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Dissociating emotional and cognitive empathy in pre-clinical and clinical Huntington's disease

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ABSTRACT

Huntington's disease (HD) is centrally characterized by motor, neurocognitive and psychiatric symptoms, but impaired emotional decoding abilities have also been reported. However, more complex affective abilities are still to be explored, and particularly empathy, which is essential for social relations and is impaired in various psychiatric conditions. This study evaluates empathic abilities and social skills in pre-clinical and clinical HD, and explores the distinction between two empathy sub-components (emotional-cognitive). Thirty-six HD patients (17 pre-clinical) and 36 matched controls filled in the Empathy Quotient Scale, while controlling for psychopathological comorbidities. At the clinical stage of HD, no global empathy impairment was observed but rather a specific deficit for the cognitive sub-component, while emotional empathy was preserved. A deficit was also observed for social skills. Pre-clinical HD was not associated with any empathy deficit. Emotional deficits in clinical HD are thus not limited to basic emotion decoding but extend towards complex interpersonal abilities. The dissociation between impaired cognitive and preserved emotional empathy in clinical HD reinforces the proposal that empathy subtypes are sustained by distinct processes. Finally, these results underline the extent of distinct affective and social impairments in HD and the need to grasp them in clinical contexts.

1. Introduction

Huntington's disease (HD) is a genetically inherited neurodegenerative disease classically associated with a triad of motor, neurocognitive and psychiatric symptoms (Roos, 2010). Beyond these well-established impairments, other deficits have been documented, particularly the presence of emotional disturbances as HD is characterized by impaired ability to identify the six classically described (Darwin 1872; Ekman, 1993; Ekman and Friesen, 1971) basic facial emotional expressions: several studies have initially evidenced a specific deficit for the identification of disgust (Sprengelmeyer, 2007), but more recent works have shown that this deficit is generalized to other negative emotions like anger, fear or sadness (Johnson et al., 2007). Critically, this deficit is present at the clinical stage of the disease (i.e. among symptomatic patients presenting motor impairments) but might already exist at the pre-clinical stage [i.e. among non-symptomatic persons carrying HD's gene (Johnson et al., 2007)]. Moreover, as this impairment appears specific to emotional processing (Sprengelmeyer, 2007) and is also present for emotional prosody (Snowden et al., 2008) as well as for facial expression of emotions (Trinkler et al., 2013), HD appears associated with a generalized impairment in the detection and expression of emotions (Henley et al., 2012).

Efficient emotional processing is a crucial skill to maintain adapted interpersonal relations, and these emotional deficits thus negatively impact social life in HD as they are correlated with reduced functional capacity in everyday life (Craufurd and Snowden 2002). It is now clearly established that emotional

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impairments have deleterious consequences on personal and professional life in HD (Ille et al., 2011a). Moreover, at a clinical level, it has been shown in several psychiatric and medical conditions that the quality of social support and interpersonal environment has a crucial impact on treatment compliance (Dour et al., 2014), underlining the role of affective and social factors on treatment response. In view of these arguments, it appears crucial to further explore the extent of these emotional deficits and their links with interpersonal impairments in HD. Indeed, despite the exploration of basic emotional decoding and the proposal that higher-level affective abilities could also be impaired, these more complex emotional abilities involved in social interactions have been little explored in HD, hampering to obtain an exhaustive view of the emotional impairments.

Among the emotional competences that should be more thoroughly investigated, empathy occupies a core position as it is an essential ability to build and maintain affective bonds between mother and child, partners, and then larger social groups (Singer, 2006). Empathy is globally defined as the aptitude to understand and respond to other's feelings, thoughts, or emotions by imagining oneself in another individual's position (Decety and Jackson, 2006). Empathy is not a unitary concept but rather a multifaceted construct involving at least two distinct components (Lawrence et al., 2004), namely: (1) an emotional component linked to the ability of experiencing others' emotional states, and (2) a cognitive component related to perspective-taking ability allowing to understand others' mental states (e.g., thoughts, goals). The validity of this emotional-cognitive distinction has been experimentally explored by the observation of specific deficits in psychiatric states: autism (Smith, 2009) and euthymic bipolar disorder (Shamay-Tsoory et al., 2009) are associated with marked cognitive empathy deficit but preserved emotional empathy. Conversely, alcohol-dependence leads to impaired emotional empathy with preserved cognitive empathy (Maurage et al., 2011). These results clearly call for completing the classical exploration of global empathy by a separate exploration of its two sub-components.

Empathic abilities have also been recently explored in a wide-range of neuropsychiatric conditions, notably showing a general empathy deficit in Parkinson's disease (Narme et al., 2013), and a dissociation between preserved emotional and impaired cognitive empathy in Alzheimer's disease (Nash et al., 2007). These results underline the critical role played by empathy deficits in neurodegenerative states and lead to crucial theoretical and clinical implications (Kemp et al., 2012). Surprisingly however, there is currently a striking scarcity of knowledge on empathy abilities in HD. Indeed, on the one hand, some studies have explored cognitive processes that are related to empathy, showing deficits for social cognition (Snowden et al., 2008), perspective taking (Brüne et al., 2011) or intention attribution (Baez et al., 2015) in clinical HD. Nevertheless, the use of cognitive-demanding tasks do not allow to exclude that these deficits are partly related to more general cognitive impairments (e.g., working memory), and these studies did not directly measure empathy. On the other hand, only one study has explored empathy in HD (Trinkler et al., 2013), describing preserved empathic abilities in clinical HD. Although constituting a valuable first exploration, these preliminary results presented three main shortcomings: First, the evaluation of empathy relied on highly criticized questionnaires (Baron-Cohen and Wheelwright, 2004) unable to dissociate emotional and cognitive sub-components. Second, while clinical psychiatric diagnoses constituted exclusion criteria, sub-clinical anxiety and depression were not controlled for and might have influenced empathy scores (Grynberg et al., 2010). Third, the experimental sample was exclusively constituted of clinical HD patients presenting motor and cognitive impairments, preventing any conclusion concerning the presence of empathy deficits in pre-clinical HD and their evolution across the successive stages of HD.

To overcome these limitations, the present study explored empathy in pre-clinical and clinical HD, with a strict control of psychopathological variables and by means of a validated questionnaire [Empathy Quotient questionnaire, EQ (Baron-Cohen and Wheelwright, 2004)] allowing the separate exploration of emotional and cognitive empathy. As earlier results have suggested significant perspective taking and social cognition impairments in clinical HD (Baez et al., 2015; Brüne et al., 2011; Snowden et al., 2008), we hypothesized that this group would show massive deficits for cognitive empathy. Conversely, as interpersonal and emotional functions have been repeatedly described as preserved in pre-clinical HD (Kipps et al., 2007; Sprengelmeyer et al., 1996), it can be hypothesized that this group will present unaltered empathic abilities. Finally, a "social skills" subscale was included in the EQ, allowing to explore the global ability to behave appropriately in interpersonal situations (Lawrence et al., 2004). A last hypothesis was thus that clinical HD participants would, in view of their reduced capacity to maintain efficient social interactions, be impaired on this subscale compared to healthy controls and pre-clinical HD.

2. Methods

2.1. Subjects

Thirty-six adults (16 women) with a genetically confirmed HD diagnosis (Huntington's Disease Participants, HDP) were recruited in the HD care units of four Belgian hospitals. Participants were first contacted by their general practitioner or neurologist who explained the aims of the study, and were then referred to the principal investigator. All participants had a family history of HD and completed a genetic blood test assessing the HD's cytosine-adenine-guanine (CAG) expansion. HD is characterized by elongated CAG repeat on at least one allele of the chromosome 4 on the Huntingtin gene. All participants presented an expansion of at least 36 CAG repeats (Roos, 2010). Among them, 17 were at pre-clinical phase (carrying the HD's gene but non-symptomatic, HDP-) while 19 were at clinical phase (symptomatic participants with motor impairments, HDP+). The disease stage was assessed by their neurologist, their nurse, and a psychologist with expertise in HD, according to Roos' criteria (Roos, 2010): among HDP−, 14 were at the A2 stage (i.e. gene carrier, premanifest stage) and three were at the A3 stage (i.e. transition phase, ongoing changes at behavioural and motor levels); among HDP+, 12 were at the B1 stage (i.e. clinical stage I, with initial neurological, cognitive and psychiatric symptoms, chorea being the most prominent symptom) and seven were at the B2 stage (i.e. clinical stage II, with generalized motor disturbance and increased cognitive-psychiatric symptoms). The mean illness duration among HDP+ was 6.87 years ($SD=5.41$). The mean number of CAG repeats of the longer allele was 42.82 ($SD=3.81$) in HDP− and 42.84 ($SD=3.30$) in HDP+. Moreover, HD participants' global functioning was assessed by a neurologist through the Clinical Global Impression Scale (CGI), a widely-used clinical tool evaluating the psychological, social and occupational abilities on a scale ranging from 1 (normal) to 7 (among the most ill patients). In HDP−, CGI scores were between 1 (normal) and 2 (borderline) ($M=1.24$, $SD=0.44$). In HDP+, CGI scores were between 3 (mildly ill) and 5 (markedly ill) ($M=4.26$, $SD=0.65$).

HD participants were matched for age, gender, and education with 36 control participants (CP). Two subgroups of CP were determined (CP−, CP+) respectively matched with HDP− and HDP+. Groups' characteristics appear in Table 1. Exclusion criteria for both groups included major medical problems, neurological disease (except HD for the HD groups), psychiatric disorder and