Title
A neuroscientific perspective on the nature of altered self-other relationships in schizophrenia

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2014

Abstract
By empirically investigating the neural correlates of the basic experience one makes of oneself as bodily self and of its alterations, new light can be shed on the relationship between self-disturbances and social deficits in schizophrenia. We review recent neuroscientific evidence showing how a pre-reflective, experiential understanding of others can be accomplished, so that others are conceived as bodily selves by means of neural reuse of our own sensorimotor and visceromotor resources, and how a clear distinction between self and other is normally preserved. By conjugating identity and alterity, a putative neural mechanism is provided underpinning the blurring of such distinction in schizophrenic patients. The reviewed empirical data suggest brain function anomalies at the levels of multisensory integration, differential processing of self- and other-related bodily information, and mediating self-experience at the basis of an imbalance in the pre-reflective relationship of the embodied self to the world including the social environment.

Introduction

A new neuroscientific approach to the study of the human mind is gaining momentum. It capitalizes upon the study of the bodily dimension of knowledge: the so-called "embodied cognition" approach. Cognitive neuroscience can today provide new insights on two aspects of social cognition: intersubjectivity and the human self. Such insights also benefit from a parallel "phenomenologization" of cognitive neuroscience whose main goal is the naturalization of intersubjectivity and the self (see Gallese 2007, 2009). This approach attempts at deconstructing the concepts usually employed to describe these aspects of human social cognition by literally investigating what they are made of at the level of description of the brain-body system. Starting from the analysis of subjective experience and of the role that the living body plays in the constitution of our experience of material objects and of other living individuals, the empirical study of the causal determinants of subjectivity and intersubjectivity can be pursued on new bases if compared to those so far adopted by classic cognitivism. The classic cognitivism approach inherited the limitations intrinsic to the Cartesian solipsistic approach to the human mind, an approach that, according to the psychiatrist and philosopher Erwin Straus, separated the mind from the body, the self from the world and the I from the Thou (Straus 1960).

Intersubjectivity requires, on the one hand, shared neural networks grounding an implicit understanding of others' behaviors and mental states. On the other, it requires processes enabling one to maintain a coherent and unique sense of self, allowing for self-other discrimination. Thus, intersubjectivity rests on the possibility to conjugate identity and alterity. In the present paper we focus on the nature of altered self-other relationships in schizophrenia by presenting and discussing recent empirical evidence on the nature of the social self and contrasting it with that obtained from schizophrenic patients. We show that a pre-reflective, experiential understanding of others can be accomplished by means of neural reuse. In other words, we conceive others as bodily selves by mapping others' actions, emotions and sensations onto one's own sensorimotor and visceromotor representations in bodily format of the same actions, emotions and sensations. We show how, in spite of shared representations generating vicarious experiences of others, a clear distinction between self and other is normally preserved. We also show the putative neural mechanisms underpinning the blurring of such distinction in schizophrenic patients. We conclude by discussing these results on the social self within the theoretical perspective looking at schizophrenia as a self-disturbance disorder.

Schizophrenia: self-experience and the social self

Schizophrenia is a pervasive as well as complex neuropsychiatric disorder with prominent social cognition deficits among its key characteristics (Pinkham et al., 2003; Burns, 2006; Derntl et al., 2009). It has been proposed that social impairments in schizophrenia may exist independently of neurocognitive impairments, are related to functional outcome and community functioning, can be detected during the prodromal phase of schizophrenia, and persist when patients are in remission (Couture et al., 2006; Bellack et al., 2007; Addington et al., 2008; Horan et al., 2009; Fett et al., 2011). Nevertheless, the exact nature and the dysfunctional neural mechanisms underlying social deficits in schizophrenia remain a topic of speculation. Although various types of social impairments may be identified in schizophrenia with an origin at different, more specific levels, the present paper will specifically focus on how certain social impairments in schizophrenia can possibly be grounded in self-experience disturbances. In particular, recent evidence from functional neuroimaging studies will be explored explaining how disturbances at the level of experiences and awareness of the bodily self could pervade self-other relationships and alter the experience of social interaction in schizophrenia.

The idea of altered self-other relationships in schizophrenia is not a new one. Already in the early 20th century psychopathology attributed a crucial role to the self and its pre-reflective relationship with the external world in schizophrenia. Eugen Bleuler coined the term "autism" to describe a detachment from the outside world coupled with a predominance of inner life as one of

the defining symptoms of schizophrenia (Bleuler, 1911). Subsequently, Eugéne Minkowski elaborated Bleuler's view, proposing a lack of vital contact with reality ("*perte du contact vital avec la réalité*"), like an inability to resonate with the world and to empathize with others, caused by a subjective deficit in the pre-reflective attunement between individuals and their world, as the core aspect of schizophrenia (i.e., its "*trouble générateur*"; Minkowski, 1927; see also Henriksen et al., 2010).

More recently, it was suggested that social deficits in schizophrenia could have their primary basis in anomalous self-experience and awareness (Haug et al., 2014; Nelson et al., 2009; Sass & Parnas, 2003; Fisher et al., 2008). Finally, reconciling these notions, it was proposed that social dysfunction in schizophrenia could result from a transformation of the structure of subjectivity in all its intertwined dimensions including ipseity, intentionality and intersubjectivity (Parnas et al., 2002). Of particular interest in the context of a subjectivity disorder, an explanation of certain phenomena concerning the social self in schizophrenia in terms of brain function pathology was proposed. Such proposal suggested a possible link between alterations in neural circuits vicariously involved in processing (bodily) self-experience and intentional motor behavior and basic intersubjective experiences, including the empathic sharing of others' actions, emotions and sensations (Gallese, 2003). In other words, disturbances of ipseity, intentionality and intersubjectivity could be directly linked within the embodied cognition approach as based on “shared” neural systems modelling what we as well as others do and feel as intentional experiences (e.g., Gallese, 2006). Before moving to the brain basis of the anomalous social self in schizophrenia, it is useful to briefly discuss the social self in the healthy brain.

The social self in the healthy brain: vicarious neural circuits

Establishing an intersubjective experience when socially interacting with other individuals is a basic aspect of vital social interaction, contributing to the understanding of others as intentional agents, like ourselves. With respect to the vicarious functions of the brain underlying such social phenomena, it has been suggested that multimodal brain regions underlying bodily self-experiences facilitate a pre-reflective understanding of others' experiences and mental states (Gallese, 2003; Gallese et al., 2004; Keysers and Gazzola, 2009).

A key empirical finding concretely subscribing this view is the discovery of mirror neurons in macaque monkeys. Briefly, neurophysiological studies in the early 90's found neurons in premotor area F5 (di Pellegrino et al. 1992; Gallese et al. 1996) and regions of the inferior parietal lobe (Gallese et al. 2002; Fogassi et al., 2005; Bonini et al. 2010) to be activated both when the monkey performed particular goal-related motor acts, like hand and mouth grasping, and when the monkey observed someone else performing the same object-related actions. Mirror neuron mechanisms have been interpreted as reflecting one out of several mechanisms that allow understanding others’ behavior, like motor goals and motor intentions. In particular, they could contribute to social understanding from “the inside” by unifying action perception and action performance within a joint neural substrate (Gallese, 2001; Rizzolatti & Sinigaglia, 2010). Subsequent studies using neuroimaging and neurophysiological techniques suggested the existence of similar neural mechanisms in humans. For instance, they demonstrated that the same, multimodal, neural circuits involved in our own bodily self-experiences are also involved with the pre-reflective understanding of others' actions (Rizzolatti et al., 1996; Caspers et al., 2010) as well as of some other mental states, like emotions and sensations (Keysers and Gazzola, 2009; Molenberghs et al., 2012).

Thus, multimodality probably could apply to motor as well as somatosensory and emotion circuits (Gallese, 2003). For example, when witnessing the affective or somatosensory experiences of other individuals, neural activity is modulated in brain networks also involved in the first-person experience of the same experiences (see for reviews Gallese at al., 2004; Lamm et al., 2011; Decety, 2011; Keysers et al., 2010). Neurological as well as artificial reversible lesions of brain circuits involved with sensory, motor or affective experiences interfere with the understanding of

these experiences in others (see for reviews Bastiaansen et al., 2009; Hillis, 2014). According to the model of embodied simulation, such multimodal sharing mechanisms allow an individual to establish a meaningful, intersubjective link with others by means of reusing one's own mental states or processes in functionally attributing them to others (Gallese, 2001; Gallese & Sinigaglia, 2011; Gallese, 2014).

The social self in the healthy brain: differentiating self from other

Neuroscientific literature largely subscribes the functional relevance as well as the neural feasibility of a pre-reflective, experiential sharing of other individuals' feelings and behaviors. However, a critical and complementary issue that received much less attention is the ability to discriminate to whom "shared" experiences belong during social interaction (e.g., Banissy et al., 2009; Lamm et al., 2011). In other words, functional intersubjective experiences respect a trenchant border between self and other. This implies that empathically shared experiences of others nevertheless are experienced as belonging to the other. Considering psychopathological accounts of schizophrenia, a disorder of self-experience that transpires through its symptomatology (Parnas et al., 2002), like confusion and blurring of self-other relationship, this aspect might be of crucial importance for the study of social interaction in schizophrenia.

From a phenomenological point of view, self-other discrimination has been considered constitutive for empathy. For instance, in her fundamental work "On the problem of empathy", Edith Stein (1989) defined empathy as "*the experience of foreign consciousness in general*" (p. 11), also clearly emphasizing the preservation of otherness in intersubjective experience: "*the subject of empathizing is not the subject empathizing, but another, a foreign mind*" (p.10). According to Stein (1989), unlike our own experiences, which are primordially given, empathy does not have this primordiality. Indeed, as Edmund Husserl put it, "*had one had the same access to the other's consciousness as to one's own, the other would have ceased being another, and would instead have become a part of oneself*" (Husserl, 1973, p. 139, transl. Zahavi, 2010). As further described by Zahavi (2010, 2001), it is because of this asymmetry that the minds we experience are experienced as *other minds*. Other's mental states are usually not experienced in the same way as the other does, but as belonging to another individual that is not us. We experience the other as having experiences similar to ours.

How can this self-other discrimination be reconciled with an empathic sharing of others' states based on vicarious brain activity in sensory-motor and emotion systems and be translated into brain function? Let's examine this issue more in detail by focusing on the domain of touch playing a peculiar role in our interactions with the environment. For example, being the first sense to develop and present all over the body, touch is the most important sense to initially learn about and create bonds with the environment. Furthermore, touch reflects a primary form of socio-emotional interaction and communication, and it supports both external perceptions (e.g., tactile sensation) and internal perceptions (e.g., proprioception) at the basis of the awareness of an elementary relationship between our body and the external world (Gallese and Ebisch, 2013).

Firstly, although sensorimotor cortices activate for both the first-person perception of touch and the observation of the same experiences in others, usually neural activity is much weaker under the latter circumstances. Moreover, Blakemore and colleagues (2005) investigated by means of fMRI brain activation for the observation of touch in a case of vision-touch synaesthesia, a woman in which the observation of another person being touched is experienced as tactile stimulation on the equivalent part of her own body. Her brain activity during the mere vision of touch was compared with a group of healthy participants without this condition that watched the same touch videos. The results showed stronger activation for the vision of touch in the case of vision-touch synaesthesia in somatosensory, premotor and anterior insula cortices, brain regions also responsible for first-person tactile experiences. Therefore, abnormal intensity of neural activity in sensorimotor circuits might contribute to the phenomenon that the other's experience is perceived as ours even though not primordially given.

Secondly, the extent to which neural activity is shared could contribute to the distinction between self and other. Watching others being touched activated only a part of somatosensory cortex that is activated when actually experiencing touch (Keysers et al., 2010). In particular, vicarious activation is commonly detected in the second somatosensory area (SII) as well as in Brodmann areas 1 and 2 of the primary somatosensory area (SI), while BA 3 seems to be confined to the exclusive processing of one's own tactile perceptions. This absence of vicarious activation in certain regions of primary sensory cortex when witnessing others being touched could be co-responsible for the absence of real first-person bodily feelings during social perception.

Thirdly, this issue was addressed more specifically in an fMRI study (Ebisch et al., 2011a). In this study, healthy participants watched videos depicting other individuals being touched on their hands in different ways and, at the end of the experiment, were touched on their hands themselves. Consistent with other studies, overlapping activation for the experience and observation of touch was found in several sensory-motor regions, including SII and premotor cortex. However, posterior insular cortex exhibited differential activation for touch experience and observation. Whereas neural activity in posterior insular cortex increased when participants were touched themselves, the same part of posterior insular cortex behaved in the opposite way by deactivating when participants observed touch in other individuals. This effect specifically occurred when the touch was characterized by an affective valence, like a hit or a caress. In other words, neural activity of posterior insular cortex was suppressed below baseline in the social perception condition. Note that the experimental task required participants neither to actively simulate other's experiences nor to explicitly distinguish between self and other experiences. Thus, maintaining a sense of self that is distinct from the other could concern a naturally occurring, active neural process. Indeed, psychophysical interaction analysis suggested that posterior insular cortex deactivation could depend on a wider functional neural network including right temperoparietal junction, right posterior parietal cortex and left precentral gyrus, all brain regions related by previous studies to multisensory integration used to maintain a coherent sense of one's bodily self.

For a better understanding of the role of posterior insular cortex in this context, it also might be useful to take a closer look at its functions and connections. Posterior insular cortex is a central node in a network for interoception (Craig, 2002), and its interactions with limbic, somatosensory and motor regions could be at the basis of a role in the processing of bodily feelings (Augustine, 1996; Craig, 2009). A series of studies also suggests that posterior insular cortex contributes to bodily self-awareness. Tsakiris et al. (2007) found a relationship between neural activation in posterior insular cortex and the subjective experience of the rubber hand illusion. This illusion concerns a condition in which an observed rubber hand synchronously stroked with participants' unseen hand is subjectively experienced as if it actually were one's own hand. Furthermore, posterior insular cortex has been related to the awareness of body parts in anosognosia patients with hemiplegia/hemiparesis (Karnath et al., 2005), and to the sense of agency, i.e. the feeling of being causally involved in an action (Farrer et al., 2003). Most recently, a lesion study (Heydrich and Blanke, 2013) showed a link between lesions in left posterior insular cortex and heautoscopy, a condition associated with the visual perception of a second own body, a strong self-identification with that second own body, and the experience of existing at and perceiving the world from two places at the same time. Importantly, autoscopic hallucinations where a second own body is seen without any changes in bodily self-consciousness, were found to be related to lesions in right occipital cortex, but not in posterior insular cortex.

Hence, taking into account the view of posterior insular cortex as a brain region crucially involved with bodily feelings and bodily awareness, the opposite activation patterns reported for touch experience and observation in posterior insular cortex (Ebisch et al., 2011a) could reflect its role in a pre-reflective, embodied differentiation between self and other conditions during social interaction.

Neuroimaging the social self in schizophrenia

Summarizing, the fMRI experiment by Ebisch et al. (2011a) allowed to investigate the contribution of neural mechanisms involved with the processing of our first-person bodily experiences to the simulation and sharing of others' (sensorimotor) experiences on the one hand, and self-other differentiation on the other hand. Notably, this offers an effective setting to extricate the nature of certain social difficulties in schizophrenia taking into account the complex interaction between sharing and differentiating self and other.

Consistent with psychopathology literature, empirical findings in schizophrenia demonstrate deficits at the level of self-other differentiation, for example, revealing misattributions of agency of action or body ownership. A reduced capacity in distinguishing between self-generated and externally perceived information has been reported consistently in patients affected by schizophrenia (Bentall et al., 1991; Blakemore et al., 2000; Franck et al., 2001; Vinogradov et al., 2008; Voss et al., 2010). For example, schizophrenic patients may have the impression that their actions or thoughts are controlled by external forces, or that they are in control of events that are not caused by their own actions. Such deficits have been explained in terms of loss of a coherently experienced sense of self, epitomized by aberrant integration of multisensory information, or failures to predict one's action effects, resulting in mismatches between predicted and actual sensory consequences. In a study were patients with schizophrenia were required to execute simple finger and wrist movements, while the image of either their own hand or a stranger's hand executing the same or a different movement was presented in real time on a screen, it was reported that patients with hallucinations and delusions had an increased tendency to interpret the alien hand as their own hand, compared to healthy controls and patients without hallucinations (Daprati et al., 1997). Furthermore, under some circumstances also the incorporation of external bodies linked with sensory experiences has been reported, like in the case of the rubber hand illusion. Interestingly this illusion was found to be enhanced in schizophrenia (Peled et al., 2000) as well as when healthy participants received infusions of ketamine, a noncompetitive N-methyl-D-aspartate antagonist reproducing symptoms of schizophrenia (Morgan et al., 2011). These findings suggest that the social self in schizophrenia could be impaired not only or necessarily in the ability to empathically share others' experiences, but also in attributing those experiences to their original source. Such misattributions of one's own or others' bodily experiences and thoughts could significantly interfere with social interaction in schizophrenia by blurring self-other relationship and distinction.

The same experiment as in Ebisch et al. (2011a) was employed to shed light on the question whether in first-episode schizophrenia (FES) functional abnormalities during social perception specifically concern the intersubjective domain or primarily have their roots in disturbances of self-experience (Ebisch et al., 2013a). Strikingly, fMRI results showed that differential activation in posterior insular cortex for first-person touch experiences and the observation of touch in another individual was absent in the FES group; no deactivation was found in posterior insular cortex during the observation of touch, though normal activation patterns were found in posterior insular cortex for first-person touch experiences. Specifically, significant differences in posterior insular cortex between the schizophrenia and the healthy control group were found for the observation of affective social touch, either with a positive (a caress) or with a negative (a hit) valence. A reduced BOLD suppression in posterior insular cortex in first-episode schizophrenia when witnessing others' tactile experiences could indicate a deficit in the pre-reflective suppression of self-oriented affective arousal. This suppression likely normally contributes to the differentiation between self and other conditions during social interaction. Corroborating the relevance of posterior insular cortex for a disturbed relationship with the external world, including the social one, in schizophrenia, aberrant posterior insular cortex activation has been related in previous studies to an impaired sense of agency of action in schizophrenia (Farrer et al., 2004).

In addition to posterior insular cortex, the study by Ebisch et al. (2013a) also yielded significant differences in neural activation in right ventral premotor cortex during touch observation and experience. Whereas in healthy control participants ventral premotor cortex activated in response to tactile bodily experiences as well as to videos depicting others' touch experiences,

activation in this region was absent in FES both during touch experience and during touch observation.

The functional and spatial properties of this ventral premotor cluster suggested that this region may reflect the putative human homologue of monkey premotor area F4 (Bremmer et al., 2001; Galati et al., 2001; Serino et al., 2011). For example, consistent with the multisensory properties of F4 and of its putative human homologue (Rizzolatti et al., 1981a/b; Bremmer et al., 2001), this region responded to visual as well as somatosensory stimuli in healthy participants. Of particular relevance, ventral premotor area F4 in the agranular frontal cortex of the macaque monkey (Matelli et al., 1985) is part of a circuit mapping sensory events in the space near the body onto arm and head movements (Rizzolatti et al., 2002). A large proportion of F4 neurons responds to both somatosensory and visual stimuli, whereas visual receptive fields of F4 neurons are mostly located in the space near the monkey (peripersonal space) and typically extend in the space adjacent to the tactile receptive fields of the same neurons (Fogassi et al., 1996). Generally, it has been proposed that this region could be involved in the integration of multisensory information from vision, audition, touch and proprioception onto the motor representations of different body-parts (Fogassi et al., 1996; Graziano et al., 1994; Rizzolatti et al., 2002). A putative human homologue of monkey area F4 has been identified by various studies in premotor cortex. For instance, Bremmer et al. (2001) demonstrated by means of fMRI that the ventral aspect of human premotor cortex responds to visual, auditory and tactile stimuli. Moreover, Serino et al. (2011) showed by using repetitive TMS a specific disruption of audio-tactile interactions around the hand, i.e. the processing of multisensory stimuli within peripersonal space.

On the basis of this evidence, aberrant ventral premotor cortex function – normally supporting a coherent sense of the bodily self – might be interpreted as part of a neural mechanism underlying the disruption of multisensory integration in schizophrenic pathology. Impaired multisensory integration could be an under-estimated disturbance in schizophrenia (Stekelenburg et al., 2013). An intriguing hypothesis that follows is that the integration of multisensory bodily information also contributes to the capacity to tell apart one's own bodily experiences from someone else's experiences during social interaction. Indeed, it would be plausible to presume that the online experience (e.g., self or source monitoring; Nelson et al., 2014) of integrated multisensory information, including visual, auditory, tactile, proprioceptive input, facilitates the definition of the asymmetry between primordially given bodily self-experiences and empathically shared experiences of others. As Edith Stein put it: "*non-primordial experiences that announce a primordial one*" (Stein, 1989, p. 10). The lack of a coherent, primordial perception of bodily experiences due to disrupted multisensory integration, as may be the case in schizophrenia, likely disrupts such an asymmetry in self-other relationship, accompanied by a blurring of the relationship and the distinction between self and other (e.g., Sass and Parnas, 2003).

Further support for these data comes from a recent fMRI study associating ventral premotor cortex with disrupted self-monitoring and self-other distinction in schizophrenia (Backasch et al., 2014). The results of this study showed that ventral premotor cortex activation and connectivity patterns could be related to a deficient integration of time-characteristics of sensory feedback with intentional movements in schizophrenia. In turn, such interrupted sensorimotor integration led to disruptions in self-other distinction, i.e., sense of agency (Backasch et al., 2014).

Finally, the relationship between impaired multisensory representations in ventral premotor cortex and anomalous self-experience was further corroborated by correlation analysis (Ebisch et al., 2013a): activation in right ventral premotor cortex during the social perception task correlated negatively and specifically with basic symptoms of the FES patients. Basic symptoms approximate anomalous self-experiences, representing subjective, subclinical, experiential disturbances in the domains of cognition, perception, bodily experience, action, and emotion (Huber, 1983; Klosterkotter, 1992). To some extent, they overlap with self-disturbances conceived as trait-phenomena captured by the EASE (Examination of Anomalous Self-Experience; Parnas et al., 2005). Of additional interest, basic symptoms reflect the first changes in experience that set off the

development of schizophrenic psychotic symptoms, and have been reported in all stages of the illness, i.e., in the prodrome to the first psychotic episode, in prodromes to relapse, in residual states, and during psychotic episodes (Schultze-Lutter, 2009). Therefore, they might represent a predisposition as well as a link between a more phenomenological approach to psychopathology, and a more categorical approach based on positive and negative symptoms (Parnas, 1999; Klosterkotter et al., 2001; Schultze-Lutter, 2009). From this perspective, correlations between neural activity and basic symptom severity reported by Ebisch et al. (2013a) suggest a close link between impaired multisensory representations in ventral premotor cortex and a disrupted sense of a coherent self in everyday life (Parnas et al., 2002).

Functional connectivity in the schizophrenia brain

Whereas the findings described above started shedding some light on how anomalous self-experience in schizophrenia connects with self-other relationship at the level of brain function, the exact neural mechanisms explaining aberrant focal brain function in social contexts remain poorly understood. Given that the brain-behavior relationship depends on neural activity organized in dynamic functional networks rather than on the function of brain regions in isolation, the study of functional connectivity could provide essential insights on how impairments in self-other relationship in schizophrenia could be related to aberrant brain function. Indeed, altered neural connectivity has been proposed as an elementary aspect in the pathophysiology of schizophrenia (Friston and Frith 1995; Pettersson-Yeo et al., 2011; Fornito et al. 2012; Uhlhaas and Singer 2010).

It could be hypothesized that brain regions associated with disturbances of the self-other relationship are characterized by anomalous functional connectivity patterns in schizophrenia, framing focal brain dysfunction within a network perspective. Therefore, in a subsequent study, it was investigated whether ventral premotor cortex and posterior insula could be characterized by altered neuro-functional interactions (Ebisch et al., 2013b). One of the principle findings was that both ventral premotor cortex and posterior insula were characterized by altered functional connectivity with posterior cingulate cortex.

Posterior cingulate cortex is a crucial node of the Default Mode Network (Buckner et al., 2008; Raichle et al., 2001) commonly implicated in schizophrenia (Skudlarski et al., 2010; Northoff et al., 2005). Particularly relevant for self-other relationship is that previous studies linked posterior cingulate cortex with self-referential processing (Northoff et al., 2006; Johnson et al., 2006), the integration of self-experience (Northoff and Bermpohl, 2004), and the regulation of the balance between internally and externally directed cognition (Leech et al., 2010; Wiebking et al., 2012). Furthermore, the results in healthy participants, in accordance with previous studies (Fox et al., 2009), showed that posterior cingulate cortex and ventral premotor cortex appeared to belong to distinct, possibly antagonistic functional networks. This segregation was decreased in FES. Based on the functions of posterior cingulate cortex indicated above and of ventral premotor cortex in multisensory integration, peripersonal space representation and goal-directed behavior (Fogassi et al., 1996; Serino et al., 2011), a decreased functional segregation between posterior cingulate cortex and ventral premotor cortex in schizophrenic patients could explain a deranged interaction between the intrinsic self (e.g. self-referential cognition mediated by posterior cingulate cortex) and the extrinsic self (e.g. the self interacting with the environment mediated by ventral premotor cortex) in FES. Highlighting the clinical relevance of this interaction in terms of anomalous self-experience, not only BOLD-response to social stimuli, but also the strength of functional connectivity of ventral premotor cortex with posterior cingulate cortex correlated significantly with basic symptoms (Ebisch et al., 2013b). Finally, since cortical midline (like posterior cingulate cortex) and ventral premotor cortices also have complementary functions for social cognition (Johnson et al., 2006; Caggiano et al., 2009; Gallese 2003; Uddin et al., 2007), altered interactions between these structures likely interfere with social cognition.

With respect to posterior insula interactions, a diminished functional connectivity was found with posterior cingulate cortex and primary somatosensory cortex/postcentral gyrus (Ebisch et al.,

2013b). These regions usually function together as a unique functional network (Leech et al., 2012; Taylor et al., 2009; Kurth et al., 2010). Such abnormal functional connectivity pattern resembles what has been found in autism spectrum disorder (Ebisch et al., 2011b). Considering the sensory-affective functions of posterior cingulate cortex and postcentral gyrus, and self-referential processing in posterior cingulate cortex (Northoff et al., 2006; Johnson et al., 2006; Northoff et al., 2004), reduced integrity within this network arguably can lead to deficient processing of bodily sensory-affective information as belonging to the self. An additional noteworthy result was that connectivity analysis yielded a disproportionally strong functional interaction between posterior and anterior insula. By contrast, although these regions have anatomical connections (Augustine, 1996), neuroimaging studies in the healthy brains show minimal functional connectivity between them (Taylor et al., 2009; Kurth et al., 2010). It was proposed that anterior insula facilitates the integration of cognitive and emotional responses, and underpins conscious evaluation of affective experience, that is, the subjective awareness of bodily feeling states (Craig, 2009; Critchley et al., 2004). Collectively, altered functional connectivity patterns of posterior insula demonstrate abnormal functional organization of a neural network involved in the awareness of the sensory-affective self. Posterior cingulate cortex could constitute a central node for the self interacting with its social environment. While in the healthy brain it has strong connections within the sensory network including posterior insula and somatosensory cortex (Leech et al., 2012) as well as an antagonistic relationship with ventral premotor cortex (Fox et al., 2009), anomalies were found at both levels in FES (Ebisch et al., 2013b). Therefore, the functional connectivity findings reported by Ebisch et al. (2013b) could reflect a neural mechanism underlying a deregulated interaction between an afflicted sensory-affective self and its (social) environment in FES.

Conclusions

In the present paper we proposed that the disruption of multisensory integration at the level of ventral premotor cortex might contribute to the disrupted sense of self characterizing schizophrenic patients. Studies in first-episode schizophrenia patients showed reduced activation in right ventral premotor cortex during the social tactile perception task, while this activation also correlated negatively and specifically with basic symptoms. Such self-disturbance, in turn, would contribute to alter self-other distinctions. Indeed, differently from healthy controls, first-episode schizophrenia patients were also characterized by the absence of differential processing of self- and other-related bodily information in posterior insular cortex during the same task.

In addition, from a neural network perspective, abnormal functional connectivity was found for both ventral premotor and posterior insular cortex with posterior cingulate cortex. Moreover, aberrant functional connectivity between the ventral premotor cortex and posterior cingulate cortex strongly correlated with the severity of basic symptoms in schizophrenic patients. Hence, the reviewed empirical data in patients affected by schizophrenia open a new window on the functional brain alterations at the basis of an imbalance in the pre-reflective relationship of the embodied self to the world including other individuals, possibly mediated by cortical midline structures.

These results confirm the heuristic power of this new neuroscientific approach to schizophrenia. Starting from a meticulous analysis of the phenomenal contents of the aberrant first-person perspective of schizophrenic patients and correlating such contents with the study of brain activity and connectivity during social tasks, one can shed new light on the neurobiological basis of this psychiatric disease. By empirically investigating the neural correlates of the basic experience one makes of oneself as bodily self and of its alterations, new light can be shed on the relationship between self-disturbances and social deficits in schizophrenia.

Acknowledgments

This work was supported by the EU grant Towards an Embodied Science of InterSubjectivity (TESIS, FP7-PEOPLE-2010-ITN, 264828) to V.G.

Reference list

Addington, J., Penn, D., Woods, S.W., Addington, D., Perkins, D.O. (2008). Social functioning in individuals at clinical high risk for schizophrenia. *Schizophrenia Research*, 99,119–124.

Augustine, J.R. (1996). Circuitry and functional aspects of the insular lobe in primates including humans. *Brain Research Reviews*, 22, pp. 229–244.

Backasch, B., Sommer, J., Klöhn-Saghatolislam F, Müller MJ, Kircher TT, Leube DT. (2014). Dysconnectivity of the inferior frontal gyrus: Implications for an impaired self-other distinction in patients with schizophrenia. *Psychiatry Research*, doi: 10.1016/j.pscychresns.2014.05.007 (e-pub ahead of print).

Banissy, M.J., Cohen Kadosh, R, Maus, G.W., Walsh, V., Ward, J. (2009). Prevalence, characteristics and a neurocognitive model of mirror-touch synaesthesia. *Experimental Brain Research*, 198, pp. 261-272.

Bastiaansen, J.A., Thioux, M., Keysers, C. (2009). Evidence for mirror systems in emotions. *Philos Trans R Soc Lond B Biol Sci*, 364, pp. 2391-2404.

Bellack, A.S., Green, M.F., Cook, J.A., et al. (2007). Assessment of community functioning in people with schizophrenia and other severe mental illnesses: a white paper based on an NIMH-sponsored workshop. *Schizophrenia Bulletin*, 33, pp. 805–822.

Bentall, R.P., Baker, G.A., Havers, S. (1991). Reality monitoring and psychotic hallucinations. *British Journal of Clinical Psychology*, 30, pp. 213–222.

Blakemore, S.J., Bristow, D., Bird, G., Frith, C., Ward, J. (2005). Somatosensory activations during the observation of touch and a case of vision-touch synaesthesia. *Brain*, 128, pp. 1571-1583.

Blakemore, S.J., Smith, J., Steel, R., Johnstone, C.E., Frith, C.D. (2000). The perception of self-produced sensory stimuli in patients with auditory hallucinations and passivity experiences: evidence for a breakdown in self-monitoring. *Psychological Medicine*, 30, pp. 1131-1139.

Bleuler, E. (1911). Dementia praecox oder Gruppe der Schizophrenien. In: Aschaffenburg G, ed. *Handbuch der Psychiatrie*. Leipzig, Germany: Deuticke; 1911.

Bonini, L., Rozzi, S., Serventi, F.U., Simone, L., Ferrari, P.F., Fogassi, L. (2010). Ventral premotor and inferior parietal cortices make distinct contribution to action organization and intention understanding. *Cerebral Cortex*, 20, pp. 1372-1385.

Bremmer, F., Schlack, A., Shah, N.J., et al. (2001). Polymodal motion processing in posterior parietal and premotor cortex: a human fMRI study strongly implies equivalencies between humans and monkeys. *Neuron*, 29, pp. 287–296.

Buckner, R.L., Andrews-Hanna, J.R., Schacter, D.L. (2008). The brain's default network: anatomy, function, and relevance to disease. *Ann N Y Acad Sci*, 1124, pp. 1–38.

Burns, J. (2006). The social brain hypothesis of schizophrenia. World Psychiatry, 5, pp. 77–81.

Caggiano, V., Fogassi, L., Rizzolatti, G., Their, P., Casile, A. (2009). Mirror neurons differentially encode the peripersonal and extrapersonal space of monkeys. *Science*, 324, pp. 403–406.

Caspers, S., Zilles, K., Laird, A.R., Eickhoff, S.B. (2010). ALE meta-analysis of action observation and imitation in the human brain. *Neuroimage*, 50, pp. 1148-1167.

Couture, S.M., Penn, D.L., Roberts, D.L. (2006). The functional significance of social cognition in schizophrenia: a review. *Schizophrenia Bulletin*, 32, pp. S44–63.

Craig, A.D. (2002). How do you feel? Interoception: the sense of the physiological condition of the body. *Nature Reviews Neuroscience*, 3, pp. 655–666.

Craig, A.D. (2009). How do you feel–now? The anterior insula and human awareness. *Nature Reviews Neuroscience*, 10, pp. 59–70.

Critchley, H.D., Wiens, S., Rotshtein, P., Ohman, A., Dolan, R.J. (2004). Neural systems supporting interoceptive awareness. *Nature Neuroscience*, 7, pp. 189–195.

Daprati, E., Franck, N., Georgieff, N., Proust, J., Pacherie, E., Dalery, J., Jeannerod, M. (1997). Looking for the agent: an investigation into consciousness of action and self-consciousness in schizophrenic patients. *Cognition*, 65, pp. 71-86.

Decety, J. (2011). The neuroevolution of empathy. Ann N Y Acad Sci, 1231, pp. 35-45.

Derntl, B., Finkelmeyer, A., Toygar, T.K., et al. (2009). Generalized deficit in all core components of empathy in schizophrenia. *Schizophrenia Research*, 108, pp. 197–206.

Ebisch, S.J., Ferri, F., Salone, A., Perrucci, M.G., D'Amico, L., Ferro, F.M., Romani, G.L., Gallese, V. (2011a). Differential involvement of somatosensory and interoceptive cortices during the observation of affective touch. *Journal of Cognitive Neuroscience*, 23, pp. 1808-1822.

Ebisch, S.J., Gallese, V., Willems, R.M., Mantini, D., Groen, W.B., Romani, G.L., Buitelaar, J.K., Bekkering, H. (2011b). Altered intrinsic functional connectivity of anterior and posterior insula regions in high-functioning participants with autism spectrum disorder. *Human Brain Mapping*, 32, pp. 1013-1028.

Ebisch, S.J., Salone, A., Ferri, F., De Berardis, D., Romani, G.L., Ferro, F.M., Gallese. V (2013a). Out of touch with reality? Social perception in first-episode schizophrenia. *Social Cognitive and Affective Neuroscience*, 8, pp. 394-403.

Ebisch, S.J., Mantini, D., Northoff, G., Salone, A., De Berardis, D., Ferri, F., Ferro, F.M., Di Giannantonio, M., Romani, G.L., Gallese, V. (2013b). Altered Brain Long-Range Functional Interactions Underlying the Link Between Aberrant Self-experience and Self-other Relationship in First-Episode Schizophrenia. *Schizophrenia Bulletin*, epub ahead of print.

Farrer, C., Franck, N., Georgieff, N., Frith, C.D., Decety, J., Jeannerod, M. (2003). Modulating the experience of agency: a positron emission tomography study. Neuroimage, 18, pp. 324–333.

Farrer, C., Franck, N., Frith, C.D., et al. (2004). Neural correlates of action attribution inschizophrenia. *Psychiatry Research*,131, pp. 31–44.

Fett, A.K., Viechtbauer, W., Dominguez, M.D., Penn, D.L., van Os, J., Krabbendam, L. (2011). The relationship between neurocognition and social cognition with functional outcomes in schizophrenia: a meta-analysis. *Neuroscience and Biobehavioral Reviews*, 35, pp. 573–588.

Fisher, M., McCoy, K., Poole, J.H., Vinogradov, S. (2008). Self and other in schizophrenia: a cognitive neuroscience perspective. *American Journal of Psychiatry*, 165, pp. 1465-1472.

Fogassi, L., Gallese, v., Fadiga, L., Luppino, G., Matelli, M., Rizzolatti, G. (1996). Coding of peripersonal space in inferior premotor cortex (area F4). *Journal of neurophysiology*, 76, pp. 141-157.

Fogassi, L., Ferrari, P.F., Gesierich, B., Rozzi, S., Chersi, F., Rizzolatti, G. (2005). Parietal lobe: from action organization to intention understanding. *Science*, 308, pp. 662-667.

Fornito, A., Zalesky, A., Pantelis, C., Bullmore, E.T. (2012). Schizophrenia, neuroimaging and connectomics. *Neuroimage*, 62, pp. 2296–2314.

Fox, M.D., Zhang, D., Snyder, A.Z., Raichle, M.E. (2009). The global signal and observed anticorrelated resting state brain networks. *Journal of Neurophysiology*, 101, pp. 3270–3283.

Franck, N., Farrer, C., Georgieff, N., et al. (2001). Defective recognition of one's ownactions in patients with schizophrenia. *American Journal of Psychiatry*, 158, pp. 454–459.

Friston, K.J., Frith, C.D. (1995). Schizophrenia: a disconnection syndrome? *Clinical Neuroscience*, 3, 89–97.

Galati, G., Committeri, G., Sanes, J.N., Pizzamiglio, L. (2001). Spatial coding of visual and somatic sensory information in body-centred coordinates. *European Journal of Neuroscience*, 14, pp. 737–746.

Gallese, V., Fadiga, L., Fogassi, L., Rizzolatti, G. (1996). Action recognition in the premotor cortex. *Brain*, 119, pp. 593-609.

Gallese V. (2001). The "Shared Manifold" Hypothesis: from mirror neurons to empathy. *Journal of Consciousness Studies*, 8, pp. 33-50.

Gallese, V., Fogassi, L., Fadiga, L., and Rizzolatti, G. (2002). Action Representation and the inferior parietal lobule. In Prinz, W., and Hommel, B. (Eds.) *Attention and Performance XIX*, (pp. 247-266). Oxford, UK: Oxford University Press.

Gallese V, Keysers C, Rizzolatti G. (2004). A unifying view of the basis of social cognition. *Trends in Cognitive Science*, 8, pp. 396-403.

Gallese V (2003). The roots of empathy: the shared manifold hypothesis and the neural basis of intersubjectivity. *Psychopathology*, 36, pp. 171-180.
Gallese, V. (2006). Intentional attunement: a neurophysiological perspective on social cognition and its disruption in autism. *Brain Research*, 1079, pp. 15-24.
Gallese, V (2007). Before and below 'theory of mind': embodied simulation and the neural correlates of social cognition. *Philos Trans R Soc Lond B Biol Sci*, 62, pp. 659-669.
Gallese, V (2009). Motor abstraction: a neuroscientific account of how action goals and intentions are mapped and understood. *Psychological Research*, 73, pp. 486-498.
Gallese, V., Sinigaglia, C. (2011). What is so special about embodied simulation? *Trends in Cognitive Science*, 15, pp. 512-519.
Gallese V., Ebisch S. (2013). Embodied simulation and touch: The sense of touch in social cognition. *Phenomenology & Mind*, 4, pp. 269-291.
Gallese, V. (2014). Bodily Selves in Relation: Embodied simulation as second-person perspective on intersubjectivity. *Phil. Trans. R. Soc. B* 2014 369, 20130177, pp. 1471-2970.
Graziano, M.S., Yap, G.S., Gross, C.G. (1994). Coding of visual space by premotor neurons. *Science*, 266, pp. 1054-1057.
Haug, E., Oie, M., Andreassen, O.A., Bratlien, U., Raballo, A., Nelson, B., Møller, P., Melle, I. (2014). Anomalous self-experiences contribute independently to social dysfunction in the early phases of schizophrenia and psychotic bipolar disorder. *Comprehensive Psychiatry*, 55, pp. 475-482.
Henriksen, M.G., Skodlar, B., Sass, L.A., Parnas, J. (2010). Autism and Perplexity: A Qualitative and Theoretical Study of Basic Subjective Experiences in Schizophrenia. *Psychopathology*, 43, 357-368.
Heydrich, L., Blanke, O. (2013). Distinct illusory own-body perceptions caused by damage to posterior insula and extrastriate cortex. *Brain*, 136, pp. 790-803.
Hillis, A.E. (2014). Inability to empathize: brain lesions that disrupt sharing and understanding another's emotions. *Brain*, 137, 981-997.
Horan, W.P., Kern, R.S., Shokat-Fadai, K., Sergi, M.J., Wynn, J.K., Green, M.F. (2009). Social cognitive skills training in schizophrenia: an initial efficacy study of stabilized outpatients. *Schizophrenia Research*,107, pp. 47–54.
Huber, G. (1983). The concept of substrate-close basic symptoms and its significance for the theory and therapy of schizophrenic diseases. *Nervenarzt*, 54, pp. 23–32.
Johnson, M.K., Raye, C.L., Mitchell, K.J., Touryan, S.R., Greene, E.J., Nolen-Hoeksema, S. (2006). Dissociating medial frontal and posterior cingulate activity during self-reflection. *Social Cognitive and Affective Neuroscience*, 1, pp. 56–64.
Karnath, H.O., Baier, B., Nagele, T. (2005), "Awareness of the functioning of one's own limbs mediated bythe insular cortex? ", *Journal of Neuroscience*, 25, pp. 7134–7138
Keysers, C., Gazzola, V. (2009). Expanding the mirror: vicarious activity for actions, emotions, and sensations. *Current Opinion in Neurobiology*, 19, pp. 666-671.
Keysers, C., Kaas, J.H., Gazzola, V. (2010). Somatosensation in social perception. *Nature Reviews Neuroscience*, 11, pp. 417-428.
Klosterkotter, J. (1992). The meaning of basic symptoms for the genesis of the schizophrenic nuclear syndrome. *Japanese Journal of Psychiatry and Neurology*, 46, pp. 609–630.
Klosterkotter, J., Hellmich, M., Steinmeyer, E.M., Schultze-Lutter, F. (2001). Diagnosing schizophrenia in the initial prodromal phase. *Archives of General Psychiatry*, 58, pp. 158–164.
Kurth, F., Zilles, K., Fox, P.T., Laird, A.R., Eickhoff, S.B (2010). A link between the systems: functional differentiation and integration within the human insula revealed by meta-analysis. *Brain Structure and Function*, 214, pp. 519-534.

Lamm, C., Decety, J., Singer, T. (2011). Meta-analytic evidence for common and distinct neural networks associated with directly experienced pain and empathy for pain. *Neuroimage*, 54, pp. 2492-2502.

Leech, R., Braga, R., Sharp, D.J. (2012). Echoes of the brain within the posterior cingulate cortex. *Journal of Neuroscience*, 32, pp. 215–222.

Matelli, M., Luppino, G., Rizzolatti, G. (1985), "Patterns of cytochrome oxidase activity in the frontal agranular cortex of the macaque monkey", *Behavioral Brain Research*, 18, pp. 125-136

Minkowski, E. (1927). *La schizophrénie. Psychopathologie des schizoïdes et des schizophrènes*. Paris, France: Payot.

Molenberghs, P., Cunnington, R., Mattingley, J.B. (2012). Brain regions with mirror properties: a meta-analysis of 125 human fMRI studies. *Neuroscience and Biobehavioral Reviews*, 36, pp. 341-349.

Morgan, H.L., Turner, D.C., Corlett, P.R., Absalom, A.R., Adapa, R., Arana, F.S., Pigott, J., Gardner, J., Everitt, J., Haggard, P., Fletcher, P.C. (2011). Exploring the impact of ketamine on the experience of illusory body ownership. *Biological Psychiatry*, 69, pp. 35-41.

Nelson, B., Whitford, T.J., Lavoie, S., Sass, L.A. (2014). What are the neurocognitive correlates of basic self-disturbance in schizophrenia?: Integrating phenomenology and neurocognition. Part 1 (Source monitoring deficits). *Schizophrenia Research*, 152, pp. 12-19.

Nelson, B., Sass, L.A., Thompson, A., Yung, A.R., Francey, S.M., Amminger, G.P., McGorry, P.D. (2009). Does disturbance of self underlie social cognition deficits in schizophrenia and other psychotic disorders? *Early Interv Psychiatry*, 3, pp. 83-93.

Northoff, G., Bermpohl, F. (2004). Cortical midline structures and the self. *Trends in Cognitive Science*, 8, pp. 102–107.

Northoff, G., Richter, A., Bermpohl, F., et al. (2005). NMDA hypofunction in the posterior cingulate as a model for schizophrenia: an exploratory ketamine administration study in fMRI. *Schizophrenia Research*, 7, pp. 235–248.

Northoff, G., Heinzel, A., de Greck, M., Bermpohl, F., Dobrowolny, H., Panksepp, J. (2006). Self-referential processing in our brain–a meta-analysis of imaging studies on the self. *Neuroimage*, 31, pp. 440–457.

Parnas, J. (1999). From predisposition to psychosis: progression of symptoms in schizophrenia. *Acta Psychiatr Scand*, 395, pp. 20–29.

Parnas, J., Bovet, P., Zahavi, D. (2002). Schizophrenic autism: clini-cal phenomenology and pathogenetic implications. *World Psychiatry*, 1, pp. 131–136.

Parnas, J., Møller, P., Kircher, T., Thalbitzer, J., Jansson, L., Handest, P., Zahavi, D. (2005). EASE: Examination of Anomalous Self-Experience. *Psychopathology*, 38, 236-258.

Peled, A., Ritsner, M., Hirschmann, S., Geva, A.B., Modai, I. (2000). Touch feel illusion in schizophrenic patients. *Biological Psychiatry*, 48, pp. 1105–1108.

di Pellegrino, G., Fadiga L, Fogassi L, Gallese V, Rizzolatti G. (1992). Understanding motor events: a neurophysiological study. *Experimental Brain Research*, 91, pp. 176-180.

Pettersson-Yeo, W., Allen, P., Benetti, S., McGuire, P., Mechelli, A. (2011). Dysconnectivity in schizophrenia: whereare we now? *Neuroscience and Biobehavioral Reviews*, 35, pp. 1110–1124.

Pinkham, A.E., Penn, D.L., Perkins, D.O., Lieberman, J. (2003). Implications for the neural basis of social cognition for the study of schizophrenia. *American Journal of Psychiatry*,160, pp. 815–824.

Raichle, M.E., MacLeod, A.M., Snyder, A.Z., Powers, W.J., Gusnard, D.A., Shulman, G.L. (2001). A default mode of brain function. *Proceedings of the National Academy of Sciences USA*, 98, pp. 676–682.

Rizzolatti, G., Scandolara, C., Matelli, M., Gentilucci, M. (1981a). Afferent properties ofperiarcuate neurons in macaque monkeys. I. Somatosensory responses. *Behavioural Brain Research*, 2, pp. 125–146.

Rizzolatti, G., Scandolara, C., Matelli, M., Gentilucci, M. (1981b). Afferent properties ofperiarcuate neurons inmacaque monkeys. II. Visual responses. *Behavioural Brain Research*, 2, pp. 147–163.

Rizzolatti, G., Fadiga, L., Gallese, V., Fogassi, L. (1996). Premotor cortex and the recognition of motor actions. *Brain Research Cognitive Brain Research*, 3, pp. 131-141.

Rizzolatti, G., Sinigaglia, C. (2010). The functional role of the parieto-frontal mirror circuit: interpretations and misinterpretations. *Nature Reviews Neuroscience*, 11, pp. 264-274.

Rizzolatti, G., Fogassi, L., Gallese, V. (2002). Motor and cognitive functions of the ventral premotor cortex. *Current Opinion in Neurobiology*, 12, pp. 149–54.

Sass, L.A., Parnas, J. (2003). Schizophrenia, consciousness, and the self. *Schizophrenia Bulletin*, 29, pp. 427-44.

Schultze-Lutter, F. (2009). Subjective symptoms of schizophrenia in research and the clinic: the basic symptom concept. *Schizophrenia Bulletin*, 35, pp. 5–8.

Serino, A., Canzoneri, E., Avenanti, A. (2011). Fronto-parietal areas necessary for a multisensory representation of peripersonal space in humans: an rTMS study. *Journal of Cognitive Neuroscience*, 23, pp. 2956–2967.

Skudlarski, P., Jagannathan, K., Anderson, K., et al. (2010). Brain connectivity is not only lower but different in schizophrenia: a combined anatomical and functional approach. *Biological Psychiatry*, 68, pp. 61–69

Stein, E. (1989). *On the Problem of empathy*, translated by W. Stein, ICS Publications, Washington, DC.

Stekelenburg, J.J., Maes, J.P., Van Gool, A.R., Sitskoorn, M., Vroomen, J. (2013). Deficient multisensory integration in schizophrenia: an event-related potential study. *Schizophrenia Research*, 147, pp. 253-261.

Straus, E. (1960). *Psychologie der menschlichen Welt*. Berlin,-Gottingen-Heidelberg: Springer, pp. 224-235.

Taylor, K.S., Seminowicz, D.A., Davis, K.D. (2009). Two systems of resting state connectivity between the insula and cingulate cortex. *Human Brain Mapping*, 30, pp. 2731–2745.

Tsakiris, M., Hesse, M.D., Boy, C., Haggard, P., Fink, G.R. (2007). Neural signatures ofbody ownership: a sensory network for bodily self-consciousness. *Cerebral Cortex*, 17, pp. 2235–2244.

Uddin, L.Q., Iacoboni, M., Lange, C., Keenan, J.P. (2007). The self and social cognition: the role of cortical midline structures and mirror neurons. Trends in Cognitive Science, 11, pp. 153–157.

Uhlhaas, P.J., Singer, W. (2010). Abnormal neural oscillations and synchrony in schizophrenia. *Nature Reviews Neuroscience*, 11, pp. 100–113.

Vinogradov, S., Luks, T.L., Schulman, B.J., Simpson, G.V. (2008). Deficit in a neural correlate of reality monitoring in schizophrenia patients. *Cerebral Cortex*, 18, pp. 2532–2539.

Voss, M., Moore, J., Hauser, M., Gallinat, J., Heinz, A., Haggard, P. (2010). Altered awareness of action in schizophrenia: a specific deficit in predicting action consequences. *Brain*, 133, pp. 3104–3112.

Wiebking, C., Duncan, N.W., Qin, P., et al. (2012). External awareness and GABA-A multimodal imaging study combining fMRI and [(18) F]flumazenil-PET. *Human Brain Mapping*, doi: 10.1002/hbm.22166

Zahavi, D. (2001), "Beyond empathy: Phenomenological approaches to intersubjectivity", *Journal of Consciousness Studies*, 8, pp. 151-167

Zahavi, D. (2010), "Empathy, embodiment and interpersonal Understanding: From lipps to Schutz", *Inquiry*, 53, pp. 285-306