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Research report

Embodied emotion impairment in Huntington's Disease

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ABSTRACT

Theories of embodied cognition suggest that perceiving an emotion involves somato-visceral and motoric re-experiencing. Here we suggest taking such an embodied stance when looking at emotion processing deficits in patients with Huntington's Disease (HD), a neurodegenerative motor disorder. The literature on these patients' emotion recognition deficit has recently been enriched by some reports of impaired emotion expression. The goal of the study was to find out if expression deficits might be linked to a more motoric level of impairment. We used electromyography (EMG) to compare voluntary emotion expression from words to emotion imitation from static face images, and spontaneous emotion mimicry in 28 HD patients and 24 matched controls. For the latter two imitation conditions, an underlying emotion understanding is not imperative (even though performance might be helped by it). EMG measures were compared to emotion recognition and to the capacity to identify and describe emotions using alexithymia questionnaires. Alexithymia questionnaires tap into the more somato-visceral or interoceptive aspects of emotion perception. Furthermore, we correlated patients' expression and recognition scores to cerebral grey matter volume using voxel-based morphometry (VBM). EMG results replicated impaired voluntary emotion expression in HD. Critically, voluntary imitation and spontaneous mimicry were equally impaired and correlated with impaired recognition. By contrast, alexithymia scores were normal, suggesting that emotion

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representations on the level of internal experience might be spared. Recognition correlated with brain volume in the caudate as well as in areas previously associated with shared action representations, namely somatosensory, posterior parietal, posterior superior temporal sulcus (pSTS) and subcentral sulcus. Together, these findings indicate that in these patients emotion deficits might be tied to the "motoric level" of emotion expression. Such a double-sided recognition and expression impairment may have important consequences, interrupting empathy in nonverbal communication both ways (understanding and being understood), independently of intact internal experience of emotion.

1. Introduction

Patients with Huntington's Disease (HD), a rare, inherited neurological disorder, causing motor, cognitive and emotional dysfunctions, are impaired at recognizing emotional facial expressions. In recent years, consensus has emerged that most emotions are concerned (Henley et al., 2008, 2012; Johnson et al., 2007; Milders, Crawford, Lamb, & Simpson, 2003; Novak et al., 2012; Snowden et al., 2008). Recognizing joy/happiness was long thought to be relatively spared, however, a recent study demonstrated that the impairment extends to positive emotions when the number of positive and negative stimuli is balanced (Robotham, Sauter, Bachoud-Levi, & Trinkler, 2011). However, an integrative explanatory model of the emotion recognition deficit is still outstanding, possibly because emotion processing has mostly been tested from the recognition side only. Here, we will adopt a perspective of embodiment (see Decety & Jackson, 2004; Gallese, 2007; Keysers & Gazzola, 2007; Niedenthal, 2007), arguing that our perception of actions and emotions in others builds upon our own action and emotion representations. Our question then is whether HD patients show impaired representations for both own and others' emotions. There are two different aspects to sharing emotions: a) on the motor level of emotion expression, and b) on the level of internal experience. Evidence for both levels has been gathered by different experimental studies: a) Carr, Iacoboni, Dubeau, Mazziotta, and Lenzi (2003, see also Blair, 2005) have suggested that an emotion recognition deficit in autism might stem from an impaired action-based network (see also Dapretto et al., 2006). Overlapping fMRI activation for observing and imitating emotional facial expressions has been found in a network comprising posterior superior temporal sulcus (pSTS), posterior parietal, anterior insula (AI), amygdala and premotor cortices (Carr et al., 2003; Hennenlotter et al., 2005). Briefly, the respective contributions of these areas might be as follows. pSTS relays higher order visual information, such as information coding gaze, expression, and lip movement (Atkinson & Adolphs, 2011; Halgren, Raij, Marinkovic, Jousmaki, & Hari, 2000; Haxby, Hoffman, & Gobbini, 2000; Hein & Knight, 2008; Kesler-West et al., 2001; Pizzagalli et al., 2002; Said, Haxby, & Todorov, 2011), and biological motion in general (Giese & Poggio, 2003). Information is forwarded to posterior parietal neurons, which code kinesthetic aspects, and further to inferior frontal (BA 44/45) neurons, coding action goals. Somatosensory cortex also plays an important part in the network (Adolphs, Damasio, Tranel, Cooper, & Damasio, 2000; De Gelder, 2006, 2016), perhaps by representing aspects of the body and body surface (Keysers, Kaas, & Gazzola, 2010; Keysers et al., 2004). In sum, this network seems to support shared visuo-motor action representations (Blakemore & Decety, 2001; Grezes, Armony, Rowe, & Passingham, 2003; Grezes & Decety, 2001). b) On the other hand, evidence for a shared network on an internal experiential level has been demonstrated in the AI and anterior cingulate cortex (ACC) for observing and experiencing pain (Jackson, Meltzoff, & Decety, 2005; Singer et al., 2004), but further for a wide range of shared emotions, such as pleasant affect, social exclusion, disgust and anger (summarized in Bernhardt & Singer, 2012). The insular cortex is known to integrate diverse forms of "interoceptive" information (Craig, 2002; Critchley, Wiens, Rotshtein, Ohman, & Dolan, 2004; Kurth, Zilles, Fox, Laird, & Eickhoff, 2010), i.e., the ensemble of information about the visceral and physiological states of the body including itch, coldness, hunger, sensation of fatigue after physical exercise, pain, etc. and also visceral affective states (Craig, 2002). Interoceptive information, mapped to more posterior insular segments, is subsequently re-represented in the AI, where it may become more consciously accessible. This enables various subjective affective experiences and global (homeostatic) feeling states (Craig, 2002, 2009). The cingulate cortex may represent the motivational-premotor counterpart for the sensory-perceptual affective feelings integrated by the insula, conjointly implementing general monitoring and control processes across multiple domains (Paus, 2001). Note that AI-A/MCC stand as neuroanatomical markers of more extensive and complex networks that include brainstem, and midbrain (see Damasio, Damasio, & Tranel, 2013). Further, beyond the interoceptive brain representations that might be shared between emotions, additional differential neural representations presumably exist, tied to each emotion's intrinsic psychobiological functions. Since with regards to HD, consensus has emerged that their emotion processing deficit is not specific to one emotion (Henley et al., 2008, 2012; Milders et al., 2003; Robotham et al., 2011; Snowden et al., 2008), we do not detail these here. Importantly, AI activity during affect sharing (Bernhardt & Singer, 2012; Bird et al., 2010) and reflecting on feelings across the emotion spectrum (Silani et al., 2008), is correlated with empathy self-report scores