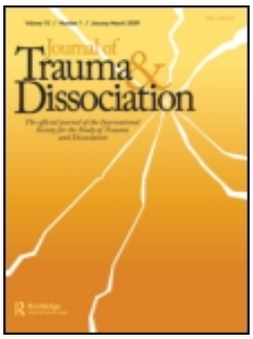

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EDITORIAL

Schizophrenia, Trauma, Dissociation, and Scientific Revolutions

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In my mid-20s, at the beginning of my training as a clinical psychologist, I was placed on a psychiatric day treatment ward in one of the poorer parts of Boston. One day, the experienced therapist with whom I led a men's group was sick, and I was called on to do the group by myself. A ball of nerves, I decided to ask the men about their ancestry (with the helpful presence of a globe in the room) rather than risk silence. I briefly spoke of my Russian and Eastern European great-grandparents to set the tone and then spoke with each man in turn. After a few minutes of this exercise, there was a pause. A fellow from across the room looked at me and said softly, "You think you're better than us, don't you? You think this could never happen to you."

I was stunned. Somehow I stammered a denial, but of course he was right. Perhaps I didn't think I was better than them, but I certainly thought I was *different* from them. Like most of us in Western societies, I had grown up believing that psychiatric disorders were illnesses—*diseases* like any other—and there had been nothing in my training until then to convince me otherwise.

But learning about trauma, dissociation, and attachment in the ensuing decades has changed my mind. And I am not the only one.

PARADIGMS IN CONFLICT

Over the past several decades, the study of schizophrenia and the study of the dissociative disorders have been dominated by opposing paradigms. For schizophrenia, the assumption of a genetic basis and biological causation has

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reigned supreme. Adverse childhood experiences are viewed as irrelevant at best and adult stressful or traumatic experiences as only "releasing" underlying disease mechanisms. Symptoms are considered meaningless—unrelated to a person's life circumstances—and psychotherapeutic approaches, when used at all, are limited to supporting medical interventions. In diagnosing schizophrenia for clinical or research purposes, posttraumatic and dissociative disorders are rarely considered or ruled out; indeed, in adherents to this paradigm, posttraumatic disorders are frequently disdained, discredited, or simply ignored.

In contrast, the overriding paradigm for the study of dissociative disorders has focused almost exclusively on life events—traumatic or otherwise—that *are* assumed to be meaningfully related to the symptoms a person experiences. A wide range of psychotherapeutic approaches to treatment are supported and advocated, whereas most medical interventions are viewed as anathema. At the same time, many trauma-oriented clinicians and researchers think of schizophrenia only as something dissociative disorders are *not*—but are often confused with; schizophrenia's validity as a biologically based entity is rarely questioned.

Consider how these two paradigms deal with auditory verbal hallucinations. To persons adhering to the dominant biological paradigm (or "medical model"), voices are psychotic symptoms to be treated with medications or coped with using distraction techniques. As Colin Ross (2008) put it, from this perspective the notion of talking with someone's voices would be as absurd as "asking a patient's knee a question" (p. 284). In contrast, in a trauma/dissociation paradigm, voices are split-off parts of the personality that are ignored at one's own peril—acknowledging and engaging these disowned parts, though often challenging, is typically advocated. The schizophrenia field views voices as biologically generated indications of a brain disorder, whereas the dissociation field views them as psychological indications of unresolved trauma or loss. Two more disparate perspectives cannot be imagined. Currently, these fields eye each other with considerable suspicion and, to a large extent, do not speak the same language or experience the world in the same way.

EUGEN BLEULER: THE MARRIAGE OF DISSOCIATION AND SCHIZOPHRENIA

But it was not always this way. When Eugen Bleuler published his *Dementia Praecox oder Gruppe der Schizophrenien* (*Dementia Praecox or the Group of Schizophrenias*) 100 years ago, the construct of schizophrenia was infused with dissociative concepts (Moskowitz, 2008; Moskowitz & Heim, 2011). While insisting on an organic basis for the disorder, Bleuler recognized the symptoms his patients described as meaningfully related to their life

experiences and used hypnotherapy and psychotherapy in his clinical work. He justified changing the name of the disorder largely on the basis that the "splitting" of the "different psychic functions" was central to its pathology (Bleuler, 1911/1950, p. 8). Bleuler's 1911 "definition" of *schizophrenia* reads almost as a calling card for dissociative disorders:

> If the disease is marked, the personality loses its unity; at different times different psychic complexes seem to represent the personality . . . one set of complexes dominates the personality for a time, while other groups of ideas or drives are "split off" and seem either partly or completely impotent. (p. 9)

The profoundly dissociative nature of Bleuler's concept of schizophrenia has been ignored for many decades but should be apparent to any unbiased reader, as has been recognized by Colin Ross (2004) and myself (Moskowitz, 2008; Moskowitz & Heim, in press).

However, Bleuler's ideas about schizophrenia have little currency in today's nosological world; all but the name has been jettisoned, and even that has been retained with considerable squeamishness—requiring constant vigilance against its interpretation as "split personality." Instead, the architects of our current diagnostic system harked back to Bleuler's predecessor, Emil Kraepelin, for inspiration.

EMIL KRAEPELIN, TAXONOMIES, AND *GENERAL PARESIS*

Despite Kraepelin's experimental psychology pedigree (he studied with Wilhelm Wundt early in his career), his ideas on *Dementia Praecox* were far less informed by psychology than those of Bleuler (who used Jung's word association experiments to aid his understanding), and he saw concepts of dissociation as irrelevant to diagnostic conceptualization. Rather, Kraepelin's approach to parsing mental disorders was strongly influenced by biological classifications, such as Linnæus's taxonomy of plants and the system developed by his own esteemed older brother, the biologist Karl Kraepelin (Weber & Engstrom, 1997). In addition, the model on which Kraepelin based his concept of *Dementia Praecox* was *General Paresis of the Insane*—sometimes called *Dementia Paralytica*. General Paresis was a terminal condition that combined psychotic symptoms with paralysis and ultimately death and was widespread in Europe during the early part of the 19th century. The triumphant linking of its symptoms with a brain disorder caused by late-stage syphilitic infections in the mid-19th century *clearly* provided Kraepelin with a template or paradigm—a "model disease entity"—for mental disorders in general and dementia praecox in particular (Jablensky, 1995, p. 186).

THE *NEO-KRAEPELINIAN* PARADIGM OF MENTAL DISORDERS

The example of *General Paresis*, with the assumption not only that mental disorders were brain disorders but that any classification of psychopathology was best pursued through identifying brain pathology, not only drove Kraepelin's typology but also still underpins that of the current diagnostic systems influenced by his thinking—the *Diagnostic and Statistical Manual of Mental Disorders* (3rd ed. [*DSM–III*]), the International Classification of Diseases–9, and their related progeny (Jablensky, 2007). For the past three or four decades, the classification of mental disorders has been dominated by this approach, which came out of a group of primarily American psychiatrists self-identified as *neo-Kraepelinian* (frequently referred to as a *movement* or even a *revolution*).

As the neo-Kraepelinians set about revising the psychiatric diagnostic system in the 1970s, and reached their goal with the 1980 publication of the *DSM–III*, they were ostensibly creating an atheoretical system with improved reliability over its precursors. But in reality, they were clearly motivated by the belief that these conditions were medical disorders like any other; indeed, in a publication from that time, two prominent researchers spoke of "coveting" for schizophrenia the solid genetic grounding of "pellagra, paresis, tuberculosis, polio, and PKU [phenylketonuria]" (Gottesman & Shields, 1973, p. 15).

A fundamental task for the neo-Kraepelinians was to shore up the distinction between schizophrenia and manic depression, a distinction that had been blurred by Bleuler's broad category. They accomplished this primarily by strongly emphasizing specific psychotic symptoms in the diagnostic criteria for schizophrenia (particular auditory hallucinations and delusions proposed by Kurt Schneider—so-called *first rank* symptoms) and by undermining the validity of the *schizoaffective* disorder category in a number of ways (Moskowitz & Heim, in press-a). The *Kraepelinian dichotomy* of schizophrenia and bipolar disorder has been explicitly seen as providing the foundation for a biologically based nosology; indeed, challenges to the clear differentiation of schizophrenia and bipolar disorder are often viewed as undermining the validity of the entire diagnostic system (Kendell, 1987). In addition, the neo-Kraepelinians have articulated a number of more general assumptions, including (a) that mental disorders are discrete from one another and from "normality" and (b) that advances in understanding mental disorders will come primarily from focusing on neurobiology (Klerman, 1978). This level of domination over research and practice (for example, *DSM–IV* diagnoses are required for insurance payments and frequently for journal article acceptance) clearly constitutes what Thomas Kuhn termed a scientific *paradigm*.

PARADIGMS AND SCIENTIFIC REVOLUTIONS

According to Kuhn (1970), in *The Structure of Scientific Revolutions*, the idea that science advances in a linear fashion with knowledge continually accruing so that "reality" or "truth" is more and more closely approximated over time is a myth. Rather, he argued, a field advances under the influence of a dominant *paradigm*, meaning both a particular past scientific achievement held up as a model or *exemplar* (as in the case of *General Paresis* and psychopathology) and the generally accepted beliefs and attitudes of a particular scientific community. A paradigm exerts an organizing influence on a field and guides research, determining to a large extent what types of research questions are considered legitimate and what sorts of answers are considered acceptable.

Kuhn (1970) argued that paradigms change and a *scientific revolution* ensues when three conditions are met: (a) a period of crisis develops in which the paradigm fails to adequately answer questions considered fundamental; (b) serious "anomalies" occur in which phenomena not clearly compatible with the paradigm are observed; and, importantly (c) a suitable alternative paradigm that explains many of the previous findings and at least some of the observed anomalies comes to light. Kuhn saw scientific revolutions as taking time to resolve; he argued that changing such strongly held beliefs involved a process of persuasion and fundamental reorganization not unlike that of *religious conversion*: "Conversions will occur a few at a time until, after the last holdouts have died, the whole profession will again be practicing under a single, but now different paradigm" (Kuhn, 1970, p. 152).

Since the publication of the *DSM–III* in 1980, the ascendance of the *neo-Kraepelinian* paradigm in the psychiatric world has been paramount. It has driven our view of schizophrenia and marginalized acceptance of the dissociative disorders and posttraumatic stress disorder (PTSD). However, this paradigm is now under threat from many quarters—from within its ranks as well as from outside—and there is good reason to view it as a paradigm *in crisis*.

FAILURES OF THE NEO-KRAEPELINIAN PARADIGM

Evidence for fundamental tenets of the neo-Kraepelinian paradigm—that there are clear genetic or biological bases for schizophrenia and other mental disorders and that mental disorders are discrete from one another and from normal experiences—have not been supported.

Comorbidity of diagnoses, incompatible with viewing diagnoses as discrete categories, is rampant in the *DSM–IV* system and typically viewed as a major problem. Psychotic symptoms are now recognized as common to many disorders other than schizophrenia, and their presence in a significant

portion of the community with no diagnosed mental disorder firmly suggests that the line between "normality" and "pathology" is not hard and fast (Van Os, Linscott, Myin-Germeys, Delespaul, & Krabbendam, 2008). In addition, evidence for the validity of *schizoaffective* disorder, a fundamental challenge to the Kraepelinian dichotomy, has accumulated over the years. The demonstrated existence of persons with prominent schizophrenic and affective symptoms undermines the core distinction between schizophrenia and bipolar disorder and provides an argument for viewing even severe psychopathology as a dimension or series of dimensions instead of as categories. Finally, the abject failure of genetic-based research to find any strong link with schizophrenia or bipolar disorder provides a further anomaly for the *neo-Kraepelinian* paradigm to explain or attempt to ignore (if anything, the genetic evidence points to a "shared neurobiology across the two disorders," Thaker, 2008, p. 720).

All of this is taking its toll on the medical model. As the neo-Kraepelinian edifice begins to crumble, adherents resort to stronger and stronger biological language, as though words such as *neuro*psychiatry and *endophenotypes* have the power to restore its once shining façade. The emphasis on endophenotypes is particularly telling, as this concept involves exploring putative underlying biological variables that may have only an *indirect* relationship to the signs and symptoms of mental disorders. For example, a recent large-scale twin and family study focused on apparent genetic impairments in memory and intelligence as conveying liability for schizophrenia (Toulopoulou et al., 2010). The strong emphasis on endophenotypes, arising from a failure to find clear connections between genetic makeup and psychiatric diagnoses or symptoms, suggests that the *neo-Kraepelinian* stalwarts have beaten a strategic retreat; at the same time that psychological approaches to treating and understanding psychiatric symptoms, including delusions and hallucinations, have made great strides, the dominant paradigm has given up the traditional territory of mental disorders—the signs and symptoms that people suffer from and that treatments target.

So, the neo-Kraepelinian, categorical, medically based diagnostic system clearly seems to be in a state of crisis. But, as Kuhn has noted, a discipline such as psychopathology will not loosen its grip on a paradigm unless a suitable alternative is available to take its place. What is the evidence that one is appearing?

THE EMERGING TRAUMA/DISSOCIATION PARADIGM

In recent years, evidence has accumulated that traumatizing events are strongly linked to psychopathology in general and psychotic symptoms in particular. Kenneth Kendler, a prominent psychiatric geneticist, concluded

from a carefully designed large-scale twin study that childhood sexual abuse was "causally related" to the development of psychiatric and substance abuse disorders (Kendler et al., 2000, p. 953). In a subsequent commentary, he noted that the more than threefold increase in major depression attributable to severe sexual abuse was "much greater" than the odds ratios associated with *any* gene putatively linked to schizophrenia or bipolar disorder (Kendler, 2006, p. 1140); he soberly concluded, "The project to ground our messy psychiatric categories in genes . . . may be in fundamental trouble" (Kendler, 2006, p. 1145). Psychotic symptoms in particular appear to be strongly linked to trauma, both adult trauma (particularly when associated with PTSD; e.g., Scott, Chant, Andrews, Martin, & McGrath, 2007) and childhood interpersonal traumas (including in longitudinal studies such as Arseneault et al., 2011). These studies are becoming increasingly well designed, typically controlling for many potentially confounding variables, even apparently genetic ones. Furthermore, psychological trauma has been strongly linked to the development of delusions and hallucinations (Moskowitz, Read, Farrelly, Rudegeair, & Williams, 2009), and dissociation has been found to consistently and powerfully predict auditory hallucinations (but not delusions) in a range of populations (Moskowitz & Corstens, 2007; several recently published studies have supported this relationship). Finally, brain changes long assumed to indicate a core genetic or biological neurodevelopmental disturbance in schizophrenia have been linked with chronic stressful or traumatic childhood experiences (Read, Perry, Moskowitz, & Connolly, 2001; Teicher et al., 2003). And these trauma-based brain changes are entirely consistent with emerging evolutionary-based explanations for psychotic symptoms (Grace, 2010; Moskowitz, 2004).

IS THERE A SCIENTIFIC REVOLUTION IN THE HOUSE?

The failures of the current dominant medically based *neo-Kraepelinian* paradigm, coupled with the successes of an alternative paradigm focusing on adverse life experiences (including attachment disturbances) and dissociation, could herald the approach of a scientific revolution. Evidence that this may be occurring includes the increased willingness of prominent medical journals such as the *American Journal of Psychiatry* and *Archives of General Psychiatry* to publish studies supportive of this view (e.g., Arseneault et al., 2011; Kendler et al., 2000; Scott et al., 2007). As more and more psychiatrists are shifting paradigms, it must be recognized that many medically trained individuals within the trauma and dissociative disorders field have long championed this perspective (of course, there are psychologists and other non-physicians who continue to firmly embrace the "medical model" as well, but these paradigms to a large extent do map onto disciplinary distinctions and tensions).

Should a new paradigm emerge, it will be a *genuine biopsychosocial* one, recognizing that genetics plays a role in psychopathology, likely in providing vulnerability to certain broad forms of mental disorders or to mental disorders in general. It will also recognize that life experiences *from gestation on* play a major role not only in the expression of psychiatric symptoms but also in the *expression of the genes* that underlie vulnerability to mental disorders. This new paradigm must also recognize some form of dimension or dimensions across apparently different types of mental disorders (evaporating the comorbidity "problem") and between pathology and so-called normality. It will require recognition of the extent and severity of childhood trauma, a reality that has long faced considerable resistance from adherents to the medical model. Finally, the presence of dissociative conditions, with the corollary that such individuals are radically different at different times, must be taken into account not only clinically but also in the design of research—something to which the current paradigm has been blind.

The *DSM–5* committees appear to have some awareness of these challenges. Dimensional perspectives are being considered for personality disorders and possibly as an axis alongside other categories. What is striking is that the schizophrenia committee is recommending the elimination of the (currently pathognomic) first rank symptoms (voices conversing or commenting, delusions involving intrusions or withdrawals of thoughts or behavior), belatedly recognizing that they have "no unique diagnostic specificity" for schizophrenia (American Psychiatric Association, 2011). This is obviously welcome news (an early indication of a paradigm shift?), as the association of these clearly dissociative symptoms with schizophrenia has led to substantial misdiagnosis of dissociative identity disorder patients. But it also reminds us of the enigma that practically every attempt to define schizophrenia, from Bleuler to the present day, has invariably called forth dissociative identity disorder. That the paradigmatic biological disorder can be so easily confused with the paradigmatic environmental disorder should already be shaking the rafters of this house (but of course, as the dominant paradigm does not recognize dissociative identity disorder, it does not recognize this enigma!). The explanation for this puzzle should help us to understand the nature of schizophrenia—until then, we can firmly state that whatever schizophrenia *is*, it is *not* psychotic symptoms and *certainly not* auditory hallucinations. Unfortunately, the *DSM–5* schizophrenia committee has not gone this far and continues to emphasize psychotic symptoms, even as the head of that committee, William Carpenter, warns against this approach ("Psychotic experience is to the diagnosis of mental illness as fever is to the diagnosis of infection—important, but non-decisive in differential diagnosis," Fischer & Carpenter, 2009, p. 2081).

If a new paradigm does emerge, we can be sure that Kraepelin's paradigmatic disease entity—*General Paresis of the Insane*—will be replaced. Perhaps it may not be possible to find a new exemplar for mental

disorders in general, but PTSD would seem a worthwhile candidate for at least some of them—those clearly linked to trauma and characterized by dissociation (as, for example, has been proposed by Van der Hart, Nijenhuis, & Steele, 2006, in their *structural dissociation* model). And the possibility that schizophrenia, or at least some form of psychotic disorder, could fit this model should not be rejected outright. Even Bleuler, the progenitor of schizophrenia, despite his commitment to an organic etiology, seemed to recognize this. A growing appreciation of this possibility could, quite literally, trigger a scientific revolution in our view of mental disorders altogether.

> The stronger the affects, the less pronounced the dissociative tendencies need to be in order to produce the emotional desolation. Thus, in many cases of severe disease, we find that only quite ordinary everyday conflicts of life have caused the marked mental impairment; but in milder cases, the acute episodes may have been released by powerful affects. And not infrequently, after a careful analysis, we had to pose the question whether we are not merely dealing with the effect of a particularly powerful psychological trauma on a very sensitive person, rather than with a disease in the narrow sense of the word. (Bleuler, 1911/1950, p. 300; Sünje Matthiesen, translation)

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