Schizophrenia and Attentional Deficit Disorder (ADD): Reply to Kaffman

by Joseph Marcus

Abstract

Kaffman's critique (1986) of the National Institute of Mental Health-Israeli High-Risk Study pointed to the existence of children who were considered to be suffering from Attentional Deficit Disorder (ADD) among the offspring of the schizophrenic parents—who were considered adult ADD patients rather than schizophrenic patients. We feel this viewpoint represents a misunderstanding of the nature and genesis of ADD and schizophrenia. While a subgroup of the offspring of schizophrenic parents do show "ADD-like" behavior, we believe that their disorder represents a separate and different entity. This is based upon the epidemiological evidence of the differing family patterns on the one hand, and some findings of differing attentional deficits on the other hand. A literature review of these findings is presented as a basis for our view about the differences between ADD and schizophrenia.

Kaffman's critique (1986) of the Israeli High-Risk Study pointed to a very relevant issue which deserves some clarification. Having noted the many signs of central nervous system dysfunctioning that we found in the children born to schizophrenic parents, he suggested that these findings "point to the presence among the index children of a typical syndrome of attentional deficit disorder" (p. 155). Kaffman then hypothesized that there was a subgroup of Attentional Deficit Disorder (ADD) parents in the sample of schizophrenic patients who were included in the project.

It is our opinion that this represents a common misunderstanding which exists in the literature and among practicing psychiatrists. Therefore, we feel it is imperative to clarify this issue.

In the early literature on Minimal Brain Dysfunction (MBD) which has more recently been appropriately designated as Attentional Deficit Disorder (ADD), there were several reports regarding adults who were schizophrenic. Psychiatrists who took the histories of these patients found evidence of some behaviors in childhood that seemed typical of MBD as it was viewed at that time. Wender (1971), in one of the first complete descriptions of this syndrome, alluded to the possibility that some children with MBD might develop schizophrenia, but his present opinion (personal communication) is that this is clearly not so. The same view was put forward by Bellak (1979) based on his clinical experience with adults—which he tried to support with secondhand information regarding the initial reports of our findings in the Israeli High-Risk Study. In a retrospective "catchup" study, Menkes et al. (1967) also reported on four psychotic cases who had been considered to have had MBD during childhood. However, all of these earlier reports have now been supplanted by very different results from other studies, some retrospective, but more importantly, others that are longitudinal and prospective.

Presently, there is a large body of data from many studies that reveals a clear picture—ADD seems to run in families characterized by adults suffering from the behaviors of residual type ADD accompanied by antisocial personality disorders, alcoholism, and drug abuse who give birth to children who have ADD. A subgroup of these children later grow...
up to be normal adults while others develop psychopathology that inevitably presents as antisocial personality disorders, alcoholism, or drug abuse, but not schizophrenia. Some of the principal studies that have demonstrated this pattern include those of Cantwell (1975), Goodwin, Schulsinger, and Hermansen (1975), Blouin, Bornstein, and Trites (1978), Hopkins et al. (1979), Dykman and Ackerman (1980), Loney et al. (1981), Satterfield, Hoppe, and Schell (1982) and Weiss et al. (in press). This issue has been carefully reviewed by Weiss (1985). Bellak (1985) also recently revised his earlier observations and described ADD psychosis as a separate entity from schizophrenia.

At the same time, there is now a growing body of evidence that in many studies of the offspring of schizophrenic parents there exists a very clear subgroup of children who have neurobehavioral deficits, and that these children are similar and appear in similar frequency in different studies and in different countries. This finding became clear in the recent meeting of the Consortium of At-Risk Researchers in Schizophrenia in April 1985. Those results are also reported by Fish (1977), Erlenmeyer-Kimling et al. (1984), and Marcus et al. (1981, 1985).

Thus, from the epidemiological point of view, it would seem that these two disease entities are different and separate, even though the childhood behavior appears on the surface to be similar—both being characterized by attentional deficits, motor deficits, and perceptual deficits.

However, it is our belief that when we begin to investigate these external behaviors on a more basic neurophysiological level, we will discover that the disturbances are in fact different. Although there is not yet a great deal of evidence to support this hypothesis, there is a body of promising work by Nuechterlein (1983, in press) and Nuechterlein and Dawson (1984) that demonstrates distinctly different defects in sustained attention that are found in groups of attentionally deficient schizophrenic offspring and in children with a diagnosis of ADD.

Nuechterlein (1983) examined groups of children using his version of the continuous performance test (Rosvold et al. 1956) and analyzed the data using signal detection theory indices rather than the traditional raw error score. This procedure allowed the investigators to characterize the subjects in terms of the accuracy of their discrimination of target (signal) and nontarget (noise) stimuli, as well as by the response criteria levels—i.e., the amount of perceptual evidence that the subject demands before responding that a given stimulus is a signal. The results suggested that the schizophrenic offspring showed impaired signal/noise discrimination (i.e., a more perceptual-cognitive disturbance), while the ADD children had low response criterion levels consistent with an impulsive cognitive style. Subsequent studies (O'Dougherty, Nuechterlein, and Drew 1984; Nuechterlein, personal communication) have suggested that the lowered response criterion in ADD children may be the primary discriminator between these two groups, as some ADD children also show impaired signal/noise discrimination.

In summary, we would conclude that although a subgroup of offspring of schizophrenic parents do exhibit "ADD-like" behavior, they are not suffering from a disorder identical to that of other children from other families who have ADD. Furthermore, as will be presented in our upcoming article in Schizophrenia Bulletin (Marcus et al., in press), it is specifically these offspring who are truly at risk of becoming schizophrenic during their early adulthood.

References


Fish, B. Neurobiologic antecedents of schizophrenia in children: Evidence for an inherited, congenital neurointegrative defect. Archives of...


Loney, J.; Whaley-Klahn, M.; Kosier, T.; and Conboy, A. A paper presented at meeting of the Society of Life History Research, Monterey, California, November 1981.


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