



Review article

Beyond emotion recognition deficits: A theory guided analysis of emotion processing in Huntington's disease



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ABSTRACT

Deficits in facial emotion recognition in Huntington's disease (HD) have been extensively researched, however, a theory-based integration of these deficits into the broader picture of emotion processing is lacking. To describe the full extent of emotion processing deficits we reviewed the clinical research literature in HD, including a consideration of research in Parkinson's disease, guided by a theoretical model on emotion processing, the Component Process Model. Further, to contribute to understanding the mechanisms underlying deficient emotion recognition, we discussed the literature in light of specific emotion recognition theories. Current evidence from HD studies indicates deficits in the production of emotional facial expressions and alterations in subjective emotional experiences, in addition to emotion recognition deficits. Conceptual understanding of emotions remains relatively intact. Impaired recognition and expression of emotion in HD might be linked, whereas altered emotional experiences appear to be unrelated to emotion recognition. A key implication of this review is the need to take all the components of emotion processing into account to understand specific deficits in neurodegenerative diseases.

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1. Introduction

Huntington's disease (HD) and Parkinson's disease (PD), like other neurodegenerative disorders, are associated with a broad spectrum of affective symptoms and syndromes, including depression, anxiety, apathy, irritability, and mood fluctuations. Emotion-related symptoms and syndromes have detrimental influences on the lived experience of patients, families, and on close others. Thus, extensive clinical research has been conducted to determine the types and extent of symptoms and impairments relevant to emotional functioning in the neurodegenerative disorders. In HD, the processing of emotion-related stimuli, such as facial expressions, scenes, descriptions of situations, or smells and tastes, has been extensively investigated. In particular, deficits in the recognition of emotional facial expressions have been well established in more than 25 studies, including a meta-analysis (e.g., Bora et al., 2016; Gray et al., 1997; Harrington et al., 2014; Hennenlotter et al., 2004; Johnson et al., 2007; Labuschagne et al., 2013; Sprengelmeyer et al., 2006; Sprengelmeyer et al., 1996; Tabrizi et al., 2009), and have been an ongoing topic of interest and debate in the clinical literature. Despite the extensive body of research, the overall progress in understanding emotional aspects of neurodegenerative diseases has been constrained by a lack of integration of this research into the broader theoretical context of emotion processing, which has developed in parallel in the fields of psychology and cognitive and affective neuroscience.

In this review, we provide a theory-based overview of research on the processing of emotional stimuli in HD, by drawing explicit links between the clinical research literature and a theoretical framework for understanding emotion processing. For practical reasons, due to the large body of literature, we have limited the scope of this review specifically to the processing of emotional stimuli, and have excluded the broader syndromic expressions of emotional dysfunction, such as apathy and depression, as well as other emotion-related functions, such as empathy and Theory of Mind. By providing the reader with a comprehensive picture of emotion processing in HD, we aim to deliver a theoretically-guided characterization of the preserved and impaired areas of emotion processing that may be responsible for problems in emotional well-being and changes to social relationships in HD. With respect to the well-known emotion recognition deficit, by integrating emotion recognition impairments into the broader evidence of problems with processing emotion-related stimuli more generally, we provide new insights into the mechanisms underlying the deficit. Furthermore, the characterization of emotion processing in HD within emotion theoretical frameworks in this review is not only useful to research in HD, but also to research in other neurodegenerative disorders and to affective neuroscience more generally. That is, we bring contemporary ideas about emotion processing from the theoretical literature to the neurodegenerative disease literature, and we believe this review will be useful by elucidating the role of brain regions affected in HD and PD for emotion processing.

HD is a genetic progressive neurodegenerative disease associated with motor symptoms and cognitive decline (Folstein, 1989; Stout et al., 2012; Tabrizi et al., 2012). HD is autosomal-dominant, and is caused by an expanded CAG repeat on chromosome 4 (Myers,

2004). A large body of research over the past 15 years has revealed that neurodegeneration begins decades before symptoms appear, starting in the brain's striatum, which is a component of a deep subcortical brain region, the basal ganglia (Aylward et al., 1997; Aylward et al., 2004; Paulsen et al., 2010), and eventually affects a wide range of brain regions (Rosas et al., 2003; Vonsattel, 2008). Disease diagnosis is typically in mid-adult life, but can occur at any age. Despite the known brain changes, individuals in the pre-diagnosis phase appear healthy, but many display subtle changes in motor functions, personality, and cognition in the years leading up to the diagnosis of symptomatic HD (Snowden et al., 2002). Currently, there is no effective treatment for HD, and patients die on average within 15 years (Folstein, 1989). In addition to motor and cognitive dysfunctions, HD is associated with a broad range of neuropsychiatric symptoms that are high in prevalence (Paulsen et al., 2001; Van Duijn et al., 2014; Van Duijn et al., 2007), with some studies finding affective symptoms in over 90% of HD patients (Paulsen et al., 2001; Thompson et al., 2012). The three most frequent emotion-related symptoms are apathy, depression, and irritability (Craufurd et al., 2001; Van Duijn et al., 2007).

We focused this review on HD, because it is the main focus of our own research, but included additional findings on PD because of the related nature of the neural circuitry dysfunction in PD and HD. PD, like HD, is a neurodegenerative disorder that affects the function of the basal ganglia (Hornykiewicz and Kish, 1987; Taylor et al., 1986). Thus, unsurprisingly, PD shares a number of similar symptoms with HD. PD patients suffer from a range of motor symptoms that have differences and commonalities with HD. In particular, common movement symptoms in PD patients include tremors, rigidity, and slowness of movement (Jankovic, 2008). Like HD patients, they also display cognitive decline (Litvan et al., 2012; Svenningsson et al., 2012) and psychiatric symptoms (Den Brok et al., 2015; Rihmer et al., 2014). Relevant to this review, PD is also associated with deficits in emotion processing, many of which appear similar to the impairments found in HD (Gray and Tickle-Degnen, 2010). Many aspects of emotion processing have been researched more extensively in PD than in HD.

We begin this review with a consideration of emotion theories from the psychological and affective neuroscience literature (Section 2). We then describe our selection of a particular theoretical framework, the Component Process Model (CPM), in detail (Section 2.1) and also describe specific theories on emotion recognition (Section 2.2), to guide our subsequent review of the literature in HD. Next, we apply the CPM to the clinical research. We start by first describing the methodological approaches used to study emotion processing in HD (Section 3), and then the findings that have emerged from the literature, including findings in PD where appropriate (Section 4). In addition to studies that use purely behavioural methods for studying emotion processing, we also consider those that have used neurophysiological methods to examine the neural correlates of impairments (Section 5). To understand the practical significance of emotion processing deficits, we review evidence regarding the association of these deficits with measures of social functioning (Section 6). Finally, we provide a general discussion of the findings of this review, including a synthesis across the studies within the theoretical framework (Section 7).

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