INTRODUCTION

The symptoms of what is now called schizophrenia have fascinated physicians and philosophers for thousands of years. Nevertheless, what is now known as schizophrenia was not even described as a disease entity until 1896, when Emil Kraepelin brought together, under the term dementia precox, a variety of psychotic syndromes previously believed to represent separate diseases. Inspite of advances in molecular biology, cytogenetics, neuro-chemistry and brain behaviour, we can not pinpoint any necessary biological defect in all or most schizophrenics. Evidently, not only are there many theories for the causes of schizophrenia, but also there are insufficient data to decide which is the correct theory.

Even as Eugen Bleuler (1911), introduced the term schizophrenia, Rosanoff and Orr (1911), published the first family pedigree study designed to explain the etiology of insanity in Mendelian terms. Ever since the first attempt of Rudin (1916) to study schizophrenia according to Mendelian principles, tremendous strides have been made in the genetic aspects of schizophrenia. There can be little doubt that genetic factors play a role in predisposing persons to some forms of schizophrenia. The position has been clearly stated by Gottesman and Shields (1972), who concluded that the results of family and pedigree studies fit equally well with several possible modes of transmission. The search for genetic linkage offers one possible way of avoiding this impasse.

In using linkage to investigate a disease, such as schizophrenia, the disease itself acts as one characteristic and some

genetic marker is the other.

The requirements for detecting linkage in man are that there should be two specific and readily ascertainable characterestics which are known to segregate in families (Race and Sanger, 1975). The marker is a well established genetic system known to occur throughout the population, but with different frequencies in subjects with mental diseases and it can be used to estimate useful parameters for genetic understanding of the disease being studied.

Dermatoglyphics, determined by polygenic inheritance, has been demonstrated as one of such tools for the diagnosis and understanding of the genetics of many human pathogenic abnormalities (Alter, 1966; Penrose, 1968; Holt, 1973). The earliest studies of dermatoglyphics in schizophrenia (Poll, 1935; Moller, 1935;) have attracted many researchers into this field of study. The conflicting findings of these workers and the limitations of these studies have been pointed out by Balgir and Srinivasa Murthy (1982). Further, to my knowledge, none of these studies have focussed on the schizophrenic families.

Of great interest is another genetic marker, HLA, which was first reported as a possible marker of schizophrenia by Cazullo et al (1974). But, recently, Rudduck et al, 1984, have stated that most of the significant associations, between HLA and schizophrenia reported in the literature are non-confirmed or contradictory. Furthermore they have mentioned that a true association can not be excluded for some antigens where the results have been confirmed.

Hence, a need exists for more data, not only on schizophrenic patients but also on their families with regard to these genetic markers.