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7

UNDERSTANDING EMPATHY AND PSYCHOPATHY THROUGH COGNITIVE AND SOCIAL NEUROSCIENCE

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Social relationships between humans and domesticated animals can often be mutually beneficial and affectionate. Humans and domestic animals provide each other with various instrumental benefits, including food provision, protection, and play. But humans' relationships with domesticated animals are not simply instrumental. Interactions of humans with their pets, and even with unfamiliar animals, are often marked by warmth, affection, and empathy. The capacity for humans to exhibit empathy toward nonhuman animals represents something of a puzzle. *Empathy*, a term that refers to a constellation of phenomena, including the capacity to know or understand the contents of another individual's mind and the capacity to care about another individual's well-being (de Waal, 2008; Decety, 2015), is generally believed to be more commonly experienced toward similar individuals than toward dissimilar individuals (Krebs, 1975). That humans can experience empathy for nonhuman animals that are extremely unlike

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themselves indicates that important dynamics other than similarity must be in play. Considering the possibility of empathy across species may aid in understanding both the dynamics of empathy and of human–animal interactions (HAIs). This chapter considers the various forms of empathy, explores the neural bases of those forms most relevant to compassionate social behavior within and across species, and discusses the consequences that may result from empathy impairments.

The study of empathy has become increasingly popular among psychology and neuroscience researchers. However, research on empathy is often hampered by the use of the term *empathy* to describe a variety of overlapping but distinct phenomena, including cognitive perspective taking, sympathetic concern, and emotional contagion. An exploration of empathy as it relates to important behavioral phenomena, including aggression, altruism, and HAIs, requires that care be taken to distinguish among these phenomena.

Cognitive perspective taking (sometimes called *theory of mind*) is the most cognitively complex form of empathy (de Waal, 2008). Perspective taking, the process of acquiring insight into another person's intentions, desires, and beliefs via attempts to infer or adopt his or her point of view (Baron-Cohen, 1997), is often described colloquially as putting oneself into another's shoes. It is typically achieved by considering prior knowledge of the person and his or her current context. For example, if I know that a friend is hoping to adopt a companion dog and I am in the room when he takes a call from the agency handling rescue adoptions, I can infer that he will not want me to make any noise or disturb him while he takes the call. The sophisticated processes that compose perspective taking are essential for many higher level social functions, such as understanding deception (Hala, Chandler, & Fritz, 1991). Impaired perspective taking characterizes some neurodevelopmental disorders that result in profound social functioning deficits, most notably schizophrenia and autism (Crespi & Badcock, 2008).

Sympathetic concern is the form of empathy typically associated with feelings of sorrow, compassion, or pity for the suffering of another being (de Waal, 2008; Nichols, 2001). For example, if a friend or your dog were injured and showing signs of distress, you might experience sympathetic concern as a result. Sympathetic concern entails two distinct phenomena: being aware of another person's distress and desiring to help ameliorate the distress. It is particularly this latter element—the desire to help—that is difficult to capture using currently available methods. In general, self-report is the predominant means of assessing sympathetic concern. Self-report is an essential means of acquiring psychological information, but this method has a variety of limitations and shortcomings that often must be mitigated by, for example, enabling anonymous responding, or

by supplementing self-report with complementary information-gathering strategies such psychophysiological or neurophysiological measurements (Brener, Billy, & Grady, 2003).

Alternatively, some of the difficulties inherent in measuring sympathetic concern can be ameliorated by focusing on the first component of this phenomenon: the simple awareness or detection of another person's distress. This most basic form of empathy is sometimes referred to as *emotional contagion*, a term that suggests emotions may spread among individuals through very low-level processes outside of conscious awareness (de Waal, 2008). Because it is relatively simple, this is a form of empathy that is much more amenable to multiple types of measurement. The detection of another person's distress can be measured by asking study participants to label emotion expressed in another's face, voice, or body or by using psychophysiological or neurophysiological measurements to identify changes in the participant's nervous system that indicate detection of another's distress. So, for example, we could infer that you experienced emotional contagion in response to a human or a dog who was injured if you were able to correctly label that individual's nonverbal cues as signs of distress or if viewing these cues caused you to exhibit changes in autonomic arousal such as alterations in heart rate or respiration.

RESPONDING TO DISTRESS

Responses to nonverbal distress cues are particularly illuminating for the study of empathy, as they appear to promote both emotional contagion and sympathetic concern and therefore enhance affiliative, prosocial responses. For example, fearful facial expressions, which convey acute and urgent distress, elicit self-reported sympathetic concern from perceivers (Marsh & Ambady, 2007) as well as promoting affiliative behaviors such as behavioral approach. Approach can be measured using a lever and comparing the response times required to push versus pull the lever in response to the presentation of a stimulus (Hammer & Marsh, 2015; Marsh, Ambady, & Kleck, 2005). That fearful expressions consistently elicit more rapid pulling (approach) than pushing (avoidance) is consistent with their being perceived as appetitive stimuli, despite fear typically being considered a "negative" emotion (Izard, 2007).

To understand why fearful facial expressions and other distress cues elicit sympathetic concern and behavioral approach, it may help to consider how fear-related behaviors are used in other social species, such as wolves and domestic dogs. Species-specific fear behaviors displayed by wolves and dogs when they anticipate attack from another wolf or dog

include crouching, tucking the tail, pinning back the ears, licking the antagonist's jowls, and rolling on the back (Schenkel, 1967). The primary purpose of these cues is to signify submission—that the expresser does not represent a threat or competition to the antagonist (Schenkel, 1967; Smith & Price, 1973). These stereotyped behaviors are thought to serve this function by making the fearful, submissive wolf or dog appear smaller and more helpless by mimicking appearance cues of pups (Schenkel, 1967). In fact, across multiple social species, it is thought that fear and submission cues mimic infantile cues because adults typically inhibit aggression toward juveniles (Lorenz, 1966).

Considering fear across species permits the generation of a hypothesis for the social function served by human expressions of fear: to appear submissive or infantile, thereby inhibiting aggression or eliciting help from perceivers. This hypothesis is consistent with findings that fearful expressions appear affiliative, as a babyish facial appearance is consistently perceived as highly affiliative (Zebrowitz & Montepare, 1992), and with the fact that fearful expressions elicit approach, as this is a behavioral response associated with protective behaviors like nurturing the young (Burgdorf & Panksepp, 2006). It is also consistent with findings that fearful expressions are perceived as looking babyish by observers, even when the expressions are manipulated to prevent observers from identifying the emotion being expressed (Marsh, Adams, & Kleck, 2005). This finding suggests that the appearance of the features in a fearful face—the widened eyes, raised brows, flattened brow ridge—makes the expresser appear more babyish physically. Fearful expressions may serve appeasing functions by eliciting the same responses that people have toward infants, just as fear and submission cues in wolves and other social species do (Hammer & Marsh, 2015).

Together, these lines of evidence may help clarify why normal responses to fear are associated with empathy, as has been demonstrated by several paradigms. People report experiencing high levels of sympathetic concern in response to fearful faces, even if the fearful faces are presented subliminally and observers have no conscious awareness of having seen them (Marsh & Ambady, 2007). Moreover, the extent to which people feel sympathetic concern in response to others' fearful expressions is associated with how sympathetic those people are in general. People who recognize fearful expressions better are more likely to respond altruistically in response to others' distress (Marsh, Kozak, & Ambady, 2007). It has been theorized that perhaps the most important predictor of the capacity for sympathetic concern in response to others' distress is the simple capacity to correctly recognize others' distress (Nichols, 2001). This capacity may extend to concern for other species' distress as well; in one recent study, approach behavior was consistently observed in mother deer exposed to the distress vocalizations of several species of mammalian infants, including human infants (Lingle & Riede, 2014).

THE NEURAL BASIS OF EMPATHY

An understanding of the neural processes that underlie any psychological phenomenon can be acquired in a variety of ways. Neural functioning in healthy and atypical brains can be measured and compared, an approach that has been enabled by the development of novel neuroimaging techniques in recent decades. Human lesion cases can also be used to investigate neurocognitive deficits that arise following injury to specific structures. For example, it has been shown that deficits directly relevant to impaired empathic functioning, including impaired recognition of and responses to distress cues, arise in individuals with damage to the amygdala.

The amygdala is a subcortical structure located within the temporal lobe that comprises several subnuclei. The lateral and basolateral nuclei of the amygdala receive sensory information via the thalamus and cortex, and relay this information to the central nucleus of the amygdala. The central nucleus projects to other subcortical regions, including the lateral hypothalamus and regions of the brain stem, which directly mediate physiological and behavioral aspects of fear responding, such as changes in heart rate, skin conductance, and freezing or flight behavior (Davis & Whalen, 2001). Reciprocal connections between the nuclei of the amygdala and the orbitofrontal cortex, hippocampus, and striatum mediate other processes central to fear responding, including fear learning and memory and instrumental approach or avoidance behavior (Davis & Whalen, 2001; LeDoux, 2003; Maren, 2001). That intact amygdala function underlies fearful responding to incipient threats has been established from numerous animal studies—in, for example, rats, rabbits, cats, and dogs—documenting reduced fear responses following amygdala damage and heightened fear responses following amygdala stimulation (Davis, 1992; Goddard, 1964).

More recent research on humans with focal amygdala lesions has established that the amygdala is important for the recognition of others' fear responses as well. One of the best-studied lesion patients is SM (Adolphs, Tranel, Damasio, & Damasio, 1994), a woman with Urbach-Wiethe disease that caused selective and almost complete calcification of her amygdalae. Repeated testing found SM to exhibit marked deficits in recognizing fearful facial expressions, although her recognition of other emotional expressions like happiness, sadness, and disgust was largely intact. By contrast, she showed no impairment in other aspects of face processing, such as recognition of facial identity (Adolphs et al., 1994).

Although very few individuals show both complete and selective amygdala damage akin to SM's, research on other patients with lesions that include the amygdala, either unilaterally or bilaterally, generally has confirmed that individuals with amygdala lesions are impaired in recognizing fear expressed via the face, body, or voice (Adolphs et al., 1999; Scott et al., 1997; Sprengelmeyer et al., 1999).

PSYCHOPATHY AS A CLINICAL MODEL OF IMPAIRED EMPATHY

Amygdala dysfunction may be still more pernicious when it is developmental. Whereas most adults with amygdala lesions acquired these lesions in adolescence or adulthood, some developmental disorders also impair the functioning of the amygdala. One notable example of such a disorder is psychopathy. This disorder is of particular importance to understanding empathy because of the suite of impairments typically observed among individuals with psychopathic personality traits, which include impaired amygdala functioning; impaired ability to recognize and respond to nonverbal distress cues, such as fearful facial expressions; and persistent and severe antisocial behavior, including aggression toward both humans and nonhuman animals.

Psychopathy is characterized by emotional deficits such as shallow affect; difficulty forming close bonds with others; a lack of remorse or empathy; a parasitic orientation toward others; irresponsible and disinhibited behavior; and goal-directed aggression, such as threats, intimidation, and physical violence (Hare, 1991; Skeem, Polaschek, Patrick, & Lilienfeld, 2011). Accumulating social neuroscience literature links psychopathy to characteristic abnormalities in neural structure and functioning in both clinical and community samples (Blair, Finger, & Marsh, 2009).

These neural abnormalities may also represent a risk factor for cruelty toward nonhuman animals, which may be a specific early indicator of psychopathy (Dadds, Whiting, & Hawes, 2006). Animal abuse has long been associated with patterns of generally disruptive and violent behavior, such as violence toward family members. In one recent notorious case, a 19-year-old Washington State man responded to his mother's threat to cut off his allowance by butchering his family's two dogs, then locking his mother and grandmother inside their home, describing what he had done to the dogs, and threatening to kill his mother and grandmother as well unless they agreed to keep paying his allowance (Cuniff, 2010). Individuals who engage in this type of behavior are at risk for more serious behavior disruptions and worse long-term outcomes than are youths with conduct problems who do not abuse nonhuman animals (Luk, Staiger, Wong, & Mathai, 1999). This suggests that

cruelty toward nonhuman animals may result from risk factors beyond the family conflicts and parenting problems that place children at risk for generally disruptive behavior. One recent study suggests that psychopathy may be a key factor in the emergence of animal cruelty (Dadds et al., 2006). In this study, affective traits associated with psychopathy, such as reduced empathy, predicted animal abuse better than overall levels of aggressive behavior or levels of family conflict. This suggests that animal cruelty may represent an early indicator that may later manifest as aggression and violence toward humans. Ultimately, then, a more complete understanding of psychopathy may be important for understanding animal abuse as well as for understanding empathy more generally.

Psychopathy is not associated with impairments in all forms of empathy. Individuals who are psychopathic frequently show intact perspective taking (Blair, 2008b), suggesting that perspective taking (or lack thereof) may not be tightly linked to callousness or the types of aggression most closely linked to psychopathy. De Waal (2008) suggested as much, proposing that, "Without emotional engagement . . . perspective taking would be a cold phenomenon that could just as easily lead to torture as to helping" (p. 287). To take the earlier example, simply knowing that a friend will not want you to disturb him may or may not motivate you to act as he wishes. Inferring the nature of others' internal states is not identical to caring about whether those states are good or bad. Indeed, psychopathic individuals may rely on intact perspective-taking abilities to con, manipulate, and otherwise take advantage of others.

By contrast, responses to others' distress, which as mentioned previously are indicative of the low-level form of empathy known as emotional contagion, are unquestionably deficient in psychopathy. In response to depictions of others' distress, individuals with psychopathic traits are less likely to show psychophysiological or neurophysiological changes (Blair, 1999) associated with emotion perception than typical individuals (Blair, 1999; Jones, Laurens, Herba, Barker, & Viding, 2009; Marsh et al., 2008; White et al., 2012). They are also impaired in recognizing distress expressed via the face, body, or voice (Marsh & Blair, 2008; Muñoz, 2009). Moreover, accumulating data suggest that empathic deficits in psychopathy are particularly evident in response to fearful facial expressions. A meta-analysis by Marsh and Blair (2008) found largely intact facial expression recognition abilities for emotions like anger, disgust, happiness, and surprise in participants who were psychopathic or otherwise antisocial, but found that these individuals were roughly 20% worse than control participants at recognizing facial expressions of fear. In other words, the ability to simply recognize others' distress, perhaps the most fundamental form of empathy, is compromised in psychopathy.

EMPATHY AND RESPONSES TO FEAR

Returning to a consideration of the role of distress cues in social interactions may help to illuminate the mechanisms by which psychopathy is associated with aggression toward humans and nonhuman animals. Distress cues appear to function across many species, humans included, to promote affiliative, prosocial behavior, and sympathetic concern. That individuals with psychopathy cannot reliably recognize these cues and show aberrant physiological responses to them suggests dysfunctional processing of distress cues in psychopathy, which may impair empathic responses to others' distress (Blair, 1999; 2008b). This may make psychopathic individuals more likely to engage in aggressive interpersonal behaviors that cause others distress (Blair, 2005; Marsh & Cardinale, 2012). A consideration of the neurocognitive impairments associated with psychopathy may help to clarify why this is the case.

NEUROCOGNITIVE IMPAIRMENTS IN PSYCHOPATHY

Behavioral and cognitive similarities between individuals with psychopathy and those with amygdala lesions led to early hypotheses that amygdala dysfunction may underlie core features of psychopathy (Blair, 2003; Blair, Colledge, Murray, & Mitchell, 2001). These hypotheses are supported by recent experiments using structural and functional neuroimaging that suggest atypical amygdala morphology and functional hypoactivation in individuals with psychopathic traits.

A large and well-controlled volumetric study in 296 incarcerated men found that psychopathy was associated with reduced bilateral gray matter volume in the amygdala (Ermer, Cope, Nyalakanti, Calhoun, & Kiehl, 2012). Yang, Raine, Narr, Colletti, and Toga (2009) identified reduced amygdala volume in both the basolateral and central nuclei of individuals with psychopathy. Other studies have found reduced structural integrity in key white matter connections between the amygdala and frontal areas implicated in executive and emotional control (Craig et al., 2009; Motzkin, Newman, Kiehl, & Koenigs, 2011). Although at least one study found evidence for mixed patterns of increased and reduced size within amygdala subnuclei (Boccardi et al., 2011), on the whole, the evidence is largely consistent regarding structural abnormalities in the amygdala in psychopathy.

Given indications that psychopathy impairs recognition of distress cues like fearful expressions and that amygdala damage impairs fear recognition, it follows that individuals with psychopathic traits may exhibit amygdala dysfunction when viewing fearful expressions. The results of several recent neuroimaging studies support this prediction. Both adolescents and adults with psychopathic traits appear to show amygdala hypoactivation in response to fearful expressions but not other expressions (Jones et al., 2009; Lozier, Cardinale, VanMeter, & Marsh, 2014; Marsh et al., 2008; White et al., 2012). In addition, psychopathy is associated with reduced functional connectivity between the amygdala and the prefrontal cortex during expression processing tasks (Marsh et al., 2008), indicating that impaired processing of fearful expressions may have broad implications for learning or behavioral responses associated with these expressions.

It is theorized that dysfunction in the amygdala and the structures with which it is reciprocally connected (particularly orbitofrontal cortex, anterior cingulate cortex, and striatum) can explain not only low-level empathic impairments in psychopathy, such as recognizing and responding to others' distress, but also impairments in higher level socioemotional processes such as cooperation, judgments about aggression, and moral reasoning (Blair, 2008a; Glenn, Raine, & Schug, 2009; Harenski, Harenski, Shane, & Kiehl, 2010; Rilling et al., 2007). For example, Rilling et al. (2007) found that psychopathy was associated with aberrant patterns of activation in amygdala and orbitofrontal cortex during a Prisoner's Dilemma task, in which study participants gain or lose points depending on whether they choose to cooperate with other players. Individuals with psychopathic traits were less likely to cooperate and more likely to defect (choose noncooperation) during the task, and they showed reduced amygdala activation when their partners defected (Rilling et al., 2007). Other studies have found that psychopathy is linked to reduced amygdala and medial prefrontal cortex activation and coactivation during moral reasoning tasks, in which participants typically must make valenced judgments about the severity of immoral behaviors (Harenski et al., 2010; Marsh et al., 2011). Differences between neural responses in psychopathic and control groups may be most likely to emerge when moral judgments are emotional in nature (Glenn et al., 2009).

NEUROCOGNITIVE MODELS OF EMPATHIC DYSFUNCTION IN PSYCHOPATHY

Accumulating research findings exploring the behavioral, physiological, and neural correlates of psychopathy have led to relatively consistent outcomes, and it is widely accepted that psychopathy is associated with dysfunction in amygdala and associated structures such as orbitofrontal cortex, anterior cingulate cortex, and striatum (Blair, 2007; Harenski et al., 2010; Raine, 2008). However, a variety of interpretations of the mechanisms by which dysfunction in these structures leads to the patterns of cognition, emotional responding, and behavioral characteristics of psychopathy have been proposed.

For example, the low-fear hypothesis (Lykken, 1995) posits that psychopathy derives from heritable traits that reduce harm avoidance and increase the penchant for risk taking. However, this model does not directly address the link between fearlessness and low-level empathy and sympathetic concern, the state that motivates people to inhibit aggression or provide help to a distressed individual. Alternatively, the integrated emotion systems model (Blair, 2005) identifies the amygdala as a primary source of dysfunction in psychopathy, focusing on its essential role in aversive conditioning. This model does not focus on empathy per se, although it provides a clear mechanism by which psychopathy would lead to increases in antisocial behavior (and perhaps reductions in prosocial behavior). However, its underlying assumption is that distress cues like fearful facial expressions are unconditioned aversive stimuli, which is inconsistent with evidence that fearful expressions elicit behavioral approach (Marsh, Ambady, & Kleck, 2005). Finally, the response modulation model (Newman, 1998) views distress cues as unconditioned aversive stimuli and posits that psychopathy impedes one's ability to allocate attentional resources to peripheral emotional information if considered irrelevant to current goal-directed behavior. This model suggests that response modulation deficits result in failures to link the affective consequences of antisocial behaviors to the actions themselves (Newman, 1998). However, this model does not adequately reflect dominant models of attention (Blair, Mitchell, & Blair, 2005; Desimone & Duncan, 1995), and recent findings raise doubts that attentional processes are central to emotion processing deficits in psychopathy (Anderson & Stanford, 2012; Sylvers, Brennan, & Lilienfeld, 2011).

As yet, then, no single model of psychopathy captures the totality of the available evidence regarding the neurocognitive and neurobiological features of this disorder. As researchers' understanding of psychopathy and its neurobiological correlates evolves, the models that have been developed to explain features of the disorder will evolve as well. This may enable the improved identification of targets of intervention and treatment for individuals with psychopathy.

CONCLUSION

Individuals with psychopathic traits are dramatically overrepresented among violent criminal offenders (Blair et al., 2005). This fact adds urgency to the ongoing search for explanations of why some individuals engage in cruel behavior toward both humans and nonhuman animals. Low-level deficits in recognizing and responding to the distress of others are central to psychopathy, and this kind of deficit may disrupt the most fundamental form of empathy (de Waal, 2008). Perhaps the similarities between human distress cues and their counterparts among other social species like dogs provide some clue as to why animal cruelty is an apt predictor of aggression toward humans in individuals with psychopathic traits. In social species, cues that signal distress, particularly fear, normally serve to inhibit aggression and elicit nurturing responses among those who see them. But for individuals with psychopathic traits, in whom characteristic patterns of brain dysfunction may have disrupted the ability to understand and respond to these cues, the ability to regulate behavior in response to the fear and distress of others, whether human or canine, may be disrupted as well. Using cognitive neuroscience methods like functional neuroimaging to explore the neurobiological roots of empathy and psychopathy in humans and corresponding behavioral responses in other social species, particularly dogs, may enable us to develop a clearer understanding of these disruptions.

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INTEGRATIVE COMMENTARY II: SHARED NEUROBIOLOGICAL MECHANISMS AND SOCIAL INTERACTIONS IN HUMAN–ANIMAL INTERACTION

LISA S. FREUND

A major goal of this volume is to bridge the discipline of social neuroscience with research involving human–animal interactions (HAI). Using a social neuroscience approach to HAI research will help identify the neurobiological mechanisms underlying the effects of HAI and offer new paradigms for investigating those effects. Additionally, a focus on HAI, as a social activity for both the humans and nonhuman animals involved, has the promise of opening new areas of understanding of human social functioning within the domain of social neuroscience. The chapters in this part, Applying Neuroscience to Human–Animal Interaction, provide a strong rationale for making the effort to build a bridge between social neuroscience and HAI.

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