Eugen Bleuler’s thoughts and views about heredity in schizophrenia

In 1917, Eugen Bleuler addressed himself to the heredity-environment issue in schizophrenia when Ernst Rüdin published his own findings regarding heredity in schizophrenic illness. Bleuler had much at stake in the Rüdin monograph, which he studied thoroughly, and he decided to publish his own evaluation of Rüdin’s study. The studies of both men were, of course, written in German and are not well known in this country. I plan to have a translation of the Bleuler article prepared, but it will take additional time. In reading the article, one gets a fair sense of Bleuler’s thinking about genetics in schizophrenia and his ideas and beliefs regarding this illness, or syndrome. For current purposes, I have selected out a number of his sayings, thoughts, opinions, and speculations which provide a view of the man and his ideas.

1. Bleuler thought that drawing preliminary conclusions from studies of psychiatric diseases, especially one of a particular type of inheritance, was childish. He encouraged his son, Manfred Bleuler, and his students, to do studies on heredity in schizophrenia.

2. He held that Rüdin nevertheless had developed valuable material and important new points of view in the clinical field.

3. He was skeptical about genetics in psychiatry, which he described as “only numbers which chance to fall into the hands of psychiatrists.”

4. He nevertheless made it a point to be knowledgeable about psychiatric genetics, and his criticisms of the findings in the Rüdin study suggested that he had a reasonable grasp of the young discipline, including the new contributions of Weinberg, who was a renowned, innovative statistician and geneticist.

5. Bleuler agreed that the existence of the sick member in the family might show that the parents possess a hereditary predisposition to the disorder.

6. Bleuler was very much concerned about problems of sampling in Rüdin’s study (an issue that I strongly emphasized again, many years later, in evaluating twin studies of schizophrenia).

7. He concluded that manifested schizophrenia could not be a monohybrid Mendelian trait or a dominant disease and that a simple recessive gene was not implicated. But he allowed that schizophrenia could involve a dihybrid, complex gene.

8. He pointed out that what is inherited does not arise out of Rüdin’s analysis, and that the analyses, if correct, indicated that there is no homogeneous gene for schizophrenia.

9. Bleuler thought that different factors must come together in order for the sickness to evidence itself.

10. He also stated that a polymorphous inheritance was not out of the question at that present time. Thus, he may have been the first to accept a polygenic mode of transmission in schizophrenia.

11. He said that the most important questions were so closely connected that no single one seemed to be completely answered without answering the others; “they only approached the problem from another side.”

12. Doubtful psychotics, upon examination in the institution, turned out to be the largest group of schizophrenic patients. “Many who later are clear cases of schizophrenia are considered otherwise, some not even psychotic, but only neurotic or psychopathic for years.”
   - “We have here in Burghölzli perhaps the broadest concept of the sickness. The acknowledgment of a broader concept of schizophrenia brings up new difficulties.”
   - “I am convinced that within the concept of schizophrenia, different illnesses are hidden.”

13. He believed that the primary process, which was the background of the psychosis, was not as yet diagnosable.

14. Dementia praecox, he held, was far more widespread than the institution cases with an absolutely certain diagnosis. “One does not need to believe, as I do, in an especially frequent latent schizophrenia and does not need to consider it true that those cases which do not come to the notice of institutions are much more numerous than the institution cases.”

15. “Many schizophrenics come
to the outpatient clinic who would not otherwise be discovered and who are even now not considered incapable of functioning adequately.

16. "If other plant and animal pairs of traits had not forced the Mendelian theory upon us, we would never have considered any explanation for dementia praecox other than intermediate inheritance with infinite gradations in the intensity of the illness."

17. "Social unfitness, which must be defined differently in different environments, is no criterion for the biological boundaries of a sickness."

18. He states: "It appears most probable to me that a considerable part of the 'other psychoses' are genetically identical to dementia praecox." This view is a forerunner of the schizophrenia spectrum concept employed by Kety, Wender, and Rosenthal, but Bleuler's view is much broader.

19. He says: "For the present, the delineation of the different forms within the group schizophrenia presents unsurmountable difficulties."

20. "The basis of every genetic study on schizophrenia rests on very shaky ground."

21. "No one denies that an inherited predisposition plays an important role, although that has not at all been proven with the desired certainty."

22. "Does the addition of certain destructive forces to an existent predisposition first develop the sickness?"

- "Is it only the reaction of a psyche predisposed to sickness to various destructive forces?"

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**References**


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**the diagnostic ambiguity of postpsychotic depression**

The clinical and prognostic significance of severe depression in the aftermath of acute psychotic episodes has been pointed out recently by McGlashan and Carpenter (1976b). On the basis of case studies and clinical observations, they estimated that a clearly differentiated episode of depression follows acute psychoses in approximately 25 percent of all patients hospitalized with an acute schizophrenic reaction, although this incidence may be an underestimation due to the scarcity of detailed posthospitalization followup studies and confounding effects of medication. The syndrome appears to be associated with a good prognosis for recovery (although not from further episodic relapse) and occurs infrequently in chronic schizophrenic patients with acute exacerbations of their illnesses or in patients with poor premorbid psychosocial functioning (McGlashan and Carpenter 1976b; Roth 1970). Rather, the syndrome is typically observed in younger patients (in their twenties) who have a good premorbid level of functioning (McGlashan and Carpenter 1976b; Roth 1970).

While postpsychotic depression (PPD) may be a valuable prognostic sign, we disagree with McGlashan and Carpenter's (1976b, p. 234) conclusion that PPD is a phase in the course of schizophrenia. The level of premorbid adjustment, symptomatology, and clinical course of patients exhibiting PPD strongly suggest an alternative hypothesis—that many of these patients may be bipolar depressives who were misdiagnosed schizophrenic while in the acute manic phase of their disorder. Such cases of misdiagnosis are not infrequent, as evidenced by the recent findings that 50 to 92 percent of good prognosis schizophrenics studied fulfill research criteria for bipolar depressive disorder (McCabe et al. 1971, 1972; Taylor and Abrams 1973, 1975; Taylor, Gaz-