

# **Sense of agency disturbances in movement disorders: a comprehensive review**

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## **Abstract**

Sense of agency refers to the experience that one's self-generated action causes an event in the external environment. Here, we review the behavioural and brain evidence of aberrant experiences of agency in movement disorders, clinical conditions characterized by either a paucity or an excess of movements unrelated to the patient's intention. We show that specific abnormal agency experiences characterize several movement disorders. Those manifestations are typically associated with structural and functional brain abnormalities. However, the evidence is sometimes conflicting, especially when considering results obtained through different agency measures. The present review aims to create order in the existing literature on sense of agency investigations in movement disorders and to provide a coherent overview framed within current neurocognitive models of motor awareness.

**Keywords:** Sense of agency; Movement disorders; Parkinson's disease, Gilles de la Tourette syndrome, Corticobasal syndrome, Functional movement disorders.

## Introduction

The sense of agency refers to the feeling that one voluntarily initiates and controls actions, and generates, through them, effects in the external world (P. Haggard, 2017). The sense of agency is altered in several psychiatric and neurological conditions (Moore & Fletcher, 2012). Schizophrenia represents a classic agency disorder. It has been the subject of most of the agency research in pathological conditions. Passivity phenomena in schizophrenia clearly implicate the misattribution of thoughts or actions to an external agent (Synofzik, Thier, Leube, Schlotterbeck, & Lindner, 2010). In the condition of delusion of control, an individual firmly believes that her/his own action has been initiated and controlled by another agent (C. D. Frith, S. Blakemore, & D. M. Wolpert, 2000a). Similarly, the thought insertion symptoms entail thoughts to be perceived as externally generated, as they have been inserted in a patient's mind without permission (Frith et al., 2000a). However, aberrant experiences of agency are not restricted to schizophrenic patients but can be observed in other clinical conditions, like obsessive-compulsive behaviour (Gentsch, Schütz-Bosbach, Endrass, & Kathmann, 2012), borderline personality disorder (Colle, Hilviu, Rossi, Garbarini, & Fossataro, 2020), and movement disorders (Moore, Schneider, et al., 2010; Saito et al., 2017). This latter condition is the specific topic of the present review.

Movement disorders are clinical syndromes characterized by either a paucity or an excess of movements unrelated to the patient's intention. They include, among others, Gilles de la Tourette Syndrome, Parkinson's disease, Huntington's disease, corticobasal syndrome, and other psychogenic conditions, like functional movement disorders.

There are several reasons for expecting an altered sense of agency in movement disorders. First, the neural correlates of the sense of agency partially overlap with the neurofunctional network of voluntary movements, with particular reference to the supplementary and pre-supplementary motor areas (SMA, pre-SMA, Kühn, Brass, & Haggard, 2013; Moore, Ruge, Wenke, Rothwell, & Haggard, 2010; Zapparoli, Seghezzi, Zirone, et al., 2020). It follows that the sense of agency might be generated by the same sensorimotor system that generates and controls motor execution and is differently compromised in movement disorders. Therefore, it is reasonable to expect that any malfunctioning of the sensorimotor system, particularly involving the SMA/pre-SMA network, may affect the subjective experience of agency to various degrees. Movement disorders might be associated with an impaired agency experience also because of the motoric symptoms. For example, it has been hypothesized that voluntary movements may be hard to distinguish from tics in Gilles de laTourette's syndrome patients due to the high level of noise in the sensorimotor system induced by the hyperkinetic production (Ganos et al., 2015). Furthermore, agency disturbances in movement disorders might be either a consequence of the disease or a side-effect of the disease's pharmacological treatment, as suggested, for instance, for dopaminergic treatment in Parkinson's disease (Moore, Schneider, et al., 2010). These considerations make movement syndromes particularly interesting for agency studies and have motivated the investigation of the sense of agency in movement disorders over the years. However, to date, a comprehensive understanding of the disturbances of the sense of agency in relation to movement disorders is still missing.

The present review provides a comprehensive overview of the behavioural, structural, and functional brain abnormalities linked to the sense of agency experience in movement disorders patients, framed within current neurocognitive models of motor awareness. We start this review by describing the different theoretical frameworks that address the arising of the sense of agency from a neurocognitive perspective. Then, we introduce the experimental paradigms typically used to measure the agency experience and its neural correlates. Finally, we analyse the existing literature on the sense of agency in movement disorders. We describe the behavioural and brain evidence that has revealed specific abnormalities in motor awareness in populations affected by movement disorders. We address whether there is a consistency in agency disturbances across different movement disorders or whether specific alterations characterize each condition. We consider if such agency disturbances are coherent across the different measures of the agency experience (i.e., implicit and explicit measures). When possible, we also include the neural correlates of the described sense of agency

disturbances. We complemented the revision by discussing how the current neurocognitive models of motor awareness can address these aberrant manifestations.

### **What is the sense of agency, and where does it come from?**

In social sciences, the term “agency” refers to individuals' ability to act on their own will. Besides the objective facts of agency, linked to motor preparation and action execution, agency is also a subjective experience, or “sense” (Patrick Haggard & Eitam, 2015). The term “sense of agency” refers to this subjective experience, and typically entails “the experience of controlling one’s own actions and, through them, the course of events in the outside world” (P. Haggard, 2017).

The origins of the sense of agency in the human mind are still under investigation. Several theories have been proposed to address the arising of the sense of agency. These theories imply different levels of description, from the top-down inferential processes to the fine-grained neural mechanisms. These are summarized in Table 2.

The apparent mental causation theory (Wegner, 2003) offers a theoretical explanation of the sense of agency generation as a top-down inferential process. In particular, this theory describes the experience of agency as a retrospective insertion to consciousness or a post-hoc reconstruction of events and their likely causes (Wegner & Wheatley, 1999). Accordingly, the sense of agency would arise from an inferential sense-making process computed after the end of the movement, in accordance with three main principles: priority, consistency, exclusivity. If (1) a thought becomes conscious just before an action (priority), (2) the thought is consistent with the action (consistency) and (3) it is not accompanied by apparent alternative causes of the action (exclusivity), the agent ascribes the generated sensory consequences to one’s own action (Wegner, 2003). This theory is intriguing, but it accounts for a theoretical explanation level that refrains from any explanations of the *mechanisms* involved in the sense of agency generation.

A mechanistic account of the sense of agency belongs to the so-called Comparator Model (Blakemore, Wolpert, & Frith, 2002; Frith et al., 2000a), which is based on concepts from *optimal motor control theory* (Franklin & Wolpert, 2011; Wolpert & Ghahramani, 2000). Central to this theory is the idea that the motor control system relies on the so-called “internal models”, which represent the motor-to-sensory transformations and how these are implemented in the physical world. Action control mainly depends on the joint activity of these internal models and a series of comparators, namely mechanisms that compare signals and use the result of the comparison to regulate the system. Within this framework, the sense of agency arises from the comparison between predictive signals generated by the internal models during motor planning (the so-called “efference copy”) and the actual sensory effects of one’s action (hence the name “Comparator Model”, Blakemore et al., 2002; C. D. Frith, S. J. Blakemore, & D. M. Wolpert, 2000b). An action is perceived as self-caused when there is a match between the predicted and experienced sensory effects (P. Haggard, 2017). This theory offers a mechanistic explanation of the sense of agency that may seem incompatible with the theoretical proposal of the apparent mental causation theory. Indeed, while the optimal motor control theory and comparator model highlighted the role of “internal” motor signals in the generation of the agency experience, the apparent mental causation theory seems to favour inferential processes based on “external”, contextual signals. This dichotomy has been solved by a recent proposal that stresses the role of both sources of information in the emergence of the sense of agency (Moore, Wegner, & Haggard, 2009; Synofzik, Vosgerau, & Newen, 2008a; Wolpe, Haggard, Siebner, & Rowe, 2013). Moore et al. (2009) suggested that the sense of agency arises from a weighted integration of internal and external cues, together with prior beliefs. This theory – called “cue integration theory” – also suggested that the relative influence of the different information sources may be linked to their reliability, with the more reliable source of information dominating the agentic experience (Moore et al., 2009).

However, these theories still lack a biologically plausible explanation of the processes of agency in terms of their underlying brain mechanisms. This further level of explanation appeals instead to theories that have tried to link the putative cognitive processes of agency to biologically plausible mechanisms.

Among them, active inference rests upon the idea that the brain uses “internal generative models” to explain the source of the incoming sensory information with the ultimate purpose of minimizing the free energy of the model, its “entropy”, or more simply the “surprise” associated with sensations (Friston, Mattout, & Kilner, 2011). Crucially, active inference proposes that the cerebral cortex, and the larger-scale organization of different brain networks, are hierarchically organized. The upper levels represent abstract, high-level, domain-general, multimodal beliefs. Intermediate levels contain modality-specific beliefs related to sensations. Lower levels address specific predictions for specific sensations, like immediate proprioceptive predictions. There is a set of neurons encoding predictions at each level, and another set encoding prediction errors, which corresponds, in the context of motor control, to the amount to unexpected proprioceptive and exteroceptive sensations contingent on the action.

The fit between model predictions – what it can explain – and sensations is assured in two ways. On the one hand, the brain could update its predictions about the sensory data through perceptual inference. Alternatively, the fit could be optimised by changing the sensory data (Parr & Friston, 2019). This latter purpose can be realised by acting on the world, such that the sensations are more consistent with the model expectations. The mechanism by which the prediction error is resolved (changing predictions or changing sensory evidence through actions) is determined by the relative *precision-weighting* of predictions and prediction errors, with the more precise source of information dominating the agentic experience. Predictions that initiate a movement prevail when the precision of the current somatosensory state is down-weighted relative to predictions (Brown, Adams, Parees, Edwards, & Friston, 2013). From a neurobiological point of view, the precision is proposed to be encoded by synaptic gain, i.e., the inverse of the variance in the fluctuation of neuronal activity. In turn, these modulations in synaptic gain are thought to underlie the physiological sensory attenuation seen prior to and during the movement in the somatosensory cortex (Brown et al., 2013). This physiological sensory attenuation seen before and during movements should not be confused with the so-called *psychophysical* (or *perceptual*) sensory attenuation evoked by the consequences of the generated movements and recorded after the execution of the movement (please see the methods session).

Within the generative model, the sense of agency is implicit in the cycle of active sampling of sensations. Precisely, the sense of agency is thought to emerge from the successful balancing of the precision of prediction errors within the cortical hierarchy for action, and the ability of this balanced hierarchy to converge on the most likely cause of a self-generated sensation, i.e., the agent himself. Thus, active inference approaches the study of the sense of agency by reducing it to an active inference problem (Friston et al., 2013). Precisely, the inference that has to be made by the agent corresponds to the estimation of the probability that one was the agent of the action (“who is the agent that is minimizing the free energy?”) given the sensory attributes of that action (proprioception, sensorimotor feedback, external cues) and the prior probability that the action was executed by the agent (i.e., the probability that the agent was optimal in minimizing the free energy associated with the desired outcomes of its actions). In this way, the active inference framework is able to contain in itself both the theoretical cues from the apparent mental causation theory and the mechanistic explanation of the comparator model, giving at the same time new hints on the neurobiology of the agency generation.

In this paper, we will take advantage of those (neuro)cognitive models to review the several expressions of aberrant experiences of agency in movement disorders by discussing, at the same time, the manifestations that are largely expected and those that escape the current explanations.

**Table 1 | Major theories and models of the sense of agency.**

Theories	Sense of agency characterization	Sense of agency generation
Apparent mental causation theory (Wegner, 2003; Wegner & Wheatley, 1999)	Top-down inferential process	The sense of agency arises from an inferential sense-making process computed after the end of the movement, in accordance with three main principles: priority, consistency, exclusivity. If (1) a thought becomes conscious just before an action (priority), (2) the thought is consistent with the action (consistency) and (3) it is not accompanied by apparent alternative causes of the action (exclusivity), the agent ascribes the generated sensory consequences to one's own action.
Comparator model (Blakemore et al., 2002; Frith et al., 2000b)	Intrinsic part of action programming and execution	The sense of agency results from the comparison between predictive signals generated from the internal models during motor planning (the so-called "efference copy") and the actual sensory effects of one's action. An action is perceived as self-caused when there is a match between the predicted and experienced sensory effects.
Cue integration theory (Moore et al., 2009)	Weighted integration of internal and external cues	The sense of agency arises from a weighted integration of internal and external cues, together with prior beliefs. The relative influence of the different information sources is linked to their reliability, with the more reliable source of information dominating the agentic experience.
Active Inference theory (Friston et al., 2011)	Implicit in the cycle of active sampling of sensations	The sense of agency emerges from the successful balancing of the precision of prediction errors within the cortical hierarchy for action, and the ability of this balanced hierarchy to converge on the most likely cause of a self-generated sensation, i.e., the agent himself.

### Methods and measures for the study of the sense of agency

In the last twenty years, several experimental paradigms have been designed to capture the subjective experience of agency in experimental settings. These are summarized in Table 2.

Explicit measures of agency rest upon humans' ability to reflect on their agentic role when directly asked about it. The most widely used task in the research setting is the so-called "action-recognition task." In a typical example, participants perform specific actions without having the possibility of seeing their movements. Rather, they see feedback through a screen that can refer to either the participant's movement or a temporal/spatial experimental manipulation of it. Participants are then asked to judge whether the video they are watching is showing their own movements or those of another person (i.e., the experimenter), or the degree of control they experience over the seen scene (Farrer, Bouchereau, Jeannerod, & Franck, 2008; Farrer, Frey, et al., 2008).

Implicit measures of agency rely on unconscious behavioural biases that provide indirect clues of our agentic experience. A major one is the intentional binding effect (P. Haggard, Clark, & Kalogeras, 2002). Intentional binding refers to a phenomenon whereby voluntary actions, but not passively induced movements, are perceived as temporally shifted towards their effects (i.e., action-binding effect), and their effects (e.g., a tone) as shifted back towards the actions that caused them (i.e., tone-binding effect). As a result, a time compression between voluntary actions and effects is reported (i.e., overall binding-effect, P. Haggard et al., 2002).

Another implicit marker of the sense of agency is the sensory attenuation phenomenon (or sensory suppression). This phenomenon describes the human tendency to perceive the sensory consequences of voluntary actions as less intense than the same stimuli generated by passive movements (Blakemore, Wolpert, & Frith, 2000; Blakemore, Wolpert, & Frith, 1998). This phenomenon can be observed at both behavioural (e.g., different behavioral responses in the "force-matching type-tasks", see, for example, Shergill, Samson, Bays, Frith, & Wolpert, 2005) and neural level (e.g., reduced ERPs for action-triggered versus externally triggered stimuli, see, for example, Hughes & Waszak, 2011 for attenuation of visual responses; and Martikainen, Kaneko, & Hari, 2005 for attenuation of auditory responses).

Although explicit and implicit measures of agency are useful in capturing the sense of agency experience in experimental settings, both measures have theoretical and methodological limitations.

Explicit paradigms are undoubtedly intuitive, but they can be vulnerable to cognitive biases, typical of self-report measures. For example, people can overestimate their agency, claiming authorship of actions or consequences that are not their own (see, for example, Wegner & Wheatley, 1999). Moreover, explicit judgments of agency are rare in everyday life. While they might have a significant role in social settings where individuals may be held responsible or liable for the consequences of their behaviour, the everyday experience of agency mostly relies on the implicit background feelings of being in control (Kühn et al., 2013).

Implicit measures have their methodological weaknesses as well. For example, intentional binding is usually observed on a group level, but there is a large variability between subjects. The source of this variability is largely unknown (Wolpe & Rowe, 2014). More importantly, it has been shown that an intentional binding effect can sometimes be also observed even in complete absence of intentionality (Buehner, 2012; Suzuki, Lush, Seth, & Roseboom, 2019).

Furthermore, the relationship between implicit and explicit measures of agency is still a matter of debate. While it is widely accepted that there are separable, and to some extent independent, agency processing systems (Synofzik et al., 2008a), the two measures sometimes co-occur and sometimes dissociate (Moore, Middleton, Haggard, & Fletcher, 2012).

**Table 2** | Methods and measures of the sense of agency.

Methods	Measure	Description	Main findings
<b>Explicit methods</b>			
Action-recognition tasks	Explicit judgments	Participants perform actions without seeing their movements. Rather, they see feedback through a screen that can refer to either the participant's movement or a temporal/spatial experimental manipulation of it. Participants are asked to judge whether the video they are watching is showing their own movements or those of another person (i.e., the experimenter), or the degree of control they experience over the seen scene.	Participants tend to attribute movements to other agents when there is a high temporal/spatial discordance between their hand movements and sensory feedback, even when movements are their own.
<b>Implicit methods</b>			
Time estimation type-task	Intentional binding tasks	Participants are asked to report the perceived timing of voluntary action, the timing of a subsequent sensory effect, or the elapsed time interval between them.	Voluntary actions, but not passively induced movements, are perceived as shifted towards their effects, and their effects are shifted back towards the voluntary actions that caused them. As a result, when the action is voluntarily executed, a time compression between actions and effects is reported.
Force-matching tasks	Sensory attenuation	Typically, a target force is applied to the participant's hand. Participants are then required to match the force they experienced, either directly by pushing down on the same hand using their finger until they perceived the same force, or indirectly by using a joystick to control the force production on the hand.	Participants typically apply a greater force when directly using their finger to match the externally applied target force. At the same time, they reproduce the original force much more accurately when they match the target force using the joystick.

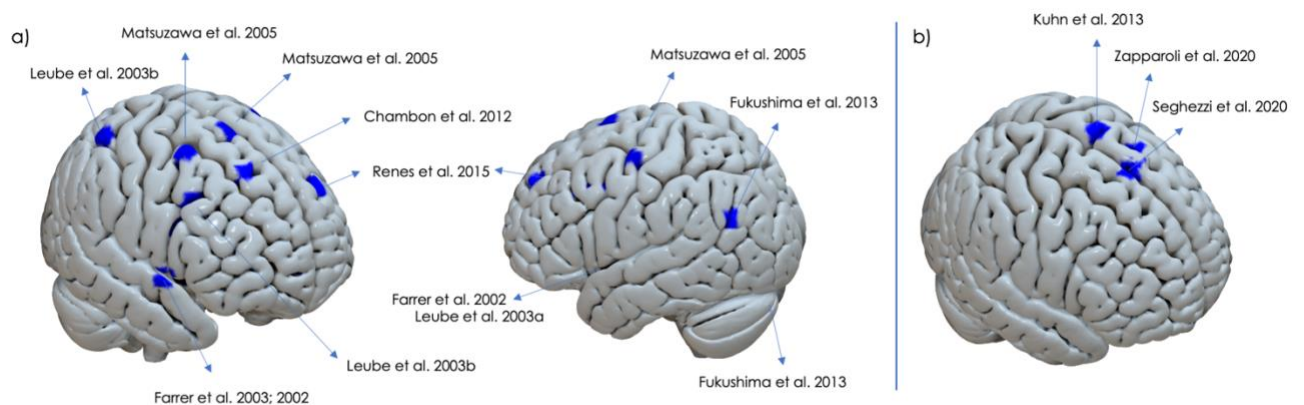
### The neurobiology of the sense of agency

In the past two decades, both explicit and implicit measures have been extensively used to explore the brain mechanisms underlying the sense of agency through neuroimaging, non-invasive brain stimulation, and electrophysiological studies.

Most of the studies measuring agency with explicit attribution judgments have consistently highlighted the involvement of the parietal cortex, especially the inferior parietal cortex and the angular gyrus, in the sense of agency experience (Farrer et al., 2003; Farrer, Frey, et al., 2008; Farrer & Frith, 2002; Preston & Newport, 2008; for a meta-analysis of 15 neuroimaging studies see also Sperduti, Delaveau, Fossati, & Nadel, 2011). However, the parietal activations observed in neuroimaging studies seem associated with the perturbed experience of agency, i.e., when the (visual) feedback is judged as externally-generated in the so-called “no-agency condition”. The neural correlates of self-agency attribution are still unclear. Several studies have hypothesized the role of the frontal and prefrontal cortex in the sense of agency generation (see for example Chambon, Wenke, Fleming, Prinz, & Haggard, 2013; Renes, van Haren, Aarts, & Vink, 2015). Other studies emphasized the role of the parietal cortex (see for example Leube, Knoblich, Erb, & Kircher, 2003), insula (see for example Farrer et al., 2003; Farrer & Frith, 2002; Leube, Knoblich, Erb, Grodd, et al., 2003) and cerebellum (see for example Fukushima, Goto, Maeda, Kato, & Umeda, 2013). For a graphical representation of the brain regions that have been associated with the self-agency experience, please see Figure 1a.

New insights come from studies that explored the brain mechanisms of the sense of agency by taking advantage of the intentional binding phenomenon. Those studies have consistently shown a link between the activity of the supplementary motor area (SMA), with reference to its anterior portion (pre-SMA), and the magnitude of the intentional binding effect (Cavazzana, Penolazzi, Begliomini, & Bisiacchi, 2015; Kühn et al., 2013; Moore, Ruge, et al., 2010; Seghezzi & Zapparoli, 2020; Zapparoli, Seghezzi, Zirone, et al., 2020). For a graphical representation of the brain regions that have been associated with the intentional-binding effect, please see Figure 1b.

**Figure 1** | The neurobiology of the sense of agency: (a) results of studies using explicit measures of agency; (b) results of studies using intentional binding paradigms.



However, it is becoming increasingly clear that such a complex phenomenon like the sense of agency experience is more likely to be associated with the functioning of brain in networks instead of single structures. One hypothesis is that the key neural substrate of the sense of agency lies in the effective connectivity between the pre-SMA that initiates actions and parietal areas that monitors the generating consequences of actions (P. Haggard, 2017).

### **Sense of agency disturbances in movement disorders**

To provide a comprehensive overview of the behavioural, structural, and functional brain abnormalities linked to the sense of agency experience in movement disorders patients, we performed literature research in the PubMed database (<https://pubmed.ncbi.nlm.nih.gov>).

Specifically, the studies included in this review were retrieved through the following queries: [“Sense of agency” AND [ “Movement disorders” OR “Parkinson’s disease” OR “Gilles de la Tourette” OR “Functional Movement Disorders” OR “Psychogenic Movement Disorders” OR “Huntington’s disease”]]. The initial set of studies included 41 papers, updated to April 2021. After the removal of duplicates, the set included 33 papers. Papers were then included in the review when fulfilling the following inclusion criteria:

- Populations involved: adult subjects; no minimum sample size was required.
- Movement disorder: we selected papers focused on primary/idiopathic movement disorders (e.g., Parkinson’s Disease, Gilles de la Tourette Syndrome, corticobasal Syndrome, Functional Movement Disorders and Huntington’s disease), rather than secondary/acquired disorders (e.g., movement disorders after trauma, vascular accidents or secondary to pathological conditions, like schizophrenia).
- Methods: explicit judgments of agency or implicit measures of agency (i.e., intentional binding and/or sensory attenuation).

Following these inclusion criteria, we selected twelve studies. Among those, four studies explored the sense of agency experience in Parkinson’s disease patients; two studies involved Gilles de la Tourette patients; one study assessed a sample of corticobasal syndrome patients, and five studies assessed the sense of agency experience in patients with functional movement disorder.

### **Parkinson’s disease**

Parkinson’s disease (PD) is a degenerative disorder characterized by motor impairments, including tremor at rest, rigidity, akinesia (or bradykinesia) and postural instability. In addition, marked difficulties in planning, initiating and executing voluntary movements have been included among classic features of PD (Jankovic, 2008). The symptoms’ appearance relates to degeneration of dopamine-producing neurons in the nigrostriatal pathway, resulting in a dopaminergic deficiency in the striatum, particularly the putamen (Agid & Blin, 1987). Accordingly, dopaminergic drugs represent the standard treatment.

Given the difficulty in initiating and controlling voluntary movements, sense of agency disturbances in PD patients are largely expected. Saito et al. (2017) tested this hypothesis by using both explicit and implicit measures of the sense of agency. PD patients showed less attribution of the given feedback to themselves compared to the control group. Moreover, while the control group showed the canonical intentional binding phenomenon for both the action (action-binding) and the effect (tone-binding), actions were not experienced as shifted towards their subsequent effects in the patients’ group (i.e., reduced action-binding effect). These findings were consistent regardless of motor symptoms’ side, supporting a linkage to primary deficits in central sensorimotor processing. Importantly, patients were under regular dopaminergic medication. Differently, Moore et al. (2010) investigated the sense of agency experience in patients both off and on dopaminergic drug therapy with the intentional binding paradigm. The magnitude of the overall-binding in PD patients OFF medication was similar to healthy controls. However, the same patients tested while ON medication showed a significant increase in the overall-binding. This suggests the disease itself may not directly induce changes in the patient’s sense of agency. Instead, changes in the sense of agency might be mainly related to the dopaminergic medication.

Moore et al. (2010) also suggested that an increased agency experience may contribute to impulsive-compulsive behaviours (ICBs) in PD, which is a common neuropsychiatric complication associated with dopaminergic treatment (Weintraub, David, Evans, Grant, & Stacy, 2015). Perceiving one’s actions as highly effective might lead to a tendency to perform actions that would otherwise be inhibited. This hypothesis would explain why PD-ICB patients show stronger action-binding than PD without ICB (Ricciardi et al., 2017).



The effect of the dopaminergic medication on changes in the sense of agency is also evident from sensory attenuation data in PD. Wolpe et al. (2018) tested PD patients on a force matching task to measure the sensory attenuation. Overall sensory attenuation did not differ between medicated PD patients and controls. However, the degree of attenuation was negatively related to PD motor symptoms and positively related to individual dopamine dose, measured by levodopa dose equivalent scores. For a synopsis of these studies, see Table 3.

**Table 3** | Studies on the sense of agency in Parkinson’s disease patients.

Study			
Author and year	Participants	Agency measure collected	Main findings
<b>Parkinson’s disease (PD)</b>			
J.W. Moore et al. (2010)	9 PD patients ON and OFF dopaminergic medication and 9 healthy controls (HC).	Implicit (Intentional binding measure).	<ul style="list-style-type: none"> <li>Stronger overall-binding in PD patients ON medication.</li> <li>No difference between PD patients OFF medication and HC.</li> </ul>
L. Ricciardi et al. (2017)	19 PD patients on dopaminergic medication with ICB (PD-with impulsive compulsive behaviours), 19 PD-no-ICB and 19 HC.	Implicit (Intentional binding measure).	<ul style="list-style-type: none"> <li>Stronger action-binding in PD-ICB.</li> <li>No difference between PD-no-ICB and HC.</li> <li>No difference in tone-binding.</li> </ul>
N. Saito et al. (2017)	9 PD patients on dopaminergic medication and 25 HC.	Explicit judgments about visual feedback. Implicit (Intentional binding measure).	<ul style="list-style-type: none"> <li>Reduced agency attribution of a given feedback to themselves in PD.</li> <li>Less action-binding in PD.</li> <li>No difference in tone-binding.</li> </ul>
N. Wolpe et al. (2018)	18 Patients with PD on dopaminergic medication and 175 HC.	Implicit (Sensory attenuation measure).	<ul style="list-style-type: none"> <li>No difference in the overall sensory attenuation.</li> <li>The degree of attenuation is negatively related to PD motor symptoms.</li> <li>The degree of attenuation is positively related to individual patient dopamine dose.</li> </ul>

In general, the most notable tendency in the PD patients’ behaviour is the great predominance of external cues rather than internal goals to guide behaviour. In PD, motor deficits are often observed in the absence of external signals, when movements must be internally generated (Georgiou et al., 1993), and providing patients with external cues can improve movement performance (Jahanshahi & Frith, 1998). It follows that agency disturbances in PD patients might be associated with a failure to weigh internal and external cues to provide normal agency experiences, with an exaggerated reliance on external cues and a reduced precision of internal cues. This would explain, for example, why PD patients show less self-agency attribution when their actual movements are concealed, making it difficult for the patients to use external cues to form correct judgments of agency (Saito et al., 2017). However, the brain mechanisms underlying such predominance of external cues rather than internal goals in agency attribution are still unknown.

In the context of active inference theory (Friston et al., 2011), it has been suggested that bradykinesia in PD can be modelled as a failure of downweighing the precision of the incoming sensory evidence that facilitates the motor execution through the fulfilment of the internal motor predictions (Macerollo et al., 2016). This might explain why PD patients strongly rely on external sensory cues while executing motor tasks (Abbruzzese & Berardelli, 2003). The proposal of a dysfunctional physiological sensory attenuation in PD has been confirmed. Macerollo et al. (2016) showed that, off medication, PD patients had a decreased sensory attenuation at movement onset. This returned at normal levels with dopaminergic treatment. These results, together with clinical reports indicating that dopaminergic treatment makes patients less dependent on ongoing visual control (Baroni, Benvenuti, Fantini, Pantaleo, & Urbani, 1984), suggest that dopamine has a positive

effect on movement impairment in PD by restoring the precision of sensorimotor predictions through sensory attenuation. The agency indexes in PD show the same correlation between the physiological sensory attenuation and the dopaminergic treatment. On the one hand, psychophysical sensory attenuation is negatively associated with disease severity and positively correlated with the dopamine dosage. On the other hand, dopaminergic medication boosts action-effect binding in PD patients. Therefore, agency disturbances in PD patients can be traced back to the failure of adequate physiological sensory attenuation in downregulating the external sensory cues. This pathological mechanism may explain the psychophysical disturbances of the sense of agency observed in PD patients, namely less explicit attribution of the feedback to themselves (Saito et al., 2017) and alterations of the intentional binding effect (Moore, Schneider, et al., 2010; Ricciardi et al., 2017). Moreover, it looks plausible that dopamine treatment, acting by restoring the precision of sensorimotor predictions through sensory attenuation, improves not only motor symptoms in PD but it also re-establishes accurate agency judgments, normal psychophysical sensory attenuation and intentional binding effect (Wolpe et al., 2018).

### **Gilles de la Tourette Syndrome**

Gilles de la Tourette Syndrome (GTS) is a childhood-onset movement disorder characterized by the presence of hyperkinetic movements and abnormal vocalizations called tics (Leckman, Bloch, Smith, Larabi, & Hampson, 2010). The voluntary or involuntary nature of tics is still unclear (Cavanna & Nani, 2013). Moreover, several environmental factors can modulate the severity and the occurrence of tics (Cohen, Leckman, & Bloch, 2013). Stress and anxiety are the most common factors that induce and exacerbate tics. In contrast, when patients are relaxed or engaged in specific activities requiring concentration or physical effort, tic severity and frequency are attenuated (Misirlisoy et al., 2015). Most GTS patients report that tics are often preceded by “premonitory urges”, namely uncomfortable sensory phenomena characterized by restlessness, pain, pressure, mounting tension or vague discomfort. These sensations can only be relieved by the tic expression, similar to the relief following scratching or sneezing. Importantly, most GTS patients can also voluntarily suppress their tics for a short while. Therefore, the imperative nature of the premonitory urge imposes the expression of the tic that is performed against the individual’s will; however, the decision to actuate the tic is usually perceived as a voluntary response to the unpleasant sensation (Cavanna & Nani, 2013).

Delorme and colleagues (2016) investigated the sense of agency in GTS patients testing the subjects’ ability to recognize incongruences between their actions and visual feedback and to make appropriate explicit judgments of agency. GTS patients reported an illusory perceived sense of agency when their performance was artificially enhanced, suggesting that they did not realize that they were not fully responsible for the observed outcome and they inflated their judgment of agency (Delorme, Salvador, et al., 2016). Moreover, GTS patients did not experience the overall-binding effect, showing a reduced implicit agency experience (Zapparoli, Seghezzi, Devoto, et al., 2020). This result was mirrored by the absence of the significant correlation between the pre-SMA activity and the binding effect observed in the healthy controls. Importantly, the degree of overall-binding was negatively related to GTS motor tic severity, with reduced binding associated with a more severe motor impairment (Zapparoli, Seghezzi, Devoto, et al., 2020). For a synopsis of these studies, see Table 4.

**Table 4** | Studies on the sense of agency in Gilles de la Tourette patients.

Study			
Author and year	Participants	Agency measure collected	Main findings
<b>Gilles de la Tourette syndrome (GTS)</b>			
C. Delorme et al. (2016)	37 GTS patients and 19 HC.	Explicit judgments about the degree of control over a cursor, that could be normal, disrupted or artificially enhanced.	<ul style="list-style-type: none"> <li>GTS patients show an illusion of agency in the task condition where their performance was artificially enhanced.</li> <li>Illusion of agency negatively correlates with global disease severity.</li> </ul>
L. Zapparoli et al. (2020)	25 GTS patients and 24 healthy controls (HC)	Implicit (Intentional binding measure).	<ul style="list-style-type: none"> <li>Reduced overall-binding effect in GTS.</li> <li>The binding reduction significantly correlates with the severity of the motoric symptoms.</li> <li>GTS did not show any correlation between the overall binding effect and the activity of a broad sensory-motor system that correlates with overall-binding in HC.</li> </ul>

Contrary to PD, where external cues can improve movement performance (Jahanshahi & Frith, 1998), the severity of tics in GTS patients it's rarely influenced by environmental cues (Wolpe, Hezemans, & Rowe, 2020). Instead, tic expression in GTS can vary as a function of psychological, internal, factors and attention (Misirlisoy et al., 2015). One can suggest a greater predominance of internal cues rather than external signals in guiding motor behaviour. The same mechanism would explain why GTS patients rely predominantly on their perceived performance to provide agency judgments and less on their objective degree of control signalled by external feedback on their performance (Delorme, Salvador, et al., 2016). However, the etiopathogenesis of such an imbalance between internal and external cues to guide GTS' behaviour is still unknown, as its relationship with the motoric manifestation of the syndrome is still unclear.

Rae et al. (2019) have recently proposed a radical mechanistic explanation of the motor (and not motor) symptoms that characterize Gilles de la Tourette syndrome (Rae, Critchley, & Seth, 2019). Precisely, tics would be generated through over-precise action predictions within the intermediate levels of the cortical hierarchy, specifically the putamen, and lack of correspondingly precise priors for action at highest levels (i.e., pre-SMA). According to the active inference framework, normal behaviour requires that precise higher-level beliefs about intended outcomes are spread down the cortical hierarchy to 'explain away' prediction errors from lower sensorimotor levels. However, when intermediate sensorimotor priors are overly precise, they fail to be informed by higher-level goals and beliefs. Instead, they are informed by the somatosensory input from lower hierarchical levels (Wolpe et al., 2020). From a neuroanatomical point of view, the abnormally precise priors within the putamen would arise through an overactivity of the SMA (but not pre-SMA) leading to increased glutamatergic (excitatory) inputs to the putamen and reduced density of GABAergic interneurons in the putamen, which causes aberrant synaptic integration. Consequently, over-precise predictions for action would be generated from the putamen, and movement would resolve the corresponding proprioceptive prediction errors, even though the precision of priors for these actions within higher levels of the motor circuit is low (perhaps reflected in diminished pre-SMA activity). This would result in thalamic disinhibition and the release of signals for movement in M1. Moreover, since the induced movement is inconsistent with the patient's determined intentions and goals, represented by high-level beliefs, this would determine a concomitant abnormal experience of agency or "alienness" for the generated action. Consequently, implicit measures of agency, such as the psychophysical sensory attenuation and the intentional binding paradigm should be reduced, as suggested by Zapparoli et al. (2020). Contrary to what was observed in healthy controls, GTS patients did not show any noticeable correlation of the pre-SMA activity with the magnitude of the intentional

binding phenomenon, supporting the idea of a lack of precise priors for action within the pre-SMA. Finally, the magnitude of binding was negatively related to GTS tic motor severity, with less binding effect for more severe motor impairment (Zapparoli, Seghezzi, Devoto, et al., 2020).

### Corticobasal syndrome

Corticobasal syndrome (CBS) is a movement disorder characterized by progressive asymmetric cortical and extrapyramidal dysfunctions. CBS can occur in the absence of an identifiable biological cause, but it is usually due to an underlying neurodegenerative disorder (Gibb, Luthert, & Marsden, 1989). CBS is typically associated with two disorders of volitional actions: alien limb and apraxia. The former refers to the execution of semi-purposeful movements in the absence of will; the latter consists of an impairment in the performance of complex movements despite the understanding of their goal. One can expect that such abnormalities in voluntary motor control may also affect the conscious experience that normally accompanies voluntary action, including the sense of agency.

Wolpe et al. (2014) used the intentional binding paradigm combined with multimodal brain imaging to investigate possible abnormalities in the sense of agency experienced over the more severely affected limb. They found increased action-binding in patients with CBS relative to control subjects, limited to the affected hand. Tone-binding was similar to controls, for both hands. The increased action-binding positively correlated with the severity of alien limb and apraxia symptoms for the affected hand. Moreover, action-binding increase was positively associated with structural changes in pre-SMA, and with augmented functional connectivity at rest between pre-SMA and a fronto-parietal network. Even though there is evidence of impairments in the ability to formulate correct agency judgment in patients who had developed apraxic symptoms following left parietal lesion (Sirigu, Daprati, Pradat-Diehl, Franck, & Jeannerod, 1999), to date, no investigations have formally assessed the explicit subjective experience of agency nor sensory attenuation in CBS patients. For a synopsis of these studies, see Table 5.

**Table 5** | Studies on the sense of agency in corticobasal patients.

Study			
Author and year	Participants	Agency measure collected	Main findings
<b>Corticobasal syndrome</b>			
N. Wolpe et al. (2014)	10 patients meeting clinical diagnostic criteria for corticobasal syndrome (CBS) and 16 healthy subjects.	Implicit (Intentional binding measure).	<ul style="list-style-type: none"> <li>Increased action-binding in CBS.</li> <li>Behavioural variability was related to changes in grey matter volume in pre-SMA.</li> <li>Changes in functional connectivity at rest between the pre-SMA and prefrontal cortex were proportional to changes in action-binding.</li> </ul>

Corticobasal syndrome patients share with Gilles de la Tourette syndrome the experience of “alienness” for the generated movements. However, contrary to the GTS condition, CBS patients’ hyperkinetic production is deeply influenced by environmental cues (Wolpe et al., 2020). The alien hand condition represents one of the most obvious expressions of abnormal reliance on an environmentally triggered motor schema for action selection (Wolpe et al., 2014). The prominent predominance of external cues to guide behaviour in CBS is also evident in the manifestations of agency. In particular, the over-reliance on external cues results in an increased temporal bias in the estimated time of the action, which is more pronounced than in healthy controls (Wolpe et al., 2014). In other words, unreliable information about the action event would then lead to an over-reliance on external cues, resulting in an increased bias towards action timing (Wolpe et al., 2014).

Wolpe et al. (2020) have recently proposed a biologically plausible account of both corticobasal syndrome (Wolpe et al., 2014), and alien limb syndrome (Wolpe et al., 2020). Accordingly, the pre-SMA dysfunctions

at higher levels of the hierarchy would lead to over-precise predictions at the intermediate level, which in turn propagate down to the motor cortex to induce semi-purposeful movements that are perceived as involuntary (Wolpe et al., 2020). Although this model of alien limb sounds very similar to the model proposed to account for tics in Gilles de la Tourette syndrome (Rae et al., 2019), the GTS model involves different neuroanatomical mechanisms, namely the increased excitatory activity from the SMA to the putamen, and reduced striatal inhibition, which lead to relatively precise priors for action in the putamen. However, these models still lack a solid corpus of experimental studies able to confirm their predictions.

### **Functional movement disorders**

Functional movement disorders (FMD) are part of the spectrum of functional neurologic disorders (Morgante, Edwards, & Espay, 2013). The definition of FMD is controversial. DSM-5 categorizes most FMD as Conversion Disorder, or Functional Neurological Symptom Disorder, within the general category of Somatic Symptom and Related Disorders (Hallett, 2016). In general terms, FMD conditions are considered as psychological or psychiatric rather than neurologic conditions (Morgante et al., 2013). However, making the diagnosis is often challenging because an overt psychological dysfunction is not always evident (Morgante et al., 2013). Moreover, morphometric magnetic resonance imaging techniques have detected subtle changes in FMD patients' brain volume and cortical thickness (Perez, Matin, et al., 2017; Perez et al., 2018; Perez, Williams, et al., 2017), challenging the assumption of a non-organic etiopathogenesis of the condition. FMD clinical manifestations are highly heterogeneous: involuntary movements may affect multiple body parts or be isolated to a single body segment, and they could be present at rest or appear only during specific tasks (Albanese et al., 2013). Symptoms are generally modulated by attention, significantly reducing their severity when patients do not specifically focus on their symptoms. Conversely, during the examination, abnormal movements are often present with considerable strength (Edwards, Fotopoulou, & Pareés, 2013). Patients with functional disease have no sense of agency towards their functional movements (Hallett, Weiner, & Kompoliti, 2012). However, functional movements have characteristics that seem to imply some voluntary control. For example, when patients are asked to tap the same rhythm as the examiner, the tremor can sometimes assume the same frequency as the tapping (Schwingenschuh et al., 2011). Moreover, functional symptoms may occur at latencies after sensory triggers that resemble voluntary reaction times and may be preceded by cortical potentials characteristic of self-paced voluntary actions (Edwards and Bhatia, 2012). This implies that agency attribution in FMD might be far from easy (Edwards et al., 2013).

Nahab et al. (2017) showed that FMD patients report abnormal agency ratings compared to healthy subjects. In that study, all subjects wore a data glove on their right hand, and they were asked to make sequential finger movements. They observed a computer screen that displayed feedback that mimicked their movements completely (100% control), randomly (0% control), or in an intermediate way (25%, 50%, or 75% control). Contrary to healthy subjects, FMD patients reported a continued sense of agency over the movement feedback despite having lost most or all control. This impairment was selectively associated with dysfunctions at the level of the agency neural network, whereby the dorsolateral prefrontal cortex and pre-SMA did not respond differently to the loss of movement control (Nahab, Kundu, Maurer, Shen, & Hallett, 2017). Further evidence is provided by a study with the intentional binding paradigm. Kranick et al. (2013) reported that patients with conversion disorder manifestations showed reduced overall-binding compared to healthy volunteers, suggesting a decreased sense of control over their actions. Finally, a loss of sensory attenuation during self-generated movements (e.g., abduction of the thumb) has been found in different types of FMDs compared to healthy volunteers (Pareés et al., 2014). For a synopsis of these studies, see Table 6.

**Table 6** | Studies on the sense of agency in functional movement disorder patients.

Study			
Author and year	Participants	Agency measure collected	Main findings
<b>Functional movement disorders (FMD)</b>			
S.M. Kranick et al. (2013)	20 patients with motor conversion disorder	Implicit (Intentional binding measure).	<ul style="list-style-type: none"> <li>Reduced overall-binding in patients.</li> </ul>
I. Pareés et al. (2014)	14 FMD patients and 14 HC.	Implicit (Sensory attenuation measure).	<ul style="list-style-type: none"> <li>Reduced sensory attenuation in FMD.</li> </ul>
A. Marotta et al. (2017)	21 Patients with FMD and 21 HC.	Explicit judgments during a moving Rubber Hand Illusion, in which passive and active movements can differentially elicit agency.	<ul style="list-style-type: none"> <li>No differences between FMD patients and HC in explicit judgments of agency.</li> </ul>
F. Nahab et al. (2017)	21 patients with FMD and 20 HC.	Explicit judgments while participants observed a computer screen that displayed a moving hand that mimicked their movements completely (100% control), was completely random (0% control), or was an intermediate mixture (25%, 50% or 75% control).	<ul style="list-style-type: none"> <li>FMD experience the tendency to overestimate control over the virtual hand.</li> <li>FMD patients lacked the ability to recognize the loss in control.</li> <li>The dorsolateral prefrontal cortex and pre-supplementary motor area on the right did not respond differentially to the loss of movement control as in HC.</li> </ul>
A. M. L. Huys et al. (2020)	23 patients with FMD and 26 HC.	Explicit judgments during subliminal and supraliminal priming conditions.	<ul style="list-style-type: none"> <li>No differences between FMD patients and HC in explicit judgments of agency.</li> </ul>

It is evident that sense of agency manifestations in FMD patients are highly heterogeneous. FMD patients showed a reduced sense of control over their actions in the classical action-recognition tasks (Delorme, Roze, et al., 2016; Nahab et al., 2017), reduced implicit measures of agency in sensory attenuation (Pareés et al., 2014), and in intentional binding tasks (Kranick et al., 2013). In this regard, an important difference with respect to PD (Saito et al., 2017), which showed a reduced action-binding but a normal tone-binding, is that in FMD patients, the action-binding is preserved while the tone-binding is reduced (Kranick et al., 2013). These results might suggest a greater predominance of internal cues rather than external goals to guide behaviour (Stenner & Haggard, 2016). However, the etiopathogenesis of such an imbalance between internal and external cues to guide behaviour is still debated.

Stenner and Haggard (Stenner & Haggard, 2016) emphasize the role of the so-called “precipitating physical events”, in explaining the greater predominance of internal rather than external cues in FMD patients’ behaviour. In particular, the authors suggested that “precipitating events”, like physical injury or panic attacks, in FMD patients are subjectively interpreted as the consequence of a loss of control, resulting in increased monitoring of action. The increased monitoring would then generate expectations (or predictions) of a strong, conscious, and vivid experience of being in control with actions. According to Edwards et al. (2012), these beliefs would be unconsciously generated at an intermediate level in a cortical hierarchy. Those expectations of a strong, conscious experience in controlling actions would then collide with conscious control over actions that the motor system is physiologically unable to provide (Stenner & Haggard, 2016). Therefore, FMD patients would then interpret this “thin” phenomenology of action as abnormal. The enhanced attention to motoric details of the action, like the parameters of motor execution, would thus result in a more accurate perception of the sensory consequences of movement. This is evident in a reduced physiological sensory attenuation (Macerollo et al., 2015), less psychophysical sensory attenuation (Pareés et al., 2014) and reduced tone-binding in FMD patients compared to healthy subjects (Kranick et al., 2013). Moreover, the same excessive attention towards the mechanics of movement is likely to increase the weighting of top-down priors

and decrease, in turn, the weighting of incoming proprioceptive information. This hypothesis has been confirmed by a recent study showing that movement perception of the tonic vibration reflex is reduced in patients with functional weakness, and the proprioceptive deficit is independent of motor impairment (Tinazzi et al., 2021).

## Conclusions

Our review indicates that an altered sense of agency characterizes several movement disorders. Agency disturbances in Parkinson's disease were revealed by a reduction of the subjective agency and an altered action-binding in intentional binding paradigms (Saito et al., 2017). Sense of agency disturbances in Gilles de la Tourette syndrome may occur as an inflated subjective experience of agency (Delorme, Salvador, et al., 2016) or as the absence of an overall-binding effect (Zapparoli, Seghezzi, Devoto, et al., 2020). This latter was mirrored by the absence of any significant correlation between the pre-SMA activity and the binding effect (Zapparoli, Seghezzi, Devoto, et al., 2020). Corticobasal syndrome patients showed increased action-binding, which correlated with structural changes in pre-SMA grey matter, and functional connectivity at rest between the pre-supplementary motor area and a fronto-parietal network (Wolpe et al., 2014). FMD patients overestimate their control over their action outcomes, and they lack the ability to recognize when they lose control over their actions (Nahab et al., 2017). This impairment is associated with dysfunctions at the level of the dorsolateral prefrontal cortex and pre-SMA (Nahab et al., 2017). FMD patients also showed a reduced action-binding (Kranick et al., 2013) and a loss of sensory attenuation during self-generated movements (Pareés et al., 2014).

However, from the present review, it is evident that the literature often provides incomplete and divergent results, with some important critical issues when considering evidence from different – explicit and implicit – measures of agency. For example, in GTS patients, inflated agency judgments (Delorme, Salvador, et al., 2016) coexist an absent intentional binding effect (Zapparoli, Seghezzi, Devoto, et al., 2020). Similarly, agency overestimations (Nahab et al., 2017) co-occur with reduced intentional binding (Kranick et al., 2013) and sensory attenuation (Pareés et al., 2014) in FMD patients. A possible explanation for these contradictory results lies in the hypothesis that there are separable and to some extent independent, agency processing systems (Synofzik, Vosgerau, & Newen, 2008b). Explicit indexes capture the conceptual, interpretative experience of agency (the so-defined "Judgement of agency", Synofzik et al., 2008a). Implicit measures, like the intentional binding effect (P. Haggard et al., 2002) or sensory attenuation (Blakemore et al., 1998), are designed to study the elusive low-level feeling of being the agent of an action (the so-defined "Feeling of agency", Synofzik et al., 2008a). It follows that the implicit and explicit agency systems may be differently affected by the syndromes. However, psychophysical and physiological validations are required to explain why explicit and implicit agency measures are sometimes coherent and sometimes dissociate in patients.

A second important criticality regards the limited evidence of the brain correlates of sense of agency disturbances in different movement disorders. From this review, a mismatch emerges between the amount of data available regarding the investigation of sense of agency in movement disorder and on the physiological basis of the sense of agency in healthy participants - the latter far outweighing the former. Therefore, it is difficult to understand to what extent different movement disorders might share similar recruitment of the sense agency brain network. Wolpe et al. (2014) and Zapparoli et al. (2020) drew attention to the role of pre-SMA in accounting for the sense of agency disturbances in corticobasal and Gilles de la Tourette syndromes, respectively. Yet, evidence is restricted to specific syndromes and limited to implicit measures, while there is no evidence in favour of a generalization of the results to other agency indexes.

Another critical issue concerns the clinical heterogeneity characterizing movement disorders. Several conditions (for example, PD) are neurodegenerative disorders with a plethora of different signs and symptoms, each with specific pathophysiology. These have been only rarely considered in the mentioned studies. For example, Saito et al. (2017) reported that PD patients showed less attribution of the given feedback to

themselves and reduced action-biding effect than the control group, and the impairments were consistent regardless of motor symptoms' side. However, motor symptoms in PD are very diverse (e.g., rest tremor and bradykinesia) and they may be sometimes difficult to isolate on one side of the body coherently. Also, one of the aspects that makes hyperkinetic movement disorders (e.g., GTS) quite difficult to investigate is the phenomenology of the involuntary movement themselves, which has been rarely considered in the mentioned studies.

The present review shows that impaired agency experiences are dramatically common in a wide range of different movement disorders. However, in addition to the agency experience, voluntary movements also entail another level of conscious action experience, namely the experience of being the source of the actions (P. Haggard & Clark, 2003). This is the experience of motor intentionality. While the sense of agency implies the experience of the consequences of the action ("I" caused this, rather than another agent), intention relates to the experiences of action preparation and effort that precede the motor execution (P. Haggard & Clark, 2003). Sense of agency and intention are closely linked since a conscious experience of intending is somehow necessary for the arising of a sense of agency. Here we focused primarily on the sense of agency rather than on intention. However, abnormal experiences of intention in movement disorders have been largely described. For example, awareness of intention to act has been shown to be delayed in PD patients (even though those results have not been replicated by Di Costa et al., 2020; Tabu et al., 2015), GTS patients (Moretto, Schwingenschuh, Katschnig, Bhatia, & Haggard, 2011) and FMD patients (Baek et al., 2017; Edwards et al., 2011). A primary impairment in voluntary motor intention at an early processing stage might explain some cases of abnormal agency experience. However, to date, no study has investigated the relationship between delayed awareness of action and abnormal sense of agency in those patients.

A final consideration concerns the (neuro)cognitive theories developed to address the arising of the sense of agency. Those theories imply different levels of description, focusing on the top-down inferential processes (Wegner, 2003), the motor processes (Franklin & Wolpert, 2011; Wolpert & Ghahramani, 2000) and the fine-grained neural mechanisms (Brown et al., 2013). Crucially, none of the hypothesized (neuro)cognitive theories per se can account for all the facets of the sense of agency and its disturbances in different clinical populations. However, a promising breakthrough may be offered by combining the levels of explanations to provide a coherent and exhaustive explanation of the sense of agency alterations in different movement disorders. The present review represents the first step in this direction, showing that the combination of internal and external cues - which has been separately claimed by the comparator model (Blakemore et al., 2002; Frith et al., 2000b) and the apparent mental causation theory (Wegner, 2003) - can be solved within the active inference framework. This framework offers a different explanatory level that emphasizes the role of physiological phenomena in accounting for the psychophysics correlates of the agency attribution.



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