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SHORT REPORT

Corticostriatal signatures of schadenfreude: evidence from Huntington's disease

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ABSTRACT

Schadenfreude—pleasure at others' misfortunes—is a multidetermined social emotion which involves reward processing, mentalising and perspective-taking abilities. Patients with Huntington's disease (HD) exhibit reductions of this experience, suggesting a role of striatal degeneration in such impairment. However, no study has directly assessed the relationship between regional brain atrophy in HD and reduced schadenfreude. Here, we assessed whether grey matter (GM) atrophy in patients with HD correlates with ratings of schadenfreude. First, we compared the performance of 20 patients with HD and 23 controls on an experimental task designed to trigger schadenfreude and envy (another social emotion acting as a control condition). Second, we compared GM volume between groups. Third, we examined brain regions where atrophy might be associated with specific impairments in the patients. While both groups showed similar ratings of envy, patients with HD reported lower schadenfreude. The latter pattern was related to atrophy in regions of the reward system (ventral striatum) and the mentalising network (precuneus and superior parietal lobule). Our results shed light on the intertwining of reward and socioemotional processes in schadenfreude, while offering novel evidence about their neural correlates.

INTRODUCTION

Huntington's disease (HD) is an autosomal-dominant neurodegenerative disorder characterised by cognitive, motor and behavioural abnormalities associated with neuronal loss within corticostriatal circuits.[1] At the neuroanatomical level, regional cerebral atrophy occurs in carriers of the HD gene mutation before clinical symptoms appear.[2] Neuroimaging studies[3–5] have revealed selective grey matter (GM) atrophy in HD, with the earliest changes progressing from the dorsolateral to the ventromedial portions of the neostriatum. The distribution of white matter degeneration is widespread, suggesting extensive loss of structural connectivity early in the clinical course of the disease. Moreover, atrophy is more prominent over more posterior cortical regions earlier in the disease.[6] Subsequently, progressive atrophy compromises the insula; the primary sensory, motor and visual cortices; and then the primary auditory cortex. Finally, atrophy extends to the entorhinal cortex and cortical regions subserving high-order functions.[3–5] Specifically, structural and functional abnormalities in the basal ganglia and frontostriatal pathways have been associated with behavioural changes[7] and impairments in several social cognition domains, including emotion recognition,[8] mentalising abilities[9] and social emotions.[10]

The latter includes schadenfreude, which refers to the perceiver's experience of pleasure at another's misfortune.[11] This is a multidetermined emotion which can be evoked by hostile feelings and envy.[12] Schadenfreude is relevant in maintaining stability during social interaction and in regulating social behaviour.[13] Moreover, the experience of schadenfreude engages mechanisms implicated in diverse social cognitive processes. For instance, schadenfreude involves heightened reward processing, accompanied by increased striatal engagement,[12] and it interacts with mentalising and perspective-taking abilities.[14] Indeed, crucial hubs of the mentalising network may be crucial mediators of this emotion.[14 15]

In a recent behavioural study,[10] we evaluated patients with HD and asymptomatic first-degree relatives with a novel paradigm assessing schadenfreude and envy (another social emotion acting as a control condition). Schadenfreude was selectively reduced in both groups relative to controls, with no abnormalities in control conditions (ie, ratings of envy or ratings for neutral conditions). As mentioned above, schadenfreude has been associated with mentalising abilities, which may also be impaired in patients with HD. Indeed, these individuals tend to draw faulty inferences from social situations, and they are impaired in both affective and cognitive aspects of theory of mind.[16–18]

To extend such findings, here we investigated whether GM atrophy in patients with HD correlates with schadenfreude ratings. First, we compared the behavioural performance of patients with HD and controls on the above-mentioned task. Then, we compared GM volume in both samples using voxel-based morphometry (VBM). Finally, we examined the brain regions where atrophy might be associated with reduced schadenfreude in the patients. We expected that such a reduction would be associated with the patients' striatal atrophy as well as with reduced GM volume in areas subserving other social cognition domains (eg, mentalising and emotion recognition). Confirmatory evidence could pave the way for novel research on the relationship between reward processing and socioemotional processes in neurodegenerative diseases.

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Table 1 Demographic, clinical and executive functions assessments

	Patients with HD (n=20), mean (SD)	Controls (n=23), mean (SD)	Patients with HD versus controls
Demographics			
Age (years)	44.9 (11.1)	41.5 (17.3)	NS
Gender (F:M)	12:8	12:11	NS
Education (years)	9.2 (3.8)	10.3 (4.5)	NS
Clinical assessment			
UHDRS motor score	46.6 (36.5)		
PDRS	77.5 (14.4)		
Cognitive assessment			
Total MoCA score	17.4 (4.5)	25.1 (3.9)	0.0000
Total IFS score	14.6 (5.6)	23.1 (2.4)	0.0000
IFS subscales			
Motor series	2.1 (1.1)	2.9 (0.3)	0.0007
Conflicting instructions	2.0 (0.9)	2.7 (0.4)	0.0008
Go/no-go	1.4 (1.2)	2.5 (0.7)	0.0002
Backward digit span	2.1 (1.1)	3.2 (0.9)	0.0002
Verbal working memory	0.7 (0.7)	1.6 (0.7)	0.0002
Spatial working memory	1.6 (0.9)	2.7 (0.9)	0.0003
Abstraction capacity	1.5 (1.1)	2.5 (0.7)	0.0012
Verbal inhibitory control	3.4 (1.9)	5.0 (1.3)	0.0021

HD, Huntington's disease; IFS, INECO Frontal Screening battery; MoCA, Montreal Cognitive Assessment; NS, non-significant; PDRS, Physical Disability Rating Scale; UHDRS, Unified Huntington's Disease Rating Scale.

MATERIALS AND METHODS

Participants

We recruited 20 patients genetically and clinically diagnosed with HD (see online supplementary file S1), and 23 healthy controls matched in age, sex and years of education with the patients (table 1). These samples of patients and controls are different from those assessed in our previous behavioural study.[10] Patients with HD underwent a neurological examination and were assessed using the Unified Huntington's Disease Rating Scale (UHDRS)[19] motor examination section and the Physical Disability Rating Scale (PDRS)[20] (table 1). As in previous studies on this population,[21–25] we only included the motor examination section of the UHDRS because of its robustness as a measure of disease progression and severity.[24 26] All participants provided written informed consent in agreement with the Declaration of Helsinki. The institution's ethics committee approved the study.

Behavioural assessment

The participants' general cognitive state and their executive functions were assessed with the Montreal Cognitive Assessment (MoCA) and the INECO Frontal Screening (IFS) battery, respectively (see online supplementary file S2 and supplementary references).

Experimental task

Levels of envy and schadenfreude were measured through a task previously employed in this population.[10] Before testing, each participant was shown a real-life photograph and a description of two target characters matched in age and gender with the participant—relevant data were obtained in a brief initial interview. The task comprised two experimental blocks. In the first block, participants read eight sentences describing fortunate events involving two characters. After each sentence, participants rated the event in terms of how much envy they felt for the character. In the second block, participants read and reported the intensity of their pleasure (schadenfreude) in response to eight unfortunate events happening to the characters (see details on online supplementary file S3). Given that the presence of envy increases the probability of experiencing schadenfreude,[12 13] experimental blocks were presented to all subjects in the same order. Stimuli were selected taking into account the general cognitive state of patients with HD.[10]

MRI scanning

All participants were scanned in a 1.5T Siemens Magnetom equipped with a standard head coil (see online supplementary file S4).

Data analysis

Behavioural data were compared using one-way analysis of variances. Gender was analysed with a χ^2 test. To control for the influence of general cognitive status on experimental results, we applied analysis of covariance tests adjusted independently for total MoCA and IFS scores. Only those effects that remained significant after covariation were reported. Effect sizes were calculated through partial eta squared (η^2). In addition, to explore the relationship between disease severity and patients' performance, we conducted correlation analyses between UHDRS and PDRS scores and envy and schadenfreude ratings. Correlation analyses were also conducted to explore the relationship between MoCA and IFS scores and performance in the experimental task.

VBM analysis

Images were preprocessed using the DARTEL Toolbox, in accordance with previously described procedures[27] (see online supplementary file S5).

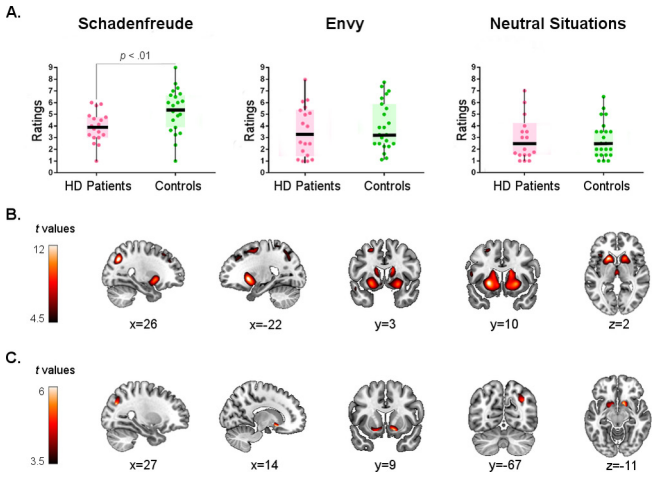

Figure 1 Correlations and comparisons between patients with HD and controls. (**A**) Differences between the groups in the social emotions task. Significant differences were found only in schadenfreude ratings. (**B**) Regions of significant GM volume loss in the HD group compared with the control group (p<0.05, FWE-corrected). (**C**) Atrophied brain regions related to reduced schadenfreude in patients with HD (p<0.05, FWE-corrected). GM, grey matter; HD, Huntington's disease.

Relationship between regional brain atrophy and performance of patients with HD

Using SPM-12, we performed multiple regression analyses to explore the association between regional GM reduction and schadenfreude ratings in patients with HD. In addition, we tested whether ratings of envy or ratings for neutral situations were associated with GM atrophy in patients with HD. These analyses were restricted to areas of significant GM atrophy in patients relative to controls. Age and total intracranial volume were included as covariates of no interest (p<0.05, family wise error (FWE)-corrected, extend threshold=50 voxels).

RESULTS

Patients with HD performed significantly worse than controls in the MoCA ($F(1,41)=36.17$, $p<0.001$, $\eta^2=0.47$) and the IFS ($F(1,41)=44.49$, $p<0.001$, $\eta^2=0.52$) total scores (table 1). Moreover, patients with HD were outperformed by controls in all the IFS subscales (see table 1).

Ratings of schadenfreude and envy

Patients showed significantly lower schadenfreude ratings than controls ($F(1,39)=7.81$, $p<0.01$, $\eta^2=0.17$). No significant between-group differences emerged in ratings of envy ($F(1,39)=0.33$, $p=0.57$, $\eta^2=0.01$) or ratings for neutral situations ($F(1,39)=0.31$, $p=0.58$, $\eta^2=0.01$) (see online supplementary file S6)
figure 1A).

Correlation analyses between envy ratings and UHDRS ($r=0.06$, $p=0.88$) or PDRS ($r=-0.08$, $p=0.84$) scores showed no significant associations. Likewise, schadenfreude ratings did not correlate with UHDRS ($r=-0.12$, $p=0.76$) or PDRS ($r=-0.18$, $p=0.66$) scores.

Correlation analyses between envy ratings and total MoCA scores showed no significant associations in any group (patients with HD: $r=-0.21$, $p=0.38$; controls: $r=0.26$, $p=0.21$). Similarly, schadenfreude ratings did not correlate with total MoCA scores in any group (patients with HD: $r=-0.03$, $p=0.90$; controls: $r=-0.38$, $p=0.08$). No significant associations were found either between envy ratings and total IFS scores in any group (patients with HD: $r=-0.10$, $p=0.66$; controls: $r=0.31$, $p=0.14$). Finally, schadenfreude ratings were not associated with total IFS scores in any group (patients with HD: $r=0.09$, $p=0.70$; controls: $r=-0.04$, $p=0.85$).

VBM results

HD brain atrophy

Compared with controls, patients with HD showed reduced GM volume in the basal ganglia (including the bilateral putamen and nucleus accumbens, and the right caudate), the left thalamus, the left middle frontal gyrus (including the premotor cortex), the right superior parietal lobule, the right precuneus and the right postcentral gyrus extending to the supramarginal gyrus (figure 1B; online supplementary table 1).

Atrophied brain regions related to schadenfreude impairments in patients with HD

We found a significant positive correlation between schadenfreude ratings in patients and their GM volume in the bilateral ventral striatum (specifically, the ventral portion of the striatum, including the nucleus accumbens) and the right superior parietal lobule and precuneus. Thus, lower GM volume in those areas was associated with a reduced schadenfreude (figure 1C). No significant correlations emerged between atrophied brain regions and ratings of envy or ratings for neutral situations in patients with HD.

DISCUSSION

This is the first study investigating the relationship between regional GM atrophy and reduced schadenfreude in patients with HD. We replicated the findings of a recent behavioural study[10] showing lower schadenfreude in this population. This effect was present even after controlling for cognitive impairment/executive dysfunction. Moreover, reduced schadenfreude in patients with HD was not associated with severity of motor symptoms, functional disability or cognitive impairments. These findings are line with those of a previous study showing that schadenfreude may be diminished even in asymptomatic first-degree relatives of patients with HD.[10] Ratings of envy and ratings for neutral situations were similar between patients with HD and controls. Also, lower schadenfreude ratings were associated with lower GM volumes in regions subserving reward processing (ventral striatum) and mentalising (superior parietal lobule and precuneus). Such findings illuminate social emotion impairments in HD while reinforcing the multidetermined nature of schadenfreude.

In line with previous studies,[28 29] patients with HD exhibited the reported atrophy pattern involving the basal ganglia (putamen and caudate) and the thalamus, as well as the frontal (premotor cortex) and parietal (precuneus and superior parietal lobule) lobes. No prefrontal cortex atrophy was observed in our sample, replicating results from previous neuroimaging studies on patients with HD.[29–32]

Multiple regression analysis revealed that reduced schadenfreude was associated with lower GM volumes in the ventral striatum, the superior parietal lobule and the precuneus. These findings may reflect some cognitive mechanisms implicated in schadenfreude which are impaired in HD. For instance, the ventral striatum has usually been involved in processing of reward information. In line with our results, previous functional neuroimaging reports have systematically associated feelings of schadenfreude with ventral striatum activity.[12] Compatibly, this

structure plays a central role in processing reciprocity of rewards during social comparisons.[33] The ventral striatum exhibits early compromise in HD[4] and reduced activity of this region has been associated with impaired reward processing in patients with HD.[34] Thus, our results suggest that specific reductions of schadenfreude in this population may be directly linked to reward system impairments following atrophy of the ventral striatum. As we did not explore the relationship between schadenfreude and reward processing, future studies in patients with HD should investigate this specific association.

On the other hand, the superior parietal lobule and the precuneus have been associated with mentalising abilities.[35] Specifically, the precuneus is involved in processing intentions related to the self. In line with these findings, previous studies have been associated the feeling of schadenfreude with mentalising and perspective-taking abilities.[14] Specifically, mentalising abilities are involved in schadenfreude since this emotion implies at least two individuals in whom one's emotional state depends on the other's mental state. Also, schadenfreude involves comparisons between one's own outcomes and those of another person.[36] In line with evidence that mentalising and perspective-taking abilities are impaired in HD,[9 16–18] our results showed that atrophy of mentalising network regions is associated with diminished schadenfreude in this population. Such a reduction may thus be linked to mentalising and perspective-taking impairments. Further studies should explore the specific relationship between mentalising and schadenfreude in patients with HD.

Note that mentalising processes have been observed to engage other brain areas, including the medial prefrontal cortex.[37–40] However, we found no significant GM reduction of prefrontal regions in HD. Therefore, atrophy of prefrontal regions seems not to be related to mentalising processes implicated in the reduced schadenfreude observed in patients with HD (see online supplementary file S7 and supplemtary references for supplementary discussion).

Recent studies in other neurodegenerative conditions[41–44] (ie, frontotemporal dementia) have shown that patients exhibit deficits in integrating self-perspectives with those of others and rewarding benefits. These impairments are associated with atypicalities in frontotemporal networks. The integration of self-preferences with the outcomes of another person seems to be a crucial aspect of the schadenfreude experience. In this study we showed that reduced schadenfreude in patients with HD is associated with brain regions supporting reward processing and mentalising. Future studies should investigate the potential overlap in neural basis of social emotions and social bargaining in patients with different neurodegenerative disorders characterised by different patterns of GM atrophy.

Ratings of envy were similar between patients with HD and controls. Moreover, no significant relationship was found between GM atrophy and envy ratings of patients with HD. This aligns with evidence of differential activation patterns for envy and schadenfreude, as the former engages ventral prefrontal areas, in particular the anterior cingulate cortex.[12 36] Our results and previous neuroimaging studies show that GM in these areas is preserved in patients with HD.[29] The differential pattern we observed arguably reflects the distinct nature and neural basis of schadenfreude relative to envy. The preservation of this closely related emotion supports the distinctiveness of schadenfreude impairments following atrophy in hubs of the reward and mentalising systems.

In conclusion, the results of this study showed that the reduced malicious pleasure of schadenfreude in patients with HD is associated with GM atrophy of regions of the reward and mentalising systems. Our findings contribute to the development of a new field of research regarding social emotions and neurodegeneration. From a theoretical point of view, our results reinforce the notion that schadenfreude is a multidetermined social emotion which involves reward processing, mentalising and perspective-taking abilities.[14 15 45] Our findings may also have some clinical implications. Schadenfreude may be included in the clinical assessment of patients with HD. Given that this emotion is also reduced in asymptomatic first-degree relatives of patients with HD,[10] it may constitute a potential marker of HD onset or vulnerability. Future large-scale longitudinal studies are required to test this possibility. Moreover, the reduction of schadenfreude may be linked to relevant clinical symptoms characterised by diminished experience of pleasure, such as depression and anhedonia. Future studies should explore whether these clinical symptoms are associated with reduced schadenfreude in HD. Finally, our results open the door to future studies investigating social emotion processing in other clinical populations (eg, Parkinson's disease, schizophrenia and autism spectrum disorders) or non- clinical populations [46] characterised by social-cognitive, striatal or mentalising network impairments .

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Contributors SB, MP, MB, LS and AI developed the study concept and the study design. MP and MB performed testing and data collection. SB, LS, HS-G and SF performed the data analysis and interpretation under the supervision of AI and AMG. SB, HS-G and SF drafted the manuscript, and MP, MB, AI and AMG provided critical revisions. All authors approved the final version of the manuscript for submission.

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Notes