

EDITORIAL

Degeneration Theory and the Stigma of Schizophrenia

Like many of his contemporaries, Emil Kraepelin (1856-1926) thought of schizophrenia as an essentially deteriorating illness that was unresponsive to environmental intervention. The dominance of this pessimistic view at the turn of the century and its persistence in attenuated form today—despite considerable evidence to the contrary—merit a fuller exploration. We offer here some brief reflections.

There are at least three possible sources of this pessimism: a truly different course and outcome for schizophrenia at the turn of the century, sampling bias, and the prevailing psychiatric theory. Whether in fact schizophrenics in 1900 experienced a prognosis poorer than today is a question of considerable historical and scientific interest. Unfortunately, no studies adequate to address this question have been published to date. However, it is likely that the focus of Kraepelin's clinical work was on the more severe or long-term institutional cases, especially those already on a chronic debilitating course. Manfred Bleuler, for instance, attributes his father's pessimism to summer visits to the provincial clinic at Rheinau, which Eugen had directed earlier in his career. Seeing his old chronic patients come out to greet him made Eugen happy, but he was also struck by their deterioration. He forgot about those who had long ago recovered and left the institution (Bleuler 1978). The dismal assessment of prognosis based upon such biased samples is a fallacy commonly overlooked by many practicing clinicians (Susser 1984; Cohen and Cohen 1984).

A contributing factor to this baleful view of schizophrenia was the conviction held by many of Kraepelin's contemporaries that mental illness had organic origins in a tainted constitution (Decker 1977). The most extreme expression of this position, the degeneration theory, had emerged earlier in France in the writings of Benedict Morel (1809-1873) and, later, of Jacques Magnan (1935-1916). This theory held that mental disease represented the presence of a familial degenerative strain that became increasingly more severe over successive generations, eventually causing extinction of the line (Genil-Perrin 1913; Burgener 1964; Ackerknecht 1968). The theory was popularized in Germany by Griesinger (1817-1868), then developed by Schuele (1840-1916) and Krafft-Ebing (1840-1902), both of whom incorporated the idea of degeneration into their respective nosologies (Genil-Perrin 1913). Kraepelin, while critical of this vision of irrevocable familial mental decline over succeeding generations (Kraepelin 1889, 1909-1915, 1962), nonetheless considered degeneration a component of the hereditary predisposition to psychiatric disorders found in certain families. In his 1904 lectures on clinical psychiatry, Kraepelin argued that "seeds of decay" could be passed from one generation to the next unless neutralized by an admixture of healthier blood (Kraepelin 1904). For Kraepelin, what might be called a heredity-degeneration theory served as a partial explanation for hysteria, manic-depressive psychosis, and dementia praecox (Kraepelin 1909-1915, 1971; Genil-Perrin 1913; Decker 1977). He referred, for example, to physical and psychic signs of degeneration in the childhood cases of dementia praecox (Kraepelin 1909-1915, 1971). In the same work, he stated bluntly that "degeneration certainly plays a part in the development of dementia praecox" (Kraepelin 1971, p 239). In general, then, Kraepelin's views underscored the predetermined nature and relentless outcome of a range of psychiatric disorders including, perhaps most especially, dementia praecox.¹

¹Emil Kraepelin's use of degeneration theory as a partial explanation of the etiology of some psychiatric disorders marks him as a man of his time, influenced by contemporary scientific and social thought. It in no way detracts from the greatness of his nosological work or his importance as a clinical researcher and teacher.

The theory of degeneration tended to dissociate radically the mentally ill from those not so labeled. Morel defined degeneration as a morbid deviation from normal humanity (Genil-Perrin 1913). For Kraepelin and his colleagues, the hereditary etiology of mental illness also rendered the patient substantially different from the normal citizen. To a historian of the period the picture of humanity drawn by these nineteenth-century physicians "was almost [of] two different species, physically alike but mentally different" (Decker 1977). Mental disease and mental health were therefore mutually exclusive, rather than points on a continuum. A relationship by blood with a psychotic was shameful and stigmatizing, giving rise to doubts about one's place in the brutal dichotomy between well-being and disease.

The degeneration theory was not unique to late nineteenth and early twentieth century psychiatry. It also informed etiological theories in other branches of medicine (particularly those concerned with chronic, deteriorating disorders like alcoholism, syphilis, mental retardation, and tuberculosis [Kevles 1984; Brandt 1985; Carlson in press]) and in the social sciences. The theory influenced Cesare Lombroso (of stigmata fame), and through him, the school of American criminology that conceived of the criminal as a reversion to an earlier biological type (Carlson in press). Degeneration theory is discernible in Social Darwinism and the eugenics movement in Britain, where Karl Pearson and Sidney Webb, among others, tried to halt the "degeneration" of the British "race" by advocating selective breeding and selective immigration (Kevles 1984). Finally, in the United States, the studies of the Kallikaks and the Jukeses were heavily influenced by degeneration theory, and similar considerations motivated the legislatures of 24 states to pass eugenic sterilization legislation during the first third of this century (Kevles 1984).

That the degeneration theory, despite its questionable scientific status, can still produce fear and shame is suggested by stories like the one told by Torrey (1983) of the mother who would have preferred that her daughter have fatal leukemia than schizophrenia. In much the same vein is the reported comment of another parent of a schizophrenic patient that "I've known people who were cured of cancer, of all these illnesses, but I know of no one who was cured of schizophrenia" (Schultz et al. 1982).

Certainly an assumption about deterioration, inextricably part of the degeneration theory, persists today in the neo-Kraepelinian movement. This movement's definition of schizophrenia as a chronic illness leading almost inevitably to a deteriorated end-state is, in essence, a resuscitation of dementia praecox. The neo-Kraepelinian's insistence, both on a 6-month duration before a diagnosis of schizophrenia can be applied, and on severe impairment in at least two areas of living (including inability to hold a job) as part of the diagnosis, exemplifies the movement's incorporation of inexorable decline into its definition of schizophrenia.

One explanation of this negative view is that the neo-Kraepelinians, like their predecessors at the turn of the century, are targeting a skewed sample. In stark contrast to this rather dismal prognostic outlook, some European investigators, using less biased samples, have been able to furnish a far more benign view. Their long-term longitudinal cohort studies of first-admission schizophrenics show that a majority of patients either recover or follow a mild chronic course (Bleuler 1978; Ciompi 1980; Huber et al 1980). Thus, only one-third to one-quarter of schizophrenic patients do not progress toward more favorable outcomes (Bleuler 1978).

Supposing no marked geographic differences between schizophrenics, the European evidence suggests the need for an alternative model for schizophrenic disorder. One that has been advanced is the vulnerability model (Zubin and Steinhauer 1981; Zubin et al. 1983), which proposed that whereas schizophrenia is not continuous, vulnerability to it is so, in certain individuals. In these persons, episodes may be triggered by exogenous or endogenous life event stressors and may continue until the stress and its sequelae dissipate. Thereafter, the patient returns to his premorbid level of adjustment and remains there in the absence of further triggering events. Since in the vulnerability model chronicity is not an essential aspect of the natural history of schizophrenia, deterioration is viewed as a reflection of iatrogenic, ecogenic, or nosocomial artifacts (Zubin et al. in press) rather than as the product of the disease process itself. According to this model, only the removal of the conceptual curse that attends schizophrenia as a so-called chronic ailment will make

it possible to develop appropriate therapeutic interventions to prevent the vulnerable from suffering either the initial or recurring attacks.

The history of attitudes toward and evidence for the prognosis of epilepsy offers a striking parallel to this account of schizophrenia. In classical times both the natural origins of epilepsy and the existence of marked variations in prognosis depending on age of onset were well recognized. However, the nineteenth-century medical literature on epilepsy was generally more pessimistic about recovery, and the concluding decades witnessed the familiar embrace of epilepsy by the degeneration theorists: "The rule of progressive degeneracy . . . proves true very often, so that with a relatively mild neuropathic morbid condition in the first generation, possibly epilepsy will arise in the second and severe mental disease in the third" (Eulenberg 1878). The epileptic, assumed to suffer from a hereditary affliction, was predisposed to criminality, suicide, vagabondage, and religiosity (Lombroso 1891).

Today, "once an epileptic, always an epileptic" (Kurtzke 1972) sums up, to some degree, the public's view and governs much clinical practice, as reflected in recommendations for prompt placement and prolonged maintenance—indeed "for the major portion of a lifetime" (Baker and Baker 1982)—on antiepileptic drugs. As with schizophrenia, the hardness of this negative assessment of prognosis in epilepsy probably reflects the inertial continuation of nineteenth-century perspectives on seizure disorders. No doubt it springs, too, from clinicians' inevitable tendency to judge a disorder's natural history on that subset of incident cases that become chronic clinic attenders, in this example at tertiary care epilepsy centers. However, research in the 1960s (Juul-Jensen 1964) and more recent, growing evidence from controlled trials and observational studies have disclosed a more positive prognostic picture. Hauser et al. (1982), for example, have shown that only 26% of persons experiencing a first seizure (without a history of prior neurological insult) proceed to have a recurrence within the next 3 years. According to Thurston et al. (1982) 28% of children withdrawn from anticonvulsants after being seizure free on anticonvulsants for at least 4 years and followed for a median period of 19 years experience a recurrence of seizures.

This parallel between schizophrenia and epilepsy helps to corroborate our basic contention about the endurance of older beliefs regarding chronicity in the midst of opposing facts. Having exhibited somewhat similar chronologies over the past century, we wonder whether the trajectories of public and medical attitudes towards schizophrenia and epilepsy will now continue in tandem towards a more benign view of prognosis, or remain yoked together in their pessimism.

The persistence of aspects of the degeneration theory, despite much contrary evidence, is an example of how one can be imprisoned by an archaic way of thinking. Sometimes it is easier to change genes than to change culture! To excise from ourselves an old, dominant zeitgeist, it may be necessary to give schizophrenia a name that is not scarred by the old stigma; possibly we should call it Kraepelin-Bleuler syndrome or even Vaslav Nijinsky's disorder.

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