Environment and Inheritance: Opposing Forces?*

Harry Wiener

Abstract

High risk of schizophrenia requires high genetic predisposition to develop schizophrenia, plus an environmental trigger. A schizophrenic family environment is commonly believed to represent this trigger. The hypothesis is presented here that, on the contrary, a high predisposition to schizophrenia in significant others protects against overt illness. The trigger may be the predominance around the index individual of significant others with low predisposition to develop schizophrenia.

A footnote in the recent article in the Schizophrenia Bulletin (Vol. 4, No. 4), "Social Networks and Schizophrenia" by Hammer, Makiesky-Brarrow, and Gutwirth (1978, p. 530), states:

...it may be worth commenting that the assumption (e.g., Wender et al. 1977) that social etiological processes, like genetic ones, should be reflected in a higher incidence of schizophrenia in the "agents" (e.g., adoptive or biological parents) is not logically warranted.

I agree, and I would like to go one step further. What if this common belief were 180° wrong? What if the "agents" were characterized by a predisposition to schizophrenia lower than that of the population in general?

Let us consider a simple form of the multifactorial hypothesis: \( P + E = HR \) — something in the patient combines with something in the environment to produce a high risk of overt schizophrenia. Let us call the "something" in the patient a high predisposition to develop schizophrenia (HPD), and let us, in view of the above, see what happens if we call the "something" in the (human) environment a low predisposition to develop schizophrenia (LPD). In brief: \( P(\text{HPD}) + E(\text{LPD}) = HR \).

This equation suggests: \( P(\text{HPD}) + E(\text{HPD}) = LR \). A person with high predisposition to develop schizophrenia, in a human environment dominated by significant others with a high predisposition to develop schizophrenia, will be at a (relatively) low risk of developing overt schizophrenia. This sounds absurd — and yet ...

1. On page 701 of the same issue of the Bulletin, we see an abstract of an article in which Benjamin (1976) argues that proper statistical analysis of the work of Kety and associates gives a result that "violates the principle that genetic effects increase with greater consanguinity" (p. 1129). Without going into the controversy involving Benjamin, Kety (1976), and Lidz (1977), it may be pointed out that if Benjamin and Lidz are right in their criticism, one way out of the paradox is shown by the equation above. It suggests that the risk of overt schizophrenia increases with the number and genetic closeness of schizophrenic relatives, but decreases with the number of schizophrenics in the immediate family (parents and siblings). Whether HPD half-siblings are at greater risk of illness depends, in part, on whether they have been removed from the physical presence of the HPD parent.

2. Nyman, Nyman, and Nylander (1978) find that the incidence of regressive (classical, more severe) schizophrenia is greater among first-degree relatives of patients with nonregressive (pseudoneurotic, less severe) schizophrenia than among patients with the regressive form of the disease. Since, as they point out, the incidence of schizophrenia among first-degree relatives of patients with nonregressive schizophrenia is greater than among patients with the regressive form of the disease. Since, as they point out,
there could be no reason to expect patients with non-evident, neurosis-like states to have more diseased relatives than those who fall ill with classical schizophrenia (p. 188), they worry about errors such as sampling bias that may have produced this result. They do not consider the possibility of a countervailing protective effect.

3. Bleuler (cited by Teschke 1978) points out that the importance of genetic factors is less for severe cases than for episodic and benign cases. Higgins (1976) compared children of schizophrenic mothers reared by them or reared apart: There was somewhat more pathology and poorer academic adjustment in the reared-apart than in the mother-reared group.

4. There have been several reports of the inheritance of subtypes of schizophrenia (Kleist 1923; Baron and Stern 1976). This is a puzzle, for longitudinal studies clearly show changes from one subtype to another in the same patient. How can something so variable and environmentally dependent be inherited? It isn't. P(LPDP) + E(LPDB) = HR(subtype). Children with high predisposition to develop schizophrenia brought up in a family with a particular constellation of persons with a predisposition to cause schizophrenia will tend to show similar patterns of reaction, i.e., similar subtypes of schizophrenia. The familial trend of subtypes is social, not genetic.

5. Critics of the environmental view of schizophrenia have pointed out that findings of disturbed mother-child communications in future schizophrenic patients do not explain why one child in a given family develops schizophrenia, while others do well. Again, the family environment does not act alone to cause schizophrenia; it triggers an existing predisposition. If the patient's siblings have low predisposition to schizophrenia, they will find nothing amiss: P(LPD) + E(LPD) = LR.

The protection afforded by one or two parents with predisposition to schizophrenia, if it exists, can only be partial. As the child grows up, he is likely to enter an environment that is normal for us but pathological for him. Perhaps the longer the delay, the milder and more reactive the illness. Process schizophrenia may involve exposure from birth to an LPD mother, with far more severe and irremediable developmental changes.

Benjamin (1976) speaks with disapproval of the concept of poor (destructive, defective, illness-inducing) genes, to which she opposes the notion of good (constructive, superior) genes. One might go further. What are poor and what are good genes? Good for whom? Krebs (1976) has said: "good genes are ones which not only code for good characters, but also get on well with their fellow genes" (p. 13). I would add: A carrier of good genes is a carrier of genes of his fellow men. Risk and opportunity are determined not by your genotype alone, but by how well it fits in with the genotypes around you.

References


Kleist, K. The view of the schizophrenias as psychologic systemic illnesses (heredodegenerations) (Ger). Klinische Wochenschrift, 2:962-963, 1923.


The Author

Harry Wiener, M.D., is Director of Professional Information at Pfizer Inc., 235 E. 42nd St., New York, N.Y.