



Atypical empathic responses in adolescents with aggressive conduct disorder: A functional MRI investigation

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ABSTRACT

Because youth with aggressive conduct disorder (CD) often inflict pain on others, it is important to determine if they exhibit atypical empathic responses to viewing others in pain. In this initial functional magnetic resonance imaging (fMRI) study, eight adolescents with aggressive CD and eight matched controls with no CD symptoms were scanned while watching animated visual stimuli depicting other people experiencing pain or not experiencing pain. Furthermore, these situations involved either an individual whose pain was caused by accident or an individual whose pain was inflicted on purpose by another person. After scanning, participants rated how painful the situations were. In both groups the perception of others in pain was associated with activation of the pain matrix, including the ACC, insula, somatosensory cortex, supplementary motor area and periaqueductal gray. The pain matrix was activated to a specific extent in participants with CD, who also showed significantly greater amygdala, striatal, and temporal pole activation. When watching situations in which pain was intentionally inflicted, control youth exhibited signal increase in the medial prefrontal cortex, lateral orbitofrontal cortex, and right temporo-parietal junction, whereas youth with CD only exhibited activation in the insula and precentral gyrus. Furthermore, connectivity analyses demonstrated that youth with CD exhibited less amygdala/prefrontal coupling when watching pain inflicted by another than did control youth. These preliminary findings suggest that youth with aggressive CD exhibit an atypical pattern of neural response to viewing others in pain that should be explored in further studies.

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1. Introduction

Conduct disorder (CD) is a serious mental disorder of childhood and adolescence that is characterized by a longstanding pattern of violations of rules and laws. Symptoms of CD include physical aggression, manipulative lying, theft, forced sex, bullying, running away from home overnight, and destruction of property. CD is a major public health problem because youth with conduct disorder not only inflict serious physical and psychological harm on others, but they are at greatly increased risk for incarceration, injury, depression, substance abuse, and death by homicide and suicide themselves (Loeber et al., 1998). Furthermore, CD is important because it is the major childhood precursor to antisocial

personality disorder in adulthood (Lahey et al., 2005). Thus there is a pressing need to understand the biopsychological processes at multiple levels of analysis that give rise to CD. Biological studies of CD should lead to new approaches to its treatment, both by understanding the mechanisms underpinning CD and by matching treatments to specific deficits in different individuals with this heterogeneous disorder (Van Goozen and Fairchild, 2008).

Empathy, the capacity to understand and appreciate the emotional states and needs of others in reference to oneself, has been one psychological characteristic repeatedly proposed as a core deficit in CD (Lovett and Sheffield, 2007 for a review). Here we consider empathy as a construct accounting for a sense of similarity in feelings experienced by the self and the other, without confusion between the two individuals (Batson et al., 1987; Decety and Batson, 2007; Decety and Jackson, 2004; Decety and Moriguchi, 2007; Eisenberg et al., 2006). The experience of empathy can lead to sympathy or empathic concern for another based on the apprehension or comprehension of the other's emotional state or condition, but empathy can also lead to personal distress, i.e., an aversive, self-focused emotional reaction to the

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emotional state or condition of another (Batson et al., 1987; Decety and Lamm, 2009; Eisenberg and Eggum, 2009; Lamm et al., 2007a).

Interestingly, some developmental psychologists have hypothesized that empathy and sympathetic concern for others are essential factors inhibiting aggression toward others (Eisenberg, 2005; Hoffman, 2000). Empathy may be regarded as a proximate factor motivating prosocial rather than antisocial behavior (Batson, 1991). It is commonly defined as an affective reaction that is appropriate to someone else's situation rather than one's own. Some researchers have theorized that there should be a relation between aggressive behavior and a lack of empathy (e.g., Zahn-Waxler et al., 1995). Similarly, other scholars have proposed that lack of sympathy (callous disregard for the welfare of others) is an important risk factor for CD (Frick et al., 2005; Lahey and Waldman, 2003).

The propensity for aggressive behavior has been hypothesized to reflect a blunted empathic response to the suffering of others (Blair, 2005). Such a lack of empathy in aggressive individuals may be a consequence of a failure to be aroused by the distress of others (Raine et al., 1997). Similarly, it has been suggested that aggressive behavior arises from abnormal processing of affective information, resulting in a deficiency in experiencing fear, empathy, and guilt, which in normally developing individuals inhibits the acting out of violent impulses (Davidson et al., 2000; Herpertz and Sass, 2000). Consistent with this hypothesis, one functional MRI study found reduced left amygdala response in 13 adolescents with CD in response to the visual presentation of pictures with strong negative emotional valence compared to 14 control adolescents (Sterzer et al., 2005).

However, an alternative hypothesis regarding the relation between affect and aggression also can be drawn from previous research that has shown that negative affect is generally positively associated with aggression (Anderson and Bushman, 2002; Berkowitz, 2003), suggesting that empathic mimicry in conjunction with poor emotion regulation might produce negative affect that increases aggression (Campbell, 1990; Gill and Calkins, 2003). For instance, there are many empirical studies that document that physical pain often instigates aggressive inclinations (Berkowitz, 1983, 1993). This is particularly interesting in the light of recent work in cognitive neuroscience of empathy for pain.

Indeed, a growing number of functional magnetic resonance imaging (fMRI) studies have demonstrated striking similarities in the neural circuits involved in the processing of both the first-hand experience of pain and the experience of observing other individuals in pain (Jackson et al., 2006a). These studies have consistently shown that the perception of pain in others elicits activation of the neural circuit subserving the processing of the affective and motivational dimension of pain in oneself (Botvinick et al., 2005; Cheng et al., 2007; Gu and Han, 2007; Jackson et al., 2005, 2006b; Lamm et al., 2007a; Moriguchi et al., 2007; Morrison et al., 2004; Saarela et al., 2007; Singer et al., 2004; Zaki et al., 2007). This circuit includes the dorsal anterior cingulate cortex (ACC), the anterior midcingulate cortex (aMCC), the supplementary motor area (SMA), and anterior insula (Derbyshire, 2000; Price, 2000). In addition, somatosensory-evoked potentials (Bufalari et al., 2007), magnetoencephalographic (Cheng et al., 2008) and fMRI studies (Cheng et al., 2007; Lamm et al., 2007b; Moriguchi et al., 2007) have demonstrated that areas processing the sensory dimension of pain (posterior insula/somatosensory cortex) may also be elicited by the visual perception of pain in others.

Recently, one functional MRI study investigated empathy and intentionality in typically developing middle-school children (Decety et al., 2008) while they watched dynamic visual stimuli depicting either a person whose pain was accidentally caused or a person whose pain was intentionally inflicted by another individual. Interestingly, when watching a person inflicting pain

on another, regions that are engaged in representing social interaction and moral behavior including the temporo-parietal junction, the paracingulate, orbital medial frontal cortices, and amygdala (Moll et al., 2003, 2007) were additionally recruited, and increased their connectivity with the frontoparietal attention network.

There also is evidence that specifically associates the amygdala and paralimbic prefrontal regions, including the dorsal and ventral/orbital medial prefrontal cortex (dMPFC and vMPFC/OFC, respectively), with human aggression (Coccaro et al., 2007; Davidson et al., 2000). In humans, amygdala atrophy and/or lesions have been associated with impulsively aggressive behaviors (van Elst et al., 2000). Specific damage to the OFC is associated with impulsive and aggressive behavior, and individuals with such damage show little control over their emotions as well as limited awareness of the moral implications of their actions (Anderson et al., 1999; Grafman et al., 1996). Since the amygdala and OFC are anatomically and functionally connected (Amaral and Price, 1984), their interactions may be critical for interpreting emotionally significant information and guiding goal directed behaviors (Saddoris et al., 2005). Furthermore, the OFC is hypothesized to play a key role in modulating limbic reactivity to threat (Davidson et al., 2000; Izquierdo et al., 2005), and in general is important for the interpretation of social cues.

Thus, there is evidence that perceiving others in pain triggers an automatic somatic and sensorimotor resonance between other and self, which activates almost the entire neural pain matrix including the periaqueductal gray (PAG) which is a major site in pain transmission and for processing fear and anxiety (Jenck et al., 1995), the SMA that programs defensive skeletomotor impulses to avoid the stimulus in the context of nociceptive information (Morrison et al., 2006), and thalamus. Such a mechanism provides a functional bridge between first person and third person information, which allows for analogical reasoning, and offers a possible, yet partial, route to understanding others (Decety and Sommerville, 2003; Decety and Grèzes, 2006). It also provides a clear signal of the other's distress that usually inhibits aggressive behavior.

So far, there is no published work on how youth with CD react to viewing others in pain. If the blunted empathic emotional response hypothesis is correct, adolescents with aggressive CD should react less to stimuli depicting others in pain than healthy controls. Furthermore, this lack of signal from the pain matrix and amygdala could account for impairment in recognizing information about the distress of others. If the pain-aggression hypothesis is correct, however, youth with aggressive CD should exhibit greater activation than healthy controls in the amygdala, PAG, and related areas in response to stimuli depicting others in pain. This strong activation of structures subserving negative emotion and less functional amygdala/OFC connectedness could reflect a general tendency to experience personal distress that elicits aggression in some circumstances.

Including a condition in which pain is intentionally inflicted onto another allows us to examine the respective contribution of mechanisms that contribute to theory of mind and moral reasoning in the context of pain perception (Decety et al., 2008). This is particularly interesting to investigate with respect to antisocial behavior given that these individuals have been reported to lack guilt and empathic concern.

2. Methods and materials

2.1. Participants

Two groups of 16–18-year-old adolescents (exactly matched on age, sex, and race-ethnicity) were scanned. The participants were purposively selected from a 9-year longitudinal study of 127 adolescents with attention-deficit/hyperactivity disorder and a matched healthy comparison group of 125 youth (Lahey et al., 2005).

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