

Disgust and Huntington's disease

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Received 17 May 2006; received in revised form 20 October 2006; accepted 26 October 2006

Available online 28 November 2006

Abstract

The disproportionate impairment for the recognition of facial expressions of disgust in patients with Huntington's disease (HD) forms a double dissociation with the impaired recognition of fear that has been reported in amygdala patients. The dissociation has generated discussion regarding the potential existence of neural substrates dedicated to the recognition of facial signals of specific emotions. The aim of this study was to establish whether the impairment for disgust in HD was restricted solely to the domain of facial perception, or whether HD patients also demonstrate impairment in other kinds of disgust. Fourteen HD patients and fourteen age and education matched healthy controls participated in seven disparate emotion processing tasks. (1) A measure of knowledge for the situational determinants of distinct emotions; (2) recognition of emotion expressed in nonverbal vocalisations; (3) recognition of the emotional content of explicit lexical stimuli; (4) recognition of emotional content in pictures of emotion scenes; (5) a disgust experience questionnaire; (6) a measure of olfactory hedonic responsiveness; (7) a measure of gustatory perception. While verbal aspects of disgust processing were preserved, parallel impairments were revealed for olfactory disgust, vocal disgust expressions, the classification of disgusting pictures, and declarative knowledge of disgust elicitors. The finding of impaired perception of disgust signalled through different input domains suggests that the inability to recognise the facial expression in this population reflects a fundamental problem with disgust processing.

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Keywords: Disgust; Huntington's disease; Emotion processing; Facial emotion; Recognition; Insula

People with Huntington's Disease (HD) can demonstrate a disproportionate impairment for the recognition of facial expressions of disgust (Sprengelmeyer et al., 1996; Sprengelmeyer, Young, Sprengelmeyer, et al., 1997; Wang, Hoosain, Yang, Meng, & Wang, 2003). Impaired disgust recognition has been found in preclinical carriers of the HD gene, suggesting this deficit may be one of the earliest correlates of the disease (Gray, Young, Barker, Curtis, & Gibson, 1997; Hennenlotter et al., 2004; Sprengelmeyer, Schroeder, Young, & Epplen, 2006). Impaired disgust recognition in HD is most interesting when viewed in parallel with evidence that recognition of facial expressions of fear is selectively impaired in patients with bilateral damage to the amygdala (Adolphs, Tranel, Damasio, & Damasio, 1994). The differential deficits form a double dissociation

between the recognition of facial disgust and the recognition of facial expressions of fear. Functional neuroimaging research has supported the lesion data: facial expressions of disgust engage different parts of the brain (the insula and basal ganglia) than facial expressions of fear, which engage the amygdala (Sprengelmeyer, Rausch, Eysel, & Przuntek, 1998). The converging evidence indicates that the neural substrates subserving the recognition of facial expressions of fear and the recognition of facial expressions of disgust are independent.

The aim of the current research was to investigate whether other aspects of disgust are also selectively affected in HD patients. We were interested in this for several reasons. First, given the accumulating evidence that impaired processing of disgust via facial expressions in HD is not always dramatically disproportionate (Milders, Crawford, Lamb, & Simpson, 2003), we sought to confirm the selectivity of the deficit for disgust using nonfacial stimuli. This approach would also inform whether the deficit for disgust reflects a process that is specific to the retrieval of information about facial expressions, or is a symptom of a more global process affecting the detection of

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disgusting stimuli in general. We were particularly interested in whether data from HD patients could be used to illustrate the link between the experience of disgust and the ability to recognize it.

Authors interested in the neuropsychology of emotion often attribute great significance to the selective deficit for recognition of facial disgust in HD. This belies the fact that the impairment is not always dramatically disproportionate. In studies of facial expression recognition, HD patients have presented with difficulty recognizing the facial expression of disgust, together with a broader profile of difficulty recognizing negatively valenced emotions in general (Milders et al., 2003; Sprengelmeyer et al., 1996). The lack of consistent convincing evidence for the selectivity of impaired emotion recognition in HD has led some researchers to argue that data from this population cannot be used in support of a double dissociation between fear and disgust (Milders et al., 2003). On the basis of the facial data alone, one might suggest that the role played by the insula and basal ganglia in emotion recognition is not specific to any particular emotion category. This would be contrary to the view expressed routinely in the literature.

The inconsistency in HD findings warrants some investigation of whether the selectivity of patient's disgust impairment can be confirmed using nonfacial emotion stimuli. An additional benefit of this approach is that these tasks may illustrate whether deficits for disgust in HD exist outside the domain of facial emotion perception. The cross-modal hypotheses entails that selective deficits for emotion recognition reflect processes impacting not just the recognition of facial expressions, but also the processing of signals of the equivalent emotion from other modalities (Calder, Lawrence, & Young, 2001; Calder & Young, 2005). While there is some evidence to the contrary (Adolphs & Tranel, 1999; Anderson & Phelps, 1998), supporting evidence for the cross-modal hypothesis include case studies of amygdala patients with impaired recognition of facial expressions of fear, mirrored by deficits for recognition of fear expressed vocally (Scott et al., 1997; Sprengelmeyer et al., 1999) and gesturally (Sprengelmeyer et al., 1999). Similarly, functional imaging has indicated overlapping amygdala activation in response to both facial and vocally expressed fear (Phillips et al., 1998). The cross-modal data has not been limited to social signals (i.e., expressions) of fear. For example, amygdala activation has also been reported in response to pictures of frightening things (Hamman, Ely, Grafton, & Kilts, 1999; Lane et al., 1997) and in response to threatening linguistic stimuli (Isenberg et al., 1999).

Evidence of a cross-modal deficit for disgust in HD remains modest. In one study HD patients presented with parallel difficulty recognising both disgusted facial expressions and emotional sounds (Sprengelmeyer et al., 1996). But more recently, Sprengelmeyer et al. (2006) failed to find any evidence for impaired recognition of vocal disgust in preclinical HD gene carriers, and found no correlation between the inability to recognise disgust in the face and disgust in the voice. Despite this lack of HD findings, there is evidence that a cross-modal system for disgust may exist. The insular cortex, repeatedly implicated in recognition of the disgust expression (Phillips et al., 2004,

1998, 1997; Sprengelmeyer et al., 1998) may also be activated by disgust-inducing pictures (Phillips et al., 2000; Shapira et al., 2003; Wright, He, Shapira, Goodman, & Liu, 2004)¹ and in response to disgusting statements (Moll et al., 2005). Patient NK has left sided damage to the insula and striatum, and presents with impaired recognition of facial disgust, paralleled by marked impairment in recognising vocal disgust and its prosodic cues (Calder, Keane, Manes, Antoun, & Young, 2000). Similarly, in healthy subjects the ability to detect facial disgust correlates with the ability to detect disgust conveyed through posture (Rozin, Taylor, Ross, Bennett, & Hejmadi, 2005).

A natural extension of the cross-modal question is whether such impairments reflect an underlying central problem with the experience of disgust itself (Gray et al., 1997; Sprengelmeyer, Young, Pundt, et al., 1997). In support there is accumulating evidence of substantial overlap between the structures important for the recognition of disgust and those involved in disgust experience. While the insula has been repeatedly linked to the perception of offensive tastes (Small et al., 2003; Zald, Lee, Fluegel, & Pardo, 1998), and offensive smells (Royet, Plailly, Delon-Martin, Kareken, & Segebarth, 2003; Wicker et al., 2003; Zald & Pardo, 2000), Heining et al. (2003) recently showed that the right anterior insula and right ventral striatum were activated by disgusting odours to a significantly greater degree than by unpleasant or pleasant odours. Wicker et al. (2003) linked this disgust experience in the insula with disgust recognition through fMRI evidence that the insula was activated during both the observation of disgust-expressive faces and during the experience of disgust evoked by unpleasant odorants. A subsequent study found insula activation during the interoceptive experience of disgust via recall of recent salient life events (Fitzgerald et al., 2004).

Attempts to demonstrate aberrant disgust experience in HD patients have yielded little. HD patients have not reported generally reduced levels of disgust on emotion experience questionnaires (Sprengelmeyer et al., 1996). There was a trend towards reduced self-reported disgust, but responses on anger and fear experience questionnaires were similarly low, suggesting results may have reflected generally reduced responsiveness, rather than a process specific to disgust. In a recent study, presymptomatic carriers of the HD gene failed to differ from gene negative controls on reported disgust sensitivity, despite poor recognition of facial disgust found among the same individuals (Sprengelmeyer et al., 2006).

A recent study using olfactory and gustatory stimuli as a means for estimating disgust experience reported that HD patients were less disgusted by both disgusting odours and unpleasant food combinations (Mitchell, Heims, Neville, & Rickards, 2005). However, an important limitation of this study was the examination of olfactory and gustatory processing in isolation. Mitchell et al. (2005) did not confirm the existence of

¹ Some authors have not found insula activation in response to disgusting scenes (Schienle et al., 2002; Stark et al., 2003). Wright et al. (2004) propose the discrepancy may be the use of 1.5T MRI by these researchers, which it is suggested may not be sufficiently sensitive to BOLD effects to detect differential activation in the insula.

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