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Phenomenology and neurobiology of self disorder in schizophrenia: Secondary factors☆

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ABSTRACT

Schizophrenia is a diverse and varying syndrome that defies most attempts at classification and pathogenetic explanation. This is the second of two articles offering a comprehensive model meant to integrate an understanding of schizophrenia-related forms of subjectivity, especially anomalous core-self experience (disturbed ipseity), with neurocognitive and neurodevelopmental findings. Previously we discussed the primary or foundational role of disturbed intermodal perceptual integration (“perceptual dys-integration”). Here we discuss phenomenological alterations that can be considered secondary in a pathogenetic sense—whether as consequential products downstream from a more originary disruption, or as defensive reactions involving quasi-intentional or even volitional compensations to the more primary disruptions. These include secondary forms of: 1, hyperreflexivity, 2, diminished self-presence (self-affection), and 3, disturbed “grip” or “hold” on the cognitive/perceptual field of awareness.

We consider complementary relations between these secondary abnormal experiences while also considering their temporal relationships and pathogenetic intertwining with the more primary phenomenological alterations discussed previously, all in relation to the neurodevelopmental model. The secondary phenomena can be understood as highly variable factors involving overall orientations or attitudes toward experience; they have some affinities with experiences of meditation, introspectionism, and depersonalization defense. Also, they seem likely to become more pronounced during adolescence as a result of new cognitive capacities related to development of the prefrontal lobes, especially attention allocation, executive functions, abstraction, and meta-awareness. Heterogeneity in these secondary alterations might help explain much of the clinical diversity in schizophrenia, both between patients and within individual patients over time—without however losing sight of key underlying commonalities.

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1. Introduction

Schizophrenia is a heterogeneous syndrome in which current categorical diagnostic assessment and associated pathogenetic research has shown severe limitations (Carpenter et al., 2013; Insel, 2009, 2010; Silveira et al., 2012; Silverstein et al., 2014; Tandon, 2012; Tandon et al., 2009). Some experts view recent research as a failed enterprise, and blame the very concept of schizophrenia for this state of affairs (Carpenter et al., 2013; Tandon, 2012). But as mentioned in the previous article, an alternative explanation for this failure may be the prevailing propensity for superficial description and conceptualization of mental or experiential symptoms that, paradoxically, remain the primary basis for current psychiatric nosology (Andreasen, 2007; De Leon, 2013; Kendler, 2009; Marková and Berrios, 2009, 2012; Parnas et al.,

2013; Stanghellini, 2009). Here we continue our project of offering a complex, neuro-phenomenological model that attempts to integrate an understanding of schizophrenic subjectivities with neurocognitive and neurodevelopmental findings. In the previous article we discussed the primary or foundational factors; here we discuss the secondary ones.

Phenomenology, the study of lived experience, can complement the clinical panorama by describing and helping to explain the subjective dimension of mental diseases in more precise terms (Sass, 2010; Sass et al., 2011). It is important to stress that phenomenology can suggest explanatory hypotheses relevant to both synchronic and diachronic issues: i.e., to complementary relationships or simultaneous interactions between aspects of experience at a given phase or point in time, as well as to progressive appearance, over time, of different subjective experiences (Sass, 2014a; Sass et al., 2011). It is encouraging that multiple findings associate abnormal experience described by phenomenology with neurobiological correlates in diverse mental syndromes or diseases, including schizophrenia (Fletcher and Frith, 2008; Lutz and Thompson, 2003; Postmes et al., 2014; Varela, 1996).

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Disruption or dissociation of the self has historically been described as a central psychopathological feature of schizophrenia (Sass, 2001). In the contemporary self-disorder model, the primary disturbance in schizophrenia is specifically ascribed to disturbance of *core* or *minimal self*, also termed *ipseity*—which refers to the most basic sense of self-hood, that is, to the subjective sense of existing as a subject of experience and agent of action (Sass, 2014b; Sass and Parnas, 2003). In the previous article, we discussed three interrelated aspects of ipseity disturbance—primary disrupted “grip” or “hold” on the world, primary (or “operative”) (Merleau-Ponty, 2012, p. lxxxii) hyperreflexivity, and primary diminished self-presence (a.k.a. diminished self-affection: the term “affection” refers here not to liking, but to a process of being *affected* by something) (Sass, 2014b; Sass et al., 2011). We considered their possible biological correlates, in particular the relevance of disrupted sensorimotor functions and disrupted intermodal perceptual integration (what we term “perceptual dys-integration”) found in early and premorbid stages of patients with schizophrenia or at risk for the illness. These sensorimotor and other cross-modal functions, which play a paramount role in the acquisition of other basic abilities during structuring of nuclear self (Parnas et al., 1996, p. 19; Piaget and Inhelder, 1969; Postmes et al., 2014; Rochat, 2009; Stern, 2000), have been found to be compromised even in eight-months-old offspring of parents with schizophrenia (Gamma et al., 2014). This suggests that disruption of cross-modal functions may have a pathogenetically primary status, appearing earlier in time, and perhaps in a more passive or automatic fashion, than do other abnormal mental phenomena requiring more advanced cognitive functions. It is well known, of course, that all diseases—psychiatric or non-psychiatric—involve complex combinations of instigating factors or vulnerabilities together with various kinds of organismic reactions to these more primary factors.

In this article we discuss phenomenological alterations in schizophrenia that can be considered secondary in a pathogenetic sense—whether as consequential products downstream from a more ordinary disruption, or as defensive reactions involving quasi-intentional or even volitional compensations or reactions to the more primary disruptions discussed in our first article. It is noteworthy that these secondary changes often involve more active or defensive responses on the patient's part; and that, as Marková and Berrios (2009) suggest, mental experiences involving forms or structures of an individual's subjectivity seem especially unstable or susceptible to fluctuation in their clinical presentation.

We shall argue that heterogeneity in the nature and overall impact of these secondary alterations might help explain much of the clinical diversity in schizophrenia, both between patients and within individual patients over time—without however losing sight of some underlying commonalities; these commonalities are inherent in the shared presence of ipseity or core-self disturbance of one kind or another. In particular we will consider certain complementary relations between these various secondary abnormal experiences, postulating possible

neurobiological correlates while also considering their temporal relations with the neurodevelopmental model and with the primary phenomenological alterations discussed in the first article. An articulated understanding of interrelated phenomenological aspects, coordinated with more complex models of diverse neurocognitive factors, offers an encompassing model able to grasp this enigmatic illness in its unitary as well as diverse dimensions. See Table 1 for a listing of the various types of ipseity disturbance at issue, both primary and secondary, together with indications of associated neurocognitive abnormalities as well as of certain non-schizophrenic analogues discussed below.

2. Reflective hyperreflexivity

“Hyperreflexivity” refers to a kind of exaggerated self-consciousness, a tendency for focal, objectifying, or alienating attention to be directed toward processes and phenomena that would normally be experienced as part of oneself, i.e., that would normally be “inhabited” (in the sense of being tacitly lived), but now come to be targeted as focal objects of attention. It involves a rendering-explicit—and thus a transformation—of what would normally be implicit dimensions of experience (Sass, 1992, 2014b; Sass et al., 2011). Two types of hyperreflexivity can be distinguished, one primary, the other more secondary in a pathogenetic sense. *Operative* (or primary) hyperreflexivity was discussed in the preceding article; it refers to *automatic* and *non-volitional* emergence or popping-out of phenomena (e.g., kinesthetic sensations, inner speech) that would normally remain in the tacit background of awareness, but are now experienced in an objectified and alienated manner (Sass et al., 2011). *Reflective* hyperreflexivity (also termed *hyper-reflectivity*) refers to attentional processes of a more intentional or volitional, perhaps quasi-volitional sort. Here the subject turns attention toward what would normally be but tacitly experienced or presupposed aspects of himself or of the background context of his thinking or perceiving, with this occurring in response to, or as an attempt to control or understand, the experiential mutations being undergone.

Despite its largely defensive motivation, this directing of attention in reflective hyperreflexivity may have the paradoxical effect of *increasing* rather than *diminishing* the abnormal experiences of world and self, and also the patient's associated distress, due to the potentially alienating and fragmenting effect of focal attention itself (Sass, 2003). One schizophrenia patient, e.g., stated, “my downfall was insight... Too much insight can be very dangerous, because you can tear your mind apart” (Sass, 1992, p. 337). As indicated, hyper-reflection may be closely related to the intentional or personal levels of experience; as such, it may be bound up with personal orientation and values, which can involve quasi-willful forms of antagonism, alienation, and idiosyncratic attitudes (Sass, 2007a; Stanghellini and Ballerini, 2007; Stanghellini et al., 2013).

Both the elicitation and the consequences of such hyper-reflective modes can be understood in light of neurocognitive models.

Table 1

Types of ipseity disturbance (=disturbances of core or minimal self), with postulated neurocognitive correlates and non-schizophrenia analogues discussed in this article.

	Phenomenological abnormality	Neurocognitive factors	Non-schizophrenia analogues
Primary factors	Primary disturbed grip Operative (or primary) hyperreflexivity Primary diminished self-presence (a.k.a. diminished self-affection)	Disturbed intermodal perceptual integration including motoric, proprioceptive, kinesthetic factors (“perceptual dys-integration”)	
Secondary factors	Reflective (or secondary) hyperreflexivity (a.k.a. hyper-reflectivity)	Altered salience of interoceptive stimuli and subsequent defensive allocation of attentional resources toward these stimuli (dysregulation of Salience Network)	Introspectionism
	Secondary diminished self-presence	Hyperactivation of the Default-Mode Network (DMN) with hypoactivation of Central Executive Network and diminished emotional processing of external stimuli	Depersonalization disorder, meditation
	Secondary disturbed grip	Hypoactivity of Central Executive Network, and hyperactivity of Default-Mode Network (DMN), both associated with dysregulation of Salience Network (Same as above)	Introspectionism; depersonalization disorder, meditation

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