

Schizophrenia as a disorder of embodied self

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Summary

Theories of schizophrenia propose numerous mechanisms underlying development of this disorder, yet the account including satisfactory etiological explanation is still lacking. Current trend is to indicate core, basic factors which may further give rise to whole diversity of symptoms. Experimental data and patients' reports show that such key factor may be self-experience disturbed on a basic, pre-reflective level, which can then lead to many higher-order symptoms. In this article we review and analyze these data, as well as the most influential cognitive theories focusing on mechanisms influencing the clinical picture of schizophrenia and giving rise to the anomalous experience of embodied self. These approaches pay attention to different, but complementary aspects of experience, such as body schema and body image, sense of ownership and sense of agency, or hyperreflexivity and diminished self-affection. Predictive coding approach is also introduced – the theory which, by appealing to the disturbance in a process of neural prediction, complements previous accounts and links various cognitive functions by means of single common predictive mechanism. Although very broad in its possible meanings, the self appears to be a concept of high explanatory value, grasping in the single framework many schizophrenic symptoms – these observable on neural, low-level of information processing, as well as on the level of phenomenology of subjective experience. Such approach appears to be valuable and useful for both research and practice.

Key words: schizophrenia, body image, embodied self

Introduction

It has long been recognized that schizophrenia is a heterogeneous disorder with strikingly different clinical manifestations. Experts disagree whether schizophrenia is a temporary, oversimplifying diagnostic convention, which under the common name combines many subtly differing dysfunctions, or if there is something that unifies them in a single illness. It seems that at least part of the inconsistencies comes from the fact that the picture of schizophrenia which emerges from the descriptions of the ICD-10 [1] or DSM-5 [2] is incomplete in light of current evidence. Although diagnostic criteria allow to objectively recognize the presence of the illness, there is still

lack of a cohesive theory of etiology of schizophrenia. Such theory would allow us to describe the mechanism responsible for its course and suggest potential causative treatment. Notably, textbooks of medical diagnosis do not ascribe much of importance to the early prepsychotic (prodromal) stage of the illness, in which one can distinguish far more drastic changes in behavior and experience of patients than just anxiety and social withdrawal. These changes are presumably anomalies of the embodied self, discussed in more detail in the following paragraphs. There are reasons to believe that symptoms of schizophrenia, although manifested differently in the developed stage of the illness, are secondary to disorders of embodiment. Such disorders appear even before the development of the so-called diagnostic symptoms [3, 4]. In other words, an evident loss of contact with reality, specific to psychotic episodes (manifested by delusions, hallucinations, disorganized speech and behavior [2]) may result from an earlier loss of contact with one's own body, understood as a transparent medium of interaction with the environment. So far, psychological theories of schizophrenia rarely have taken into account the aspect of body, focusing more on the delusional nature of the illness and exploring deficits of higher cognitive functions such as memory, attention, or cognitive control [5–7]. Yet considering the complexity of the clinical picture of schizophrenia, we aim to propose that anomalies of more basic functions of the self provide a much greater explanatory power.

At the most fundamental, phenomenological level self is tantamount to a sense of having a first-person perspective and being a specific entity with clearly demarcated physical boundaries between oneself and environment [8]. Many cognitive models propose that this preconceptual experience is a body and mind dynamic construction, the effect of proprioceptive, interoceptive and exteroceptive information integration [9, 10]. As such, it constitutes necessary condition for the development of self-image – the higher forms of metacognitive elaboration on the self, related directly to emotions, episodic memory, conscious, long-term concept of the self.

The recognition of the embodied self as a phenomenon strongly rooted in information integration [11] implies that it can be manipulated experimentally (for example, through the induction of multisensory conflicts) and furthermore, that it is susceptible to pathological disturbances. Deformation of the self is evident in a number of psychiatric disorders such as dysmorphophobia [12], eating disorders [13, 14] or some personality disorders [15]. Yet the most distinctive anomalies concerning self-consciousness are manifested in schizophrenia. Understanding this illness as resulting from disordered self has been present in the scientific literature for a long time, usually though with emphasis put on deformation of higher forms of self-consciousness. Identity delusions, delusions of grandiosity, inadequate long-term concept of the self seem to be pertinent examples here [9]. Nonetheless, more recently it became evident that the self of a patient suffering from schizophrenia is distorted on a much more basic, prereflective level of body awareness.

In this paper we attempt to join the discussion on the core symptoms of schizophrenia. Anomalies of the embodied self constitute a potential etiological core, unifying all

subtypes of schizophrenia. The following review of first-person reports and empirical studies supports such a view.

Phenomenology of schizophrenic experience

The only available source of information about what it is like to experience schizophrenia are reports of patients. Analysis of 552 self-reports has consistently indicated that patients experience distorted forms of self-awareness. Here are some typical examples, provided by Scharfetter [16]: “I was no longer sure that I was still the same person”; “I became one with other creatures or objects. I lost the sense of my own boundaries”; “When I had an experience I often did not know if it was mine or the experience of someone else” [16, p. 286–287]. Unstable sense of self often leads to unpleasant sensations of depersonalization and derealization – altered perception of time and space, feeling of distance in regard to one’s body [17]. In one of the case studies Parnas [18] reported about a patient who, in attempt to prevent this type of sensations, decided to wear an extremely wide and tight strap so to clearly stabilize the boundaries between the self and not self. Other attempts to regain a sense of stable embodiment are manifested in self-mutilation and reports such as: “I did not feel my skin anymore, so I had to rub or pinch or punch it”; „I had to hurt myself deliberately in order to cause pain” [18, p. 288; see also 19].

Many verbal reports concerning phenomenology of prepsychotic stage of schizophrenia have been collected by Zadęcki [20]. He described, *inter alia*, the case of Zosia, who after experiencing a school failure, said: “I do not know what happened to me, who I am, how to tell you all this. I do not know if I have hands, feet, or I do not know if I have face, am I very pale?” [20, p. 117]. Zadęcki suggests (as cited in: [21]) that in schizophrenia body begins to be treated instrumentally, experienced as an external object, thereby analyzed only by the “observational self”. Another case concerns a patient manifesting increasing “quasi-objectification” of introspective experience. In his statements one can note a growing distance between the self and the very stream of consciousness, as well as kind of decentralization: “thoughts appear as if out of nowhere, they are not mine”; they often take on characteristics of physical objects: “my thoughts are dense and encapsulated”, “my thoughts feel mainly in the right side of the brain”; “it feels as if my thoughts were slightly behind my skull” [18, p. 228]. Some of the patients experience utter confusion when watching his or her own reflection in a mirror [18] and complain about a problem with identifying which side presents the real self (the so-called *heautoscopy*, see [8]). Some report even more extreme aberrations: “If I look at someone else in the mirror, I am not able to distinguish him from myself any more. When I am feeling worse, the distinction between me and a real other person gets lost, too. While watching TV, I don’t know any more, whether I am speaking in the TV-set or whether I am hearing the words here. [...] Are there perhaps two <I>s?” [22, p. 104].

Disembodiment is usually accompanied with disturbed sense of agency, “I feel directed by alien forces”, “I could no longer do what I intended to do, my movements

and actions were directed and controlled. I felt like a tool, a puppet” [16, p. 273]. Others seem to feel that they cannot perform any action automatically, without explicit elaboration. This in turn can give rise to states of helpless freezing in a catatonic stupor, sometimes lasting for hours. An interesting illustration of “deautomatization” experience is the case of a patient described by Buerger (as cited in: [22]). The patient reported the persistent need to devote full attention to his body and its movements, which eventually began to feel alien.

Delusions associated with schizophrenia are particularly distinctive. Not only do patients report the illusion of external control over their motor actions but also call into question the intentional origin of their own thoughts (thought insertion), for example: “The thoughts of Eamonn Andrews come into my mind. He treats my mind like a screen and flashes his thoughts on to it like you flash a picture” [23, p. 51]. Detaching mental states from the very feeling of mineness may exacerbate the impression that a boundary between one’s mind, the world, and the minds of others becomes blurred and permeable. As a consequence, this feeling may contribute to the increase in anxiety and enhance the paranoid model of events interpretation.

There is, therefore, a lot of phenomenological evidence showing that patients with schizophrenia to some extent experience anomalies regarding their own corporeality and self-awareness.

Theoretical frameworks of the embodied self in health and pathology

Contrary to the classical symbolic approach, which assigned peripheral meaning to embodiment in the genesis and functioning of the mind, embodied cognition puts special emphasis on the body as a bridge between the mind and the world [24]. Philosophical grounds for the development of this paradigm were the works of phenomenologists and existentialists, including Heidegger [25], Merleau-Ponty [26] and Jaspers [27] – who clearly shared the view on embodied nature of the self. Contemporary cognitive science, drawing upon philosophers’ ideas, indicates that embodiment is the key to solve the semantic problem (how do representations gain meanings?) and the mind-body problem (how to determine the relationship between a mind and a body?) [28]. Proponents of the embodied view stress the fact that cognition is always situated in the environment, restricted by time pressure and task-oriented [24]. The body is therefore an inevitable medium of any interaction with the physical environment, and each thought process, to a certain extent, depends on the dynamic interactions between the neuronal substrate, action and external feedbacks [29].

From this point of view, transparent and stable representation of the body always imposes a specific, contextual frame on perception and essentially, makes the perspective subjective, first-person. As a consequence, according to the embodied view, any distortion in the body image necessarily influence information processing that may lead to psychological anomalies. There are several models to explain how this process

takes place and provide immediate context for understanding disorders of embodied self in schizophrenia.

The minimal and the narrative self

The first theoretical framework essential for further consideration of self-awareness in schizophrenia was provided by Gallagher [30–32]. Two levels of self indicated already in the introduction, refer directly to his famous distinction between the minimal self and the narrative self. While the minimal self denotes the phenomenology of subjective “now”, the narrative self is characterized by a higher level of conscious cognitive elaboration and a coherent self-image dependent on individual history. In this approach the minimal self is closely related to the aspect of embodiment; it creates nonconceptual, but constantly implicitly present “in the background”, sense of the first-person action in the environment and can be further described by drawing a division into its two aspects: the sense of agency – the experience that self is the author and initiator of voluntary action, and the sense of ownership – a natural, automatically taken for granted, implicit belief that self is the subject of first-person experience. Both aspects of the self-experience seem to be concurrent and indistinguishable. However, some research paradigms and clinical observations suggest the possibility of their dissociation and by that means, the separation of the mechanisms underlying them [33, 34].

The covert and reflective level of self-experience corresponds to the another, significant terminological distinction between body schema and body image. Although both systems in healthy individuals operate in strictly coordinated manner, they are characterized by different properties. Body image constitutes its conscious, active and dynamic representation; it underlies emotions, bodily sensations and explicit intentions. On the other hand, body schema remains consistently unavailable to consciousness; it is a collection of hidden rules and neurological mechanisms operating prior to or outside of conscious intention. However, body schema is necessary for monitoring, coordinating, planning of movement or maintaining body posture. Heavily distorted body schema sooner or later affects its conscious image [35].

Gallagher points out that schizophrenia, above all, is distinguished by the primary anomaly of the minimal self, which can in turn contribute to the problems with the notion of self at the reflective level. In the case of psychotic patients one can sometimes observe the abnormal body image [36, 37], but presumably it is the erroneous body schema that is deregulating the embodied self. Body schema, disturbed in schizophrenia [38], has an undeniable impact on perception, action and phenomenology, but in itself is essentially inaccessible to consciousness. Therefore, patients usually do not explicitly report the peculiar bodily experiences, especially given that the described anomaly is difficult to be articulated in propositional terms. The impact of disordered schema can, however, be observed indirectly.

Ferri et al. [39] confirmed the hypothesis that in patients with schizophrenia not only conscious access to the representation of the body is disturbed but also covert

access. One of the tasks was to present participants with three images of the upper or lower limb and ask them to identify which image, upper or lower, was identical to the target picture. All healthy controls identified limbs faster when the screened image depicted their own arm or leg (the so-called self-advantage effect). This effect was not observed in patients, which seems to indicate pathology of covert access to the body representation. The second task involved the overt recognition on which of the three displayed pictures showed one's own leg or arm – in this task patients also performed worse than the healthy controls.

Similarly, Kircher and David [9] showed that patients suffering from schizophrenia cannot recognize their own faces as effectively as healthy participants. Such deficit seems to correspond to a reduction of volume of fusiform gyrus, documented in post mortem and neuroimaging studies on patients [40, 41]. Furthermore, abnormalities concerning volume of fusiform gyrus are progressive in schizophrenia [42].

There are therefore serious grounds to believe that schizophrenia is an illness associated with anomalies both in the body schema and body image. Yet still, bearing in mind the priority of bodily self-awareness anomalies over higher reflective levels of the self, it is worth to introduce some concepts dealing directly with embodied self.

Minimal Phenomenal Selfhood Theory

Minimal Phenomenal Selfhood Theory [43, 44] stems from the ground of functionalism and representationalism and pertain precisely the embodied experience of self. According to this theoretical view, the self does not exist as a single construct; it is also not identical with the body or specific neuronal structure. The sense of subjective situatedness in the environment is rather a process of information processing through a subjective model of the self (self-model). Metzinger defines this model quite generally, as an active data structure, possibly encompassing vast areas of the prefrontal cortex, involved in constant, dynamic operations of simulation and re-representation of states of the system for its current, ongoing needs. To give rise to phenomenology of the self, model must satisfy some functional conditions such as making information globally accessible for motor action or its availability in introspection. A crucial feature of the self-model is transparency – self-model cannot be recognized by the agent as a model, but it is permanently used as an invisible tool for active perception. Transparency results here directly from the fact that certain neural computation processes remain inaccessible for attentional exploration, therefore they always take place out of consciousness, implicitly. The subject has access only to the final results of information processing, which underlay a natural “certainty” and create the illusion of direct contact with reality. Notably, owing to the transparency, the body can be experienced as a real, dynamic medium of interaction with the world, not just the internal construct.

Schizophrenia, on the ground of Metzinger's theory, can therefore simply come down to the incorrect integration of content in the self-model. When the integration proceeds adversely, the incorrect result of processing is also taken for granted. Pre-

sumably, it is what stays behind depersonalization, hallucinations, delusions and other anomalies associated with the experience of self. In this perspective, some of the positive symptoms would be understood as active cognitive representations that remain conscious, but progressive loss of transparency deprives them of the phenomenological quality of “mineness”. As a result, one’s own thoughts cease to be perceived as internally generated; therefore, they arouse suspicion and foster persistent delusions regarding their external origin. The disappearance of a clear boundary between “mine” and “not mine” also raises a very characteristic fear that other people “read” the mind of the patient.

Hyperreflexivity and diminished self-affection

Metzinger’s theory seems to properly illustrate phenomenology of experiencing schizophrenia. However, it does not identify the specific mechanisms responsible for errors in information integration within a consistent, coherent model of self. The approach of Sass and Parnas [45, 46] may be used for the purpose of complementary extension. Both models assume that a healthy self-awareness is characterized by transparency. They also indicate that the central axis of the pathogenesis of schizophrenia is a disordered experience of the self, in regard to which symptoms listed in the DSM-5 and ICD-10 are secondary. An important postulate of Sass is the introduction of the notion analogical to the Gallagher’s minimal self – ipseity, which refers to a prereflective, covert awareness that every experience is always fundamentally perceived from the first person perspective – naturally and unconsciously classified as “mine”. Schizophrenia is accompanied by two complementary irregularities of ipseity: hyperreflexivity and diminished self-affection.

The first component refers to the exaggerated self-consciousness and the loss of its transparency. A person can experience usually hidden, inhibited or unconscious properties of his/her experience in a way so explicit and clear, as would happen in the case of experience of an object or external event. In the long term, this contributes to the perception of oneself and one’s own body rather as an alien object than a subjective entity. Diminished self-affection is in turn a phenomenon of significant reduction of primary, preconceptual sense of presence of oneself in the world – limiting and shallowing usually implicit feeling of “being myself”. Both aspects of the disorder well reflect the characteristic schizophrenic fear, reported in the words: “Do I think? Since there is nothing that would confirm that I think, I cannot know whether I exist” [18, p. 229]. The continuous cognitive elaboration, theming, touching upon oneself, entail the loss of implicit gestalt structure of the body (loss of transparency of experience). Hyperreflexivity and diminished self-affection can condition each other and constitute a consequence of a single, common pathological process.

The model of Sass and Parnas has been supported by many phenomenological interviews [8, 16, 22, 47, 48]. It seems that it adequately describes the experience of psychotic patients, especially in the early stages of the illness. However, despite the

large number of theoretical articles and collections of case studies on phenomenology of “disembodiment” in schizophrenia, there is relatively small number of empirical studies conducted in this area. Some of them provide support for Sass’ theoretical framework. Maintaining a reflective focus on usually unattended aspects of experience leads to diminished sense of agency, diminished sense of ownership and the experience of “ego permeability”. As demonstrated by Huff and Chefurka [49], such effect can be elicited by unnaturally prolonged, intensive concentration on the sense of bodily self, permanent monitoring of “now” status. Similar results were provided by Petitmengin and Bitbola [50], who induced an excessive focus on usually unattended aspects of auditory perception: instead of concentrating on heard sound’s source or meaning, subjects were asked to attend to the “bodily felt sound” (“what it does to me”). According to Sass, even relatively short induction of hyperreflexivity on healthy participants can lead to temporary, psychotic-like symptoms [51].

Hypothetically, a prolonged state of hyperreflexivity can cause a permanent reorganization of body representation. One study indicates that in the case of schizophrenia patients it is much easier to induce the rubber hand illusion – the feeling of embodiment of artificial hand, caused by a multisensory conflict – in comparison with the healthy controls [52]. Authors of the study interpret this relationship as evidence for much more flexible body representation among patients. This is also confirmed by research on the neural organization of the associative regions in this group. During rubber hand illusion patients show an abnormal SEP (somatosensory evoked potential) response [53]. As for both early and late components of SEP above associative regions healthy subjects exhibit significant difference between pre-illusion and illusion condition, yet such difference is not significant among schizophrenia patients. Moreover, temporo-parietal junction, described as essential in dynamic maintenance of healthy, self-updating representation of embodied self [11], is characterized by diminished functional connectivity with Broca’s area in a psychotic group (what corresponds well with the occurrence of auditory hallucinations) [54].

Predictive coding and embodiment

A common distinction of positive symptoms in schizophrenia is the one made between hallucinations and delusions. A classic Maher’s theory [55] links these two kinds of symptoms by claiming that abnormalities in the perceptual system (or in other systems related to it, such as attentional one), responsible for emergence of hallucinations, are primary. Delusions are understood as secondary symptoms, which emerge in the process of higher-order rationalizations and interpretations of the experienced, disordered percepts. Such distinction between hallucinations and delusions, clear on phenomenological level, can actually overrate the actual degree of separation of processes underlying these symptoms.

An alternative approach, which treats such kinds of symptoms as an effect of abnormality of the mechanism common not only for perception and thinking but also for

a number of other cognitive functions, is the Bayesian hierarchical system approach, also referred to as predictive coding [56–62]. Within this view, the most fundamental neurocognitive function performed by the brain is a neural prediction and minimization of error and surprise. Here, prediction and surprise should not be understood as taking place on a personal level, but rather are sub-personal processes, i.e., they take place on a relatively low neural levels and are unavailable for conscious access. According to this approach, perceptual content is created in upper layers of hierarchy by means of a so-called generative model. The role of such model is to estimate the structure of external and internal environment on the basis of currently available data. Nature of such model is always probabilistic, because incoming sensory signal is always ambiguous and noisy – system needs to hypothesize about world's structure on the basis of this imperfect information [63]. The crucial point in this approach is the nature of incoming information processing: bottom-up pathways do not convey content in the standard understanding, because it is created in a top-down manner by the generative model. Instead, bottom-up pathways convey prediction error, i.e., a difference between predicted and actual stimulation. Predictions generated on the higher levels of the system thus serve as a kind of a frame imposed on lower levels, which, if accurate, explains away the sensory signal. In the case of incongruence of prediction and the input, bottom-up prediction error signals that model is not optimal. This leads to updating of model and its the higher level predictions and thus minimization of future errors. Minimization of errors is done not only in perception (via modifications of the model) but also in embodied action, by means of active inference. In the latter case, motor control proceeds in a way that probing of the environment is congruent with the interoceptive prediction [64].

In predictive coding framework, the distinction often made between perception and reasoning is much less sharp. Perception is always controlled and limited from the upper levels of the system – what we perceive is determined by what we expect. This framework is capable to explain many empirical observations – both everyday perceptual illusions and apparently difficult to explain experimental results (for a review, see [56]). Fletcher and Frith [23] use this approach to show how abnormal prediction error signaling leads to hallucinations and delusions.

Many research show abnormalities in predicting different kinds of signals and events in psychotic patients. For example, Voss et al. [65] manipulated probability of action's effect occurrence and demonstrated that subjects with schizophrenia present a deficit in predicting sensory consequences of their own actions. Moreover, this deficit's amplitude positively correlated with the intensity of positive symptoms. Patients also present abnormalities in smooth eye pursuit which depends on predicting the movements of tracked object [66]. Yet another example here is a sensory attenuation effect. In sensory attenuation, intensity of predictable stimuli (such as sensory effects of own actions) is subjectively lower (attenuated) as compared to objectively identical stimuli that are unpredictable (e.g., effects of external events). It has been shown that this prediction-dependent phenomenon is diminished in schizophrenic patients [67, 68].

This type of data fits well to a relatively well known and studied deficit of source monitoring resulting from inaccurate neural mechanisms discriminating endo – and exogenous stimulation. Disturbed attenuation of sensory effects of own actions can lead to experiencing endogenous stimulation similarly to the exogenous one [69]. This kind of problem can lead to disturbances of sense of agency, present in this group of patients [70, 71], as well as to different symptoms such as hallucinations. It is being postulated that optimal connectivity between sensory and motor areas in the brain, which is known to be significantly diminished in schizophrenia, is necessary for optimal source monitoring [72]. For example, it has been shown that patients' disturbed connectivity between superior temporal cortex and anterior cingulate cortex is related to inaccurate speech attribution and auditory hallucinations [73].

Accurate, dynamic prediction is crucial for perception of external events. Predictable stimulation, being incorporated to the system of beliefs about given situation (prior), has different functional meaning than the stimulation which is unpredictable and surprising. Fletcher and Frith [23, see also 57] propose that in psychopathology the main dysfunction lies in abnormalities of prediction error signaling (or, more precisely – abnormalities in its weigh and relative impact on further cognitive processing and action). Such claim is supported by empirical data. For example, two EEG event-related potential components can serve as electrophysiological markers of prediction errors. First is a decrease of an amplitude of a potential evoked in response to repeating presentation of identical, predictable stimuli (repetition suppression), which reflects processes of adaptation and matching predictions to sensory input (closely related to described sensory attenuation effect). Second is a mismatch negativity, which is a negative event-related component evoked by recording of the difference between predicted and actual stimulation. It has been shown that these two components are diminished in the group of psychotic patients [74, 75].

Prediction error is a crucial signal for learning [76]. Stimuli leading to error are significant for the system as they convey novel information – they require adaptation (model's updating) in order to make future estimations more accurate. This is closely related to the process of setting significance and perceiving salience of stimuli, which is greater for unpredictable ones.

Disturbances in predictive processes observable in psychotic patients can also lead to abnormalities in setting significance and perceiving salience of signals coming from the body and from the external environment. In this respect, predictive coding framework is compatible with Kapur's theory [77] of psychosis, which describes it as a state in which stimuli and events receive erroneous, aberrant salience. Neurophysiological research also provide support for such account. Dopaminergic system is strongly activated in learning based on errors and rewards [78]. Disturbances of this system observed in schizophrenia, can easily be linked to abnormalities in probabilistic learning [79] and diminished latent inhibition (i.e., an effect of inhibited learning about stimuli which previously did not convey any novel information) [23, 80], as well as in assigning meaning.

Predictive coding theory is strongly rooted in embodied approach to cognition, emotion and action, and is capable of linking these functional domains by common mechanism of prediction and prediction error minimization –by means of perception (modification of generative model so that it matches stimulation) and action (modification of stimulation so that it matches the model). As it has been shown, abnormalities in setting adequate significance and weight to the signals coming from the internal and external environment can contribute to the problems in distinguishing the source of stimulation. Such distinguishing is crucial for forming both explicit and implicit concept of “self” (and “not-self”), together with its bodily (physiological) basis.

As accurately noted by Nelson et al. [81], patients’ self-experience problems (phenomenological level) directly correspond to two kinds of deficits on neurocognitive level, i.e., to the source monitoring deficit and aberrant salience. Also, according to Seth et al. [62], predicting and accurate signaling (interoceptive) of prediction error are necessary for abovementioned processes and also for the subjective experience of presence and consciousness as such. In this sense self (together with its elements or aspects) is dependent on prediction. Prediction error approach thus unifies the abovementioned concepts by appealing to the common predictive mechanism. Disturbances in the predictive processing will clearly influence further cognitive processes and experiences, and thus also subject’s functioning in domains which relay on it. Such association can be seen not only in schizophrenia but also in other psychiatric disturbances (such as depression[82]).

Applications and conclusions

So far, cognitive behavioral therapy for schizophrenia focused mainly on the cognitive formation of positive symptoms [83]. One of its significant purposes was to identify the deficiencies in patient’s reasoning through the use of classical techniques of Socratic questioning [84]. Nevertheless, following the reasoning presented in this paper, already build-up delusional constructions should not be treated as a primary aim of psychotherapy. An attempt to modify the paranoid beliefs by detecting and discussing the errors in patient’s reasoning may be unsuccessful, mainly because subjective experiences underlying the delusions are real and indisputable for the patients. It is therefore worth to differentiate the individuals susceptible to the development of psychosis from healthy individuals by identifying anomalies at the level of prereflective self-experience. It is difficult, however, to reveal such subtle irregularities in the course of standard, structured medical interviews. Phenomenological interviews seem to be much better suited to these purposes [85, 86]. Sensitive, particularly to a subjective side of the illness, they enable broader, qualitative insight into “what-it’s-like” to be in a particular pathological state.

In addition to early detection of the illness and conducting prevention, the above findings are of potential use also for treatment. There are evidence showing that the

activation of AMPA and GABAA receptors may correspond to a bottom-up prediction error coding, while the slower activation of NMDA – to specification of the current generative model [76]. It can be proposed that the precise identification of functional, neural Bayesian substrate of information processing would allow for the improvement of the pharmacological model of psychosis, and, in consequence, the development of more effective drugs. A promising complement to pharmacotherapy would also be a therapy focused on training of processes related to the monitoring of the source of incoming stimuli. A significant improvement of the condition of patients diagnosed with schizophrenia after regular participation in body-oriented psychological therapy has been documented in several studies [86–88]. One of the core assumptions of such therapy is a close connection of motor activity with dimension of experience and social interaction. Group exercises include, inter alia, mirroring of partner's movements and monitoring one's body boundaries in an organized action. According to theories of motor simulation such training is to influence covert bodily representation [89]. Systematic work on the insight into the nature of one's symptoms is also emphasized. Phenomenological interviews reveal the positive impact of this type of therapy on the experience of the self among patients [90].

Self should therefore be understood as a fully embodied structure, dependent on processes enabling distinguishing the source of stimulation, ascribing meanings to signals from the environment, as well as forming transparent models of that environment. Despite – or thanks to – the multiplicity of perspectives from which schizophrenia can be seen and studied, it appears as a disorder which originates from anomalies in the embodied self, revealing on many levels of the organization, and in many functional aspects of the system/person. Such an approach to this illness appears to be cognitively accurate but also to be useful for practical applications.

References

1. World Health Organization, *The ICD-10 classification of mental and behavioural disorders: Clinical descriptions and diagnostic guidelines*. Geneva: World Health Organization; 1992.
2. American Psychiatric Association. *Diagnostic and statistical manual of mental disorders*, 5th ed. Washington, American Psychiatric Pub; 2013.
3. Parnas J. *A disappearing heritage: The clinical core of schizophrenia*. *Schizophr. Bull.* 2011; 37(6): 1121–1130.
4. Nelson B, Thompson A, Yung AR. *Basic self-disturbance predicts psychosis onset in the ultra high risk for psychosis "prodromal" population*. *Schizophr. Bull.* 2012; 38(6): 1277–1287.
5. Hutton SB, Puri BK, Duncan LJ, Robbins TW, Barnes TR, Joyce EM. *Executive function in first-episode schizophrenia*. *Psychol. Med.* 1998; 28(02): 463–473.
6. Barch DM, Csernansky JG, Conturo T, Snyder AZ. *Working and long-term memory deficits in schizophrenia: Is there a common prefrontal mechanism?* *J. Abnorm. Psychol.* 2002; 111(3): 478.
7. Lee J, Park S. *Working memory impairments in schizophrenia: A meta-analysis*. *J. Abnorm. Psychol.* 2005; 114(4): 599.

8. Blanke O, Metzinger T. *Full-body illusions and minimal phenomenal selfhood*. Trends Cogn. Sci. 2009; 13(1): 7–13.
9. Kircher T, David A. *Self-consciousness: An integrative approach from philosophy, psychopathology and the neurosciences*. In: Kircher T, David A ed. *The self in neuroscience and psychiatry*. Cambridge: Cambridge University Press; 2003. S. 445–473.
10. Ehrsson HH. *The concept of body ownership and its relation to multisensory integration*. In: Stein BE ed. *The New Handbook of Multisensory Processes*. Cambridge: MIT Press; 2012. p. 775–792.
11. Tsakiris M, Costantini M, Haggard P. *The role of the right temporo-parietal junction in maintaining a coherent sense of one's body*. Neuropsychologia 2008; 46(12): 3014–3018.
12. Bjornsson AS, Didie ER, Phillips KA. *Body dysmorphic disorder*. Dialogues Clin. Neurosci. 2010; 12(2): 221–232.
13. Izydorczyk B. *Psychologiczny profil cech ja cielesnego u młodych kobiet polskich – analiza porównawcza struktury ja cielesnego u kobiet chorych na zaburzenia odżywiania się i zaburzenia psychiatryczne*. Psychiatr. Pol. 2011; 45(5): 653–670.
14. Spitoni GF, Serino A, Cotugno A, Mancini F, Antonucci G, Pizzamiglio L. *The two dimensions of the body representation in women suffering from Anorexia Nervosa*. Psychiatry Research 2015; 230(2): 181–188.
15. Asai T, Tanno Y. *The relationship between the sense of self-agency and schizotypal personality traits*. J. Mot. Behav. 2007; 39(3): 162–168.
16. Scharfetter C. *The self-experience of schizophrenics*. In: Kircher T, David A ed. *The self in neuroscience and psychiatry*. Cambridge: Cambridge University Press; 2003. p. 272–289.
17. Sedeño L, Couto B, Melloni M, Canales-Johnson A, Yoris A, Baez S et al. *How do you feel when you can't feel your body? Interoception, functional connectivity and emotional processing in depersonalization-derealization disorder*. PloS One 2014; 9(6): e98769.
18. Parnas J. *Self and schizophrenia: A phenomenological perspective*. In: Kircher T, David A. ed. *The self in neuroscience and psychiatry*. Cambridge: Cambridge University Press; 2003. p. 217–241.
19. Kubiak A, Sakson-Obada O. *Repetitive Self-Injury and the body self*. Psychiatr. Pol. 2016; 50(1): 43–54.
20. Zadęcki J. *„Ja” we wczesnej schizofrenii*. Krakow: Jagiellonian University Press; 2015.
21. Laing R. *The divided self: A study of sanity and madness*. London: Tavistock Publications; 1960.
22. Fuchs T. *Corporealized and disembodied minds: A phenomenological view of the body in melancholia and schizophrenia*. Philosophy, Psychiatry, and Psychology 2005; 12(2): 95–107.
23. Fletcher PC, Frith CD. *Perceiving is believing: A Bayesian approach to explaining the positive symptoms of schizophrenia*. Nat. Rev. Neurosc. 2009; 10(1): 48–58.
24. Wilson M. *Six views of embodied cognition*. Psychon. Bull. Rev. 2002; 9(4): 625–636.
25. Heidegger M. *Being and time*. New York: Harper & Row; 1968.
26. Merleau-Ponty M. *Phenomenology of perception*. London: Routledge & Kegan Paul; 1962.
27. Jaspers K. *The phenomenological approach in psychopathology*. Br. J. Psychiatry 1968; 114(516): 1313–1323.
28. Chalmers DJ. *The conscious mind: In search of a fundamental theory*. Oxford Paperbacks; 1997.
29. Wierchoń M, Łukowska M. *Ucieleśnione poznanie*. In: Bremer J ed. *Przewodnik po kognitywistyce*. Krakow: WAM Publishing House; 2016.

30. Gallagher S. *Philosophical conceptions of the self: Implications for cognitive science*. Trends Cogn. Sci. 2000; 4(1): 14–21.
31. Gallagher S. *Self-reference and schizophrenia*. In: Zahavi D. ed. *Exploring the self. Philosophical and psychopathological perspectives on self-experience*. Amsterdam–Philadelphia: John Benjamins; 2000. S. 203–239.
32. Gallagher S. *How the body shapes the mind*. Oxford: Clarendon Press; 2005.
33. Paillard J. *Body schema and body image – a double dissociation*. In: Gantchev GN, Mori S, Massion J ed. *Motor control, today and tomorrow*. Sophia: Academic Publishing House; 1999. p. 197–214.
34. Moore JW, Fletcher PC. *Sense of agency in health and disease: A review of cue integration approaches*. Conscious Cogn. 2012; 21(1): 59–68.
35. Gallagher S. *Body schema and intentionality*. In: Bermudez JL, Marcel AJ, Eilan NM ed. *The body and the self*. Cambridge, MA: MIT Press; 1995. S. 225–244.
36. Stanghellini G, Ballerini M, Blasi S, Mancini M, Prezenza S, Raballo A et al. *The bodily self: A qualitative study of abnormal bodily phenomena in persons with schizophrenia*. Compr. Psychiatry 2014; 55(7): 1703–1711.
37. Graham KT, Martin-Iverson MT, Holmes NP, Jablensky A, Waters F. *Deficits in agency in schizophrenia, and additional deficits in body image, body schema, and internal timing, in passivity symptoms*. Front. Psychiatry 2014; 5: 126.
38. Tamming C, Shad M, Ghose S. *Neuropsychiatryczne aspekty schizofrenii*. In: Yudofsky C, Hales R ed. *Neuropsychiatria*. Wrocław: Elsevier; 2012. p. 451–468.
39. Ferri F, Frassinetti F, Mastrangelo F, Salone A, Ferro FM, Gallese V. *Bodily self and schizophrenia: The loss of implicit self-body knowledge*. Consc. Cogn. 2012; 21(3): 1365–1374.
40. Lee CU, Shenton ME, Salisbury DF, Kasai K, Onitsuka T, Dickey CC et al. *Fusiform gyrus volume reduction in first-episode schizophrenia: A magnetic resonance imaging study*. Arch. Gen. Psychiatry 2002; 59(9): 775–781.
41. Onitsuka T, Shenton ME, Salisbury DF, Dickey CC, Kasai K, Toner SK et al. *Middle and inferior temporal gyrus gray matter volume abnormalities in chronic schizophrenia: An MRI study*. Am. J. Psychiatry 2004; 161(9): 1603–1611.
42. Kasai K, Shenton ME, Salisbury DF, Hirayasu Y, Lee CU, Ciszewski AA et al. *Progressive decrease of left superior temporal gyrus gray matter volume in patients with first-episode schizophrenia*. Am. J. Psychiatry 2003; 160(1): 156–164.
43. Metzinger T. *The ego tunnel: The science of the mind and the myth of the self*. Nowy Jork: Basic Books; 2009.
44. Metzinger T. *Being no one: The self-model theory of subjectivity*. Cambridge, MA: MIT Press; 2004.
45. Sass LA. *Self-disturbance in schizophrenia: Hyperreflexivity and diminished self-affection*. In: Kircher T, David A. ed. *The self in neuroscience and psychiatry*. Cambridge: Cambridge University Press; 2003. p. 242–271.
46. Sass LA, Parnas J. *Schizophrenia, consciousness, and the self*. Schizophr. Bull. 2003; 29(3): 427–444.
47. Fuchs T, Schlimme JE. *Embodiment and psychopathology: A phenomenological perspective*. Curr. Opin. Psychiatry 2009; 22(6): 570–575.
48. Irrázaval L. *The Lived Body in Schizophrenia: Transition from Basic Self-Disorders to Full-Blown Psychosis*. Front. Psychiatry 2015; 6: 9.

49. Hunt HT, Chefurka CM. *A test of the psychedelic model of altered states of consciousness: The role of introspective sensitization in eliciting unusual subjective reports*. Arch. Gen. Psychiatry 1976; 33(7): 867.
50. Petitmengin C, Bitbol M. *Listening from within*. Journal of Consciousness Studies 2009; 16(10–12): 363–404.
51. Sass LA. *Self-disturbance and schizophrenia: Structure, specificity, pathogenesis (Current issues, New directions)*. Schizophr. Res. 2014; 152(1): 5–11.
52. Thakkar KN, Nichols HS, McIntosh LG, Park S. *Disturbances in body ownership in schizophrenia: Evidence from the rubber hand illusion and case study of a spontaneous out-of-body experience*. PLoS One 2011; 6(10): e27089.
53. Peled A, Pressman A, Geva AB, Modai I. *Somatosensory evoked potentials during a rubber hand illusion in schizophrenia*. Schizophr. Res. 2003; 64(2): 157–163.
54. Vercammen A, Knegeting H, den Boer JA, Liemburg EJ, Aleman A. *Auditory hallucinations in schizophrenia are associated with reduced functional connectivity of the temporo-parietal area*. Biol. Psychiatry 2010; 67(10): 912–918.
55. Maher B. *Delusional thinking and perceptual disorder*. J. Individ. Psychol. 1974; 30(1): 98–113.
56. Clark A. *Whatever next? Predictive brains, situated agents, and the future of cognitive science*. Behav. Brain Sci. 2013; 36(3): 181–204.
57. Clark A. *Surfing uncertainty: Prediction, action, and the embodied mind*. Oxford: Oxford University Press; 2016.
58. Friston K. *The free-energy principle: A rough guide to the brain?* Trends Cogn. Sci. 2009; 13(7): 293–301.
59. Friston K. *The free-energy principle: a unified brain theory?* Nat. Rev. Neurosci. 2010; 11(2): 127–138.
60. Hohwy J. *The predictive mind*. Oxford: Oxford University Press; 2013.
61. Seth AK. *Interoceptive inference, emotion, and the embodied self*. Trends Cogn. Sci. 2013; 17(11): 565–573.
62. Seth AK, Suzuki K, Critchley HD. *An interoceptive predictive coding model of conscious presence*. Front Psychol. 2011; 2: 395.
63. Vilares I, Kording K. *Bayesian models: The structure of the world, uncertainty, behavior, and the brain*. Ann. N. Y. Acad. Sci. 2011; 1224(1): 22–39.
64. Brown H, Friston K, Bestmann S. *Active inference, attention, and motor preparation*. Front. Psychol. 2011; 2: 218.
65. Voss M, Moore JW, Hauser M, Gallinat J, Heinz A, Haggard P. *Altered awareness of action in schizophrenia: A specific deficit in predicting action consequences*. Brain 2010; 133(10): 3104–3112.
66. Sereno AB, Holzman PS. *Antisaccades and smooth pursuit eye movements in schizophrenia*. Biol. Psychiatry 1995; 37(6): 394–401.
67. Ford JM, Mathalon DH. *Anticipating the future: Automatic prediction failures in schizophrenia*. Int. J. Psychophysiol. 2012; 83(2): 232–239.
68. Shergill SS, Samson G, Bays PM, Frith CD, Wolpert DM. *Evidence for sensory prediction deficits in schizophrenia*. Am. J. Psychiatry 2005; 162(12): 2384–2386.
69. Feinberg I. *Efference copy and corollary discharge: Implications for thinking and its disorders*. Schizophr. Bull. 1978; 4(4): 636–640.
70. Frith C. *The self in action: Lessons from delusions of control*. Conscious. Cogn. 2005; 14(4): 752–770.

71. Synofzik M, Thier P, Leube DT, Schlotterbeck P, Lindner A. *Misattributions of agency in schizophrenia are based on imprecise predictions about the sensory consequences of one's actions.* Brain 2010; 133(Pt. 1): 262–271.
72. Kubicki M, Mccarley R, Westin C, Park H, Maier S, Kikinis R, Shenton ME. *A review of diffusion tensor imaging studies in schizophrenia.* J. Psychiatr. Res. 2007; 41(1–2): 15–30.
73. Mechelli A, Allen P, Williams SCR, Brammer MJ, McGuire PK. *Misattribution of speech and impaired connectivity in patients with auditory verbal hallucinations.* Human Brain Mapping 2007; 28(11): 1213–1222.
74. Fisher DJ, Labelle A, Knott VJ. *Alterations of mismatch negativity (MMN) in schizophrenia patients with auditory hallucinations experiencing acute exacerbation of illness.* Schizophr. Bull. 2012; 139(1–3): 237–245.
75. Rentzsch J, Shen C, Jockers-Scherübl MC, Gallinat J, Neuhaus AH. *Auditory mismatch negativity and repetition suppression deficits in schizophrenia explained by irregular computation of prediction error.* Plos One 2015; 10(5): e0126775.
76. Corlett PR, Honey GD, Krystal JH, Fletcher PC. *Glutamatergic model psychoses: Prediction error, learning, and inference.* Neuropsychopharmacol. 2011; 36(1): 294–315.
77. Kapur S. *Psychosis as a state of aberrant salience: A framework linking biology, phenomenology, and pharmacology in schizophrenia.* Am. J. Psychiatry 2003; 160(1): 13–23.
78. Waelti P, Dickinson A, Schultz W. *Dopamine responses comply with basic assumptions of formal learning theory.* Nature 2001; 412(6842): 43–48.
79. Averbeck BB, Evans S, Chouhan V, Bristow E, Shergill SS. *Probabilistic learning and inference in schizophrenia.* Schizophr. Res. 2011; 127(1–3): 115–122.
80. Vaitl D, Lipp O, Bauer U, Schüler G, Stark R, Zimmermann M, Kirsch P. *Latent inhibition and schizophrenia: Pavlovian conditioning of autonomic responses.* Schizophr. Res. 2002; 55(1–2): 147–158.
81. Nelson B, Whitford TJ, Lavoie S, Sass LA. *What are the neurocognitive correlates of basic self-disturbance in schizophrenia? Integrating phenomenology and neurocognition. Part 1 (Source monitoring deficits).* Schizophr. Res. 2014; 152(1): 12–19.
82. Gradin VB, Kumar P, Waiter G, Ahearn T, Stickle C, Milders M et al. *Expected value and prediction error abnormalities in depression and schizophrenia.* Brain 2011; 134(Pt. 6): 1751–1764.
83. Škodlar B, Henriksen MG, Sass LA, Nelson B, Parnas J. *Cognitive-behavioral therapy for schizophrenia: A critical evaluation of its theoretical framework from a clinical-phenomenological perspective.* Psychopathol. 2012; 46(4): 249–265.
84. Tai S, Turkington D. *The evolution of cognitive behavior therapy for schizophrenia: Current practice and recent developments.* Schizophr. Bull. 2009; 35(5): 865–873.
85. Nordgaard J, Sass LA, Parnas J. *The psychiatric interview: validity, structure, and subjectivity.* Eur. Arch. Psychiatry Clin. Neurosci. 2013; 263(4): 353–364.
86. Janusz B, Bobrzyński J, Furgał M, Barbaro de B, Gdowska K. *O potrzebie badań jakościowych w psychiatrii.* Psychiatr. Pol. 2010; 44(1): 5–11.
87. Röhricht F, Priebe S. *Effect of body-oriented psychological therapy on negative symptoms in schizophrenia: A randomized controlled trial.* Psychol. Med. 2006; 36(5): 669–678.
88. Röhricht F, Papadopoulos N, Suzuki I, Priebe S. *Ego-pathology, body experience, and body psychotherapy in chronic schizophrenia.* Psychol. Psychother. 2009; 82(Pt. 1): 19–30.
89. Jeannerod M. *Neural simulation of action: A unifying mechanism for motor cognition.* Neuroimage 2001; 14(1 Pt. 2): 103–109.

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90. Röhrich F, Papadopoulos N, Holden S, Clarke T, Priebe S. *Therapeutic processes and clinical outcomes of body psychotherapy in chronic schizophrenia – An open clinical trial*. Art. Psychother. 2011; 38(3): 196–203.

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