

The Phenomenology and Neurobiology of Delusion Formation During Psychosis Onset: Jaspers, Truman Symptoms, and Aberrant Salience

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Following the publication of Karl Jaspers' *General Psychopathology* (1913), delusions have been characterized as being nonunderstandable in terms of the person's biography, motivations, and historical-cultural context. According to Jaspers, this loss of understandability is due to an underlying neurobiological process, which has interrupted the normal development of the individual's personality. Inheriting the 19th-century division between the natural- and human-historical sciences, Jaspers emphasizes the psychological understanding of mental disorders as narrative-based, holistic, and contextual. By doing so, he embraces cultural, ethnic, and individual differences and anticipates a person-centered medicine. However, he also affirms the value of explanatory neurobiological approaches, especially in the research and diagnosis of delusions. The phenomenological approach leads to neurobiological hypotheses, which can be tested experimentally. The present article addresses these issues by illustrating Jaspers' fundamental contribution to current neurobiological research concerning the formation of delusions during early phases of psychosis. Specifically, we present delusional mood and Truman symptoms as core phenomenological features at the origin of psychosis onset, and we discuss their neurobiological substrate with the aberrant salience and dopamine dysregulation models. Jaspers and his successors' phenomenological approach suggests that delusion is formed through loss of context in its experiential-perceptual origins. This is consistent with the more recent neurobiological models.

Key words: psychosis/schizophrenia/delusion/phenomenology/at risk/prodromal

Introduction

Karl Jaspers (1883–1969) is recognized as the first major psychiatrist to bring scientific foundation to psychopathology.¹ He is also seen as the first to bring order to delusion research

in German-speaking psychopathology.² All that followed on the topic, supportive or critical, may be considered a kind of footnote to his work.³ In the present article, published in a special issue devoted to the centenary of Jaspers' *General Psychopathology* (*Allgemeine Psychopathologie* [AP], 1913),⁴ we describe the phenomenological bases of delusion formation, with a specific focus on early phases of psychosis. We illustrate how seminal concepts developed by Jaspers 100 years ago may be useful in developing current neurobiological models of delusion onset investigated by clinical neuroscience. Our aim is to demonstrate the fundamental impact and continued importance of AP to current clinical research and practice, especially prodromal and early psychosis.

Primary Delusions vs Delusion-Like Experiences

In psychiatry, Jaspers is known for his masterwork AP (1913), in which he proposes 3 crucial criteria for diagnosing delusions:

1. subjective certainty, incomparable to other convictions;
2. imperviousness to counterarguments;
3. implausibility of content.^{4(p45),5(p80),6(pp95–6)}

Nevertheless, Jaspers states that these criteria are merely external. They do not help us understand delusion formation phenomenologically in terms of the patient's subjective experience. They are also not helpful in defining delusions operationally⁷ nor with the practical problem of how to differentially diagnose a genuine, primary delusion (*echte Wahnidee*) from merely delusion-like ideas (*wahnhaftes Idee*) and, therefore, whether we are dealing with schizophrenia or personality disorder. Jaspers' answer is that we must consider the patient's original subjective experience (*Wahnerlebnis*), which gives rise to primary delusion. This, however, presents enormous methodological problems. Because we are unable to

directly see into another person's mind and the original delusional experience, we are dependent on patient reports: "The content of the delusions which the patient may disclose to us in the course of the interview is always a secondary product. . . . We are always confronted with the question—what is the primary experience traceable to the illness."^{5(p80),6(p96)} Every verbal report is mediated by the patient's memory, which is a possible confound. Moreover, as one of Jaspers' patients expresses it—these experiences are "uncommonly difficult to describe in words, in part frankly impossible."^{8(p1319)} Therefore, Jaspers must develop a reliable method that does not exclusively rely on such reports for the differential diagnosis of primary delusions.

The Cultural Environment Surrounding Allgemeine Psychopathologie

When Jaspers first arrived in 1908 as a voluntary assistant at the Heidelberg psychiatry clinic,⁹ he missed Kraepelin who had just left the directorship at Heidelberg to become Chair at Munich. Still, "the common conceptual framework of the hospital was Kraepelin's psychiatry along with deviations."^{9(pp7,15)} At this time, Kraepelin's proposal that dementia praecox (parallel to the progressive paralysis of neurosyphilis) is a unitary clinical entity with common course and outcome was challenged on several fronts.¹⁰ The Tübingen psychiatrist, and previously Kraepelin's assistant, Gaupp¹¹ proposed that delusions of reference indicating paranoia may be reactive, based on a psychasthenic, obsessive character, and had a psychological basis. Writing his Habilitation thesis under Gaupp, Kretschmer¹² described these delusions as arising from a sensitive personality, who—as Jaspers paraphrases Kretschmer—is "tender, thin-skinned but full of self-conscious ambition and obstinacy."^{6(p409)} Additionally, Freud¹³ had proposed that paranoid delusions could be explained by repressed unconscious contents. For Jaspers, it was critical to distinguish delusions due to character, or reactive to experience, from those involving an underlying, yet-to-be-discovered neurobiological process (for reviews, see Westerterp¹⁴ and Berze and Gruhle¹⁵).

Schizophrenia Process as Nonpsychologically Derivable Change

In his early 1910 study, Jaspers¹⁶ notes that jealousy delusions may be indicative of personality disturbances and/or alcohol-related disorders but may involve something entirely different, a schizophrenia psychosis based on unknown neurobiological process. In the former case, the delusion emerges slowly. If there is an acute phase, "the personality, so far as one can judge, remains unchanged"^{16(p111)} (our translation). Following this, the previous status quo is reestablished. The whole occurrence may be understood as a change or development of

personality.^{16(p121)} In contrast, there are cases that indicate a process schizophrenia in which there is an irreversible, inexplicable, suddenly emergent change. The change persists, and, in contrast, has poor prognosis. *Something entirely new enters without any connection to what was before.* There is constant interrupting of the previous personality with "heterogeneous moments . . . random irregularities." These cannot be derived psychologically from the personality but are secondary to some "brain disorder."^{16(p121)} The "previous personality may all at once completely disappear." This may lead to an entirely "new personality, developing in its own way analogously to the original one."^{16(pp.117,121)} In other words, *the neurobiological process interrupts the development of personality with something completely foreign.* At the point where the self is threatened by losing continuity with what was before, the patient may develop a "double" in the form of a shadowy presence (a nonperceived, embodied presence [*leibhafte Bewusstheit*]). The double is *felt*—but not seen—to be in the self's proximity (the "feeling of a presence"). This may indicate an early, prodromal transitional step in delusion formation. Jaspers reports a patient who says: "I had the feeling that somebody was inside me and—how would you say—left me by my side? . . . If I stood up, he stood up. If I started to walk, he started to walk. He always remained at the same place [behind me]. If I turned around to see him, he also turned around at the same time so that I could never see him . . . ' Nevertheless, the patient feels himself observed [by the presence] and insists that it is not an illusion "^{17(p415)} (our translation; for review, see Mishara¹⁸). By invoking the opposition *development of personality* vs schizophrenia *process* based on unknown neurobiological change, Jaspers was able to distinguish delusion-like ideas from primary delusions. The latter are nonderivable from any psychological continuity in the patient's personality and thus indicate a process schizophrenia. Still, Jaspers' descriptive psychopathology required a method to distinguish the different delusion types. The Heidelberg psychiatrist, Gruhle, who influenced Jaspers' writing *AP* with "sharp" criticisms,^{19(p21)} later writes: "What is critical at this point is to demonstrate what is specific to schizophrenic delusions."^{15(p123)} Jaspers would find the answer in the clinical interaction itself.

The Diagnosis of Delusions Is Interactive

Jaspers proposed that delusion-like ideas, eg, Kretschmer's sensitive delusions of reference, could be understood in terms of the psychological connectedness made to form these ideas, a psychologically understandable personality development. In contrast, primary delusions cannot be understood in terms of prior psychological origin or motivation. They seem to come from nowhere and cannot be derived from anything else.^{4-6,16} As "something ultimate" (etwas Letzes),^{4(p45),5(p80)} a new "original phenomenon"

Table 1. Steps in the Clinician's Experience of Diagnosis Parallel Stages of Delusion Formation in the Patient

Steps of understanding in the diagnosing clinician	Parallel processes in the the patient
1. Naïve realism Default self-perspective is one's own common habitual world	1. Loss of common sense (Blankenburg), or default naïve realism Delusional mood, marked artificiality of the environment, "Truman symptoms"
2. Effortful perspective taking (to transcend default self-perspective) Ethnic, cultural, and individual diversity is embraced	2. Loss of ability to shift/transcend current perspective (Binswanger, Blankenburg, and Conrad) Temporal shrinking to the present in which the moment takes precedence over continuity (aberrant salience)
3. Recourse to neurobiological explanation Clinician unable to understand the patient's frame of reference contextually Explains this loss of context or psychological connectedness in terms of an underlying neurobiological process	3. Separation of the delusional theme from the inner life story or history Everything so "new" (aberrant salience) Continuity with old self lost and delusions are encoded acontextually Delusional theme spreads to larger and larger spheres of the patient's life (reconsolidation of the delusional theme) New delusional themes emerge (delusion's absoluteness, incorrigibility preserved but shifted to new themes; Binswanger and Blankenburg)

Note: Although this process can only be determined interactively, it also points to the underlying neurobiology.

(Urphaenomenon), they interrupt "normal" continuity of personality, directly reflecting a brain disturbance. In order to differentiate these 2 possibilities, diagnosis needs to be interactive according to the following steps (see [table 1](#)).

Naïve Realism. In everyday experience, we believe that what we directly see and experience is real. Jaspers writes "we live uncritically ... in an *immediate* world,"^{19(p171)} (our translation) a world given to us naïvely and effortlessly.²⁰ We remain blind to the interpretive lens through which we experience this "immediate" world, the product of implicitly functioning, unnoticed biases. "The other (foreigner) is misunderstood, reduced to the motives and goals of one's own world."^{19(p171)} In "common sense,"²¹ "we often seem to [directly] experience another's feelings, intents or traits. ... Using an intuitive ... relatively automatic process, people do not think about making attributions, they just do it."^{22(pp84,100)} We believe our perceptions and judgments, being a direct reflection of reality, are more objective and less biased than those of others.²³ We experience our own actions as arising in response to a situational context but attribute others' behaviors as due to their traits and dispositions, overlooking the situational contextual factors they experience from their perspective.²⁴ Our default mode of reasoning about others is biased toward a self-perspective.²⁵ Jaspers writes: "As matter of course, we take the current moment, what is familiar, the more or less stable social milieu, and our internal mental life ... as the only one that exists. Everything else is naïvely experienced and judged as fully in harmony with our perspective"^{19(p171)} (our translation).

Empathy as Effortful Perspective Taking. Now, it is not the case that we always understand others' behaviors from the unquestioned context of naïve realism. We shift from habitual to effortful attributions about the reasons for others'

behavior when we are motivated to do so or when their motives are not obvious. We shift our attention from our own experiential context to their inner frame of reference, their "subjective psychological experience."¹⁹ By intuitively reproducing (*anschaulich vergegenwaertigen*) in ourselves the inner connectedness (*psychischen Zusammenhänge*) of their thinking, feelings, and motivations, "we try to empathically place ourselves in their shoes, to imagine the world as they see it"^{26(p113)} (our translation). However, we do so in steps of comparison with our point of view: "people adopt others' perspectives by initially anchoring in their own perspective and only subsequently, serially, and effortfully accounting for differences between themselves and others until a plausible estimate is reached."^{27(p328)} By acknowledging the biases of naïve realism, we "bracket or set aside all ... prejudgments and purely describe what is occurring in the patient's mental life."^{28(p373)} Jaspers' phenomenology is thus "presuppositionless" in ... that it does not impose features to mental processes ... [but] ascribes only ... those features ... intuitively re-presented based on ... the patient's speech and behavior."^{28(p374)} This requires its own methodology. Drawing on contemporaries Brentano, Dilthey, Husserl, Simmel, Weber, and others, Jaspers emphasizes, "we *understand* psychic-connections, how the mental arises from the mental, how actions arise from motives, how moods and affects arise from situations. ... To the extent we ascribe internal states as underlying motives to observed behavior we are empathically making connections. ... Methodical-empathic understanding is psychology itself"^{28(p330)} (our translation). By focusing on their internal experiences, we understand others' minds as "inner in contrast to our experience of external nature."^{19(p169)}

Shifting to Causal (Neurobiological) Explanation. When attempting to empathically understand primary delusion,

the clinician is unable to access the experiential source of the delusion. Unlike delusion-like ideas, the primary delusion remains alien. The new or primary in the delusion cannot be derived from some prior experience, motivation, or inner psychological connectedness in the patient. Failing to find any context that makes the patient's behavior or statements understandable, the clinician's empathic understanding falters and she/he experiences the limits of this understanding.^{4(p46),5(p82)} The clinician reverts to *explaining* this failure in terms of a nonunderstandable *process* schizophrenia. This can only be determined interactively: "Why a patient starts to sing in the middle of the night ... why a key on the table excites him so much. All this will seem the most natural thing in the world to the patient but he cannot make us understand."^{5(p486),6(p581)} By recognizing the limits of subjective, psychological understanding, we transcend it. In this sense, we introduce the subject or clinician back into physical medicine.^{5,6,29} For Jaspers, every being who has the capacity for understanding, also understands her- or himself reflectively.^{5,6} Kurt Schneider later observes that it is not always the delusional content in schizophrenia process that is nonunderstandable but rather the fact that the delusion is present at all.³⁰

Jaspers and the Birth of Person-Centered Medicine

When writing the *AP*, Jaspers finds himself in the middle of a debate carried over from the 19th century. In this debate, which concerns the opposed methodology of the natural- vs. the human-historical sciences (the so-called explanation-understanding controversy, see figure 1), Jaspers emphasizes psychological or historical understanding. In psychiatry, the psychological understanding of the patient occurs in the interactive context of the diagnosis and treatment of mental disorders. This understanding is narrative-based; holistic; sensitive to ethnic, cultural, and individual differences; and ultimately oriented to patient strengths. In this sense, Jaspers' work anticipates a person-centered medicine.^{31–33} (See also Stanghellini et al. in the present issue.) Jaspers (1910) writes: "We experience in the other a unity which we cannot define but only experience"^{16(p115)} (our translation). Therefore, Jaspers underscores the importance of the contextuality in the developing expertise of diagnostic practice.^{31,32} However, he also affirms the value of explanatory neurobiological approaches. Delusions are characterized as being nonunderstandable in terms of their historical-cultural context and the person's biography (or motivations) because the underlying neurobiological process has interrupted the person's development.

As can be seen in figure 1, natural scientific explanation (which is reductive) and understanding (which is holistic and contextual) work in opposite directions, yet the clinician must be able to move from one to the other in diagnosing delusions. This occurs, according to Jaspers, precisely when psychological "understanding"

fails. Jaspers^{4–6,16} proposes that diagnosis must freely move between being explanatory and using contextual approaches and therefore, as stated above, anticipates a "person-centered medicine." Although explanation and understanding are mutually exclusive, they presuppose one another when working together in diagnostic clinical practice.²⁹

The Construct of Prodromal Psychosis

According to Jaspers' definition, primary delusions cannot be derived from prior psychological content or motivation. They are ultimate, an original phenomenon (Urphänomenon) in the sense that they arise from a delusional experience.^{5(pp78,89),6(pp93,106)} Something new (unprecedented, nonderivable) must be present.^{5(p165),6(p196)} In a passage added in the 4th edition in 1946, Jaspers writes: "Only where there is thinking and judging can a delusion develop"^{5(p80),6(p95)} (translation modified). Nevertheless, "the delusion is something basic and primary which comes before thought ... the primary event has to be related to some radical change in personality since, otherwise, the insurmountable character of the delusion and its essentially distinctive incorrigibility would be quite incomprehensible ... delusion proper shows itself as a whole primarily in the fact that it creates a *new world* for the deluded person."^{5(p165),6(p196)}

Over the past 2 decades, a major innovation in psychiatry has allowed extensive research into the dynamic process of creation of such a new world. The clinical high-risk construct has been used to investigate psychopathological changes predating onset of illness in subjects presenting with potentially prodromal symptoms (for review, see Fusar-Poli et al.³⁴). Psychosis onset is usually preceded by a variable prodromal phase, characterized by subtle positive and negative symptoms,³⁴ cognitive deficits,³⁵ and psychosocial/functional impairment.³⁴ These changes are correlated with significant alterations in brain function,³⁶ structure,³⁷ and neurochemistry.^{38,39} Despite increasing research, the core phenomenological features associated with a putative prodromal psychotic phase have been relatively underinvestigated.

Delusional Mood at the Dawn of Psychosis

For Jaspers, delusional mood is the first subjective phenomenological experience of something radically new, or alien, entering in prodromal psychosis (indicating an underlying neurobiological process). In prodromal delusional mood, which can last for "days, months or even years ... Something is in the air but one is unable to say what."^{7,40,41} Jaspers writes: "Patients feel uncanny ... that there is something suspicious afoot. Everything gets a *new meaning*. The environment is somehow different ... this general delusional atmosphere with all its vagueness of content must be unbearable. Patients obviously suffer

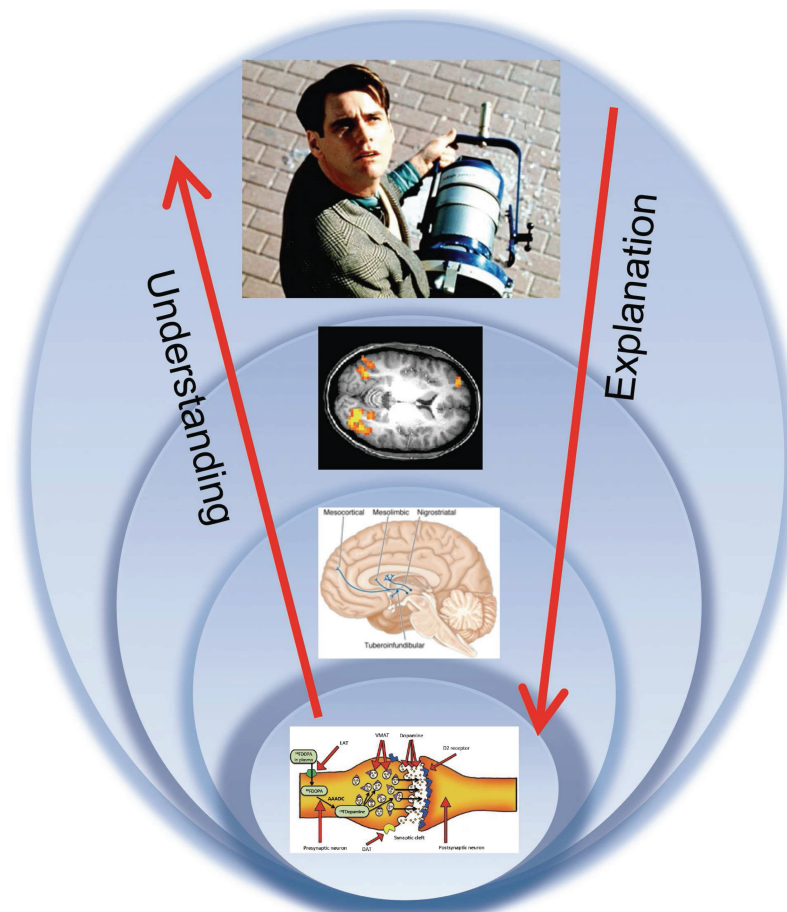


Fig. 1. Understanding and explanation of delusion formation during psychosis onset. The respective methods of the natural and historical human sciences are indicated in the figure by the opposite directions of the arrows. Explanation (arrow pointing to the smallest circle) and understanding (arrow pointing to the largest circle) indicate the opposed methodology of the natural and human-historical sciences, ie, explanation and understanding, respectively. Natural sciences proceed in terms of the “classic reductionist hierarchy” from history and sociology to psychology to biology, chemistry, and physics. They generally proceed from larger, rather nebulous wholes to seek out explanatory relationships among ever-smaller, abstract parts of these wholes. Conversely, understanding is contextual by situating parts in ever-greater wholes, even if these totalities are ultimately unavailable to the individual perspective but transcend or “encompass” it.^{4-6,16,29,31} Each discipline requires an “abstraction,” or “idealization” (ie, “naming,” Husserl)²⁰ of the “objects” or entities of its discipline (which in turn excludes the objects of neighboring disciplines). Gray areas between disciplines indicate interdisciplinary relationships that are often more fuzzy, involving destabilizing relationships within interdisciplinary vocabulary and concepts. Each level is a provisional but also necessary abstraction that presupposes the greater whole from which it was abstracted. As Husserl,²⁰ the founder of phenomenological philosophy, puts it, we can only access a lower-level domain of research “objects” by provisionally abstracting them from their context, what he calls the “life world.” Thus, the movement from the levels of molecular neurochemistry (altered dopaminergic neurotransmission)^{51,52} to systems neuroscience (aberrant salience)^{50,57} to the psychopathology (Truman symptoms), as depicted in the diagram, requires, at each step, a contextual understanding to resituate the neurobiological explanation in its context. As we move up the classical reductionist hierarchy from physics and molecular neurobiology to systems neuroscience, we are nevertheless working from abstractions. Each level is a provisional step and presupposes the greater whole from which it was abstracted. As Jaspers^{4-6,16} emphasizes, the methods of the natural and the human-historical sciences are opposed but also interdependent.

terribly under it and to reach some definite idea at long last is like being relieved from some enormous burden.”⁵ (p82),6(p98)

The delusional mood is experienced by the patient as unprecedented, something entirely new.^{4-6,40,41} Unable to find continuity with his/her previous life history (ie, autobiographical episodic memory), the patient experiences a radical change in personality. “The onset of psychosis happens, by definition, only once.”⁴²(p104) These observations are key to our effort to develop

neurobiological hypotheses from the phenomenology. Jaspers writes: “At the beginning of process disorders, no clear definite meaning accompanies the perceptions. Objects, persons, and events are simply eerie, horrifying, peculiar or ... remarkable, mystifying, transcendental ... something must be going on; the world is changing, a new era is starting. ... The dog scratches oddly at the door. ‘I noticed particularly’ is the constant remark these patients make, though they cannot say why they take such particular note of things nor what it is they suspect. ...

The patients arrive at defining the meaning more clearly when there are delusions of reference. Here the objects and events perceived are experienced as having some obvious relation to the patient himself.” Gruhle comments, “the very sudden emergence of these references to self, often surprising to the patient himself, is the primary non-derivable pathological fact in the matter”^{43(p172)} (our translation). Therefore, the primary delusion arises without any understandable reason or cause (Anlass).^{43(p170)} “There is an immediate, intrusive knowledge of the meaning [which] ... is itself the delusional experience.”^{5(pp83),6(p99)}

This taking note, finding significance in things, and then finally the intrusive meaning, which spreads monotonously to more and more of the patient’s experience is consistent with the later work of the phenomenologically oriented psychiatrists, Conrad⁴⁰ and Binswanger.^{44,45} Conrad calls the stage of the onset of delusions following the delusional mood “apophany” (revelation).^{40,41} He “coined the term *apophany* on the basis of Jaspers’ observation that “the immediate, obtrusive knowing of significant meanings” occurs as a revelation. This is essential to primary delusion”^{40(p46)} (our translation). However, Conrad extends Jaspers definition of delusion as the “abnormal consciousness of meaning” to the patient’s perceptual experience (for review, see Mishara^{41,46}). When perceptual processing is diminished or disrupted (a possibility Jaspers did not consider), we impose meaningful order on the ambiguous sensations. Interestingly, Conrad’s observations anticipate later results that find an association between delusion proneness and jumping to conclusions as well as need for closure (see [supplementary text box S1](#)). In apophany, “the experiential structure is transformed such that each aspect of the patient’s field is related back to the patient. ... Similar to a neurologic symptom, we see the same monotonous structure in each clinical case. ... Apophany describes this process of repetitively, *monotonously* experiencing abnormal meanings in the entire surrounding perceptual field, eg, ‘being observed, spoken about ... followed by strangers’”^{47(p405)} (our translation).

Truman Symptoms and the Creation of a New World

More recently, the delusional mood experiences by prodromal subjects have been reinterpreted as Truman symptoms. The 1998 satirical comedy-drama film (Peter Weir, director; Andrew Niccol, screenwriter) chronicles the life of a man—Truman Burbank (Jim Carrey)—who lived his entire life, since before birth, in front of cameras for *The Truman Show*, although he is unaware of this fact. Truman’s life is filmed through thousands of hidden cameras, 24h a day, 7 days a week, and broadcast live around the world to capture real human emotion and behavior. Truman becomes suspicious of his perceived reality and embarks on a quest to discover the truth about his life and to ultimately discover a “new world.” In 2008, Fusar-Poli

and Howes first reported cases of subjects at risk for psychosis who experienced Truman symptoms.⁴⁸ These individuals first noticed subtle perceptual changes: auditory perceptions became increasingly muffled and associated with a clicking noise, while vision became tunnel-like, resulting in difficulties taking in the whole picture. At the same time, they felt detached from the environment, with a profound alteration of subjective experience and self-awareness accompanied by feelings of depersonalization/derealization. They felt their familiar surroundings were somehow different in an unreal world as if they were living in the *Truman Show* film set. Some patients developed the delusional conviction that they were in a *Truman Show*-like program, followed by a production team, and that everything happening around them was part of this program, in order to test how they would respond. Similar more vivid and intense Truman experiences have been recently (2012) described in established schizophrenic patients.⁴⁹ Overall, these studies point to the fact that alterations in perception of the environment and self are core expressions of an impending vulnerability to psychosis.

This artificiality of the environment and others’ behavior, coupled with the self being center of these artificial arrangements, have also been noted by Jaspers and later phenomenologists. In beginning schizophrenia, “patients often report that everything appears to *revolve* about me.”^{40,41} Conrad reports a patient for whom “every component of his experiential field appears to stand in special relation to him, eg, the instructions given to others about how to behave in front of him, the preparations, the being staged. His ‘world’ transforms itself into a singular field specifically meant to ‘test’ him. ... Once the apophany takes over, no aspect of the field remains untouched. Then, *everything becomes conspicuously salient (auffällig)*. The patient often interprets the course of events as if a film were being made or a theater-piece performed”^{40(p53)} (our translation). Therefore, Conrad calls the delusional mood prior to delusion onset *Trema* (stage fright).^{40,41} Binswanger⁴⁴ describes the delusion as taking place on a delusional theater stage (Wahn Bühne) where the inner contents of the patient’s own mind are played out in front of her/him.⁵⁰

Aberrant Salience: Linking Phenomenology and Neurobiology of Delusion Formation

What is causing the delusional mood and Truman symptoms during psychosis onset? One of the most enduring neurochemical theories of schizophrenia centers upon dysregulation of dopaminergic neurotransmission.⁵¹ In particular, subcortical and striatal hyperdopaminergia has been postulated to be fundamental to psychosis onset.⁵² A recent meta-analysis has confirmed that striatal dopamine synthesis capacity is one of the most robust neurobiological findings in psychosis, with an average elevation of 14% in patients compared to controls.³⁹ Of interest, these alterations in dopaminergic neurotransmission also

have been observed during prodromal psychotic phases in subjects at enhanced clinical risk,³⁸ which affect their neurocognitive cortical functioning.⁵² Therefore, increased subcortical dopamine activity is already present before the full expression of schizophrenia, consistent with dopamine's putative role in the pathophysiology of psychosis.³⁸ Furthermore, longitudinal elevation in striatal dopamine synthesis is specifically associated with emergence of illness.⁵³ But how do dopaminergic alterations relate to delusional mood, Truman-like experiences, and delusion formation in prodromal psychosis? How do the dopaminergic alterations affect the creation of a “new (psychotic) world”? There remains an explanatory gap between what we know about the neurobiology of early psychosis and what we understand about its subjective psychopathological experience.

In recent years, Kapur⁵⁴ advanced the idea that dopamine mediates the “salience” of environmental events and their mental representations, filling such a gap. The broad challenge for any organism negotiating a complex world is how to efficiently and effectively choose and respond to relevant stimuli. This selection involves attentional processes of filtering, sensory and behavioral orientation, and searching.⁵⁵ Stimuli are prioritized according to their “saliency”/relevance: Their features are compared with their context. Stimulus-driven processing interacts with internal factors, eg, goals, beliefs, and history, to determine the most salient stimulus. A key influence on goal-directed behavior is the pursuit of reward and avoidance of punishment. Basic neuroscientific research has highlighted the key role played by dopamine in these behaviors. Drugs of addiction work by increasing or prolonging the action of dopamine on its main projection targets,⁵⁶ and animals with electrodes implanted in dopamine-related areas repeatedly stimulate these over food and sex, sometimes until death. Neuroimaging techniques have further prompted neurobiological investigations of dopaminergic alterations underlying salience processing. Neuroimaging studies using tasks that involve salience processing indicate that the salience network involves midbrain dopamine neurons, their projections to the striatum, and the frontotemporal and frontoparietal cortical regions. There is thus converging evidence indicating that dopaminergic neurotransmission is essential for signaling reward and salience. Abnormal striatal dopamine firing in early psychosis could thus lead to the aberrant assignment of salience to innocuous stimuli^{51,57} and therefore underlie the emergent delusional mood and Truman-like symptoms. Primary delusions may arise from the prodromal state as the individual's own explanation of the experience of aberrant salience (see also [supplementary text box S2](#)).⁵¹ This is in line with what Jaspers called delusional experiences (*Wahnerlebnisse*), which eventually lead to errors in judgment.^{5(p87),6(p104)} Further developments in the phenomenology of delusion formation following Jaspers are detailed in [supplementary text box S3](#).

Reconsolidation of Delusions as Impaired Learning About Salient Events

Because the continuity or development of personality has been disrupted by an underlying neurobiological process,^{4-6,16} the delusional theme, wrested from the originally experienced perceptual saliences, becomes detached from how memories are normally encoded in an ongoing narrative event memory of self.^{44,45,50,58} This “inner life history” is both the continuity of the flowing of present experience and also how this experience becomes memory in terms of an autobiographical self. Coherent self-experience plays a key role in integrating episodic memories into more enduring self-knowledge (see [supplementary text box S4](#)).⁵⁹

Mishara and Corlett interpreted the monotonous repetitiveness of the delusion to be the reconsolidation phase of impaired learning about salient events⁶⁰ (see also Corlett et al.⁶¹). Unexpected events, prediction errors, are registered inappropriately in an eventual shifting of control from goal-directed learning to the striatal habit system. These processes may lead to reconsolidation of delusions from an initial early acute onset to a chronic phase as the sense of self becomes disrupted.

For Blankenburg,⁵⁰ based on Häfner's review,⁶² it is precisely the repetitiveness of the delusional theme, with its greater and greater insistence on “absoluteness” (see [table 1](#))—no longer processed as part of the patient's previous autobiographical self—that points to Jaspers' distinction between understandable psychological-historical development of personality and an unknown underlying neurobiological process (see [supplementary text box S3](#)). Jaspers' “nonunderstandability theorem” is not only the clinician's failure to understand the patient's goals, intentions, and motivations, which give rise to incomprehensible actions/utterances, but with Schneider,³⁰ something much more embracing. Blankenburg^{1,50} calls this a reduction in social relatedness, a context blindness, or the loss of common sense⁶³ that otherwise underlies and makes comprehensible everyday interactions (see also Stanghellini et al. in the present issue). This is experienced by the clinician as a lapse in coming to mutual understanding in a reciprocal exchange of perspectives.^{1,40,44,45} In this scenario, one of the prodromal symptoms of psychosis is a loss of common sense, which may be associated with positive symptoms but may also indicate the early onset of a symptom-impoverished simple schizophrenia.⁶³ In this sense, the clinician's experience parallels the patient's in what we have described as a 3-step process (see [table 1](#)).

Conclusions

Although not well known in English-speaking psychiatry, Jaspers' *AP* played a profound role in the development of views about delusion formation in prodromal and early schizophrenia in Europe (see [supplementary text box S5](#)).

Jaspers' approach to delusion formation and its subsequent development by phenomenological psychiatrists supports more contemporary neurobiological models of delusion formation in terms of aberrant salience and dopamine dysregulation. Finally, it is demonstrated that there is an explanatory gap between the phenomenology and current neurobiological models of delusion formation in early psychosis. The explanatory gap between phenomenology and neurobiology has been debated for many years and several models have been proposed, largely unsuccessfully, to bridge this gap. We do not claim to solve this difficult problem with the current contribution but only offer one further step in how to approach it. The phenomenological approach leads to neurobiological hypotheses, which can be tested experimentally. We believe that returning to Jaspers' *AP* will help us overcome this gap by encouraging models that incorporate both the phenomenology of the patient's experience of delusion formation and its underlying neurobiology. Jaspers and his successors' phenomenological approach suggests that the delusion is formed through loss of context in its experiential origins and memory. This is consistent with more recent neurobiological models.

Supplementary Material

Supplementary material is available at <http://schizophreniabulletin.oxfordjournals.org>.

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