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Reframing schizophrenia and autism as bodily self-consciousness disorders leading to a deficit of theory of mind and empathy with social communication impairments

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ABSTRACT

Prior observations and studies suggest self-consciousness disorders in schizophrenia and Autism Spectrum Disorder (ASD), two neurodevelopmental disorders sharing social communication impairments. First, the relationships between schizophrenia and autism are explored regarding social communication impairments. Then, self-consciousness disorders in schizophrenia and autism are described and discussed in relation with impairments of body self leading to impairments of self-other differentiation, a deficit of theory of mind and empathy, and their consequences on social communication. Also, neurological dysfunction involved possibly in self-consciousness disorders in schizophrenia and autism is presented. In conclusion, a new model is proposed integrating results of studies presented here and stating the existence of bodily self-consciousness disorders in schizophrenia and autism associated with altered/absent intermodal sensory integration (especially visual-kinesthetic-tactile integration). This would result in problems of self-other differentiation, leading in turn to a deficit of theory of mind and empathy as well as social communication impairments. This model opens new perspectives to understand better self-consciousness disorders and social communication impairments in schizophrenia and ASD and to develop therapeutic strategies.

1. Introduction

The question of relationships between autism and schizophrenia is not recent. The term autism (etymologically, *autos* in old Greck means *self-centered)* was introduced by Bleuler (1993) to describe social withdrawal in adults with schizophrenia. At that time, the existence of infantile forms of schizophrenia had already been recognized thanks to De Sanctis who since 1906 reported, among his patients, cases of "précocissime dementia" referring to the early dementia of Kraeplin (McClellan and Stock, 2013; Hochman, 2009). Kanner (1943), borrowed the term "autism" from Bleuler and applied it to a group of 11 children who were described as suffering from "child schizophrenia", but whose clinical signs, notably social withdrawal, seem to inscribe them in a different psychopathological framework. Until 1970, all different child psychotic disorders and pathologies implying global developmental processes were grouped together in the same category entitled "child schizophrenia". Based on the work of Kolvin (1971) and Rutter (1972), the boundaries between schizophrenia and autism were redesigned. This led to the separation of these two disorders in the main international classifications (ICD-9 then ICD-10, and DSM-III, DSM-IV and DSM-5).

Even today, many authors question the relationships between autism and schizophrenia, especially for early-onset schizophrenia, with possible common dimensions and psychopathological mechanisms. Early-onset schizophrenia is a rare disorder. Its prevalence is estimated at 0.03% or 50 times less than adult-onset schizophrenia (Remschmidt and Theisen, 2012) with a sex ratio that is approximatively 1.4 boy for 1 girl (Bailly et al., 2003). It corresponds to schizophrenia that begins before the age of 18 and includes two forms of schizophrenia: a form of schizophrenia with a very early onset before the age of 13 (prevalence estimated at 0.002%), and a later onset form beginning between the ages of 13 and 17 that would be closer to the adult form. The concept of early-onset schizophrenia raises many questions about diagnosis. Indeed, early-onset schizophrenia refers strictly to the diagnostic criteria for schizophrenia in its "classical" form, as described in the latest versions of the international diagnostic

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classifications, the ICD-10 (World Health Organization, 1993) and the DSM-5 (American Psychiatric Association, 2013). However, the evolution of the place of this disorder in the nosography is of interest because it questions the possible links between schizophrenia, as a chronic psychiatric disorder, and developmental disorders, in particular autism. One of the limitations of the current diagnostic classifications (ICD-10 and DSM-5) is that by separating completely schizophrenia from Autism Spectrum Disorder (ASD), many young patients can no longer be formally related to one or the other diagnosis. The current classification system appears often to be too strict and is associated with frequent diagnostic errors (Remberk et al., 2014; Castro-Fornieles et al., 2011). Also, these classifications elude almost any developmental perspective that appears to be indispensable in individuals whose psychic construction is still at work. The early diagnosis of schizophrenia but also the screening of people at high or very high risk of schizophrenia/psychosis are important issues in contemporary psychiatry. However, the diagnostic criteria for schizophrenia, as defined in the DSM or ICD classifications, do not make it possible to achieve such an objective. Indeed, these diagnostic criteria do not have sufficient sensitivity and validity for early diagnosis of schizophrenia (Nordgaard et al., 2012; Jansson et al., 2002). Several authors considered that the diagnostic criteria for schizophrenia in DSM and ICD classifications have simplified drastically the clinical symptoms without taking into account alterations of subjective experience, such as self-distortions, and therefore reflect only partially the clinical picture of schizophrenia (Parnas and Handest, 2003). However, the identification of such subjective impairments could be of great clinical utility to identify individuals at high risk of psychosis.

Schizophrenia and ASD share social communication impairments that may rely partly on self-consciousness disorders. Self-consciousness is a multidimensional concept that involves as much the awareness of one's own body and image as the recognition of oneself with awareness of one's own identity. For the psychologist William James (1890), the self involves two intertwined aspects: self-awareness, the "*I*", and the representations of self, the "*Me*". This dual concept allows people to understand the multi-faceted nature of self while keeping a sense of continuity and unicity by the feeling of being only one person. Inspired by the ideas of the French psychologist Henri Wallon (1959), the American psychologist Ulric Neisser (1991) described two distinct ways of building the self, highlighting the central role of the body and the other: through bodily perceptions and interactions with surrounding objects, and through interactions with the other. As early as 1921, Freud stated that "The Ego is above all a corporeal entity". The philosopher Maurice Merleau-Ponty (1945) insisted on the importance of the "lived body" in self-consciousness, giving access to intersubjectivity through intercorporality. During fetal life, the body self develops through intermodal sensory perception (particularly, tactile-kinesthetic perception) and perception-action coupling which lead to the construction of a sensory-motor repertory, the development of a body schema and the sense of a coherent bodily unity. This sense of a coherent bodily unity through congruent sensori-motor informations is the basis of the fetal development of an implicit sense of self. Philippe Rochat (2019) hypothesizes that self-unity at birth is the necessary ground zero enabling very early learning and development. From birth, the baby is able to discriminate his/her own tactile stimulation from others'tactile stimulation, suggesting the existence of a body self (Butterworth and Hopkins, 1988; Rochat and Hespos, 1997). When the baby's hand touches his/her mouth (self-stimulation), this contact is not followed by the rooting reflex whereas this reflex appears following tactile stimulations from another person. This discrimination results from intermodal sensory integration: when the baby touches his/her cheek, tactile and proprioceptive sensations are integrated. Ultrasonography (ultrasound) shows that bodily exploration behaviors are already present and visible in utero, suggesting that the development of the body self could begin during fetal life. In the third trimester of pregnancy, the fetus sucks his or her thumb (Prechtl, 1984). Movement of the hand to the mouth occurs between 50 and 100 times per hour in fetuses of 12 to 15 weeks of gestational age (De Vries et al., 1985). Between 14 and 37 weeks of gestational age, the fetus touches frequently with his/her hands, not only his/her face and mouth but different parts of the body as well as the uterine wall, even though this frequency fluctuates during the weeks until delivery (Sparling et al., 1999). An active hand-mouth coordination is also observed at birth. Korner and Kraemer (1972) reported that babies spend more than 20% of their waking hours touching their faces with their hands. For some authors, hand-mouth coordination is a primary form of tactile-kinesthetic exploration of one's own body (Kravitz et al., 1978). The presence of fetal and infant tactile-kinesthetic sensory intermodality suggests a very early development of body self.

A few months after birth, the baby develops first a consciousness of the other through imediate spontaneous imitation providing a shared bodily sensori-motor experience (Nadel, 2004; Nadel-Brulfert and Baudonnière, 1982) with other-image recognition in the mirror around 8 months of age. Then, the baby develops self-consciousness with self-image recognition in the mirror around 2 years of age and spatio-temporal permanence of self (identification of a permanent self invariant overtime and location in pictures and movies) between 2.5 and 3 years of age (Zazzo, 1948). For Henri Wallon (1959), it is through the consciousness of the other that the concept of body self develops; the young child, in interaction with his or her environment, gradually becomes aware of his or her own body. The American social-psychologist Charles Cooley (1902) explains that the self is built like a mirror and changes with each interaction with the "other ". The other, like a mirror, is the reflection of the infant. This is the "self-mirror" theory (looking-glass self). According to Winnicott (1975), it is the link to the mother which plays very early the role of a "first mirror" for the infant. Winnicott (1975) writes: "in the emotional development of the individual, the forerunner of the mirror, is the face of the mother". Also, Jean-Paul Sartre (1946) describes well the importance of the other in the construction of self: "The other is essential to my existence, as well as to the knowledge that I have of myself ".

Self-consciousnessness is therefore a skill that develops gradually in the human being. It goes through the distinction and differentiation between self and others. It is through the body, interface between self and others, that this distinction will develop. From fetal life, the body is the seat of the first perceptive experiences of the baby and perception-action coupling allows the acquisition of a body schema. At birth, the physical and emotional interactions participate to the development of self-consciousness. The foundation of mental representations of ourselves will be built from the mental representations of others. Studies have also highlighted later in the development the role of intermodal sensory perception, such as visual-tactile perception, in bodily self-consciousness, including self-image recognition, and self-other differentiation. Synchronous tactile stimulation when observing another person receiving the same facial touch stimulation leads to a change in self-image recognition with confusion with others. This has also been experimented with a tactile stimulus in the back. These experiments can induce a perception of self as localized outside of one's own body (Lenggenhager et al., 2007).

Self-consciousness disorders are related to various perturbations of the pathways implied in self-consciousness. Disturbances in the recognition of self-image are common in certain neurodevelopmental disorders such as schizophrenia or autism (Blanke, 2007), and will be described later in this article. In schizophrenia, self-consciousness disorders are also expressed through impairments in self-other differentiation. Evidence of self-consciousness disorders appears more complex in autism than in schizophrenia, due particularly to autistic language impairments, but similarities exist and will be also described and discussed in this article. First, the relationships between schizophrenia and autism are explored with regard to social communication impairments. Then, self-consciousness disorders in schizophrenia and Autism Spectrum Disorder are described and discussed in relation with

body-self impairments leading to self-other differentiation impairments, a deficit of theory of mind and empathy, and their consequences on social communication. Also, neurological dysfunction possibly involved in self-consciousness disorders are presented. Finally, new perspectives are proposed to assess more objectively self-consciousness impairments in schizophrenia and Autism Spectrum Disorder based on self- and other-recognition in the mirror and to develop therapeutic strategies.

2. Relationships between schizophrenia and autism regarding social communication impairments

Autism and schizophrenia (more precisely, the negative syndrome of schizophrenia as observed often in early-onset schizophrenia), far from being diametrical disorders of the social brain, share common psychopathological dimensions in terms of social communication deficits. Since the early 1970s, autism and schizophrenia have belonged to two different diagnostic categories. However, they share some common features: they are both considered as neurodevelopmental disorders involving psychiatric symptoms with impairments in the same main behavioral domains (especially in communication and social interactions). Communication impairments are reported in autism as well as in early-onset schizophrenia (Alaghband-Rad et al., 1995; Asarnow et al., 1994; Baum and Walker, 1995; Cantor et al., 1982). Thus, impairments in verbal communication (delay in the development of spoken language, poor or disorganized speech) and non-verbal communication (reduced facial expression or body language, poor eye contact, and abnormal emotional expression such as flat, bizarre, or inappropriate affects) are found in individuals with early-onset schizophrenia or autism. In addition, social development is impaired in both schizophrenia and Autism Spectrum Disorder. Several authors consider nowadays schizophrenia and autism as neurodevelopmental disorders, and more specifically as social developmental disorders (Howes and Murray, 2014). The degradation of social skills is a hallmark of schizophrenia (Mueser et al., 1991) and the distinguishing feature of subgroups within schizophrenia (Carpenter et al., 1988). Deterioration of social skills is associated with the chronic phase of schizophrenia and its deficit form (Carpenter et al., 1988) or negative syndrome. Social isolation observed in schizophrenia, especially in the childhood of individuals with early-onset schizophrenia, is similar to the autistic withdrawal described by Kanner (1943). Tantam (1988) even argued that individuals with autism display symptoms that can also be considered as schizoid personality traits. In addition, Konstantareas and Hewitt (2001) reported that none of 14 men with paranoid schizophrenia met criteria for autism, whereas 7 out of 14 males with autism met criteria for schizophrenia on the Structured Clinical Interview (SCID). More precisely, symptom overlap concerned negative symptoms of schizophrenia, such as affective flattening, alogia, avolition, apathy, anhedonia, and poor communication. This symptom overlap can be observed particularly between high functioning autism and early-onset schizophrenia which tends to be characterized by negative symptoms with social interaction impairments (Bailer et al., 1996) and a chronic course (Krauss et al., 2000). Furthermore, retrospective studies conducted on patients with schizophrenia or longitudinal studies of children with autism reported frequent associations between these two disorders (antecedents of autism in the childhood of the schizophrenia group and appearance of schizophrenia in the autism group) (Alaghband-Rad et al., 1995; Bender and Faetra, 1972; Jansen et al., 2000; Petty et al., 1984; Van Engeland and Van Der Gaag, 1994).

From a biological point of view, abnormal stress responses (in particular hyper-reactivity of the hypothalamo-pituitary-adrenal axis to psychosocial stress with high cortisol levels — cortisol is a stress and arousal neurohormone involved in the regulation of emotions) were reported in individuals with autism (for a review, see Tordjman et al., 1997, 2014, 2018), schizophrenia and high risk individuals for schizophrenia (Coulon et al., 2016; Jansen, 1998; Jansen et al., 2000). This common biological feature is associated in both disorders with major anxiety focused often on body image and difficulties to adapt to novel and/or stressful situations (Jacobson and Ackerman, 1990; Tordjman et al., 1997, 2014, 2018; Van Den Bosch et al., 1992; Wiedl, 1992). Concerning body image, some studies suggest that the apparent pain analgesia observed in both autism and schizophrenia is more related to a different mode of pain expression due to social communication impairments and body image problems, than to a real endogenous analgesia (Guieu et al., 1994; Tordjman et al., 1999, 2009, 2018; Watson et al., 1979). Body image problems observed in schizophrenia and autism are described later in this article with more details in the section on body self and body image impairments. Also, altered circadian patterns of cortisol have been reported in both schizophrenia and autism suggesting abnormal circadian rhythms in these disorders (for a literature review in schizophrenia and autism, see Coulon et al., 2016, and Tordjman et al., 2014). It is noteworthy that in the context of stable circadian rhythms, fluctuations in the physiological rhythms (variants) occurring in a background of regular repetition of identical sequences (invariants), may allow the individual to develop and maintain throughout the lifespan (from the foetal period to old age) the ability to adapt to change in a secure internal environment characterized by high regularity; it is through the regular repetition of identical sequences of discontinuity provided by circadian rhythms, that a continuum is constructed associated with the development of adaptation to changes (Tordjman et al., 2015). Therefore, abnormal circadian rhythms observed in schizophrenia and autism may prevent the establishment of a secure internal environment contributing to stress vulnerability with difficulties to adapt to changes in these disorders. Sameness, a major autistic symptom described initially by Kanner (1943) which is currently part of the DSM-5 criteria of ASD (2013), can be related to these difficulties to adapt to changes. Furthermore, abnormal secretion and circadian rhythm of melatonin were also reported in schizophrenia and autism (Tordjman et al., 2017). Melatonin is a neurohormone involved in sleep-wake rhythms but also in synchronization of peripheral oscillations (peripheral clocks). This neurohormone plays a major role in the adaptation of the individual to his/her external environment (such as day-night alternance) and internal environment (internal homeostasis) through synchronization of physiological rhythms. The ability to be rhythmically synchronized with the environment appears important for infant development in the sensorimotor, emotional, cognitive, and social realms (Feldman, 2007a; Trevarthen and Aitken, 2001). Numerous studies have been emphasizing the importance of parent-infant synchrony and the construction of shared timing in social communication development (Feldman, 2007b). More generally, the importance of rhythmicity and synchrony of emotional, motor, behavioral and relational (interpersonal) rhythms in social communication was highlighted (Tordjman et al., 2015). For example, synchronization of neural rhythms recorded by EEG was found between distant brain regions of interacting individuals through a free imitation task (Dumas et al., 2010). Interestingly, previous studies reported disorganized emotional and relational rhythms with poor synchrony in children with autism (Trevarthen and Stuart Daniel, 2001). These results taken together suggest that impaired circadian rhythms with the absence of synchronization of the circadian clock network might alter the synchrony of emotional and interpersonal rhythms leading to social communication impairments and vulnerability to psychiatric disorders with social communication deficit such as ASD or schizophrenia. Autism and schizophrenia provide challenging models of physiological, behavioral and relational rhythm disturbances with possible effects on social communication impairments.

Finally, genetic factors possibly involved in both early-onset schizophrenia and autism spectrum disorder have been discussed by several authors (see Yan et al., 2000; Jones and Mormede, 2002, and Table 2 in Tordjman et al., 2018). The serotonin transporter gene (HTT) is of particular interest given the reported association of HTT promoter polymorphism with anxiety-related traits in the general population (Lesch et al., 1996). Furthermore, in a genetic study conducted on

families of 71 children with autism, we showed that the HTT promoter polymorphism modifies the behavioral phenotypic expression of autism: the data consistently pointed to the short(s) allele being significantly associated with more severe impairments in the communication and social interaction domains (Tordjman et al., 2001). These data were confirmed by another study (Brune et al., 2006). Of special relevance are reports that the HTT promoter polymorphism also influences the severity of hallucinations in schizophrenia: Intensity of hallucinations was positively associated with the long (l) allele (Malhotra et al., 1998) and negatively associated with the short (*s*) allele (Golimbet et al., 2003). It suggests that the HTT promoter polymorphism influences the phenotypic expression of positive symptoms (hallucinations) and negative symptoms (social withdrawal and communication deficits). In this regard, our results fit within the following hypothesis previously developed (Tordjman, 2008): autism and the deficit form of schizophrenia would be at one extreme (negative symptoms), and paranoid schizophrenia at the other extreme (positive symptoms) of this continuum. It opens the path to future common research on the genetics of autism and schizophrenia and underlines the importance of using a methodology in which genetic transmission is studied in concert with a detailed examination of clinical phenotype. Future research on the genetics of autism and schizophrenia should develop multi-trait models (Tordjman et al., 2007) and a multidimensional approach, such as studying social communication impairments, instead of focusing on diagnostic categories.

3. Schizophrenia and autism as self-consciousness disorders

In their literature review, Brent et al. (2013) suggested that the onset of self-consciousness disorders in childhood may be part of premorbid signs of schizophrenia, reflecting some of the neurodevelopmental disturbances involved in schizophrenia. In other words, these self- consciousness disorders would be present before the appearance of prodromal symptoms of schizophrenia that could be expressed many years later. This hypothesis of an atypical neurodevelopmental continuum in schizophrenia evades possible childhood psychiatric disorders whose manifestation would also be this self- consciousness disorder. In line with this hypothesis, it would be of interest to conduct a study on individuals with catatonia, characterized by a very early onset of schizophrenia but also severe social communication impairment and longer episodes of schizophrenia (Bonnot et al., 2008), to verify the presence of self-consciousness disorders in this population. It is noteworthy that clinical observations (Tordjman and Maillhes, 2009) suggest body self impairments reflected by severe problems of coordination and prolonged primary enuresis in children with very early onset of schizophrenia (onset before the age of 13). However, further research is necessary to explore rigorously self-consciousness disorers in very early onset schizophrenia and early onset schizophrenia (onset before the age of 18). Knowing that self-consciousness disorders might be part of premorbid signs of schizophrenia, their early detection might help to identify and treat earlier schizophrenia and improve therefore, according to Zaytseva et al. (2013), schizophrenia prognosis. The existence of such early impairments can be observed in both schizophrenia and Autism Spectrum Disorder.

Self-consciousness disorders in schizophrenia refer particularly to first rank symptoms described by Schneider and Demers (1955). These Schneiderian first rank symptoms cover various symptoms that come to mark a rupture of the boundary between oneself and others. Through these symptoms, patients with schizophrenia assign some of their actions and thoughts to others. It includes both verbal intrapsychic or auditive hallucinations (rather described as a common internal language that the individual attributes mistakenly to another), imposed thoughts phenomena, flight of thought, forced desires and acts (as in influence syndrome), as well as some delusional perceptions. Schneider pointed these positive symptoms as mandatory for the diagnosis of schizophrenic illness. Tandon and Greden (1987) evaluated that 60% of patients with schizophrenia present such symptoms but the frequency of Schneiderian first rank symptoms can be variable. In early-onset schizophrenia, some of the first rank symptoms described above may be observed, particularly auditory hallucinations (Bailly et al., 2003) and dysmorphophobia (Tordjman and Maillhes, 2009). However, delusional or dissociative symptoms are less frequent in children than negative symptoms. Indeed, the preferential expression of behavioral impairments in early-onset schizophrenia is the "autistic" social withdrawal that questions again the relationship between schizophrenia and autism (Sporn et al., 2004).

In a more global approach of schizophrenia, references to alterations of the sense of self are present in seminal texts (Bleuler, 1993; Jaspers, 1933; Kraepelin, 1970) and phenomenological descriptions of schizophrenia (Blankenburg, 1971; Tatossian, 1976; Minkowski, 2002). Several studies suggest the existence of impairments in the development of body self, and in particular problems in the most elementary component of the self, the minimal self (Gallagher, 2000; Hur et al., 2014). Body-self impairments are detailed in the section below. Also, their consequences on self-other differentiation, and therefore on social development with a deficit of theory of mind and empathy are discussed in the following sections.

3.1. Body self and body image impairments in schizophrenia and autism

The results of empirical studies using semi-structured interviews such as the phenomenological Examination of Anomalous Self-Experience (EASE) (Parnas et al., 2005), suggest that impairments in the minimal self, described below, are main features of schizophrenia. Indeed, impairments in the minimal self have been observed in the prodromal phase of schizophrenia as well as early and chronic states of schizophrenia (Molloy et al., 2003; Raballo et al., 2011; Parnas and Henriksen, 2014), and may differentiate schizophrenia from other mental disorders (such as bipolar disorder). Impairments in the minimal self can be expressed through alterations in the flow of consciousness, alterations of the presence, and alterations of body experience. Alterations in the flow of consciousness are characterized by an alteration of the sense of "mineness" of mental contents, i.e., by a sense of distance between the self and the mental content (mental content becomes virtually autonomous). Thoughts can then appear as coming from nowhere or can be experienced as spatially localized in a specific part of the body and thus be experienced as "things" that are different from the individual. Alterations of the presence correspond to the feeling of a default of immersion in the first-person experience and a mitigated feeling of being the subject of his/her own experience. One of its most common features is the sense that the self no longer "saturates" nor "lives" completely in the perception of the subject, which would explain the experience of a "mismatch" between the self and the perception of the external world (“*my vision will always appear with a few seconds of difference with me*” said one of our patients). Alterations of body experience are characterized by an experience of embodiment default. The subject can then experience him/herself as an abstract entity contemplating his/her own life from the outside, i.e., in a third person perspective, or even a view from nowhere. This experience is characterized by a radical dualism between the "thinking subject" and the "object-body" (the “Körper” in German, i.e., the body as an object external to the self) — a dualism between a pure conscience and a pure body/bodily materiality that constitutes the central sign of experienced self-disembodiment. Investment difficulties of the body (negligence) and body control (catatonic or agitated behavior) are also common in schizophrenia and might be related to disorders concerning the course of thought (Bouvet, 2005). All these alterations suggest a weakening of the limits of the self with blurred boundaries between the self and other. The subject is no longer able to distinguish between what is his/her own experience and what belongs to the experience of others (Parnas et al., 2005).

Body-self impairments are also expressed through abnormal

recognition of self-image in its physical dimension. The existence of altered self-image and perception of the body itself has been recognized in schizophrenia since several decades (Chapman et al., 1978; Kokonis, 1972). In their typical presentation, these abnormalities are observed in schizophrenia during partial cenesthetic hallucinations (aberrant bodily sensations) or dissociative symptoms with a feeling of depersonalization. Abely (1930) studied self-image recognition in schizophrenia. He described "a requirement of certain subjects to discuss at length and frequently in front of a reflective surface" (p.677). He reports here a phenomenon that has been observed at the dawn of the appearance of early dementia in some of his patients. In his descriptions, the fixing of the mirror can be accompanied by a search dialogue with the reflection that is considered as another. François Achille Delmas (1929) published similar observations. These two authors, almost simultaneously but separately, referred to the same concept: the "sign of the mirror". This sign would be for them a clinical marker of psychotic disorganization. They consider the sign of the mirror, more than a fascination for his/her reflection, a search of his/her own image in the mirror — image that disintegrates more and more durably with the development of psychotic disorders. It is noteworthy that the sign of the mirror would be more related to the prodromal phase of disorders and would disappear after diseases are established. Later, other authors have also studied self-image recognition through the mirror in schizophrenia. It has been observed in schizophrenia that confrontation of patients with their own image in the mirror can produce the feelings that their reflection is distorted, independent of themselves or sinister, but also reactions (such as strange verbalisations, smiles) when facing in the mirror someone other than oneself (Harrington et al., 1989). In addition, an association has been found between the difficulties of recognizing one's own image and the severity of schizotypic disorder or disorganization (Laroi et al., 2007). Some studies have shown abnormalities in self-image recognition through the use of photographs (Kircher and David, 2003), but other studies reported preserved self-image recognition compared to healthy controls (Bortolon et al., 2016).

Concerning childhood disorders, Shentoub was one of the first theorists to study self-recognition in children with developmental disabilities, including intellectual disabilities (Shentoub et al., 1955). He observed the reaction of intellectually disabled children blocked in front of the mirror compared to typically developing controls. The reactions described were various, ranging from virtual absence of recognition of self-image to complex emotional events, and from interactions with reflection to stereotyped behaviors. Among the most important finding was the apparent lack of self-recognition which also questions a possible lack of recognition of the other, and more generally a disturbance of self-consciousness. These blocked-in-front-of-the-mirror children did not present a psychotic disorder marked by internal disorganization or a break with reality. Shentoub ended his studies on a positive observation showing that repeated experiences in front of the mirror with the same child allows him/her to become familiar with others and with his/her own image, which is associated with an overall positive evolution of behavior. This raises the question of potential remediation work which could occur through the mirror image.

In Autism Spectrum Disorder, body image impairments have been also described. Geneviève Haag et al. (2005, 2010) reported impairments in cross-modal sensory integration and sensorimotor integration, leading to instability of body image and developmental problems of body-self in children with autism. These observations are supported by early motor defects found in autism (Sutera et al., 2007). Also, autistic body impairments, such as delayed postural control described in autism (Molloy et al., 2003; Minshew et al., 2004), increase when several sensory inputs are recruited, confirming the lack of sensorimotor integration as a core feature in autism (Minshew et al., 2004). Furthermore, body-related anxieties have been observed in children with autism (anxieties of falling or being liquified, first described by Winnicott in, 1958 and applied after to autism by Tustin in, 1981 and 1986), through the expression of fear of slopes (anxieties of falling) and fear of water draining from the sink or toilet flushing (anxieties of being liquefied). These anxieties can be also reflected by the need to control them through hypertonicity of the body and stereotyped behaviors such as running water on the body incessantly or dropping the same object and picking it up repeatedly (Haag et al., 2010). According to Ayres (1980), auditory, vestibular, proprioceptive, tactile and visual senses are progressively integrated as a body percept in which are rooted different functions such as the coordination of the two sides of the body, motor planning, activity level, attention span and emotional stability. Considering the sensory processing impairments observed in autism, sensory integration dysfunction, as described by Ayres, can be viewed as a core deficit on which treatment interventions should focus (Haag et al., 2010). Autistic impairments in body image and development of body-self, which require cross-modal sensory integration through emotional communication with motor representations, would lead to problems of spatial-temporal organization and self-others representation in autism (Haag et al., 2010). Taken together, neuroscience studies (cognitive neuroscience but also neurophysiology and neuroendocrinology) and the psychodynamic experience of children with autism emphasize the interrelated development of sensory integration and social communication in which emotions play an important role by creating bodily sensations and reinforcing social cognition. Finally, Tordjman and Maillhes (2009) proposed the hypothesis that developmental disorders in body image, already present since early childhood, may be a shared dimension between schizophrenia and autism relevant to a possible problem in the development of bodily self-consciousness leading to problems of self-nonself differentiation, and consequently problems of social communication development. These problems would be expressed very early in some cases such as autism, very early-onset schizophrenia or early-onset schizophrenia (when psychomotor and sensory development is highly altered) and at puberty in other cases such as late-onset schizophrenia (when psychomotor and sensory problems are less severe). The physical changes of puberty occurring in an already vulnerable individual may add indeed to the pre-existing difficulties in body image construction and bodily self-consciousness development, and therefore be a decompensatory factor participating to the onset of schizophrenia at adolescence.

3.2. *Impaired self-other differentiation: a shared dimension in schizophrenia and autism*

Many authors report major impairments in the ability of self-other differentiation in individuals with schizophrenia (Moe and Docherty, 2014). Disorders of attribution and agency (agency is the perception that one is the initiator of his/her own actions; Spence et al., 1997) have been often studied in schizophrenia through various behavioral experimental paradigms. For example, Daprati et al. (1997) developed the paradigm where patients with schizophrenia were supposed to differentiate between their own hands and those of an examiner they saw through a television screen with conflicting visual-tactile information. In this study, attribution errors were always in the direction of excessive appropriation of the other's hand. We saw in the introduction the importance of intermodal sensory perception in typical development, such as visual-tactile integration as shown by Lenggenhager et al. (2007), for bodily self-consciousness, including self-image recognition, but also for self-other differentiation. The effect of intermodal sensory stimulation on self-image recognition and self-other differentiation has been reported in schizophrenia to be either absent (Keromnes et al., 2019) or impaired (Daprati et al., 2007). Furthermore, the Daprati et al. study (1997), based on the video presentation of the patient's hand or the experimenter's hand, showed more pronounced attribution errors in self-recognition for patients with schizophrenia compared to control individuals without schizophrenia. Individuals with schizophrenia made more mistakes than controls by pointing more often to the examiner's hand as their own hand (excess of attribution). Also, when two hands were presented, the patients made

more errors by default attribution. These errors were associated with the presence of main productive symptoms of schizophrenia (hallucinations, etc.). Taken together, the results of several studies suggest that self-image recognition, but also body ownership and agency, might be impaired in schizophrenia (Niznikiewiczj et al., 2000; Bortolon et al., 2017); these impairments appear to be associated with productive symptoms (Jeannerod, 2003). Brébion et al. (2000) have shown similar results. This approach echoes Janet (1937) who described in patients with schizophrenia frequent self-other disorders, either by excess or by default. In this model, each individual would have in him/herself a representation of his/her actions and thoughts and a representation of the actions and thoughts of the other. Agency errors observed in patients with schizophrenia (who mistakenly ascribe the thoughts of others, for example) are the result of an imbalance between these two representations.

The sense of agency corresponds to the ability to identify that one is the cause of an action or thought (Gallagher, 2000) and to distinguish the consequences of actions caused by oneself from those caused by actions of others (Balconi, 2010). Thus, the sense of agency makes it possible to distinguish actions and their consequences in the environment that are self-generated from those generated by others.

The meta-analysis of Hur et al. (2014) highlighted the existence in schizophrenia of a global disruption of self-consciousness. The Hur et al. meta-analysis suggested more particularly altered bodily self-consciousness and frequent agency impairments with increased rather than decreased self-consciousness. However, this self-focused functioning might be related to defence mechanisms in individuals with schizophrenia to cope with decreased self-consciousness (in fact, the opposite of increased self-consciousness) with fragilization and even fear of disappearance of the self, especially the body-self.

In autism, body image disorders found in early childhood may be related to problems in the development of body self, which would hinder the self / non-self differentiation (Meltzoff, 2007; Meltzoff et al., 2017; Tordjman and Maillhes, 2009). As we have just seen, the disturbances of the sense of agency were first studied in schizophrenia where they make it possible to explain positive symptoms such as reference illusions, control illusions and acoustico-verbal hallucinations (Frith et al., 2000; Synofzik and Voss, 2010). According to the model of Synofzik and Voss (2010), the distinction between self-generated actions from those generated by others may be implicit or explicit. The meaning of implicit agency is limited to an elementary representation of the action as being caused or not by oneself. Whereas the meaning of explicit agency is a conscious causal attribution of an action to a particular agent, implying an implicit level and clues (contextual information, prior beliefs, etc.). In autism, regarding the sense of implicit agency, the intentional coupling was intact for the auditory modality and the multimodal condition, but not for the visual modality (Sperduti et al., 2014), which suggests an alteration of sensorimotor indices with visual contents. Cascio et al. (2012) proposed the paradigm of the rubber hand applied to children with and without autism. In this task, the examiner brushes, simultaneously or not, the child's left hand (hidden) and a left (visible) rubber left hand. The coupling of visual-tactile perceptions creates a proprioceptive illusion giving the subject the impression that the rubber hand is his/her own. In children with autism, this illusion appears later, which shows that they take less into account visual information and they rely more on proprioceptive information. Moreover, they are less sensitive to the dyssynchrony of visual-tactile information (simultaneous or alternating brushing), which illustrates that the temporal processing of the sensory information is disturbed. Concerning the explicit agency, it seems to be preserved in individuals with autism (Hill and Russell, 2002; Williams and Happé, 2009; Grainger et al., 2014; Zalla et al., 2010), although some authors find discrepant results (Russell and Jarrold, 1999).

The sensitivity to one's own agency, that appears between 9 and 18 months (Johnson, 2003), makes it possible to establish a clear distinction between oneself and the other and could thus be a prerequisite for the attribution of intentions and other mental states and imitation (Pacherie, 1997; Nadel, 2004). In autism, the presence of repetitive behaviors and deficits in postural control, perception of movement, motion sequence planning, praxis execution may reflect the alteration of agency (Von Hofsten and Rosander, 2012; Mostofsky and Ewen, 2011). As social skills require both engaging in a series of movements and understanding the movements of others, as well as the intentions associated with them, an alteration of the mechanisms of control and awareness of action may limit social interactions and impair communication (Beilin and Fireman, 1999).

Alteration of control and awareness of action in individuals with autism may thus reveal impairments in self-other differentiation and limit social interactions, as any social interaction requires the dynamic treatment of one's own actions and those of others.

3.3. Social withdrawal as a typical manifestation of social cognition disorders

Problems of self-other differentiation, as described above, impair social development and may contribute to the social withdrawal observed in schizophrenia and autism. In this context, social withdrawal can be considered as a defence mechanism resulting from difficulties to interact with the social environment due to impaired self-other differentiation. It is noteworthy that, as indicated in the introduction, social withdrawal was one of the main behavior reported by Kanner (1943) to characterize autism, and autism was introduced by Bleuler (1993) to describe precisely social withdrawal in schizophrenia.

In a more general approach of self-disorders in schizophrenia, Franck (2010) described disorders of the *lived experience* in schizophrenia, which include both self-consciousness disorders and social cognition disorders (mainly in the form of social withdrawal and social interaction impairments). In this perspective, the *lived experience* disturbances are present in the main dimensions of schizophrenia as well as autism, which can question the relevance of a categorical approach in favour of a more dimensional one applied to a transnosographic approach of self-consciousness disorders and social cognition disorders in schizophrenia and autism.

Social cognition disorders encompass the resulting symptoms of a disruption of cognitive processes underlying the relationship with others. Their most typical manifestation is social withdrawal. Today, to describe social withdrawal observed in patients with schizophrenia, the term “autistic” withdrawal is often used. This self-centered tendency is not limited to schizophrenia and autism but is exacerbated there. For Wallon (1959), children in their typical development go through an “autistic” phase before achieving initial consciousness of the other. Social cognition disorders observed in schizophrenia and autism could then reflect a failure of the construction of the self in childhood, including the early development of body self, and be the expression of self-consciousness disorders. Furthermore, it is noteworthy that social withdrawal is part of a broader disturbance of the self-consciousness process, including a deficit in theory of mind and empathy. These problems represent a major dimension of schizophrenia and autism and are developed in the next section.

3.4. Deficit of theory of mind and empathy in schizophrenia and autism: towards a better understanding of social communication impairments in these disorders

3.4.1. Definitions of the concept of empathy and theory of mind

According to Langdon et al. (2006), empathy is a complex mental process that can be defined as the ability to put oneself into the psychological frame of reference of others, in order to understand what they think, feel and act based on sensory, motor and emotional experience. Empathy involves the development of self-other differentiation. It includes main cognitive and affective components (Benedetti et al., 2009). From a cognitive perspective, theory of mind (ToM) is one

Table 1
Historical conceptualizations of affective empathy (Emp) and theory of mind (ToM).

Author	Conceptualization of empathy and theory of mind
Hoffman (1987)	Someone's emotional response to other's affective states (Emp)
Fesbach (1987)	Three processes: identify other's affective signals, infer perspectives and role of others, experience and express emotions (Emp)
Lee et al. (2004)	To be inside and experience other people's feelings (Emp)
Premack and Woodruff (1978)	Capacity to represent mental states and to make inferences regarding other people's mental states to predict their behavior (ToM)
Benedetti et al. (2009)	Put oneself into the psychological frame of reference of others (ToM)

of these components and can be defined as the capacity to represent mental states and to make inferences regarding other people's mental states like their knowledge, needs and intentions, in order to understand and predict their behavior (Premack and Woodruff, 1978). From an affective perspective, empathy (affective empathy) is, according to Lawrence et al. (2004), the capacity to understand and deduce people's emotional experiences by showing an appropriate emotional response towards them. Langdon et al. (2006) described different conceptions of affective empathy outlining principally the Hoffman (1987); Fesbach (1987), and Lee et al. (2004) work presented in Table 1 along with some other authors' studies on ToM conceptualizations. In summary, cognitive empathy can be brought closer to the concept of ToM and defined as the capacity to understand other people's emotions, whereas affective empathy can be defined as the capacity to experience other people's emotions with emotional responses. Both, empathy and ToM are substantial competencies closely linked and depending on the process of self-consciousness. Preston and de Waal (2002) indicate that still today, concepts and definitions related to the empathetic processes remain confusing when trying to establish distinctions between empathic responsiveness, emotional contagion, sympathy, compassion, perspective-taking and prosocial-behavior.

3.4.2. Deficit of theory of mind and empathy in schizophrenia and autism

Both schizophrenia and autism present ToM impairments (Langdon et al., 2006). As highlighted above, both schizophrenia and autism can be understood as social disorders related to self-consciousness disoders. This relationship has a direct impact on ToM and empathic abilities, considering that both "affective" empathy and "cognitive" empathy are social abilities related to self-consciousness.

Some authors suggested that the development of ToM is impaired in schizophrenia (Shamay-Tsoory et al., 2007; Lee et al., 2004). They mentioned also that individuals with schizophrenia show a lack of success when trying to understand other people's mental states. Furthermore, other authors reported that patients with schizophrenia show an important deficit in the following abilities: a) perspective taking ability which would have a direct impact on the recognition of other people's beliefs, percepts, and emotions; b) ToM ability; c) executive function ability, and d) other general cognitive impairments (Benedetti et al., 2009; Langdon et al., 2006; Brüne, 2005). A meta-analysis conducted by Sprong et al. (2007) showed that on average, ToM performance in patients with schizophrenia was more than one standard deviation below ToM performance in healthy participants.

Also, studies suggested that cognitive empathy (in terms of understanding others' feelings, beliefs and intentions) is more impaired in individuals with autism than affective empathy (in terms of experiencing others' emotions) (Baron-Cohen, 1995). However, it is noteworthy that Langdon et al. (2006) considered it difficult to compare schizophrenia and autism in terms of cognitive impairments to understand mental states because patients with schizophrenia who show impairments in ToM attribute wrong mental states to other people, whereas autistic impairments in ToM are more related to a "lack of theory of the representational nature of mind" (p. 138). The same authors reported that in terms of task performance and clinical phenomenology, individuals with autism show *intact visual perspective-taking*, whereas patients with schizophrenia show *impaired visual perspective-taking* (Langdon et al., 2006).

3.4.3. Consequences of a deficit of empathy and theory of mind: social communication impairments

Several studies suggested that empathy and ToM deficits contribute to poor social interactions displayed by individuals with schizophrenia (Morrison et al., 1988; Bell et al., 1997; Penn et al., 1996; Roncone et al., 2002; Brüne, 2005; Shamay-Tsoory et al., 2007). Similarly, Spain et al. (2017) reported that individuals with autism showed social impairments such as a difficulty to initiate and sustain reciprocal social interactions co-occuring with deficits in ToM (Baron-Cohen et al., 2001 quoted by Spain et al., 2017). Furthermore, the deficit of empathy and ToM impairs also the communication domain given that the capacity to represent other people's mental states (ToM) as well as the capacity to understand and experience other people's emotions (empathy) are necessary to communicate with others. It is noteworthy that nonverbal communication involves emotional gestures requiring to be at least understood in order to engage a reciprocal communication. It appears difficult to separate completely the behavioral domain of social interactions from the one of communication, to the point that the DSM-5 criteria for Autism Spectrm Disorder have combined the social and communication domains into a single total domain. The issue of social communication impairments is important as patients presenting these impairments show frequently other clinical issues, such as social anxiety which could benefit from social group interventions (Spain et al., 2017).

4. Neurological dysfunction and self-consciousness disturbance in schizophrenia and autism

Taken together the points developed previously, it appears that schizophrenia and autism are characterized by self-consciousness disturbance with impairments in sensory integration associated with body-self disorder and impairments in psychic and bodily boundaries between the self and the other involving particularly a deficit of theory of mind, empathy and sense of agency. According to Tononi and Edelman (2000), self-consciousness is based on a global activation of the central and peripheral nervous systems, allowing the establishment of a network permanently connected in which each link is important. Sass (2014) hypothesized that global mechanisms of self-consciousness are impaired in schizophrenia and several authors pointed out a disruption in connectivity between some of these links in schizophrenia (Backasch et al., 2014). This disruption in the chain would prevent a global integration of information and would be the basis of the disintegration observed in patients with schizophrenia but also a wider cognitive disorganization interfering with the relationship between the individual and the other (Tononi and Edelman, 2000). The underlying neurobiological mechanisms are still insufficiently understood. However, some brain areas seem to play a key role and deserve to be mentioned. Indeed, neuroimaging studies have suggested, as developed below, the existence of a network of brain areas involved in making inferences to others' mental states, and therefore related to the theory of mind (such as the mirror neurons, the prefrontal cortex, and the temporo-parietal junction).

4.1. The mirror neuron system

Mirror neurons were described in 1990 by Giacomo Rizzolati

(Rizzolatti and Sinigaglia, 2008). It is a group of motor neurons that activate when we perform an action but also when we see someone else performing the action, and when we think of this action or name it verbally. Their description came initially from observations in monkeys. The brain regions involved in this « mirror » response are the premotor frontal cortex, the upper temporal sulculum and parietal areas. No activation of the visual areas was found (Calvo-Merino et al., 2005). In the great ape, the brain area F5 (Broca's area in man) could play a role in the mirror neuron system showing the implication of language and ability to communicate with others. More generally, mirror neurons are involved in social cognition and empathy. Currently, the importance of the mirror neuron system is more controversial particularly because of its lack of specificity (Hickok, 2014).

4.1.1. Schizophrenia

Mirror neuron dysfunction might explain some symptoms of schizophrenia. In a literature review, Mehta et al. (2014) showed direct relationships between a decreased activity of mirror neurons in patients with schizophrenia and persistent negative symptoms, social cognition deficit and a lack of self-control. The decreased activity of mirror neurons would provoke a reorganization of neuronal tissue, followed by a secondary hyperactivation of mirror neurons, resulting in various symptoms including catatonia, emotional instability and hallucinations.

4.1.2. Autism Spectrum disorder (ASD)

Dysfunction in the mirror neuron system (MNS) has also been described in autism and was discussed with regard to social communication impairments and poor imitative performance resulting especially from imitative control deficits (Hamilton, 2013; Schunke et al., 2016; Hsiang-Yun et al., 2015). Indeed, several studies in ASD individuals, using neural activity recording and neuroimaging techniques, have reported functional deficits in the MNS network, and particularly a weaker coordination of the MNS network associated with alterations in cortical thickness and microstructural integrity of the tracts connecting the regions forming the classical MNS (Chien et al., 2015). In addition, a strong correlation was found between the integrity of the right frontoparietal tracts and the social communication scores. Furthermore, Fishman et al. (2014) observed in ASD adolescents a mixed abnormal pattern of both over- and underconnectivity in the theory of mind (ToM) network associated with social impairments; also, increased connectivity in the ASD group was detected between the MNS and ToM networks and was correlated with social communication impairments. This increased ToM-MNS connectivity may reflect immature or atypical developmental processes in brain networks involved in social cognition, especially in social communication. However, a systematic review of 25 studies related to MNS in autism, conducted by Hamilton (2013), showed discrepant results possibly due to studies using weakly localized measures of the integrity of the MNS, and therefore were difficult to interpret. In contrast, fMRI studies provide well localized measures of MNS activity and showed that group differences were observed following emotional stimuli whereas no group differences were reported following non-emotional hand action stimuli (Hamilton, 2013).

4.2. The prefrontal cortex

The prefrontal cortex, especially the right prefrontal cortex, is involved in many cognitive tasks, including self-recognition. This requires the mobilization of episodic memory, introspection and the theory of mind (Kircher and David, 2003). In particular, a 2010 meta-analysis (Van der Meer et al., 2010) highlighted the importance of the medial prefrontal cortex in the process of self-recognition and the recognition of others. Based on the analysis of 33 studies, they proposed a model where the ventral part of the medial prefrontal cortex would correspond to the localization of the self, whereas the dorsal part would be involved in evaluation and decision allowing self-other differentiation.

4.2.1. Schizophrenia

Some dysfunctions of the PFC (prefrontal cortex) have been demonstrated (Haller et al., 2014). In particular, it has been reported in patients with schizophrenia showing Schneiderian first rank symptoms an increased activity of cortical areas involved in language or action processing. This increased activity would be secondary to a failure of the inhibition process normally exercised by the prefrontal cortex on more posterior areas, such as temporal and parietal lobes as described below, leading to attribution errors in self-recognition.

The mechanism involved might be an inhibition defect in the primary auditory area (temporal lobe) (Frith, 1979). Acoustic-verbal hallucinations may be related to the perception by the patient of his/her own inner dialogue related to an overactivity of the primary auditory area (temporal lobe) due to an inhibition defect (Frith, 1979). The Dierks et al. study (1999), using functional magnetic resonance imaging (fMRI) in patients with schizophrenia during auditory hallucinatory episodes supports this hypothesis. This study showed an increase in metabolic activity in the primary auditory area (Heschl gyrus) of the temporal cortex during hallucinatory phenomena, along with the activation of associative temporal areas involved in language recognition. In healthy individuals, such activation of Heschl gyrus is not observed in inner language, probably due to inhibition exerted by the prefrontal cortex. This result could therefore suggest that patients with schizophrenia treat their inner language as an activity coming from the outside (Dierks et al., 1999). In addition, an increased activity in the lower right parietal area has been reported, and could be explained, according to Daprati et al. (1997), by the role of the parietal lobe in comparing the executed movement and its sensory consequences. The difficulties for the patient to attribute the origin of the actions might be related to this overactivity of the right lower parietal lobe leading to disturbances of the self-nonself differentiation. Also, an anatomical inversion of the two left and right lower parietal areas was reported in patients with schizophrenia (Korkmaz, 2011). Finally, the first levels of visual recognition are altered in schizophrenia by the lack of interaction between magnocellular (parietal lobe) and parvocellular (temporal lobe) visual pathways (Martin et al., 2014). These visual impairments are not specific to the face. They increase when the cognitive and perceptual load is high in individuals with schizophrenia and worsen with chronicity.

4.2.2. Autism spectrum disorder (ASD)

Abnormal functional activation and maturation of ventromedial prefrontal cortex during temporal discounting were reported in ASD (Murphy et al., 2017). Abnormal signal gating to prefrontal cortex has also been reported in ASD with basal ganglia impairments (Chantel et al., 2016; Prat et al., 2016). More precisely, abnormal synchronization between the prefrontal cortex and other cortical processing centres has been observed in ASD individuals. Such a failure to prioritize and filter signals to the prefrontal cortex could result in pervasive impairments in cognitive flexibility and executive functioning that characterize ASD and may offer a mechanistic explanation of some of the observed abnormalities in patterns of cortical synchronization in ASD.

4.3. The temporo-parietal junction

The central role of the temporo- parietal junction in self-consciousness has been reported by several studies (Ionta et al., 2011a; Sugiura et al., 2005). The temporo- parietal junction is a brain area involved in the integration of multimodal sensory information and plays a key role in the first-person perspective, self-nonself differentiation, and certain complex mechanisms of the theory of mind (ToM), especially the capacity to understand the intentions and beliefs of others (Ionta et al., 2011b). It is noteworthy that transcranial direct current stimulation of the temporo-parietal junction in healthy adults improves imitation-inhibition and perspective-taking, i.e. the control of self and other representations involved in the ToM (Nobusako et al., 2017). Interestingly, imitation-inhibition and perspective-taking are

also related to the MNS, which strengthens the hypothesis of ToM-MNS connectivity previously described in this section. Furthermore, lesions of the temporo-parietal junction provoke asomatognosy (impossibility of recognizing certain parts of one's own body). Finally, abnormalities of the temporo-parietal junction have been described as associated with disturbance of self-consciousness in certain neurological impairments, including Out-of-Body Experiences (OBE, i.e. moments outside one's own body), autoscopic hallucinations and heautoscopy (the patient sees suddenly his/her image appearing in front him/herself; one of its variants is negative heautoscopy in which the individual sees no longer his/her own image in a reflective surface) (Blanke et al., 2004). The Blanke studies on OBE showed the activation of the temporo-parietal junction during OBE electroencephalographic monitoring but also following transcranial magnetic stimulation (TMS) of the temporo-parietal junction in healthy individuals (Blanke et al., 2005; Aspell et al., 2012). Abnormalities of the temporo-parietal junction would lead to changes in the perception of body self with impairments in the integration of proprioceptive, tactile and visual information, but also in the integration of visual and vestibular signals (Blanke et al., 2004). This is important with regard to self-consciousness, knowing that bodily self-consciousness is based on proprioceptive, vestibular, tactile and visual information (Thirioux et al., 2016a, b). Complex anomalies involving the temporo-parietal junction have been reported in schizophrenia and autism and are described below.

4.3.1. Schizophrenia

Dysfunctions of the temporo-parietal junction have been reported in schizophrenia (Bodnar et al., 2014; Vercammen et al., 2010). Such abnormalities appear particularly related to auditory hallucinations but are also observed in patients with a deficit syndrome, including persistent negative symptoms with prevalent social withdrawal, and in patients with early-onset schizophrenia. These observations confirm the key role of the temporo-parietal junction in the treatment and integration of sensory information but its dysfunction in schizophrenia appears as a quite unspecific characteristic.

4.3.2. Autism spectrum disorder (ASD)

Several studies suggest a dysfunction in ASD of the temporo-parietal junction associated with impairments in mental and emotional state attribution, and therefore with a deficit of ToM. For example, a fMRI study (Murdaugh et al., 2014) reported in ASD adults reduced brain responses in the right temporo-parietal junction during a task of intentional causal attribution. Another study (Donaldson et al., 2017) reported interactions between autistic symptoms and temporo-parietal junction stimulation effects on social cognition, knowing that the pre-stimulation mental state attribution accuracy was reduced in participants with higher autistic symptoms.

4.4. White matter and synaptic abnormalities

4.4.1. Schizophrenia

Recent studies suggest diffuse abnormalities of white matter (White et al., 2015) and the existence of an exacerbated synaptic elimination in patients with schizophrenia that can either begin de novo in adolescence or occur on already reduced synaptic capital (Hoffman and McGlashan, 1997).

4.4.2. Autism spectrum disorder (ASD)

A meta-analysis of diffusion tensor imaging studies (Xim et al., 2017) showed focal white matter reduction in ASD, in particular in the left splenium of corpus callosum and the right peduncle associated with sensorimotor impairments and another meta-analysis conducted also on diffusion tensor imaging studies reported disrupted focal white matter integrity in ASD (Di et al., 2018). Furthermore, a study using also diffusion tensor imaging in children and adolescents with ASD compared to a matched group of typically developing controls showed significantly reduced hemispheric asymmetry of white matter microstructure in ASD (Carper et al., 2016). No correlations between global white matter asymmetry and social communication impairments were found.

Concerning synaptic abnormalities, many genetic studies in ASD reported mutations of genes encoding for proteins involved in synaptic development and neural communication (synapse formation, adhesion, stabilisation and homeostasis), such as for example neuroligins NLGN3 and NLGN4 (postsynaptic cell adhesion proteins), SHANK3 (a postsynaptic scaffolding protein), cell adhesion molecule-1 (CADM1 driving synapse assembly), or protocadherin10 (synaptic development) (for a review, see Tordjman et al., 2018). It is noteworthy that synaptic elimination described in schizophrenia (Hoffman and McGlashan, 1997) has also been reported in ASD (Tsai et al., 2012). In particular, the Tsai et al. study showed that multiple autism-linked genes mediate synapse elimination via proteasomal degradation of a synaptic scaffold PSD-95.

5. Conclusions: towards an integrated model of schizophrenia and autism as bodily self-consciousness disorders

It is important to understand better the development of Autism Spectrum Disorder and schizophrenia, notably early-onset schizophrenia, in order to identify early symptoms as early as possible, propose adapted therapeutic care, and improve the prognosis of these neurodevelopmental disorders. The study of self-consciousness, in its affective, cognitive and bodily aspects, appears essential in child and adolescent neurodevelopmental disorders given that self- and other-consciousness is still in construction at this period of life (Korkmaz, 2011). The study of impairments in body self and body image might have major implications, especially for an early diagnosis of schizophrenia and autism. However, the limits we are facing are that the identification of such impairments are based on the verbal expression of patients. Available data should be interpreted with caution given that body self and body image are related to non-verbal aspects of self-consciousness. Therefore, the challenge is to develop a method to study objectively body self and body image from a non-verbal approach (Mishara et al., 2014). The development of self-image which is observed from early life might be an interesting indicator to monitor the evolution of the consciousness process (Tordjman and Maillhes, 2009). A new paradigm proposed by Thirioux and collaborators (2016a,b), based on a system of alter ego developed by Moritz Wehrmann, allows self-other differentiation and self-consciousness to be specifically explored through self-image recognition but also through the recognition of the other in the mirror. This paradigm can be used to study disturbances of self-consciousness in schizophrenia and autism as we did in previous research from our team, comparing individuals with schizophrenia (early onset and adult onset schizophrenia) to typically developing controls (Keromnes et al., 2019), but also children with Autism Spectrum Disorder to typically developing controls (article in preparation). The findings in individuals with early-onset schizophrenia (EOS) and adult onset schizophrenia (AOS) compared to typically developing controls (TDC) showed that there was no significant effect of intermodal sensory stimulation on self-other recognition in EOS and AOS patients whereas self-recognition in the mirror was significantly increased by visual-kinesthetic-tactile stimulation in TDC. Similar results were observed in children with severe ASD (Tordjman and Keromnes, personal communication, Sienna, 2016). These results, taken together with prior observations developed in this article, suggest, as shown in Fig. 1, the existence of bodily self-consciousness disorders in schizophrenia and severe ASD associated with altered/absent cross-modal multisensory integration (especially visual-kinesthetic-tactile integration). This would result in problems of self-other differentiation, leading in turn to social communication impairments as well as a deficit of ToM and empathy that will reinforce, as suggested in this article, social communication impairments.

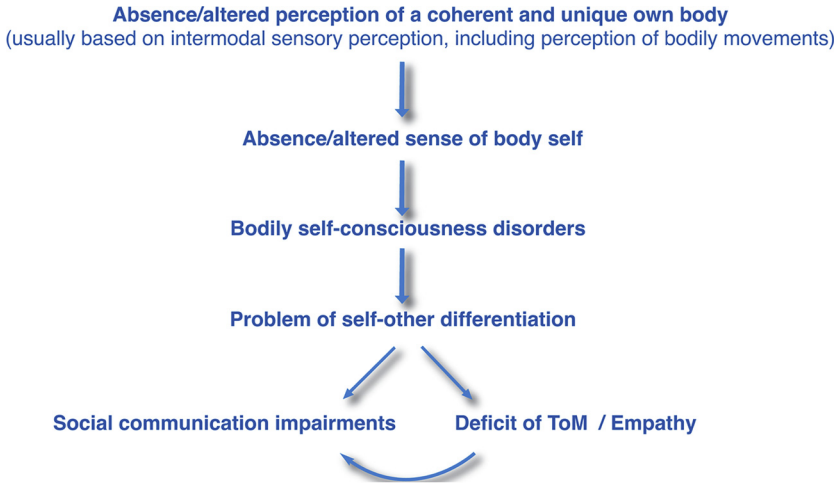

Fig. 1. Legend: Model of schizophrenia and autism as self-consciousness disorders associating a deficit of theory of mind (ToM) and empathy with social communication impairments.

Conflict of interest statement

The authors declare that they have no competing financial interests.

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