The transmission of the schizophrenia phenotype does not conform to a simple Mendelian pattern, even when we allow for incomplete penetrance. There are two models of transmission that fit the data available in the literature: a polygenic model and a multifactorial model (2). In a polygenic model, liability to develop a disease is continuously distributed in the population because of the additive effects of multiple genes at different loci. Only the individuals whose liability exceeds a certain threshold will manifest the disorder. The relatives of affected individuals have an increased mean liability compared with the population as a whole, resulting in more relatives manifesting the disorder. Multifactorial models allow extension of this concept, such that liability can be contributed by genetic and environmental factors in an additive fashion. Most of the discussion regarding the polygenic model applies to the latter.

Given the high prevalence of schizophrenia, these models suggest that the genes that give liability to the illness are not confined to schizophrenia patients and their relatives but are present in variable quantities in all humans, with a Gaussian distribution. Looking at the relatives of schizophrenia patients for a fertility advantage to explain the persistence of these genes is therefore not the right approach. These genes are an intricate part of the nature of *Homo sapiens*. They are not just an advantage to some but indispensable to all of us. The price paid is that 1% of the population develops schizophrenia.

When we look at diabetes as an analogy, it exists because evolution has developed a system dependent on insulin for the metabolism of glucose. The advantages offered by such a mechanism outweigh the disadvantages of having a proportion of the population affected by diabetes. The reason that the genes that give liability to diabetes do not disappear from the human genetic endowment, despite lower-than-average fertility in the individuals affected (3), is therefore clear when we look at the human population in its totality but would defy us if we analyzed only the fertility of siblings of diabetic patients. The same is true for schizophrenia.

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Dr. Haukka and Colleagues Reply

TO THE EDITOR: The idea in our article was to test whether mutations that increase the risk of schizophrenia would also bring some advantage that would appear as increased fertility among the siblings of patients with schizophrenia. The classic example of heterozygous advantage is hemoglobin S, which is protective against severe malaria in heterozygotes but causes often fatal sickle cell disease in homozygotes (1). If we understand correctly, Dr. Procopio feels that our approach was wrong because the genetic background of schizophrenia is polygenic. However, according to a recent review on human genetic evolution (2), "The rate of trait evolution tells us nothing about the number of genes involved. The intensity of selection and heritability are more important determinants of evolutionary rate than is the genetic complexity of the traits under selection." Given the strong selective disadvantage and high heritability of schizophrenia, our interest in whether the mutations predisposing to schizophrenia could also carry some advantage seems justified. We use the word "mutation" instead of "gene" to point out that we also believe that it is quite probable that the genes associated with liability for developing schizophrenia are quite useful but that the mutations within the genes that increase the risk of schizophrenia may not be useful and could be under selective pressure.

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Childhood Abuse and Suicidality in Women

TO THE EDITOR: Angela E. McHolm, Ph.D., et al. (1) reported recently on the relation between childhood physical abuse and suicidality among depressed women. They indicated they were unaware that prior "community-based studies have specifically examined suicidality among adults with a history of childhood physical abuse" (p. 934). In fact, a number of community-based studies have examined the specific relation between childhood physical abuse and suicidality (2). They have appeared in a number of peer-reviewed publications, including premier general medical (3) and child maltreatment (4) journals.

Dr. McHolm et al. depicted their specific focus on physical abuse as a strength. However, children are rarely subject to one form of abuse. There is considerable value in examining the ways in which combinations of child maltreatment are associated with psychiatric outcomes in adulthood (5). Other criticisms of the report by Dr. McHolm et al. can be generalized to other studies of child maltreatment and suicidality. First, they ignored the relations between the duration, intensity, and frequency of abuse and the kinds and degrees of psychiatric outcomes. Second, the kinds and degrees of psychiatric outcomes may vary with the developmental stage (cognitive and socioemotional) at which child maltreatment occurs. Third, popular awareness of physical abuse emerged in 1962 with the reports by Kempe and colleagues of battered child syndrome (6). Variations in awareness and attitudes about child maltreatment over time (7) may be associated with age cohort effects in adult psychiatric outcomes. Dependency and vulnerability in later adulthood may arouse memories of similar feelings in childhood. Older adults who reached adolescence before reports of battered child syndrome became well known, when child maltreatment was not discussed openly or publicly, may be less inclined to report el-