Self and schizophrenia: current status and diagnostic implications

The notion of self-disorders in schizophrenia emerged in contemporary psychiatry at the beginning of this millennium[1]. It was considered an unorthodox novelty, and neither the DSM-IV nor the DSM-5 contains a reference to disordered self in the schizophrenia spectrum.

However, that notion was historically co-constitutive of the concept of schizophrenia. Bleuler[2] listed experiential "ego-disorders" among the fundamental symptoms of schizophrenia and reported patients complaining of being only "reflections of themselves", unable to "catch up with themselves" or having "lost their individual self". All classic texts on schizophrenia contain a reference to disordered self[1]. The concept of "disintegration", widely used in psychiatry and psychoanalysis, makes only sense in the presence of some notion of self that is at stake.

The DSM-III glossary of terms linked disturbance in the "sense of self" to schizophrenia, and the ICD-9 definition of schizophrenia referred to disturbance of fundamental features of personality (e.g., uniqueness and autonomy), whereas in the ICD-10 the term "personality" was removed. The disappearance of "disordered self" was perhaps linked to the simplification of fundamental symptoms into the so-called "four A's" (autism, ambivalence, association and affect disorders) and a difficulty with conceptualizing the notion of autism.

What kind of self is disordered in the schizophrenia spectrum conditions? It is useful to follow a distinction of contemporary philosophy of mind and phenomenology between the so-called "narrative self" and the "core self".

The narrative self refers to features which characterize and individualize a person and which easily lend themselves to linguistic self-description (e.g., "I have a tendency to act impulsively") and descriptions from the third-person perspective ("she is acting impulsively"). These features comprise biographical, characterological and cognitive characteristics and are heavily dependent on language and memory.

The notion of core self refers, instead, to the first-person perspective which is an intrinsic structural feature of all experience and which provides us with an immediate or pre-reflective sense of subjectivity and self-familiarity as an "I-me-myself". This can be extended to comprise a sense of temporal persistency, self-coincidence, substantiality-embodiment, and demarcation. All these features are never an object of ordinary experience, but provide a first-person structure for the narrative level of experiencing oneself as, for example, "impulsive" or "suspicious". However, these features are experientially accessible when we reflect upon the way in which our experience articulates itself.

We have previously proposed that the essential feature of schizophrenia spectrum disorders is a disturbance of the core self in its immediate relation to the world[3]. It is important to emphasize that we are not talking about a lack or a deficit (as in "too much or too little") but rather of an instability or *dis*-order[4]. This basic disturbance of self-world relation is the generative component in the Gestalt of autism[3], which "appears nowhere else in this particular fashion"[2] and which imbues schizophrenia with an air of un-understandability[5].

Empirical studies[1] from different groups and on different samples clearly show a selective hyper-aggregation of disorders of core self in schizophrenia and schizotypal disorder as opposed to bipolar disorder and other psychiatric disorders. Self-disorders typically begin in childhood or adolescence, are observed in populations at ultra-high-risk for psychosis, and predict subsequent schizophrenia spectrum outcome[1].

Two studies have demonstrated temporal persistence and similarity of patterns of self-disorders five years apart[6]. Self-disorders are unrelated to IQ[1], and preliminary data fail to show any substantial correlation with neurocognitive disorders. In sum, empirical research seems to corroborate Bleuler's idea that these phenomena are to be considered as trait features of the schizophrenia spectrum.

This structural instability of self-world relation is the background for the development of psychotic symptoms, which in their form contain an imprint of disordered selfhood[4,7]. For example, the characteristic auditory verbal hallucinations are often a progression from the state of anonymization and spatialization of thinking, where the patient's "I think" becomes transformed into "it thinks in me". The phenomenon of thought broadcasting is a flamboyant expression of the loss of sense of demarcation. And the characteristic double-bookkeeping involves a construction of a private world or alternative ontological framework[7,8].

The recognition of self-disorders entails important nosological consequences. Currently, we see a decrease in the diagnosis of disorganized schizophrenia, a very uncommon use of the schizotypal diagnosis and an increasing frequency in the use of the borderline personality disorder diagnosis. This latter diagnosis is over-inclusive and often applied to patients which would in the ICD-9 be diagnosed with a schizophrenia spectrum condition[9]. It seems to us that it is nearly impossible to conceptualize a core psychopathological difference between the notion of schizotypy and the contemporary clinical application of the DSM-5 diagnosis of borderline personality disorder[9].

This diagnostic confusion is multidetermined, but mostly due to a very tolerant use of the ninth borderline disorder criterion ("transient, stress-related paranoid ideation or severe dissociative symptoms") and the unclarity of the borderline disorder criteria of "identity disturbance" and "feelings of emptiness". "Feelings of emptiness" are undefined, and the identity disturbance criterion, although apparently referring to the narrative level of selfhood, is not sufficiently differentiated from disturbances of core self[10]. We find it crucial to sharpen the distinction between schizophrenia spectrum psychopathology (involving disturbances of both core and narrative self) and disorders of personality (which do not involve structural disturbances of the core self).

Contemporary classification is striving for simplicity and reliability, with much research being performed by for-the-purpose-trained lay interviewers. The disappointment with the slow progress of pathogenetic research encourages critical voices advocating abandonment of phenotypic categories altogether. However, the story of self-disorder research may inspire us to reconsider the phenotypic classification with a more refined psychopathological approach.

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The schizophrenia spectrum anhedonia paradox

Anhedonia, defined as a diminished capacity to experience pleasure, has been considered a core symptom of schizophrenia since the earliest descriptions of the disorder. It is longitudinally stable and associated with a range of poor clinical outcomes[1]. Unfortunately, interventions targeting this symptom have produced minimal benefits, and no drug has received US Food and Drug Administration's approval for this indication.

Limited progress in effectively treating anhedonia results in part from a lack of conceptual clarity regarding the nature of the symptom. Evidence for anhedonia in schizophrenia has primarily come from data obtained via clinical interviews, which indicate that the majority of those diagnosed with that disorder are anhedonic. Clinicians have long assumed that such self-reports indicate that individuals with schizophrenia have a diminished capacity to experience positive emotion. However, laboratory-based studies provide evidence that contradicts this notion, indicating that schizophrenia patients self-report as much positive emotion as healthy controls in response to pleasant stimuli[2] and show intact neurophysiological responses in key reward circuits during receipt of rewarding outcomes[3].

It has been argued that this apparent discrepancy can be resolved if one examines the anchors and probes used in negative symptom interviews[4]. Upon careful inspection, it is clear that what interviewers are rating is the frequency of reward-seeking behavior, rather than the extent to which patients enjoy pleasurable activities when engaged in them. Based on this evidence, as well as on results from ecological momentary assessment studies, the field has gradually shifted away from the view that schizophrenia patients have a reduced hedonic capacity. Rather, schizophrenia appears to be associated with a behavioral deficit characterized by a reduction in the frequency of pleasurable activity[4].

The disconnect between behavior and hedonic capacity has been termed the "liking-wanting anhedonia paradox", and spurred research attempting to determine why apparently normal hedonic responses do not translate into motivated behaviors aimed at obtaining rewards in schizophrenia. Several conceptual models attempted to answer this question, proposing that impairments in various aspects of reward processing (e.g., reinforcement learning, value representation, effort-cost computation, reward anticipation), that rely on cortico-striatal circuitry, prevent fully intact hedonic responses from influencing decision-making processes needed to guide action selection and initiate motivated behavior[4]. These models have received significant empirical support and are beginning to influence the development of treatments targeting these underlying mechanisms.

However, there is a second "anhedonia paradox" that has emerged over recent years. We refer to this as the "schizophrenia spectrum anhedonia paradox". Specifically, there is growing evidence that, although patients with schizophrenia have intact hedonic capacity[4], individuals with schizotypy and youth in the prodromal phase of illness do not. People with schizotypy self-report less positive emotion in response to pleasant stimuli than healthy controls and show reduced neurophysiological response during the receipt of reward outcomes[5]. Youth at clinical high risk for psychosis also have diminished neurophysiological and self-reported responses to pleasant stimuli[6]. Since schizophrenia is a more severe form of psychopathology in nearly every conceivable way, this apparent discrepancy is paradoxical: why would the less severe forms of pathology show deficits in hedonic capacity, whereas the more severe form does not? Below we discuss some plausible explanations, hoping to promote future studies aimed at resolving this paradox.

A first possibility is that mood and anxiety symptoms produce diminished hedonic response in schizotypy and clinical high risk youth more than in schizophrenia. Consistent with this notion is evidence indicating that youth at clinical high risk for psychosis and those with schizotypy have higher rates of comorbid depression and anxiety than people with schizophrenia, and that greater severity of depression and anxiety is associated with reduced hedonic response in those individuals[6].