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SCHIZOPHRENIA—A GENETIC STUDY[1]

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SUMMARY

102 families of schizophrenic patients were studied by drawing pedigree charts. The affected relatives failed to manifest any sexwise difference and so were the probands. Parental consanguinity doesn't seem to augment positive family history. When Slater's computational model was applied to assess the mode of inheritance, the study favoured polygenic hypothesis.

The genetic contribution to the etiology of Schizophrenia, which was pioneered by Rudin has been substantiated by different workers. In the recent past numerous studies have been undertaken to unraval the mode of inheritance. The polygenic hypothesis has been adopted by Gottesman and Shields (1967) and by Kringlen (1967). Karlsson (1966) proposed a two gene model, but he later revoked his model in favour of Slater's (Slater, 1966). Slater and Tsuang (1968) favoured a single dominant gene hypothesis. Miron Baron (1980) proposed a polygenic theory.

The present study was designed to assess the occurrence of this illness in the families of schizophrenic patients. It was aimed to estimate the parental consanguinity and also to determine the possible mode of inheritance.

MATERIAL AND METHOD

All new cases of schizophrenia who reported to the Department of Psychiatry, Govt. General Hospital, Madras, from Jan. '81 to April '82 were selected. However, only those patients who fulfilled the Feighner's criteria for schizophrenia (1972), were chosen for the study. Further 4 cases had to be discarded for want of adequate family history. Thus the final sample studied was 102 (M-48, F-54). The sample belonged to the South Indian culture. For each patient a pedigree chart was drawn in a separate sheet, depicting the details of the family members stretched over three generations. The data about the familial occurrence of similar illness, parental consanguinity were recorded. For the purpose of the analysis only those members of the family who had schizophrenia, or schizophrenia like psychosis as made out from the history were taken as members affected with similar illness as that of the index cases. Cases of affective disorder, suicide, mental retardation, organic brain syndromes, alcoholism, neurosis, and personality disorders were ignored.

Slater's (1966) computational model was used for distinguishing polygenic inheritance and the effects of a single dominant gene. Bilateral pairs are computed according to the following formula,

$$\text{Bilateral pairs} = 2 \times \frac{\text{Paternal cases} \times \text{Maternal cases}}{N-1}$$

where N is the number of affected cases in the family, thus correcting the family size. The correct number of unilateral pairs is obtained by subtracting the corrected number of bilateral pairs as calculated by Slater's method, from the total number of affected pairs. Deviation from expected ratios was statistically determined. Only those families who had two or more ancestral secondary cases could be taken for Slater's computational model.

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RESULTS AND DISCUSSION

The affected relatives (M-51, F-41) as well as the probands (M-48, F-54) failed to manifest any difference with regard to sex distribution.

Out of 78 patients for whose parents ancestry could be traced to explore their relationship, 25 (32.05%) had consanguinous parents. In these families with parental consanguinity, positive family history was obtained in 14, is which not at variance statistically, with corresponding families without positive family history (11). Perhaps, consanguinity between parents doesn't recognizably play a role in augmenting family history of schizophrenia. Ahmed (1979), in his study among Sudaneese community reported parental consanguinity as 43.7% which was not significantly different from that of their general population.

An attempt was made to explore whether this condition is caused by a single dominant gene or by polygenes, by adopting Slater's computationl model. Some of the previous studies, which have utilized this method in schizophrenia (Slater and Tsuang, 1968; Tsuang, 1971), supported a dominant gene hypothesis. Michael et al. (1972) come to the conclusion that if highly restrictive criteria are used for family members' illness, then it favours a dominant gene hypothesis. As criteria for illness are broadened, the findings tend to favour a polygenic hypothesis. Miron Baron (1980) by adopting the same method favoured a polygenic theory, but added that his observed deviation from polygenic predictions was not far from significant. In the present sample, 21 probands had two or more ancestral secondary cases (N-56). 12 bilateral and 44 unilateral pairs were observed (corrected for family size). These figures do not deviate from polygenic predictions, i.e., a 2:1 ratio of unilateral to bilateral pairs ($X^2 = 3.524$, N.S.).

CONCLUSION

Parental consanguinity and sex doesn't appear to contribute to schizophrenic predisposition. Though the outcome of this study has favoured a polygenic hypothesis, the present investigation has its limitations, for, the identification of ancestral secondary cases rested on history furnished by the elder members of the family and psychiatric evaluation could be carried out only in some cases.

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