

DOI: 10.1515/cpp-2017-0015

Towards integrating phenomenology and neurocognition: Possible neurocognitive correlates of basic self-disturbance in schizophrenia

Barnaby Nelson^{1,2} A,E,F, Louis A. Sass³ A,E,F¹Orygen, The National Centre of Excellence in Youth Mental Health, Melbourne, Australia²Centre for Youth Mental Health, University of Melbourne, Melbourne, Australia³Department of Clinical Psychology, Graduate School of Applied and Professional Psychology, Rutgers University, Piscataway, New Jersey, USA

Abstract

Phenomenological research indicates that disturbance of the basic sense of self may be a core phenotypic marker of schizophrenia spectrum disorders. Basic self-disturbance refers to disruption of the sense of ownership of experience and agency of action and is associated with a variety of anomalous subjective experiences. Little is known about the neurocognitive correlates of basic self-disturbance. In this paper, we review recent phenomenological and neurocognitive research and point to a convergence of these approaches around the concept of self-disturbance. Specifically, we propose that subjective anomalies associated with basic self-disturbance may be associated with: 1. source monitoring deficits, which may contribute particularly to disturbances of “ownership” and “mineness” (the phenomenological notion of *presence*) and 2. aberrant salience, and associated disturbances of memory, prediction, and attention processes, which may contribute to hyper-reflexivity, disturbed “grip” or “hold” on the perceptual and conceptual field, and disturbances of intuitive social understanding (“common sense”). These two streams of research are reviewed in turn before considering ways forward in integrative models, particularly regarding the role of early neurodevelopmental disturbances, primary versus secondary disturbances, and the state versus trait nature of such pathology. Empirical studies are required in a variety of populations in order to test the proposed associations between phenomenological and neurocognitive aspects of self-disturbance in schizophrenia. An integration of findings across the phenomenological and neurocognitive domains would represent a significant advance in the understanding of schizophrenia and possibly enhance early identification and intervention strategies.

Keywords: schizophrenia; psychosis; phenomenology; self; neurocognition

Introduction

The diagnostic boundaries and pathogenetic mechanisms of schizophrenia and related disorders remain obscure. The lack of integration across different research “domains” (phenomenological, psychological, neurocognitive, neurobiological, genetic) may contribute to this situation. Integration across these domains may help researchers move towards unifying principles and themes in the study of schizophrenia, which would ultimately be beneficial for diagnostic practice, treatments, and early identification—as well, of course, as for general pathogenetic understanding. Along these lines, there have been several recent attempts at a theoretical integration of phenomenological and neurocognitive research in schizophrenia [1-5]. We have argued in previous work that there is a convergence of recent findings across these different research domains linked around the concept of disturbance of the basic (a.k.a. “minimal” or “core”) self. This concept may therefore play a useful unifying or integrative role. The purpose of the current piece is to provide a summary of this proposal and to alert readers to current empirical studies currently underway testing the proposed model.

The phenomenological domain: Basic self-disturbance in schizophrenia

A body of phenomenological research, combining clinical exploration, empirical studies and philosophical perspectives, indicates that a disturbance of the basic sense of self is at the clinical core of the schizophrenia spectrum and may therefore function as a phenotypic trait marker of these disorders, including vulnerability not (yet) expressed at diagnosable levels [6-19]. What exactly is being referred to “basic” sense of self? The term refers to a *tacit, pre-reflective* level of selfhood. All subjective experience has a first-person quality and there is a tacit or implicit “ownership” of experience, sometimes referred to as “for-me-ness” or “mineness” [20-22]. This aspect of selfhood stands in contrast to less implicit aspects of selfhood, such as the reflective self (the self as an object of reflection) or the narrative self (social identity) [7].

A range of anomalous subjective experiences indicative of some sort of disturbance of instability in the basic sense of self have been identified in schizophrenia spectrum conditions. These include disturbances in the stream of consciousness, sense of presence, bodily experience

('corporeality'), self-other/self-world demarcation ('transitivism'), and existential reorientation. Although it is useful for descriptive purposes to categorise these subjective disturbances in this way [17], they should not be thought of clearly distinct from each other, but are in fact intimately interrelated and likely to be expressive of a Gestalt or structural shift in subjectivity [7, 11, 17, 23]. They can be briefly summarised as follows:

Stream of consciousness – The onset of schizophrenia is characterised by an emerging "gap" between the self and mental content. The sense of implicit ownership of mental content is disrupted. Patients may describe, for example, thoughts as adopting an almost autonomous or anonymous identity, as pressured or as having a physical, object-like, or acoustic quality. These experiences can be the forerunner to first-rank psychotic symptoms, such as thought insertion and thought broadcasting.

Presence – A prominent feature of normal human experience consists of being engaged and absorbed in activity amongst a world of (animate and inanimate) objects (as Heidegger explicated in his concept of being-in-the-world). The "mineness" of this absorbed, engaged interaction with the world provides a form of implicit self-awareness (it is *I* who is having these experiences), which is sometimes referred to as *presence* or as *self-affection*. This can be disturbed in the schizophrenia spectrum, with the self seeming to stand alienated from itself, rather than being implicitly and constantly present. Various forms of depersonalisation and derealisation, a sense of inner void, and a reduced ability to be affected or influenced by events or other people are reflective of this disturbance.

Corporeality – The schizophrenia spectrum can be characterised by a transformation in the experience of the "lived body" [24]: experiential distance emerges between the self and bodily experience; one no longer "inhabits" one's body but, rather, experiences it as an object. This is expressed in many of the bodily basic symptoms, such as cenesthesias and impaired bodily sensations [25].

Self-demarcation – There can be diminution, permeability, or in extreme cases, a loss of self-world boundaries. Examples include confusion of boundaries between self and others (e.g., losing sense of whether thoughts, feelings, etc. originated in oneself or another person), a sense of passivity in relation to the world and others, or experiencing the physical presence and contact of others as threatening.

Existential reorientation – The early psychotic period can be characterised by a preoccupation with philosophical, supernatural, and metaphysical themes [18, 26]. The rupture in "normal" self-experience would seem to motivate this preoccupation. In cognitive psychology terms,

the person is attempting to accommodate, explain, justify, or explore his anomalous experiences.

The basic self-disturbance model of schizophrenia has gained substantial empirical support (for review see [12, 27]). The model certainly does not deny that "the self" or self-experience is disturbed in many other psychiatric disorders, but proposes that it is a different *level* or *type* of selfhood that is disturbed in most of these conditions [7, 28, 29]. The congruence of first person patient accounts of schizophrenia with the basic self-disturbance model lends support to this overall ipseity-disturbance approach [30, 31].

The precise nature of what is specific to schizophrenia is somewhat controversial, however. Sass and colleagues [32] and Madeira and colleagues [33, 34] have found evidence of a prominence of EASE items (the main measure of basic self-disturbance [35]) in depersonalization disorder and in panic disorder respectively—as well as in certain intense forms of introspectionism [36]; and they argue that certain aspects of the basic-self alteration captured in the EASE (though not all) may result from dissociative and depersonalization processes, perhaps largely defensive in nature, that can also be found outside the schizophrenia spectrum. Other aspects of basic-self disturbance—perhaps rooted in a more foundational disturbance in the integration of perceptual systems, may be absent from these disorders outside the schizophrenia spectrum.

There are three complementary psychological features or processes thought to generate basic self-disturbance [8, 9]:

1. *Hyper-reflexivity*. Hyper-reflexivity refers to an inflated self-consciousness and heightened awareness of aspects of one's experience that are normally tacit, implicit and sit quietly "in the background". Examples include awareness of the act of breathing or sensations while walking, or awareness of the "inner speech" that mediates our thinking. Normal consciousness of course involves fleeting awareness of such background features. However, when this form of awareness becomes exaggerated or difficult to control it can have the effect of objectifying these implicit features of experience, forcing them to be experienced *as if* they were external objects, and transforming them in various reifying ways (as when mental images take on a quality of "phantom concreteness" [37]). Hyper-reflexivity consists of both *reflective* hyper-reflexivity (or hyper-reflectivity), which refers to an exaggerated intellectual or reflective process, and *operative* hyper-reflexivity, which refers to acts of awareness that are not intellectual in nature, that may not occur voluntarily, as in the case of kinaesthetic experiences "popping" into awareness, and that are probably more primary in a pathogenetic sense [14].

2. *Diminished self-presence.* Diminished self-presence refers to a weakened sense of existing as a *subject* of awareness. Whereas hyperreflexivity captures how something normally tacit becomes focal and explicit, diminished self-presence emphasises the ‘reverse’ side of this same process, i.e. how what once was tacit is no longer inhabited as a medium of taken-for-granted selfhood [8].

3. *Disturbed “hold” on conceptual/perceptual field of awareness.* A standard feature of consciousness consists of being a *point of orientation/perspective* that is directed by needs, desires, and motivations. The person is situated in a world that *matters* to her in a range of ways – where objects/people appear as valuable, functional, interesting, enticing, threatening and so on [38]. We are always *in relationship* to the world/others (what phenomenologists call the “intentionality” or object-oriented quality of consciousness). This provides the “centre of experiential gravity” [10], organising and holding together the diffuse elements of conscious experience. When this is disrupted through the processes of hyper-reflexivity and diminished self-presence, there is *necessarily* a disruption of a person’s “hold” on their conceptual or perceptual field of awareness [8, 39]. The usual tacit-focal structuring of experience and the organised nature of thought and perception (the sharpness or stability with which figures are perceived or meanings emerge against a background context) is undermined. This can naturally lead to the sense of perplexity and withdrawal so common in schizophrenia.

Disorders of basic self are thought to be foundational factors in the development of virtually all types of schizophrenia symptoms. In one recent article, e.g., Sass and Byrom [40] discuss ipseity disturbance’s specific relevance for understanding the distinctively “bizarre” nature of delusions in schizophrenia (and elsewhere they speculate [41] about associated neurobiological factors that may contribute to self disorder as well as to the formation and maintenance of such delusions).

Possible neurocognitive correlates of basic self-disturbance

While the phenomenology of basic self-disturbance in schizophrenia has been well characterised, its pathogenesis is not yet clear, from a developmental, neurocognitive or neurobiological point of view [42]. To our knowledge, there have been three empirical studies of neurocognitive disturbance in relation to basic self-disturbance. Haug and colleagues [43] examined basic self-disturbance and neurocognition in a group of first episode schizophrenia patients. The neurocognitive variables included measures of psychomotor speed, working memory, executive- and memory functions. Few associa-

tions were found between basic self-disturbance and neurocognitive impairment, with impaired verbal memory emerging as the single correlate. The authors speculated that: i. the general lack of association between basic self-disturbance and neurocognitive deficits could be due to the fact that they represent different basic features of schizophrenia, ii. verbal memory deficits may be linked to basic self-disturbance as they pick up on an individual’s ability to comprehend, direct, remember and reason about his or her thoughts, functions that are likely to be disturbed by basic self-disturbance. Nordgaard and colleagues [44] and Comparelli and colleagues [45] also reported no correlation between basic self-disturbance and neurocognitive measures in a first episode schizophrenia sample and a clinical high risk for psychosis sample, respectively. Given the overlap between the basic self disturbance concept and ‘basic symptoms’ in schizophrenia, it is also noteworthy that two studies found no correlation between the basic symptoms and neurocognitive performance [46, 47].

It is possible that the lack of association between neurocognitive measures and basic self-disturbance may be due to the fact that studies have tended to use standard, reasonably broad neurocognitive measures. The neurocognitive disturbances underpinning basic self-disturbance may be more specific and subtle and require different tests to identify them. The “traditional” neurocognitive measures used in psychosis research were, after all, devised for assessing acquired brain injury and intellectual disability [48], which may limit how useful they are in detecting the specific disturbances which may be present in schizophrenia. Indeed, studies using these “standard” neurocognitive models tend to show no correlation or modest correlation between severity of cognitive impairment and symptoms, especially positive symptom severity [49, 50]. Similarly, these measures are not strong predictors of future onset of psychotic disorder in clinical high risk cohorts [51], possibly indicating that they do not index the specific neurocognitive disturbances at play in schizophrenia.

We have previously argued that there are at least two streams of neurocognitive research in psychosis that are consistent the phenomenology of basic self-disturbance and which may therefore be more useful for integrative models of schizophrenia spectrum vulnerability. Broadly speaking, they consist of: 1. source monitoring deficits and 2. aberrant salience (memory-attention disturbances). Recent work on these approaches harks back to the neurocognitive work of the psychologists Frith [52, 53], Gray [54-56] and Hemsley [57-60], experimentalists whose views were considered in a phenomenological perspective, and in relationship to self-disturbance in particular, by Sass [9]. These two streams of research will be reviewed in turn before

considering ways forward in integrative models, particularly regarding the role of early neurodevelopmental disturbances, primary versus secondary disturbances, and the state versus trait nature of such pathology.

Source monitoring deficits

A central tenet of a number of neurocognitive models of schizophrenia is the notion that there is a failure to discriminate between sources of stimuli. These so-called “source monitoring deficits” can be divided into reality monitoring (failure to discriminate between internal and external sources) and self-monitoring (failure to discriminate between two external sources or between two internal sources), both of which have been found to be present in schizophrenia [61]. To date, our integration of phenomenological and neurocognitive findings has focused on the former (reality monitoring).

The notion of “prediction errors” has played an important role in attempts to understand the mechanisms that may underlie source monitoring deficits [52, 54, 57, 59, 60, 62, 63]. These approaches propose that positive psychotic symptoms result from the *predictions* we make and the extent to which these predictions are fulfilled. In other words, predicted outcomes are continually *compared* with actual outcomes [64]. A key difference between self- and other-generated stimuli is that the former are more predictable and controllable than the latter. When stimuli are predictable (i.e., self-generated) they are “dampened” in perception (e.g., tickling yourself compared to being tickled by another person). This attenuation is thought to be due, at least in part, to the effects of efference copies, which are neural signals that accompany self-generated actions and which often act to predict and suppress the sensory activity arising from these actions [65, 66]. (The neural representation of the sensory feedback that is predicted on the basis of the efference copy is known as a “corollary discharge” [67]). That is, the prediction based on this signal is used in an unconscious, automatic fashion to “dampen” the sensation normally associated with the proprioceptive input and the associated activation of sensory areas of the brain. It is proposed that when these self-generated stimuli are not effectively “dampened” in perception they might be experienced *as if* they were external in origin [63]. Consistent with this proposal, experimental studies show that patients with auditory hallucinations (generally, hearing voices) and passivity phenomena experience self-produced stimuli in the same way as other-produced stimuli, in contrast to psychiatric patients without these symptoms and to normal controls. Specifically, patients with these symptoms experience the sensation of being touched on the palm of the hand as just

as intense, tickly and pleasant when self-produced as when the identical stimulus is produced by another person, in contrast to patients without these symptoms, who experience the self-produced stimulus as less intense, tickly, and pleasant than the other-produced stimulus [68]. Persistent inaccurate sensory experiences may lead to unusual and inaccurate interpretations and beliefs, which may crystallise as psychotic symptoms. For example, the repeated *perception* of one’s own movements as being unpredictable may lead to the *belief* that one’s movements are being controlled by an external force and, consequently, delusions of motor control. This is an example of abnormal “bottom-up” processing (i.e., sensory experiences leading to belief formation).

A range of studies have yielded data consistent with the view that in schizophrenia there is some form of disconnection between a (self-generated) motor act and the sensory consequences of that act. Ford and colleagues reported a reduction in functional connectivity in schizophrenia patients between brain regions that *initiate* an action and regions involved in the *perception* of the sensory consequences of that action [69–71]. Specifically, they found a disruption in the connectivity between frontal brain regions activated during speech production and temporal brain regions involved in auditory processing, concluding that a disruption of the connectivity between these brain regions may lead to a *mismatch* between the expected auditory consequences and the actual auditory experience of talking (i.e., a prediction error) [69]. Interestingly, the disruption of functional connectivity was particularly pronounced in patients with auditory hallucinations. Similar findings emerged from a functional magnetic resonance imaging (fMRI) study of a sentence completion task in schizophrenia patients [72]. Compared with healthy controls, patients showed reduced connectivity between prefrontal and temporal regions and the degree of disconnection correlated with the severity of hallucinations (also see consistent findings from Breakspear et al [165], Fletcher et al [73, 74], and Ford et al [75]).

Evidence for impaired corollary discharge is not restricted to speech generation. Both Ford et al. [76] and Whitford et al. [77] observed that while healthy controls exhibited significantly smaller event-related potentials (ERPs) to self-initiated, button-press initiated sounds compared to identical, externally-initiated sounds, patients with schizophrenia did not exhibit this ERP suppression. This again suggests that schizophrenia patients fail to distinguish between internally-generated and externally-generated sensations, even when the association between action and sensation is not direct (e.g., sounds caused by willed speech) but is instead indirect and more

likely to be associatively learned (e.g., sounds generated by pressing a button).

Several other recent studies are consistent with the theme of these findings. Wilquin and Delevoeye [78] found that disturbed motor agency (i.e., the immediate experience of oneself as the cause of an action) differentiated ultra-high risk (UHR) for psychosis and first episode psychosis (FEP) patients from normal controls with greater sensitivity than a more classic neuropsychological test battery. Carhart-Harris and colleagues [79] measured the effects of psilocybin (a psychotomimetic pharmacological agent) on functional connectivity between the default mode network (DMN), which supports introspection, and the task-positive network (TPN), which supports externally-focused attention. Spontaneous activity in these networks is normally independent of each other. Psilocybin had the effect of increasing the functional connectivity between these networks and decreasing their orthogonality, indicating that increased DMN-TPN connectivity in psychosis may contribute to a breakdown in how distinct internally and externally focused states are. This may lead to difficulties in distinguishing between one's internal world and the external environment.

To summarise, impaired corollary discharges may prevent brain regions responsible for generating actions (or thoughts more generally, if thoughts are considered to be a special type of action [63]) from informing other brain regions that these thoughts/actions are self-generated. These thoughts/actions will therefore not be "predicted" events and will not be dampened in perception. This leads to difficulty distinguishing events that we control (self-generated acts) from those that occur independently of us in the external world (externally-generated acts). A recent model by Taylor [80] extends this approach (originally applied to motor action) to attention. He argues that the free deployment of attention grounds our sense of agency and ownership of experience ("corollary discharge of attention movement"), and that the reduction of this process accounts for schizophrenic self-disturbance.

Possible phenomenological correlates

How might the neurocognitive dysfunctions outlined above be expressed on a phenomenological level? We have argued that many of the anomalous subjective experiences that constitute disturbed basic selfhood are highly congruent with these neurocognitive disturbances (also see Taylor [80]). In the cognitive domain, one would expect a lack of "ownership" and automatic self-identification with mental content and an

associated sense of dissociation from one's thoughts. Given the difficulty in identifying whether stimuli are internal or external in origin, thoughts may adopt an object- or "thing"-like quality, i.e., to be experienced *as if* they were external objects. In concert, *external* objects or persons could come to seem "internal" or dreamlike. Both of these sets of experiences have been well described in the phenomenological literature (see above) as constituting an aspect of disturbed basic selfhood in schizophrenia. These anomalous experiences, possibly driven by source monitoring neurocognitive disturbances, may underlie and evolve into first rank psychotic symptoms such as auditory hallucinations and passivity phenomena (such as thought insertion or withdrawal) [7, 9].

Similarly, disturbances of self-other boundaries (or "transitivistic" phenomena [9]) as a feature of basic self-disturbance are congruent with source monitoring deficits. These phenomena include loss or permeability of the self-world boundary, including experiences of confusion about self-other boundaries (e.g., whether oneself or another person had a certain thought or experience, confusion about whether a reflected image is of oneself or another person, etc.), feeling as though one cannot affect one's surroundings, and that one is in a passive position in relation to the world. If the brain is not effectively "warning" itself that stimuli are self-generated then these malleable and porous boundaries between self and other (in cognitive, emotional and physical domains) is a likely result, as is a diminished grasp of the subtle interplay of cause and effect in the relationship between self and others/world.

Finally, the lack of automatic identification of experiences (thoughts, sensations, etc.) *as one's own* may prompt consequential or compensatory forms of hyper-reflexivity of a reflective sort. Such hyper-reflection might occur as an in-the-moment reaction (e.g., the sense that this thought did not originate in me leads to reflection on what its origins might be), but it may also become a more habitual act, as the exaggerated reflection comes to be ingrained. But at a more basic, "operative" or automatic level (see above), a form of hyper-reflexivity is perhaps *already* present, since the very experience of one's thought as foreign implies that it is attended to as an object rather than being "inhabited" implicitly as one's own. In other words, something normally implicit or tacit, experienced as the very medium of selfhood, has been turned into the object of focal and objectifying awareness [14].

Table 1 Proposed neurocognitive and phenomenological correlates indicative of basic self-disturbance in schizophrenia.

Basic Self Disturbance: Research Domains			
Neurocognitive		Phenomenological	
	Neural/Electrophysiological	Behavioural/Cognitive	
Source Monitoring Deficits	- Efference copy [65, 66] - Corollary discharge [67, 77, 80] - Reduced functional connectivity [70-75, 165] - Elevated "resting state" brain activity [166, 167]	- Endogenous-exogenous confusion [68, 78, 168] - Prediction errors [52, 54, 57, 59, 60, 62-64, 169]	- Diminished ownership of mental content [35, 170] - "Transitivistic" phenomena: self-other boundary confusion [9, 35] - Hyper-reflexivity [8, 9, 14]
Aberrant Sali-ence	- Latent inhibition [54, 87, 88] - Mismatch negativity [86, 116-119, 171]	- Attention and memory disturbances [48, 58-60, 84, 85, 172] - Failed suppression of attention to irrelevant stimuli [101] - Weakening of contextual constraint [59, 60, 92, 103-108, 110, 111, 113, 114] - Prediction errors [52, 54, 57, 59, 60, 62, 63]	- Hyper-reflexivity [8, 9, 14] - Rigidity and perplexity in interaction with world [9, 23, 35] - Disturbance of "common sense" [94, 135, 137] - Disturbed "grip" or "hold" on conceptual and perceptual field [8, 14] - Diminished perspectival abridgement [8, 9] - Diminished "affordance value" of objects [9]

Aberrant salience (memory-attention disturbances)

Attention and memory disturbances have been well documented in schizophrenia. A major theme in this work is the failed suppression of attention to familiar or irrelevant information/stimuli in the environment, leading to aberrant salience of objects and associations [82, 83] – or, to reverse the terminology, excessive attention to information that is highly familiar or irrelevant. A number of neurocognitive models and experimental paradigms have produced findings consistent with this view, including the memory-prediction model of cortical function [48, 84, 85], the salience dysregulation model based on dopamine system abnormalities [55, 57, 82], mismatch negativity reduction [86], latent inhibition [54, 87, 88], and Corlett's model of ketamine as a pharmacological model of psychosis [89, 90]. Also, Hemsley [58, 59, 91] and Sass [9] drew on findings regarding malfunction in the hippocampus-based "comparator" system in schizophrenia, proposing that this dysfunction may result in an automatic, hyperreflexive awareness that disrupts the tacit/focal structure essential to normal experience of basic selfhood. Below, we review the major common themes of these models and how they accord

with the phenomenological model of basic self-disturbance.

Keefe and colleagues [48, 84, 85] recently attempted to introduce an organising principle in studies of cognition in schizophrenia in the form of a "memory-prediction" model of cortical function, which has substantial affinities with the prediction errors models mentioned above, as well as with earlier models proposed by Hemsley [57, 58, 91] and Gray [55]. The "memory-prediction" model is based on the understanding that perceptual processes do not simply involve the reproduction of stimuli, but that they involve matching and integrating sensory input, which are often fragmented and partial, with "working models" of the world. These working models are memories that the brain has encoded based on consistent, repeated aspects of the environment. Working models are used to continuously make (automatic) predictions about what will occur or appear in the temporal flow of experience. This "top-down" process (implying an interpretation of sensory input by cognitive/sensory schemas) allows us to "fill the gaps" in fragmented or partial sensory input and to facilitate efficient

interaction with a complex and fluid environment. Keefe and Kraus [84] provide the example of a partially obscured road sign: "...Because of our past experiences with stop signs, we are not confused by deviations from the archetypal stop sign; even if the lower left corner of the sign is bent and the "OP" is obscured by a tree branch, we immediately recognise the symbol and step on the brake" (p.415). In this way, memory-prediction processes infuse familiar meaning into the world, endowing perception and interaction with the environment with a significant amount of ease and automaticity.

The hierarchical structure and column-like architecture of the cortex gives rise to these memory or learning-based predictions [84]. If we encounter stimuli that do not neatly fit our predictions based on previous experience (the memory component of the model), an area of cortex will relay details of these stimuli to higher cortical areas. The signal will keep graduating to higher cortical layer until a match is achieved. In the event of no match being achieved, a new mental representation of this stimulus will be introduced. Repetition of stimuli will make them become more familiar and their mental representations will be shifted to lower cortical areas, which allows higher areas to detect high-level or superordinate patterns. In this way, familiar stimuli (i.e., input that conforms to memory-based predictions) will be processed at lower cortical levels, allowing for efficient use of cortical resources.

The model proposes that in schizophrenia these memory-prediction processes are compromised in some way. There is disturbance of both "bottom-up" and "top-down" cortical processing, probably due to early and widespread disruption of neuronal circuitry [84]. Lower cortical levels do not provide enough perceptual detail for higher levels to establish consistent and stable representations, and higher levels do not provide enough context for lower cortical levels to interpret incoming stimuli. The consequences of this are: 1) slowed, more effortful processing of incoming information, 2) increased probability of arbitrary, internally generated interpretations of stimuli. This proposal is consistent with several earlier suggestions regarding schizophrenia: the Russian psychologist Polyakov spoke of disturbances of "probability prognosis" [9, 92] and Hemsley [58-60] described a "loosening of expectations" based on previous experience.

One significant feature of the memory-based "context" provided by higher cortical levels is that it has an inherently social nature. That is, an event or situation is likely to be interpreted in a particular way *based on the context of its social significance* (e.g., shaking one's head from side to side is likely to be interpreted quite differently depending on the cultural context). It follows that when contextual memory is weakened or disturbed in an individual, as in the case of schizophrenia, the interpretation

of events or situations is more likely to be unconventional, arbitrary and idiosyncratic. A brain-based disturbance of memory and attention processes (and associated aberrant salience) would thereby contribute to forms social disarticulation. The accumulation of inaccurate (but internally meaningful) perceptions may build upon one another into idiosyncratic and incorrect beliefs, distancing the person from common sense and consensual reality [93, 94], and even leading, in some instances, to a solipsistic orientation [9, chp 9] and development of delusions and hallucinations.

A range of empirical findings lend support to this model. Post-mortem studies show that brain tissue of people with schizophrenia has abnormal cerebral cortex architecture, characterised by decreased neuropil, decreased synaptic density and disarray of neuronal location, particularly in layers that sit between bottom-up signalling and top-down contextual predictions (layers II and III) [95]. Cortical thinning has also been observed in UHR patients who later develop psychotic disorder [96]. Keefe and colleagues [84] argue that while such cortical disruption may have a wide-ranging impact on cognition, memory-prediction processes may be particularly affected due to the specific cortical layers most disrupted. Deficits in smooth pursuit eye tracking observed in schizophrenia are largely due to impairments in predictive mechanisms [97-100]. The *improved* performance of schizophrenia patients compared to controls when tracking a target that changes direction unpredictably also suggests a weakening of prediction processes, i.e. that the person is less constrained by automatic predictions. Similarly, reduced mismatch negativity (MMN, see below) and P300 amplitude indicate deficits in physiological responses to unexpected stimuli, consistent with the notion of impairments in memory-based prediction processes. Morris and colleagues [101] found that schizophrenia patients showed increased attention to irrelevant (i.e., non-predictive) cues, assessed using a causal learning test, and that this tendency correlated with intensity of positive symptoms. Excessive attention to irrelevant cues may result in learning inaccurate and irrelevant causal associations, contributing to the emergence of positive psychotic symptoms. Studies of ketamine as a pharmacological model of psychotic symptoms have yielded similar findings [89]. Interestingly, the tendency to ascribe meaning where none is present, assessed using a white-noise task, has been found to predict onset of psychotic disorder in an at-risk population [102]. In this experiment, meaningless stimuli were endowed with potential significance, consistent with the memory-prediction model's notion of an increase in arbitrary, internally generated interpretations of stimuli (aberrant salience).

Schizophrenia patients also demonstrate difficulty identifying obscured or incomplete images of common ob-

jects [103, 104] and filling in missing gaps in speech [105], and are more hindered in identifying faces by superficial differences in the face images (e.g., lighting conditions and visual angle) compared to controls [106, 107]. In the same vein, schizophrenia patients are more likely to perceive the image of a concave face when presented using the Binocular Depth Inversion Test (BDIT), rather than automatically invert the image into a standard convex face, as healthy controls typically do [108]. Interestingly, scores on the BDIT correlate with severity of psychotic symptoms [109], which is often not the case with more standard tests of cognition. These findings are all consistent with the notion of a failure to make accurate “top-down” predictions based on contextual information (drawing on memory-based processes) and an over-reliance on lower level perceptual processing. It is interesting, then, that schizophrenia patients show a pattern of *superior* performance on several kinds of tasks in which standard expectations/predictions normally cause a decline in performance: 1, recognition of unlikely scenes shown tachistoscopically [92]; 2, a tendency to be more “logical” when logic requires the ignoring of practical context [110], although contrasting results have been reported by Nordgaard and colleagues [44]; 3, greater accuracy in discerning a concave mask, as described above, or in which attention to larger Gestalt factors might be misleading [111-113]; and 4, more accurate perception of speech when paired with video footage of incongruous facial movements (the McGurk effect) [114].

Findings from a range of other neurocognitive experimental paradigms are consistent with the memory-prediction model. An example of this is mismatch negativity (MMN), which refers to a change in brain activity in response to the occurrence of novel, unexpected stimuli, leading to an attentional shift [115, 116]. The most widely used experimental paradigm to elicit MMN is the oddball paradigm, which involves presenting unexpected, deviant (i.e., “oddball”) stimuli amongst an otherwise continuous stream of stimuli and measuring the resulting electrical brain activity using electroencephalography (EEG). Studies indicate that schizophrenia patients show a consistent decrease in the amplitude of the MMN in response to oddball stimuli [117, 118] and the MMN impairment is a good candidate as a predictor of the onset of psychosis in UHR patients [119]. These data relating to electrical brain activity indicate a reduced ability to discriminate relevant from irrelevant stimuli in schizophrenia. Interestingly, the MMN abnormality has been found to correlate with compromised global functioning [120-122] and social cognition [123] in schizophrenia patients, possibly indicating that this basic information processing disturbance may have a downstream effect on a person’s ability to function in everyday life.

Another relevant construct is latent inhibition [54, 87, 88]. Latent inhibition is a neurologically-based concept that describes the phenomenon of reduced attention to stimuli upon repeated exposure. It is based on the notion of a gating mechanism that allows organisms with complex nervous systems to stop responding to stimuli with no apparent motivational value [124]. Reduced latent inhibition (i.e., *non-reduction* of attention to stimuli upon repeated exposure) has been found to characterise the schizophrenia spectrum, including both schizotypy [125-128] and schizophrenia, particularly during acute episodes [87, 129]. The integrity of sensory gating processes can be measured with EEG during an auditory dual-click paradigm. The amplitude of the positive potential occurring approximately 50ms post-stimulus (i.e., the P50) is normally decreased when the second click is presented. This decrease in amplitude is thought to reflect a sensory gating mechanism that functions to protect against information overload. Abnormal auditory gating (i.e., less of a decrease in the amplitude of the second P50) has been observed in schizophrenia [130, 131]. This indicates that patients cannot inhibit, or “gate,” irrelevant sensory input, leading to an overload of information reaching the brain and contributing to perceptual disturbances and attention deficits in schizophrenia.

The aberrant salience of experiences and information arising from these attention and memory disturbances may lead to distorted interpretations of events. For example, cognitive associations may form between irrelevant and unrelated stimuli, which may consolidate into delusional thinking. In other words, the neurocognitive disturbances may have a bottom-up influence on cognitive processes of “meaning-making” in schizophrenia. The neurocognitive disturbances may also be associated with various disturbances of *subjective experience reflective of basic self-disturbance*, such as hyper-reflexivity, disturbed perception of affordances, perspectival abridgement, disturbed “grip” or “hold” on the perceptual and cognitive world, sense of perplexity, and lack of common sense [9, 14], as described below.

Possible phenomenological correlates

The tacit process of “filling in the gaps” and “matching” events/stimuli to memory-based predictions endows consciousness with a fluidity and automaticity and facilitates ease of interaction with the environment. However, if there is a breakdown in this system, as seems to be the case in schizophrenia, one is likely to direct attention to relatively fragmentary stimuli that normally go unnoticed (aspects of the environment, but also of self-experience, such as cenesthetic sensations) rather than larger, more meaningful Gestalts. This, in turn, is likely to elicit ever more intense and ever more fragmenting forms of focal attention. If a new situation does not readily “match” its

predicted state then one must pay more attention to the constituent aspects of that situation. To return to the (rather simplistic but nevertheless useful) “STOP” sign example, if I do not automatically mentally complete the “ST” with “OP” (to form the word “STOP”) then I must pay more attention to the stimulus “ST” and put extra *effort* into determining its meaning. It is possible that this may have a cascading effect on my attentional and reflective processes. That is, my initial difficulty comprehending the meaning of “ST” and the extra attention I therefore give to this stimulus might lead to me becoming excessively aware of my own attentional processes (“Why am I paying this so much attention?”, “Why aren’t others paying this more attention?” etc.). This process is captured in the phenomenological concept of hyper-reflexivity – that is, excessive attention being paid to aspects of experience that are normally tacit and remain in the “background” of awareness. Hyper-reflexivity is one of the central features of disturbed basic selfhood.

This is further illustrated in a clinical case study that we have previously published [132]. The patient complained of all aspects of his functioning and action requiring a lot more thought and effort than was normally the case (premorbidly), prompting him to “give up” and remain immobile and unresponsive: “I lost my automatic things – they became conscious. Everything I did was a conscious effort - for example, picking up a glass.” His thoughts were slowed down and disconnected, as though they lacked a meaningful flow. While this is a form of hyper-reflexivity, the process of attention being drawn to irrelevant or background stimuli is also likely to be associated with *operative* hyper-reflexivity (see the distinction made above) – that is, acts of awareness that are not intellectual, effortful or voluntary but are automatic in nature, such as sensations suddenly “popping” into awareness, as represented by some of the cenesthetic “basic symptoms” of schizophrenia [133, 134].

Here we see that the excessive attention to tacit, background aspects of experience and stimuli (the tacit becoming focal), possibly driven by the compromised memory-prediction processes described above, can disrupt smooth interaction with and habitation in the world. Rather than automatically “knowing” how to respond to a particular situation or what sense to make of particular events or contexts, the person may be “caught up” or “stuck” on some element of their perceptual or cognitive world (and, indeed, even the *acts* of perception and cognition themselves) that would normally be incorporated smoothly into the stream of consciousness. These extra “processing” requirements may have the effect of introducing an awkward rigidity, slowness and sense of perplexity into the person’s interactions with the world, which have all been described in the phenomenological literature [9, 23, 81].

The (temporal or visual) context in which an event occurs or a stimulus appears tends to anchor its significance and directs how it is most usefully interpreted. The neurocognitive disturbances described above show how an individual stimulus (internal or external) might “come loose” from its context. These neurocognitive disturbances may lead a person to pay attention to “irrelevant” stimuli (i.e., stimuli that do not seem to be providing any “new” information) *as if* they were “new” and of potential significance – and possibly less attention to “relevant” stimuli. That is, the appropriate (and functionally useful) salience of events and objects is disrupted [59, 60, 82, 83]. This accords well with many of the subjective disturbances in schizophrenia described in the basic self-disturbance model. As described above, an element of the model is a disturbed “hold” on the conceptual or perceptual field. There is an alteration in field of awareness, a disruption of the focus or salience with which objects and meanings emerge against a background context [14]. To draw on terminology from perception research, the normal figure-ground relationship is disrupted. Individual thoughts or perceptions can seem to be atomistic, disconnected from other thoughts or perceptions, and may take on a thing- or object-like quality. Phenomenological literature [8, 9] also describes various types of perspectival shift or drift that undermine the possibility of blocking out alternative perspectives (referred to as “perspectival abridgement”), which can contribute to states of perplexity or ambivalence: “He is ‘snowed under with options’; e.g. he thinks that he probably ought to become a vegetarian even though he loves meat. Such considerations lead him into ‘doubleness’ and ‘silly, blind alleys’” [81], see EASE item 1.9). This is precisely what one would expect from a disruption of the “grounding” influence of background context (in its most inclusive sense) on how an individual stimulus (internal or external) is experienced.

Similarly, the disturbances of “common sense” in schizophrenia described in the phenomenological literature may be seen as arising from a weakening of the “grounding” effect of context. Blankenburg [94, 135] described an essential feature of schizophrenia as a loss or lack of “common sense”, a term used to refer to the ability “to see things in the proper perspective, to distinguish between *what is relevant and irrelevant, likely and improbable*, which is a more elementary ability than to distinguish between what is true and what is false” [136]. To see things “in the proper perspective” assumes being able to automatically grasp the significance of an event, act or utterance given its social context. The disruption of contextual understanding, as described in the neurocognitive models above, will undermine the ability to distinguish the relevant from the irrelevant, with possibly increased significance being assigned to irrelevant stimuli.

This will disrupt an individual's attunement to others and the world, a characteristic feature of schizophrenia [137, 138] that has been formulated in the cognitive psychology literature as disturbances of social cognition [139, 140]. The erosion of the "orienting" influence of context is consistent with philosophical work suggesting that disruption in the foundational, non-representational, non-rule-governed, or dispositional structure of everyday understanding (the "Bedrock" or "Background") underlies the formation and maintenance of delusions [141].

These neurocognitive models also have relevance to the loss of objects' "affordance" value that has been described in phenomenological literature [9], and which contributes to a disturbed self-world relationship. As described above, these models emphasise the breakdown in contextual perception/understanding of stimuli. An important feature of the self-world relationship is the *functional value* of objects in the world, how they might be *used* by us, which is referred to as their affordance value [142, 143]. Our motivations, needs and desires structure and direct how we view or approach objects, e.g. if I am lost in a forest at night and feel vulnerable I am likely to see a metal pole I am carrying as a potential weapon. This is the pole's affordance value for me in this context. In many ways, this "directed" relationship to the world (the phenomenological concept of *intentionality* [144]) provides context and organises our multifarious experience of the world. The attenuation or loss of affordance value and its replacement with a sense of an object's crude physical presence (or "mere being" [9, 145]) has been reported in psychotic states. A hammer, for example, is no longer a tool but is perceived simply as an object. In this state, the context-dependent relationship with objects recedes and "mere being" comes to the fore. This experience can be marked by a sense of disgust or horror at the arbitrariness of things, as well as a sense of wonder [9, 146].

While the above discussion focuses on aspects of self-disturbance that involve disruption in the experience of and ability to interact with others/the environment, we note that the neurocognitive concept of aberrant salience also accords with more "private" or "non-interactive" (and possibly more fundamental) aspects of self-disturbance. Aberrant salience involves focal attention being directed to the familiar or "irrelevant" and bringing the "background" or the implicit to the fore, a process captured in the concept of hyper-reflexivity (see above). This disrupts the naturalness and automaticity of various aspects of self-experience [8, 9]. If aspects of self-experience (e.g., bodily experience such as kinaesthetic sensations or cognitive processes such as "inner speech" underlying thinking) come to be noticed (i.e., enter the focus of awareness) then this can have the effect of distancing or alienating oneself from these components of selfhood and the nor-

mally unified nature of basic self-experience can become fragmented. This can result in these aspects of consciousness no longer being lived or "inhabited" aspects of selfhood (i.e. no longer *constituting* selfhood) but becoming alienated processes, acts or experiences [8, 9]. Hemsley [58] argues that this disruption in the automatic ability to appropriately "integrate" sensory input/information with stored material (aberrant salience) can disrupt the sense of continuity and consistency in sense of self, contributing to a gradually developing instability in personal identity (also see EASE, domain 2 items relating to identity confusion and instability [81]).

The "loosening" of stimuli from the usual significance bestowed on them by context (or the "openness" to stimuli regardless of their past significance) might in fact confer some benefits in addition to, or instead of, disturbing an individual's functioning [58, 147]. For example, it might be associated with increased creativity or formal logical rationality (see Nelson and colleagues [1, 2] for further discussion).

In a recent article on the phenomenology and neurobiology of delusions, Sass and Byrom [41] discuss the factors we have been considering above—source-monitoring abnormalities and abnormal experiences of hyper-salience—but also go on to take up a somewhat different, additional possibility: namely, experiences of *hypo*-salience that may be associated with an "anything goes" orientation to ongoing experience—that is, with a failure to be surprised even by what is unexpected or unusual. And this, as they note, may be correlated, on the neurobiological plane, with the failure to inhibit the "default-mode network" that has been observed in schizophrenia—a dis-inhibition that would be the neural correlate of a free-floating, ruminative, non-practical mode of experience that, if dominant in everyday life, would be conducive to accepting interpretations of the world (including "bizarre" beliefs) that most persons might quickly reject as illogical or highly implausible

Conclusions and Future Directions

Although progress has been made in understanding phenomenological and neurocognitive disturbances in schizophrenia, these domains of enquiry have tended to remain separate from each and have been poorly integrated to date. This "splintering" of research domains in schizophrenia research not only applies to these particular domains but has been a general challenge for the field [148-150]. We have argued here and elsewhere [1, 2, 151] that there is a convergence of models and empirical findings from these domains around the concept of disturbance of the basic sense of self. We have described how the neurocognitive disturbances of source monitoring (with particular focus on reality monitoring) and aberrant sali-

ence accord with many of the anomalies of subjective experience described in the phenomenological literature. These neurocognitive disturbances may constitute the neural correlates or cause of an abnormal sense of basic selfhood (and, by implication, of experience of others and the world), which may, in turn, evolve into frank psychotic symptoms. However, the distortions in the basic sense of self are arguably more primary (and often more disturbing to the person [30, 31, 152]) than the resultant psychotic symptoms themselves. The neurocognitive models discussed above may pick up more directly on the specific disturbances at play in schizophrenia than more standard neurocognitive models of schizophrenia based on measures of cognitive functioning developed mainly in the fields of general intelligence and brain injury research. The point of this approach is not to “reduce” phenomenological disturbances to neurocognitive or neurological disturbances, but rather to signal the convergences in these domains of research and how they might be integrated in “cross-domain” models of schizophrenia and guide future empirical research.

A number of directions for future research emerge from the integration of phenomenological and neurocognitive research outlined above. First, we would expect neurocognitive measures of source monitoring deficits and aberrant salience and phenomenological measures of basic self-disturbance to be positively correlated in schizophrenia spectrum samples and to possibly assist in the identification of ‘at risk’ individuals at highest risk of progressing to schizophrenia. We are currently conducting an empirical study to test these hypotheses. Second, it is not clear at this stage exactly what the relationship between source monitoring deficits and aberrant salience is. There are indications, as alluded to above, that prediction errors may generate both source monitoring deficits and aberrant salience [89, 153] (see Nelson and colleagues [1] for further discussion of this point). The focus of the above discussion has also been on the reality monitoring aspect (i.e., internal-external confusion) of source monitoring deficits, rather than on self-monitoring deficits (i.e. external-external or internal-internal confusion). How exactly do the self-monitoring deficits fit with the model outlined above? Are these neurocognitive and phenomenological disturbances of a trait or state nature and, if they do indeed fluctuate in intensity and nature, what intrapsychic and contextual factors are they reactive to? This latter question would of course have implications for treatment approaches.

The developmental factors that may contribute to the phenomenological and neurocognitive disturbances reviewed above are also not well understood at present. Borda and Sass [3] and Sass and Borda [4] propose that early perceptual and motoric disturbances that affect

perceptual organization, especially intermodal or multi-sensory perceptual integration [5, 154, 155] (and perhaps especially between exteroceptive and interoceptive forms of perception, e.g., visual and kinaesthetic/proprioceptive) may lead to a “perceptual disintegration” that undermines the ability to apprehend the world in a holistic, vital, contextually grounded fashion, or to fully identify with or experience the unity of one’s own body or thinking. This “primary factor” may generate an early disruption or diminishment of basic self and of the sense of existing in a coherent world. These neurodevelopmental disturbances of basic selfhood (via hyperreflexivity, diminished self-presence, disturbed “hold” on the conceptual/perceptual field) – perhaps together with experiences of trauma or intense stress -- may give rise to an array of secondary abnormal experiences. These secondary experiences may be consequential products downstream from a more primary disruption, but may also involve defensive reactions involving either quasi-intentional or even volitional compensations for the more primary disruptions, or else responses to various forms of challenge from the external world (stress, trauma). The secondary phenomena can be understood as highly variable factors involving overall orientations or attitudes toward experience, which has bearing on the trait versus state question raised above.

Certain forms of ipseity disturbance—involving hyperreflexivity or diminished presence—might, then, have a more primary, basic, perhaps affliction-like quality, and be prominent from early in life, whereas other forms—more reactive, reflective, and sometimes even quasi-volitional in nature—might develop later as *sequelae* or defensive reactions to these more foundational problems or in response to difficult environmental circumstances. Though “secondary” in the sense of occurring later in time (perhaps only slightly later, or perhaps especially during adolescence), these latter forms might nevertheless be equally *necessary* for the development of a characteristically *schizophrenic* type of illness.

Sass has argued that the secondary forms of self disorder may, in many cases, be closely akin to the forms of dissociation and depersonalization that occur in certain disorders *outside* the schizophrenia spectrum, such as Depersonalization Disorder, Post-Traumatic Stress Disorder, or Panic Disorder [156, 157] (and that can also be observed in various examples of the avant-garde literature and art of modernism and postmodernism, where forms of hyper-reflexivity and alienation are prominent [9]). Such a model of schizophrenia is consistent with the widely accepted findings, from the last decade or two, that demonstrate the role of prolonged stress, trauma, social defeat, and cultural collision in the pathogenesis of schizophrenia (see, e.g., [158, 159]). It may help to account as well for a related finding:

the extensive overlapping between schizophrenic and dissociative symptoms [160].

It is entirely possible that schizophrenia patients might differ according to the relative contribution of these two sets of factors. Primary factors may be more decisive in so-called “poor premorbid” patients or cases with early or “insidious” onset, who are often dominated by negative symptoms and “nonparanoid” features. By contrast, secondary factors may be more crucial for cases with more acute onset and better premorbid functioning, and with more prominent positive and perhaps especially dissociation-like symptoms, as well as for acute exacerbations later in the course of illness. It may be, then, that there are two pathways to psychosis: one more endogenous, the other more trauma-driven, but with both present—to different degrees, and interacting—in many or most cases [161, 162].

It is important to remember, in any case, that there is no reason to make the reductionist assumption—all too common in neurobiological theorizing—that the causal direction must always be from brain to mind rather than the reverse (or even that causality is the best formulation of mind-brain relationships). Although this general cautionary point concerning the mind/body problem applies even on the level of the primary factors, it seems especially obvious with regard to secondary factors, given that these latter can involve motivated (and psychologically understandable) defensive tendencies that may nevertheless entrain, and also express, changes on the neurobiological or neurocognitive level.

There is, in fact, an urgent need for research that explores the variable nature of various neurocognitive and neurobiological trends. Such research would manipulate and investigate the effects of changing mental orientation or stance (e.g., of adopting a withdrawn/introspective vs practical focus, of scrutiny vs a spontaneous orientation) in both schizophrenic and normal subjects—in order to determine the extent to which such phenomena as salience dysregulation, perceptual fragmentation, and disturbed sense of agency (or their milder equivalents), together with their neural correlates, might alter in accord with such changes. To what extent, for instance, would either the experience or neural correlates of auditory verbal hallucinations be mimicked (albeit to a lesser degree) by a person who withdraws and focuses attention on his own inner thinking? To what extent would perception of the concave mask [163] or perceptual fragmentation [164] be increased under conditions of introverted withdrawal, either in normal or in schizophrenic subjects? Such research would contribute to developing a more sophisticated model of the pathogenesis, maintenance, and variability of schizophrenic symptoms, and would also have implications for the development of psychological and psychotherapeutic interventions.

These are all important avenues to pursue in further research into integrative pathogenetic models of schizophrenia and related conditions.

Acknowledgments

BN was supported by a NARSAD Independent Investigator Award from the Brain & Behavior Research Foundation (BBRF).

Conflict of interest

The authors have declared no conflict of interest.

References:

- 1 Nelson B, Whitford TJ, Lavoie S, Sass LA: What are the neurocognitive correlates of basic self-disturbance in schizophrenia?: Integrating phenomenology and neurocognition: Part 2 (aberrant salience). *Schizophr Res* 2014;152:20-27.
- 2 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;152:12-19.
- 3 Borda JP, Sass LA: Phenomenology and neurobiology of self disorder in schizophrenia: Primary factors. *Schizophr Res* 2015;169:464-473.
- 4 Sass LA, Borda JP: Phenomenology and neurobiology of self disorder in schizophrenia: Secondary factors. *Schizophr Res* 2015;169:474-482.
- 5 Postmes L, Sno HN, Goedhart S, van der Stel J, Heering HD, de Haan L: Schizophrenia as a self-disorder due to perceptual incoherence. *Schizophr Res* 2014;152:41-50.
- 6 Parnas J, Handest P, Jansson L, Saebye D: Anomalous subjective experience among first-admitted schizophrenia spectrum patients: Empirical investigation.[see comment]. *Psychopathology* 2005;38:259-267.
- 7 Parnas J: Self and schizophrenia: A phenomenological perspective; in Kircher T, David A (eds): *The self in neuroscience and psychiatry*. Cambridge, Cambridge University Press, 2003, pp 127-141.
- 8 Sass LA, Parnas J: Schizophrenia, consciousness, and the self. *Schizophrenia Bulletin* 2003;29:427-444.
- 9 Sass LA: *Madness and modernism: Insanity in the light of modern art, literature, and thought*. Cambridge, MA, USA, Harvard University Press, 1992. Revised edition: Oxford University Press, 2017.
- 10 Nelson B, Yung AR, Bechdolf A, McGorry PD: The phenomenological critique and self-disturbance: Implications for ultra-high risk (“prodrome”) research. *Schizophr Bull* 2008;34:381-392.
- 11 Parnas J: The core gestalt of schizophrenia. *World psychiatry: official journal of the World Psychiatric Association* 2012;11:67-69.
- 12 Parnas J, Henriksen MG: Disordered self in the schizophrenia spectrum: A clinical and research perspective. *Harvard review of psychiatry* 2014;22:251-265.
- 13 Nelson B, Parnas J, Sass LA: Disturbance of minimal self (ipseity) in schizophrenia: Clarification and current status. *Schizophrenia Bulletin* 2014;40:479-482.

- 14 Sass LA, Parnas J: Explaining schizophrenia: The relevance of phenomenology; in Chung MC, Fulford KWM, Graham G (eds): *Reconceiving schizophrenia*. New York, Oxford University Press, 2007, pp 63-96.
- 15 Parnas J, Jansson L, Sass LA, Handest P: Self-experience in the prodromal phases of schizophrenia: A pilot study of first-admissions. *Neurology Psychiatry and Brain Research* 1998;6:97-106.
- 16 Parnas J: The self and intentionality in the pre-psychotic stages of schizophrenia: A phenomenological study; in Zahavi D (ed) *Exploring the self: Philosophical and psychopathological perspectives on self-experience*. Amsterdam, John Benjamins, 2000, pp 115-148.
- 17 Parnas J, Møller P, Kircher T, Thalbitzer J, Jansson L, Handest P, & Zahavi D: Ease: Examination of anomalous self-experience. *Psychopathology* 2005;38:236-258.
- 18 Møller P, Husby R: The initial prodrome in schizophrenia: Searching for naturalistic core dimensions of experience and behavior. *Schizophrenia Bulletin* 2000;26:217-232.
- 19 Sass LA, Parnas J, Zahavi D: Phenomenological psychopathology and schizophrenia: Contemporary approaches and misunderstandings. *Philosophy, Psychiatry & Psychology* 2011;18:1-23.
- 20 Gallagher S (ed) *The Oxford handbook of the self*, Oxford, Oxford University Press, 2011.
- 21 Zahavi D: Phenomenology of self; in Kircher T, David A (eds): *The self in neuroscience and psychiatry*. Cambridge, Cambridge University Press, 2003, pp 56-75.
- 22 Zahavi D: Consciousness and (minimal) selfhood: Getting clearer on for-me-ness and mineness; in Kriegel U (ed) *The Oxford handbook of the philosophy of consciousness*. Oxford, Oxford University Press, 2017
- 23 Parnas J: A disappearing heritage: The clinical core of schizophrenia. *Schizophrenia bulletin* 2011;37:1121-1130.
- 24 Merleau-Ponty M: *The primacy of perception*. Northern University Press, 1964.
- 25 Klosterkötter J, Hellmich M, Steinmeyer EM, Schultze-Lutter F: Diagnosing schizophrenia in the initial prodromal phase. *Archives of General Psychiatry* 2001;58:158-164.
- 26 Yung AR, McGorry PD: The initial prodrome in psychosis: Descriptive and qualitative aspects. *Australian and New Zealand Journal of Psychiatry* 1996;30:587-599.
- 27 Nelson B, Raballo A: Basic self-disturbance in the schizophrenia spectrum: Taking stock and moving forward. *Psychopathology* 2015;48:301-309.
- 28 Lysaker PH, Lysaker JT: Schizophrenia and alterations in self-experience: A comparison of 6 perspectives. *Schizophrenia bulletin* 2010;36:331-340.
- 29 Nelson B, Thompson A, Chanen AM, Amminger GP, Yung AR: Is basic self-disturbance in ultra high risk for psychosis ('prodromal') patients associated with borderline personality pathology? *Early Intervention in Psychiatry* 2013;In press
- 30 Kean C: Silencing the self: Schizophrenia as a self-disturbance. *Schizophrenia bulletin* 2009;35:1034-1036.
- 31 Saks ER: *The center cannot hold: My journey through madness*. New York, Hyperion, 2007.
- 32 Sass LA, Pienkos E, Nelson B: Introspection and schizophrenia: A comparative investigation of anomalous self experiences. *Consciousness and Cognition* 2013;22:853-867.
- 33 Madeira L, Carmenates S, Costa C, Linhares L, Stanghellini G, Figueira ML, Sass L: Basic self-disturbances beyond schizophrenia: Discrepancies and affinities in panic disorder - an empirical clinical study. *Psychopathology* 2017;50:157-168.
- 34 Parnas J, Nordgaard J, Henriksen MG: Panic, self-disorder, and ease research: Methodological considerations. *Psychopathology* 2017;50:169-170.
- 35 Parnas J, Møller P, Kircher T, Thalbitzer J, Jansson L, Handest P, Zahavi D: Ease: Examination of anomalous self-experience. *Psychopathology* 2005;38:236-258.
- 36 Sass L, Pienkos E, Nelson B: Introspection and schizophrenia: A comparative investigation of anomalous self experiences. *Conscious Cogn* 2013;22:853-867.
- 37 Sass LA: *The paradoxes of delusion: Wittgenstein, schreber, and the schizophrenic mind*. New York, Cornell University Press, 1994.
- 38 Ratcliffe M, Broome M: Existential phenomenology, psychiatric illness and the death of possibilities; in Crowell S (ed) *Cambridge companion to existentialism*. Cambridge, 2012, pp 361-382.
- 39 Merleau-Ponty M: *The phenomenology of perception*. London, Routledge, 1962.
- 40 Sass LA, Byrom G: Self-disturbance and the bizarre: On incomprehensibility in schizophrenic delusions. *Psychopathology* 2015;48:293-300.
- 41 Sass L, Byrom G: Phenomenological and neurocognitive perspectives on delusions: A critical overview. *World Psychiatry* 2015;14:164-173.
- 42 Maj M: The self and schizophrenia: Some open issues. *World psychiatry : official journal of the World Psychiatric Association* 2012;11:65-66.
- 43 Haug E, Oie M, Melle I, Andreassen OA, Raballo A, Bratlien U, Lien L, Møller P: The association between self-disorders and neurocognitive dysfunction in schizophrenia. *Schizophrenia research* 2012;135:79-83.
- 44 Nordgaard J, Revsbech R, Henriksen MG: Self-disorders, neurocognition and rationality in schizophrenia: A preliminary study. *Psychopathology* 2015;48:310-316.
- 45 Comparelli A, Corigliano V, De Carolis A, Pucci D, Angelone M, Di Pietro S, Kotzalidis GD, Terzariol L, Manni L, Trisolini A, Girardi P: Anomalous self-experiences and their relationship with symptoms, neuro-cognition, and functioning in at-risk adolescents and young adults. *Compr Psychiatry* 2016;65:44-49.
- 46 Schultze-Lutter F, Ruhrmann S, Pickar H, von Reventlow HG, Daumann B, Brockhaus-Dumke A, Klosterkötter J, Pukrop R: Relationship between subjective and objective cognitive function in the early and late prodrome. *Br J Psychiatry Suppl* 2007;51:s43-51.
- 47 Zanello A, Huguelet P: Relationship between subjective cognitive symptoms and frontal executive abilities in chronic schizophrenic outpatients. *Psychopathology* 2001;34:153-158.
- 48 Keefe RS, Kraus MS, Krishnan RR: Failures in learning-dependent predictive perception as the key cognitive vulnerability to psychosis in schizophrenia. *Neuropsychopharmacology : official publication of the American College of Neuropsychopharmacology* 2011;36:367-368.
- 49 Keefe RS, Bilder RM, Harvey PD, Davis SM, Palmer BW, Gold JM, Meltzer HY, Green MF, Miller DD, Canive JM, Adler LW, Marder SR, Swartz M, Rosenheck R, Perkins DO, Walker TM, Stroup TS, McEvoy JP, Lieberman JA: Baseline neurocognitive deficits in the catie schizophrenia trial. *Neuropsychopharmacology : official publication of the American College of Neuropsychopharmacology* 2006;31:2033-2046.
- 50 Heinrichs RW: *In search of madness: Schizophrenia and neuroscience*. Oxford, Oxford University Press, 2001.
- 51 Lin A, Yung AR, Nelson B, Brewer WJ, Riley R, Simmons MB, Pantelis C, Wood SJ: Neurocognitive predictors of transition to

- psychosis: Medium to long-term findings from a sample at ultra-high risk for psychosis. *Psychological Medicine* 2013;In press
- 52 Frith CD: The cognitive neuropsychology of schizophrenia. Hove, Lawrence Erlbaum, 1992.
 - 53 Frith CD, Done DJ: Towards a neuropsychology of schizophrenia. *British Journal of Psychiatry* 1988;153:437-443.
 - 54 Gray JA: Integrating schizophrenia. *Schizophrenia bulletin* 1998;24:249-266.
 - 55 Gray JA, Feldon J, Rawlins JNP, Hemsley DR, Smith AD: The neuropsychology of schizophrenia. *Behavioral and Brain Sciences* 1991;14:1-20.
 - 56 Gray NS, Snowden RJ: The relevance of irrelevance to schizophrenia. *Neuroscience and biobehavioral reviews* 2005;29:989-999.
 - 57 Hemsley DR: Cognitive abnormalities and schizophrenic symptoms. *Psychological medicine* 1992;22:839-842.
 - 58 Hemsley DR: The disruption of the 'sense of self' in schizophrenia: Potential links with disturbances of information processing. *Br J Med Psychol* 1998;71 (Pt 2):115-124.
 - 59 Hemsley DR: The development of a cognitive model of schizophrenia: Placing it in context. *Neuroscience & Biobehavioral Reviews* 2005;29:977-988.
 - 60 Hemsley DR: The schizophrenic experience: Taken out of context? *Schizophrenia bulletin* 2005;31:43-53.
 - 61 Keefe RS, Arnold MC, Bayen UJ, Harvey PD: Source monitoring deficits in patients with schizophrenia; a multinomial modelling analysis. *Psychol Med* 1999;29:903-914.
 - 62 Fletcher PC, Frith CD: Perceiving is believing: A bayesian approach to explaining the positive symptoms of schizophrenia. *Nat Rev Neurosci* 2009;10:48-58.
 - 63 Feinberg I: Efference copy and corollary discharge: Implications for thinking and its disorders. *Schizophrenia bulletin* 1978;4:636-640.
 - 64 Frith C: Explaining delusions of control: The comparator model 20 years on. *Consciousness and cognition* 2012;21:52-54.
 - 65 Stephan KE, Friston KJ, Frith CD: Dysconnection in schizophrenia: From abnormal synaptic plasticity to failures of self-monitoring. *Schizophrenia bulletin* 2009;35:509-527.
 - 66 Crapse TB, Sommer MA: Corollary discharge across the animal kingdom. *Nat Rev Neurosci* 2008;9:587-600.
 - 67 Poulet JF, Hedwig B: New insights into corollary discharges mediated by identified neural pathways. *Trends Neurosci* 2007;30:14-21.
 - 68 Blakemore S-J, Smith J, Steel R, Johnstone EC, Frith CD: The perception of self-produced sensory stimuli in patients with auditory hallucinations and passivity experiences: Evidence for a breakdown in self-monitoring. *Psychological Medicine* 2000;30:1131-1139.
 - 69 Ford JM, Mathalon DH, Whitfield S, Faustman WO, Roth WT: Reduced communication between frontal and temporal lobes during talking in schizophrenia. *Biological psychiatry* 2002;51:485-492.
 - 70 Ford JM, Gray M, Faustman WO, Heinks TH, Mathalon DH: Reduced gamma-band coherence to distorted feedback during speech when what you say is not what you hear. *Int J Psychophysiol* 2005;57:143-150.
 - 71 Ford JM, Roach BJ, Faustman WO, Mathalon DH: Out-of-synch and out-of-sorts: Dysfunction of motor-sensory communication in schizophrenia. *Biological psychiatry* 2008;63:736-743.
 - 72 Lawrie SM, Buechel C, Whalley HC, Frith CD, Friston KJ, Johnstone EC: Reduced frontotemporal functional connectivity in schizophrenia associated with auditory hallucinations. *Biological psychiatry* 2002;51:1008-1011.
 - 73 Fletcher PC, Frith CD, Grasby PM, Friston KJ, Dolan RJ: Local and distributed effects of apomorphine on fronto-temporal function in acute unmedicated schizophrenia. *J Neurosci* 1996;16:7055-7062.
 - 74 Fletcher PC, McKenna PJ, Frith CD, Grasby PM, Friston KJ, Dolan RJ: Brain activations in schizophrenia during a graded memory task studied with functional neuroimaging. *Archives of general psychiatry* 1998;55:1001-1008.
 - 75 Ford JM, Mathalon DH, Roach BJ, Keedy SK, Reilly JL, Gershon ES, Sweeney JA: Neurophysiological evidence of corollary discharge function during vocalization in psychotic patients and their nonpsychotic first-degree relatives. *Schizophrenia bulletin* 2012;In press
 - 76 Ford JM, Gray M, Faustman WO, Roach BJ, Mathalon DH: Dissecting corollary discharge dysfunction in schizophrenia. *Psychophysiology* 2007;44:522-529.
 - 77 Whitford TJ, Mathalon DH, Shenton ME, Roach BJ, Bammer R, Adcock RA, Bouix S, Kubicki M, De Siebenthal J, Rausch AC, Schneiderman JS, Ford JM: Electrophysiological and diffusion tensor imaging evidence of delayed corollary discharges in patients with schizophrenia. *Psychological medicine* 2011;41:959-969.
 - 78 Wilquin H, Delevoeye-Turrell Y: Motor agency: A new and highly sensitive measure to reveal agency disturbances in early psychosis. *PLoS One* 2012;7:e30449.
 - 79 Carhart-Harris RL, Leech R, Erritzoe D, Williams TM, Stone JM, Evans J, Sharp DJ, Feilding A, Wise RG, Nutt DJ: Functional connectivity measures after psilocybin inform a novel hypothesis of early psychosis. *Schizophrenia bulletin* 2012;In press
 - 80 Taylor JG: A neural model of the loss of self in schizophrenia. *Schizophrenia bulletin* 2011;37:1229-1247.
 - 81 Parnas J, Møller P, Kircher T, Thalbitzer J, Jansson L, Handest P, Zahavi D: Ease: Examination of anomalous self-experience. *Psychopathology* 2005;38:236-258.
 - 82 Kapur S: Psychosis as a state of aberrant salience: A framework linking biology, phenomenology, and pharmacology in schizophrenia. *Am J Psychiatry* 2003;160:13-23.
 - 83 Kapur S, Mizrahi R, Li M: From dopamine to salience to psychosis--linking biology, pharmacology and phenomenology of psychosis. *Schizophr Res* 2005;79:59-68.
 - 84 Keefe RS, Kraus MS: Measuring memory-prediction errors and their consequences in youth at risk for schizophrenia. *Ann Acad Med Singapore* 2009;38:414-416.
 - 85 Kraus MS, Keefe RS, Krishnan RK: Memory-prediction errors and their consequences in schizophrenia. *Neuropsychol Rev* 2009;19:336-352.
 - 86 Todd J, Michie PT, Schall U, Ward PB, Catts SV: Mismatch negativity (mmn) reduction in schizophrenia-impaired prediction--error generation, estimation or salience? *Int J Psychophysiol* 2012;83:222-231.
 - 87 Gray NS, Hemsley DR, Gray JA: Abolition of latent inhibition in acute, but not chronic, schizophrenics. *Neurology, Psychiatry, and Brain Research* 1992;1:83-89.
 - 88 Lubow RE, Gewirtz JC: Latent inhibition in humans: Data, theory, and implications for schizophrenia. *Psychol Bull* 1995;117:87-103.
 - 89 Corlett PR, Honey GD, Fletcher PC: From prediction error to psychosis: Ketamine as a pharmacological model of delusions. *J Psychopharmacol* 2007;21:238-252.
 - 90 Corlett PR, Honey GD, Aitken MR, Dickinson A, Shanks DR, Absalom AR, Lee M, Pomarol-Clotet E, Murray GK, McKenna PJ, Robbins TW, Bullmore ET, Fletcher PC: Frontal responses during learning predict vulnerability to the psychotogenic

- effects of ketamine: Linking cognition, brain activity, and psychosis. *Archives of general psychiatry* 2006;63:611-621.
- 91 Hemsley DR: An experimental psychological model for schizophrenia; in Hafner H, Gattaz W, Janzarik W (eds): *Search for the causes of schizophrenia*. Berlin and Heidelberg, Springer, 1987, pp 179-188.
 - 92 Polyakov UF: The experimental investigation of cognitive functioning in schizophrenia; in Cole M, Maltzman I (eds): *A handbook of contemporary soviet psychology*. New York and London, Basic Books, 1969
 - 93 Sass LA: Self and world in schizophrenia: Three classic approaches. *Philosophy, Psychiatry & Psychology* 2001;8:251-270.
 - 94 Blankenburg W: First steps toward a psychopathology of "common sense". *Philosophy, Psychiatry, & Psychology* 2001;8:303-315.
 - 95 Harrison PJ: The neuropathology of schizophrenia: A critical review of the data and their interpretation. *Brain* 1999;122:593-624.
 - 96 Pantelis C, Velakoulis D, McGorry PD, Wood SJ, Suckling J, Phillips LJ, Yung AR, Bullmore ET, Brewer W, Soulsby B, Desmond P, McGuire PK: Neuroanatomical abnormalities before and after onset of psychosis: A cross-sectional and longitudinal mri comparison. *Lancet* 2003;361:281-288.
 - 97 Thaker GK, Ross DE, Cassidy SL, Adami HM, LaPorte D, Medoff DR, Lahti A: Smooth pursuit eye movements to extraretinal motion signals: Deficits in relatives of patients with schizophrenia. *Archives of general psychiatry* 1998;55:830-836.
 - 98 Thaker GK, Ross DE, Buchanan RW, Adami HM, Medoff DR: Smooth pursuit eye movements to extra-retinal motion signals: Deficits in patients with schizophrenia. *Psychiatry research* 1999;88:209-219.
 - 99 Hong LE, Turano KA, O'Neill H, Hao L, Wonodi I, McMahon RP, Elliott A, Thaker GK: Refining the predictive pursuit endophenotype in schizophrenia. *Biological psychiatry* 2008;63:458-464.
 - 100 Hong LE, Avila MT, Thaker GK: Response to unexpected target changes during sustained visual tracking in schizophrenic patients. *Exp Brain Res* 2005;165:125-131.
 - 101 Morris R, Griffiths O, Le Pelley ME, Weickert TW: Attention to irrelevant cues is related to positive symptoms in schizophrenia. *Schizophrenia bulletin* 2012;39:575-582.
 - 102 Hoffman RE, Woods SW, Hawkins KA, Pittman B, Tohen M, Preda A, Breier A, Glist J, Addington J, Perkins DO, McGlashan TH: Extracting spurious messages from noise and risk of schizophrenia-spectrum disorders in a prodromal population. *The British journal of psychiatry : the journal of mental science* 2007;191:355-356.
 - 103 Doniger GM, Silipo G, Rabinowicz EF, Snodgrass JG, Javitt DC: Impaired sensory processing as a basis for object-recognition deficits in schizophrenia. *The American journal of psychiatry* 2001;158:1818-1826.
 - 104 Cavezian C, Rossetti Y, Danckert J, d'Amato T, Dalery J, Saoud M: Exaggerated leftward bias in the mental number line of patients with schizophrenia. *Brain Cogn* 2007;63:85-90.
 - 105 Newby D: 'Cloze' procedure refined and modified: 'Modified cloze', 'reverse cloze' and the use of predictability as a measure of communication problems in psychosis. *British Journal of Psychiatry* 1998;172:136-141.
 - 106 Whittaker JF, Deakin JF, Tomenson B: Face processing in schizophrenia: Defining the deficit. *Psychological medicine* 2001;31:499-507.
 - 107 Benton AL, Van Allen MW: Prosopagnosia and facial discrimination. *J Neurol Sci* 1972;15:167-172.
 - 108 Emrich HM: A three-component-system hypothesis of psychosis. Impairment of binocular depth inversion as an indicator of a functional dysequilibrium. *The British journal of psychiatry Supplement* 1989:37-39.
 - 109 Schneider U, Borsutzky M, Seifert J, Leweke FM, Huber TJ, Rollnik JD, Emrich HM: Reduced binocular depth inversion in schizophrenic patients. *Schizophrenia research* 2002;53:101-108.
 - 110 Owen GS, Cutting J, David AS: Are people with schizophrenia more logical than healthy volunteers? *Br J Psychiatry* 2007;191:453-454.
 - 111 Uhlhaas PJ, Silverstein SM: Perceptual organization in schizophrenia spectrum disorders: Empirical research and theoretical implications. *Psychol Bull* 2005;131:618-632.
 - 112 Uhlhaas PJ, Phillips WA, Silverstein SM: The course and clinical correlates of dysfunctions in visual perceptual organization in schizophrenia during the remission of psychotic symptoms. *Schizophrenia research* 2005;75:183-192.
 - 113 Uhlhaas PJ, Phillips WA, Schenkel LS, Silverstein SM: Theory of mind and perceptual context-processing in schizophrenia. *Cogn Neuropsychiatry* 2006;11:416-436.
 - 114 Pearl D, Yodashkin-Porat D, Katz N, Valevski A, Aizenberg D, Sigler M, Weizman A, Kikinon L: Differences in audiovisual integration, as measured by mcgurk phenomenon, among adult and adolescent patients with schizophrenia and age-matched healthy control groups. *Compr Psychiat* 2009;50:186-192.
 - 115 Naatanen R, Kujala T, Winkler I: Auditory processing that leads to conscious perception: A unique window to central auditory processing opened by the mismatch negativity and related responses. *Psychophysiology* 2011;48:4-22.
 - 116 Naatanen R, Kahkonen S: Central auditory dysfunction in schizophrenia as revealed by the mismatch negativity (mmn) and its magnetic equivalent mmm: A review. *The international journal of neuropsychopharmacology / official scientific journal of the Collegium Internationale Neuropsychopharmacologicum* 2009;12:125-135.
 - 117 Umbricht D, Krljes S: Mismatch negativity in schizophrenia: A meta-analysis. *Schizophrenia research* 2005;76:1-23.
 - 118 Oranje B, Aggernaes B, Rasmussen H, Ebdrup BH, Glenthøj BY: Selective attention and mismatch negativity in antipsychotic-naïve, first-episode schizophrenia patients before and after 6 months of antipsychotic monotherapy. *Psychol Med* 2017;1-11.
 - 119 Bodatsch M, Ruhrmann S, Wagner M, Müller R, Schultze-Lutter F, Frommann I, Brinkmeyer J, Gaebel W, Maier W, Klosterkötter J, Brockhaus-Dumke A: Prediction of psychosis by mismatch negativity. *Biological psychiatry* 2011;69:959-966.
 - 120 Light GA, Braff DL: Mismatch negativity deficits are associated with poor functioning in schizophrenia patients. *Archives of general psychiatry* 2005;62:127-136.
 - 121 Henriksen MG, Parnas J: Clinical manifestations of self-disorders and the gestalt of schizophrenia. *Schizophrenia Bulletin* 2012;38:657-660.
 - 122 Rasser PE, Schall U, Todd J, Michie PT, Ward PB, Johnston P, Helmbold K, Case V, Soyland A, Tooney PA, Thompson PM: Gray matter deficits, mismatch negativity, and outcomes in schizophrenia. *Schizophrenia bulletin* 2011;37:131-140.
 - 123 Wynn JK, Sugar C, Horan WP, Kern R, Green MF: Mismatch negativity, social cognition, and functioning in schizophrenia patients. *Biological psychiatry* 2010;67:940-947.
 - 124 Peterson JB, Smith KW, Carson S: Openness and extraversion are associated with reduced latent inhibition: Replication and commentary. *Personality and Individual Differences* 2002;33:1137-1147.

- 125 Allan LM, Williams JH, Wellman NA, Tonin J, Taylor E, Rawlins JNP, Feldon J: Effects of tobacco smoking, schizotypy and number of pre-exposures on latent inhibition in healthy subjects. *Personality and Individual Differences* 1995;19:893-902.
- 126 Baruch I, Hemsley DR, Gray JA: Latent inhibition and "psychotic proneness" in normal subjects. *Personality and Individual Differences* 1988;9:777-783.
- 127 Lubow RE, Ingberg-Sachs Y, Zalstein-Orda N, Gewirtz JC: Latent inhibition in low and high "psychotic-prone" normal subjects. *Personality and Individual Differences* 1992;13:563-572.
- 128 Braunstein-Bercovitz H, Rammsayer T, Gibbons H, Lubow RE: Latent inhibition deficits in high-schizotypal normals: Symptom-specific or anxiety-related? *Schizophrenia research* 2002;53:109-121.
- 129 Baruch I, Hemsley DR, Gray JA: Differential performance of acute and chronic schizophrenics in a latent inhibition task. *Journal of Nervous and Mental Disease* 1988;176:598-606.
- 130 Bramon E, Rabe-Hesketh S, Sham P, Murray RM, Frangou S: Meta-analysis of the p300 and p50 waveforms in schizophrenia. *Schizophrenia research* 2004;70:315-329.
- 131 Patterson JV, Hetrick WP, Boutros NN, Jin Y, Sandman C, Stern H, Potkin S, Bunney WE, Jr.: P50 sensory gating ratios in schizophrenics and controls: A review and data analysis. *Psychiatry research* 2008;158:226-247.
- 132 Nelson B, Sass LA: Medusa's stare: A case study of working with self-disturbance in the early phase of schizophrenia. *Clinical Case Studies* 2009;8:489-504.
- 133 McGhie A, Chapman J: Disorders of attention and perception in early schizophrenia. *British Journal of Medical Psychology* 1961;34:103-115.
- 134 Gross G, Huber G, Klosterkötter J: Bonn scale for the assessment of basic symptoms - bsabs. Berlin, Springer, 1987.
- 135 Blankenburg W: [approach to the psychopathology of common sense]. *Confinia Psychiatrica* 1969;12:144-163.
- 136 Bovet P, Parnas J: Schizophrenic delusions: A phenomenological approach. *Schizophr Bull* 1993;19:579-597.
- 137 Stanghellini G: Disembodied spirits and deanimated bodies: The psychopathology of common sense. Oxford, UK, Oxford University Press, 2004.
- 138 Stanghellini G, Lysaker PH: The psychotherapy of schizophrenia through the lens of phenomenology: Intersubjectivity and the search for the recovery of first- and second-person awareness. *Am J Psychother* 2007;61:163-179.
- 139 Green MF, Olivier B, Crawley JN, Penn DL, Silverstein S: Social cognition in schizophrenia: Recommendations from the measurement and treatment research to improve cognition in schizophrenia new approaches conference. *Schizophrenia bulletin* 2005;31:882-887.
- 140 Penn DL, Sanna LJ, Roberts DL: Social cognition in schizophrenia: An overview. *Schizophrenia bulletin* 2008;34:408-411.
- 141 Rhodes J, Gipps RGT: Delusions, certainty, and the background. *Philosophy, Psychiatry & Psychology* 2008;15:295-310.
- 142 Gibson JJ: The theory of affordances; in Shaw R, Bransford J (eds): *Perceiving, acting, and knowing: Toward an ecological psychology* Hillsdale, NJ, Erlbaum, 1977, pp 67-82.
- 143 Gibson JJ: The theory of information pickup and its consequences; in Yantis S (ed) *Visual perception: Essential readings*. New York, Psychology Press, 2001, pp 84-100.
- 144 Spinelli E: *The interpreted world: An introduction to phenomenological psychology*. London, Sage, 1989.
- 145 Nelson B, Sass LA: The phenomenology of the psychotic break and huxley's trip: Substance use and the onset of psychosis. *Psychopathology* 2008;41:346-355.
- 146 Sartre J-P: *Nausea*. New York, New Directions, 1964.
- 147 Nelson B, Rawlings D: Relating schizotypy and personality to the phenomenology of creativity. *Schizophr Bull* 2008;36:388-399.
- 148 Howes OD, Kapur S: The dopamine hypothesis of schizophrenia: Version iii--the final common pathway. *Schizophr Bull* 2009;35:549-562.
- 149 Cannon TD: How schizophrenia develops: Cognitive and brain mechanisms underlying onset of psychosis. *Trends Cogn Sci* 2015
- 150 Nelson B: Varieties of self-disturbance: A prism through which to view mental disorder. *Early Intervention in Psychiatry* 2013;7:231-234.
- 151 Nelson B, Fornito A, Harrison BJ, Yucel M, Sass LA, Yung AR, Thompson A, Wood SJ, Pantelis C, McGorry PD: A disturbed sense of self in the psychosis prodrome: Linking phenomenology and neurobiology. *Neurosci Biobehav Rev* 2009;33:807-817.
- 152 Kean C: Battling with the life instinct: The paradox of the self and suicidal behavior in psychosis. *Schizophrenia bulletin* 2011;37:4-7.
- 153 McClure SM, Daw ND, Montague PR: A computational substrate for incentive salience. *Trends Neurosci* 2003;26:423-428.
- 154 Gamma F, Goldstein JM, Seidman LJ, Fitzmaurice GM, Tsuang MT, Buka SL: Early intermodal integration in offspring of parents with psychosis. *Schizophr Bull* 2013
- 155 Parnas J, Bovet P, Innocenti GM: Schizophrenic trait features, binding, and cortico-cortical connectivity: A neurodevelopmental pathogenetic hypothesis. *Neurology, Psychiatry and Brain Research* 1996;4:185-196.
- 156 Sass LA: *Madness and modernism: Insanity in the light of modern art, literature, and thought—revised edition*. Oxford, UK, Oxford University Press, 2017.
- 157 Sass LA: Self-disturbance and schizophrenia: Structure, specificity, pathogenesis (current issues, new directions). *Schizophr Res* 2014;152:5-11.
- 158 Bendall S, Jackson HJ, Hulbert CA, McGorry PD: Childhood trauma and psychotic disorders: A systematic, critical review of the evidence. *Schizophr Bull* 2008;34:568-579.
- 159 Selten JP, van der Ven E, Rutten BP, Cantor-Graae E: The social defeat hypothesis of schizophrenia: An update. *Schizophr Bull* 2013;39:1180-1186.
- 160 Renard SB, Huntjens RJ, Lysaker PH, Moskowitz A, Aleman A, Pijnenborg GH: Unique and overlapping symptoms in schizophrenia spectrum and dissociative disorders in relation to models of psychopathology: A systematic review. *Schizophr Bull* 2017;43:108-121.
- 161 Kilcommons AM, Morrison AP: Relationships between trauma and psychosis: An exploration of cognitive and dissociative factors. *Acta Psychiatr Scand* 2005;112:351-359.
- 162 Myin-Germeys I, van Os J: Stress-reactivity in psychosis: Evidence for an affective pathway to psychosis. *Clinical psychology review* 2007;27:409-424.
- 163 Keane BP, Silverstein SM, Wang Y, Papathomas TV: Reduced depth inversion illusions in schizophrenia are state-specific and occur for multiple object types and viewing conditions. *J Abnorm Psychol* 2013;122:506-512.
- 164 Silverstein SM, Keane BP: Perceptual organization impairment in schizophrenia and associated brain mechanisms: Review of research from 2005 to 2010. *Schizophr Bull* 2011;37:690-699.
- 165 Breakspear M, Terry JR, Friston KJ, Harris AW, Williams LM, Brown K, Brennan J, Gordon E: A disturbance of nonlinear

- interdependence in scalp eeg of subjects with first episode schizophrenia. *NeuroImage* 2003;20:466-478.
- 166 Whitfield-Gabrieli S, Thermenos HW, Milanovic S, Tsuang MT, Faraone SV, McCarley RW, Shenton ME, Green AI, Nieto-Castanon A, LaViolette P, Wojcik J, Gabrieli JD, Seidman LJ: Hyperactivity and hyperconnectivity of the default network in schizophrenia and in first-degree relatives of persons with schizophrenia. *Proc Natl Acad Sci U S A* 2009;106:1279-1284.
 - 167 Northoff G, Qin P: How can the brain's resting state activity generate hallucinations? A 'resting state hypothesis' of auditory verbal hallucinations. *Schizophrenia research* 2011;127:202-214.
 - 168 Keefe RSE: The neurobiology of disturbances of the self: Autozoetic agnosia in schizophrenia.; in Amador XF, David AS (eds): *Insight and psychosis*. Oxford, Oxford University Press, 1998, pp 142-173.
 - 169 Graham-Schmidt KT, Martin-Iverson MT, Waters FAV: Self- and other-agency in people with passivity (first rank) symptoms in schizophrenia. *Schizophr Res* 2017
 - 170 Parnas J, Handest P: Phenomenology of anomalous self-experience in early schizophrenia. *Compr Psychiatry* 2003;44:121-134.
 - 171 Naatanen R, Kujala T, Kreegipuu K, Carlson S, Escera C, Baldeweg T, Ponton C: The mismatch negativity: An index of cognitive decline in neuropsychiatric and neurological diseases and in ageing. *Brain : a journal of neurology* 2011;134:3435-3453.
 - 172 Waters FA, Maybery MT, Badcock JC, Michie PT: Context memory and binding in schizophrenia. *Schizophr Res* 2004;68:119-125.

Correspondence address

Nelson, B., Orygen
Youth Health Research Centre,
35 Poplar Rd (Locked Bag 10), Parkville, Victoria 3052, Australia.
e-mail: Barnaby.nelson@orygen.org.au

Received: 21.05.2017

Revised: 22.05.2017, 29.05.2017

Accepted: 03.07.2017

Published: 30.08.2017