur. When the neural systems responsible for generating one particular emotion—fear—are intact, the empathic response may result in sympathetic concern and, in some cases, altruism. The breakdown of this process is associated with psychopathy. This suggests that the typical person may be equipped to respond empathically to the distress of others. It remains for ongoing research to determine how best to foster and enhance this response.

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