



Jung's views on causes and treatments of schizophrenia in light of current trends in cognitive neuroscience and psychotherapy research

I. Aetiology and phenomenology

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Abstract: Jung's writings on schizophrenia are almost completely ignored or forgotten today. The purpose of this paper, along with a follow-up article, is to review the primary themes found in Jung's writings on schizophrenia, and to assess the validity of his theories about the disorder in light of our current knowledge base in the fields of psychopathology, cognitive neuroscience and psychotherapy research. In this article, five themes related to the aetiology and phenomenology of schizophrenia from Jung's writings are discussed: 1) *abaissement du niveau mental*; 2) the complex; 3) mandala imagery; 4) constellation of archetypes and 5) psychological versus toxic aetiology. Reviews of the above areas suggest three conclusions. First, in many ways, Jung's ideas on schizophrenia anticipated much current thinking and data about the disorder. Second, with the recent (re)convergence of psychological and biological approaches to understanding and treating schizophrenia, the pioneering ideas of Jung regarding the importance of both factors and their interaction remain a useful and rich, but still underutilized resource. Finally, a more concerted effort to understand and evaluate the validity of Jung's concepts in terms of evidence from neuroscience could lead both to important advances in analytical psychology and to developments in therapeutic approaches that would extend beyond the treatment of schizophrenia.

Key words: *abaissement du niveau mental*, aetiology, archetype, cognitive neuroscience, complex, *dementia praecox*, mandala, perception, schizophrenia phenomenology

Introduction

Although it is common even today to see citations of the work of Kraepelin and Bleuler in the introductions to research papers on schizophrenia, Jung's work on schizophrenia is rarely mentioned in papers in scientific journals, despite his writing the first comprehensive theoretical treatment of the disorder. Jung's book, *The Psychology of Dementia Praecox* (original title in German: *Über die psychologie der dementia praecox: ein Versuch*), was begun in 1903, published

in German in late 1906 (with a 1907 publication date), and in English in 1909. The book is significant in several respects. First, rather than focusing on symptom groupings and nosological issues, Jung (like his early colleague and hospital chief Bleuler) moved the field in a new direction by demonstrating the psychological significance of psychotic symptoms, including what they can reveal about unconscious and symbolic processes (Taylor 1998). Further, he emphasized how they could be used to guide treatment, as when noting that ‘the patient describes for us, in her symptoms, the hopes and disappointments of her life’ (Jung 1907/1960, para. 298). In this way, the book was the first psychodynamic conceptualization of schizophrenia (Rodriguez 2003). All of this was in stark contrast to the prevailing ideology, which held that psychotic symptoms were essentially random and meaningless productions. Second, Jung proposed the first theory in which psychological factors were seen as important in causing the disorder, thereby anticipating the current vulnerability-stress model of schizophrenia (Zubin & Spring 1977) by 70 years. Third, he applied the influential work of Janet to an understanding of schizophrenia, which had not been done before (Rodriguez 2003). This allowed for the demonstration of continuities between neurotic and psychotic symptoms, and for meaningful discussions of a spectrum of schizophrenia-related illness—a concept only confirmed by family, twin, and adoption research studies in the 1960s and 70s, and by clinical work related to schizotypy from the 1950s onwards. As part of this focus, Jung demonstrated parallels between the positive symptoms of schizophrenia and aspects of hysteria (e.g., hallucinations) that have been viewed as the basis for Bleuler’s important categorization of symptoms found in schizophrenia as either ‘accessory’ (diagnostically non-specific symptoms) (Rodriguez 2003), or ‘fundamental’ (schizophrenia-specific symptoms) (Bleuler 1911/1950; Raskin 1975). For this, and other reasons, it has been claimed that Bleuler’s classic text of 1911 (*Dementia Praecox or the Group of Schizophrenias*) would be unthinkable without Jung’s *The Psychology of Dementia Praecox* (Rodriguez 2003). Indeed, on the 100th anniversary of the German publication of Bleuler’s book, Moskowitz and Heim (2011) made multiple references to the influences of Jung’s word association experiments, his concept of the complex, and his extension of Janet’s ideas to Bleuler’s thinking.

In 1909, Jung left the Burhölzli clinic (where he had been treating schizophrenia patients since 1900), although he continued to give lectures there until 1914 (Möller, Scharfetter & Hell 2002). However, he wrote several additional papers on schizophrenia in his lifetime, with the last appearing in 1958. What is remarkable about many of Jung’s ideas about schizophrenia is the extent to which they resemble many of the ‘new’ discoveries about schizophrenia in the areas of psychopathology research, cognitive neuroscience, and psychotherapy. The purpose of this, and a follow-up paper, is to review the primary themes found in *The Psychology of Dementia Praecox* and Jung’s later works on schizophrenia, to demonstrate the extent to which Jung’s ideas anticipated much current thinking about the disorder, and to assess the validity of his theories in the light of our current

knowledge base in these other fields. This paper, Part 1, will focus on aetiology and phenomenology, and will cover the following themes: 1) *abaissement du niveau mental*; 2) the complex; 3) mandala imagery; 4) constellation of archetypes; and 5) psychological versus toxic aetiology. Part 2 will focus on psychological approaches to research on, and treatment of, schizophrenia. A review of the above 5 areas reveals that: 1) Jung's ideas about schizophrenia and its treatment anticipated several important current trends; 2) Jung's ideas on schizophrenia remain useful for further exploration of the interaction of biological and psychological factors in the disorder; and 3) the ways in which Jungian concepts and cognitive neuroscience converge suggest that incorporation of ideas and data from the latter field can assist in the further evolution of analytical psychology.

Jung's theories of the aetiology and phenomenology of schizophrenia

Abaissement du niveau mental

Throughout *The Psychology of Dementia Praecox*, Jung repeatedly makes use of Janet's concept of *abaissement du niveau mental*. In Janet's formulation, *abaissement du niveau mental* relates to the concept of *tension psychologique*—or the degree to which psychic energy was channelled or focused (i.e., not to the *amount* of psychic energy): lowered psychic tension, or a reduction (*abaissement*) in the mental level (*niveau*) therefore referred to a more primitive, less differentiated, and less organized state of mental functioning (Haule 1983). Jung defined *abaissement du niveau mental* as 'a weakness in the hierarchical order of the ego' (1946/1985, para. 361). More specifically, in Jung's view, *abaissement* leads to normally inhibited contents of the unconscious entering consciousness, incoherent speech, and 'superficiality of associations, symbols, stereotypies, perseverations, command automatisms, apathy, abulia, disturbance of reproduction, and in a limited sense, negativism' (1907/1960, para. 30). Stated differently, 'the central control of the psyche has become so weak that it can neither promote the positive nor inhibit the negative acts, or vice versa' (ibid., para. 29).

This framework resembles later work from a behavioural (Hullian learning theory) tradition in which schizophrenia is viewed as involving a partial random re-ordering within the hierarchy of response tendencies to environmental stimuli, resulting in behaviour that seems odd and out of context (Broen & Storms 1966; Storms & Broen 1969). This model was later combined with concepts from the diathesis-stress model (Zubin & Spring 1977), and data on intense arousal in schizophrenia (both consistent with Jung's views, see below) to develop the *hierarchy collapse model* which has been useful in developing psychiatric rehabilitation interventions for people with schizophrenia (Spaulding et al. 1986).

Jung's view of the *abaissement*-related transformation of brain and psychological functioning in schizophrenia is similar to recent research suggesting a reduction in the reciprocal relationship between task-related and default mode brain network activity in the disorder. The default mode network is an

interconnected system of brain structures that is active when a person is not engaged in focused thought, but is instead daydreaming, letting his/her mind wander, thinking about the past or future, or involved in other internally-focused activity. Default mode network activity has also been implicated in the genesis of creativity (Buckner et al. 2008). Normally, during task-related activity, default mode activity is inhibited. However, in schizophrenia, there is evidence for a reduction in inhibition, and this is thought to lead to the emergence into consciousness of thoughts that would normally be suppressed in a given situation, and to psychotic symptoms (Anticevic et al., 2012a, 2012b; Whitfield-Gabrieli et al. 2009), a view consistent with Jung's ideas on *abaissement*.

Jung believed that two important characteristics of schizophrenia resulted from the *abaissement*: the fragmentation of psychic functioning into multiple, active, independent, complexes (Jung, 1939/1960), and, relatedly, 'a physiological and unsystematic disintegration of the psychic elements' (1957/1960/1960, para. 544), a view that is similar to the reduced cognitive coordination and disconnection hypotheses (of schizophrenia) of today, and our understanding of the neurobiological basis of cognitive fragmentation (e.g., Phillips & Silverstein 2003, 2013; see below, section on the complex, for examples of evidence supporting these models). Jung also believed that *abaissement* could explain ambivalence, which can be viewed as the simultaneous occurrence of functionally independent streams of mental activity that represent incompatible response tendencies. Importantly, however, Jung emphasized that the *abaissement* in schizophrenia was not generalized, the way it is in substance-induced intoxicated states. Rather, in his view, it was limited to those aspects of mental functioning that were influenced by complexes. An implication of this is that there will be areas of preserved reality testing and other aspects of psychic life in schizophrenia, despite the presence of symptoms. There are countless examples of this in the clinical literature and the everyday experience of clinicians (e.g., the severely thought-disordered and hallucinating hospital patient who cannot participate in group therapy but who routinely seeks out places in the hospital where cigarette butts may be found; or the patient who appears inattentive and disoriented in group therapy but gets up to leave as soon as the therapist announces the group is over, etc.), and this is the basis for the design of effective behavioural interventions that increase the frequency of appropriate behaviours by rewarding their occurrence (e.g., Paul & Lentz 1977; Silverstein et al. 2006b).

The complex

The concept of the complex is central both to Jung's theory of schizophrenia, and to analytical psychology as a whole. In this section, Jung's ideas on the complex, as he applied them to schizophrenia, will be reviewed and examined in light of current evidence from cognitive neuroscience. In doing so, some of his basic concepts about the complex will also be examined to provide a more comprehensive review of the implications of Jung's ideas on schizophrenia for current efforts to understand brain function. In the subsequent two sections,

this line of thought is extended to mandala imagery, and then to archetype constellation, as both are increased in schizophrenia, and the theory of the archetypes can be viewed as the completion or ultimate extension of Jung's theory of the complex (Haule 1984; Jaffé 1972).

Overview

In Jung's view, complexes are clusters of ideas and images, held together by a powerful feeling, that affect the contents of consciousness and behaviour—like 'a soul within a soul' (Peterson & Jung 1907/1981, para. 1067). In his later works, he noted that the complex 'has a powerful inner coherence, it has its own wholeness, and in addition, a relatively high degree of autonomy...and therefore behaves like an animated foreign body in the sphere of consciousness' (1960/1969, para. 201). In 1906, in his introduction to Bleuler's first book (*Affectivität, Suggestibilität, Paranoia*), Jung noted that psychotic symptoms such as delusions can be viewed as manifestations of complexes (Moskowitz 2006), a theme he also emphasized in his experimental works (Peterson & Jung 1907/1981) (note that Jung introduced the concept of the complex to Bleuler in 1904; Moskowitz 2006). Such ideas are supported by current research on medication-resistant psychotic symptoms (e.g., delusions), which suggests that they have become independent of increased dopaminergic activity (which may initially trigger symptom formation). That is, these circuits become self-activating (i.e., autonomous), or parasitic foci (Hoffman & McGlashan 1993, 1994), which can be viewed as attractor states that operate relatively independently of other attractors. An important feature of Jung's theory of the complex was the idea that strong complexes assimilate as many mental representations as possible, allowing them to gain in strength, and to further interfere with ego function and reduce the overall influence of the ego on behaviour and mental life. An implication of this for psychosis is that, as more and more memories (new and old) become associated with the complex, more and more of experience is interpreted through the lens of the complex rather than the ego complex. This is consistent with modern work demonstrating that any neuron can become part of multiple networks and that the activity of individual networks can become activated or inhibited depending on the focus of attention (Fuster 2003).

Symptoms of schizophrenia and complexes

In Jung's view, many symptoms of schizophrenia can be explained as effects of complexes. For example, symptoms such as not completing actions, believing one is a machine and that all movements seem mechanical, believing that one is being forced to think ideas, thought blocking and thought withdrawal, and feelings of strangeness to oneself, could all, in Jung's view, be seen as due to strong inhibition of the ego complex deriving from the activation of other complexes (see above, for the discussion of the symptom of ambivalence). Jung

observed that when a person is dominated by a complex, only the ideas associated with that complex are experienced as normal: 'all other perceptions within or without are subject to the inhibition, so that they become unclear and lose their feeling tone' (1907/1960, para. 174). This can be seen in the following clinical example: 'I am in nowhere land, no feelings, no thoughts, nothing but a blank empty space where Pam used to be. I am a broken bowl of nothing, still breathing and alive, but little else. No needs, no desires, no wants, no urges, an involuntary Buddhist dissolving into the void' (Wagner & Spiro 2005, p. 295). Jung also noted that, at its extreme, the inhibitory function of the pathological complex can become so strong that it can create a disturbance in reality testing. Moreover, the more the complex is 'split off', the more automatic the disturbances may be. A good example of this phenomenon can be seen in this statement by a person with schizophrenia: 'I feel like I am "being thought" by the zillions, as if thoughts themselves—and not even *my* thoughts—are in control' (Wagner & Spiro 2005, p. 136).

In a fascinating comment that anticipated some of his later biological focus on schizophrenia, Jung noted that

Because of its intensity the complex arrogates to itself the activity of the cerebrum on the widest scale, so that at least a very large number of impulses to other areas disappear. It can then easily be imagined that the complex creates a condition in the brain functionally equivalent to an extensive destruction of the cerebrum. Though this hypothesis cannot be proved, it might nevertheless explain many things that are beyond the reach of psychological analysis.

(1907/1960, para. 193)

The idea that the symptoms of schizophrenia can themselves cause changes in brain function is consistent with modern views. For example, several lines of evidence indicate that active psychosis is a neurotoxic process that leads to progressive deterioration in brain tissue and function, especially in the period leading up to and immediately following the first episode (McGlashan 2006; Wyatt 1991) (see also section below on psychological vs. toxic aetiology of schizophrenia).

It has also been proposed that rather than, or in addition to, deterioration, what is happening is an attenuation in synaptic plasticity, or the ability of neurons to form new networks (McGlashan 2006). There is much evidence for the latter. For example, the concept of 'functional disintegration' has been used to describe the reduced connectivity between brain regions, and resulting lack of coordinated neural and cognitive activity, observed in schizophrenia (Zhou et al. 2007). Other views of schizophrenia also highlight impaired cognitive coordination (Phillips & Silverstein 2003, 2013), or disconnection (e.g., Friston 1998, 1999, 2005) in the disorder. These views are based on a growing body of evidence, including from functional imaging studies of the connection strength between brain regions. For example, Palaniyappan et al. (2013) demonstrated a breakdown in key neural circuits in schizophrenia that process information about the meaningfulness of external stimuli. This included a significant relationship between illness severity and reduced network activity. There are also multiple examples of disorganization

across cognitive domains (e.g., in vision, audition, language, memory, affect-ideation consistency) that are thought to reflect weakened connectivity, and these are significantly correlated in schizophrenia, as would be expected if there was a single process that was impaired (Phillips & Silverstein 2003; Silverstein & Keane 2011). Psychological research has also demonstrated reduced binding between self- and action-representations in schizophrenia, so that patients are aware of events that happened, but are impaired in remembering whether they or somebody else performed the action (Danion, Rizzo & Bruant 1999). This impairment has clear implications for maintenance of a sense of self and agency in the world, including predisposing a person to develop delusions of being controlled by outside forces. Jung noted, however, that in schizophrenia, the ego complex is not totally destroyed: ‘...schizophrenics often suddenly begin to react in a fairly normal manner during severe physical illness or any other far-reaching changes’ (1907/1960, para. 180). This has implications for psychotherapy and recovery since it suggests that the core of the person remains accessible even when symptoms are active.

The status of the complex as a concept within cognitive neuroscience

To what extent has the concept of the complex been supported by later work? Evidence from several sources supports the idea. For example, long ago, Hebb (1949) proposed the concept of the cell assembly, leading to the popular notion that ‘cells that fire together wire together’. Similarly, Hayek (1952) noted that all experience is stored in networks or maps of connections between neurons in the cortex. These networks have been formed by the co-occurrence of inputs from the environment, and also co-occurrence of internal mental events and affects with these environmental inputs. Support for the existence of neural networks held together by mutual excitation comes from studies in cognitive neuroscience in areas ranging from vision to thought.

A paradigmatic and clear example of a cell assembly has been demonstrated in vision, using the concept of the ‘association field’ (Field, Hayes & Hess 1993; Hess et al. 2003; see Figure 1). The association field refers to the existence of mutual excitation between the cells in visual cortex that encode visual features that are related to each other by virtue of forming an edge, line, or shape contour (i.e., based on their spatial positions and orientations relative to each other; see Figures 1, 2 below). Support for the concept of association fields in vision comes from psychophysical (Hess et al. 2003), anatomical and electrophysiological studies (e.g., Mandon & Kreiter 2005).

But it has been noted that the nature of the structural representations underlying visual representations are the same as those involved in higher cognitive functions such as attention, memory, language, reasoning, motor control, and even consciousness (Chechile et al. 1996; Crick 1994; Fuster 2003; Glezer 1995; Phillips & Singer 1997; Prinz & Hommel 2002; Varela et al. 2001; Vidal et al. 2006). A theme emerging from this work is that mental representations (e.g., perceptions, memories, symbols) are ‘derivative gestalts’ (Fuster 2003, p. 94) defined by a set

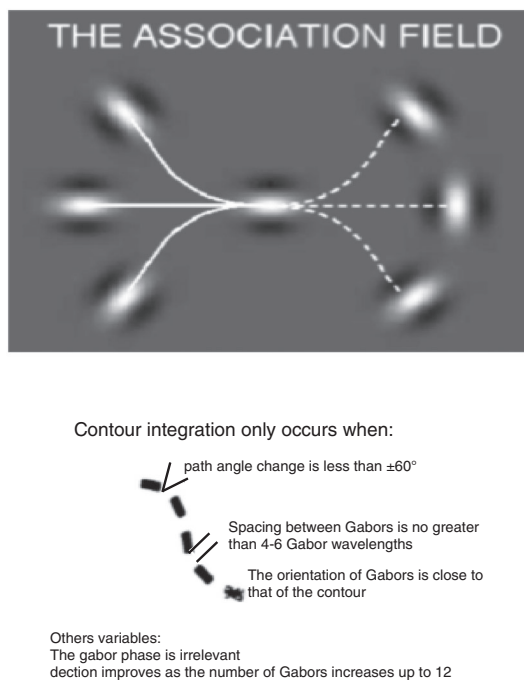


Figure 1. Top: Visual depiction of an ‘association field’. This is a low-level demonstration of how elements that are related to each other (in this case, based on spatial relationships) are linked within the nervous system. Solid lines represent excitatory linkages between elements that are likely to be part of the same edge, curve, or object—based on prior experience of the person and species with objects in the world. Dashed lines represent inhibitory links between features that are unlikely to be part of the same form (due to the statistically low likelihood of elements with these orientations being part of a single object). Bottom: Some of the relational constraints that must be met for contour elements to be linked. It can be seen that these are relevant to mandala imagery. In addition, the concept of an association field from vision science provides a model for linking of higher-level representations (based on less concrete features, such as meaning and emotional tone) into ‘derivative gestalts’ (Fuster 2003) such as complexes and archetypes. Figure from Hess, Hayes, & Field (2003). Reprinted with permission from Elsevier.

of relationships. This raises the possibility that the component parts of a complex may be ‘held together’ by a form of association field. Therefore, the study of how the brain creates some of its most basic gestalts (i.e., those involved in the visual perception of shape), and how these can become fragmented, is relevant to understanding complexes and schizophrenia, respectively.

Recent work in neuroscience suggests that binding of neural activity occurs through synchronization of the oscillatory firing activity of sets of neurons. Synchronized oscillations are thought to ‘serve as a tag of relatedness for the formation of distributed assemblies that underlie coherent action and cognition’

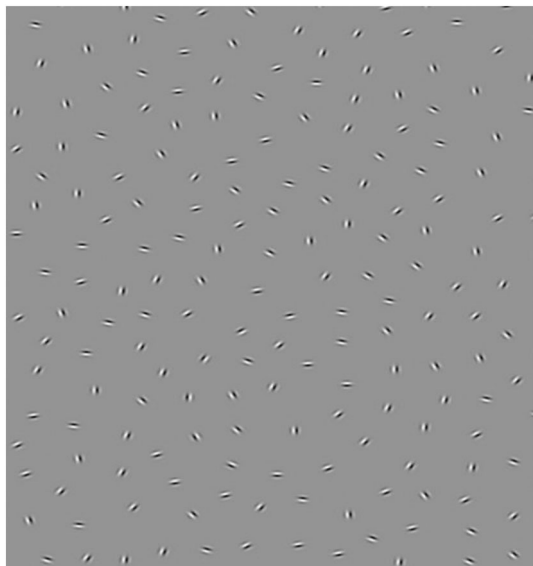


Figure 2. Most people can see an oval contour in the bottom portion of this image. Since the elements of the circular contour are further apart from each other than are adjacent background elements (on average), in order to perceive this figure the visual system must detect the regularities in degree of orientation change in order to link the contour elements into a coherent shape, as shown in Figure 1 (and conversely, to not link contour elements with background elements). These processes—linking related elements together, and not linking unrelated elements together—are deficient in schizophrenia (Silverstein et al. 2000, 2009), and are related to higher-level manifestations of reduced organization (e.g., associative disturbance in thinking; Phillips & Silverstein 2003), suggesting a general weakening of connection strength in schizophrenia—a setting that neuroscience research has demonstrated can lead to the emergence of isolated attractor states (e.g., parasitic foci subserving hallucinations and delusions that can be seen as representing powerful complexes). In addition, however, binding of contiguous elements is intact in schizophrenia, and such imagery may be released during periods of disorganization, and may serve a compensatory organizing function, according to Jung.

(Uhlhaas et al. 2008, p. 939). This process is mediated by excitatory activity at the NMDA-type of glutamate receptor, and by related inhibitory interneuron activity at GABA receptors (Phillips & Silverstein 2003, 2013). Moreover, groups of synchronously activated neurons release neurotrophins (i.e., proteins that promote the development of neurons) such as BDNF and NGF which enhance the formation of synapses and the effectiveness of communication between cells in the network, leading them to be mutually co-activated in the future (Katz & Shatz 1996). This can be helpful when forming networks related to adaptive behaviour, but maladaptive when synchronization is occurring in stable attractor systems unrelated to the statistical structure of the world (e.g., parasitic foci, complexes).

In schizophrenia, many of these processes have been shown to be deficient. For example, it has been repeatedly demonstrated that synchronized neuronal activity is reduced in schizophrenia (Uhlhaas et al. 2008), and associated with clinically significant disorganized symptoms (Grützner et al. 2013). Electrophysiological data indicate that inappropriate associations in schizophrenia (such as those studied by Jung with the word association test) result from activation of associations to words that are distant in the semantic network, and not as constrained by the context provided by preceding words, as is the case of healthy people (Mathalon, Faustman & Ford 2002). This is analogous to reduced binding of contextually related visual features in schizophrenia, which has been found in over 50 studies (reviewed in Silverstein & Keane 2011), as has been demonstrated using stimuli similar to those in Figure 2.

The astute reader may ask here: if neural network formation is reduced (due to either deterioration and/or reduced ability to form new networks) in schizophrenia, how can stronger-than-usual complexes be formed? The key to this is the idea that the weakening of the ego complex leads to the relative strengthening of non-ego complexes, and vice versa. As Hoffman and McGlashan (1993) noted, the isolation of an aberrant cortical network from the influence of other networks (i.e., a parasitic focus) occurs due to a general weakening of cortical connectivity, due to reductions in NMDA receptor activity and synchronization as noted above (the biological bases of *abaissement du niveau mental*?). When cortical connectivity is weakened in general, any newly formed network will be less influenced than it should be by ongoing brain activity, and, by extension, by the statistical structure of reality. As we know from epilepsy research, one of the effects of isolating an active network can be to strengthen that network and to have it enlarge. Although, clearly, in schizophrenia, this does not happen as quickly or with the seizure inducing electrical effects seen in temporal lobe epilepsy, rates of epilepsy are elevated in schizophrenia, and psychotic symptoms are increased in people with epilepsy relative to the general population, providing evidence for a common mechanism (see Butler et al. 2012; Uhlhaas & Singer 2006). It is the combination of reduced functional connectivity in networks that would normally represent reality via synchronized binding of related elements (Caldera & Seghier 2009; Hess et al. 2003), and that are part of the ego complex, along with hyperexcitability of aberrant networks (i.e., other complexes)—typically begun by excessive stress-related dopaminergic activity and arousal—that can lead to the formation of parasitic foci in schizophrenia that can evolve into complexes, delusions, and hallucinations.

Neuroscience research has recently demonstrated that the series of events postulated by Jung—weakening of the primary network/complex and formation of other ones that compete for the control of cognition and behaviour—does occur. For example, Olypher, Klement, and Fenton (2006) demonstrated that injection of tetrodotoxin (a neural activity blocker thought to induce an animal model of schizophrenia) into the hippocampus of rats who had learned a set of spatial relationships had the effects of reducing the correlated firing

between neurons in the network related to learning the task, and increasing the coupling of neurons that had not previously fired together. As a result, the injection impaired the ability of rats to distinguish between relevant associations among distant spatial stimuli, and increased the strength of associations between irrelevant local stimuli—an effect that can be viewed as analogous to Jung's view of what occurs during *abaissement du niveau mental*. A similar process may be at work during the onset of psychotic symptoms, although caused by endogenous neurochemistry (see the section below on psychological versus toxic causation). A revealing example of this phenomenon, as it is expressed in humans during the early phase of psychosis, came from a patient who reported that objects began to seem unconnected to their environmental contexts and therefore meaningless. At the same time, he noted that 'out of these perceptions came the absolute awareness that my ability to see connections had been multiplied many times over' (Matussek 1952/1987, p. 96).

What factor determines which networks dissipate and which form stable states? It is likely that this is determined by personal vulnerability factors, as we see clinically where delusions primarily involve threats to the self (e.g., Ben-Zeev et al. 2012). This supports Jung's concept of the feeling-toned complex. An important task for future research is to characterize how affect is bound with representations such as imagery. To date, research on stimulus binding has been restricted mainly to concrete sensations, and it is only recently, with the growth of the field of affective neuroscience, that the neurobiology of emotion is receiving adequate scientific attention.

Mandala imagery and art in schizophrenia

Discussions of mandala imagery and symbolism are prevalent in Jung's later work. However, they are also relevant to his work on schizophrenia, as such images are known to appear (in hallucinations, imagery, and art) during episodes of psychotic disorganization (Jung 1959; Trueman 2002). Jung speculated that this is a compensatory response to help re-establish order and coherence (Jung 1958a/1960, para. 582). While this hypothesis has not been investigated, it is supported by anecdotal clinical evidence, such as the sequencing in the art of Adolf Wölfl (who was institutionalized with schizophrenia for nearly his entire adult life), where mandala paintings often followed paintings depicting disasters and dangerous situations (Spoerri & Baumann 2003).

It is interesting to consider that all mandala imagery is extensively comprised of elementary visual features such as lines, circular contours, radial projections, and symmetry (see Figure 3). Such representations are generated and represented in early visual processing areas such as V1-V4 (Silverstein et al. 2009; Wilkinson et al. 2000), and, as long as the stimuli are not fragmented in space, can be processed and perceived successfully even by very psychotic schizophrenia patients (see Silverstein & Keane 2011; Uhlhaas & Silverstein 2005 for reviews).

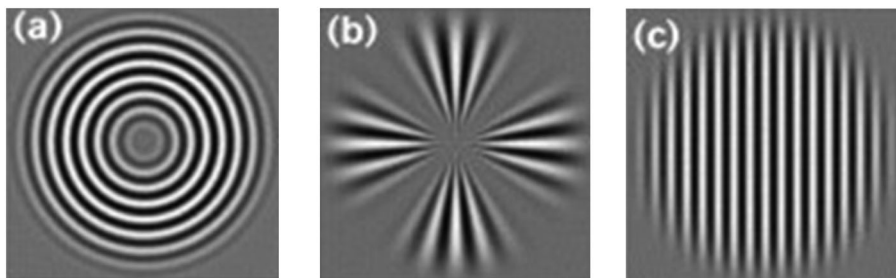


Figure 3. Basic feature arrangements to which early portions of the visual system are sensitive. Human extrastriate visual areas (e.g., V4) are more sensitive to the patterns a and b, than to c. This is relevant to the generation of the basic components of mandala imagery during psychotic states in people with schizophrenia, and such imagery may involve spontaneous activity in areas such as V4. The global aspects of much mandala imagery can be viewed as a combination of concentric and radial images, as demonstrated in panels a and b. Figure from Wilkinson et al. (2000) reprinted with permission from Elsevier.

These visual features are also among the earliest drawn by children, and the last preserved among artists with degenerating brain diseases (Weber 2002). This suggests that even in severely psychotic states characterized by cognitive disorganization, the building blocks of mandala imagery are present, and available to be harnessed by powerful, primitive, and compensatory emotional forces. It is also possible that emergence of mandala imagery reflects reduced cortical connectivity, as discussed above. For example, evidence exists that during psychosis there is reduced transmission of visual information to anterior brain regions (Pasupathy 2006). This reduced visual input to higher cortical areas may create a form of functional sensory deprivation (e.g., Baggott et al. 2010), or a situation where there is top-down input to visual cortex that is relatively unconstrained by actual external stimulation (see Clark 2013), either of which would be associated with an increase in activity in visual centres.

Consistent with Goodwyn (2012), the emergence of mandala imagery during a psychological emergency represents the rendering of ideation and affect in an older, simpler, concrete, visual-spatial format (e.g., above-below, left-right, inside-outside, light-dark, etc.), reflecting the brain's predisposition to reflect experience in visual-spatial metaphors (Lakoff & Johnson 1999; see below, section on archetypes). By predisposition, I mean that the mental representation of spatial relationships is independent of visual input—as demonstrated in work with congenitally blind individuals (see Silverstein et al. 2013)—and is the basis for both visual experience as well as much higher-level cognition, such as an appreciation of logical relationships (Glezer 1995).

Is there evidence for such compensatory mechanisms in schizophrenia, however? It has been proposed, based on relationships between symptoms

and electrophysiological recordings during task activity, that a fragmentation and disorganization process is central to schizophrenia, whereas positive and negative symptoms are compensatory phenomena. In this view, hallucinations and delusions are attempts to create and organize meaningful sensory and cognitive representations out of a disorganized psyche, whereas negative symptoms represent a form of shutdown and withdrawal in the face of disorganization (Lee et al. 2003a, 2003b). Thus, emergence of spontaneous mandala imagery may represent an extreme form of compensation for disorganization, one that makes use of the basic visual processing capabilities that remain intact even in the presence of extreme levels of psychosis. From a Jungian perspective, appearance of the mandala image represents a sustaining vision of the archetype of the Self, whose appearance is meant to promote survival in the face of disintegration, given that ‘the quaternity is an organizing schema par excellence’ (Jung 1951/1970, para. 381; 1946/1985, para. 219).

If emergence of mandala imagery is a compensatory response, it would be expected that such imagery would exhibit some evidence of the psychological ‘lesion’ that is being compensated for, as well as evidence of compensatory activity. In this way, occurrences of mandala imagery can be viewed as analogous to irruptions of affect (but in symbolic form). As Jung noted:

Affects occur usually where adaption is weakest, and at the same time, they reveal the reason for a weakness, namely a certain degree of inferiority and the existence of a lower level of personality.

(1948/1970, para. 15)

Regarding visual imagery in general, Jung implied as much himself in a 1932 newspaper interview (Jung, 1932). There, he commented that, in the art of people with schizophrenia, ‘the main characteristic is one of fragmentation, which expresses itself in the so called “lines of fracture”—that is, a series of psychic “faults” (in the geological sense) which run right through the picture’. Although this has not been studied empirically, it can be confirmed by viewing the art of people with schizophrenia, as can be seen, for example, in the cover art for the journal *Schizophrenia Bulletin* (<http://schizophreniabulletin.oxfordjournals.org/content/current>), all of which is painted by people with the disorder. However, other research does support Jung’s view that the visual renderings of people with schizophrenia are different in predicted ways from those of other people. For example, people with schizophrenia are deficient in processing low spatial frequency information (i.e., the larger aspects of images that have consistent luminance and contrast, and that typically carry information about global form), whereas their processing of high spatial frequency information (e.g., fine details) is intact. This has been shown regarding objects and human faces (Calderone et al. 2013; Silverstein et al. 2010). And, as a compensatory response to this, it appears that the art of people with schizophrenia is characterized by *excessive* low spatial frequency information, compared to paintings of artists without schizophrenia (Graham & Meng 2011). This has been interpreted as a compensatory means

to represent the world in a way that feels more right than it appears visually, and could result in the appearance of an increase in divisions between parts of the image. In addition, a reduction in perception of global form would lead to excessive reliance on fine detail during visual perception. This has been observed in laboratory tasks of object (e.g., Silverstein & Keane 2011) and face (Silverstein et al. 2010) perception. Moreover, it can be detected in subjective reports of visual distortions of people's faces, where morphing is characterized by an increase in high spatial frequency information such as lengthening of teeth and increases in facial hair and wrinkles (Berndl et al. 1986; Dewdney 1973). Finally, the art of people with schizophrenia is often characterized by excessive detail, as exemplified by the paintings of Wölfl (Spoerri & Baumann 2003) and Louis Wain (although his diagnosis has been disputed) (McGennis 1999). In short, much clinical evidence, and preliminary research evidence suggests that mandala imagery and other imagery (as expressed in visual art) reflects both the fundamental breakdown in coordinated mental activity in schizophrenia, as well as efforts to compensate for this by imposing global structure and expressing information as fine details. However, research in this area is in its infancy.

Archetype constellation in schizophrenia

It has long been appreciated in the clinical literature that people with schizophrenia, especially during psychotic episodes, can be overwhelmed by archetypal material and can also identify with it to a pathological degree (e.g., Edinger 1955; Silverstein 2007). Indeed, Jung's ideas about the existence of 'primordial images' (which later become his construct of archetypes), were heavily influenced by his observations that, among psychotic (and prodromal) patients, a great deal of clinical material bore resemblances to themes from myths, religion, fairy tales, and art from cultures all over the world (Jung 1956; Jung 1939/1960, para. 527; Stevens 2006). But to what extent are Jung's observations about archetypes and their emergence in schizophrenia consistent with what we know today about brain function?

In a late paper on schizophrenia, Jung (1958a/1960) discussed Penfield and Jasper's demonstration that stimulation of the occipital cortex in a patient with epilepsy caused the perception of a circle within a square. Based on this, Jung suggested that these are 'the raw materials from which mandala symbols originate' (ibid., para. 582). And, 'I have long thought that, if there is any analogy between psychic and physiological processes, the organizing system of the brain must lie subcortically in the brain stem...I would conjecture that such a subcortical system might somehow reflect characteristics of the archetypal forms in the unconscious' (ibid.). Only parts of this statement are supported by current evidence. The brain stem is, of course, important for biological functions such as regulating the central nervous system and sensory processing. However, we now know that there are many other important structures, such as the amygdala, that are involved in generation and processing of emotion.

Also, it is now accepted that the basic visual features that form mandala images (e.g., lines, radial patterns, curved contours, see Figure 3), and visual images in general, are processed in regions of the occipital lobe (e.g., the pathway from V1 to the lateral occipital complex; Löffler 2008; Pasupathy 2006) and that processing of these basic features, as well as emergent features such as symmetry, is intact in schizophrenia despite multiple other visual processing impairments, including in binding separated elements into integrated representations (Knight et al. 2000; Silverstein et al. 2009; Silverstein & Keane 2011). Indeed, it is curious that, as the stimulus for Jung's comment above was a study of occipital lobe stimulation, he focused on the brain stem. However, while the basic visual components of archetypal imagery may emerge from the visual processing regions of the brain, the affective components and the final choice of images that are represented also undoubtedly reflect emotion processing centres and stored memories, respectively. That is, the constellation, or emergence, of material reflecting archetypes can also be viewed as the output of an organizing process, using the association field mechanism described above, but one on a 'grand scale' that goes beyond simple image creation to include memory traces and subcortical structures involved in emotional reactions.

Consistent with this, it has been suggested that archetypal images and feeling states can be viewed as relational structures (Brooke 1991; Goodwyn 2012). As noted by Fuster (2003): 'From the point of view of neurobiology, knowledge, memory, and perception share the same neural substrate: an immense array of cortical networks or cognits that contain in their structural mesh the informational content of all three' (p. 112). In these higher-level cases, the links between components of the representation are based, not on concrete dimensions as in vision (e.g., similarities in orientations of edge components), but on more abstract features such as word meaning, affect, and the sequence of events in an episodic memory representation, etc. Goodwyn (2012) discussed how it is quite common in evolution for newer, complex processes to be based on earlier, simpler processes, a view which he convincingly argues can account for why external events and internal states (including archetypal imagery and affect) are so strongly grounded in basic visual-spatial representations and metaphors (e.g., elation as an ascent; sadness as descent; love as a journey; progress as left to right movement; regression as right to left movement, etc.). Interestingly, recent work in neuroscience has highlighted the role of emotional and motivational significance in the formation of neural networks (e.g., Fuster 2003, p. 74).

Jung did not see archetypes as involving hard-wired images or responses, but rather predispositions to organize sets of information, feelings, and response tendencies in certain ways under certain psychologically powerful conditions. Knox (2003) makes the distinction between pre-experiential potentialities, which form the basis of innate structures, and post-experiential constructions, which form the basis of internal working models. This echoes the distinction between the 'archetype-as-such' and the 'archetypal image' (Jung 1947/1988; McDowell 2001; Rodriguez 2003). In support of this, McDowell (2001)

reviewed several lines of biological evidence indicating that images are not inherited in the form of predetermined connections between cortical neurons.

The means by which neural networks emerge—both those that develop over the evolution of a species, and those that develop over the course of a lifetime—have been described by Edelman (1987, 1989), using the concept of ‘group selection’. In his model, groups or networks of cortical neurons out of all possible neurons and combinations respond to environmental stimuli and support a variety of representational functions. Neurons and groups of neurons not used in this way eventually disappear. Neurons chosen initially will reflect the history of the organism, based on those patterns of brain activity that have been associated with adaptive responses to classes of external stimulation. In this way, it can be seen that the neural basis of an archetype is likely to be established very early in development, via the combination of evolutionarily ‘selected’ networks related to survival and security (Goodwyn 2012; Halpern & O’Connell 2000) and personally experienced imagery. The network (or large scale association-field including affect, memories, and imagery) that is formed is sustained through repeated activity, and also through re-entry from other cortical areas, leading to the formation of secondary networks that are even more precise in their representation of the situation, its significance to the individual, and its response strength (Fuster 2003). Moreover, the network can be activated by only a portion of its components [the concept of *degeneracy* (Edelman 1987)], which can be seen as the basis for activation of a complex or archetypal material across a wide range of situations. Because activation can come from internally generated activity alone, it follows that abnormal mental activity (e.g., that associated with a complex) can perpetuate a vicious cycle or reactivation of personally meaningful associations and mental states (e.g., paranoid ideation) that become increasingly independent from what is happening in the external world (Fuster 2003). Moreover, as noted above, in schizophrenia, where the overall level of connectivity is reduced, networks that would not normally be sustained may become self-sustaining attractor states or parasitic foci, leading to heightened constellation of archetypal activity. In short, Jung’s views on the nature of archetype constellation have received support from evolutionary psychology and neurobiology (reviewed in Goodwyn 2012), although much more work needs to be done in this regard.

Psychological vs. toxic aetiology of schizophrenia

The development of Jung’s thinking about the aetiology of schizophrenia foreshadowed current work on the effects of adverse environments (e.g., childhood sexual abuse, poverty, social defeat) and dysfunctional cognitive schemata (e.g., negative beliefs about oneself) on brain development and function, and on the risk for schizophrenia (Fisher et al. 2013; van Os et al. 2005). At the time Jung worked at the Burghölzli, it was widely assumed that dementia praecox was caused by a biological abnormality, and this was the

view promulgated by Kraepelin (see Noll 2004, 2007). However, theories at that time focused on toxic effects on the brain secondary to abnormalities in other parts of the body (e.g., sex glands, intestines). In contrast, Jung's focus was on the effects of stress and intense affect. For example, as early as 1907, he stated that 'it is possible that in the disposition to dementia praecox, affectivity brings about certain irreparable organic disturbances, as for instance, metabolic toxins' (1907/1960, para. 1067). In that same work he noted that complexes could not be considered the only cause of schizophrenia and that a biological predisposition towards heightened affect (and therefore, toxic consequences of that affect) could be important. This has been confirmed by studies demonstrating increased stress reactivity in people with schizophrenia (Myin-Germeys & van Os 2007), and links between increased stress reactivity and emergence of psychotic symptoms in the general population (Collip et al. 2013). Speculating on even more of a primary role for an unknown toxin he referred to as 'X', he added 'at the same time I am fully aware of the possibility that this X may arise in the first place from non-psychological causes and then simply seize on the existing complex and specifically transform it, so that it may seem as if the complex had a causal effect' (1907/1960, para. 195). He noted, however, that whatever the original cause, the complex becomes ingrained to an excessive degree in the psyche. Importantly, Jung noted that intense affect in schizophrenia is not always visible to an outside observer, but can be largely unconscious and lead to compensatory reactions such as apathy. This is consistent with research demonstrating that even in patients with a high level of negative symptoms (e.g., flat affect, apathy, poverty of speech), there can be increased levels of psychophysiological reactivity (e.g., Kring & Neale 1996).

Jung hypothesized that one effect of the toxin is to decompose complexes into their component parts (1958a/1960, para. 581). It has already been noted that schizophrenia is characterized by reduced grouping of component parts of visual images (see Silverstein & Keane 2011 for review), which is consistent with the idea of network strength reduction. Whether complexes, as high-level association fields involving both imagery and affect, also deteriorate over time, as Jung hypothesized, is an open question. However, longitudinal data indicate a common profile of deterioration over the long-term, from paranoia (where, in theory, complexes are strong) to disorganization (where, in theory, complexes are weak) (McGlashan 1998). Moreover, multiple aspects of disorganization become increasingly related over time (Marengo et al. 2000) and these symptoms are associated with progressive losses in brain volume over time (Collin et al. 2012), consistent with the idea of a deteriorating condition.

Jung's emphasis on the relative importance of psychological versus biological causation of schizophrenia appeared to change over time, despite his saying, in 1939, that 'I could not say that my standpoint has had to undergo any radical change' (1939/1960, para. 504). For example, Jung stated later in the same 1939 paper that 'I admit that I cannot imagine how something "merely" psychic can cause an *abaissement* which destroys the unity of personality, only too often

beyond repair' (ibid., para. 541) while also noting that 'many relapses as well as improvements are due to psychological conditions' (ibid., para. 533), or to 'psychic facts which one would not hesitate to declare causal in a case of neurosis' (op. cit.). Finally, in the same paper he noted that it was impossible to prove, at that point in time, that schizophrenia is either an organic disease to begin with, or that it has an exclusively psychological origin. Near the end of his life, in 1956, at the age of 81 in a 'Voice of America' radio broadcast, Jung said,

Inasmuch as we have been unable to discover any psychologically understandable process to account for the schizophrenic complex, I draw the conclusion that there might be a toxic cause. That is, a physiological change has taken place because the brain cells were subjected to emotional stress beyond their capacity...I suggest that here is an almost unexplored region, ready for pioneering research work.

(*Time Magazine* 1956)

However, in 1958, he stated that: 'I have now, after long practical experience, come to hold the view that the psychogenic causation of the disease is more probable than the toxic causation' (1958a/1960, para. 570). As evidence of this, he cites many mild cases which begin after psychological stress, and also that can be cured by psychological methods. He also noted that he has even seen this in severe cases, but also commented that 'my material consisted for the most part of milder, still fluid cases and of latent psychoses. I do not know, therefore, how it is with those severe catatonias, for instance, that may have a lethal outcome and naturally do not appear in the psychotherapist's consulting-room. Consequently, I must leave the possibility open that there may also be schizophrenias for which a psychogenic aetiology can be considered only in minimal degree or perhaps not at all' (ibid., para. 577). Interestingly, many of these milder cases would not meet modern diagnostic criteria for schizophrenia. Perhaps the best summary of Jung's view is this:

To make myself clear, I consider the aetiology of schizophrenia to be a dual one: namely, up to a certain point psychology is indispensable in explaining the nature and the causes of the initial emotions which give rise to metabolic alterations. These emotions seem to be accompanied by chemical processes that cause specific temporary or chronic disturbances or lesions.

(1958b, p. 194)

Consistent with this idea of a dual aetiology, Jung saw the need to study and treat schizophrenia at both the biological and psychological levels, noting that it

has two aspects, physiological and psychological, for the disease, so far as we can see today, does not permit of a one-sided explanation. Its symptomatology points on the one hand to an underlying destructive process, possibly of a toxic nature, and on the other—inasmuch as a psychogenic aetiology is not excluded and psychological treatment (in suitable cases) is effective—to a psychic factor of equal importance. Both ways of approach open up far-reaching vistas in the theoretical as well as therapeutic field.

(1957/1960, para. 552)

As discussed below, evidence supports the importance of both psychological and biological factors in the development of schizophrenia.

What is the evidence for a toxic cause in schizophrenia? Bercel (1960) reviewed early evidence indicating that blood serum from schizophrenia patients had toxic effects on animals (e.g., alteration of spider web design) and plants. However, these studies were poorly controlled and this line of research has not continued. Throughout the second half of the 20th century, the most popular theory of schizophrenia was the dopamine hypothesis, originally suggested by clinical and research evidence of amphetamine (a dopamine agonist) psychosis, as well as by the ability of dopamine receptor blocking medications to reduce psychotic symptoms (Carlsson & Lindqvist 1963; Snyder 1976). While dopamine, as one of the major neurotransmitters, cannot be considered a toxin, excess dopaminergic activity may have toxic effects. For example, when dopamine turnover is excessive, D2 receptors (those most involved in psychotic, as opposed to movement-related symptoms) can morph into a high affinity state, which has been referred to as a D2High receptor (Seeman 2008). Recent research has clarified the extent to which various substances can lead to this transformation. Of those studied, corticosterone (the rat analogue to cortisol, the human 'stress hormone') was the major contributor to moving D2 receptors into the high-affinity state. For example, sensitization by corticosterone led to a 210% increase in the proportion of D2High receptors, whereas the rates for cocaine and caffeine were 160% and 125%, respectively. One of the known effects of this is to disrupt hypothalamic-pituitary-adrenal (HPA) axis functioning, in the sense of reducing negative feedback that normally shuts off the stress response (in order to prevent an unnecessarily prolonged fight or flight response). An additional effect of chronic corticosterone or cortisol release is cell death in the hippocampus, a subcortical structure critical to memory formation and aggression control, where size reductions have been observed in schizophrenia and in other conditions of chronic stress (Bremner & Narayan 1998; Shepherd et al. 2012).

Perhaps the leading theory of schizophrenia in the 21st century (thus far) is the glutamate (an excitatory neurotransmitter) hypothesis of schizophrenia, which attributes much of the disease process to a combination of: 1) hypoactivity at the NMDA form of glutamate receptor (the receptor most responsible for learning and short- and long-term binding of neural assemblies based on spatial, temporal, and conceptual relationships); 2) subsequent reduced excitation of GABA-ergic (inhibitory) interneurons; and therefore 3) reduced inhibition of excitatory glutamatergic activity on pyramidal cells (the most prevalent form of cortical cell) (Olney & Farber 1995; Moghaddam & Javitt 2012), leading to overstimulation of neurons (as hypothesized by Jung). It has been shown that a primary impairment in NMDA receptor activity can cause acute dopaminergic increases. In addition, however, excess glutamatergic activity is excitotoxic and can cause damage to the hippocampus, such as cell death and volume reduction (Schobel et al. 2013), and this cascade can be triggered by stress (Moghaddam 2013). In short, the evidence from studies of dopaminergic and glutamatergic function in schizophrenia

supports Jung's views of: 1) the role of stress and intense affect in causing changes in brain function; 2) evidence for a toxic effect in the brain resulting from intense affect; and 3) the presence of a mechanism whereby there could be continued deterioration of neural networks over time. Especially important here is the role of subcortical structures, especially the hippocampus, in forming the associations between cortical cell populations that are critical to the formation of memories (Fuster 2003), and therefore the sense of self, and the excitotoxic atrophy of this structure caused by a prolonged stress response.

There is currently much evidence indicating the aetiological role of infectious and inflammatory processes in schizophrenia. This work has been extensively reviewed numerous times in just the past few years (e.g., Feigenson *et al.* in press; Suvisaari & Mantere 2013; Yolken, Dickerson & Fuller Torrey 2009). In brief, evidence is accumulating for: 1) elevated rates of infection prior to first episode psychosis in people with schizophrenia (Benros *et al.* 2011, 2012; Morris 1996; Khandaker *et al.* 2012); 2) infection and autoimmune abnormalities preceding relapse (Benros *et al.* 2011, 2012; Miller *et al.* 2013); 3) elevated rates of maternal infection during pregnancy in people who go on to develop schizophrenia (Brown & Derkits, 2011); 4) increased blood levels of inflammatory markers in people with schizophrenia, including at or prior to the first episode (Niebuhr *et al.* 2008); 5) effects of antibodies and inflammation on increasing dopaminergic activity (Yolken *et al.* 2009); 6) positive effects of anti-inflammatory agents (e.g., aspirin, celecoxib, fish oil high in Omega-3 fatty acids) in reducing psychotic symptoms (Torrey & Davis 2012); and even 7) the ability of anti-inflammation treatment to prevent the development of schizophrenia in young people at high risk from the disorder (Amminger *et al.* 2010). This evidence is of course consistent with Jung's view that a toxic process is at work in schizophrenia.

Earlier, it was noted that, in 1907, Jung suggested that a biological predisposition towards heightened affect and its toxic consequences could be important in the aetiology of schizophrenia. Thus, Jung can be seen as proposing the first example of a stress-vulnerability model for schizophrenia. Evidence for such an interaction comes from genetic studies. For example, among children of mothers with schizophrenia who are adopted by other families very early in life, those who are raised in enriched environments (e.g., with low conflict and available opportunities to pursue creative outlets) have a lower than expected rate of schizophrenia (i.e., they are protected *against* schizophrenia), whereas those raised in distressed environments (e.g., characterized by family conflict, intrusiveness, and communication deviance) have a higher than expected rate of developing the disorder (Wynne *et al.* 2006a, 2006b). These data indicate that the biological diathesis for schizophrenia may include an exaggerated neuroplasticity in response to environmental stimuli, at least prior to the onset of psychotic symptoms (after which plasticity may be reduced, as noted above). While this would be adaptive in enriched environments and could lead to creativity, it is a serious vulnerability in stressful environments, as well as being, consistent with Jung's theory, a setting condition for overly intense affect and its pathological consequences.

Finally, as suggested by Jung, recent evidence is demonstrating that risk for the development of schizophrenia is significantly increased under conditions of chronic stress (which are the conditions that can lead to excitotoxic effects causing hippocampal cell death). This evidence has also been reviewed extensively in recent years, and conditions linked to later schizophrenia include: physical and sexual abuse in childhood; bullying; racial discrimination; poverty; immigration; and other instances of chronic 'social defeat' (e.g., van Os, Kenis & Rutten 2010; Morgan et al. 2013; Wicks et al. 2005), as well as high levels of negative expressed emotion from friends and caregivers (Bebbington & Kuipers 1994; Berry et al. 2011). Of course, we know now that environmental effects are not purely psychological, but exert powerful effects on brain and somatic functioning, and that the extent of these interactions is moderated by genetic predisposition (e.g., Peerbooms et al. 2012). Nevertheless, it is remarkable that Jung was the earliest proponent of a comprehensive theory of schizophrenia that included a psychological aetiology, and that today this insight is considered a basic assumption in the field of schizophrenia research.

Conclusions

The main theme that emerges from Jung's work on schizophrenia is that it can be understood at both the psychological and biological levels. At the time of his 1907 book, the aspect of this that was most groundbreaking and important was the idea that schizophrenia has any kind of psychological basis at all, let alone one that might be primary. These issues are still very relevant today. Indeed, the field appears to be moving away from a sole focus on 'the broken brain' (Andreasen 1985), to an understanding of the interaction between a person's cognitive schemata and attributional style, the quality of the interpersonal environment, chronic stress and its effects on the brain, and genetic and other biological (e.g., inflammatory, neurocircuitry) factors that may lead to heightened stress reactions and their excitotoxic consequences (Morgan et al. 2013). So, while Jung's views that schizophrenia may have a psychological basis, that its symptoms express this basis, and that this behoves clinicians to develop relationships with patients and to work with this material may not seem revolutionary today, it is important to recognize how new these ideas were and how they have influenced clinical work for schizophrenia over the past 100 years. This is not to say that Jung's approach is widely used today. Indeed, psychotherapy and other psychological treatments for schizophrenia (especially those that are evidence-based) are still grossly underutilized (Silverstein, Spaulding & Menditto 2006).

In recent years, it has been suggested that there should be a renewed approach to analytical psychology, one that involves a closer association with the natural sciences (Goodwyn 2012; Haule 2010; Knox 2003). Although it seems increasingly common for Jungian concepts to be translated into the language of quantum physics (e.g., Koestler 1972; Stevens 2006) or chaos and complexity theories (e.g.,

Cambray 2009), our understanding of key Jungian ideas from the perspective of brain function is only beginning, despite Jung's many statements on the links between biology (e.g., instinct, brain function) and psyche. A more concerted effort to understand and evaluate the validity of Jung's concepts in terms of available evidence from neuroscience could lead both to important advances in analytical psychology, in addition to advances in treatments for schizophrenia and other disorders. In doing this, I suggest a good starting point is Jung's understanding of the complex, and how it can be viewed as a 'derivative gestalt' or 'cognit' (both terms from Fuster 2003) defined by relationships between aspects of perception, memory, knowledge, and affect. Since perception, memory, and knowledge themselves can all be viewed as involving the same form of circuitry and algorithms, a complex can be seen as a high-level association field, an hypothesis supported by much neurobiological evidence (reviewed in Phillips & Singer 1997; Fuster 2003). The study of how the brain creates basic gestalts, whether in perception, memory, knowledge, or their combination into higher-level structures such as complexes, is certainly relevant to understanding schizophrenia, where a breakdown in this process at multiple levels has been demonstrated (for reviews, see Phillips & Silverstein 2003; Uhlhaas & Silverstein 2005; Silverstein & Keane 2011). And, as described above, a breakdown in gestalt creation sets the stage for the development of 'parasitic' (Hoffman & McGlashan 1993) attractor states (i.e., complexes, including those putatively involved in psychotic symptom generation) as well as for their eventual deterioration over time in the large group of patients who become increasingly disorganized as they age (McGlashan 1998).

It is important to note that Jung's contributions do not simply represent re-statements of Bleuler's positions. In several important respects (e.g., the validity of the concept of *abaissement du niveau mental*; the possibility of psychogenic causation of schizophrenia; the issue of whether uncontrollable affect is due to ego disintegration), they disagreed, and Jung's theory of the complex clearly influenced Bleuler. An interesting example of Jung's perceived differences with Bleuler is found in a 1939 (1939/1960) comment regarding the latter's view that a psychological cause can produce only symptoms but not the disease itself: 'This statement may be profound or the reverse' (para. 534).

Given the degree to which Jung's ideas anticipated much current thinking about the cognitive neuroscience of schizophrenia, and the degree to which they can inform future work in this area, why is his work on schizophrenia so rarely taught, or cited in theoretical or research papers? Perhaps Jung's reputation as a heretic within the traditional psychoanalytic movement, and as a mystic within mainstream psychiatric circles has perpetuated a resistance to reading and/or citing his work. There also appears to have been little interest among Jungians in working with people with schizophrenia (Couteau 1988). However, as has been demonstrated countless times, overcoming resistances can lead to meaningful progress. With the current (re)convergence of psychological and biological approaches to understanding and treating the disorder, the pioneering ideas of Jung in this regard remain a useful and rich, but still underutilized resource.

TRANSLATIONS OF ABSTRACT

Les écrits de Jung sur la schizophrénie sont presque complètement ignorés ou oubliés de nos jours. L'objet de cet article, de même que celui de l'article suivant, est de passer en revue les principaux thèmes trouvés dans les écrits de Jung sur la schizophrénie, et d'évaluer la validité de ses théories à propos de ce trouble en s'appuyant sur la base des connaissances actuelles dans les champs de la psychopathologie, des neurosciences cognitives et des recherches en psychothérapie. Dans cet article, cinq thèmes relatifs à l'étiologie et à la phénoménologie de la schizophrénie dans les écrits de Jung seront discutés: 1) *abaissement du niveau mental*; 2) le complexe; 3) images de mandala; 4) constellation des archétypes et 5) étiologie psychologique vs toxique de la schizophrénie. L'étude des thèmes ci-dessus amène trois conclusions. D'abord, à maints égards, les idées de Jung sur la schizophrénie ont anticipé de nombreuses idées et données actuelles sur ce trouble. Deuxièmement, avec la récente (re) convergence des approches psychologiques et biologiques dans la compréhension et le traitement de la schizophrénie, les idées pionnières de Jung au sujet de l'importance de ces deux facteurs et de leur interaction demeurent une ressource utile et riche, mais encore sous-employée. Enfin, un effort plus concerté pour comprendre et évaluer la validité des concepts de Jung avec l'étayage des neurosciences pourrait conduire à des avancées importantes en psychologie analytique ainsi qu'à des évolutions dans les approches thérapeutiques qui pourraient s'étendre au-delà du traitement de la schizophrénie.

Jungs Schriften zur Schizophrenie werden heute vollständig ignoriert oder sind vergessen. Der Zweck dieses Artikels, zusammen mit einem nachfolgenden Aufsatz, ist es, die in Jungs Schriften über Schizophrenie auffindbaren zentralen Themen durchzusehen und die Gültigkeit dieser Theorien über die Störung im Lichte unseres gegenwärtigen Wissensstandes auf den Gebieten der Psychopathologie, der kognitiven Neurowissenschaften und der Psychotherapieforschung einzuschätzen. In diesem Artikel werden fünf Themen diskutiert, die in Beziehung zur Ätiologie und zur Phänomenologie der Schizophrenie stehen, wie sie in Jungs Schriften zu finden sind: 1. *abaissement du niveau mental*, 2. der Komplex, 3. Mandaladarstellungen, 4. archetypische Konstellationen, 5. psychologische vs toxische Ätiologie der Schizophrenie. Nachprüfungen der genannten Gebiete legen drei Schlußfolgerungen nahe. Zunächst ist festzustellen, daß Jungs Überlegungen zur Schizophrenie auf mancherlei Weise viel vom heutigen Denken und Wissen über diese Störung vorweggenommen haben. Zweitens erweisen sich angesichts der neuerlichen (Re-)Konvergenz von psychologischen und biologischen Herangehensweisen an ein Verständnis und die Behandlung der Schizophrenie die wegweisenden Ideen Jungs bezüglich der Wichtigkeit beider Faktoren und ihres Zusammenspiels als nützliche und reiche, aber noch immer wenig genutzte Ressource. Schließlich könnten besser abgestimmte Bestrebungen zum Verstehen und Beurteilen der Gültigkeit von Jungs Konzepten zu den Evidenzbedingungen der Neurowissenschaften sowohl zu wichtigen Fortschritten der Analytischen Psychologie, wie auch zu Entwicklungen therapeutischer Ansätze führen, die über die Behandlung der Schizophrenie hinausreichen würden.

Oggi gli scritti di Jung sulla schizofrenia sono completamente ignorati o dimenticati. Lo scopo di questo scritto, insieme ad un ulteriore articolo, è quello di rivedere le tematiche primarie

trovate negli scritti di Jung sulla schizofrenia e di accertare la validità delle sue teorie sul disturbo alla luce della base delle conoscenze attuali nei campi della psicopatologia, delle neuroscienze cognitive e nella ricerca psicoterapeutica. In questo articolo vengono discussi cinque temi degli scritti junghiani relativi all'eziologia e alla fenomenologia della schizofrenia: 1) *l'abbassamento del livello mentale*; 2) il complesso; 3) le immagini di mandala; 4) costellazioni di archetipi; 5) psicologico vs tossica eziologia della schizofrenia. Revisioni delle areesuccitate suggeriscono tre conclusioni. Primo le idee di Jung sulla schizofrenia anticipano in molti modi vari pensieri attuali e dati sul disturbo. Secondo, con la recente (ri)convergenza di approcci psicologici e biologici nella comprensione e nel trattamento della schizofrenia le idee pioniere di Jung riguardanti l'importanza di entrambi i fattori e la loro interazione rimangono una risorsa utile e ricca, ma ancora non utilizzata. In ultimo, uno sforzo più concentrato da parte delle neuroscienze per comprendere e valutare la validità dei concetti junghiani in termini di evidenza potrebbero entrambi portare ad avanzamenti importanti nella psicologia analitica e a sviluppi negli approcci terapeutici che potrebbero estendersi oltre la cura della schizofrenia.

Цель этой статьи—сделать обзор изначальных тем, присутствующих в работах Юнга о шизофрении, и дать доступ к ценности его теорий об этом расстройстве в свете наших актуальных знаний в области психопатологии, когнитивной нейронауки и психотерапевтических исследований. В этой статье обсуждаются шесть тем, связанных с этиологией и феноменологией шизофрении из работ Юнга: 1) *abaissement du niveau mental* (снижение уровня мышления); 2) комплекс; 3) образы мандал; 4) констелляция архетипов и; 5) Психологизм версус токсичная этиология шизофрении. Обзор вышеуказанных областей приводит к трем заключениям. Во-первых, во многом идеи Юнга о шизофрении превосходили нынешние мысли и данные об этом расстройстве. Во-вторых, в свете недавней (повторной) конвергенции психологического и биологического подходов к пониманию и лечению шизофрении, первооткрывательские идеи Юнга относительно значимости обоих факторов и их взаимодействия остаются полезным и богатым, но все еще не полностью используемым ресурсом. Наконец, более согласованные усилия по пониманию и оценке ценности концепций Юнга с точки зрения научной неврологической обоснованности могут привести как к важным достижениям в аналитической психологии и к развитию новых терапевтических подходов, которые будут использоваться не только в лечении шизофрении, но и шире.

Los escritos de Jung sobre la esquizofrenia son casi completamente ignoradas y olvidadas hoy en día. El propósito de este documento, junto con un artículo para seguir, es revisar los principales temas que se encuentran en los escritos de Jung sobre la esquizofrenia, y para evaluar la validez de sus teorías sobre este trastorno a la luz de nuestra base de conocimientos en los campos de la psicopatología, la neurociencia cognitiva y la investigación en la psicoterapia. En este artículo se analizan cinco temas relacionados con la etiología de la esquizofrenia y la fenomenología de los escritos de Jung: 1) *abaissement du niveau mental*; 2) el complejo; 3) Las imágenes de mandala; 4) La constelación de arquetipos y; 5) psicológica vs tóxica etiología de la esquizofrenia. Los estudios de las áreas mencionadas sugieren tres conclusiones. En primer lugar, en muchos aspectos, las ideas de Jung a la esquizofrenia precede en

mucho al pensamiento actual y los datos sobre el trastorno. En segundo lugar, con la reciente (re)convergencia de enfoques psicológicos y biológicos para entender y tratar la esquizofrenia, las ideas pioneras de Jung sobre la importancia de ambos factores y su interacción sigue siendo un útil y rico recurso, aún infrautilizados. Por último, un esfuerzo más concertado para comprender y evaluar la validez de los conceptos de Jung en términos de pruebas de neurociencia puede conducir a importantes avances en psicología analítica y con la evolución de los enfoques terapéuticos se extenderían más allá en el tratamiento de la esquizofrenia.

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