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Social cognition in hyperkinetic movement disorders: a systematic review

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ABSTRACT

Numerous lines of research indicate that our social brain involves a network of cortical and subcortical brain regions that are responsible for sensing and controlling body movements. However, it remains unclear whether movement disorders have a systematic impact on social cognition. To address this question, we conducted a systematic review examining the influence of hyperkinetic movement disorders (including Huntington disease, Tourette syndrome, dystonia, and essential tremor) on social cognition. Following the PRISMA guidelines and registering the protocol in the PROSPERO database (CRD42022327459), we analyzed 50 published studies focusing on theory of mind (ToM), social perception, and empathy. The results from these studies provide evidence of impairments in ToM and social perception in all hyperkinetic movement disorders, particularly during the recognition of negative emotions. Additionally, individuals with Huntington's Disease and Tourette syndrome exhibit empathy disorders. These findings support the functional role of subcortical structures (such as the basal ganglia and cerebellum), which are primarily responsible for movement disorders, in deficits related to social cognition.

KEYWORDS: hyperkinetic disorders; theory of mind; social perception; emotion recognition; empathy

1. Introduction

Social cognition refers to a complex set of mental abilities that enable us to process and respond to social stimuli. These abilities are crucial for accurately interpreting social contexts and effectively interacting with others (Beaudoin & Beauchamp, 2020; Decety & Cacioppo, 2011). When social cognition is impaired, it can have a negative impact on our understanding of others and our ability to interact with them, leading to a diminished quality of life (Hasson-Ohayon et al., 2017). In experimental and clinical settings, researchers have primarily focused on three main dimensions of social cognition: social perception, theory of mind (ToM), and empathy (Henry et al., 2016). These dimensions are conceptually, behaviorally, and neurally distinguishable from each other (Preckel et al., 2018).

At its most basic level, social perception involves how we perceive and interpret social cues. These cues include biological movements (Giese & Poggio, 2003), emotions expressed through facial and bodily expressions (De Gelder et al., 2010; Todorov et al., 2008), and emotional prosody (Mitchell et al., 2003). The posterior superior temporal sulcus (pSTS) is one of the areas most consistently activated in response to biological movements (Allison et al., 2000; Jastorff et al., 2012; Urgesi et al., 2014) and emotional expressions (Deen et al., 2015; Haxby et al., 2000; Paracampo et al., 2018b). Other areas that respond to biological movements include inferior frontal, premotor, somatosensory, and posterior parietal regions that are also active during action execution (Paracampo et al., 2018a; Pitcher et al., 2008; Sliwinska & Pitcher, 2018; Urgesi et al., 2014; Valchev et al., 2017). These sensorimotor regions contribute to linking observed movements and expressions with their corresponding sensorimotor representations (Avenanti et al., 2013; Keysers et al., 2010; Rizzolatti & Sinigaglia, 2016), providing support to embodied-grounded proposals that cognition, and social perception in particular, is rooted in sensorimotor experiences and body-environment interactions (Barsalou, 2008; Borghi et al., 2013; Gallese & Sinigaglia, 2011; Niedenthal et al., 2010; Pezzulo et al., 2011; Shapiro, 2011). Social perception also relies on visual areas involved in processing morphological aspects of facial and body stimuli, including the fusiform facial area (FFA) and occipital face area (OFA) (Kanwisher & Yovel, 2006; Pitcher et al., 2011) and the extrastriate body area (EBA) and fusiform body area (FBA) (Downing & Peelen, 2016; Peelen & Downing, 2007). Additionally, social perception involves the amygdala which is part of a cortico-subcortical network responsible for identifying the salience and emotional value of social stimuli (Bagnis et al., 2020; Costafreda et al., 2008; Seeley, 2019; Tamietto & de Gelder, 2010).

Theory of mind (ToM) allows us to attribute mental states to ourselves and others. This ability enables us to make inferences and adjust our behavior in social environments (Carrington & Bailey, 2009; Gallagher & Frith, 2003). The medial prefrontal cortex (MPFC) plays a role in integrating social information with stored knowledge and norms, allowing us to infer others' mental states, including stable states such as personality traits (Amodio & Frith, 2006; Heleven & Van Overwalle, 2018). On the other hand, the temporoparietal junction (TPJ) is involved in processing temporary inner states such as belief, goals, and intentions of other people (Krall et al., 2015; Van Overwalle, 2009).

Empathy encompasses two main components: cognitive empathy, which refers to the ability to assume the point of view of others (perspective-taking) and understand their feelings, and affective empathy, which involves perceiving and experiencing at an emotional level what another person feels (Cuff et al., 2016; Zaki & Ochsner, 2012) – although some scholars tend to limit the concept of empathy to its affective component (e.g., Preckel et al., 2018). The brain networks involved in cognitive empathy overlap with those associated with ToM, including MPFC and TPJ. On the other hand, affective empathy primarily involves the anterior middle cingulate cortex (aMCC) and anterior insula (Fan et al., 2011; Singer et al., 2004; Zaki & Ochsner, 2012) which reflect individual emotional response (Corradi-Dell'acqua et al., 2016; Fan et al., 2011; Zaki & Ochsner, 2012). Sensorimotor areas also participate in empathic responses, such as when observing others in pain, which activates motor and somatosensory regions (Avenanti et al., 2005; Bufalari et al., 2007; Keysers et al., 2010) relevant for understanding others' bodily states (Adolphs et al., 2000; Pitcher et al., 2008). The collective activity of these brain regions contributes to eliciting compassions, which is a complementary social emotion associated with reward-related brain networks, including the basal ganglia (ventral striatum and nucleus accumbens), orbitofrontal cortex (OFC) and subgenual anterior cingulate (sgACC) (Preckel et al., 2018), and regulating prosocial behavior (e.g., Gallo et al., 2018; Zaki & Ochsner, 2012).

Given the critical involvement of brain networks related to sensing and controlling the body in various aspects of social cognition (Avenanti et al., 2013; Barsalou, 2008; Gallese & Sinigaglia, 2011; Keysers et al., 2010; Niedenthal et al., 2010; Rizzolatti & Sinigaglia, 2016; Shapiro, 2011), it is relevant to investigate the extent to which pathological conditions affecting sensorimotor control of body movement, specifically movement disorders, are associated with deficits of social cognition. This is particularly interesting since many of the neural structures affected in individuals with movement disorders overlap with the brain regions involved in social cognition or are directly connected with them. Therefore, it is worth examining these cognitive processes in people with movement disorders. Recent meta-analyses on Parkinson's disease (PD) (Coundouris et al., 2019, 2020) – a complex hypokinetic disorder characterized by slowness and paucity of movement (Abdo et al., 2010) – have reported deficits in social perception, involving the recognition of emotions from faces and prosody, as well as in the cognitive dimensions of ToM and empathy, despite an intact affective empathy. On the other hand, less attention has been devoted to synthesizing knowledge about hyperkinetic movement disorders, which comprise a diverse group of diseases characterized by excessive and involuntary movements (Abdo et

al., 2010). Therefore, our objective is to synthesize the current literature on social cognition processes in hyperkinetic movement disorders to address this knowledge gap.

We conducted a systematic review of social cognition studies in several hyperkinetic movement disorders, including Huntington's disease (HD), Tourette syndrome (TS), dystonia, and essential tremor (ET). These disorders are characterized by excessive and abnormal involuntary movements and are commonly associated with a dysfunction of the basal ganglia and associated cortical-subcortical-circuits; this dysfunction may extend to various brain regions, including the prefrontal cortex and the cerebellum (Albin et al., 1989; Den Dunnen, 2013; Jankovic, 2009).

We focused on studies investigating social perception, ToM, and empathy in hyperkinetic disorders. Social cognition impairments have been observed in these disorders, and understanding the nature and extent of these deficits is crucial for better comprehending their impact on social functioning and developing effective interventions.

This review aims to achieve the following objectives:

Present an overview of the state of the art of social cognition in hyperkinetic movement disorders, highlighting any points of convergence or divergences among the examined diseases.

Build on neuroimaging and clinical evidence to provide new insights into the brain networks and connectivity patterns underlying social cognition. In this regard, particular attention will be given to subcortical structures (i.e., basal ganglia, cerebellum), which are primarily responsible for hyperkinetic movement disorders.

Evaluate the consistency of social cognition deficits across studies to address whether the assessment of social cognition can serve as a tool for the evaluation, treatment, and prognosis of patients affected by hyperkinetic movement disorders.

2. Methods

To conduct the systematic literature review, we followed the guidelines of the PRISMA Statement (Page et al., 2021). The research protocol was registered in PROSPERO, the International prospective register of systematic reviews (CRD42022327459). The review process involved the following steps, based on the PICO framework:

Problem/Patient/Population (P): We analyze the presence of alterations in social cognition in patients with hyperkinetic movement disorders.

Intervention (I): we assessed different dimensions of social cognition, with or without the addition of neuroimaging techniques, to find correlations with brain structure and functioning.

Comparison/Control (C): We compared the results with control groups of healthy subjects.

Outcome (O): We investigated the extent and nature of social cognition alterations or deficits in patients with hyperkinetic movement disorders, along with any structural and/or functional neural correlates at the level of cortical or subcortical areas and/or connectivity between them.

To identify relevant articles, we consulted the semeiotic description of movement disorders from "Principles and practice of movement disorders" (Fahn, 2011; Fahn et al., 2011) and the ICD-11 classification (World Health Organization, 2019). These sources were cross-referenced with the three dimensions of social cognition mentioned above.

The search process involved entering the following string as a search term in the Scopus, PubMed, and Web of Science databases: (("abdominal dyskinesia" OR "akathisia" OR "athetosis" OR "ballism" OR "chorea" OR "dystonia" OR "hemifacial spasm" OR "hyperekplexia" OR "hypnogenic dyskinesia" OR "jumping disorder" OR "jumpy stump" OR "moving toes" OR "moving fingers" OR "myoclonus" OR "myokymia" OR "synkinesis" OR "myorhythmia" OR "periodic movements in sleep" OR "REM sleep behavior disorder" OR "restless legs" OR "stereotypies" OR "tics" OR "tremor") AND ("social cognition" OR "theory of mind" OR "social perception" OR empathy OR "emotion* recognition")). The string produced a total of 1137 results. To refine the search, duplicates were eliminated, and a first selection was made based on abstracts. The final selection was made after consulting the full texts, considering the following inclusion and exclusion criteria.

Inclusion criteria: (1) the research articles had to be original and published in English in peer-reviewed journals; (2) the studies needed to be conducted on patients with hyperkinetic movement disorders, based on the list of symptoms in “Principles and practice of movement disorders” (Fahn, 2011; Fahn et al., 2011) and ICD-11 (World Health Organization, 2019). The studies should have involved tasks or assessment measures related to the dimensions of social cognition considered; (3) The studies were required to provide a detailed description of tasks and assessment measures related to social cognition.

Exclusion criteria: (1) review articles were excluded; (2) case studies were not considered; (3) articles that did not pertain to the topic of interest, such as those lacking social cognition measures, movement disorders, or articles on genetics or pharmacology, were excluded; (4) Editorials or commentary were not included; (5) articles not written in English were excluded; (6) Studies without a healthy participants control group were not considered; (7) articles that were already included in a previous meta-analysis on HD (Bora et al., 2016), which highlighted deficits in social perception and ToM in individuals with HD, were excluded. Indeed, our aim was to explore the most recent or excluded developments in social cognition in this disorder.

After applying these criteria, a total of 50 articles were included. The literature search encompassed papers published until NaN Invalid Date . The PRISMA flow diagram (Figure 1) and the table of excluded articles and their reasons (Table 1) are presented below. The selected articles were categorized into paragraphs focusing on different pathological conditions, namely HD, TS, dystonia, and ET.

Figure 1. PRISMA flow diagram illustrating the selection process.

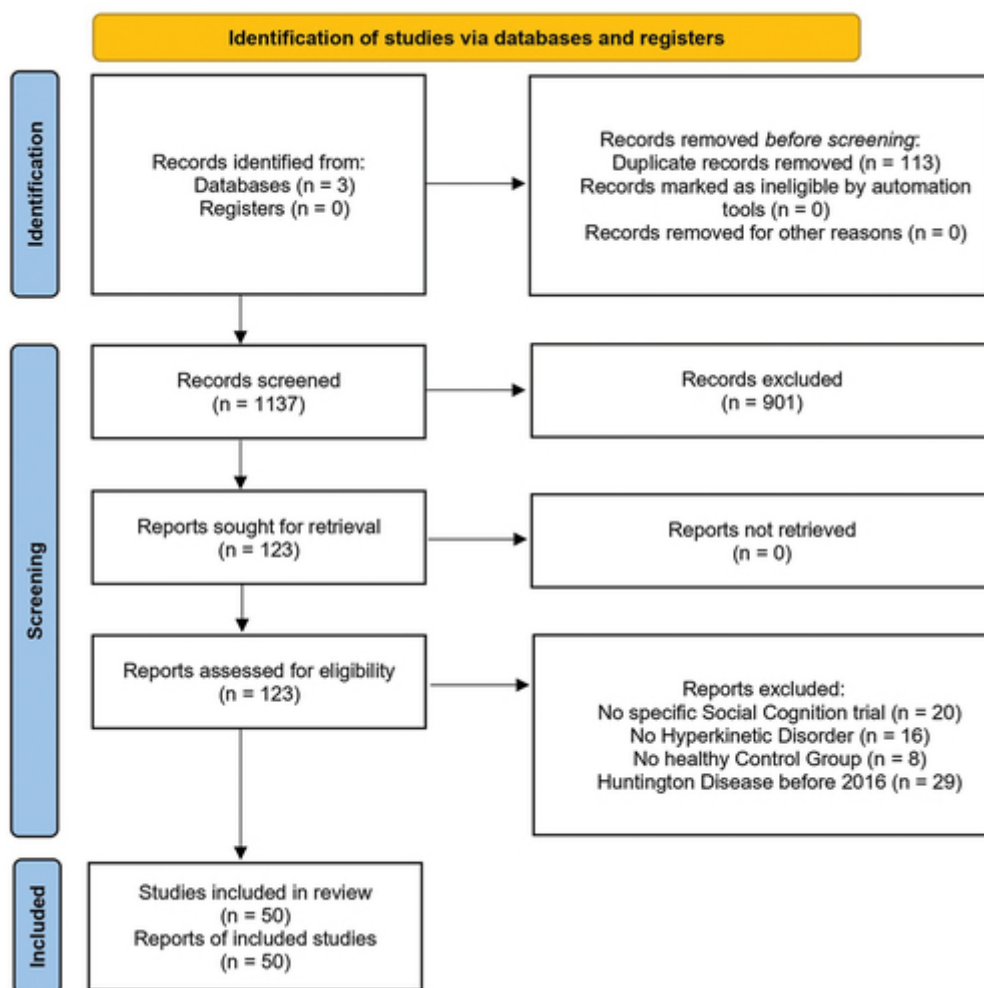


Table 1 Table 1. Social cognition impairments in hyperkinetic movement disorders.

	Social Perception	ToM	Empathy
Huntington Disease	Premanifest: individuals tend to perceive social stimuli as less salient. Manifest: difficulties in recognizing emotions, particularly negative ones. Manifest: difficulties in recognizing emotions, particularly negative ones. Manifest: difficulties in recognizing emotions, particularly negative ones.	Premanifest: ToM (cognitive and affective) worsens as motor symptoms progress. Manifest: Impairments in affective ToM (emotional attribution) and cognitive ToM (understanding others' mental states). Altered sarcasm understanding, schadenfreude, and third-party punishment.	Premanifest: Lower scores on cognitive (perspective-taking) and affective empathy (empathic distress) compared to healthy controls. Manifest: Impairments in both cognitive and affective empathy. Manifest: Impairments in both cognitive and affective empathy. Manifest: Impairments in both cognitive and affective empathy.
Tourette Syndrome	Deficits in recognizing facial expressions of anger, surprise, and sadness. Difficulty in identifying anger prosody.	Difficulties in cognitive ToM (detecting and judging faux pas, unconventional reactions to social stimuli, deficits in indirect sarcasm comprehension, hyper-mentalization). Deficits in affective ToM (reasoning about envy and gloating expressions). TS patients may not necessarily perform worse on ToM tasks but may have task-dependent gray matter alterations.	Reduced scores on cognitive empathy (perspective-taking). Increased scores on personal distress
Dystonia	Only craniocervical dystonia: difficulties in recognizing the facial expression of disgust, fear, and anger from prosody.	Only craniocervical dystonia: impairments in cognitive ToM (recognizing others' intentions) and affective ToM (attributing emotions to others in a social context).	No significant differences with controls.
Essential Tremor	Negative correlation between tremor severity and recognition of facial emotions, particularly fear, and joy.	Deficits in cognitive ToM (mental state attribution) but not in affective ToM (emotional attribution).	Unknown

2.1. Risk of bias assessment

We assessed the risk of bias using “The Scottish Intercollegiate Guidelines Network Methodology checklist: case-control study” (Scottish Intercollegiate Guidelines Network, 2012) since we reviewed non-randomized case-control studies. This tool consists of sections that evaluate the methodological quality of the studies based on sample selection methods, the validity and reliability of the measures used, control of confounding variables, and the statistical analysis methodology, which help form an overall judgment (Figure 2). The assessment primarily focused on controlling comorbidity with psychiatric and neurological diseases because their presence can influence the outcome of social cognition tasks (Rokita et al., 2018; Weightman et al., 2014). Most studies on movement disorders had a low risk of bias, while the highest risk was found in studies focusing on individuals with HD (28%). For the complete record, refer to the supplementary materials.

Figure 2. Risk of bias summary.

RISK OF BIAS		Huntington Disease	Tourette Syndrome	Dystonia	Essential Tremor	Total
(++)	Low	17 (68%)	12 (80%)	5 (71%)	3 (100%)	37 (74%)
(+)	Medium	1 (4%)	2 (13%)	2 (29%)	-	5 (10%)
(-)	High	7 (28%)	1 (7%)	-	-	8 (16%)
Total articles		25	15	7	3	50

3. Results

3.1. Huntington disease

Huntington Disease is an autosomal dominant neurodegenerative disorder caused by a mutation in the IT15 gene on chromosome 4. This mutation leads to abnormal expansion of the CAG repeat in the region coding for the protein huntingtin (HTT). This mutant HTT aggregates and accumulates within neurons, resulting in progressive neuronal dysfunction and death (Beal et al., 1986; Jimenez-Sanchez et al., 2017; Tabrizi et al., 2020; Walker, 2007). The progression of HD can be divided into three stages: premanifest, prodromal and manifest. The premanifest stage is characterized by the absence of clear symptoms. During this stage, individuals may not exhibit any noticeable signs of the disease, but genetic testing can identify the expansion of the CAG repeat. In the prodromal state, the first subtle symptoms of HD start to emerge. These symptoms can vary among individuals and may include behavioral and motor changes. The manifest stage is characterized by the progressive development of motor and cognitive symptoms until death, which usually occurs around 15–20 years after the appearance of the first symptoms (Vonsattel & DiFiglia, 1998). The classic motor symptom of HD is chorea, which involves involuntary, rapid, and arrhythmic movements. Other motor symptoms may accompany chorea, including tics. In the advanced stages or early-onset forms, bradykinesia and akinesia may become more prominent. Cognitive impairment is also a significant aspect of HD, with executive functions deficits being a common manifestation. These deficits can affect decision-making, planning, sequencing, attention and other cognitive abilities. Additionally, individuals with HD may experience psychiatric problems, including depression and apathy, which worsen as the disease progresses (for a review, see Bates et al., 2015).

Pathologically, HD is characterized by neurodegeneration of the basal ganglia, which is particularly severe in the striatum – a key structure for movement control and reward processing (Beal et al., 1986; Jimenez-Sanchez et al., 2017; Tabrizi et al., 2020). Prominent atrophy occurs mainly in its dorsal sensorimotor components (caudate nuclei and putamen) and results from the loss of GABAergic spiny projection neurons, also known as medium spiny neurons. Atrophy also occurs in the neocortex, the main input region of the striatum, and it is most pronounced in motor and premotor areas, while in advanced disease stages other brain regions become affected, including other basal ganglia nuclei and the cerebellum (Waldvogel et al., 2015). Neurotransmitter systems in the brain are also affected in HD, with a relevant decrease of GABA levels in striatum and dysregulation of other neurotransmitters, including dopamine and glutamate, has also been implicated in the pathophysiology of HD. Finally, HD is associated with mitochondrial dysfunction, oxidative stress, and impaired energy metabolism in affected neurons. These cellular abnormalities contribute to the progressive degeneration and death of neurons in the brain (Beal et al., 1986; Jimenez-Sanchez et al., 2017; Tabrizi et al., 2020; Walker, 2007).

Social cognition abnormalities in HD primarily manifest in the domain of social perception. Individuals with HD consistently experience difficulties in recognizing emotions from others' facial expressions, particularly negative emotions (Ille et al., 2011; Kordsachia et al., 2018a; Labuschagne et al., 2018; Larsen et al., 2016; Philpott et al., 2016; Sprengelmeyer et al., 2016; Trinkler et al., 2017; Unti et al., 2018; Vicario et al., 2017; Yitzhak et al., 2020). These difficulties have been extensively discussed in recent studies and were highlighted in a meta-analysis by Bora and colleagues (Bora et al., 2016). However, additional research suggest that these difficulties can also extend to the recognition of facial expressions of calmness (Hünefeldt et al., 2020) and happiness (Hünefeldt et al., 2020; Osborne-Crowley et al., 2019), as well as the recognition of emotions conveyed by body

expressions (Zarotti et al., 2019). Moreover, individuals with HD, even in the premanifest or early stages of the disease, tend to perceive a lower salience of social stimuli. When looking at a face, they exhibit fewer fixations and less time scanning the eye region, which is crucial for recognizing various expressions (Kordsachia et al., 2018b).

Emotion recognition impairments are further supported by a recent study using regression-based normative data, which showed that 80% of people with HD perform worse in emotion recognition than healthy participants (Vogel et al., 2022). It should be noted that while individuals with HD demonstrate reduced ability to recognize emotional faces and provide lower intensity ratings of facial expressions compared to controls, they tend to give higher intensity ratings for affective scenes across different emotions. This suggests various impairments in emotion recognition and emotion experience in HD (Ille et al., 2011). Notably, individuals with HD also exhibit reduced spontaneous facial mimicry when faced with others' emotions and demonstrate reduced voluntary imitation (Kordsachia et al., 2018a; Trinkler et al., 2017). The impairment in emotion recognition is correlated with gray matter volume in the caudate as well as in areas previously associated with shared action representations, such as the somatosensory, posterior parietal, pSTS, and subcentral sulcus regions, as shown by voxel-based morphometry (VBM) (Trinkler et al., 2017). On the other hand, studies have reported no correlation between emotion recognition deficits and alexithymia in individuals with HD (Trinkler et al., 2017). Alexithymia refers to a deficit in the cognitive processing of emotions, causing difficulties in identifying and verbally describing experienced feelings and emotions by oneself and others (Barchetta et al., 2021; Craparo et al., 2016; La Rosa et al., 2022; Martino et al., 2021; Taylor & Bagby, 2013). This suggests that the emotion deficit in HD is unrelated to alexithymia but might be tied to the "motoric level" of emotion expression (Trinkler et al., 2017).

Emotion recognition deficits in HD are generally observed across all negative emotions, but particularly compromised in the recognition of fear, disgust, and anger (Bora et al., 2016). These deficits have been reported in both pre-manifest and manifest HD individuals (Bora et al., 2016). Early studies have also shown that emotional experience itself can be affected, as indicated by research on disgust (e.g., Hayes et al., 2007; Mitchell et al., 2005). Interestingly, individuals with HD also struggle with recognizing trustworthiness and dominance from observed facial expressions (Sprengelmeyer et al., 2016). These deficits have been associated with reduced fractional anisotropy, primarily in the MPFC, middle frontal gyrus, anterior corpus callosum, left somatosensory cortex, bilateral insula, amygdala, cerebellum, and pons (Sprengelmeyer et al., 2016).

Furthermore, intranasal oxytocin administration has shown promise in improving social perception of disgust in people with HD, bringing it to levels similar to those of control participants. This improvement is associated with increased activity in the putamen and middle frontal gyrus (Labuschagne et al., 2018). Low levels of oxytocin in individuals with HD have also been found to correlate with poor performance in social perception and ToM tasks (Unti et al., 2018). These findings suggest that oxytocin administration may have a therapeutic potential for improving social cognition in HD.

In their meta-analysis, Bora et al. (2016) reported consistent difficulties in ToM tasks, such as the Reading the Mind in the Eyes Test (RMET) and faux pas recognition, in people with HD. They also found a trend for impairment in individuals with premanifest HD. Subsequent studies have further supported these findings, showing deficits in cognitive and affective ToM in individuals with HD (Bayliss et al., 2019; Belardinelli et al., 2019; Brüne et al., 2021; Eddy et al., 2018; Unti et al., 2018). These deficits are not accounted for by impaired global cognitive functioning, at least in HD patients in mild to moderate disease stages (Lagravinese et al., 2017).

An fMRI investigation conducted by Eddy and colleagues showed reduced RMET performance in people with HD, associated with reduced activation of dorsolateral prefrontal-parietal areas involved in executive functions (Eddy et al., 2018). This study also reported reduced activations in the left insula and supramarginal gyrus in manifest HD and increased activations in MPFC/anterior cingulate cortex (ACC) in premanifest HD (Eddy et al., 2018). People with premanifest HD have been reported to show changes in eye movements, including alterations in the number of fixations, average fixation time, and percentage of time spent fixating images during performance of the RMET (Olivetti Belardinelli et al., 2021). Additionally, individuals with premanifest HD have been reported to experience a decline in social perception and ToM abilities, alongside the progression in motor symptom severity (Bayliss et al., 2019; Belardinelli et al., 2019; Eddy et al., 2018). It is important to note, however, that certain studies have also found preserved emotional attribution skills in individuals with premanifest HD (Zarotti et al., 2018).

Other investigations of ToM abilities and social reasoning in individuals with HD have reported impairments in understanding sarcasm. One study found that only “simple sarcasm”, where the described situations imply the opposite meaning to what is being said (Philpott et al., 2016), is affected (Larsen et al., 2016), while another also identifies abnormalities in recognizing “paradoxical sarcasm”, which refers to sentences where the exchange of words is meaningless unless one understands the sarcasm involved (Philpott et al., 2016). The larger sample size in the first study suggests that individuals with HD have greater difficulty identifying simple sarcasm, which may be attributed to challenges in perceiving the emotional nuances during social interactions.

Recent research has expanded the exploration of ToM in HD to include neuroeconomic games such as the ultimatum game and dictator game. These games assess the appreciation of fairness rules and reciprocity (Brüne et al., 2021). Interestingly, people with HD tend to reject unfair offers in the ultimatum game similar to control subjects but tend not to punish unfairness observed from a third-party perspective. This diminished altruistic punishment correlates with deficits in executive functioning, which is related to social decision-making (Lucifora et al., 2021c), social cognition and empathy (Eslinger et al., 2011).

A few studies have investigated empathy in people with manifest and premanifest HD compared to control participants using self-report empathy questionnaires (Maurage et al., 2016; Puig-Davi et al., 2021). Maurage et al. (2016) used the Empathy Quotient Scale and found that people with manifest HD reported lower scores on cognitive empathy scales, while affective empathy scores appeared similar to healthy controls (Maurage et al., 2016). Individuals with premanifest HD showed no significant difference compared to healthy controls in both cognitive and affective empathy scores (Maurage et al., 2016). Puig-Davi et al. (2021) administered the Cognitive and Affective Empathy Test (TECA) and found that individuals with premanifest HD scored lower on perspective-taking (cognitive empathy) and empathic distress (affective empathy) subscales. In contrast, lower scores in almost all TECA subscales were observed in early manifest HD individuals. This study also demonstrated an association between empathy measures and decreased gray matter volume and cortical thickness in several brain regions, including frontotemporal, parieto-occipital, basal ganglia, and limbic regions.

A recent profiling method by Turner et al. (2022) reported that a large proportion of people with premanifest HD fall below healthy volunteers’ median values on ToM (>80%), social perception (>57%), empathy (>54%), and social behavior (>40%) (Turner et al., 2022). However, only a small proportion of these individuals displayed marked problems with social cognition, highlighting the need for individual-level assessments.

Finally, Baez et al. (2016) found lower presence of *schadenfreude* (the pleasure in others’ misfortunes) in people with HD, which may be linked to anomalies in the cortico-striatal reward system (Baez et al., 2016). Lower *schadenfreude* ratings were associated with lower gray matter volume in the bilateral ventral striatum (including the nucleus accumbens) and the right superior parietal lobule and precuneus in people with HD (Baez et al., 2018).

3.2. Tourette syndrome

Tourette Syndrome (TS) is a childhood-onset neurodevelopmental disorder characterized by the presence of tics, which are repetitive and patterned movements and vocalizations that escape volitional control despite being potentially inhibited by subjects on demand (Ganos & Martino, 2015; Vicario et al., 2016). Motor and vocal tics can vary in complexity, ranging from simple movements like excessive blinking to more elaborate actions such as repeatedly touching objects. Similarly, vocal tics can be as simple as clearing the throat excessively or as complex as repeating words and phrases. In some cases, tics may lead to echolalia or, less frequently, coprolalia. The defining characteristic of tics is the sense of urgency that accompanies them, which presents as an intrusive and premonitory sensation and dissipates once the tics are performed. Tics typically emerge around the age of 6–7 and reach their peak during early adolescence. However, symptoms may gradually diminish in certain instances and even disappear in adulthood (Müller-Vahl et al., 2019).

The disorder is believed to involve dysfunction in the cortico-basal ganglia-thalamo-cortical circuits, which play a crucial role in motor control and sensorimotor integration (Ganos & Martino, 2015). Disruptions in the balance of neurotransmitters, particularly dopamine and gamma-aminobutyric acid (GABA), within these circuits have been implicated in the development of tics (Ganos, 2016). The increased excitability of brainstem interneurons, as observed in studies on blink reflex and startle reflex, suggests abnormalities in the brainstem and subcortical structures (Berardelli et al., 2003). Some animal models suggest involvement of cholinergic and histaminergic neurotransmission in addition to GABAergic inhibition disruption (Ganos, 2016). Neuroimaging studies have

consistently observed abnormalities in the cortico-striatal dopaminergic circuitry of individuals with TS (Felling & Singer, 2011; Plessen et al., 2009). These anomalies manifest as asymmetry or reduction in the putamen and caudate nucleus, as well as cortical hyperactivity in the insula, supplementary motor area (SMA), ACC, and parietal operculum. These brain regions are implicated in generating the sense of urgency that precedes tic expression (Gagné, 2019; Plessen et al., 2009). Disruption of cortico-cerebellar regulatory loops has also been suggested based on the findings of reduced volumes of cerebellar structures (Tobe et al., 2010; see also Eidelberg et al., 1998; Schirinzi et al., 2018; Brüggemann, 2021).

Regarding social perception, Mermillod et al. (2013) found that individuals with TS have deficits in recognizing emotional facial expressions of anger, surprise, and sadness. However, Drury et al. (2012) did not observe differences in facial emotion recognition in children and adults with TS, but did observe difficulty in identifying anger prosody in adults with TS.

In a study conducted by Neuner et al. (2010), individuals with and without TS were examined using fMRI to perform a gender recognition task involving emotional faces (Neuner et al., 2010). The study found that individuals with TS exhibited hyperactivity in the left amygdala compared to controls. This hyperactivity was negatively correlated with the extroversion trait and involved various brain areas including the MFC, dorsolateral prefrontal areas, inferior frontal gyrus, medial temporal gyrus for all the emotions presented (Neuner et al., 2010).

Another study by Kalsi et al. (2019) investigated a small sample of children with TS using EEG. They found that children with TS showed shorter latency of the P1 and N170 components of event-related potentials (ERPs) in response to anger faces compared to controls. Source reconstruction suggested greater activation in the occipital cortex and lower activation in the amygdala, temporal cortex, and cingulate cortex in response to anger stimuli in children with TS (Kalsi et al., 2019). However, given the small sample size and the limitations of EEG in recording deep nuclei activity, the generalizability of these findings is challenging.

Studies have also investigated the role of the insula in TS during emotion perception tasks. Rae et al. (2018) found hyperactivation of the anterior insula in individuals with TS compared to controls, both for angry and neutral faces. This hyperactivation was associated with increased functional connectivity of the insula with sensorimotor areas involved in the generation of tics such as the premotor cortex, SMA, primary motor cortex and putamen. Furthermore, increased connectivity of the insula with the globus pallidus and thalamus correlated with tic severity, while the SMA correlated with premonitory urgency (Rae et al., 2018). These findings suggest that individuals with TS may exhibit hypersensitivity toward interoceptive experiences associated with social stimuli due to the altered connectivity with striatal-corticothalamic circuits involved in symptom generation.

Six studies reported ToM impairments in individuals with TS (Channon et al., 2012; Drury et al., 2018; Eddy & Cavanna, 2015; Eddy et al., 2010a, 2010b, 2011). These studies have reported various deficits. One study by Channon et al. (2012) found that individuals with TS alone have difficulties detecting faux pas (social errors) but can accurately explain detected faux pas. Similarly, Eddy et al. (2010b) reported that individuals with TS alone do not have problems explaining noticed faux pas but have difficulty judging whether a faux pas has occurred (Eddy et al., 2010b). However, in a mixed TS sample, individuals had increased difficulties in explaining faux pas even when accurately detected, and it is unclear if this also applies to the subset with TS alone (Eddy et al., 2010a). Individuals with TS may exhibit unconventional responses to social stimuli. For example, they may have atypical reactions to unfair offers in the ultimatum game and interpret humorous material unconventionally (Eddy et al., 2011). Consistent with this, people with TS may show deficits in comprehending indirect sarcasm (Drury et al., 2018) and a tendency to hyper-mentalization, attributing meaningful behaviors to random movements of triangular figures, which has been linked to an overactivity of the dopaminergic system (Eddy & Cavanna, 2015).

Other studies found no significant differences in ToM task performance in individuals with TS when compared to controls (Channon et al., 2004). Instead, they showed substantial differences in the brain's structural, functional, and connectivity levels using brain imaging techniques (Eddy et al., 2016, 2017). In particular, it was noted that functional alterations at the level of the rTPJ, posterior cingulate cortex and right amygdala during a ToM task involving reasoning about false belief (Eddy et al., 2016). Activity in rTPJ correlated with the symptoms of echophenomena (imitation of other people's speech and actions), and altered impulse control, whereas activity

in rTPJ and amygdala correlated with tic urgency. This suggests a possible affective component in the generation of tics and a difficulty in distinguishing one's perspective from that of others, due to altered activity of the rTPJ (Eddy et al., 2016).

Regarding affective ToM, Eddy et al. (2011) suggested that people with TS had difficulties reasoning about envy and gloating expressions, as measured by the Socially competitive emotions task (SCET). The authors also reported deficits in interpreting facial expressions, evaluated by the RMET. During an RMET task, individuals with TS exhibited altered brain activity compared to controls. Specifically, increased activity emerged in the left orbitofrontal cortex (OFC), posterior cingulate, right amygdala and rTPJ. Conversely, reduced activity was found in the left inferior parietal cortex. These findings suggest that individuals with TS have altered processing of predominantly negative emotions and mentalizing abilities (Eddy et al., 2017).

Two studies assessed empathy in people with TS using the Interpersonal Reactivity Index (Channon et al., 2004; Eddy et al., 2015). Channon et al. (2004) found no significant differences, but Eddy et al. (2015) with a larger sample reported that individuals with TS showed a reduced tendency to assume the others' perspective (perspective taking, PT scale) and a high degree of distress in interpersonal situations (personal distress, PD scale) compared to controls. However, no significant differences emerged in other affective empathy (Empathy Concern) and cognitive empathy (Fantasy) subscales.

3.3 Dystonia

Dystonia is a movement disorder characterized by sustained or intermittent muscle contractions that lead to abnormal, often repetitive movements, postures, or both (Albanese et al., 2013). It encompasses various symptoms, including torticollis, limb and trunk dystonia, writer's cramp, blepharospasm, and spastic dysphonia (Breakefield et al., 2008). Isolated adult-onset dystonia includes different types of dystonia, such as cranial dystonia (e.g., blepharospasm, oromandibular, and lingual dystonia), cervical dystonia, laryngeal dystonia, and hand dystonia (Phukan et al., 2011). The involvement of basal ganglia deficits in developing dystonia is strongly supported by several lines of evidence derived by from animal models (Jinnah et al., 2017). Basal ganglia lesions resulting from brain injuries, metabolic diseases, and neurodegenerative conditions are linked to secondary dystonia (Kojovic et al., 2013). Studies on people with dystonia undergoing deep brain stimulation (DBS) have demonstrated abnormal neural firing patterns, synchronized oscillations, and expanded receptive fields within the basal ganglia-thalamic circuit (Gatev et al., 2006; Hendrix & Vitek, 2012). Finally, there is evidence that dystonia may be considered a network disorder involving not only the cortico-basal ganglia networks but also the cerebellar pathways (Cerasa & Quattrone, 2016; Louis, 2018; Mavroudis et al., 2021; Tewari et al., 2017), as well as collicular – pulvinar–amygdala subcortical networks (Rafee et al., 2021).

Regarding social cognition, a few studies investigated the impairments and differences in emotion recognition in people with dystonia. Individuals with cranial or cervical dystonia have shown impairments in recognizing negative emotional expressions, particularly the facial expression of disgust (Rinnerthaler et al., 2006). Additionally, they may experience difficulties in recognizing anger from prosody, which relates to the dimension of arousal and valence, as assessed through self-report measures during a listening task (Nikolova et al., 2011). Another study focusing on young individuals (9 children and 12 adolescents) with myoclonus dystonia, a form of early-onset dystonia primarily affecting the upper limbs, found no differences in emotion recognition compared to controls. However, they discovered that a mutation in the ϵ -sarcoglycan gene (SGCE) was associated with greater difficulty in emotion recognition (Coenen et al., 2021). Moreover, a study specifically investigating individuals with cervical dystonia identified significantly more difficulties in recognizing fearful faces in this group (Ellement et al., 2021).

While these findings suggest altered recognition of facial emotion expressions in cranial or cervical dystonia, but not when dystonia affects the upper limbs, it is important to note that there are also discrepant findings that may attributed to the various instruments used in these studies. For instance, people with cervical dystonia exhibited lower performance than the standard norms on naming facial expressions and emotional prosody when assessed using subtests of the Florida Affect Battery (Burke et al., 2020). However, most patients showed normal performance on social cognition subtests from the Wechsler Adult Intelligence Scale IV (Ellement et al., 2021). Furthermore, research indicate that a group of patients with a form of dystonia primarily affecting the upper limbs, demonstrate brain activation patterns similar to controls when exposed to emotional faces or intense emotional scenes (Espay et al., 2018a). In contrast, individuals with functional dystonia, a type of dystonia that arises from psychogenetic causes and affects various body parts, showed distinct changes in

brain activation compared to both healthy controls and individuals with organic dystonia (Espay et al., 2018a). These changes were primarily observed in visual areas such as the occipital cortex and fusiform gyrus, as well as sensorimotor regions, during observation of emotional faces and extremely disgusting or offensive scenes (Espay et al., 2018a).

Social perception deficits in dystonia are thought to result from altered connections between subcortical structures, such as the basal ganglia and cerebellum, and cortical regions involved in social perception. However, scholars have also proposed that changes in collicular – pulvinar–amygdala subcortical networks could underlie non-motor symptoms in dystonia, including social perception deficits (Rafee et al., 2021). Moreover, deficits in social perception abilities can be associated with the psychiatric comorbidity profile. Specifically, better performance on an Affect Naming task was linked to comorbid and more severe anxiety in individuals with cervical dystonia (Ellement et al., 2021).

Recently, studies have investigated affective and cognitive ToM in individuals with dystonia. Lagravinese et al. (2021) investigated different motor phenotypes in cervical dystonia (Lagravinese et al., 2021) and hypothesized that the malfunctioning cerebral network nodes might differ between different dystonia phenotypes. Specifically, they suggested that abnormal cerebellar processing could contribute to the motor phenotype with tremors (Avanzino et al., 2018, 2020; Bologna & Berardelli, 2017; Prudente et al., 2014). The study's results showed that both affective and cognitive aspects of ToM were impaired in people with cervical dystonia (Lagravinese et al., 2021). Individuals with cervical dystonia and tremors showed more significant impairments in cognitive ToM compared to those without tremors, suggesting that the cerebral network responsible for motor and non-motor impairments might be more widespread in patients with tremor (Lagravinese et al., 2021). Another study by Czekóová et al. (2017) examined deficits in recognizing social faux pas in cervical dystonia. They found that individuals with cervical dystonia mistakenly perceived behavior as inappropriate more frequently than controls in situations where no faux pas had occurred, suggesting an impairment in ToM and the ability to recognize social norms (Czekóová et al., 2017). Finally, a recent study quantified the proportion of individuals with cervical dystonia who perform below normative values on ToM tests (Ellement et al., 2021). The study revealed that only about 22% of these patients underperformed in the domain of social cognition (Ellement et al., 2021). Consistent with this, a study highlighted the tendency of people with cervical dystonia to present impairment in basic social perception tasks (naming facial affect and emotional prosody) but not in tasks related to ToM, like the RMET (Burke et al., 2020). Also, in most people with cervical dystonia cognitive, despite deficits in social perception and some aspects of TOM, cognitive and affective empathy appears comparable to that of healthy controls (Czekóová et al., 2017; Ellement et al., 2021).

3.4. Essential tremor

Essential Tremor (ET) is a common movement disorder that primarily manifests with upper limb postural and kinetic trembling. These tremors can be observed during daily activities and are elicited during neurological examination using various maneuvers (Louis, 2019). The diagnosis of ET requires the tremors to persist for at least three years and not be associated with other conditions such as dystonia, ataxia, or Parkinson's disease. The pathophysiology of ET remains poorly understood. Post-mortem examinations, neurophysiological investigations, and animal studies suggest the involvement of a cerebello-thalamo-cortical network encompassing the cerebellum, brainstem, thalamus, and sensorimotor areas of the cortex (Hallett, 2014; Hopfner & Deuschl, 2018). Functional imaging studies have consistently shown cerebellar hyperactivity during rest and during tremors (Sharifi et al., 2014). Several studies have also shown alterations in the Purkinje cells and basket cells of the cerebellum, and the olive-cerebellar climbing fibers, along with possible compensatory mechanisms involving hypertrophy of the GABAergic basket cells to restore inhibitory cerebello-cortical function. When this compensatory mechanism becomes insufficient, the symptoms and progression of the disease can occur (Louis & Faust, 2020).

In the domain of social perception, researchers have found an inverse relation between tremor severity in ET and facial emotion recognition (Auzou et al., 2014). Another study did not find activation differences between individuals with ET and healthy controls during an emotion perception task (Espay et al., 2018b), although it reported greater volume in the right amygdala and reduced volume in the left cerebellum and occipital pole in the ET group (Espay et al., 2018b).

Individuals with ET exhibited deficits in tasks requiring the attribution of mental states (cognitive ToM), but not in tasks requiring emotion attribution (affective ToM) (Santangelo et al., 2012). These deficits persist even when controlling for memory performance (Santangelo et al., 2012).

Finally, research on empathy function in ET is currently lacking.

4. Discussion

In this article, we present a systematic reviewed of 50 published studies that investigate social cognition in hyperkinetic movement disorders. Our comprehensive analysis of these studies reveals consistent evidence of altered social cognition in individuals with these neurological conditions. The key findings and associated social cognition impairments for each movement disorder are summarized in Table 1.

4.1. Altered social cognitive functions in hyperkinetic movement disorders

Our review highlights social perception deficits in people with manifest HD. These individuals have difficulties recognizing emotions, particularly negative ones, and exhibit deficits in ToM (emotional attribution, understanding of others' mental states, understanding of sarcasm), social decision-making, and both cognitive and affective empathy. They also tend not to experience *schadenfreude* due to alterations in the reward circuit. People with premanifest HD show worsening social perception and ToM as their motor symptoms progress. They also perceive a lower salience to social stimuli and tend to score lower on perspective-taking and empathic distress compared to healthy controls. These deficits in social cognition in premanifest HD are usually mild to moderate and progress as the disease advances.

People with TS may exhibit altered cognitive ToM, although not all studies confirm this. Some cognitive impairments observed in TS include difficulties detecting faux pas, unconventional reactions to social stimuli, deficits in indirect sarcasm comprehension. Additionally, people with TS may show hyper-mentalization but deficits in affective ToM, particularly in reasoning about envy and gloating expressions. Social perception deficits, such as difficulties recognizing emotional facial expressions and identifying anger prosody, have also been observed. Neuroimaging studies have shown hyperactivity in several brain regions, including the left amygdala and prefrontal areas, during emotion recognition tasks individuals with TS. Additionally, these individuals exhibit increased functional connectivity between insula and sensorimotor areas involved in tic generation. Furthermore, empathy research show that individuals with TS may show reduced perspective-taking ability and high social distress.

People with dystonia, specifically cranial and cervical dystonia, exhibit difficulties in the recognition of negative emotions, such as facial expression of disgust or fear and emotional prosody of anger. These deficits are not observed in dystonia affecting the upper limbs. Additionally, individuals with cervical dystonia show deficits in cognitive ToM tasks requiring to recognize other people's intentions. ToM performance correlate with deficits in executive functions, suggesting a contribution of domain-general abilities in explaining ToM impairments, in line with the literature in healthy individuals (Carlson et al., 2002) and other clinical populations (Roca, 2016). On the other hand, cognitive and affective empathy appear intact in people with dystonia.

People with ET experience specific impairments in their cognitive abilities related to the understanding of other people's mental states (cognitive ToM), while their emotional attribution abilities (affective ToM) remains intact. These individuals may exhibit altered social perception, as evidenced by a negative correlation between the severity of tremors and the recognition of facial emotions, particularly fear, and joy. However, there is currently a lack of research on empathy specifically related to ET.

4.2. Altered brain networks in hyperkinetic movement disorders

Overall, the reviewed evidence from HD, TS, dystonia and ET consistently indicates the presence of social perception and ToM deficits in these conditions. These deficits can be at least partially attributed to alterations in the basal ganglia and cerebellum and their pattern of connectivity with other subcortical and cortical regions (Fahn et al., 2011).

HD is characterized by basal ganglia degeneration, which occurs many years before the onset of symptoms (Tang et al., 2019). Imaging studies have revealed progressive degeneration in the striatum and other brain areas, with the caudate and putamen exhibiting the greatest and earliest degeneration (Tang et al., 2019). Moreover, cerebellar anomalies are common in HD (Franklin et al., 2021; Padron-Rivera et al., 2021).

TS is marked by alterations in the cortico-striatal dopaminergic circuitry, with reduction or asymmetry of the putamen and caudate nuclei, along with cortical hyperactivity in specific regions associated with the feeling of urgency preceding tic expression. These regions include the insula, SMA, ACC, and parietal operculum (Gagné, 2019). Also, alterations of the cerebellum have been reported in individuals with TS (Lerner et al., 2007; Tikoo et al., 2021).

Several lines of evidence, including research in animal models (Neychev et al., 2011), support the role of basal ganglia deficits in dystonia development. It has been proposed (Tewari et al., 2017) that dystonia may represent a network disorder involving not only cortico-basal ganglia networks but also cerebellar pathways. Abnormalities in cerebellar structural, function, and connectivity with other areas have been found in people with dystonia, along with the finding of a decrease in symptoms following cerebellar transcranial magnetic stimulation (TMS) (Tewari et al., 2017). Additionally, collicular – pulvinar–amygdala subcortical networks could also play a role in dystonia and contribute to social cognition deficits (Rafee et al., 2021).

In people with ET, alterations have been identified in the cerebellum, brainstem, thalamus, and sensorimotor areas of the cortex (Hopfner & Deuschl, 2018; Louis & Faust, 2020). A compensatory mechanism involving GABAergic cerebellar circuits aims to restore the inhibitory function of the cerebellum on the cortex. However, when this compensatory mechanism becomes insufficient, symptoms and disease progression occur (Louis & Faust, 2020).

In summary, hyperkinetic movement disorders are associated with social perception and ToM deficits. These deficits can be at least in part attributed to alterations in the basal ganglia and/or cerebellum, along with involvement of other subcortical and cortical regions, with distinct patterns of impairments observed in different clinical categories. For example, individuals with ET display deficits in cognitive ToM but maintain intact affective ToM (Santangelo et al., 2012). Moreover, impaired emotion recognition is found only in individuals with more severe tremors. This contrasts with individuals with HD, TS, and cervical dystonia, who exhibit impairments in social perception and both cognitive and affective domains of ToM, although to a different degree. These differences could be attributed to the minor involvement of the basal ganglia in ET's pathophysiology (Bhalsing et al., 2013; Klaming & Annese, 2014), with little or no alterations in motor and limbic dopaminergic pathways associated with these subcortical regions (Di Giuda et al., 2012). On the other hand, the observed deficit in cognitive ToM in ET could be associated with dysfunction in the prefrontal-cerebellar circuit, as suggested by Cerasa and Quattrone (2016).

4.3. Functional role of subcortical regions in social cognition

Traditional attempts to identify the neural correlates of social cognition have often overlooked the relevance of subcortical structures, with the exception of the amygdala, which is well known for its role in emotions and social perception (Adolphs, 2010; Adolphs et al., 2001; Calder, 1996). Our systematic review of social cognition deficits in hyperkinetic motor disorders emphasizes an important role played by the basal ganglia and cerebellum. We report that these subcortical regions, crucial for motor control, are critical in emotion recognition-based social perception tasks, as evidenced by impaired performance across individuals with HD, TS, cervical dystonia and severe ET. Interestingly, similar impairment has been observed in hypokinetic movement disorders associated with basal ganglia degeneration, such as PD, with reduced facial mimicry and reduced recognition of emotions in facial expressions and prosody (Coundouris et al., 2019). Taken together these results are relevant for embodied-grounded cognition models. These models propose that cognitive processes, including those involved in emotion perception, are deeply rooted in sensorimotor experiences and bodily interactions with the environment (Barsalou, 2008; Borghi et al., 2013; Gallese & Sinigaglia, 2011; Niedenthal et al., 2010; Pezzulo et al., 2011; Shapiro, 2011). In this vein, impaired sensorimotor processing would make observers less able to motorically simulate perceived facial or vocal emotional expressions, leading to poor emotion recognition (Borgomaneri et al., 2020; Niedenthal et al., 2010; Wood et al., 2016). Therefore, the pattern of social perception deficits following alteration of motor control in hyperkinetic movement disorders provide clinical support to these theoretical models.

Another major result of our review is that hyperkinetic movement disorders show impairments even in more abstract and complex cognitive processes related to ToM and empathy. Also in this case, this impairment is also shared by hypokinetic movement disorders, as people with PD show impairments in cognitive and affective ToM (Coundouris et al., 2020). In hyperkinetic disorders, the ToM deficit mainly refers to the cognitive component in ET, whereas for cervical dystonia, and TS and HD in particular, it also involves affective components. Regarding empathy, people with HD and TS report reduced perspective taking (cognitive empathy). On the other hand, personal distress (a disposition negatively associated with more mature forms of affective empathy) seems to be enhanced in TS. This different pattern of results is likely due to different dysfunctions in brain regions involved in social cognition and emotional processing such as the amygdala, dorsal striatum, and insula.

While the functional relevance of basal ganglia and cerebellum to motor control is well established, there is a growing recognition of their importance in affective and cognitive functions. These subcortical structures, along with their direct connections to the cortex, form an expanded limbic and associative circuit (Bostan & Strick, 2018; Pierce & Péron, 2020). The basal ganglia have a fundamental role in action selection and modulation of movement vigor, but contemporary accounts of their function, suggest a general role in controlling, or “gating”, the influx of signals from other cortical areas to the prefrontal cortex (Frank et al., 2004; Pierce & Péron, 2020; Stocco et al., 2010), which could explain the impact of basal ganglia degeneration in specific cognitive deficits, including cognitive aspects of ToM and empathy found in hyperkinetic motor disorders. The basal ganglia are also involved in the reward circuitry, contributing to various cognitive and emotional functions (Bostan & Strick, 2018; Pierce & Péron, 2020; Sesack & Grace, 2010). They are modulated by dopaminergic signals and play an active role in behavioral reinforcement mechanisms (Montague et al., 2004). The basal ganglia are known to contribute to learning through stimulus-response associations and influence decision-making (Pennisi et al., 2023; Vicario et al., 2020a). They also play a role in predicting stimulus salience and contribute to the production of positive emotions (Schultz, 1998, 2015). They are also implicated in cognitive functions such as time perception (Rao et al., 2001; Vicario et al., 2020c). As suggested by the results of our work, some deficits in social cognition, like the perception of social stimuli, might depend on imbalances in the reward circuitry, as observed for the lack of *schadenfreude* in HD patients.

Evidence of a link between basal ganglia and deficits in social cognition also comes from research on other diseases where dysfunction of the basal ganglia is likely to occur. For instance, Wilson’s disease, a condition characterized by degenerative changes in the basal ganglia, has been associated with impairments in the recognition of anger, fear, disgust, as well as mentalization abilities (Peyroux et al., 2017). Similarly, research has indicated that schizophrenia is characterized by aberrant patterns of basal ganglia activation (Pierce & Péron, 2020) and deficits in social cognition, including face and prosody perception, affect sharing, mentalizing, emotion experience and emotion regulation (Green et al., 2015; Yu et al., 2014). Furthermore, autism, which is characterized by compromised reasoning about intentions and emotions (Boucher, 2012) and reduced empathy (Baron-Cohen, 2010), has been linked to dysfunction in the basal ganglia (Subramanian et al., 2017).

Another essential structure involved in the pathophysiology of hyperkinetic movement disorders is the cerebellum. Indeed, cerebellar anomalies are common in HD (Franklin et al., 2021; Padron-Rivera et al., 2021), TS (Lerner et al., 2007; Tikoo et al., 2021), dystonia (Brüggemann, 2021; Eidelberg et al., 1998; Schirizzi et al., 2018), and ET (Cerasa & Quattrone, 2016; Louis, 2018; Mavroudis et al., 2021). Recent research has shed light on the link between the cerebellum and social cognition, indicating its role beyond motor functions (Sokolov, 2018; Van Overwalle et al., 2020). A meta-analysis involving over 350 fMRI studies revealed that the cerebellum is activated during tasks related to social cognition, such as observing human actions, inferring others’ intentions, and forming inferences about personality traits and abstract thinking (Van Overwalle et al., 2014). Leggio and Molinari (2015) have shown how the cerebellum can play a central role in predictive processes, generating internal models based on temporal and spatial patterns, which are fundamental for mentalizing and constructing models of sequences of events. Studies have also highlighted the connectivity between the cerebellum and cortical areas responsible for social cognition, such as the TPJ, MPFC, OFC and the posterior STS (Nguyen et al., 2017; Sokolov et al., 2012). Non-invasive stimulation techniques, like repetitive TMS (rTMS) and transcranial direct current stimulation (tDCS), have shown the potential to improve performance in social perception tasks. For example, high-frequency rTMS (20 Hz for 15 min) applied to the cerebellar vermis enhances reactivity to positive emotional stimuli, such as happy faces (Schutter et al., 2009), while anodal and cathodal tDCS (2 mA for 20 min) applied bilaterally in the cerebellar hemispheres reduces the reaction time in identifying negative emotions from faces (Cattaneo et al., 2021; Ferrucci et al., 2012).

Similarly, our review highlights associations between deficits in social perception and other sensorimotor areas, such as the premotor and motor areas, which are considered key nodes of the Mirror Neuron System (Keysers

et al., 2010; Rizzolatti & Sinigaglia, 2016). Non-invasive brain stimulation studies in healthy participants have demonstrated that interfering with these regions impairs social perception (Avenanti & Urgesi, 2011; Paracampo et al., 2017, 2018a, 2018b). Interestingly, enhancing the same regions can improve social perception (e.g., Avenanti et al., 2018). This suggests the possibility of using brain stimulation techniques to address social perception deficits in patients with movement disorders. These techniques have been successfully used to promote neural plasticity and sensorimotor functioning in healthy humans and clinical populations (Avenanti et al., 2012; Casula et al., 2023; Lefaucheur et al., 2020; Markovic et al., 2021; Vicario et al., 2020b, 2020d).

Given the altered connectivity patterns observed in hyperkinetic movement disorders, it could be particularly interesting to test the efficacy of TMS protocols aimed at strengthening cerebellar-cortical and cortico-cortical connectivity (Chiappini et al., 2018, 2020; Lu et al., 2012; Turrini et al., 2022, 2023c). One promising approach is the use of cortico-cortical paired associated stimulation (ccPAS), which has been successfully applied to enhance brain connectivity (Buch et al., 2011; Trajkovic et al., 2023) and improve motor functions (Fiori et al., 2018; Turrini et al., 2023b, 2023a) and perceptual functions in humans (Di Luzio et al., 2022; Romei et al., 2016), including social perception functions (Borgomaneri et al., 2023). By modulating the neural circuits involved in social perception, ccPAS may offer a potential strategy to address social cognition deficits in clinical populations characterized by altered connectivity.

4.4. Methodological issues, limitations, and future perspectives

In order to understand the extent to which deficits in social cognition may be related to general cognitive disorders, such as executive functions, studies included in the review have investigated the correlation between social cognition tests and executive function tests. For instance, Burke et al. (2020) found a correlation between social cognition tests, such as the RMET, and encoding and executive skills in dystonia. Similarly, Czekóová et al. (2017) reported a correlation between cognitive ToM, working memory, and semantic verbal fluency (Czekóová et al., 2017). However, most studies on TS do not show a relationship between executive functioning and social cognition (Channon et al., 2004, 2012; Drury et al., 2018; Eddy et al., 2010a, 2010b, 2011). On the other hand, conflicting results exist for HD. For example, Bayliss et al. (2019) did not find any correlation between RMET and Montreal Cognitive Assessment (MoCA), while Unti et al. (2018) report correlations between MoCA and MMSE with various social cognition tests in HD, including faux pas, strange stories test, and recognition of emotional faces. Finally, in ET, executive function and social cognition are predominantly associated. Auzou et al. (2014) found that total Ekman task scores were related to semantic and letter fluency, and Santangelo et al. (2012) showed correlations between cognitive theory of mind, measured through the ATT (Advanced Test of ToM), and the FAB (Frontal Assessment Battery) score, the phonological fluency score, and the global score of WCST (Wisconsin Card Sorting Test), all tests that involve executive function. Taken together, these reports suggest that social cognition deficits may be partially attributed to general cognitive dysfunctions. However, due to the variability in the results and the absence of quantitative meta-analyses, further investigations are required to reach firm conclusions in hyperkinetic movement disorders.

Among the included studies in this systematic review, approximately 58% (29 studies) provided clarification on whether the examined patients were under pharmacological treatment, which could have influenced their social cognition performance. These findings emphasize the necessity for further investigation to understand the impact of drugs and other treatments on social cognition performance in individuals with hyperkinetic movement disorders. It should be noted, however, that the reviewed studies generally exhibited adequate methodology quality, with minimal comorbidity observed in the analyzed samples. While a few studies indicated a high risk of bias (see Figure 2 and supplementary materials), most of the reported studies demonstrated suitable methodology and acceptable reliability. Therefore, although a quantitative meta-analysis was not conducted to assess the specific impact of methodological factors on the patterns of social cognition deficits observed across different studies, the qualitative results reported in this review offer an adequate summary of current state-of-the-art, and these results are based on methodological robust studies.

In the existing literature, there has been a predominant focus on studying social cognition in HD, while comparatively less attention has been given to the investigation of TS, dystonia, and ET. Therefore, additional research is crucially needed to explore and elucidate the underlying mechanisms of social cognition across the different hyperkinetic movement disorders. There is need to carry out more systematic research addressing different aspects of social cognition within the same sample of patients. In particular, there is a need for more research focusing on empathy, to better understand its different facets. For example, in addition to relying solely

on self-report measures of cognitive and affective empathy, future investigations should incorporate complementary approaches, including gathering judgments from caregivers or other observers to provide external perspectives on empathic abilities (Eslinger et al. 2011). Furthermore, neurophysiological investigations of empathic brain responses, using paradigms such as empathy for pain (Avenanti et al., 2005, 2005; Lamm et al., 2019, 2019; Singer et al., 2004, 2004) or empathy accuracy (Gallo et al., 2018, 2018; Paracampo et al., 2017, 2017, 2018b, 2018b; Zaki et al., 2009, 2009), can offer valuable insights into the neural mechanisms underlying impaired empathy in movement disorders.

Developing more ecological methodologies for assessing social cognition is also crucial. In this regard, virtual reality technology could serve as a valuable tool, allows patients to immerse themselves in controlled social contexts that could facilitate more realistic experiences requiring social perception, empathy and mentalizing processes (Daher et al., 2021; Grasso et al., 2019, 2020; Lucifora et al., 2020, 2021a, 2021b). However, it is important to note that social cognition encompasses various processes, including attribution, attitudes, social schema, social attention, and social memory. Currently, these aspects remain underexplored in the literature on hyperkinetic movement disorders. On the other hand, it is worth acknowledging that literature on moral decision-making and/or moral reasoning was not included in this review, despite the presence of some evidence in existing studies on hyperkinetic movement disorders (Vicario et al., 2021). Investigating these aspects would provide a more comprehensive understanding of social cognition deficits in this class of clinical conditions.

To advance our understanding of how hyperkinetic movement disorders impact social cognition, future research should embrace an integrative approach, combining multiple neuroimaging, neurophysiological, and brain stimulation techniques (Chiappini et al., 2022; Ritter & Villringer, 2006; Turrini et al., 2023a; Ystad et al., 2011; Zanon et al., 2018) with a systematic investigation of multiple behavioral measures of processes implicated in social cognition. In this vein, network neuroscience, an emerging discipline that focuses on modeling and analyzing brain networks composed of interacting neural elements, holds great promise in this endeavor and can enrich our understanding of the broader neural context in which social behaviors and interactions between brain regions occur (Baek et al., 2021; Barrett & Satpute, 2013; Bassett & Sporns, 2017).

Our current understanding of social cognition suggests that it involves various processing levels, including bodily, emotional, motivational, cognitive, and neural aspects. Therefore, it is essential to go beyond unidimensional approaches and mere localizationist methods and instead identify and integrate each processing level. Our study emphasizes the relevance of basal ganglia and cerebellum, which are known to be linked together to form an integrated functional network (Bostan & Strick, 2018; Bostan et al., 2010; Pierce & Péron, 2020; Sesack & Grace, 2010). These subcortical structures play a crucial role in bridging corporeality and cognition, integrating the processing of motor control, emotion, and inter-subjectivity (Koziol et al., 2012). However, we have limited knowledge about how changes in connectivity between the basal ganglia, cerebellum, and cerebral cortex, as well as their interactions with bodily process, affects social cognition in hyperkinetic movement disorders. To gain a better understanding of the neural mechanisms that support social cognition, it is crucial for future research to prioritize investigating this specific issue.

It is increasingly recognized that assessing social cognition is important to understand individuals' abilities for successful social interactions, as well as, their overall mental health and well-being (Etchepare & Prouteau, 2018). Our review provides extensive evidence of deficits in social perception, empathy, and ToM across distinct hyperkinetic movement disorders. However, traditional clinical assessment focuses on motor symptoms and functional impairments only. By incorporating the assessment of social cognition deficits into the clinical evaluation, it is possible to gain a more comprehensive understanding of the individual's overall cognitive and functional profile. This integrated approach could enable proactive interventions aimed at improving social functioning and overall quality of life.

Disclosure statement

No potential conflict of interest was reported by the author(s).

Supplementary data

Supplemental data for this article can be accessed online at <https://doi.org/10.1080/17470919.2023.2248687>

Additional information

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