Emotions in action: The relationship between motor function and social cognition across multiple clinical populations.

Clare M. Eddy[1] and Jennifer L. Cook[2]

1. National Centre for Mental Health, BSMHFT, The Barberry, Birmingham, UK
2. School of Psychology, University of Birmingham, Birmingham, B15 2TT, UK

Corresponding author: Dr Jennifer Cook; School of Psychology, University of Birmingham, Birmingham, B15 2TT; j.l.cook@bham.ac.uk

GLOSSARY

- Alexithymia: difficulties reflecting on, identifying and describing feelings
- Akinesia: loss of power over voluntary movement
- Apraxia: problems with planning movements that impair production (e.g. on command)
- Ataxia: impaired muscle control and co-ordination of movement
- Autosomal dominant: process of inheritance where the disorder will be present when the gene is passed through one parent
- Bradykinesia: slowness of movement
- Choreiform movements: involuntary writhing or 'dance-like' movements, characteristic of Huntington's Disease
- Corrugator supercilii: a narrow pyramidal muscle above the eye that permits frowning
- Dysphagia: impaired swallowing
- Dysphasia: impaired speech production
- Dystonia: movement disorder whereby sustained or repetitive muscle contractions lead to repetitive writhing movements or abnormal static postures
- Echopraxia: the copying of others actions, or 'mirroring tics' characteristic of Tourette Syndrome
- Hyper/hypo-kinetic: an excess/a lack of movement
- Idiopathic: arising spontaneously with no identified cause
- Interoception: sense of the internal states within one's body
- Micrographia: a reduction in the size of handwriting, characteristic of Parkinson's Disease
- Mirror Neurons: brain cells that fire during both action execution and action observation
- Stereotypy: purpose-less (as opposed to goal-directed) repetitive action

SECTION 1: INTRODUCTION

1.1 Introductory statement

Although a growing body of literature documents bodily movement difficulties in Autism Spectrum Disorder (ASD[1]), this condition has classically been associated with differences in socio-cognitive ability. The converse is true for motor disorders: for conditions such as Parkinson's Disease (PD), Huntington's Disease (HD) and Tourette Syndrome (TS), clinical focus rests on motor symptoms and has somewhat overlooked socio-cognitive difficulties. Previous work has explored the possibility that, in ASD, motor and social ability are not merely co-occurring but are functionally linked such that (for example) atypical movement patterns can result in atypical interpretation of other people's body movements (Cook, 2016). Contrasting functional links between social and motor function in late-onset conditions like PD, with the developmental disorder ASD, may be especially illuminating – enabling us to disentangle the contributions of long-term, developmental, influences and short-term changes. However, at present there is a poverty of literature concerning functional links between motor and social ability with respect to movement disorders. This review paper therefore aims to explore the relationship between social and motor ability in PD, HD, TS and ASD. In particular we focus on non-verbal socio-cognitive processes and the interpretation of intentions and desires where these can be inferred from visual cues representing eye gaze or bodily movements.

In Section 1, we provide background literature about the relationship between action and perception in the general population. We conclude that compared to two people who move in dissimilar ways, two people who move similarly will likely experience more fluid action perception and prediction during interaction and may be better at inferring each other's mental states and emotions. In Section 2, we summarise impairments in motor function and social cognition exhibited by individuals with HD, PD, and TS comparing them to individuals with ASD. In Section 3 we further reinforce the link between motor and socio-cognitive function by highlighting particular studies which have examined both social and motor ability within the same participants and we comment on likely directions of causality. Finally, Section 4 begins to unpack the complex relationship between social and motor function by considering two additional factors which might impact upon both social and motor function: alexithymia and dopamine system dysfunction. We conclude that there is good evidence for both social and motor difficulties that span multiple clinical conditions but that, to fully understand the aetiology of many clinical symptoms, further studies must unpack the way in which social and motor atypicalities feed into and influence each other.

1.2 The relationship between action production and perception

Watching another person perform an action evokes activity (often referred to as 'motor resonance') in one's own motor system. The earliest studies providing evidence for this claim were single cell recording studies, which found that neurons in the motor system of the macaque (subsequently labelled 'mirror neurons') fire when the monkey passively observes an action (di Pellegrino et al., 1992). Subsequently, research using a range of methods including functional magnetic resonance imaging (fMRI), and electroencephalography (EEG) has provided strong evidence for overlapping activity during action execution and action perception in a network of brain regions, commonly referred to as the human mirror neuron system (MNS). The MNS is thought to include the inferior frontal gyrus (e.g. Iacoboni et al., 1999), inferior parietal lobe (Aziz-Zadeh et al., 2006; Grèzes et al., 2003), ventral and dorsal premotor cortex (Buccino et al., 2001; Gazzola et al., 2007), anterior intraparietal sulcus (Dinstein et al., 2007; Shmuelof and Zohary, 2006) and the superior temporal sulcus (Gazzola et al., 2006). Cross-modal repetition suppression, where a reduced response is seen

[1] Although we abide by the terminology of the *Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition* (DSM-V; APA, 2013), we refer readers to Kenny et al. (2016) for the terminology preferred by the UK autism community.

for observation following execution of an action, or vice-versa, also implicates frontal (Kilner et al., 2009) and parietal regions (Chong et al., 2008) in the human MNS.

Behavioural studies demonstrate strong, reciprocal, influences of action production on perception and vice versa. For instance, an actor's perceptual judgments about an object can be influenced by their own concurrent actions and their past sensori-motor experience: one study attesting to this showed that, while lifting a heavy weight, subjects tended to underestimate the weight of objects lifted by others (Hamilton et al., 2004). Similarly, Jacobs and Shiffrar (2005) demonstrated that judgments about an observed walker's speed were altered when the perceivers themselves were walking. In sum, our own actions can influence our perception of the actions of others.

Further demonstration of the reciprocal relationship between action production and perception is apparent from studies that show that the degree of overlap, or similarity, between the movements we produce and those we perceive can influence our recognition of observed actions. Casile and Giese (2006) taught participants a novel upper-body movement while blindfolded, meaning that they received verbal and haptic, but not visual, feedback. Before and after training, point-light stimuli were used to test the visual recognition of the learned movement. Despite the absence of visual stimulation during training, trained participants demonstrated an enhanced ability to visually recognise the trained movement versus other un-trained movements. Furthermore, visual recognition performance after training correlated strongly with the accuracy of the execution of the learned movement. Thus, the more similar a participant's executed movements were to the observed movement, the better their recognition of that movement. In the following section we will consider how the similarity between our own actions and those made by others may also enhance our ability to predict and interpret others' actions and thereby influence social cognition.

1.3 Movement similarity influences action prediction and imitation

The similarity between our own performance of a movement and someone else's performance of that movement influences our ability to predict what that person will do next. For example, professional basketball players can predict the success of free shots at a basket earlier and more accurately than coaches or sports journalists, despite comparable amounts of visual experience (Aglioti et al., 2008). Movement similarity also influences the extent to which individuals imitate each other. Kilner, Hamilton and Blakemore (2007) tracked participants' arm movements during execution of vertical sinusoidal arm "waving" movements while watching a video of an actor making incongruent horizontal waving movements. The video was experimentally manipulated such that the arm moved either with typical human kinematics (in a smooth, fluid manner), or at constant velocity (i.e. like a traditional robot). They found that observing videos of a person moving with human kinematics interfered with participants' on-going actions such that they subtly imitated the observed movement. In contrast, signs of imitation were not apparent for the constant velocity movements. Thus, imitation was enhanced (or greater interference was seen) for movements that were similar to the participants' own movements relative to movements that were dissimilar. Existing research therefore demonstrates prediction and imitation of movements is facilitated for movements that are similar to one's own established motor patterns.

1.4 Movement similarity influences internal state attribution and emotion recognition

Visual motor cues, such as the speed of body movements, or the contraction of certain facial muscles, can provide important information about a person's internal mental states and emotions. Correspondingly, movement similarity between two individuals can influence mental state and emotion attribution. For instance, if participants are required to watch point light displays depicting an actor walking in a happy, sad, or angry fashion, their emotion judgements are biased by their own typical walking speed (Edey et al., 2017). That is, a person who typically walks slowly is likely to judge other slow walkers as being in a neutral frame of mind, whereas the same walker might be judged to appear sad by an individual who typically walks fast. Likewise, a fast walker would likely be judged by other fast walkers as appearing in a neutral frame-of-mind but by other slow walkers as appearing angry.

Internal emotions and mental states can be signalled by a variety of cues, not restricted to full bodily movements. Studies have shown that even relatively simple motion cues can be interpreted as conveying mental states, as illustrated by the work of Heider and Simmel (1944), and exploited in the Animations Task (Abell et al., 2000). During these tasks, participants view video-clips showing the movements of simple geometric shapes (two triangles). The animations are designed to elicit mental state attribution (e.g. one triangle *persuading* the other). A recent study building on the Animations Task has shown that mental state attribution also varies as a function of movement similarity. Edey and colleagues (2016) asked participants to produce their own videos depicting two triangles coaxing, mocking, surprising, or seducing each other. The videos were subsequently shown to other participants, who were asked to judge the extent to which each video depicted coaxing, mocking, seducing or surprising. Edey and colleagues found that participants were better at estimating mental states for videos that moved with kinematics that were similar to their own, compared to videos that proceeded with dissimilar kinematics.

1.5 Summary: Motor resonance and perception of social cues

In this section, we have presented evidence that, compared to two people who move in dissimilar ways, two people who move similarly will likely experience more fluid action perception and prediction during interaction, be more likely to imitate each other, and may be better at inferring each other's mental states and emotions. It follows from this that individuals with clinical conditions involving movement disorder may experience difficulties with socio-cognitive functions including action prediction, imitation, mental state attribution and emotion recognition. This possibility is explored in sections 2-4. We begin, in section 2, by briefly reviewing the literature concerning movement and social cognition in clinical conditions chiefly associated with abnormalities in the motor or socio-cognitive domain.

SECTION 2: MOVEMENT AND SOCIAL COGNITION IN CLINICAL POPULATIONS

2.1 Opening statement

In Section 1 we have discussed how the way a person moves can have a significant impact on various aspects of socio-cognitive function, from action perception to emotion interpretation. If it is indeed the case that motor and socio-cognitive function are linked, it should follow that conditions that are typically thought of as 'motor disorders' should be associated with socio-cognitive atypicalities, and conditions typically linked with socio-cognitive difficulties may be associated with motor abnormalities. In Section 2 we investigate the first claim by examining the literature regarding the movement disorders PD, HD, and TS, and we investigate the second claim by reviewing the ASD literature (though see Cook, 2016 for a more in-depth investigation). Motor impairments are listed against each clinical condition in Table 1; while findings on tests of social cognition are given in Table 2. We focus on tasks involving non-verbal cues associated with movement that convey emotions or intentions.

2.2 Parkinson's disease

2.2.1 Motor and behavioural aspects of Parkinson's disease

Patients with PD exhibit hypokinetic movement disorder as a result of neurochemical dysfunction within frontostriatal circuitry, arising through a loss of dopaminergic neurons within the substantia nigra pars compacta. The cardinal signs of PD are resting tremor, bradykinesia, rigidity and postural instability. Other common motor features include swallowing difficulties, loss of facial expression, micrographia, shuffling gait, dystonia, slowed blink rate, and freezing (Jankovic, 2008). With disease progression, cognitive impairments arise, affecting executive functions such as working memory (Montoya et al., 2006; Dirnberger and Jahanshahi, 2013), and often leading to subcortical dementia (Bonelli and Cummings, 2008). Depression is also common (Marsh, 2013). Treatment is based on dopamine replacement therapy (e.g. L-Dopa or dopamine agonists), although deep brain stimulation of globus pallidus or subthalamic nucleus may be used in more severe cases (e.g. Bronstein et al., 2011).

Bradykinesia affects the planning, initiation and execution of movement. It can therefore influence a range of motor functions. PD is typically associated with impairments in internally generated actions not elicited by environmental cues, which has been suggested to reflect involvement of the supplementary motor area rather than the premotor cortex (Marsden, 1989). Indeed, external environmental triggers may help some patients to overcome immobility. Perhaps of most interest are reported instances of sudden and brief improvement in mobility in situations involving extreme emotion or physical stress (e.g. Bonnani et al., 2010). This could suggest an important relationship between movement and emotion in PD, or between motor control and motivational state (Kojavic et al., 2014). Such relationships have been argued to be mediated by the neuromodulator dopamine (Mazzoni et al., 2007) discussed further in Section 4.

2.2.2 Overview of social cognition in Parkinson's disease

Many studies have investigated recognition of emotional facial expressions in both medicated PD patients (receiving dopamine replacement therapy) and unmedicated patients. A range of results have been reported including impairments in recognising fear and disgust (Kan et al., 2002); specific problems with disgust (Suzuki et al. 2006); impairments in all emotions but particularly negative emotions (Gray and Tickle-Degnen, 2010; Sprengelmeyer et al., 2003; Yip et al., 2003); and no impairment (Dujardin et al., 2004). Recognition of facial expressions can be impaired both on and off medication (Bediou et al., 2012). A meta-analysis of facial emotional expression recognition deficits in PD concluded that patients are worst at recognising negative emotions (Gray and Tickle-Degnen, 2010). Although this literature is mixed, there are a number of factors that may explain conflicting findings. For instance, work by Ille and colleagues (2016) suggests that (own) emotion regulation abilities and disease stage may be important predictors of emotion recognition abilities: more specifically, Ille and colleagues (2016) found that PD patients with poorer control over feelings of disgust exhibited less accurate recognition of angry faces and that emotion recognition capacity typically deteriorates with disease progression.

The Reading the Mind in the Eyes Test (RMET: Baron-Cohen et al., 2001) assesses recognition of internal states (e.g. preoccupied, tentative, fantasising) from photographs of only the eye region of human faces. Some studies have reported little evidence for impairment on the RMET in early PD (Peron et al., 2009; Roca et al., 2010) while others have indicated poorer performance on this task in medicated PD patients in comparison to neurotypical individuals (Bodden et al., 2010; Mimura et al., 2006; Tsuruya et al., 2011). For example, Tsuruya et al. (2011) report impairments in RMET performance in medicated early stage PD patients, and furthermore show that impairments are not attributable to the perceptual problems in the visual processing of faces (as patients showed intact performance on a gender judgment task) or a failure to discriminate emotional adjectives.

The possibility that at least some patients with PD exhibit deficits in emotion attribution is supported by performance on a range of tasks. For example, studies (Bodden et al., 2010; Narme et al., 2013) have reported impairments on another task requiring following eye gaze direction. In this task (Shamay-Tsoory et al., 2007), a cartoon face (named Yoni) is surrounded by four other faces or items from a semantic category (e.g. fruit), Yoni's eyes are directed towards one of the items/faces. Participants are asked to indicate which picture matches a given statement (e.g. "Yoni likes/does not like" or "Yoni is thinking of"). For the control condition, participants are asked to select the item that is closest to the face. Patients with PD pass the control task but struggle with the gaze following condition (e.g. Bodden et al., 2010). Thus, evidence from a number of different studies suggests deficits in PD when reasoning about, or recognising, emotions and mental states. Although it can be hard to completely rule out the impact of executive deficits (Eddy et al., 2013), PD patients' socio-cognitive problems typically extend beyond domain general cognitive difficulties which affect performance on control tasks.

2.3 Huntington's disease

2.3.1 Motor and behavioural aspects of Huntington's disease

HD is an inherited autosomal dominant movement disorder involving neurodegeneration of medium spiny neurons within the striatum. It is caused by an unstable trinucleotide cytosine-adenine-guanine repeat in the coding region of the HTT gene, which leads to the production of mutant huntingtin protein (MacDonald et al., 1993). The number of trinucleotide repeats is inversely correlated with the age of onset of disease, but most gene carriers typically develop motor symptoms around middle age (Snell et al., 1993; Stine et al., 1993). Characteristic motor symptoms include involuntary choreiform movements, dyskinesia, dystonia, rigidity, bradykinesia, dysphagia, dysphasia and difficulties with balance, gait and motor co-ordination (e.g. Roos, 2010). In addition to the limbs, upper and lower body, motor chorea can involve nose and mouth, abnormal eye opening and corrugator supercilii contractions (Fekete and Jankovic, 2014).

With disease progression, neuropathological changes affect a range of brain areas from frontal lobes to thalamus (e.g. Vonsattel, 2008). Cognitive problems become more frequent and may begin with more specific impairments in executive functions such as working memory (You et al., 2014), which are then surpassed by more widespread difficulties affecting concentration, comprehension and communication. In one study (Hamilton et al., 2003), apraxia was associated with greater motor dysfunction involving voluntary movements, and aspects of cognition (i.e. verbal fluency). In turn, motor symptoms may be correlated with performance on many cognitive tasks in HD including those thought to assess executive functions (Eddy and Rickards, 2015a). This could reflect an incidental contribution to performance or an underlying mediating factor linked to disease stage and underlying neural dysfunction.

Psychiatric symptoms are also common in HD (Craufurd et al., 2001). Depression has been reported as more prevalent earlier in the disease and declining later (Paulsen et al., 2005), though some cases could be reactive rather than more closely tied to the disease process. Apathy appears more central to the psychiatric phenotype and is considered to track more closely with disease progression (Tabrizi et al., 2009). Some patients can exhibit disinhibited behaviours or aggression, and poor interpersonal skills can become apparent (e.g. Duff et al., 2010). Impairments in social cognition may make a particularly important contribution to these latter behavioural problems (Eddy, Parkinson and Rickards, 2016).

2.3.2 Overview of social cognition in Huntington's disease

Early studies investigating the social abilities of people with HD found that these patients had substantial difficulty in recognising facial emotion (e.g. Jacobs et al., 1995; Sprengelmeyer et al., 1996). It was suggested that the recognition of disgust (Sprengelmeyer et al., 1996; Hayes et al., 2007; Wang et al., 2003) or anger (Snowden et al., 2008; Calder et al., 2010) may be disproportionately affected. However, subsequent studies have argued for more generalised impairment (Johnson et al., 2007; Henley et al., 2008). In addition, the understanding of emotional vocal tone (Speedie et al., 1990) can be impaired, including both positive as well as negative vocalised emotions (Robotham et al., 2011). Deficits in recognition of emotional facial expressions can extend to premanifest gene carriers (Gray et al., 1997; Labuschagne et al., 2013).

One recent study using the Yoni cartoon task (Shamay-Tsoory and Aharon-Peretz, 2007) reported that patients with manifest HD demonstrate impairments in following eye gaze direction, but do not make significantly more errors than neurotypical individuals in the control condition requiring physical state judgments. Patients with HD also exhibit difficulties in recognising mental states on the RMET (Eddy et al., 2012; Eddy et al., 2014), inferring intentions from pictures and video-clips (Allain et al., 2011; Brune et al., 2011; Eddy and Rickards, 2015b) and interpreting humorous cartoons (Snowden et al., 2003). A few studies have reported links between executive deficits and poor socio-cognitive task performance in HD (e.g. Allain et al., 2011; Eddy et al., 2012; Eddy et al., 2014). However, at least some patients with the HD gene show differences compared to control adults in terms of their attribution of emotions and intentions during socio-cognitive tasks, despite intact executive function (Eddy and Rickards, 2015b; Eddy and Rickards, 2015c). In addition, recent studies suggest that while patients with HD struggle to recognise mental states during the RMET, they are as

accurate as neurotypical individuals when judging physical states (i.e. judging age) based on the same stimuli (Eddy, Rickards and Hansen, 2018).

2.4 Tourette syndrome

2.4.1 Motor and behavioural aspects of Tourette syndrome

Tourette syndrome (TS) is a neurodevelopmental disorder involving tics: repetitive movements and vocalisations. The most frequent time of onset is around 7-10 years of age, with a peak in severity around the age of puberty, and a general improvement in tics in early adulthood (e.g. Leckman et al., 1998; 2006). However, tic frequency characteristically waxes and wanes over time, and there is a wide spectrum in the severity and complexity of tics across the TS population. Common simple tics include eye blinking, facial grimace, shoulder shrugging and limb extension. Sequences and combinations sometimes occur. More complex tics can include touching objects, repeating movements a certain number of times, mirroring tics (echophenomena), and tics linked to socially inappropriate behaviour (e.g. coprolalia, non-obscene socially inappropriate behaviours: Kurlan et al., 1996; Eddy and Cavanna 2013a,b). A range of co-morbid behavioural problems are present in many patients (e.g. Cavanna et al., 2009) including obsessive-compulsive symptoms, attention deficit and hyperactivity, mood disorder and impulse control disorders. Current treatments for tics include clonidine (an alpha-adrenergic agonist), dopamine antagonists such as aripirazole and risperidone, and behavioural therapy (Eddy, Rickards and Cavanna, 2011).

Tics tend to be prompted by a premonitory urge which can be described as a feeling of itch or pressure, which is relieved on releasing the tic. This urge is hard to resist but can usually be suppressed for at least short periods of time, leading tics to be described as semi-involuntary, or 'unvoluntary' movements (The Tourette Syndrome Classification Study Group, 1993). Tics are thought to reflect dysfunction of the basal ganglia (e.g. Mink, 2006; Peterson et al., 1993; Singer et al., 2003), and/or poor inhibitory control (e.g. Nowak et al., 2005) over inappropriately generated movements. It has also been suggested that motor compulsions in TS could be linked to sensori-motor integration (Eddy, 2016), and a further possibility is that motor function is differently distributed in the brain in TS (Biswal et al., 1998; Georgiou et al., 1997). However, while some tics may occur consistently across all contexts (e.g. eye blinking), a simple basal ganglia model is less able to explain environmentally dependent complex tics (Eddy and Cavanna, 2014), such as touching objects, echophenomena or socially inappropriate behaviours. Furthermore, as tics can worsen in association with stress, anxiety, or other strong emotions (Singer, 2013), it is likely there is some interaction with the limbic system.

2.4.2 Overview of social cognition in Tourette syndrome

Both the possibility of limbic involvement in TS, and the instance of socially inappropriate behaviours in many of these patients (Kurlan et al., 1996; Eddy and Cavanna, 2013a) has prompted consideration of social cognition in this population. Early studies included patients with TS as a control group (Baron-Cohen et al., 1997) or reported no differences compared to neurotypical individuals (Channon et al., 2004). However, further experiments showed subtle differences in the way that people with TS respond to tasks involving interpretation of social cues including complex emotional facial expressions (Eddy et al., 2010a&b; Eddy et al., 2011). Performance differences between patients with TS and neurotypical individuals do not usually indicate a failure to attribute mental states, and more often comprise less conventional interpretations of social communications.

Overall, adults with TS have been found to respond conventionally on some tasks, but not on others. In general, impairments seem more likely on tasks involving visual cues (Eddy and Cavanna, 2013b) e.g. when making judgments about a cartoon character's socially competitive emotion (e.g. feelings of envy or gloating towards other characters) based on cartoons featuring emotional expressions and eye gaze cues (Eddy et al., 2011). Imitation processes also seem to be unusual in TS, such that these patients can show greater interference in their own actions while observing the actions of others (Jonas et al., 2010). The involvement of visual motor cues linked to emotion in the task may therefore be an important factor, and it has been found that adults with TS are more affected than neurotypical individuals by other people's non-verbal expression of negative emotions in everyday life (Eddy et al., 2015). More recently, people with TS were found to show an increased tendency to

view the random movements of geometric shapes in the Animations Task (Abell et al., 2000) as reflecting goals, emotions and intentions (Eddy and Cavanna, 2015). This indicates that people with TS may be particularly sensitive to visual motor cues that can evoke social cognitive attributions and may exhibit a bias towards 'hyper-mentalizing'(Eddy, 2018).

2.5 Autism Spectrum Disorder

2.5.1 Motor and behavioural aspects of Autism Spectrum Disorder

ASD is a neurodevelopmental condition associated with social and communication difficulties, repetitive behaviours and restricted routines (APA, 2013). Although the social and communicative features of the condition have received the most interest in terms of academic research, there is a growing interest in movement atypicalities associated with this condition (Table 1). Early clinical observations noted hypokinetic signs including posturing, freezing, bradykinesia (Wing and Attwood 1987), dystonia, and "striatal toes" (a Babinski-like spontaneous reflex; Damasio and Maurer, 1978). In a cohort of 154 children with ASD from ages 2 to 7, the prevalence of motor abnormalities was substantial, with 51% exhibiting dystonia and 34% motor apraxia (Ming et al., 2007). Furthermore, a number of studies have reported signs of catatonia in ASD, with current prevalence rates being estimated between 12 and 17% (Ghaziuddin et al., 2012). Signs of hypokinesis have led some researchers to draw similarites between ASD and PD (Damasio and Maurer, 1978). More recent studies investigating movement in ASD have noted postural instability (Chen and Tsai, 2015; Doumas et al., 2015; Fournier et al., 2010, 2014; Graham et al., 2014; Greffou et al., 2011; Kohen-Raz et al., 1992; Minshew et al., 2004; Molloy et al., 2003; Morris et al., 2015; Stins et al., 2015), atypical gait characterised by an increased step width and reduced velocity (Kindregan et al., 2015), increased time to initiate and execute manual aiming movements (Glazebrook et al., 2006, 2007), longer movement times for reach-to-grasp actions (Yang et al., 2014), increased jerkiness of arm movements (Cook et al., 2013; Edey et al., 2016), and reduced legibility of handwriting and letter formation (Kushki et al., 2011).

Though many studies have documented movement atypicalities in ASD, much remains to be known about the origin of such atypicalities. Various accounts have been proposed. Brain based accounts have noted differences in the structure and/or function of brain areas involved in movement. For example, structural abnormalities in the cerebellum predict functional deficits on a simple motor learning task (Allen et al., 2004; Courchesne et al., 1988); fractional anisotropy of the brainstem's corticospinal tract predicts both grip strength and ASD symptom severity (Travers et al., 2015) and an unbiased meta-analysis of voxel-based morphometric studies highlights the basal ganglia as a brain region with consistent structural atypicalities in ASD (Nickl-Jockschat et al., 2012). At present whether such brain atypicalities are the *cause* or consequence of behavioural movement differences is unknown. Others have argued that movement atypicalities may in fact stem from socio-cognitive deficits since many motor skills are learned through interaction with, and observation of, others (e.g. primary caregivers).

2.5.2 Overview of social cognition in Autism Spectrum Disorder

On the Frith-Happé Animations Task, high-functioning children with ASD provide less typical mental state descriptions when describing social interactions depicted by the triangles (Abell et al., 2000). Performance differences on the Animations Task persist into adulthood in ASD (White et al., 2011). Individuals with ASD also exhibit atypicalities with respect to categorising biological motion. For example, Cook et al. (2009) reported differences in categorising arm movements as natural or unnatural on the basis of kinematics. In line with this, others have reported difficulties with classifying point light stimuli as representing 'human' or 'non-human' movement (Kaiser et al., 2010). These difficulties are unlikely to reflect purely perceptual problems, given that fewer differences are reported when making stimulus-driven judgements e.g. judging the facing direction (left versus right) of point light stimuli (Saygin et al., 2010).

Numerous studies have reported differences in ASD with interpreting representations of emotion. This includes interpreting emotion from facial expressions (Braverman et al., 1989; Brewer et al., 2015; Capps et al., 1992; Davies et al., 1994; Hobson et al., 1988; Macdonald et al., 1989; Tantam et al., 1989; though see Gepner et al., 2001; Ozonoff et al., 1990), from the eyes alone

(Baron-Cohen et al., 2001), reading emotions from bodily postures (Hadjikhani et al., 2009) or body movement (Atkinson, 2009; Hubert et al., 2007; Nackaerts et al., 2012), and judging emotion from vocal stimuli (Golan et al., 2006).

2.6 Summary

In conditions associated with atypical motor control (PD, HD, TS) we see atypical social cognition. Likewise, in conditions such as ASD, typically associated with socio-cognitive differences, we see atypical motor function. Co-occurring impairments may suggest that the motor system plays an important role in at least some aspects of social cognition (or vice versa). However, observing deficits within the social and motor domains, in separate experiments, within a population is not the same as observing within-individual overlap. In other words, it could be that some individuals, within a clinical group, exhibit motor difficulties, some individuals exhibit socio-cognitive atypicalities, but few individuals exhibit both. In the following section we report studies which have investigated both social cognition and movement atypicalities within the same individuals and we comment on the likely direction of causality.

SECTION 3: WITHIN SAMPLE EVIDENCE OF THE RELATIONSHIP BETWEEN SOCIAL COGNITION AND MOVEMENT DISORDER

3.1. Correlations between social cognition and motor disorder in clinical populations

3.1.1 Parkinson's disease

Multiple studies have demonstrated within-sample correlations between motor dysfunction and socio-cognitive ability in PD. For example, Xi et al. (2015) found that for medicated early to moderate stage PD patients, RMET scores were negatively correlated with disease severity (which is largely based on motor symptom severity). Similarly, Raffo de Ferrari et al. (2015) found that performance on the RMET was worse in PD patients who showed more evidence of later stage hypokinetic symptoms, such as freezing of gait. This relationship remained when psychiatric symptoms and executive functions (e.g. visuoconstruction ability) were controlled for. Buxton et al. (2013) found that the more impaired a PD patient was based on the motor subscale of the Unified Parkinson's Disease Rating Scale, the worse they were at recognising emotions from facial expressions and vocal tone. Simons et al. (2004), investigated spontaneous, posed and imitated emotional expressions in PD, revealing reduced facial emotion expressivity across various situations compared to neurotypical individuals, and evidence that the origin of the expression deficit was more motoric than emotional given patients' additional difficulties in posing non-emotional expressions. Such difficulties in the production of facial expressions may play a functional role in emotion recognition. For example, after removing the effect of disease severity, Marneweck et al. (2014) found that voluntary ability to control facial muscles in PD correlated with recognition of emotional expressions specifically, but not recognition of faces more generally, and Ricciardi et al. (2015) reported a correlation between PD patients' ability to express facial emotion and recognise facial emotion. At present, whether motor deficits precede or succeed socio-cognitive difficulties in PD is unclear. One challenge for investigations within the PD population is the idiopathic nature of the disorder i.e. PD is typically diagnosed on the basis of motor symptoms. Thus, it is possible that any pre-dating of socio-cognitive symptoms by motor symptoms could be an illusion created by diagnostic method. The application of highly sensitive tests of social cognition and motor function in patients at risk of developing PD will offer insight into the chronological emergence of social and motor abnormalities.

3.1.2 Huntington's disease

A handful of studies have demonstrated correlations between social and motor function in HD. De Gelder and colleagues (2008) reported impaired recognition of non-verbal whole-body anger expressions in HD, in addition to poor understanding of instrumental acts such as putting clothes on. Poorer recognition of anger was correlated with certain (though not all) motor symptoms (e.g. gait, rigidity and postural abnormalities). Eddy, Sira Mahalingappa and Rickards (2014) report correlations between motor symptom severity and RMET performance, and a recent study highlighted a relationship between patients' motor symptom severity and mental state attribution in response to the

Animations Task (Eddy and Rickards, 2015b). Deficits in both the expression and recognition of emotion in HD have also been reported (Trinkler et al., 2013).

While it was once assumed that socio-cognitive difficulties emerge *after* the onset of motor symptoms in HD, there is growing evidence that this is not always the case. Studies assessing HD gene carriers who have yet to express overt motor signs, have reported evidence of impairment in the recognition of (Gray et al., 1997; Labuschagne et al., 2013), and neural response to (Novak et al., 2012), emotional facial expressions. Furthermore, recent studies have highlighted that HD gene-carriers without motor symptoms can demonstrate behavioural differences when compared to neurotypical individuals, on tasks such as the RMET and the Animations Task (Eddy and Rickards 2015a & b). Importantly, the same patients were reported to exhibit few differences compared to neurotypical individuals on tests of executive function, demonstrating a relatively selective impairment in social cognition. Thus, there is mounting evidence that social cognition is affected early in the course of HD, and that some socio-cognitive deficits may predate motor dysfunction. However, existing studies in this field have not been able to ensure that motor and socio-cognitive assessments are equally sensitive to individual differences. Thus, further studies should test whether socio-cognitive difficulties emerge before motor difficulties even when assays of these abilities are matched in terms of sensitivity.

3.1.3 Tourette syndrome

The study of social cognition in TS is in early stages and therefore few studies have focused on the relationship between motor and socio-cognitive function in patients with TS. In a review of research conducted so far, Eddy and Cavanna (2013b) found that there was no significant correlation between ratings of the frequency and severity of motor tics and behavioural performance on social cognitive tasks. However, a link between social cognition and motor symptoms was highlighted by two recent fMRI studies (Eddy et al., 2016; Eddy Cavanna and Hansen, 2017), which reported that activity within brain regions involved in social cognition (e.g. temporo-parietal junction) during tasks including the RMET, was correlated with symptoms such as mirroring tics. Further research is clearly needed and should specifically focus on testing the hypothesis that more extreme motor abnormalities in TS should be significantly positively predictive of socio-cognitive atypicalities.

3.1.4 Autism Spectrum Disorder

Correlations between social cognition and movement atypicalities in ASD are attracting increasing attention. Using motion-tracking technology Cook, Blakemore and Press (2013) showed that individuals with ASD produced arm waving movements that were more jerky, and which proceeded with greater acceleration and velocity, than those produced by neurotypical individuals. In a separate perception task, these same participants were required to label the movement of an animated human hand as ‘natural’ or ‘unnatural’. Results showed that the degree to which kinematics were atypical when *executing* arm movements was significantly correlated with biased responding when *observing* motion of a human hand. In other words, the more atypical an autistic participant’s kinematics (relative to kinematics exhibited by neurotypical individuals), the less likely they were to classify movements that follow typical kinematics as “natural”. This finding is consistent with Edey and colleagues’ (2017) findings that neurotypical observers’ emotion perception from gait information is modelled on their own walking kinematics.

Cattaneo and colleagues (2007) investigated the link between action execution and automatic imitation of others’ actions (including grasping and eating) in children with ASD and a matched group of typically developing children. During both action execution and observation conditions, the activity of the mouth-opening mylohyoid (MH) muscle was recorded using electromyography. During the execution condition MH muscle activity from neurotypical children started to increase several hundreds of milliseconds before their hand grasped the food, kept increasing during actual grasping, and peaked when the child started to open the mouth. For autistic children, no activity increase was found during reach or grasp, but only as the food was brought to the mouth. These group differences during action execution translated into group differences during action observation: for neurotypical children MH activity was observed when they passively observed another child reach and grasp a piece of food. In contrast, the autistic children did not show MH activation during the observation of either reaching or grasping phases.

At present, there is no consensus as to whether socio-cognitive atypicalities are the cause or consequence of movement atypicalities in ASD, though a growing literature informs us about whether movement atypicalities precede or succeed socio-cognitive atypicalities. A number of studies have reported that socio-cognitive atypicalities are not necessarily present from birth but tend to gradually emerge over the first few years of life in children that go on to develop ASD (Johnson et al., 2015). Orienting to faces and eyes, for example, is commonly reported to be typical during the 1st year of life (Elsabbagh et al., 2013a, 2013b, 2014; Ozonoff et al., 2010; Young et al., 2009) but subsequently decreases (Jones and Klin, 2013; Ozonoff et al., 2010).

In contrast with the protracted emergence of socio-cognitive atypicalities, movement differences have been observed in 1-month old infants that go on to develop ASD. Karmel and colleagues (2010) noted abnormal upper extremity tone in 1-month old infants that later developed ASD. Other studies have noted delays in early motor milestones. For example, displaying abnormal head lag when pulled to sit at 6 months is associated with later ASD diagnosis (Flanagan et al., 2012). During free play sessions conducted at 6, 9, 12, and 14 months, four infants later diagnosed with ASD showed substantial delays in the emergence of new postures, spent more time in less developmentally advanced postures (e.g. lying, sitting) and shifted posture less often (Nickel et al., 2013). Delays in performance on measures of fine and gross motor function can also be seen from 6 months (Nickel et al., 2013). These studies show that movement atypicalities can be observed within the first year of development, and have been observed as early as 1 month, in children that later develop ASD. Thus, it is plausible that motor abnormalities precede socio-cognitive atypicalities in ASD, and therefore that atypical movement abilities may contribute to the atypical development of socio-cognitive functions. However, caution should be exercised in drawing this conclusion since, at present, studies have not investigated the development of movement and socio-cognitive abilities within the same individuals, thus it may be the case that, for example, the individuals in the above-mentioned movement studies were more severely autistic compared to those in the social cognition studies. Furthermore, it should be noted that two processes may be developmentally associated due to a third factor of importance to both. Therefore, developmental evidence should not be taken as definitive evidence of causation (see Happé et al., 2017, for further discussion about establishing cause and effect in socio-cognitive studies), and intervention studies should also be conducted in order to establish the direct of causality.

Section 3.2 Summary

In Section 3 we have shown that in PD, HD, TS and ASD, socio-cognitive and movement deficits are frequently reported within-subject as well as within sample. Furthermore, in each population, problems within these two domains are typically positively correlated. However, though it may be the case that movement difficulties are the *cause* of socio-cognitive atypicalities in these populations, it is feasible that the opposite direction of causality may apply: socio-cognitive atypicalities could lead to movement abnormalities. Here we have reviewed existing literature and suggested the likely direction of causality. However, additional research addressing this issue is much needed and must address a number of obstacles: tests of social cognition and motor function must be matched in terms of sensitivity such that tests are equated in their capacity to reveal impairments in both domains, and such tests should be used in 'at risk' individuals to avoid any biases which may result from diagnosis.

SECTION 4: UNPACKING THE RELATIONSHIP BETWEEN SOCIAL COGNITION AND MOTOR FUNCTION

4.1 Social and motor function: simulation and template-matching

In section 1 we presented evidence that, compared to two people who move in dissimilar ways, two people who move similarly will likely experience more fluid action perception and prediction during interaction, and may be better at inferring each other's mental states and emotions. It follows from this that individuals with clinical conditions involving movement atypicalities may experience difficulties with socio-cognitive functions. We demonstrated evidence consistent with this hypothesis in sections 2 and 3. In section 4 we ask, why is it the case that socio-cognitive and motor difficulties are so commonly co-occurring?

The relationship between motor and social function has received a great deal of attention within embodied cognition and mirror neuron frameworks (Aglioti et al., 2008; Cook et al., 2014; Cross, Hamilton and Grafton, 2006; Gallese and Sinigaglia, 2011; Goldman and Vignemont, 2009; Patel, Fleming & Kilner, 2012). These accounts postulate that socio-cognitive and motor difficulties co-occur because impairments in motor function directly impact on socio-cognitive function via two possible routes. The first is a 'simulation' route: observation of others' movements activates the motor codes, in the observer, that would be activated if they were to execute the same movement. This covert simulation of the observed movement allows inference of the cognitive/emotional state that most likely caused the movement (Kilner, Friston and Frith, 2007; Wolpert, Doya and Kawato, 2003). The second is a template matching route: through a lifetime of experience we develop 'templates' comprising visual representations of bodily movements associated with particular internal states (Cook et al., 2013; Bird and Viding, 2014). These associations arise either through observing and labelling one's own actions, and/or through communicating with others about the internal states that underlie their movements. Subsequently, each time a bodily movement is observed one can use 'template matching' to infer the most likely internal state. Notably, although template and simulation processes are not necessarily dependent on each other, both routes may operate concurrently.

In neurodevelopmental disorders such as TS and ASD, both simulation and template matching routes could provide possible means by which motoric atypicalities result in socio-cognitive differences. For example, if an individual's bodily movements are, on average, different from those exhibited by neurotypical individuals that individual's simulations of neurotypical movements may result in atypical inferences (or no inferences) about the internal states of neurotypical people. It may also be the case that the individual has different templates of movements. For example, their visual representation of a happy movement (highly influenced by seeing their own happy movements (e.g in the mirror)) may be different from the way in which most neurotypical individuals move when they are happy, thus neurotypical happy movements will not fit the template.

The way in which social and motor function are linked in late-onset conditions such as PD and HD is harder to determine. The template matching explanation is less likely for such conditions, especially in the early stages of the disorder. This is because templates are based on experience accumulated over the lifetime, thus an individual who develops a motor disorder late in life may be able to rely on templates developed prior to the onset of the disorder. However, as the disease progresses, individuals gain increasing experience of observing and labelling their own, atypical, movements. Thus, over time movement templates may become increasingly atypical. The simulation route is perhaps more plausible with respect to HD and PD, especially in the early stages of these conditions. Previous studies have suggested that both executed, and imagined movements are atypical in individuals with PD (Maidan et al., 2016; Peterson et al., 2014). If covertly simulating a movement is akin to imagining that movement, this research suggests that the process of simulation may be atypical and may therefore result in atypical inferences in PD.

Simulation and template matching provide two routes by which motor and social function might be directly linked. However, there are various intermediary factors, which may independently contribute to social and motor function, or play a mediating role in the relationship between social and motor ability. Future comprehensive investigations of the relationship between social and motor function, must also consider these intermediary factors. Here, in Section 4.1, we begin to unpack the relationship between social and motor function by looking at two potential intermediary factors: alexithymia and dopamine system function.

4.2 Social cognition and movement – intermediary factors.

4.2.1 Alexithymia and awareness of internal states

Alexithymia is a sub-clinical condition traditionally characterised by difficulties identifying and describing one's own emotional states. A large body of work demonstrates that this difficulty with labelling own emotions has a knock-on effect on recognising others' emotions (Grynberg et al., 2012), and empathising with others' emotional states. Thus, co-occurring alexithymia may explain many socio-cognitive difficulties in the clinical conditions that we have focused on here. Indeed, the

suggestion that alexithymia may explain socio-cognitive impairment is increasingly being recognised in the ASD literature (Bird and Cook, 2013). Recent studies have demonstrated that individuals that have ASD without co-occurring alexithymia do not exhibit difficulties with emotion recognition or empathy, whereas individuals that have both conditions have emotion recognition and empathy difficulties, as do individuals with alexithymia alone (Bird et al., 2010, 2011, 2016; Bird and Cook, 2013). Thus, emotion-related socio-cognitive difficulties are due to the presence of alexithymia, not ASD. The appreciation of the importance of alexithymia is also growing in the motor disorders research field. In comparison to neurotypical individuals, patients with PD are more likely to experience alexithymia (Assogna et al., 2012; Costa et al., 2006; Costa et al., 2010), and increased alexithymia in PD has been related to reduced empathy and perspective taking in everyday life (Narme et al., 2013). Significant levels of alexithymia have also been reported in both manifest and premanifest patients with HD (Eddy and Rickards, 2015b). Alexithymia does not, however, appear to be more prevalent in adults with TS than control adults (Eddy et al., 2015). On the contrary, compared to neurotypical individuals, adults with TS exhibit significantly higher ratings on measures of interoception (Eddy et al., 2014) i.e. the perception of the physical condition of the body including visceral sensations (Craig, 2002), whereas alexithymia has been linked to interoceptive deficits (Brewer et al., 2015; Brewer et al., 2016; Gaigg et al., 2016; Murphy et al., 2017; Shah et al., 2016).

In addition to socio-cognitive deficits, alexithymia has also been associated with motor production (Brewer et al., 2015; Trevisan et al., 2016) and control (Sowden et al., 2016) abnormalities. For example, Trevisan and colleagues (2016) demonstrated that atypical production of emotional facial expressions is better explained by alexithymia than by ASD. In addition, Sowden and colleagues (2016) have demonstrated abnormalities in the control of action imitation in individuals with high levels of alexithymia. High rates of alexithymia in PD, HD, TS and ASD may therefore explain some of the co-occurring socio-emotional and movement difficulties, though further studies are needed to examine motor production using tasks with no requirements for emotion or socio-cognitive processing. Importantly, though rates of alexithymia are high in these clinical conditions, there are some individuals that have "pure" disorders, without alexithymia. It is crucial that further studies investigate whether movement and social cognition difficulties co-occur in these different patient subgroups.

4.2.2 Dopamine

It is important to note that all of the disorders we have focused on have been associated with some degree of dysfunction in one of the brain's primary neuromodulatory systems: the dopamine system. In parallel work, dopamine has been linked with both social and motor function. Could it be that dopaminergic dysfunction, at least in part, underpins difficulties across both socio-cognitive and motor domains in PD, HD, TS and ASD?

4.2.2.1 Dopamine and motor function

Dopamine plays a central role in motor function, and dopamine system dysfunction has been linked to movement abnormalities across various clinical conditions. In neurotypical individuals, the pharmacological manipulation of dopamine impacts on the execution of motor acts including foot waggling, finger tapping and handwriting (Badgaiyan et al., 2003; Egerton et al., 2009; Goerendt et al., 2003; Lappin et al., 2009; Larisch et al., 1999; Ouchi et al., 2002; Schommartz et al., 2000), as well as on the process of motor learning (Badgaiyan et al., 2007, 2008; Garraux et al., 2007). In PD the primary cause of movement abnormalities is degeneration of dopamine-producing neurons in the substantia nigra pars compacta (e.g. Obeso et al., 2008). Ultimately a cascade of neurochemical disruption leads to over stimulation of basal ganglia output nuclei that inhibit activation of motor nuclei, leading to problems with movement. Dopaminergic drugs (agonists or precursors such as Levodopa) aim to replace the lost neuromodulator and restore normal motor function (e.g. Hagan et al., 1997). A popular view links dopamine to movement via its role in reporting average reward rates (Collins and Frank, 2014; Niv et al., 2006). That is, if (tonic) dopamine is high, the average rate of reward is high, thus every second in which a reward is not delivered is costly, and the benefit of moving speedily outweighs the energetic costs of doing so. In the context of PD it has been suggested that dopaminergic dysfunction increases sensitivity to the energetic costs of movement, resulting in a bias against vigorous movements (Mazzoni et al., 2007).

Dopaminergic dysfunction has also been linked to other motor disorders including HD and TS. Early-stage patients with HD exhibit increased dopamine levels (Garrett and Soares-Da-Silva, 1992) and late-stage patients exhibit reduced levels (Bernheimer et al., 1973; Kish et al., 1987) which are linked to hyperkinesis (e.g. chorea) and akinesia (i.e. loss of movement; Bird, 1980; Spokes, 1980) respectively. The link between hyperkinesis and increased dopamine levels is further supported by the TS literature wherein hyperkinesis is accompanied by elevated levels of dopamine, as evidenced by studies of post-mortem brains (Singer et al., 1991; Minzer et al., 2004; Yoon et al., 2007), nuclear imaging studies (e.g. George et al., 1994; Malison et al., 1995; Wolf et al., 1996; Wong et al., 1997, 2008; Ernst et al., 1999; Müller-Vahl et al., 2000a; Singer et al., 2002; Cheon et al., 2004; Gilbert et al., 2006; Yeh et al., 2007; Albin et al., 2009; Steeves et al., 2010; Liu et al., 2010) and cerebro-spinal fluid analyses (Singer et al., 1982). Indeed, dopamine antagonists are often the most effective drugs for reducing motor tics in TS (e.g. Eddy, Rickards and Cavanna, 2011).

Despite a lack of studies there is some evidence for dopaminergic dysfunction in ASD. Early studies noted similarities between movements generated by individuals with ASD and PD (Damasio and Maurer, 1978; Maurer and Damasio, 1982; Vilensky et al., 1981). Studies have shown reduced levels of dopamine in isolated platelets (Launay et al., 1987) and urine (Martineau et al., 1992) from individuals with ASD and symptom modulation by dopaminergic drugs in autistic individuals and in animal models of ASD (Chadman et al., 2012; Ghanizadeh and Moghimi-Sarani, 2013). Furthermore, genes encoding the dopamine transporter (Hamilton et al., 2013), several dopamine receptors (Hettinger et al., 2012; Qian et al., 2013; Reiersen and Todorov, 2011) and enzymes involved in dopamine synthesis, have previously been linked to ASD. Thus, convergent evidence points towards dopamine system differences in ASD. Further studies are needed to establish whether dopamine system atypicalities play a functional role in the movement patterns seen in this clinical condition.

4.2.2.2 Dopamine and social function

Although less is known about the relationship between dopamine and social cognition there is some evidence, from both non-human animal and human studies, supporting a link between the two. Studies of macaque monkeys have shown that the amount and availability of dopamine D2 receptors can vary according to social environment and social dominance (Grant et al., 1998; Morgan et al., 2002; Nader et al., 2012). In rats, dopamine plays an important role in various aspects of social behaviour including social interaction (Homberg et al., 2016), scent marking (Homberg et al., 2016) and social play (Vanderschuren et al., 2016). In humans, pharmacological manipulations of dopamine modulate social behaviours including social learning (Eisenegger et al., 2013), social conformity (Campbell-Meiklejohn et al., 2012; Egerton et al., 2010; Pedroni et al., 2014; Stokes et al., 2014), social moral decision making (Crockett et al., 2015; Sáez et al., 2015) and social reward processing (Kampe et al., 2001). Genetic analyses have also supported a role for dopamine in social cognition, though in many cases replication studies with larger sample sizes are required. For instance, variation in genes affecting dopaminergic function has been associated with individual differences in social functions including mentalising (Lackner et al., 2012), altruism (Reuter et al., 2011), social facilitation (Walter et al., 2011) and the ability to store and manipulate information about other people (Dumontheil et al., 2014). Genes influencing dopamine metabolizers, such as the enzyme Catechol O-Methyltransferase, have been linked to performance on emotion expression recognition tasks (e.g. Weiss et al., 2007), and may influence neural responses in brain regions such as the amygdala and ventromedial prefrontal cortex during processing of emotional facial expressions (Lelli-Chiesa et al., 2011). Finally, evidence from human Positron Emission Tomography (PET) suggests associations between social desirability, social conformity and social status, and dopamine D1/D2 receptor binding in the striatum (Egerton et al., 2010; Martinez et al., 2010; Plavén-Sigray et al., 2014; Reeves et al., 2007; Stokes et al., 2014), a key neural structure implicated in the movement disorders reviewed here. Thus, evidence from non-human animal studies, and human studies utilising PET, genetics and psychopharmacology, suggests an important relationship between the dopamine system function and social cognition.

4.2.2.3 Linking dopamine, motor and social function

Cognitive and motor difficulties in PD, and to some extent TS (Tremblay et al., 2015), have previously been united under a dopamine dysfunction thesis (Frank, 2005). While of use in elucidating the aetiology of cognitive and motor symptoms and in guiding treatment interventions,

this thesis is silent about **socio-**cognitive function and has not been extended to other conditions such as ASD. Indeed, dopamine system dysfunction may contribute to both the social and motor symptoms of many conditions. For example, dopaminergic medication in PD has been noted to improve both the recognition of emotional facial expressions as well as basic motor function (Sprengelmeyer et al., 2003). At present, however, it is unclear whether dopaminergic dysfunction would independently contribute to social *and* motor difficulties, or whether neurochemical disruption in one domain (e.g. motor) mediates effects on other domains (e.g. socio-cognitive). For example, it is conceivable that dopamine system dysfunction impairs social cognition *via* effects on motor function. That is, dopamine loss may impair motor simulation (in the same way it would impair motor imagery: Maidan et al., 2016; Peterson et al., 2014), or it may have long term effects on movement production resulting in atypical motor templates, this may then impair performance on particular socio-cognitive tasks that rely on these motor templates. Such a mechanism might operate in parallel with dopaminergic impairments in decision making and executive function. In other words, there could be multiple mechanisms by which dopamine dysfunction impairs both social and motor function.

SECTION 5: CONCLUSIONS

Building on the observation that visual and motor representations of one's own movements play an important role in the perception, prediction and interpretation of others' movements we have previously argued that socio-cognitive atypicalities in individuals with ASD may, at least in part, be a function of motoric abnormalities (Cook et al., 2013; Cook, 2016; Edey et al., 2016, 2017). The current review extends this logic to three additional clinical conditions in which both socio-cognitive and motor difficulties have been observed: PD, HD and TS.

In Section 1 we briefly reviewed literature concerning the relationship between socio-cognitive and motor function, ultimately concluding that, compared to two people who move in dissimilar ways, two people who move similarly will likely experience more fluid action perception and prediction during interaction, be more likely to imitate each other, and may be better at inferring each other's mental states and emotions. It follows from this that individuals with clinical conditions involving movement disorder may experience socio-cognitive difficulties when interacting with neurotypical individuals (and, conversely neurotypical individuals might experience socio-cognitive difficulties when interacting with individuals with clinical conditions: see Edey et al., 2016). Correspondingly, in Section 2 we presented evidence of abnormalities in both motor and socio-cognitive functions including action prediction, imitation, mental state attribution and emotion recognition in PD, HD, TS and ASD. In Section 3 we further reinforced the association between motor and socio-cognitive functioning by highlighting studies which have examined both social and motor ability within the same participants. We concluded that, though more studies in this vein are required, the extant literature suggests a correlation between social and motor function, such that greater motor impairment is associated with greater socio-cognitive dysfunction. Finally, in Section 4 we began to unpack the complex relationship between social and motor function. First, in Section 4.1, we outlined two possible routes by which motor atypicalities might result in socio-cognitive differences: the simulation and template matching routes. We suggest that whilst both routes are plausible for neurodevelopmental conditions, the simulation route may be most likely in the case of neurodegenerative disorders. Second, in Section 4.2, we considered two additional factors which might impact upon both social and motor function: alexithymia and dopamine system dysfunction. The difficulties in identifying and describing one's own emotional states that characterise alexithymia are common in PD, HD and ASD. Alexithymia has been linked to particular difficulties in emotion-related socio-cognitive tasks (e.g. recognition of emotional facial expressions) and motor tasks such as producing emotional facial expressions. More broadly, alexithymia is thought to be indicative of a general 'interoceptive' deficit in identifying internal bodily signals, thus we suggested that further research should investigate the role of interoceptive awareness in socio-cognitive and motor function across diverse conditions including PD, HD, TS and ASD. We also acknowledge the central role of dopamine in motor function and that PD, HD, TS and ASD are all associated with dopamine system abnormalities. Given empirical evidence that modulating dopamine system function can impact on both social and motor performance, we acknowledged that dysfunction of the dopamine system may play a role in the co-occurrence of social and motor abnormalities across various clinical conditions. We noted that whilst there is evidence linking dopamine with motor dysfunction for PD, HD, and TS,

there is less evidence linking dopamine with social dysfunction across these disorders. Finally, we observed that the relationship between dopamine, social and motor function is highly complex. More specifically, it is not clear whether a dopaminergic abnormality would independently contribute to motor and social difficulties, or impact on one function (e.g. motor) with a concomitant effect on the other function (e.g. social). Thus, additional work is required to elucidate the mechanism by which a dopamine system abnormality might give rise to both social and motor difficulties.

In sum, we have shown that comparing multiple clinical disorders characterised by atypicalities in both motor and social domains suggests an intricate, potentially reciprocal, relationship between motor and social functions. Studying PD, HD, TS and ASD in parallel could shed light on similarities and differences within underlying neural and neurochemical mechanisms that may help us to better understand the aetiology of these conditions. Future research must now seek to further specify exactly how and why this co-occurrence between motor and socio-cognitive impairment exists.

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	Parkinson's disease	Huntington's disease	Tourette syndrome	Autism Spectrum Disorder
Balance, posture, gait	Characteristic posture, shuffling gait (Jankovic, 2008). Observing actions of others can improve freezing of gait in PD (Pelosin et al., 2010).	A range of motor signs of HD, such as dysarthria, ataxia, postural instability, and broad-based gait have been linked to the cerebellum (Rüb et al., 2013).	Tics can affect gait and posture (Fasano et al., 2012). Possibly subtle gait abnormalities (e.g. irregular step length: Liu et al., 2014).	Postural instability (Chen and Tsai, 2015; Doumas et al., 2015; Fournier et al., 2010; Graham et al., 2014; Morris et al., 2015; Stins et al., 2015), plus atypical gait characterised by increased step width and reduced velocity (Kindregan et al., 2015)
Hyperkinesis	Dyskinesia, tremor, stereotypical movements e.g. punding (medication related) and perseveration (Stoffers et al., 2001).	Dyskinesia, chorea (Mann et al., 2012).	Tics (including context dependent and sequences), echopraxia	Stereotypy (Bodfish et al., 2000). Flapping (Damasio and Maurer, 1978).
Hypokinesis	Cogwheel rigidity, bradykinesia, freezing, dystonia, dysarthria, hypomimia (Shahed & Jankovic, 2007).	Rigidity, bradykinesia, dystonia, dysarthria (e.g. Hart et al., 2013; Louis et al., 2000). Poor imitation of facial expressions and postures (e.g. De Gelder et al., 2008; Trinkler et al., 2013).	Catatonic signs (Cavanna et al., 2008).	Catatonia (Ghaziuddin et al., 2012); posturing and freezing (Wing and Attwood, 1987); dystonia, bradykinesia, rigidity and "striatal toes (Damasio and Maurer, 1978); reduced range of facial expressions (Brewer et al., 2015)
Grip, co-ordination	Abnormally elevated grip force (Fellows et al., 1998). More movement and slower than neurotypical individuals during both precision and power grasp (Pradhan et al., 2015). Increased grip force, bradykinesia, and abnormalities of between-hand load force coordination (Gorniak et al., 2013).	Poor regulation of reach-grasp transition and greater variability in force application and temporal coordination (Rao et al., 2011). Excessive grip force (Schwarz et al., 2001) and poor coordination (Serrien et al., 2001), with absence of the long-latency stretch reflex and a delay in response to loading (Fellows et al., 1997).	Nowak et al. (2005) found that patients had a proportionally larger grip force than neurotypical individuals. The neural correlates of a grip-load force task were also found to be altered in TS (Serrien et al., 2002), though it may be hard to disentangle activations linked to having tics.	Increased time to initiate and execute manual aiming movements (Glazebrook et al., 2006, 2009, 2007); longer movement times for reach-to-grasp actions (Yang et al., 2014)

Kinematics (speed, smoothness)	Bradykinesia, poor modulation of motor plans in social context off medication (e.g. Straulino et al., 2015; Straulino et al., 2016). Dopamine medication therapy influences movement kinematics (Foreman et al., 2014).	Patients can exhibit poor modulation of action based on visual cues (Georgiou et al., 1997). Bradykinesia can interfere with motor tasks, and some movement abnormalities on kinematic tasks in HD are suggestive of impairments in sequencing and motor strategies (Quinn et al., 2001).	Abnormal lateralisation effects during concurrent cognitive and motor tasks (e.g. Georgiou et al., 1997; Yazgan et al., 1995). Differences in bimanual movements have also be reported in relation to symmetry and movement accuracy (Avanzino et al., 2011).	Jerky kinematics for reach-to-grasp actions (Yang et al., 2014) and sinusoidal arm movements (Cook et al., 2013).
Fine motor control	Micrographia, fine motor control impaired by bradykinesia (e.g. van Gilst et al., 2015). Ideomotor apraxia (i.e. deficits in planning and executing motor activity) as determined by tasks such as demonstration of tool use (Hödl et al., 2008).	Poor handwriting, speech deterioration. Fine motor control on the Purdue Pegboard is impaired in HD although this may be influenced by cognitive impairment (Andrich et al., 2007). Patients may fail to adapt kinematics to target size (Georgiou et al., 1997)	Children with TS can exhibit difficulties on visuomotor tasks (Brookshire et al., 1994) including the Purdue Pegboard (e.g. Bloch et al., 2006; Margolis et al., 2006), and grooved pegboard (e.g. Bornstein et al., 1990) although few studies have controlled for comorbid ADHD (Eddy et al., 2009).	Atypical handwriting (Grace et al., 2017; Kushki et al., 2011). Atypical Purdue pegboard task performance (Barbeau et al., 2015).

Table 1. Motor symptoms: Motor atypicalities in Parkinson's Disease (PD), Huntington's disease (HD), Tourette Syndrome (TS) and Autism Spectrum Disorder (ASD).

	Parkinson's disease	Huntington's disease	Tourette syndrome	Autism Spectrum Disorder
Recognising emotional facial expressions (e.g. basic emotions, RMET)	Impairment in basic emotion recognition, particularly bad in patients with more severe motor symptoms (e.g. freezing of gait).	Impaired, perhaps even early in disease stage. RMET errors correlate with motor symptom severity. Patients remain confident.	Mild difference in performance on RMET, patients show anxiety at task; neural differences could enhance greater sensitivity to negative emotional expressions	Impairment in emotion recognition. However, this is argued to be due to co-occurring alexithymia not ASD per se (Brewer et al., 2015; Trevisan et al., 2016)
Reading mental states from non-biological motion cues (e.g. animations task)	Can still process e.g. helps with gait freezing. PD less sensitive in recognising biological movement than neurotypical individuals as measured by temporal dilation effects Cao et al. (2015). Worse at detecting biological motion linked to walking (Jaywant et al., 2016).	Impaired interpretation, reduced attribution of intentions to goal directed movements and social interactions.	Increased attribution of intentions/emotions in response to random movements	Less appropriate mental state descriptions (Abell et al., 2000; Castelli et al., 2002; White et al., 2009)
Interpreting biological motion (e.g. body language, imitation)	Poor at understanding communicative gestures from point light displays (e.g. Jaywant et al., 2016).	Poor recognition of angry and instrumental body postures (de Gelder et al., 2008). Poor recognition of anger in film clips (Rees et al., 2014).	More interference from observed biological movements not compatible with current motor task (Jonas et al., 2010). Mirroring tics (echopraxia) support greater salience of observed non-verbal action.	Atypical categorization of / orienting towards biological motion (e.g. Annaz et al., 2012; Cook et al., 2009; Hubert et al., 2007; Kaiser et al., 2010)
Tasks involving eye gaze cues	Deficits on Yoni cartoon task for desire and attention, not for control condition involving physical judgments (Bodden et al., 2010)	Deficits on Yoni cartoon task for desire and attention, no significant difference for control condition (Adjeroud et al., 2016)	Adults with TS were worse than neurotypical individuals on a version of the Yoni task which asked them to judge who a cartoon face was jealous of/gloating at based on facial expression and gaze direction (Eddy et al., 2011).	Some atypicalities noted on tasks involving processing of eye gaze cues such as the RMET and gaze following tasks (Baron-Cohen *et al*., 1999*a*, 2001; Leekam *et al*., 1998, 2000)

Table 2. Socio-cognitive ability: Socio-cognitive atypicalities in Parkinson's Disease (PD), Huntington's disease (HD), Tourette Syndrome (TS) and Autism Spectrum Disorder (ASD). RMET = reading the mind in the eyes.