

"THOUGH THIS BE MADNESS, YET THERE IS
METHOD IN IT": A SOCIOLOGICAL APPROACH
TO MADNESS

By

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A Sociological Approach to Madness

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ABSTRACT:

Sociological approaches to mental illness have been dominated by the epidemiological approach. The lack of success of this approach is evidenced in the paucity of definitive findings it has produced, and in the lack of any generally accepted theory of the etiology of "schizophrenia" and the other functional mental disorders. It is the fundamental thesis of this work that the model of "schizophrenia" used by the sociologist is essentially misconstrued. Consequently, rather than suggesting methodological refinements in case finding techniques, the whole rationale underlying the epidemiological approach is subjected to a thoroughgoing critique. By treating the recurrent problems which have hampered research as investigable problems in themselves, a radically different approach to doing the sociology of mental illness is suggested. Rather than accepting psychiatric definitions of what constitutes a case, it is suggested that sociologists (and by implication psychiatrists) should concern themselves with the social meanings of mental illness; and treat "schizophrenia" as a label which defines the relationship between individuals and not as defining a property of an individual.

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Classifications in social research are mainly used to establish relations between a number of variables. The crucial question, therefore, is whether these relations, the empirical findings we are looking for, are much affected if we interchange one reasonable index with another.

P. F. Lazarsfeld, "Evidence and Inference in Social Research," in D. Lerner (ed) Evidence and Inference, N.Y. The Free Press, 1959:108

We begin with a paradox!

How is one to understand irrational behaviour? If one holds that schizophrenic behaviour is bizarre, unintelligible and meaningless, what is the appropriate methodology for understanding the phenomenon? What do we accept as appropriate data, and what exactly are we seeking to understand and explain? At first glance, it would seem fruitless to examine the phenomenon in terms of the motives and intentions of the schizophrenic himself. The schizophrenic, if we accept the disease model of the disorder, cannot be said to be acting of his own volition. He is not responsible for what he is doing, rather his behaviour must be attributed to the disease process. It does not make sense to ask him why he did such and such, or why he lives where he does, for he is not held responsible for his behaviour.

The proper focus of sociological enquiry within this frame of reference would then seem to be not what causes the behaviour we recognize as Madness, because we know that it is caused by the illness, but what causes the illness itself. One method which can be employed

is to examine the rates of its occurrence in different areas; and then assess the contribution of social and environmental factors in determining those rates.

Sociological approaches to mental illness have been dominated by the epidemiological method. MacMahon et al. (1960:3) have defined epidemiology as the "study of the distribution and determinants of disease prevalence in man." Accordingly, if schizophrenia is accepted under the rubric of the disease label the two main tasks of sociology in the epidemiological exercise are first, estimating if significant rate differentials exist in social structures; and second, providing an adequate explanation of those rate differentials. While this latter may be interpreted as a search for the etiological factors associated with the disorder the specific task is to explain the rate differentials. While these two types of explanation are not necessarily exclusive they do suggest different levels of analysis. For example, while we may be able to isolate factors associated with significant rate differentials (high mobility of schizophrenics may explain their overrepresentation in a particular census tract) it remains to further analysis to determine the factors of etiological significance in the development of the disorder. (Is it the stress associated with mobility which precipitates the disease?)

The significance of this point will become more apparent as we proceed; but for the moment we would be justified in commenting that the success of the epidemiological approach to unravelling the etiology of schizophrenia can be measured in the paucity of definitive

findings which have emerged from the considerable research effort, and the lack of a widely accepted theory of the relationship between sociological factors and psychoses.

Wallace (1965:299) recently pointed out that:

(the) evaluation of the progress so far made in social epidemiological studies of schizophrenia cannot be a complacent one. Not only are there few studies which meet rigorous methodological standards: the studies as a whole are remarkably non-cumulative. By this I mean that, considered in series, the studies either do not deal with the same variables, or if they do, do not so much pursue leads revealed in the earlier studies as attempt to repeat an essentially similar study on a different population.

On a more pessimistic note, Wardle (1962) has expressed the opinion that in the absence of unequivocal proof of the relationship between sociological variables and the psychoses, sociology should limit itself to understanding and changing the public's attitudes towards mental illness. This, to the writer, seems an unduly nihilistic approach to the problem, but does sum up the current dissatisfaction with epidemiological research in this area.

Dohrenwend (1966) after attempting to resolve some of the anomalies in the research literature, insists that before sociologists can conduct effective research in this area, they must agree on an adequate definition of what constitutes a case. Such agreement would meet certain of Wallace's criticisms about the non-cumulative nature of research, and focus the different researchers' attention on the same problem.

But Dohrenwend's is only a partial answer for it stops short of suggesting that sociologists should actively criticize the

perspective within which they approach the problem. It is the contention of this research that significant progress by sociologists in the field of the epidemiology of schizophrenia has been impeded by their uncritical acceptance of the disease entity model of the disorder.

But what happens if we take the disease entity model as problematic, and suspend for the moment judgments as to the rationality or not of the schizophrenic's actions? what implications does this have for methodological approaches to the study of schizophrenia (as patterned action)? and to the type of theoretical explanation which has been given to the epidemiological research?

(i) Predominance of the Disease Model

It is a common practice in any scientific enterprise to develop a model to explain the data generated during the research. Models can be extremely useful in clarifying the nature of the relationship between variables, in suggesting new areas to investigate and eventually in leading to the construction of a substantive theory. In Greer's terminology (1970:141-142), they serve as guiding "metaphors", which are created by analogising:

Forms useful in other contexts are applied to the problem at hand; they are forms that lead us to abstract certain aspects in terms of their interrelations in the observed events.

However, it is often because of their very utility that it is common for those who espouse a particular model to be reluctant to abandon it, and instead concentrate on forcing all new information,

or information which appears to contradict into the model they
2.
already hold. It is suggested that this situation has obtained in
the study of mental illness, a field which has been dominated by
what has been termed the medical model, or disease model of mental
disorder.

Anyone attempting to legitimate a sociological approach to
the study of mental illness can be excused a certain trepidation
and diffidence on entering a field which has been so dominated by
models of personality which are akin to the concepts and practices
of physical medicine. (see Szasz, 1962; Laing, 1964; Leifer, 1971;
Cooper, 1970) Within the framework of this medical, or disease
model the basic cause of any emotional or psychiatric abnormality
is assumed to be inside the individual organism, and the reason
for any behavioural disturbance is sought in terms of some
physiochemical imbalance or unique patterning of interpersonal
relationships.

Mental disorders are thought to be analogous, or at least
3.
not radically different from physical diseases. When a patient is
diagnosed by a psychiatrist as having schizophrenia, the psychiatrist
is in effect asserting that there is something wrong with the
"identified" patient which causes the disturbance between his
experience and behaviour. The research exercise becomes one of
seeking the cause of schizophrenia in the same manner one would
attempt to isolate the cause of any physical disease.

Sociological variables are assumed to have, at most, a
secondary significance in the onset of psychiatric breakdown, the

recurrent argument being that environmental tension, stress, strain, pressure, precipitate psychiatric disorder in a predisposed personality. As such, environmental factors may be necessary conditions, but it is questionable whether they are ever sufficient conditions for breakdown.

Given this climate of opinion, it is not surprising that sociologists have been reluctant in deriving a sociological definition of illness which is independent of psychiatric models, and have either implicitly or explicitly accepted a psychiatric definition of what constitutes a case in terms of rates of treated disorder drawn from hospital records, or the psychiatric evaluation of individuals drawn from a non-patient population. In a very real sense it can be argued that sociologists involved in epidemiology have been concerned with rates of mental illness as a sui generis phenomena, rather than mental illness per se, and have left the investigation of the behaviour associated with the diagnosis of a case to the psychiatrists themselves.

This attitude is echoed by Schatzman and Strauss (1966:4) who have claimed that:

'it would be more fruitful for sociology if more research were done about psychiatry than in it or for it.' (emphasis added)

The authors' are specifically concerned with issues of professional growth and identity. To the writer, two more fundamental problems present themselves within this frame of reference - (1) what factors are associated with the formulation of a psychiatric diagnosis? and (2) what factors are associated with an individual's identification as being in need of psychiatric care and his subsequent presentation to a psychiatric functionary?

An analysis of these factors would have a major bearing on Dohrenwend's statement above, and suggest an alternate perspective for sociological approaches to the etiology of schizophrenia. Of immediate concern, if this viewpoint is accepted, is an investigation of the correspondence between the sociologist's categorisations and the phenomena to which they refer. Specifically, if it can be shown that the data which the sociologist takes as objective and true indices of mental disorder are themselves the product of a complex social process, then explanations of those phenomena are inadequate, and investigation should concentrate, not on rates of mental illness, but on the social meanings of mental illness.

This latter needs more detailed explanation, which will become more apparent as the thesis proceeds, but for the moment we would argue that in order to derive formal definitions of a phenomenon, in this case schizophrenia, it is necessary to study the real-world patterns of action and meaning associated with the use of that term. In Schutz' (1954) terminology, in order to understand social action we must first understand that action in terms of the meanings actors give to the situations in which they interact. The implications for a sociology of mental illness are apparent. We must first understand the behaviour associated with the label "schizophrenia" or "mental illness" and the situations in which such a label is applied (or contexts in which the same behaviour is not so labelled). This involves, as a corollary, an investigation of the processes by which individuals come to enter psychiatric care and become "cases" in the epidemiological studies.

To return to the paradox, it might be argued, with some substance, that mental illness is inherently irrational behaviour and is thus not susceptible to the type of analysis Schutz and others have suggested is appropriate for social action. We will argue this point in a later chapter (Chapter 3) but even if we concede for the moment (and this point has not been decided by any means) that the schizophrenic's behaviour is irrational, this does not preclude us examining the social processes involved in arriving at psychiatric decisions (unless these too are considered as irrational) which itself has important implications for a sociological approach to mental illness.

(ii) Statement of Concern

What I want to do in the thesis is almost the opposite of what Dunham (1968) suggests in his recent evaluation of epidemiological studies. Dunham consistently misses the point of using certain of the recurrent difficulties which arise in the epidemiological research constructively. Rather than treating these difficulties as investigable problems in themselves, Dunham treats them as methodological difficulties which must be overcome or controlled. Consequently, in what follows, rather than suggesting methodological refinements in case finding techniques the whole rationale underlying the epidemiological approach as it has been used by sociologists will be subjected to a thorough-going critique. By treating the recurrent problems which have hampered research as investigable problems in themselves a radically different approach to doing the sociology of mental illness will be suggested.

What seems to be needed is a reformulation of the problem, specifically a sociological approach to the problem which is not informed by an uncritical acceptance of the disease entity model and the methodology it implies.

Wide reading of the extensive literature on schizophrenia suggests to the writer, that the schizophrenic phenomenon, or the phenomenon of mental illness is not what those sociologists engaged in epidemiological research have assumed it to be, and that an adequate sociological approach has been hampered by uncritical acceptance of psychiatric definitions in the criteria of case identification. In the light of statements like that of Schatzman (1971:164) a psychiatrist, that:

all that is certain about "mental illness" is that some people assert that other people have it, Epistemologically, mental illness has the status of an explanatory concept, or as a working hypothesis. No one has proven it to exist with scientific precision and reliability.

and Cooper (1970:16) that:

the term schizophrenia has done much to confuse the real problem and there is not one shred of unequivocal credible evidence to support the inclusion of schizophrenia as a disease-entity in the field of medical nosology.

such definitions should at least be problematic.

These conclusions have been reached not on the basis of the macrosociological research typical of the epidemiological approach; but first, by a phenomenological examination of the actions of the "diagnosed" schizophrenic in relation to the groups with whom he has meaningful interaction; and second, by examining the manner in

which his construction of reality or imputations of meaningfulness (or meaninglessness) to his shared social relations conflict with those of the individuals with whom he interacts.

The assumption that the operational definitions used by the conventional psychiatrist, or psychiatric functionary, correspond to his own definitions of these phenomena, has resulted in the sociologist imposing classifications on to his data rather than having them emerge from the data to guide the course of analysis and collection of further data. The resulting consequences are an unwillingness to consider alternative explanations of the relationships among the data, and the collection of inadequate information to test the validity of competing explanations. It is suggested that rather than imposing what are arbitrary definitions on the phenomena (by accepting psychiatric classifications, or examining rates), we should attempt to understand the meaning of those phenomena for those involved.

Sociologists have typically been concerned with the "why?" and the "how much?" of mental illness; but such questions presuppose an answer to prior questions about the nature of the phenomena. Apart from the work of Scheff (1967) and the research this has stimulated, there has been little systematic questioning of the assumptions of the medical model in the sociological literature. It is true that Scheff's work does not examine the nature or causes of the disorders themselves; but by treating them as an aspect of deviance, and treating deviance nominalistically, he raises the whole issue of the reality of the disease entity by highlighting disparities in the imputations of

mental-disease labels. This work presents a fundamental critique of the practice of those involved in epidemiological research of assuming that the operational definitions used by the psychiatrist or other psychiatric functionary correspond to their own definitions of those phenomena; and of the nature of the phenomena themselves.

In the research that follows, the status of schizophrenia and by implication other functional mental disorders is taken as problematic. Schizophrenia will be treated as more a hypothesis to be tested, than an assumptive framework to guide the analysis of the data. If schizophrenia is considered as not a disease entity, i.e. if it does not submit to nosological classification, then it is not accurate (nor worthwhile) to ask questions about etiology. Instead, if schizophrenia is considered as not a disease entity existing within the individual; but rather as a pattern of mutual accommodation between individuals, then it will be possible to examine the relationship between the individual's signs and symptoms which are diagnosed by the psychiatrist as the 'disease' "schizophrenia", to more or less specifiable patterns of interaction within the patient's family (or other significant groups with whom he interacts). If such interactions can be shown to be meaningful, and when viewed in the light of the individual's interactions with others as a form of communication, then it is not accurate to relegate that behaviour to the 'process' of a disease.

4.

(iii) Purpose and Outline of the Thesis

The purpose of this thesis is not to develop a grand theory of the etiology of schizophrenia; but to suggest a way sociologists might go about doing the sociology of mental illness which is radically different from that employed in the epidemiological research.⁵ One reason for this comparatively modest aim is that the research will rely on the analysis of secondary data and while this may be adequate to suggest fruitful methodological and theoretical lines of enquiry, it will not be sufficient for the formation of any substantive or formal theory. The choice of material has been eclectic, and in each instance guided by the inadequacies of the previous material reviewed.⁶ While this may give a broader picture of the problem it is difficult to synthesise this material because of these very methodological and conceptual difficulties. To attempt a synthesis without the appropriate data would leave the writer open to the same criticisms of missing data, and fallacy of the wrong level to be made of the research reviewed.

In Chapter Two an attempt is made to make sense of the epidemiological literature on schizophrenia. The functions of epidemiology as a method are discussed, and then after a brief review of the data on social class and mental illness we plunge into a discussion of the methodological problems which have been encountered by this epidemiological approach. The rationale for treating methodology before the various theoretical explanations is simply that by examining their data base and adequacy of the concepts employed the discussion of the theories will be considerably more

than a choice between competing hypotheses.

Two problems recur in this methodological discussion: the fallacy of the wrong level and the identification of a case. The fallacy of the wrong level is exhibited in the tendency, of researchers in this field, to reason from aggregate data on, say, areas and rates of illness to the properties of individuals. The reasons for the illegitimacy of these inferences are discussed and recognised as an aspect of the more general problem of missing data. While the problems associated with the identification of a case are an aspect of this missing data problem, the more salient question is raised of the correspondence between the sociologist's categories and what he seeks to explain.

In the second section of Chapter Two, after a brief digression on causes, the discussion turns to the major theoretical explanations which have been offered of the epidemiological data. We begin by discussing studies using incidence rates of reported illness and it soon becomes apparent that the problems discussed in the methodological section have a major bearing on how much credence we give to each of the theories which have been offered. The early explanations were ecological in nature and could be grouped under the two general headings of the 'breeder' and 'drift' hypotheses. We discuss these two approaches in detail and find that the evidence for neither is particularly convincing. A modified drift hypothesis is suggested which considers the disorder as not being caused by pathogenic factors in the environment, and in which the model of the schizophrenic is not that of an aimless individual. It is suggested that the differences

in rates for different areas of the city may be explained by the schizophrenic's deliberate choice of those areas as havens from a stressful environment.

A recurrent problem with the ecological explanations is that they either implicitly or explicitly assume something about the interpersonal environment and experiences of their cases. Unfortunately, much of this speculation is irrelevant because their data has been of the wrong order.

This same criticism can be made of much of the explicitly sociopsychological explanation. In this category 'social isolation' is the most glaring example of a hypothesis which has been suggested without the requisite interpersonal data.

A common feature of much of the epidemiological theorising has been the implication of stress in the etiological process. After discussing the status inconsistency approach (which is inconclusive) we move to a discussion of the community studies which do include some data on the interpersonal experience of their cases, which is lacking in the studies relying on hospital or other statistical records. It is suggested (with a slight modification of Leighton et al's viewpoint, 1963) that the response to stress may be adaptive unless it is labelled as maladaptive by self or others and this label is confirmed by a psychiatric agent.

The discussion of the epidemiological theories is inconclusive but does set the scene for the remainder of the thesis which revolves around the twin questions:

a/ what is it that sociologists are seeking to explain?

b/ what is the interpersonal experience of individuals who come to be labelled as cases?

It is suggested that the family experience may be the most salient among the experiences of individuals (or most individuals) labelled as cases; but this topic is left to one side until, in Chapter Three, the concept of schizophrenia used in the epidemiological studies is examined.

Chapter Three begins with a discussion of the case finding techniques used in the hospital and community studies. Throughout we are concerned with the legitimacy of using psychiatric diagnoses as definition of cases when there is so much confusion within the psychiatric literature as to what schizophrenia refers. We take the position of Bannister (1968) and Laing (1970) (among others) that 'schizophrenia' is not the diagnosis of a fact; but an assumption or hypothesis and should not be accepted uncritically by sociologists working in this field. We take up again the point raised in Chapter Two that actions in response to stress become maladaptive if they are regarded as such by self or others and become psychiatric disorder when diagnosed (labelled) as such by a psychiatric functionary.

By treating 'schizophrenia' as a hypothesis, and by not regarding case identification as a process identifying or recognising a fact, we move into a discussion of 'mystification' in which the illness label is considered literally as a dehumanising event which serves to deny the rationality of an individual's actions by attributing them to a disease process. This provides a useful

introduction to the work of Scheff (1967) and the labelling approach to mental illness.

After discussing the main points of this approach which treats deviance nominalistically, we consider some of the empirical work it has stimulated. This is almost exclusively concerned with the official labelling process and it is suggested that there is a complementary approach which examines the stabilisation of patterns of action prior to the official act. The stimulus for this approach is in Scheff's concept of residual rules which, contrary to Scheff, we consider not as cultural stereotypes but as the rules which are invoked to make actions accountable within the family or other significant group. Consequently, it is possible to consider the interaction within the family or group as normal with reference to these rules when such action if taken out of this context would be a candidate for an illness label. The actions, which may seem strange and bizarre, are not deviant unless they are so labelled by being brought to the attention of a psychiatrist. Normalising, as we term it, is considered as an aspect of mystification and is taken to refer to the concerted actions of the family as a whole to deny the actions and experiences of one of its members. The ideas developed in this Chapter suggest a radically different way of looking at mental illness and in Chapter Four we look at the implications of this approach.

If the status of schizophrenia is treated as problematic, and if the ideas developed in Chapter Three have any validity, the concern of sociology should be not with etiology or causes,

but with examining an event or action as part of an interacting sequence and with examining its intelligibility as such. An alternative approach to doing the sociology of mental illness is suggested which directs our attention to the social meanings of the schizophrenic label.

Much of the epidemiological literature suggested a need for a mediatory variable intervening between the socio-cultural and environmental process and the individual response. In Chapter Four the epidemiologists' approach to the family is discussed and found to be lacking because they assume, but fail to analyse the interpersonal processes occurring within the family. To fill this gap the clinical studies are discussed. After considering the inadequacies and implications of the 'trait studies' we consider the work of the Lidz, Wynne and Bateson groups on the total family. The really significant point to emerge from this discussion is that the type of approach developing in this area is consistent with the ideas developed in Chapter Three.

In these studies an attempt is made to relate the schizophrenic's actions to the patterns of communication and/or action within his family. In this sense the schizophrenic's actions are considered as normal for particular families. These actions are not caused by the family interaction but are a pattern of accommodation to the actions of others which have developed over a period of time. To understand these actions it is not sufficient to consider them in isolation but with reference to the interaction within the family as a self

defined whole. If these actions are normal for a particular family, it is not clear why they should ever come to be labelled as schizophrenia. It is suggested that the labelled actions of the schizophrenic are essential to the maintenance of the status quo within the family. The label is applied when an individual's actions threaten this stability. The label serves to invalidate this threat, and the individual may actively seek to invalidate his actions in order to maintain the family stability. Adolescence is suggested as the period in which these threats are most likely to occur, a period which has been associated with the onset of the disorder in the clinical, and epidemiological studies and in the modified 'drift hypothesis' suggested in Chapter Two.

At this point it would have been satisfying to pull together the disparate evidence from the clinical and epidemiological research and suggest a grand theory of the social meanings of schizophrenia. This is not possible because of the restrictions in the data reviewed. Rather than attempting a synthesis, it is suggested that sociologists should reject the journeyman role they have assumed in psychiatric research and actively criticise psychiatric models of mental disorder by focusing on the social meaning of the mental illness label for the individual and the group from which he originates. Rather than accepting the diagnosis as the recognition of a fact it should be considered as defining a relationship amongst people and necessary for the maintenance of situational definitions.

(iv) A Note on Terminology

It is something of a truism to argue that the categories used in organising data have an inordinate influence on the type of explanations offered to explain the relationships observed amongst that data. One of the fundamental arguments of this research is that the adoption of psychiatric categories (and the attendant assumptions which underlie them) has hampered the development of a sociological approach to the phenomenon of mental illness. The temptation is great to engage in neologisms which, because this research relies on secondary data, would serve only to obfuscate the issue. Accordingly we retain the terminology used in the original research reports. However, it must be clear at the outset that the use of such terms as 'patient', 'schizophrenic', etc. do not carry with them the assumptions of an underlying disease process, but are considered simply as labels, which have been used to identify the behaviour we are interested in. This usage is reflected in the term 'identified patient' or 'identified case' by which we understand a person who has been labelled as being mentally ill by a psychiatrist or other psychiatric functionary.

Concepts such as leadership, dependency, introversion and extraversion, nurturance and many others become the object of detailed study. The danger is, of course, that all these terms, if only thought and repeated long enough, assume a pseudoreality of their own, a measurable quantity in the human mind which is itself conceived as a phenomenon in isolation. Once this reification has taken place, it is no longer recognised that the term is but a shorthand expression for a particular form of ongoing relationship.

Watzlawick, P. et al. The Pragmatics of Human Communication. 1968, N.Y. Norton: p. 27

The research on the etiology of schizophrenia may be divided into two broad areas: the clinical, and the epidemiological. We will look more fully at some of the clinical work in Chapter Four, however our immediate concern is with the epidemiological research, for it is in this area that sociologists have been most completely involved.

Section One: Conceptual and Methodological Problems

a/ The Empirical Findings

It would be impossible to do justice to the variety of techniques employed in the analysis of the epidemiology of schizophrenia. The sheer number of independent variables considered prohibits exhaustive treatment in the space of a short chapter. Our concern is not exclusively with the substantive findings in this area; but is to examine the relationship between the type of methodology employed, and the type of theoretical explanation offered.

As such it will suffice to concentrate on one aspect of the problem which is representative in terms of the methodologies employed, and give a very brief indication of the generally accepted findings in the field. More detailed discussion is left to the theoretical section.

Of all the variables which have been studied in relation to schizophrenia, those associated with social class have yielded the most, seemingly clear cut and provocative results. That is not to say that they have provided accurate data to furnish adequate etiological explanations; but that the data which in general are consistent across studies, are exceedingly difficult to interpret in terms of etiological explanations.

To make matters more interesting, there is a small minority of studies which do not conform to the expected pattern and present contradictory results to the general findings. These studies are generally the better controlled, which have been conducted in an effort to decide the validity of competing theories. It is these very contradictions which suggest avenues of etiological explanation not yet attempted by the epidemiological studies.

Although predated by the Nolan study (1917) it would be fair to argue that the majority of recent research in epidemiology has been stimulated by the problems raised by Faris and Dunham's (1939) study of the distribution of reported cases of schizophrenia in Chicago. Relying on ecological data they revealed that the highest rate for schizophrenia was found in the central areas of the city,

with diminishing rates as one moved towards the periphery of the city. These central areas were characterised by large numbers of single unit dwellings, and in general were the poorer areas of the town. Clark (1949) reanalysed their data in terms of the occupational distribution of schizophrenia and established an inverse relationship between socio-economic class and first admissions for schizophrenia, the highest rate being for the lowest socio-economic class, with diminishing rates as one moves up the socio-economic ladder.^{1.}

These findings have been replicated in study after study, with substantially supportive findings for Providence, R.I. (Faris & Dunham, 1939); Peoria, Ill., Kansas City, St. Louis, Milwaukee (Schroeder, 1942); Rochester, N.Y. (Gardner & Babigan, 1966); and Bristol, England (Hare, 1956a, 1956b).^{2.}

Despite the considerable criticisms which have been made of the inferences which have been drawn from the type of data collected, the high rate for the lower class areas, and areas characterised by single unit dwellings, and among those having lower socio-economic status has been 'confirmed' over and over again.

However, there are some important exceptions to the general drift of the findings. Principal amongst these is Clausen and Kohn's (1959) study of Hagerstown. In a well controlled study (controls were selected on the basis of their names appearing adjacent on a school register to those of people later identified as having the 'illness') they found that there was no discernible relationship between socio-economic status, or ecological area and the rates

for illness. While this is not sufficient in itself to discount the great weight of confirmatory evidence, Clausen and Kohn after a re-examination of the previous studies discovered a curious trend. The correlation between socio-economic class and reported rates was not uniform, rather, it was shown that the larger the city the stronger the correlation became. A metropolis the size of Chicago produced striking correlations between socio-economic class and rates of reported schizophrenia. In smaller cities such as Peoria (population in 1939 - 105,000), Kansas City (400,000), Milwaukee (578,000) and Omaha (214,000) the correlation was much smaller, and in Hagerstown (population 36,000) the relationship disappears. (1959:82)

This hypothesised relationship between city size and rate differentials has substantive support in the work of Sundby and Nyhus (1963) in Oslo; and Hollingshead and Redlich's (1958) data on first admissions in New Haven. In one of the few Canadian studies, Buck and others (1955) have substantiated the findings on social class, finding smaller correlations between median wage and first admissions as the community size decreases below 10,000. Similarly, in the Stirling county study (Leighton, D., 1963) the authors suggest that while the rate for the county as a whole follows the general pattern, in a community the size of Bristol (Digby, N.S.) there is no relationship between social class and mental disorder.

How is one to explain these findings? Why the preponderance of illness among those cases having low socio-economic status on admission? What is there about the area which is associated with rate differentials? And how is one to explain the curious relationship with city size?

(i) The issue of explanation

City size itself cannot provide a complete explanation of the rate differentials, there is no convincing argument in the literature, and such speculation must remain at the level of hypothesis until the specific factors associated with city size can be identified. Clausen and Kohn have proffered an explanation in terms of social cohesion; but this was purely speculative, and explanations of this sort can have no validity when made on the basis of the bald statistics alone, unless the dimensions of social cohesion can be defined and identified, and the manner in which they operate on the rate to effect the differentials specified.^{3.} Nor can city size offer any insights into an etiological explanation of schizophrenia.

One might appeal to the confusion of the city dweller that Simmel so aptly describes in his Metropolis and Mental Life. This profuse world of words and images, of objects and sensations which cannot be ordered, controlled, or organised corresponds to the stereotypical schizophrenic confusion; but until one can specify the dynamics of the factors associated with the city size, such an explanation must remain adduced, as speculation, and not stand as a complete explanation. We do not know why this should be so, nor why it is the schizophrenic who is unable to deal with these problems when so many others are able.^{4.}

While there is considerable agreement on the main lines of the social and ecological distribution of schizophrenia in the population, there is, unfortunately considerably less agreement

about why this should be so. Several sociological, and socio-psychological explanations have been advanced; but none has met with widespread recognition, and it still remains a mystery just how the socio-economic status of an individual affects his health, in much the same way that it is a mystery why city size should be associated with differences in rate.

One source of this confusion is the type of 'top down' explanation the epidemiologists have engaged in. They have tended to rely on data concerning the aggregate characteristics of individuals and areas and have attempted to infer from these models of individual functioning and breakdown. However, in order to substantiate any thesis like that of social cohesion data on the level of the individual are essential.

It is worth pursuing this point in more detail. Consequently, before examining some of the various theoretical explanations of the data, it will be profitable to examine the type of methodological criticisms that can be made to explain the statistical relationships which have been found.

b/ Methodological problems

It is extremely easy to confuse epidemiological research with ecological theory, and as we examine the theories this research has stimulated it will be readily apparent why this confusion exists. There are a considerable number of excellent critical reviews of the epidemiological literature on schizophrenia (for example see Dunham, 1961, Hoch & Zubin, 1961; Hollingshead, 1961; Plunket & Gordon, 1960) however not all are in agreement as to what

constitutes the scope of epidemiology. Mishler and Scotch (1965:285) have criticised the epidemiologists for their concentration on macro-sociological variables: social class; urban area; migration; etcetera, in explaining the distribution and etiology of schizophrenia. This limitation may be considered as stemming from a confusion as to the nature of epidemiological research.

Dunham (1966) has made the useful distinction between ecology as a field of study, concerned with the manner in which man's interactions with his social and physical environment affect his health, (in this case mental health); and epidemiology as a method of study examining how the rate of illness varies with the strength of environmental factors. In his reply to Clausen & Kohn (1954) he outlined some of the main assumptions of ecological theory:

1. That human communities have a certain organic character in that they expand, change and decline with the probability that this process will be repeated. This cycle constitutes a dynamic equilibrium.
2. That in this expansion a process of distribution takes place which sorts and relocates individuals and groups by residence and occupation over a given land area. In the ecological theory this expansion is a function of competition, and it has been demonstrated that certain conscious motives operate in the relocation of persons.
3. That this selective process creates "natural areas" which develop their own characteristics and can be delimited.
4. That each area with its particular characteristics leaves its cultural "stamp" upon the people who reside there and affects them in numerous and diverse ways.

5. That this cultural stamp will be registered in each area by frequencies of numerous types of both acceptable and unacceptable behaviour which will differ according to the character of the area.

(1954:149-150)

By contrast, the primary purpose of epidemiology is to establish whether there is an association between rate and strength and on the basis of the isolated relationship to state the direction of the relationship in causal terms. It follows that within this purview, the scope of epidemiology need not be limited to the field of ecology. The only characteristic required of populations studied is that they be alike in all respects except the factor under consideration. (We might draw an analogy with the controlled experiment.)

Consequently, epidemiological research need not limit itself to investigation and explanation solely in terms of macro-sociological variables. This, unfortunately, has been too often the case. A major criticism made by Mishler & Scotch (1965:285) in their critique, is that whereas with almost any other problem, the establishment of a relationship between social class, or urban area and the dependent variable would be the starting point of a more intensive investigation of exactly what factors in the social and physical environment are associated with the rate differentials: "in the investigation of the etiology of schizophrenia the relationship often stands for the complete analysis". Accordingly, etiological explanations on the basis of the data collected at the macro-sociological level are extremely vacuous. Excluding the

community studies, the type of information collected usually from hospital records of diagnosis, residence and occupation, does not provide any information about the interpersonal environment, or the experience of the identified case. The researcher is left to supplement his data with either an implicit psychological model of man, or the disease process, or a theory of schizophrenic causation⁵ adduced from another source, and not derived directly from his data.

Thus evidence that high rates were associated with areas of the city characterised by single unit dwellings fostered the etiological conclusion that schizophrenia was caused by social isolation (for example, Jaco, 1965). It took anomalies in the data reported from other studies (Clausen & Kohn, 1959), which contradicted the original findings, to stimulate the researchers to ask whether the high representation of cases in these areas was not so much a function of the etiological significance of social isolation; but of the identified case's desire to escape from stressful interpersonal relations, which led him to choose areas of the city in which he could be alone. (Characteristically no one thought to ask the case why he in fact moved to these areas.)

This is not to say that epidemiological research should ignore the macro-sociological factors; but that a complete explanation of etiology must take account of the interpersonal environment of the identified case. This will become apparent if we examine more closely the functions of epidemiological research.

(i) Functions of Epidemiology

As mentioned above, MacMahon et al (1960) consider the importance of epidemiology in the estimation of the determinants and distribution of disease prevalence. Thus the two main tasks of sociology in epidemiological research are first, estimating if significant rate differentials exist between different areas or social structures; and second, providing adequate explanations of those rate differentials.

The accurate assessment of such rate differentials is extremely important in the estimation of the need for psychiatric facilities; determining the population at risk (from which it may be possible to identify factors associated with the onset of the disorder) and estimating the demographic distribution of cases. Within this frame of reference, it is not of paramount importance to determine the reasons for the rate differentials. On purely pragmatic grounds, accurate knowledge about rates has important implications for the training of personnel, provision and location of new facilities, estimating the cost (and effectiveness of such facilities) and in terms of the contemporary emphasis on treatment in the community (see Pasamanick, 1967) locating the type of facility and therapy available to the type of disorder. (See Hany, 1970; for an example of the use of this approach).

The first task of specifying the determinants of the distribution involves adequate explanation of the rate differentials. (Dunham, 1965:8). This latter function can be interpreted on two

levels, and it is suggested that a confusion of these two levels is responsible for much of the inadequacy in the epidemiological research and explanation.

On one level, the ecological, the goal is an explanation of the rate differential (if any) of the identified disorder in different groups, classes, or geographical areas. Such explanations need not, and more accurately should not, involve etiological explanation of why particular individuals contract, or develop a particular disorder. If the sociologist operates on an ecological level, then in order to explain the rate differentials in his findings he must emphasise those processes within the environment of the individuals studied, and attempt to show which variables on the level of the social system are associated with the rate differentials. An example of such an explanation would be Dunham's (1965) interpretation of the difference in incidence rates between two groups of census tracts in Detroit in terms of the mobility of people into those tracts. This would explain why some areas have higher rates than others but would not provide an etiological explanation (and is not offered as such).^{6.}

As yet, sociologists have not been particularly successful in deriving such 'theories' at a social system level. Dunham, has outlined the various hypotheses which come under the rubric of social selection:

- (1) that certain persons because of personality inadequacies or proneness to mental disease have a tendency to drift into certain social classes.

- (2) the visibility of and tolerance for mental disorder vary with the attitudinal structure of different types of community.
- (3) that certain persons because of their psychic needs to break social ties, tend to select and segregate themselves in areas, cultural or spatial, marked by anonymity.
- (4) that as the size of the city decreases, rate differentials between areas decrease.

(1965:8)

It is significant that none of these hypotheses make statements about what causes the disorder, or what factors in the physical and social environment are associated with high rates of disorder. The concern is in explaining why one area should have a higher rate than another. If an attempt is made to explain why particular individuals become mentally ill, and to isolate the factors associated with an individual's presentation, and the diagnosis, of psychiatric symptoms, then one moves to a second level of explanation of the determinants.

Here the concern is no longer with what causes the rate of reported cases and their distribution; but, more fundamentally what causes an individual to exhibit the symptoms which will be diagnosed as the disease. Such etiological statements must incorporate some assumptions about the socio-psychological functioning of the individual. If the sociologist attempts to make statements about the social factors which cause the illness in a particular person, then he moves to a different level of explanation than the ecological which can only indicate factors which may be significant.

For example, it is one thing to establish a preponderance of cases originating in the lower socio-economic classes, and quite another to attribute that distribution to lower-class 'way of life' on the basis of data gleaned from hospital records. Questions about the strength of certain social or cultural factors which predispose individuals to, or precipitate, the 'illness' are better asked on a socio-psychological level at which the behaviour and experiences of individuals are examined.

The attempt to make statements on a socio-psychological level on the basis of data on rates of illness and characteristics of areas is what Dunham (1961) has termed the 'fallacy of the wrong level'. It will be treated here as an aspect of a more general problem of missing data.

(ii) The fallacy of the wrong level

The fallacy of the wrong level, may be considered as an aspect of a much wider problem of aggregation and disaggregation.

That is, the general problem of inferring relationships on one level from data collected at another level. In the epidemiological studies, excluding the community studies like those of Srole et al. (1962); Leighton, D.C. et al. (1963); Krupinski et al. (1967); these data have consisted of rates of reported disorder usually measured by the incidence of cases to state mental hospitals, and/or private hospitals and/or private practitioners; with the demographic data being drawn from incomplete hospital records, or more usually features of the environment from which the cases originated. Typically one is working with a reported rate of diagnosed illness of more or less completeness (depending on the range of facilities

covered) and certain data about the social class characteristics, type of housing, or ethnic composition of the areas from which the cases derive. It is a simple matter, on the basis of elementary correlational techniques to associate the rate with the particular area and draw conclusions about the relationship between the, say, lower class 'way of life' in a particular area and risk or susceptibility to mental disorder. (See Ikeda, 1962; for a perfect example of this sort of reasoning.)

There is nothing essentially wrong with collecting data on this level. Indeed, it may be crucial in identifying populations of high risk as a basis for more intensive study. It is, however, illegitimate to use it as a basis for statements about the individual characteristics of the cases, or to make causal statements about the etiology of the disorder on the basis of the inferred individual characteristics. The reason is very simple and rests with what Robinson (1950) has termed the ^{8.} 'ecological fallacy'.

Readers interested in the statistical complexities of Robinson's argument are referred to the original publication. Here a simple example will suffice to illuminate the issue: If we plot the relationship between madness and socio-economic class in a 2 x 2 contingency table we might arrive at this arrangement:

| | Mad | Sane | |
|-------------|-----|------|---|
| Upper Class | a | b | X |
| Lower Class | c | d | Y |
| | W | Z | |

where the Pearsonian fourfold correlation depends on the within class individual correlations, while the ecological correlation depends upon the marginal frequencies of the within class correlations. (see Robinson, 1950; Goodman, 1953) To state the obvious, the marginal frequencies of the contingency table do not determine the internal frequencies -- that is, there are a large number of internal frequencies which will satisfy exactly the same marginal frequencies for any 2×2 table. Consequently there are a large number of individual correlations which might correspond to any ecological correlation. Which stated simply means that there need be no correspondence whatsoever between the individual correlation and the ecological correlation. The implication of this is that one cannot assume that because a person is admitted to hospital from an area of single unit dwellings he necessarily suffers from a pathogenic 'social isolation' which has etiological significance in the development of the disorder. This type of explanation requires data of a different order -- specifically on the interpersonal experience of the diagnosed case prior to his admission which is not derivable from data on rates of illness and characteristics of areas.

On a more general level, if we treat the ecological fallacy as suggested as an instance of the wider problem of aggregation and disaggregation the work of Blalock provides us with a useful explanation of the city size hypothesis. Blalock argues that 'in shifting from one unit of analysis to another we are likely to affect the manner

in which outside and possibly disturbing influences are operating on the dependent and independent variables under consideration.'

(1964:98)

In the case of city size and schizophrenia what this means is that prior to formulating grand theories linking a hypothetical increase in anomie or what have you with an increase in city size and explaining the relationship between city size and schizophrenia in terms of this increase in anomie we must first establish whether or not the relationship is a statistical artifact. As Blalock argues, the key to this problem may be changes in the degree to which other unknown or unmeasured variables are affecting the rate as we change units of analysis. By using the larger unit of the city we may be controlling for certain types of disturbing influences which affect the distribution in the smaller units.

For example, the potential schizophrenic in the small community may be 'saved' from breakdown because he happens to meet a clergyman who has the solution to his problems. These chances or "idiosyncratic" (1964:99) variables may significantly affect the rate in a small area or town; but may cancel each other out as we move to the larger units of analysis, presenting us with a more stable rate or accentuating different features than in the smaller units. By taking the larger unit, then, we tend to iron out the individual differences which in the case of schizophrenia may be so important in explaining why some people do, while others do not, appear as case statistics. The explanation of the differentials may be made then on pure probability grounds and

owe nothing to any inherent effects of city size.

The most obvious question to ask at this juncture is why bother using ecological correlations as a basis for generalisation (particularly etiological generalisation)? The simplest answer to this question is in terms of the problem of missing data.

(iii) Problem of missing data

It would be something of a miracle in any field if it were possible to obtain exactly that data which would allow one to generate an inclusive theory, or to decide between competing explanations of the same phenomena. The epidemiological field is no different from any other.

There are a variety of situations in which the missing data problem is likely to arise. For example, we may have data for a range of units or areas, but have no way of disaggregating, or identifying the community contexts of their behaviour. That is, there may be no way to reallocate individuals to any known primary sampling area. An example is the use of hospital records in epidemiological research in which for reasons of secrecy or, more usually, poor information we may not be able to identify individuals and their social contexts although we may have available general socio-economic class data and initial diagnosis.

A second situation in which the problem arises is that in which we have no individual data; but aggregate data are available for territorial units at different levels. The major source of such data would be official statistics in which the primary individual data are kept secret from the outset, or cannot be made

available for administrative or economic reasons. So, while we may know how many people become ill from census tract 15 the only data we have available are this rate and certain characteristics of this tract. While it would be preferable to match each individual incident of disorder with individual characteristics, the best we can do is compare the rate in this tract with that of others, in the hope that in the process we might unearth a variable which is significantly related to rate differentials.

There are a number of dangers with this procedure of using rates as indices of the occurrence of mental disorder. First, we accept a psychiatric diagnosis as the criterion of who is and is not mentally ill; and second, for objectivity (or simplicity?) we adopt this psychiatric model of what constitutes a case in terms of incidence measures of first admissions to private and public mental hospitals, or less frequently, diagnosed cases under private psychiatric care. Unfortunately there are a number of problems with this approach. First, there is a considerable literature on the unreliability and questionable validity of psychiatric diagnoses (Blum, 1962; Ash, 1949; Mehlman, 1952) reflected in the statement of Laing, (1970:11-12):

If anyone thinks that 'schizophrenia' is a fact, he would do well to read critically the literature on 'schizophrenia' from its inventor Bleuler to the present day. After much disbelief in the new disease more and more psychiatrists adopted the term, though few English or American psychiatrists knew what it meant...But though the term has now been generally adopted and psychiatrists trained in its application the fact it is supposed to denote remains elusive. Even two psychiatrists from the same medical school cannot agree on who is schizophrenic independently of each other more than eight out of ten times at

best; agreement is less than that between different schools, and less again between different countries. These figures are not in dispute. But when psychiatrists dispute the diagnosis there is no court of appeal. There are at present no objective, reliable, quantifiable criteria -- behavioural or neurophysiological or biochemical -- to appeal to when the psychiatrists differ.

(emphasis added)

Second, it tends to ignore the nosocomial factors (that is factors influencing the availability of beds, the willingness of patients to be admitted and differences in admitting policies between hospitals,) which may produce differential rates. For example Bickford (1967) takes a rather commonsensical (obvious?) stance on the epidemic of senile psychosis which hit Britain in the 1950's:

The increase in mental illness is directly due to the passage of the National Health Act in 1948, when the government of the day in effect created an entirely new mental illness. The decision was made to turn workhouses into general hospitals. In order to do this the previous occupants of the workhouses (tramps and old people) had to be got rid of. Tramps are now, of course, an eyesore and largely settled in London, but the old people could not be so dispersed. They had to go into mental hospitals because there was nowhere else for them. In order for them to do this they had to suffer from a mental illness, and the small number of people who were old and had to go into mental hospitals became a flood, and senile psychoses assumed epidemic proportions.

While this may seem a facetious (though tragic) example, Terris (1965) has explained the decline in admissions for manic-depressive psychoses in New York State Hospitals (1930-1950) in similar terms, as mainly due to changes in diagnostic criteria.

A third factor which may contribute to the problem of missing data is that the required data are seldom complete and are often in the wrong form or order to provide information on the interpersonal environment from which the cases originate. (Mishler and Scotch, 1965; for example, see Gerard & Houston, 1953, who were unable to

secure life history data for more than half their sample.) If such data are desired, and it is argued that a complete analysis of the phenomena requires such data, then one cannot rely solely on the data drawn exclusively from hospital records.^{9.}

Fourth, the use of hospital data suffers from a further deficiency in that it reports only those cases which have come to the attention of psychiatric facilities. While such data may be of considerable importance in the epidemiology of 'treated cases' it must be of limited applicability in a discussion of the etiology of the particular disorder. Scheff, has estimated that the ratio of treated to untreated cases in the United States may be as high as 1:14 (1966:49) which means that for every treated case there are at least fourteen at large in the community which have not come to the attention of a psychiatrist.

This would not in itself invalidate the findings from the hospital studies if it could be shown that the factors associated with commitment or non-commitment were not themselves associated with the type of diagnosis made, or the decision to commit itself. Unfortunately, this seems not to be the case. Both the incidence of disorders, and the decision to commit with what diagnosis have been shown to be influenced by factors which are not directly related to the psychiatric status of the patient. (See, Hollingshead & Redlich, 1958; Hughes & Marshall, 1971; Waxler & Mishler, 1963; Lieberman, 1965) Consequently, it is not possible to make accurate statements or even suggestions about etiology.

A variety of strategies have been employed to overcome this problem. In recent years, at least in Britain, there has been a considerable effort to improve hospital reporting procedures, specifically the collection of data which may be pertinent for etiological statements. (See Hill et al., 1962) (Although such efforts are only as good as the willingness of the admitting staff to co-operate; and, if we follow Garfinkel (1967) the willingness of the staff to make their behaviour 'accountable' to the researcher by making explicit, the taken for granted assumptions which underly their decisions to commit.)

The use of prevalence measures (that is the number of persons who can be judged to be ill at a particular time; as opposed to the number of new cases occurring) goes some way to avoiding some of these problems. The researchers involved in the community studies have attempted to derive some objective measure of Mental Disorder, usually in the form of a questionnaire which is administered by interviewers with varying degrees of psychiatric training. (Srole et al., 1962; D. Leighton et al., 1963; Manis et al., 1963; Manis et al., 1964) Such questionnaires take the form of a 'supermarket' of symptoms from which the interviewee selects. The questionnaires are validated against criterion groups of 'normal' individuals and psychiatric inmates and in the 'better' studies the identified cases are interviewed by a team psychiatrist. (See however A. H. Leighton, 1959; for a discussion of the difficulties in diagnosing the 'non-patient groups' in the Srole et al. (1962) study.) However most of the community studies have tended to lump together a variety of different diagnostic categories because of

the difficulties in sampling sufficient numbers to facilitate statistical testing; consequently it is difficult to isolate the rate for a particular disease. (Mishler & Scotch, 1963:279; Manis et al., 1963.) In some cases traditional diagnostic categories have not been used which again poses the problem of estimating particular disease rates, with the attendant consequences for etiological explanations. (See Srole et al., 1962; in which level of impairment was used instead of specific diagnostic categories.)

The findings of these incidence studies pose us with some extremely perplexing problems. The problem of total prevalence in relation to the hospital incidence studies was mentioned above. In Srole's study in Midtown, Manhattan twenty-four percent of the sample was rated as disturbed to a 'marked', 'serious' or 'extreme' degree. As we see in Table 1, the findings of the D. Leighton Sterling County Study speak for themselves. The team psychiatrists termed those most in need of psychiatric care "most abnormal". In types 2 and 3 psychiatric treatment was thought to be advisable but not mandatory. Individuals most in need of psychiatric attention tended to have more symptoms and more different kinds of symptoms than those less in need of attention. They also tended to be more impaired by their symptoms. (1963:169) The Leightons conclude that if the population of Stirling county were studied intensively by "competent" psychiatrists, approximately two-thirds would be found to have been suffering, at some time during their lives, from

TABLE 1

| | Bristol Health Score | Family Life Score |
|---|----------------------|-------------------|
| Type 1. Most abnormal | 1 % | 3 % |
| Type 2. Psych. disorder significant impairment | 38 % | 17 % |
| Type 3. Probable Psych. disorder | 36 % | 37 % |
| Type 4. Doubtful | 14 % | 26 % |
| Type 5. Probably well | 11 % | 17 % |

(Source, D. Leighton, 1963:139: The differences in the two columns are attributed to the greater amount of data available for the Family life study. Leighton believes the Family Life data to be more stable because it was based on 1010 respondents as opposed to the 140 of the Bristol Health score. However the Bristol score was believed to be more accurate because of greater information obtained in the protocols. Bristol is the name given by the authors to Digby, N.S. and is not to be confused with Bristol, England!)

a psychiatric disorder ('for the most part low grade and chronic') according to the criteria of the American Psychiatric Association Diagnostic Manual. (1963:355)

These findings raise a number of important issues, which we will discuss in more detail below. But for the moment we are faced with the problem of exactly what criteria are adopted for a judgement of who is and who is not mentally ill, particularly, as it seems clear from the community studies, when the individual himself does not consider himself to be mentally ill, and nor, apparently do the members of his community. This problem becomes of increasing importance when we consider psychiatric symptoms as adaptive responses to the environment, (as do Leighton, et al., 1963:357, 14) which serve to relieve tension, rather than as maladaptive responses which indicate an underlying illness. While Leighton et al reach a similar conclusion; we differ somewhat with their emphasis. They argue that in terms of their findings:

it means that a very large number of people, however efficient they may be in earning a living, keeping house and in conducting the round of daily activities dictated by their culture, are nevertheless unhappy and discomforted by these symptoms, or are a source of difficulty to others, or both.

(1963:357)

In terms of the large number of people involved it would seem that the type of behaviour uncovered by the questionnaires, were very normal responses to stressful situations. If this is the case, then it leads one to certain important questions about why only certain people seek psychiatric care, while others, (if the results

of these various studies are accurate; see also Manis et al., 1964) with equally strong reasons do not. Since this line of reasoning anticipates items which will be discussed more fully below, it would be best to postpone discussion until later. However, it will suffice to raise the problem of the correspondence between a sociological definition of illness, and a medical definition.

(iv) Identification of a case

As we have seen, the epidemiological literature can be divided into two distinct areas on the basis of its classification of case material. On the one hand are those studies dealing with treated disorders, or identified cases -- studies using hospital records and/or data from private practitioners -- on the other hand are the community studies, which intentionally pay little attention to treated disorders (see Srole et al., 1962:350-353), and concentrate on determining some index of global adjustment, or assessing the true prevalence of disorder in a specific community (that is their total social and physical environment).

Given the two distinct areas of study, the problem which immediately presents itself, is to what do the different definitions of a 'case' correspond? Are the researchers in the two areas all talking about the same thing, or do their 'cases' refer to different phenomena? Do these differences have any implications for etiological statements?

One way of distinguishing between the two groups of studies is that of Roman and Trice (1967:5) who argue that the 'patient' studies adopt a societal definition of mental illness, for they

appear to consider psychiatric labelling by a societal functionary to be an implicit dimension of the condition mental disorder.

This in contrast to the community studies which, according to the same authors, employ the medical concept of mental disorder; proceeding on the assumption

that the condition of psychological pathology may exist in the community without coming to the attention of psychiatric functionaries.

(1967:8)

The use of the medical definition in the latter is explicit in their use of psychiatric diagnoses to decide on the mental status of individuals on the basis of data recorded in interview protocols (while the interview is not normally conducted by a psychiatrist, and he may never see the person he diagnoses as mentally ill).

Even allowing for gross inaccuracy in the estimation of the figures for total prevalence reported above, it is arguable, in statistical terms, that treated cases constitute a potentially biased sample on which to make statements of etiological significance. Unless, that is, controls are employed for the factors which influence psychiatric referrals by self or others. (A condition which is, of course dependent upon successful identification of those factors.) As a corollary, the reverse is also true in that it is inaccurate to make statements of etiological significance on the non-treated samples without considering those same factors which influence decisions to commit.

Identification of the relevant factors has not been done in those studies using identified cases as data; which brings into

question Roman and Trice's classification of these studies as using a societal definition of illness, or, at least the implication that the individual researchers were aware of the implications of this interpretation of their data while conducting their studies.

The interactionist conception of mental illness will be discussed in more detail below, but it is sufficient to ask at this juncture what correspondence there is between the cases used in epidemiological research, and the phenomena sociologists seek to explain. This is extremely important in assessing the validity of etiological statements of the rate differentials. If hospitalised, or treated, individuals are accepted as cases, and it is recognised that there are individuals in the community with symptomatology as severe as those treated, then it is argued that the proper focus of study, before any attempt is made at etiological explanation is:

- (a) accurate assessment of the rate differentials of treated disorder;
- (b) estimation of the distribution of disorders in various treatment facilities;
- (c) estimation of the rate of non-treated cases; and their distribution;
- (d) explanation of rate distribution within and between facilities on a number of independent variables;
- (e) assessment of the characteristics of the non-treatment cases and their similarities or differences to the treatment group;

(f) examination of the factors associated with non-treatment;
and finally

(g) having identified the relevant population, description and explanation of the various factors associated with the development of a particular disorder.

We must know accurately to which populations statements of etiological significance refer.

If we examine the epidemiological 'patient' studies chronologically we find a progressive concern with the representativeness of case finding techniques, evinced in a concern with the representativeness of the sampling procedures. The three fold classification of patient studies, outlined above, correspond to an almost chronological concern with inclusiveness. The earlier conclusions of epidemiological research that highest rates for schizophrenia occur in the lowest socio-economic areas has been tempered by considerations of the representativeness of the samples studied. The work of Nolan (1917), Odegaard (1932), Frumkin (1955), are questionable for their reliance on data from state mental hospitals alone, which may not be representative of the distribution in the population as a whole. While these studies may be legitimate in determining the distribution of treated cases, it is questionable whether they can ever provide a solution to the etiology of the disorder -- unless, that is, schizophrenia, or mental illness is defined exclusively in terms of those receiving a psychiatric diagnosis. In this case, it still remains to be explained why there are people with disorders of equal severity who do not come to

psychiatric attention.

While it can be argued, as do Roman and Trice that community studies accept a medical definition of illness, it is questionable whether the authors of the 'patient' studies accept the societal definition as these authors imply. While agreeing with Roman and Trice (1967:18-21) that a sociological definition of illness must include some consideration of the societal processes involved in the labelling, or diagnosis, of a case, it is not accurate to attribute this awareness to the authors of the 'patient' studies. The 'patient' studies accept an implicit medical or psychiatric definition of mental illness, or schizophrenia, in terms of their choice of diagnosed patients as cases.

Indeed, it is this uncritical acceptance of the medical definition of what constitutes a case which has tended to obfuscate the issue of an adequate sociological approach to schizophrenia. This, particularly in their lack of concern with the processes involved in arriving at a psychiatric diagnosis. While these studies may be of crucial importance in determining the distribution of treated disorder their findings are of limited importance in an approach to etiology. Similarly, while the community studies may be crucial in estimating total prevalence they can be only of limited use in answering this question while they ignore the factors associated with commitment.

It is worth bearing these problems in mind as we move to an examination of some of the myriad theoretical explanations which have been offered.

Section Two: Theoretical Explanations

a/ A digression on causes

In the previous section we examined some of the problems involved in epidemiological research, and their implications. It is against this background that the theoretical explanations will be examined.

One problem which was not considered above; but which could be termed non-theoretical, though not strictly methodological, is that of identifying which variables are taken to be dependent, and which independent. At first glance this might seem something of a 'red-herring' -- it is perfectly obvious that schizophrenia is to be properly treated as the dependent variable. The exercise is one of explaining its distribution, and hopefully, its etiology. However the issue is not as simple as it seems, particularly in light of the earlier discussion of the scope of epidemiology. It was argued, that while the aims of the method were in explaining the distribution and determinants of disease, an explanation of the distribution is not necessarily synonymous with an etiological explanation. Distribution may be explained in terms different from such an etiological explanation. Indeed, the disease itself may be a factor in explaining the distribution.

Taking schizophrenia as the independent, or intervening variable may have profound effects on an explanation of rate differentials. As an exercise it is useful to speculate what would constitute an adequate explanation of the over-confirmed relationship between social class and schizophrenia. If this problem is approached

in total ignorance of the existing literature, we have a choice
of taking schizophrenia as either dependent or independent.^{9.}

Taking it as dependent, implies that there is something
about the lower-class life style which is conducive to high risk
for the disorder. That is:

lower class —————> illness.
life style

If we adopt this latter type of explanation we must be prepared
to explain why there are people of higher status who contract
the disorder. If the relevant variables can be identified, we may
arrive at an explanation which is relatively free of the original
class bias. For example, the etiological factor may be stress.
The lower-class environment is more 'stressful' and consequently
subjects its population to a higher risk. That is:

lower class ———> stress ———> illness.
life style

This would constitute a particularly parsimonious explanation in
that it offers an explanation of rate differentials, and goes some
way to providing an etiological explanation when the relevant
variables are identified and refined.

Alternatively, if the disorder is an independent variable,
then a completely different picture emerges. It would be possible
to argue that the schizophrenic because of his disability is unable
to hold down a steady job. He is unable, because of his thought
confusion, to perform tasks of any complexity, and consequently

finds himself in the lowest socio-economic class by virtue of his inability to maintain or achieve a higher position. That is:

illness \longrightarrow social disability \longrightarrow decline in S.E.S./
 thought disorder. or inability to achieve.

Though less inclusive than the previous explanation, it gives an explanation of the rate differentials without providing any clues as to etiology. It is an explanation of rates of a different order than a corresponding etiological explanation.

The interesting point is that while data like that of Faris and Dunham, Clark, Nolan, Jaco, and many others will support either explanation, they have all offered explanations taking schizophrenia as the dependent variable. It was not until recently, particularly after the work of Mare (1956a, 1956b.) that researchers have seriously considered the disorder as the independent variable. (See Srole et al's discussion of these problems, 1962.) This is extremely significant; the importance of epidemiology has been thought to be in isolating factors which may be of importance in questions of etiology. However, if, within the framework of these studies the variable to be explained can be treated as an independent variable in its own right, then this function is impossible to achieve.

This problem is attributable, in many cases, to a confusion of the two types of explanation involved in epidemiology, and to the confusion of explanations of rates with etiological explanations of illness. If rates are taken as the subject matter than it is perfectly feasible to make epidemiological statements about factors

associated with those rates; but these should not be confused with etiological statements about the development of the illness which are of a different order, given the restricted definition of a case. If data were available to permit etiological explanation, as with the explanation of rate differentials, there would still remain the problem of specifying the nature of the relationship, specifically, in determining the direction of the relationship. Etiological explanation would be impossible without some notion of causality, however crude. For example, in studies of the relationship between lung cancer and smoking, the establishment of a relationship precedes a statement of the causal relationship between smoking and the development of cancer. (See Doll & Hill, 1950.)

It would be a mistake to go into a long discussion of causality here, indeed the exercise would probably prove fruitless in relation to the types of criticism which will be made of the literature. While etiological explanation implies causal explanation (and concern with rates stimulates questions about the causes of those rates), it will suffice for our present purposes to take Greer's point:

Indeed in scientific theory there is really no need for the concept of causation. It is an esthetic dimension added for esthetic reasons. In operations, causality turns out to be invariant associations in time and space. Most logical analyses of causality insist on invariant associations in time -- sequence. But, relationships can frequently be specified as invariant with no attention to sequence. Which comes first, the circulation system or the state of irritability and mobility we call animal life? The question is meaningless. The statement of scientific laws does not include or require causality; it simply requires statements of invariant association.

(1969:120-121)

However, while for our purposes it will suffice to consider etiological explanations in terms of invariant association this does not necessarily imply that the association need necessarily be symmetrical. For purposes of etiological explanation we will be concerned with what Dubin has termed 'sequential' laws of interaction (1969:100) in which a "time dimension is used to order the relationship among two or more units (variables)". It should be emphasised that the statement of sequence is just that; a statement that variable A precedes B, or that B succeeds A, and is not to be interpreted as a causal statement.

As Dubin argues, sequential laws may be made more specific by the inclusion of two constraints (1969:103):

- (a) by an exclusive choice of the first appearing variable, making it the only one which is followed by the second appearing variable; e.g. "If and only if a person eats cranberry jelly will he exhibit psychotic symptoms".
- (b) by specifying that the sequence is unidirectional, that values of A will only appear before values of B (1969:104): e.g. "Presentation of psychotic symptoms always follows and never precedes the ingestion of cranberry jelly".

It is of course possible to combine these two restrictive instances into one law of interaction (1969:104): e.g. "If and only if a person eats cranberry jelly will he exhibit psychotic symptoms; and the presentation of psychotic symptoms never precedes the ingestion of cranberry jelly".

By not stating these sequential laws as causal laws we avoid the knotty philosophical problems of specifying the causal agent and lose nothing in predictive capacity or explanatory power. To Dubin's analysis we would add a further restriction that the association be sequential and nonspurious. That is, the association does not disappear when other antecedents are held constant. (Spurious correlations may themselves be useful in identifying the relevant antecedent variable, see Simon, 1957.) It would seem that this further restriction is essential in explanations of rate differentials or etiologial explanations.

While this may solve the problem of what will be accepted as an adequate explanation, there still remains the problem of inferences of associations between variables based on data which are of a different order than the relationship to be explained. As argued in the discussion of ecological correlation, there may be serious logical difficulties in developing theories which deal with collectivities and their individual units particularly if all the data are only on one level. (See Lazarsfeld & Menzel, 1961; "On the relation between individual and collective properties.") Hence, etiologial explanation cannot be made on the basis of data which takes the form of reported rates of illness and associated demographic and socio-economic data. It can only approximate by controls on the appropriate dimensions and the collection of appropriate data. If the data are limited, as they are for many of the theories to be discussed, then other

consideration must be brought to the analysis. This problem will crop up over and over again in the substantive discussion of the various theories and is apparent in the confusion as to what it is the reported rates refer.

It has not been clear what it is these rates refer to. On the one hand they may be taken as indicators of illness qua illness and used as the basis for etiological statements. On the other, they may be treated simply and solely for what they are: rates of reported illness. If they are treated as rates of reported illness then the whole exercise takes on different implications as the concern becomes not one of establishing etiology; but of isolating the factors associated with becoming a case. That is not to say that the presence or absence of disorder may not be an important factor in the decision to commit; but that this may not be sole criterion and factors other than the disorder may be involved.

b/ Ecological explanations

(i) The breeder vs the drift hypothesis

If the rates are taken as synonymous with the disease, a whole crop of problems arise. The early research in epidemiology was conducted within the framework of ecological theory. While there are still attempts to implicate certain features in the social and physical environment as increasing the risk of acquiring a disorder, within the framework of ecological theory

it was assumed that there was a direct correspondence between the characteristics of areas, and the rates of particular diseases. Despite Dunham's rejoinder (1954) to Clausen and Kohn (1954) it would be fair to argue that in his work with Faris, this sort of assumption lead to their postulating social isolation as quite a plausible explanation of the high rates for the rooming house districts of Chicago. Single unit dwellings are characteristic of these areas, and it is a small step from this to arguing that schizophrenics or potential schizophrenics live alone and that this factor has etiological significance. There should be no need to re-iterate here Robinson's criticism that because an individual comes from an area having certain aggregate characteristics, there is no reason to assume that he shares those characteristics.

It was this sort of reasoning from aggregate to individual data which prompted critics to offer alternative explanations to Faris and Dunham's findings. Suppose that those people who eventually become identified as cases had merely drifted into those areas a short time before their commitment and had not, after all, developed the illness in response to factors operating in those areas? That is, what happens if we treat the disorder as the independent variable? We are able to explain the differential rate; but no longer able to offer an etiological explanation.

Such an explanation of Faris and Dunham's findings was offered by Myerson (1940:995-997) and Mary Bess Owen (1941).

Here the argument was that during the course of the development of his illness the schizophrenic 'drifted' into the rooming house districts of the city. The higher rate differentials were to be explained not by factors operating in these environments; but by the movement of cases into these areas.

Unfortunately it was not possible to test this hypothesis on Faris and Dunham's original findings, for while they had data on the last residence of cases before admission to the Chicago hospitals, the records they used did not contain information about the length of stay at this residence, nor from which areas the cases had originally moved (that is, if they had moved prior to admission).

(ii) Drift and social class

The "drift" idea is easily extended to the studies which associated high rates with lower social class, and not solely poorer ecological areas. One explanation of Clark's (1949) results is that the lower class way of life has schizogenic properties which results in a higher risk for those with low status occupations. In similar terms to the explanation of a real distribution we could argue that the higher rates for the poorer classes are a consequence of the tendency of individuals in higher positions to decline in social position as a consequence of their illness, moving into the occupations associated with lower class position. The drift of people into the lower status occupations would tend to exaggerate the rates for those occupations, and result

in a corresponding understatement of the rate of the class from which they originate. Again, it is not possible to interpret the original findings in these terms because of the lack of adequate (or appropriate) information.

One specific criticism of this type of hypothesis is the type of image of the schizophrenic which emerges. Drift is only one variety of the social selection hypotheses which regards "society" as selecting out its weaker members by some, as yet, unspecified process. The drift hypothesis conjures up a picture of the schizophrenic as

a highly incoherent, bizarre, confused and impulsive person and that in no social situation does he know what he is doing or why he is doing it. He is seen as a very abnormal personality whose overtures are being rebuffed and rejected at every turn, repudiated by his family and permitted to drift almost in a haphazard fashion until he gets into an area where his disturbance becomes too obvious for acceptance and he is sent into the public mental hospital.

(Dunham, 1965)

As Dunham has argued, the hypothesis does not account for the behaviour of the schizophrenic in the pre-psychotic period -- which behaviour may have profound implications for his social characteristics when he is identified as a case. While it may be possible to describe the 'full-blown' psychotic in the manner outlined above, to deny the possibility of choice and voluntary action to the pre-psychotic is clearly inadequate. If we leave aside for the moment the knotty problem of the meaningfulness, or voluntary nature of the psychotics' or pre-psychotics' action it would seem possible to resolve the

question of the social and geographical mobility of the identified cases, and thus the issue of drift, by examining their histories of geographical and social mobility.

Unfortunately, there is inadequate information in the early studies to substantiate either viewpoint and as late as 1966 Dohrenwend was still calling for studies to assess the validity of social causation as opposed to drift as explanatory hypotheses. (see also Hare, 1969)

(iii) Drift and social mobility

Later studies have been more specifically concerned with the issue of social mobility per se as a causative factor. Unfortunately, the evidence is, to say the least, inconclusive.

If the concern is with mobility, then two problems immediately present themselves. First, it is extremely important to determine the onset of the disorder; second, it is difficult to derive an adequate index of mobility which is independent of the disease process.

To take the first problem, in order to make statements about the antecedent conditions associated with the onset of the disorder (a prerequisite for making statements of causality, or the direction of the relationship) it is important to specify the period in which the disorder first appeared. This would not be so severe a problem if the model of etiology espoused traces its source to some childhood experiences. In this case it would still be necessary to discuss the nature of that experience; but it could be assumed that the

social and environmental factors associated with those experiences could be identified in terms of the class membership of the parents, and their area of residence (See Myers & Roberts, 1959). However, if the etiological model is phrased in terms of present stresses, that is, if there are factors in the immediate environment of the pre-psychotic which precipitate the onset of the disorder (Hare, 1969:10), (and these stresses may work on a predisposition to the disorder engendered in childhood experience) then it is necessary to be able to specify the initial onset.

This is virtually impossible in those studies which rely on data from hospital records for the identification of their cases; and again, the problem of what it is the records represent arises. If the rates are taken as being true indicators of the disease, then there would be no real reason why the date of commitment should not serve as a reasonably accurate approximation of the date of onset: that is, if commitment could be shown to be dependent on the presence of the disorder, and not influenced by other condoning factors. This unfortunately is not the case. Hollingshead and Redlich found that there may be considerable class differences in the speed in which cases come to the attention of psychiatric functionaries, in the type of facilities entered, and the type of diagnosis made. (1958) It would thus be difficult to legitimate statements about etiological factors associated with social class, if it could be shown that the possibility of becoming a case with a particular diagnosis depended as much (if not more) on class as on the affliction of the illness itself.

If the rates are treated for what they are, as rates of illness, then these limitations become legitimate problems in their own right, the task becoming one of specifying the factors associated with commitment, as a prelude to the identification, and definition of what in fact the disorder is.

Unless the onset of the disorder can be specified then the second problem of indexing mobility is merely academic. It is possible to examine factors associated with commitment without specifying the onset of the disorder. The same is true if the problem is treated in terms of past stresses (the important factors are then the parental characteristics). If however the explanation is in terms of present stresses, it is essential to specify the onset, especially in a hypothesis like that of drift.

There would be little point in attempting to isolate etiological factors associated with a person's status with the presence or absence of disorder if it were impossible to establish a measure of status which was independent of the disease process. Similarly, it would not be accurate to attempt to associate mobility with the disorder if it could not be shown that this mobility was independent of the disorder.

Mobility may be analysed either within a person's own lifetime, or between generations. In the former instance of intra-generational mobility it is necessary to establish a person's occupation at two distinct points in time and establish whether or not there has been a significant change in status. The evidence on this point is inconsistent. Some studies find downward mobility

associated with high rates (Schwartz, 1946; Lystad, 1957) others that upward mobility is the significant association (Hollingshead, Ellis & Kirby, 1954) and still others that there is no significant association (Hollingshead & Redlich, 1958; Clausen & Kohn, 1959; Gerard & Houston, 1953; Lapouse, Monk & Terris, 1956). It is, as always, difficult to know which of these conclusions to accept. Some of the studies do not use control groups (see Lystad, 1957) and fail to compare the experiences of the identified cases with that of a 'normal population', others use inappropriate controls, or like Clausen and Kohn, (whose study is impressively designed and controlled) choose a city which has an inadequate concentration of schizophrenics in the lowest socio-economic class. On the balance of the evidence of the intragenerational studies there is little to suggest that schizophrenics have been more upwardly or downwardly mobile than their 'normal' contemporaries, or that mobility per se is a sufficient explanation of the concentration of schizophrenics in the lowest socio-economic class.

An alternative approach is to compare the status of the schizophrenic at the time of the onset of the illness (more accurately at the time of his entry into an institution) with the social class of his parents. The comparison provides us with an index of the patient's mobility relative to his original social-class of which parent's social-class is assumed to be a measure. By using this measure the influence that the disorder may have on mobility is explicitly recognised. Srole's rationale for using this sort of

index is that it overcomes some of the problems of the synchronic studies which focus on the status of an individual at a single point in time and thus " (1) only provide correlations between concurrent variables (and) (2) if a correlation emerges, they provide no firm basis for identifying the antecedent and consequent" (1962:25, f.n. 42). It was the hope of the Midtown researchers that by concentrating on the demographic parental factors they would be able to convert their study from synchronic to longitudinal, and because of the choice of parental S.E.S. as the independent variable, 'to avoid the possible confounding influence of the disease on the cases' S.E.S..

If fathers of schizophrenics are concentrated in the lowest socio-economic class, then this would be fairly conclusive proof of a relationship between lower class origin and the risk of disorder. (Thus avoiding the problem of having to specify the onset of the disorder.) This would not of course be the complete solution. There would still be the question of exactly which features of the lower class life-style were significantly associated. As with the studies on the patient's own social class, the results are equivocal. The data from the Midtown Study suggests that the parent's social class correlates almost as well with the rates for mental illness as with the subject's own social-class. (Srole et al, 1962:212-222)

However, in an extremely well controlled study in Bristol, England, Goldberg and Morrison (1963) found that if patients were regarded as having the same social class as their fathers, then the

rate for schizophrenia is the same for all social classes.

The major problem with the study is the high number of cases for whom no data are available on social class (25%), however, their findings have received substantive support in the work of Dunham (1965, 1966) and Morris (1959) who found an excess of schizophrenics in Class V when classified by their occupation at commitment; but whose fathers were distributed evenly over the five social classes used.

Likewise in the Goldberg and Morrison study, schizophrenics were found to be downwardly mobile with respect to their fathers' social class, and tended to be overly represented in Class V because of their inability to secure occupations commensurate with their abilities. Close examination of the data presented, on the education of the identified cases, is illuminating. The cases showed no apparent difficulties with their school work, some having considerable academic achievements; but characteristically, their occupational history was one of progressive failure. It is significant that a similar sort of finding was reached in a much more detailed study of families in Puerto Rico by Rogler and Hollingshead. (1965) In their book Trapped the authors report the results of an intensive study of three generations of slum families living in San Juan. The study consisted of intensive interviews with families with either one or both spouses certified as schizophrenic by one of the team psychiatrists compared with families acknowledged, after examination, to be mentally well. The findings indicate that the adolescent and childhood experience of the future schizophrenics was

no different than that of the normals, and that the disorder manifested itself on entry into the job market.

That this intergenerational mobility does not provide the complete answer is suggested by Turner and Wagonfeld (1967) in their data from Rochester, New York. While in their sample, rates for first admissions for schizophrenia are disproportionately high both for patients with lowest socio-economic status and for patients whose fathers had lowest occupational status, they are not necessarily the same patients. Some of those whose fathers were in the lowest occupational status had origins in the higher status positions. There is thus evidence to support the proposition that most schizophrenics come from the lowest socio-economic classes, and that schizophrenics are characteristically downwardly mobile. However it is still not apparent what causal factors are operative in the process.

While it might be argued that entry into the lowest socio-economic class is the precipitating factor in the onset of the disorder, and consequently that, in all cases, some aspect of the lower class life style is associated with the breakdown, we cannot identify with any accuracy what factors in this way of life are associated with breakdown, nor can the possibility that breakdown might precipitate a descent in socio-economic status be ruled out. (The same precipitating factor could also operate amongst the lower classes; but it is not apparent because they have no further to fall.)

Comparing their sample with a cross section of the total

population Turner and Wagonfeld produced two important findings, (a) that schizophrenics are more downwardly mobile than the control group of normals; and (b) downward mobility is not attributable to loss of an established occupational position; but reflects a failure to achieve an occupational position commensurate with their abilities.

It might be argued that these occupational failures are associated with the onset of the disease in the middle or late teens (Morris, 1959:305; argues that his results indicate that the patients experience a downward drop in S.E.S. because of the illness) or legitimately, that occupational failure causes stress in the predisposed personality which precipitates the psychotic breakdown; but these explanations can only be inferred and not derived from the data. At this point it is sufficient to note that the appearance of the disorder is associated with that period in which the individual is attempting to assert (or at least is customarily expected to assert) his independence from his family group. Why this should be so is not derivable from either Goldberg and Morrison's or Rogler and Hollingshead's studies because their data are of the wrong order.

(iv) A modified drift hypothesis

This raises some important questions about the "drift hypothesis" as originally formulated, and suggests that the high rate differentials in the lower socio-economic classes are not

simply a result of the misclassification of an individual's occupation at the time of his commitment after he has suffered a decline. Rather, a more sophisticated hypothesis is required which, it seems, should deal specifically with an individual's inability to achieve the occupational levels expected of him. Dunham and others (1966) have reformulated the hypothesis in terms of status inconsistency (and incidentally find substantive support in the work of Jackson, 1962; Jackson & Burke, 1966; on psychosomatic symptoms) and attribute breakdown, in part, to be stresses associated with failure in achievement of the expected position. (Discussed in more detail below in section on stress)

Thus the important problems raised by these studies would seem to be the extent to which the high incidence rates in the lowest socio-economic stratum is explicable in terms of new cases appearing in that stratum and the contribution underachievement makes to these rates.

These questions are not soluble with the data presented in the studies reviewed. If it were possible to solve these problems, it would still not be clear why these effects should be as they are. Dunham, (1965) in his Detroit study argues on an inferential basis that psychotic symptoms are the forces which produce mobility -- the pre-psychotic's

traits, attitudes, mannerisms, and verbal reactions become only too obvious and operate against his securing a position in the work force, and, -- if he does secure some position -- operate to restrict his advancement in the job.

(1965:113)

Unfortunately, (characteristically?) he gives no data on the problems of occupational mobility, nor does he analyse any of the many extraneous factors which may be associated with downward mobility. A useful palliative to such monocausal explanations is the literature which seeks to implicate mobility per se as an etiological factor (see Odegaard, 1932, 1936; Astrup & Odegaard, 1960: for details on geographical mobility, also Tietze, et al., 1942; Leacock, 1957), Kleiner and Parker (1963) and Myers and Roberts, (1959) have suggested that the stresses associated with mobility have etiological significance; but significantly, the issue is still not resolved and given the data they present, it is still not clear whether it is the stresses of mobility which have the etiological effect, or whether it is that psychotic or pre-psychotic people have a tendency towards mobility.

As with the social class data, it is not clear what it is about mobility that is etiologicaly significant. If we refer to the two explanatory functions of epidemiology mentioned above it could be argued that while a sophisticated 'drift hypothesis' might go some way to explaining the distribution of identified cases, it cannot stand for a complete etiological explanation. While it may indicate factors which may be causative it can give no definitive answer to which factors are causative.

This type of explanation requires speculation on a psycho-social level, and it is to this we turn in the following section.

c/ Psycho-social 'causation'

It should be apparent from the discussion of the 'drift' and 'breeder' hypotheses that in order to move from statements which concern themselves exclusively with an explanation of rate differentials to etiological explanation data of a different order on the immediate social and physical environment of the identified case prior to his commitment are required. These data cannot be derived from the aggregate data available in hospital records or similar sources. The attempts which have been made to give psycho-social explanations to ecological data commit the 'fallacy of the wrong level' discussed above. Because of the level of the data collected; such inferences are not legitimate and should rather be considered problematic and as objects for research rather than accepted as assumptions in the analysis. Needless to say, the 'ecological fallacy' will be rampant in what follows. I now turn to an examination of the various socio-psychological theories which have been offered to explain the rate differentials.

A feature of the explanations which involve social-class or particular areas of a city is the implicit assumption that there are schizophrenogenic factors in these physical and social environments. It has not been clear what these factors are; but two which have received recurrent attention are social isolation and stress.

(i) Social isolation

Of all the psycho-social explanations of the etiology of schizophrenia social isolation is perhaps the most aesthetically

pleasing because it reflects so much of the typical reported symptomatology of the schizophrenic: in particular the retreat from reality, and virtual autism in social relationships.

Faris (1934) gave what is perhaps the earliest expression of major assumptions of the hypothesis when he argued that:

any form of isolation which cuts the person off from intimate social relations for an extended period of time may possibly lead to this form of mental disorder. The eccentric behaviour is a result of the seclusiveness of the person, and the seclusiveness is the result of the long period of isolation. The isolation may not be voluntary and indeed seems to be rarely, if ever, of the individual's own choice; but rather to circumstances beyond his control. Typically the isolated person makes a struggle to establish intimate social relations and feels lonely when he fails. In the beginning of the process the "seclusiveness" or "shut-in" trait is not the cause, but the result of isolation. The other eccentricities follow from this seclusiveness.

(1934:157)

This hypothesis received substantive support in Faris' work with Dunham (1939). (See also Dunham 1944) High rates for schizophrenia were found to be associated with areas characterised by (a) a high proportion of single unit dwellings; (b) high residential mobility; or (c) among ethnic group members who lived in areas dominated by other ethnic groups. These all seem to be situations in which social isolation would be likely to occur; but, unfortunately, to reason from these characteristics of areas to the properties of individuals is clearly to commit the 'ecological fallacy'. (See the discussion of Clausen & Kohn, 1954)

This same criticism may be made of Jaco's work (1954, 1957, 1960) which examined the characteristics of the communities from

which his identified cases originated. His findings support Faris and Dunham, as do his conclusions:

At least the prevalence of a high degree of social isolation in those communities known to have a high incidence of rates of this mental disorder has been empirically established and warrants serious consideration as a precipitating influence in the social etiology of schizophrenia.

(1954:577)

Unfortunately he omits all mention of the possibility that schizophrenics moved into those areas after 'contracting the disorder', thus making isolation not so much an etiological factor, but a situation which may be sought to alleviate certain of the problems associated with the disorder. (Again, the image of the schizophrenic is that of a mindless creature.) Nor is it clear that because the case originated in an area characterised by isolation, he necessarily was isolated himself.

A study which concentrated on the characteristics of the individual and not those of his community was that of Clausen and Kohn (1955) which did not support the social isolation hypothesis. The authors examined the 'basic background data' for all persons hospitalised at any public or private psychiatric facility between 1940 and 1952 from Hagerstown and the surrounding Washington County area. They sought to establish whether the degree of social isolation in their patient group differed from that of a control group matched on the basis of their names appearing next in the school register. Interviews were conducted and focused on the "residential and occupational history, relationships in the parental family, friendship and activity patterns in early adolescence, dating patterns, social participation as an adult, and a brief

psychosomatic inventory." On this basis they claim to have established the recalled interaction patterns of cases and controls at the age of 13 to 14 years. (It is significant that the authors do not consider the quality of the relationships, nor the meaningfulness of the friendship and activity patterns -- although given this type of study it would clearly have been impossible.)

In certain respects the findings of the study are incomparable with those of Faris and Dunham. In Hagerstown, as illustrated earlier, there was no significant concentration of schizophrenics in the lowest socio-economic class. If, however, social isolation is regarded as a necessary, if not sufficient 'cause', regardless of social class, and this is what Faris suggests in the quotation above, then the pattern should exhibit itself despite the differences in class distribution of the reported cases.

Their general conclusion was that

for the group ... studied the data do not support the hypothesis that social isolation in adolescence is a predisposing factor in either schizophrenia or manic depressive psychosis.

(1955:272)

It could be argued that one reason for the discrepancy between these and earlier findings was the authors' concentration of childhood experiences of their cases. It could be argued that Faris was referring in the quotation to the immediate experience of the cases prior to their commitment. However data supporting the Clausen

and Kohn findings and concentrating on the contemporary experience of the cases are provided in an earlier study by Weinberg (1950) who focused on the backgrounds of 53 reactive schizophrenics in two State Hospitals.

No significant evidence of social isolation was found. However, in his explanation of his findings and on the basis of his examination of case histories Weinberg sought to explain his data in terms of a social withdrawal hypothesis. (An explanation which is not anathema to Clausen or Kohn.) Social withdrawal, or 'disruption in role taking'

has a protective effect upon the schizophrenic insofar as it spares him from accepting evaluations of others and looking back on himself.

(1950:256-257)

This withdrawal is a consequence of numerous irreconcilable personal conflicts in the pre-psychotic individual:

These conflicts are so unbearable because they are so self involving. The schizophrenic regards himself as a failure and/or completely loses confidence in his ability to manipulate his environment... The crucial forms of isolation of schizophrenics emerge from the following personal experiences: (1) they reject the self-image but strive for a self acceptance and social acceptance; (2) they are unable to communicate their conflicts to other persons or do not have accessible persons to whom they can communicate their conflicts; and (3) they resort to withdrawal as a medium of self-protection.

(1950:256-257)

The isolation of the schizophrenic is not considered as an etiological factor; but as a technique he utilises to alleviate certain of the pressures his disorder brings to him in his interactions with others. This difference in interpretation is reflected in the differences between this quotation and that of Paris above.

Paris extrapolates from one symptom of the disorder to the aggregate characteristics of high rate areas; and in the correspondence identifies an etiological factor. However, an examination of the experience of the schizophrenics suggests that this very factor may be purposely sought to alleviate the disorder by the person afflicted. The image of the schizophrenic is changed. He need no longer be considered as a mindless creature; but one who is aware of the difficulties he is experiencing, and seeks in some way to adapt to them.

Weinberg's analysis marks a radical break with the conception of the schizophrenic we have been dealing with so far. Instead of the mindless creature of the 'breeder' and 'drift' hypotheses, Weinberg presents us with an individual who actively, and voluntarily segregates himself from significant social relationships. This he may achieve in one of two ways: (a) by a process we may term 'social autism' which involves him breaking off; or insulating himself from, emotional commitment within his family or original community; or (b) by moving into another area of the city and in this fashion severing his emotional ties. His choice of area will be determined by the availability of appropriate accommodation which in Weinberg's study seemed to be the poorer central sections of town.

It should be apparent that 'voluntary segregation' is not an etiological explanation but an explanation of why it is that certain areas of the city have higher rates of reported schizophrenia than

others. The problem of why it is that an individual should feel it necessary to seek isolation, or why he should exhibit the behaviour which comes to be labelled remains unsolved.

Gerard and Houston (1953) use an explanation similar to that of Weinberg in their explanation of the distribution of schizophrenics in Worcester, Mass.. The authors found that patients living with their families prior to admission had definite patterns of residential stability compared with those not living with their parents. When all the cases are considered together their distribution through the city corresponds to that found in previous studies. If only those cases living with their families prior to admission are examined the distribution throughout the city is random. Whereas, the cases living alone, or away from home tend to come from the central areas of the city and share a marked residential instability.

The authors suggest that choosing to live alone is an escape from disruptive family relationships. While this fits well with Weinberg's speculations, there are no specific data to support these inferences in Gerard and Houston's research. The latter authors fail to show that those patients living outside their family settings were not schizoid before they moved, nor that they had moved by choice. It could be argued equally from their data that the cases were forced to move by familial pressure when their disorders became intolerable. Hare (1956a, 1956b) in two important papers adds much to the resolution of this issue. His earlier findings (1956a) broadly confirm those of Paris and Dunham, with an important refinement. High rates occurred not only in the poor central areas; but in the

good central areas. The common factor in both these areas is not high population density, but the high number of persons living alone. The peripheral areas where rates were low, were not (as in Faris and Dunham) the high class residential areas; but council estates (rent supplemented) characterised by a low proportion of people living alone. (see also Sainsbury on suicide, 1955) His conclusions in this paper are cautious and he admits that these high rates are attributable to either the 'breeder' or 'segregation' ('withdrawal') hypotheses. However, in his reworking (1956b) of the data although he finds support for both hypotheses he comes down in favour of the segregation hypothesis. Hare suggests two causes of cases leaving home prior to their commitment: (a) personality difficulties of a schizoid nature result in the patients leaving their families and moving to boarding-house accommodation; (in this instance he argues that the transition from personality disorder to schizophrenia is relatively slow and may occur over a period of years). (This explanation is not incompatible with that of Weinberg.) (b) The prospective patients are separated from their families by force of circumstances, not connected with their disorder, and this isolation is probably a factor in their mental illness.

The issue is by no means settled. While, again, there is considerable agreement on what is to be explained, there is considerable confusion as to what the explanation is. These confusions stem from the lack of the appropriate data which would decide the issue. It is still not clear why individuals move into the high rate areas, nor in what ways they are different from the occupants

of those areas who do not become schizophrenic, or, at least,
are not identified by their use of psychiatric facilities.

(Remembering Srole's findings on total prevalence of disorders.)

No data are presented on appropriate control groups of 'normals' living in the same areas giving 'normal' reasons as to why people choose to move into (or remain in) those areas. And, despite Hare's inferential conclusions, it is still not clear whether the disorder is a consequence of the area, movement is a consequence of the disorder, or the disorder is a consequence of the movement (given that the movement could have occurred in the pre-psychotic period).

What emerges is a psycho-social explanation without the attendant psycho-social data in support. Consequently, it is still not clear what the significant factors are, or why or how they are significant. One factor which may be important is the response individuals make to stress.

(ii) Response to stress

A common feature of the etiological-type explanations so far has been either an implicit, or explicit recognition of the possible relationship between some aspect of stress and the etiology of the disorder. Aspects of the environment are considered stressful, or it is the stresses associated with lower-class 'ways-of-life'; or it is the factors associated with mobility, isolation etcetera which precipitate the disorder. In all cases stress is used as an hypothetical intervening variable, to mediate between the ecological data and the risk of becoming a case. In the 'hospital studies' it

is not possible to measure the stress directly because this sort of data is not recorded on hospital records. It is an inference with no basis in the data collected; but one which nevertheless is essential if an explanation is to be forthcoming.

(iii) Stress and status integration

This lack of necessary data is equally true of the literature which adopts an inconsistency model of stress. Dunham et al (1966) were mentioned previously and the similarities between their work and that of Jackson, and Jackson and Burke commented upon. The basic thesis of the approach is that inconsistency of ranking on the different dimensions by which status is accorded in a group, organisation, or society produces stress within the individual which manifests itself in particular types of response.

This explanation is of a different order than that of Srole, Langer and Michael, also Rogler and Hollingshead or Myers and Roberts who concern themselves with stressful events; death in the family, unemployment, inadequate income etcetera, as specific problems. Status integration concerns itself mainly with the expectations made of an individual as a result of his incumbency of a particular status position, expectations he has of himself, and that others have of him. It is assumed that unequal ranks on the different dimensions carry with them discrepant expectations which produce stress within the individual because of the ambiguity of his role position.

This model would fit nicely the explanation offered by Weinberg (see also, Hammet, 1965; Hinkle & Wolff, 1957; Sewell & Haller, 1959) and is akin to that offered by Kleiner and Parker (1966) in their review of the literature and examination of the data from Philadelphia. Again while this study does not concern itself with schizophrenia per. se. their explanation in terms of the wider discrepancy found between the achieved and ascribed goals amongst those exhibiting psychopathology than 'normals' is relevant for a discussion of schizophrenia. It is suggested that the frustration and striving which are consequences of the discrepancy may have etiological significance in the development of schizophrenia or mental disorder in general.

But, as the authors themselves point out, it is not possible from the data they review and those collected in their own study, to determine whether the pathology is a consequence of the discrepancy, whether the discrepancy is a consequence of the disorder, or whether the relationship is spurious and attributable to their common relationship to some third factor not identified.

This problem of defining the independent variable is common to the community studies discussed below. Apart from variables like a death in the family, or parental socio-economic status which we would feel confident in arguing are not a consequence of the disorder, it is crucial that it be shown that the variables chosen as elements of the stressful environment (and therefore independent) are not themselves associated with the disorder. Dohrenwend (1965) is arguing a similar point when he asserts that it is important that

it be shown that the individual is not responsible for the stressful environment. That is, he is powerless to change the environment; but must accommodate to it. But phrasing the issue in these terms tends to obfuscate the issue of those problems which may be a consequence of the disorder, for example unemployment, but which the individual is powerless to influence. These factors would not be of etiological significance, although it might be argued that they aggravate the disorder.

As with all of the 'hospital studies' we have discussed so far, it is not possible in these studies, on the basis of the information given to examine the salience to the individual of the dimension which is given etiological significance. This is a problem which plagues inconsistency studies as has been argued elsewhere. It is not accurate to make statements about the salience of the discrepancy between status expectations, or ascribed and achieved goals on the basis of the aggregate data customarily employed, unless we know how actors perceive those situations themselves, and what alternatives they see open to them. Stress has still the status of an intervening variable, and no data are collected with the specific purpose of testing its etiological significance.

The field studies have concentrated more closely on stress as a possible etiological factor.

(iv) Stress and the field studies

Myers and Roberts in their complementary volume to Hollingshead and Redlich's New Haven Study (1958) argue that their theoretical position

is that tensions and adjustments of both groups and individuals are related to the social class system. More specifically, we hypothesise that persons occupying different positions in the stratification system are subjected to stresses and strains characteristic of those positions which create personality problems. Our problem is to determine if persons at different levels face different social and emotional problems and if they develop a psychiatric illness in working out an adjustment to them.

(1959:13)

The authors are not exclusively concerned with explaining rate differentials but are concerned with the class factors associated with the development of functional mental illness among two groups of patients (from Class III and Class V on the Hollingshead Index of Social Position, 1958). (Nor are they concerned to look solely at the contemporary experience of their cases but examine also their past stresses.) In general the writers found significant differences between the patients of the two groups in "intrafamilial role relationships, sex role development, external community pressures, attitudes towards psychiatric illness, the therapy process, and symptomatology." Significantly mobility was found to be associated with Class III but not in Class V. (1959:247)

Unfortunately it is not possible to draw etiological conclusions from the study. Because of limitations on the size of the sample, its restriction to treated cases, the lack of data on

early infancy, and the omission of a control group, Myers and Roberts are quick to point out (1959:29, 246) that their study cannot tell why certain people in a class become ill and others do not. Nor, and this point is particularly significant because it has not arisen before, can it tell why certain persons in a family become 'ill' while their siblings do not. The significance of this last point cannot be overemphasised particularly in sociological explanations of mental disorder. In this situation we have not only to explain why certain people become ill while others do not; but why members of the same family, who we would expect to be subject to similar external stress associated with their class position, do not all become afflicted. The question becomes confounding, to say the least, when we consider those studies which have revealed the startling fact that in certain families when one member is removed because of mental disorder, another develops the disorder. (Jackson, 1957) (This could perhaps be explained in terms of the communality of external stresses; but such an explanation would not suffice to explain why it is characteristic for there to be only one schizophrenic child per family, and extrapolating from Jackson's observation, why it is that in certain families it seems to be necessary that there be one schizophrenic child. Bateson, 1959)

Although it did not focus specifically on schizophrenia, (using instead a global index of adjustment) the Midtown Study (Srole, 1962) presented a hypothesis which may be pertinent. Langer and Michael (1963:9) in the companion volume to the Midtown study offer the following definition of stress:

any influence, whether it arises from the internal environment or the external environment which interferes with the satisfaction of basic needs or which disturbs or threatens to disturb the stable equilibrium.

Stress causes strain within the individual which may manifest itself in what may be identified as symptoms. It is the testimony of the Srole study that not everybody who goes through this process comes to the attention of a psychiatric functionary. Using data from the standard, structured interview schedules (see p.40ff) the researchers derived a stress score for each person interviewed. (See the original volume for the rather complicated computation of credits to arrive at stress scores.) From there it was a relatively easy task to relate the stress score for each person to the degree of impairment experienced by that particular individual. As expected, on the basis of the previous research in the area, the poorer respondents experienced, on average, more 'life-stress' than their rich counterparts, a finding which on surface value, would go a long way to explaining the differential class distribution of 'global' disorder, and by implication schizophrenia.

However, it was not simply that lower-class people experienced more stress per se; when the number of 'life-stresses' was controlled

it was found that lower class people exhibited a higher degree of impairment for the same stress scores. The researchers are extremely cautious in the manner in which they explain the relationship. They argue that it appears that those in the lowest stratum are equipped with poorer defense mechanisms, and poorer abilities to cope with stress in their social and physical environments.

There are at least two possible explanations for this inability to cope. First, it may be a function of faulty socialisation and the acquisition by the child of inappropriate cognitive models of his environment (this type of explanation is akin to Kohn's (1969) recent thinking.) Or, second, it may be a function of the lack of supportive mechanisms in the lower class subculture. This particular approach to explanation has been elaborated by Srole and other contributors to the Conference on Mental Health and Poverty (1969) and is the impression drawn from Rogler and Hollingshead's work in San Juan (1965). These two explanations are not necessarily exclusive, and should be considered as complementary. Unfortunately, the data will support neither hypothesis. It is significant that in both the New Haven and the Manhattan studies the attempt was made to examine the social experience of the schizophrenics. The attempts were limited and the results which ensued valuable more for the research they will stimulate than in the definitive answers they provide. It was clearly recognised that it is necessary to specify the mediation between the gross aggregate data on class membership and the like, or individual data on occupation, income etcetera and rates of

disorder or the identification of individual disorder. By identifying those factors which were considered as stressful in the environments of their respondents this mediation was approached, and the more complicated models derived. We may graphically illustrate the more complicated models as follows: in Myers and Roberts:

social status → status specific stress factors → strain within individual → presentation of psych. symptoms

and Dunham:

status inconsistency → stress → psychiatric symptoms

(v) Labelling and stress

The Langer and Michael model is complicated by the fact that their cases were not drawn from the population of treated cases. In this instance it is possible to consider the behaviour which may be labelled as symptoms as

enduring, stable, self-perpetuating manifestations of personality defects, or may be regarded as the normal response of individuals to the stressful events in their environment.

(Dohrenwend, 1965)

That is, the response to stress may not necessarily be maladaptive; but an adaptive response. (See the Army Medical Service Graduate School Reid Symposium on Stress, 1953) The situations in which it becomes maladaptive are reflected in Myers and Roberts' (1959:15) definition of stress as

an unpleasant emotional tension engendered in an individual when he feels that he is unable to satisfy his needs within his situation of action.

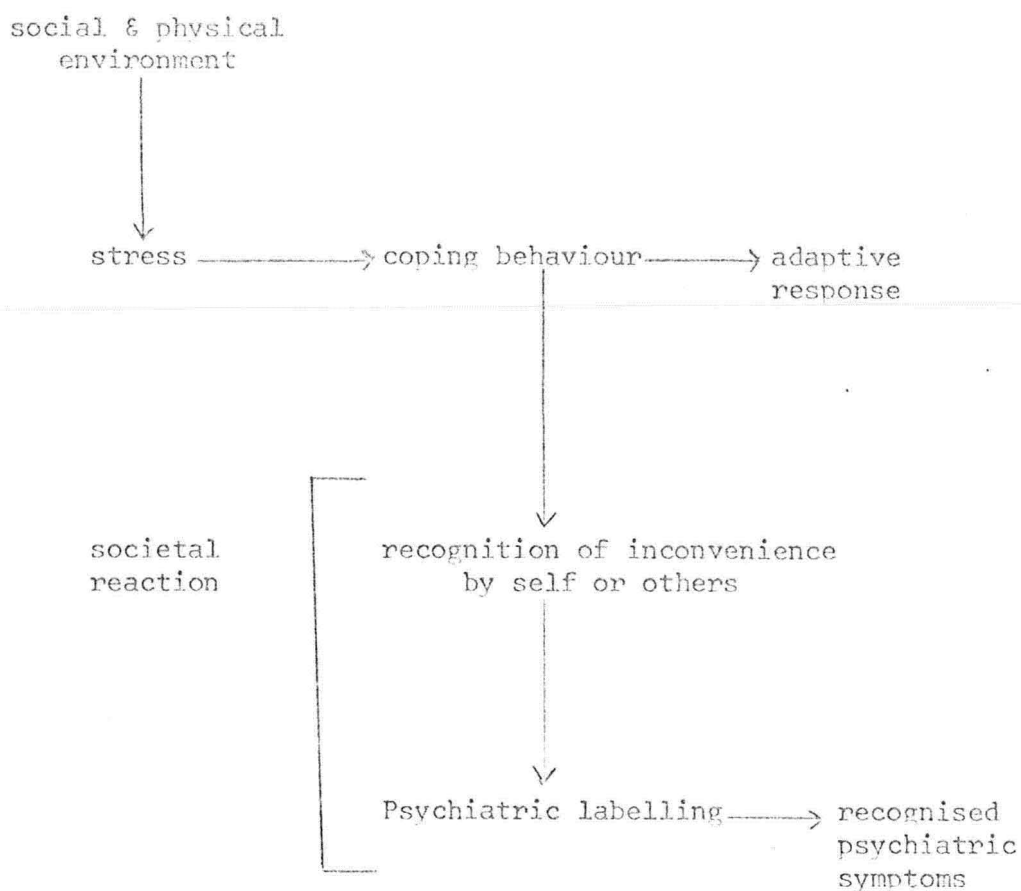
(Emphasis added)

This does not involve the person being actually labelled as ill; but he must be aware of the problems caused by the stress. Dohrenwend in his definition of psychological disorder explicitly recognises the labelling aspect:

(s)trссор induced symptomatic response judged by the individual and/or other social agents to be harmful to the individual and/or others with whom he stands in social relationship, indicate psychological disorder if the symptoms continue (a) after the stressor ceases to impinge on the individual, and (b) despite sanctions directed towards the individual by social agents who judge the response maladaptive.

(1965:68-69) (emphasis added)

The more complicated Langer and Michael model could be represented thus:



where the social and physical environment may produce stress for an individual. He engages in coping behaviour which may become adaptive and customary patterns of behaviour. Or he may become aware of difficulties engendered by these coping behaviours and thus seek psychiatric help. Alternatively, taking Dohrenwend's definition that behaviour may be considered by a psychiatric functionary as a danger to the individual, and/or to others.

A model of this nature fits well with the criticism we have made so far of the epidemiological research. However it still remains to specify the nature of the stress producing factors, and the factors associated with the labelling of the coping response as evidence of mental disorder.

d/ Conclusions

Having run the gamut of the major theoretical explanations the impression which remains is similar to that of Mishler and Scotch who at the end of their review liken the problem of drawing conclusions to

talking with the relatives of the deceased after returning from a funeral. Other than some platitudes there is little that can be suggested that would remedy, alleviate, or eliminate the trouble.

(1963:340)

However, despite the lack of definitive theoretical explanation (and after all, the majority of studies made no claim to anything more than a modest tentative exploratory statement, see Birch in Pasamanick, 1959:90) the discussion has highlighted a number of interesting problems and suggestions which may prove fruitful if analysed without the epidemiological framework.

In certain respects we are now no nearer an etiological explanation than we were when we began the examination of the theoretical explanations. While there are fairly plausible explanations of the distributions of reported cases, particularly in the modified 'drift hypothesis' we still have no clear indication of what factors are associated with the onset of the disorder. There is no evidence that there is anything in the immediate environment of the schizophrenic which precipitates the disorder, however it was suggested in the discussion of Hare, of Goldberg and Morrison, and of Rogler and Hollingshead that the disorder is in some way associated with the individual's inability to secure an occupational position commensurate with his ability. It is not clear from the research whether this inability is the result of the disorder, as the comment quoted by Dunham would seem to suggest, or whether the inability to achieve precipitates the disorder. (Suggested by the status inconsistency appeal) There were no specific data to support either viewpoint. This lack of appropriate data reflects a problem met in all of the explanations discussed so far; that of providing a mediation between data collected on the sociocultural level and the individual's behaviour reflected in his appearance in the rates of the disorder, or in his identification as a case in the community studies.

One specific area of studies which we have not yet examined, and which may give us some insights into the childhood and early adolescent experience of the prospective patients, is those studies

which have dealt with the family. As we have seen, the epidemiological studies reviewed have taken little account of the evidence from the family studies. This is unfortunate for, as some authors note, the family mediates between the societal and cultural processes and the individual's response.^{10.} In the Midtown study, it is assumed that certain socio-cultural and demographic factors are associated with differences in intrafamilial functioning; specifically (a) in behaviour patterns culturally enjoined and inhibited through the definition of normative rules; (b) in bonds between the several kinds of family members; (c) in cohesion; (d) in life style and social resources. (Srole, 1962:18) As Srole comments,

(i)n our formulation, the independent, sociocultural type of demographic factor is seen as one potential key to inter-group differences in intrafamily dynamics.

(1962:19)

These in themselves may affect the frequency with which families internally generate noxious or crisis situations as well as their immunity and invulnerability in the face of stressful external conditions. However in the research report there is little if any discussion of the family dynamics; the implication of a pseudo-matriarchal pattern in the family is referred to in a footnote (1962:356, f.n. 30); and its importance substantiated not by the results of the study itself (for there was not adequate data collected on the family to support any but the most structural of explanations i.e. in terms of parental or sibling deaths, divorce, or evidence of severe hardship) but by reference to clinical research. In the discussion of the Langer and Michael analysis

of the Midtown data it was suggested that differences in socialisation may account for the more extreme response to stress of the lower class cases; but again there was no support in the data for this hypothesis, while the aim of the research project outlined in the quotation above would seem to call for information along this dimension.

A similar criticism could be made of the Rogler and Hollingshead research. In their statement of the research problem the authors give the impression that they will be confronting some of the issues we have raised.

Although some researchers continue to look for a genetic base to explain mental illness, others believe that the tangled skein of human misery labelled schizophrenia, may be unravelled by careful studies of the family as a social group. For the sociologically oriented researcher, the family continues to be a focal point of studies aimed at discovering the causes of schizophrenia. We studied mental illness in the family and community rather than in the hospital and clinic.

(1965:4)

Unfortunately, the authors seem to ignore much of the research they refer to in the footnote to the above quotation (1956:4 fn 1) and consequently provide little information about the internal dynamics of the families of the patients they study, (although this would have been extremely difficult given the type of data collected by introspective report).

It should be clear that we are not yet in a position to make statements about the factors associated with the onset of the disorder. We cannot yet formulate a sequential law. Much of the confusion stems from the inability to define exactly what

it is we are talking about. In the previous section the problem was confounded when it was suggested that an individual's response to stress in his environment may either become adaptive behaviour or mental illness depending on whether that behaviour is labelled as such by the individual himself (that is by his seeking psychiatric help) or by a psychiatric agent when that behaviour is brought to his attention. That the symptoms themselves may not be important in this process is indicated by the studies of total prevalence, which indicate that persons with severe symptomatology may never come to the attention of a psychiatrist. This raises further the conceptual and methodological problems involved in the definition and measurement of mental illness which surely must be solved before any statements can be made about etiology. We have up until this point accepted a medical definition of what constitutes a case, either in terms of commitment to a hospital, or the psychiatric diagnosis of a protocol in one of the field studies. In the next chapter we will look more closely at the assumptions involved in such a definition. It is significant that it was in the course of the community studies that this labelling aspect of mental illness was suggested because these studies accept an explicit medical definition of the disorder by their use of psychiatrists to diagnose protocols. While a psychiatric diagnosis in general (even if it is only of a protocol) accepts the existence of an entity which is diagnosed as an illness, the labelling approach as it has been developed by Scheff and other symbolic

interactionists in some senses denies the existence of such an entity and is concerned more with the factors involved in the application of a psychiatric label. Working within the psychiatric framework of the community studies, it is difficult to conceive how it is possible to diagnose an entity as the illness while recognising at the same time that similar forms of behaviour may be considered as adaptive, for different individuals. The issue seems to become one of specifying the nature of the inconvenience caused to the individual or to others by his response to the stress producing factors; and of specifying what factors are associated with whether that behaviour is considered as adaptive by self or others, or as being a candidate for a psychiatric label (diagnosis). And it is to this problem we turn now.

In understanding the new viewpoint on schizophrenia we might remind ourselves of the six blind men and the elephant: one touched its body and said it was a wall, another touched an ear and said it was a fan, another a leg and thought it was a pillar, and so on. The problem is sampling, and the error is incautious extrapolation.

(Laing, 1970:88)

At this point, an important distinction must be made which has remained implicit in the discussion so far. We must be able to distinguish between factors associated with the definition of an individual as having mental illness, and those factors associated with the identified patient's actions which lead to that definition. While this distinction may prove to be invalid, as the analysis proceeds -- in which cases the only factors associated with diagnosis or commitment (except for the nosocomial factors) will be the presence or absence of disorder and its severity -- it will be valuable to maintain it at the outset of the analysis so as not to obscure factors which may be associated with the diagnosis and not the presentation of symptoms, and vice versa.

a/ Definition of a case in Hospital and Community Studies

We discussed earlier the problems associated with the use of hospital statistics as true indicators of the extent of the disorder. There is no need to reiterate them here save for Gruenberg's opinion:

I don't think that it (hospital admission) is a good

definition of illness...The more we get into it, the more clear it becomes that it doesn't have any substantive meaning.

(quoted in Srole, 1962:351)

It has been argued at length that rates of reported illness are inadequate data on which to make etiological statements about a disorder, and that reliance on the data reported in the hospital records, or ecological data, may be inadequate to support all but the most general hypothesis.

Clinical judgement is used in both studies of treated and untreated disorder to establish and identify cases. The difference is that while we are presented with information about the classification procedure in the field studies, and consequently, we can attempt to assess the criteria on which diagnoses are based, this information is not available to us in the patient studies. Cases in these studies are identified on the basis of their hospital records, and although the findings are presented in the form of categories like those of the diagnostic manual of the American Psychiatric Association, neither the information available to the judge, nor the criteria for decisions are available in the reported studies. We are expected to accept such information as 'objective' fact, without any information about the actions associated with the diagnostic label, nor the situations in which the label was applied. Both factors, as we shall see, are crucially important in the adjudication of impairment.

Nor do we know, in the majority of hospital studies, who performed the diagnosis - a resident psychiatrist, first year intern,

psychiatric nurse or whoever - when the diagnosis was made, nor what changes occurred in diagnosis over the course of stay in the hospital. All are factors which would seem to be of crucial importance in establishing a reliable "sociological" diagnosis, or put differently establishing a correspondence between the sociologist's categories, and the social meanings shared by those involved in the diagnostic process.

Much of the inadequacy of the "patient studies" is highlighted by the results of the various community studies which indicate a total prevalence out of all proportion to the number of reported cases. While, as we argued, this raised the question of the legitimacy of using rates of disorder as indices of true prevalence, a more serious problem is that of deciding who is, and who is not ill, particularly when the 'afflicted' person and his peers do not consider him to be so. Implicit in this statement is the assumption that a psychiatric diagnosis constitutes the defining characteristic of mental illness. That is, "schizophrenia", rather than specifying a disease entity, establishes a relationship between a psychiatrist and the person he diagnoses, and between the person diagnosed and those to whom he relates. Even if we accept the notion of a disease entity, that same statement holds true - a psychiatric diagnosis defines a relationship between the labeller and labelled, a definition which includes a statement about the labelled's actions to the effect that they are caused by the disease. (See Friedson, 1970, esp. Chp 12, p. 244)

This latter statement may make certain readers uncomfortable, for on the one hand it questions the legitimacy of psychiatric diagnosis, and on the other contradicts the explicit aims of the community studies. While the hospital studies accept an implicit medical definition of illness in their use of reported rates (i.e. actually diagnosed persons) such medical definition is explicit in the community studies when psychiatrists are used to formulate the psychiatric questionnaires, analyse the resultant protocols and make decisions as to impairment or adjustment. The explicit aim of the studies was not to question the legitimacy of a psychiatric definition; but to establish the total prevalence of individuals whose behaviour could be classified by a psychiatric definition (see also Roman, 1972; who makes a similar point).

That is, while the psychiatrists of the Midtown study made their classifications in terms of degree of impairment the original aim was to make specific diagnoses. Unfortunately, on the basis of the limited data they were able to collect:

... it became apparent very soon that we could not make a diagnosis in the usual sense of this word, on the basis of this material. Symptoms could be listed, and complexes of symptoms could be appraised as possible diagnostic categories but the nature of the data led us more in the direction of some kind of overall evaluation of mental health functioning.

(Srole, 1962:63)

By contrast the Leighton's accepted that

... the defining of psychiatric disorder, ... would rest on judging an individual as a person who, if thoroughly studied by a psychiatrist, would be diagnosed as suffering from one or more of the specific conditions described in the Manual.

(D. Leighton, 1963:118, the manual referred to is the Diagnostic and Statistical Manual of the American Psychiatric Association)

And Essen and Muller (1966) used the probability of pathology as their defining characteristic.

In all cases the purpose was explicitly a psychiatric recognition of cases. In the Srole and Leighton studies "diagnosis" was made on the basis of a team psychiatrist's evaluation of a structured interview schedule recording the subject's self-reporting of ever having experienced any of the symptoms reported on the schedule. In the Midtown study, Srole et al selected a group of items from the Army Neuropsychiatric Screening Adjunct and the M.M.P.I. "consisting principally of the psychophysiological manifestations of these tapping the anxiety, depression and inadequacy dimensions" (Srole, et al, 1962:42). In addition the psychiatrists on the team contributed 40 items. The final decision in determining the 120 items used was made on the basis of the 'clinical experience' of the senior psychiatrist (1962:60). A similar procedure was employed in the Stirling studies which used N.S.A. and other test scores without specifying the explicit selection procedure (Leighton, D.C. et al; 1963:85). Dohrenwend (1966) has questioned the validity of such procedures, particularly the content validity in the absence of systematic sampling of items, although he concludes that such validity would be difficult to achieve because of the absence of agreement in defining the variable. However, more important for our purposes here, is the process by which psychiatric diagnoses were achieved.

The subjects themselves were not interviewed by the team psychiatrists, nor were the interviewers themselves psychiatrists (although an attempt was made to recruit people as interviewers, at least in the Midtown study - "who had technical experience in methods of intimate investigation ... These included psychiatric social workers, clinical psychologists, social caseworkers, and social scientists" (Srole, 1962:31). However, in the Midtown study, part of the data evaluated by the psychiatrists were the respondents' "free associations, elaborations and asides spontaneously given or elicited" by the interviewer, and observations gained by the interviewer in the interaction with the respondent:

aspects of the respondents behaviour, including manifestations of ease or tension, affect or mood, appropriateness of replies, apparent I.O., dress and grooming habits, muscular ties, stutter or stammer in speech, memory difficulties, psychical deviations or disabilities.

There is no indication given of how far this information was used in the evaluation of cases - whether it was an integral part of the diagnosis, crucial in borderline cases, or whatever. The inclusion of such data would seem to be in flagrant opposition to the claims to objectivity of the psychiatric diagnosis, particularly when no indication is given of how and when it is used, what criteria the interviewers used in reporting such behaviour, and what level of reliability there existed between interviewers in reporting similar behaviours. More importantly, especially in a study which claims to objectivity on the basis of the independent analysis of interview protocols by the team psychiatrists, and examines the reliability of such diagnosis, no indication is given, nor was any systematic analysis undertaken of what biases entered the data from alerting the interviewers to such phenomena. That is, rather than asking

the interviewer to remain neutral with respect to the data, he was encouraged to engage the subject in free-associations, etcetera. While this in itself does not preclude the data from analysis, analysis of that data can proceed only if the manner in which the data were elicited are taken into account, and the interpretations of the interviewer which effect the selection and reporting of information are made explicit. Needless to say we are not provided with this information.

A more severe criticism is that we are not provided with information as to how the psychiatric diagnosis was achieved. It would be something of a truism (euphemism?) to state that the processes and procedures through which individuals are identified as in need of psychiatric care are frequently unclear. We all know from our own experiences situations in which (to our minds) very "sick" individuals go unnoticed while persons exhibiting relatively mild symptoms are identified as in need of treatment and care. A re-reading of the protocols presented in Leighton's book, The Character of Danger, is illuminating for it serves only to re-inforce this impression -that is, at least to the non-psychiatrist! While certain of the cases do indeed seem bizarre, there are others, diagnosed with severe symptomatology by the team psychiatrists, who seem perfectly normal, manifesting the same types of response to stress any normal individual would. And one cannot help wondering if these cases which seem bizarre do so only because the behaviour is presented out of the context of its occurrence. (1963:150 ff)

The procedures by which these same psychiatrists identified their cases itself remains something of a mystery. The field studies claim objectivity in their rigorous discussion of methodological problems associated with the formulation of the questionnaire and agreement between psychiatrists on what constitutes a case, yet the process of evaluation is not explicitly reported. Clausen (1968) argues, that it was assumed that the use of the symptom schedules in each study would provide standard explicit, set data for psychiatric assessment; and goes on to question the use of symptoms in deciding on pathology: a more pertinent criticism, for our purposes, is of the symptom scales themselves. For the Midtown study the scales were tested against 139 diagnosed neurotic and remitted psychotic patients and 72 patients judged as "well" by psychiatrists after a 1/2 hour (!) interview. Of the questionnaire items 22 discriminated significantly between the patient and well groups ($p.01$). However (1962:3906) while the team psychiatrists reported that in rating actual cases they gave special attention to 6 of the items which did not differ significantly (see Dohrenwend, 1966 for a discussion of this point). Thus while the study made pretences to concurrent validity, the pretests were ignored in the psychiatric evaluations. A similar point can be made for the Stirling study, that is, while the test was "validated" against patient and normal community samples (though how it was possible to choose a 'normal' sample in a community of 64% impairment remains something of a mystery where impairment is the statistical norm),

the actual selection of items was not wholly defined in this study (1962:205) nor did the psychiatrists make use of objective scores in the assessment of psychiatric disorder. (See also Dohrenwend, 1966)

Thus while there are seemingly objective scales, the process of diagnosis remains a mystery. The Midtown psychiatrists admit in their comments on the study that

we used our clinical judgements to the best of our ability. It would be a mistake however, to overlook the fact that there remain some aspects of the process which are not altogether in our awareness.

(1963:62-63)

Unfortunately for the reader, little of the "conscious" process is made available!

The process is essentially private, and as such not replicable, nor particularly open to scrutiny. Leighton (1959:147-148) has commented on the difficulties involved in defining non-patient groups in the Midtown study. Six psychiatrists were asked to read the protocols of 50 white adult males and instructed to assess whether each was mentally ill or well. Fifteen were placed in the category as unequivocally ill and five of the remainder diagnosed as well. Which seems fine, until we realise that the five men who were diagnosed as well differed for each of the six psychiatrists! And it was not simply a matter of different classification of the equivocable groups. One psychiatrist's five "wells" were another's sickest group! So much for objectivity in the field studies.

So what on earth are these people talking about? There is a tremendous literature on the unreliability of psychiatric diagnoses.

(Blum, R.H., 1962; Ash, 1949; Clausen and Kohn, 1959; Hollingshead and Redlich, 1958; Hoch, 1959; Hollingshead, 1961) A case in point is Mehlman (1952) who distributed one group of 597 patients for diagnosis among 9 psychiatrists and another of 1,358 among 16 psychiatrists. There was a significant difference (p.001) between the percentage of patients assigned to organic versus psychogenic categories, and a significant difference (p.01) in patients diagnosed as manic depressive and schizophrenic. Despite the prevalence of a schizophrenic diagnosis there seems to be no term over which there is more dispute. Bannister (1968) has argued that schizophrenia is so confused a concept as to be scientifically useless and hence "research into schizophrenia as such should not be undertaken", (in Laing, 1970:II). This is very much the approach of Laing and Esterson who argue that schizophrenia as a disease entity is not so much a fact; but rather an "assumption and theory, a hypothesis" and that

... though the term has now been generally adopted and psychiatrists trained in it's application, the fact it is supposed to denote remains elusive. Even two psychiatrists from the same medical school cannot agree on who is schizophrenic independently of each other more than eight out of ten times at best; agreement is less than that between different schools, and less again between different countries. These figures are not in dispute. But when psychiatrists dispute the diagnosis there is no court of appeal. There are at present no objective, reliable, quantifiable criteria - behavioural or neurophysiological or biochemical - to appeal to when psychiatrists differ.

(1970:11-12)

This latter point -- there is no court of appeal in disputes over psychiatric diagnoses -- is crucially important when discussing

the social meaning of mental illness. Sociological approaches to mental illness have so often been dismissed, almost ad hominem, because their respective writers lack psychiatric expertise, and are thus incompetent to discuss questions relating to psychiatric issues. The above quotation which throws doubt on the factual status was made by a psychiatrist, and is supported by other psychiatrists in the field. For example Leifer, in a paper in the International Journal of Psychiatry (1971) questions the legitimacy of the medical model, and the whole notion of a disease entity. The comments on this paper are not to reject the premises, or arguments, but the conclusions, for to reject the medical model would, be premature while there is a lack of an alternative model. While this might be acceptable if the question were purely academic, it is deplorable that such a model be accepted when the attribution of disease labels has such profound personal consequences, in the deprivation of an individual's liberty.

The problem of defining mental illness cannot be treated purely as a technical psychiatric issue. The term has distinct jurial effects which are obvious in the legal procedures involved in the commitment of individuals into institutions in some of the United States (for example Maryland) and in the terms of commitment enshrined in the Ontario Mental Health Act. (A "forward looking document" in the words of the Ontario Health Departments 44th Annual Report)

Since the term defines a problem in social control, discussion of it must consider the technical and legal aspects and attempt to

resolve the essential ambiguity between the two. (Readers wanting a historical analysis of mental illness as social control are directed to Foucault, 1967; Madness and Civilisation.)

(i) The recognition of disorder

This ambiguity is compounded when it is realised that the initial definition of disorder, in the majority of cases, is performed by lay people, and not persons with psychiatric training.

The early definition of mental illness, especially in middle-class populations, are likely to take place in groups in which the person primarily operates: evaluations are made by the family, fellow employees, friends and employers. If symptoms appear and are not recognised as such by members of the individual's more primary groups, it is unlikely that he will become accessible to psychiatric personnel unless his symptoms become visible, and disturbing enough to lead to his commitment to some treatment centre by external authorities.

(Mechanic, 1967:24)

Among the working-class, these external authorities are likely to be the police or clergy, who will refer the person for psychiatric treatment. (Refer, here, may be something of a euphemism, for in Ontario, at least, such a referral may mean the unfortunate remaining in hospital for a period of 30 days before his case comes up for review.) The individual may, of course, define himself as ill, and seek psychiatric help. However, as a general rule, the madman is recognised by his peers, and it would be safe to argue that individuals who find themselves entrammelled in the formal and informal processes which lead to psychiatric treatment are there because they present a problem of some kind to others (and themselves) which cannot readily be defined within other categories of deviance. (Szasz, for one, regards mental-illness as a catch-all for behaviour which cannot

be accounted for in other terms.) This type of approach is made explicit in Dohrenwend's definition of disorder referred to earlier. That is, responses to stress become psychiatric disorders when they appear harmful to the individual or to others. The crucial phrase in Dohrenwend's definition is that "symptoms" are defined as maladaptive, if they continue

(b) despite sanctions directed towards the individual by social agents who judge the response maladaptive.

(1965:68-69)

The essential feature of this definition is that actions become symptoms if they are regarded as maladaptive by self or others, and become psychiatric disorders when diagnosed (labelled) as such by a psychiatrist. Dohrenwend does not discuss the informal aspects of the diagnosis, that is the recognition and referral by lay-people, nor does he explicitly criticise the notion of a disease entity. This diagnosis is held to be the recognition of a fact. However, such criticism is implicit, if not intended in his definition. For Dohrenwend, diagnosis is an act of social control. This act which would be legitimate if it constituted the diagnosis of a "fact" -- the fact of mental illness which causes behaviour which may be a danger to self or others -- but which is illegitimate if the nature of the phenomenon is fundamentally misconceived. It seems necessary at this point to belabour the problematic status of schizophrenia as a disease entity, and thus legitimate taking an alternative perspective on the phenomenon.

For Laing (1970:18) to regard the diagnosed patient as suffering from a pathological process as a fact is "unequivocally false", and to support this assertion he argues:

No generally agreed objective clinical criteria for the diagnosis of 'schizophrenia' have been discovered. No consistency in pre-psychotic personality, course, duration outcome, has been discovered. Every conceivable view is held by authoritative people as to whether 'schizophrenia' is a disease or group of diseases; whether an identifiable organic pathology has been, or can be expected to be found.

There are no pathological anatomical findings post mortem. There are no organic structural changes noted in the course of the 'illness'! There are no physiological-pathological changes that can be correlated with these illnesses. There is no general acceptance that any form of treatment is of proven value, except sustained careful interpersonal relations and tranquillisation. 'Schizophrenia' runs in families, but observes no genetically clear law. It appears usually to have no adverse effect on physical health, and given proper care by others it does not cause death or fore-shorten life. It occurs in every constitutional type. It is not associated with any other known physical malfunction. (1970:17-18)

Perhaps the only definitive conclusion to be drawn from this weight of negative evidence is Schatzman's (1970) comment that the only thing certain about mental illness is that some people say that other people have it. Even if we ignore any implicit or explicit assumptions of a disease entity in Dohrenwend's definition, this is all he is really saying.

However, if the status of schizophrenia as a disease entity is taken as problematic, and if as Szasz, Laing, Leifer, Cooper and others have argued, psychiatric diseases are largely human conflicts, how can the psychiatrist remain aloof, and objective when asked to decide on the sanity of an individual

who presents a problem to the person referring him. For Szasz:

The answer is he can't. Thus while ostensibly acting as neutral scientists, psychiatrists are actually partisan advocates of one party to a conflict and opponents of another.

(Szasz, 1970:6-7)

By diagnosing disorder, the psychiatrist may oppose a person's self defined interests and support those with whom the patient is in conflict. We met this idea earlier with Leighton's conception of symptoms as adaptive responses to stress, which Dohrenwend argued may be thought of as maladaptive (and possibly labelled) by others. This process is most graphically illustrated in a comment by Cooper (1967) that if a prospective patient is interviewed with his family prior to commitment, very often it is not he who is committed, but another member of the family. Whose perspective do you accept? The impression given is one of a power game (Cooper speaks of "violence") in which the family seeks to define one of its members as mentally-ill (we will discuss the reasons for this in the following chapter) and seeks psychiatric legitimation for that label.

(ii) A satirical digression

Certain acts of an individual are immediately intelligible because they conform to a typically recognisable form and do not need special interpretation. Some actions, however are simply unusual in that they are components of everyday activity, their form is not sufficiently routine to be taken-for-granted, and a special explanation might be to impugn the sanity of the actor.

Laing, in his discussion of Kraepelin in The Politics of Experience illustrates this point by reversing the role of psychiatrist and patient. (See also the similar discussion in The Divided Self (1967:31-38). He quotes verbatim a passage from Kraepelin in which he describes a clinical examination -- "Gentlemen, the cases that I have to place before you today are peculiar". In this instance the patient is a young servant girl "aged twenty-four, upon whose features and frame traces of great emaciation can be plainly seen." Kraepelin demonstrates the patient's symptoms by the manner in which she responds to his actions. However when the roles are reversed and Kraepelin is no longer considered as psychiatrist, something rather curious occurs. Laing continues:

Here are a man and a young girl. If we see the situation purely in terms of Kraepelin's point of view, it all immediately falls into place. He is sane, she is insane; he is rational, she is irrational. This entails looking at the patient's actions out of the context of the situation as she experiences it. But if we take Kraepelin's actions ... -- he tries to stop her movements, stands in front of her with arms outspread, tries to force a piece of bread out of her hands, sticks a needle in her forehead, and so on -- out of the context of the situation as experienced and defined by him, how extraordinary they are!

(1967:89) (emphasis added)

As with Szasz (1970) the question for Laing becomes one of whose viewpoint we adopt, which perspective is legitimate, which definition of reality acceptable? While he avoids the question of why we don't think of Kraepelin's behaviour as mad without the artifact, he sensitises us to the problem of power referred to above. Kraepelin's definition is accepted because he has the power to impose that

definition, and legitimate that imposition with reference to the institution of insanity (or as Szasz (1970) would have it, the "ideology of insanity".) Without looking into the development of this 'psychiatric ideology', (see Szasz, 1970) it may be considered as a 'legitimizing system' in a sense similar to that of Berger and Luckmann (1963) which in this case provides a schema for an understanding of behaviour which breaks taken-for-granted assumptions about social reality.

However, reference to such a schema, which in this case explains the odd behaviour by reference to the disease process, tends to objectify that behaviour by ignoring the relationship between the psychiatrist and his patient. In the above example, Kraepelin acts towards the patient and then ignores, in his interpretation of the patient's response, the latter's interpretation of Kraepelin's original action. (Consideration which may lend intelligibility to those actions.)

In this instance, if Kraepelin's interpretation is legitimated by the institution of insanity, it represents, in Mueller's terminology, an instance of 'repressive communication' (1970:105). That is, an attempt to semantically structure the world of another, and effectively deny his interpretation of reality and attempts to locate himself in society:

Institutionally imposed communication is the matrix of meaning imposed on individuals subjected to total institutions be it an army or a psychiatric hospital. The individual's interpretation is temporarily suspended since it is not judged as corresponding to what is defined as reality by the institution.

(1970:105-106)

(iii) Mystification

As used above, repressive communication is very similar to Laing's (1967:119) use of 'mystification', essentially, an action performed upon another to defend one's own personality. A common form of mystification, as we have seen here, may be to deny that a person is responsible for his actions (or praxis) and attribute them to a disease process. Szasz (1970:196) has argued that in psychiatric nosology there is no such thing as action towards a goal, only behaviour which is determined by causes. This makes it a perfect institutional vehicle for institutional mystification. In the above example, by denying his patient's structuring of reality, and ignoring any interpretation of his patient's actions with reference to the situation as she experiences it, Kraepelin relegates her actions from intentional to behaviour caused by the disease.

Schizophrenia is a label attached by one person to another. Schizophrenia is a dehumanising event -- a person's behaviour is no longer regarded as independent and rationally directed but attributed or regarded as the product of some pathological process or processes. It seems that we use different sorts of explanations of behaviour depending on whether that behaviour is approved or disapproved. (Nettler, 1970:1) In "normal" everyday life it is customary to accept "reasons", "purposes" or "goals" as elements of an explanation of an individual's actions, whereas the explanations we would accept for abnormal behaviour tend to be phrased in causal terms. (Leifer, 1964)

In a similar vein, Peters, in his excellent discussion on motivation (1967) argues that causal questions are asked about 'peculiar goings on'.

These are usually cases of lapses from action or failure to act -- when there is some deviation from the purposive rule-following model, when people as it were get it wrong. ... In such cases it is as if the man suffers something rather than does something. It is because things seem to be happening to him that it is appropriate to ask what made, drove, or possessed him to do that. The appropriate answer may be in terms of a causal theory.

These cases of particular goings on which look like the breakdowns of action are very similar to a whole class of general activities which seem to have no point or a very odd point -- dreams, hallucinations, obsessions, anxieties and perversions.

(1967:10; emphasis added)

And this is very much what occurs in the recognition and diagnosis of schizophrenia. The patient's actions present a problem either to himself or more generally to others. The psychiatrist observes the behaviour to be disturbed in particular ways, or is told by others that the patient's behaviour is so disturbed. Unless he analyses that behaviour in the context of its occurrence he denies it intelligibility. He organises this behaviour, now 'symptoms', about the label 'schizophrenia' which means, as does his choice of any 'appropriate diagnosis', that the patient's behaviour and experiences are disorganised because there is something wrong with him which causes him to be disturbed. Having attached the label he must seek for a cause of or cure for 'schizophrenia'. Thus, as argued earlier, he does not seek to explain the behaviour, for after

all that was caused by the disease; but he may seek for the causes of the disease.

(iv) Topic and resource

One characteristic of sociology is that it is rooted in everyday life, which not only furnishes the context of sociological explanation, but also a leading conception of "its order of fact and program of research". (Zimmerman & Pollner, 1970) To quote Zimmerman and Pollner

Sociological enquiry is addressed to phenomena recognised and described in common-sense ways (by reliance on the unanalysed properties of natural language) while at the same time such common-sense recognitions and descriptions are pressed into service as fundamentally unquestioned resources for analysing the phenomena thus made available for study. (1970:81)

Zimmerman and Pollner, term this a confounding of topic and resources, that is, rather than examining the manner in which everyday definitions and explanations are constructed and used by members of a social context as routine grounds for their everyday activity, the sociologist accepts such definitions as the topic of his enquiry, thus assuming the stable properties of the social world, rather than using them as a resource to examine the manner in which the world is given stability in ongoing interaction. Thus rather than accepting a member's definitions of another as being "schizophrenic", "mentally-ill", "strange", Zimmerman and Pollner urge that these "facts" should be treated as ongoing accomplishments by which parties to a setting, regardless of its substantive character make that setting available to one another as the kind of setting they take it to be.

Consequently, (a) without making judgements as to the existence or not of a disease entity 'schizophrenia', we can discuss the consequences of such a diagnosis, as defining a relationship, in the establishment and maintenance of a definition of a setting or situation; (b) with reference to the labelling approach, to which we will now turn in more detail, by examining the practical activity as an ongoing process by which actors give meaning to their relationships, we move away from the notion of mental-illness as deviance to one in which the attribution of a mental-illness label is essential for the maintenance of stability in situational definitions.

b/ Labelling, or what's in a name

Siegler and Osmond (1966, 1971) in their continued formulation of models of madness, have isolated in the work of Goffman (Asylums) and latterly Laing, what they term a "conspiratorial" approach to madness. The model

has as its main concern the violation of the rights of the person labelled as schizophrenic. Since it is derived that the person so labelled has an illness, his incarceration in a building called a "hospital" is inexplicable. And so it is said that there is a conspiracy among those surrounding the "patient" to exile him to a total institution which is called a hospital but is really a kind of concentration camp.

(1971:88)

The approach is epitomised in Szasz' work (1961, 1968, 1970), for example

Both psychiatry and law are concerned with defining which roles are socially legitimate and which are not, and with enforcing conformity to prescribed roles. Institutional psychiatry enforces role conformity by defining role deviance as mental illness punishable by commitment. When, for example,

a poor, uneducated, overburdened housewife escapes from her life of drudgery into the pretense that she is the Virgin Mary, the psychiatrist calls the woman sick and thus interferes with her playing the role she has selected for herself. This type of prohibition is buttressed by the sanction of confinement in a mental hospital, is similar to the prohibition of the role of bank robber, buttressed by the sanction of confinement in prison.

(1970:102)

However, the most systematic approach to the problem has been that of Scheff (1966), who has developed a sophisticated model of mental illness as deviance, and it is his work we will concentrate on here, and especially the empirical work it has stimulated. However, before looking more closely at Scheff's work it is important to outline some of the important theoretical concepts of the societal reaction approach.

The approach is essentially nominalistic, which is reflected in Scheff's adoption of Becker's concept of deviance, one, which if applied to mental illness, is fundamentally at variance with the idea that schizophrenia can admit of psychiatric nosology. The importance of the approach is that it focuses not on the acts themselves but on the quality of people's response to these acts. It is worth quoting Becker's concept of deviance here:

Some groups create deviance by making rules whose infraction constitutes deviance, and by applying those rules to particular people and labelling them as outsiders ... deviance is not a quality of the act the person commits, but rather a consequence of the application by others of rules and sanctions to an "offender". The deviant is one to whom that label has successfully been applied; deviant behaviour, is behaviour people so label.

(Becker, 1963:9; quoted in Scheff 1966:32,
Scheff's emphasis)

A deviant is a member of a social category and on this basis is assigned a particular role by enforcement institutions. This role defines his relationship with others, and the forms these relationships may take. (Goffman, 1968) For example, being a homosexual is not the same as being somebody who prefers sexual relations with the same sex.

Deviance within this framework is considered as rule-breaking activity, and the kinds of deviance which it suits best are those for which there are definite legal, moral, or conventional rules which define the deviant act. With mental illness the rules or norms which define the symptoms of mental illness are more difficult to specify. (Szasz does away with the problem by viewing the "symptoms" of mental illness as motivated by features of the role of patient as a constituent part of the medical institution. Much in the spirit of Goffman, 1968; and Braginsky et al, 1969) Scheff attempts to specify what behaviours qualify an individual for the role by suggesting that mental illness constitutes a residual category of deviance; that after all the various types of deviance have been named and identified with respect to known rules or norms there remains residual deviation which cannot be fitted into any known category. Scheff terms "residual rules" rules which are taken for granted by a group, a violation of which causes the infractor to be thought of as strange, bizarre, and somewhat frightening; a threat because the behaviour violates the assumptive world of the group and thus threatens the natural order

of things:

the diverse kinds of rule-breaking for which our society provides no explicit label, and which, therefore, sometimes lead to the labelling of the violator as mentally ill, will be considered to be technically residual rule-breaking.

(1966:32)

Individuals do not become deviants until their behaviour is so labelled, and the significant question is not the understanding of rule-breaking as such; because according to Scheff's first two propositions everybody, at one time or another, breaks residual rules;

1. Residual rule-breaking arises from fundamentally diverse sources. (1966:32)
2. Relative to the rate of treated mental illness, the rate of residual rule-breaking is extremely high. (1966:47);

The significant question is to understand how an individual comes to adopt a regular pattern of behaviour which is rule-breaking.

This constitutes the most fundamental distinction made by

labelling theorists, that between primary and secondary deviation.

To quote Lemert:

Primary deviation is assumed to arise in a wide variety of social, cultural, and psychological contexts, and at best has only marginal implication for the psychic structure of the individual; it does not lead to symbolic re-organisation at the level of self-regarding attitudes and social roles. Secondary deviation is deviant behaviour, or social roles based upon it, which becomes a means of defence, attack or adaptation to the overt and covert problems created by the societal reaction to primary deviation.

(1967:17)

Primary deviance is the behaviour which provokes a deviant label, while secondary deviance may be the response, a stable

rule-breaking career, which the individual makes to being placed in the deviant role. The imputation of a psychiatric label has implication for the identity of the person so labelled, (see Garfinkel on 'degradation ceremonies', 1967). A label defines the expectations made of another's behaviour "by suggesting that individual behaviours are to be taken as documents or indicators of some underlying essential quality of the self". (Hughes and Marshall, 1971:5) In this instance the disease label denies the intentional character of an individual's behaviour. The imputation of such a label, in Garfinkel's terms, serves to reorganise an individual's biography, in terms of our interpretation of our previous experience of him, and our response to his presented behaviour. Faced with this definition of himself as not a normal person, the individual is faced with two choices. Either, accept the label in the absence of alternative modes of behaving or because it is rewarding to him as Scheff argues:

The individual plays his role by articulating his behaviour with the cues and actions of other persons involved in the transaction. The proper performance of a role is dependent on having a co-operative audience. The proposition may also be reversed: having an audience act toward the individual in a uniform way may lead the actor to play the expected role even if he is not particularly interested in doing so.
(1966:56)

The essential point about the above quotation which we will take up in more detail below, is that this does not have to be a violent, or overt process, which is the impression given by Siegler and Osmond's use of the term "conspiratorial". Thus rather than

considering the process as one in which an individual is forced to play an unwanted role, it may represent an accommodation between actors, or as Scheff terms it an "articulation".

The second response an individual can make to the labelling process is to attempt to resist it, or rationalise the behaviour as an index of something else,

proposition (3) Most residual rule-breaking is "denied" and is of transitory significance.

(1966:51)

If it is possible to deny the behaviour, or resist a label, it does not organise into a stable role. However, fighting off the label engages the individual in a power-game in which the individual must organise aspects of his life to resist the mental illness label. The ability of an individual to resist a label depends upon the power of that individual and conversely the ability of other's to impose a label depends on the power they are able to muster. Once the label is applied and confirmed (by a social agent) Scheff expects that the individual forced or coerced to play a role will alter his self-concept or identity in keeping with his behaviour (see Hughes and Marshall 1971 for a more complete discussion of this process).

This in very general terms is the labelling approach of
 1. Scheff, and, in broad terms, it is not that different from Dohrenwend's definition outlined above. The difference is essentially one of emphasis, in that the labelling approach after Scheff, explicitly rejects the notion that we have been dealing with so far, that there

is something fundamentally different about psychiatric cases, which sets them apart from normal people. Dohrenwend does not face this issue. The crucial moment is the act of "labelling" which serves to organise an individual's behaviour into a 'deviant' role. To quote Scheff:

... under what conditions is residual rule-breaking stabilised? The conventional answer lies in the rule-breaker himself. The hypothesis suggested here is that the most important single factor (but not the only factor) in the stabilisation of residual rule-breaking is the societal reaction. Residual rule-breaking may be stabilised if it is defined to be evidence of mental illness, and/or the rule-breaker is placed in a deviant status.

(1966:54, emphasis added)

The quite radical nature of this perspective is reflected in this quotation from Lemert in which the focus is shifted away from the disorder as a property of the individual, to an understanding of that behaviour as an accommodation to the concerted actions of others.

The general idea that the paranoid person symbolically fabricates the conspiracy against him is in our evaluation incorrect or incomplete. Nor can we agree that he lacks insight, as is so frequently discussed. To the contrary, many paranoid persons properly realise that they are being isolated and excluded by concerted interaction, or that they are being manipulated. However, they are at a loss to estimate accurately or realistically the dimensions and form of the coalition ranged against them. (1962)

Thus paranoia is not considered as an illness, although the individual's behaviour may appear strange, but as the individual's interpretation of his social reality. And as such, his actions and experience are understandable only if taken in context of this social reality. The application of the disease label serves

to deny the veridity of the individual's experience, and substitute another's interpretation of it.

(i) Empirical research

The implications of this latter statement will be discussed in more detail below. In this section the concern is with the official process of conferring a psychiatric status. One of the important assumptions involved in the use of treated cases as indices of true disorder is that other factors should not be involved if the presence or severity of disorder is the sole criterion of diagnosis or commitment. (That is, of course excepting the nosocomial factors which can be accounted and allowed for.) The critical feature of the labelling approach which distinguishes it from a medical model of disorder is in its treatment of power which is taken to be the significant feature in the ability to attribute or resist a mental illness designation. As such it provides a useful explanatory adjunct to studies such as that of Hollingshead and Redlich (1958) who show that the type of treatment received and diagnosis made may be dependent more on the social-class of the recipient than the type or severity of his disorder. Similarly Myers, et al, (1968), found the same class bias in the speed of treatment and release in the Ten year follow-up of the Hollingshead and Redlich study. It would be tempting to interpret these results in power terms, which would, of course be tautological in the absence of specific analysis of the power dimension, however the labelling approach does provide

a perspective within which to examine such discrepancies and while it is not possible to interpret the material referred to in these terms there is evidence from other sources to suggest that such an interpretation is legitimate and possibly fruitful if the specific data were collected.

(ii) Involuntary patients

The situation concerning involuntary patients is by no means clear and, as in all things interpretation of the data is coloured by one's particular perspective. Thus, within a "conspiratorial" framework it is questionable how many patients are in fact voluntary when, for example, in Ontario it takes only the signature of a single doctor to commit a patient involuntarily, and once in the hospital as a voluntary patient, it is a relatively simple matter to convert a voluntary to an involuntary status. Gove (1970:877 & 877n10) in his critique of the labelling approach, quotes figures from Mishler and Wexler (1963) and Mandel and Rapport (1969) which indicate "that public mental hospitals only admitted 40% of the voluntary applicants". These figures are quoted as an argument against Brown (1961) and Mechanic (1967) who Gove suggests "feel that public mental hospitals accept virtually all such patients". This is a considerable distortion of what Mechanic, in fact, argued. Mechanic is explicit in stating that his conclusion is limited to the two hospitals he studied, and in linking the high degree of commitment to nosocomial factors. That is, given the availability of beds "it is likely that they will absorb whoever appears, at

least for a time". (1967:28) However, the main point of Mechanic's paper is the importance of community and lay definitions of psychiatric disorder intervening prior to the actual psychiatric screening process. And much in line with Scheff (1966:105-155) he argues that individuals are brought to the hospital or present themselves on the basis of lay definitions, "and once they arrive, their appearance alone is usually regarded as sufficient evidence of illness". (1967:27)

As commented above, the interpretation of the data depends upon which perspective is accepted. What is important is that the initial recognition of disorder is performed in the community, and to all intents and purposes as Blum argues:

It is likely that the psychiatrist will concur in nearly every case with the self or community diagnostic criteria, for one suspects that the incidence of persons who come to the psychiatrist seeking treatment but are turned away with a diagnosis of 'no illness present' represent but a small fraction of those who apply for care. Unless folk criteria are more perfect than the evidence would lead us to believe, it would appear that the psychiatrist's self-referred case finding encompasses a variety of folk criteria for the identification of mental disorder.

(1962)

Blum's position is similar to that of Dohrenwend discussed earlier, the essential feature being that individuals present problems for themselves or others which may come to be labelled as evidence of psychiatric disorder. The crucial problem is not in examining the frequency of voluntary commitment, but in examining the factors which precipitate an individual's seeking help, and/or the pressure brought to bear on him to seek help.

One approach to this has been the examination of public stereotypes of the mentally ill (Nunnally, 1967; Cumming & Cumming, 1957; Rootman, 1969) which seems of limited importance for our

purposes. These studies generally revolve around the presentation of a fictitious description of persons with various types of psychiatric disorder for evaluation by a particular group or member of a community (see the descriptions formulated by Star, 1955). In general it has been found that persons are not judged to be mentally ill except when their behaviour is said to be dangerous, or as Phillips has pointed out by their being labelled as mentally ill by receiving treatment (Phillips, 1967). However, there is a considerable difference between these stereotypes and the actual experience of dealing with somebody whose behaviour may draw an illness label particularly when one is personally and emotionally involved with that person. Since LaPiere (1934, Cook & Sellitz, 1967) we have been aware of the difference between stated attitude and actual behaviour, and similarly the recognition of a stereotyped symptom description, the ability to recognise such behaviour in everyday life, and the possibility of acting on such knowledge are entirely different things. Gove's discussion of this point is somewhat confused (1970:877) for he attempts to extrapolate from the studies using symptom descriptions to the processes of commitment themselves. It seems erroneous to argue, as he does, that the evidence

strongly suggests that persons, typically, are hospitalised because they have an active psychiatric disorder which is extremely difficult for themselves and/or others to handle. It would appear that the public stereotype of mental illness does not lead to persons being inappropriately labelled mentally ill through an inadvertent act of residual rule-breaking.

On the contrary, an equally plausible conclusion is that the public stereotypes have little to do with the processes of commitment. Thus, decisions to refer are not made on the basis of stereotypes but in terms of the perceived personal and interpersonal disorganisation of the individual. Indeed, Gove's discussion of Yarrow et al (1955) bears out this point. Rather than argue, as he does that the "gross exaggeration of the degree and type of disorder in the stereotype fosters the denial of mental illness, since the disturbed person's behaviour does not usually correspond to the stereotype" (1970:877), which conclusion has no basis in the material he reviews, it would be more useful to examine the human context of referral and commitment, and the processes and pathways involved in the progress towards becoming a case. This involves not the public stereotype, but the interpersonal context of a decision to refer. Perhaps the most telling comment is that of Cumming and Cumming (1957:102) that mental illness is a condition "which afflicts people who must go to a mental hospital; but up until they go almost anything they do is fairly normal". (See Phillips, 1967; Yarrow et al 1955)

The majority of research in this perspective has been done into the process of involuntary commitment (Scheff, 1966:128ff); unfortunately much of this research is limited in scope and sampling, and in some cases the conclusion drawn is a matter of emphasis or interpretation. However, given the problematic status of the disorder, it is useful to examine this material in terms of a labelling approach for evidence of a power dimension in the decision

to commit. Of the 83 cases, 73% for whom appointments had been made were committed, and only 23% of those for whom no prior appointment was made were committed. If a non-psychiatrist made the request 33% of the cases were approved. The comparable figures for psychiatrist and court psychiatrists were 98% and 100% respectively. These findings may be interpreted in two ways. As Gove (1970:879) does, in terms of meticulous initial screening which prevents the commitment of individuals who are not ill; or, in labelling terms, as evidence of a power game in which the defining power of a psychiatrist is used to reorganise an individual's biography. It is not possible to decide either way, except to suggest that there is no support for Gove's conclusion in the data presented, and that the author's admittedly crude control for severity would suggest that there are factors other than disorder operating in this instance.

More interesting for our purposes is the evidence presented by Wenger and Fletcher (1969) on the presence of lawyers at commitment proceedings. In the hearings examined it was the practice to have a referee preside while two psychiatrists examined a patient and gave their opinion. The referee was not a psychiatrist. When only the psychiatrists were present the judge invariably followed the psychiatrist's advice. When the patient was represented by a lawyer the mean time of the hearing increased from 6.15 to 16.84 minutes ($n = 81$) and unlike the decisions without lawyers present, of 15 people with legal counsel only 4 were committed; by contrast, of the 66 persons without counsel only 5 were not committed. The authors did

attempt to control for the severity of illness on the basis of their own observations (essentially placing themselves in the position of the lay referee, see Hughes & Marshall, 1971:14) dividing the cases into those meeting legal requirements for commitment; borderline; and those not meeting the legal criteria. Of the cases present at the time of the hearing (n = 72) only 27% were classified as meeting the legal requirements, 43% borderline, and 30% as not meeting the criteria. In all categories the presence of a lawyer decreased the risk of commitment, indicating that the labelling of an individual may be partially independent of the presence or absence of disorder.

As argued earlier, if commitment depends solely on the presence or absence of diagnosed disorder other factors should not be involved. The presence of legal counsel seems important in some decisions to commit, and as Haney et al have shown other factors may be involved.

Haney et al (1969; see also Haney & Michielutte, 1968) have provided the most sophisticated (though small, n = 127 from four Florida counties) examinations of the interaction between the characteristics of the petitioner and deviant in adjudication proceedings. Unfortunately, it was not possible to control for severity of disorder. There is no need to list their findings here. For our purposes, it is interesting to note their general conclusion that:

- (1) status distinctions may be of some importance in influencing the outcome of adjudication proceedings, and
- (2) a given characteristic of the petitioner may

interact with the same characteristic of the alleged to enhance or depress the probability of being adjudicated incompetent. (1969: 188-189)

The interesting feature is that the majority of the variance is explained not in absolute terms, i.e. in pure status distinctions; but in the interaction between the characteristics of the petitioner and adjudged incompetent. (1969:191) The importance of the study is that it highlights some of the non-medical and non-legal factors associated with commitment, in that the decision to commit is not "simply" a matter of the presence or absence of disorder, or fulfillment of the criteria of dangerousness to self or others; but confounded by this interaction between petitioner and incompetent characteristics.

Haney et al's conclusions revolve around the notion of "marginality". Again what is important is not the disorder:

The relationships found here all share a common element in that they imply that the alleged incompetent is restricted to a marginal role in the family or society at large. Perhaps it would be more appropriate to say that the alleged incompetent who is most likely to be declared incompetent is the one who has become a liability to those around him.

(1969:192)

This idea should be familiar from our discussion of Rogler and Hollingshead, Goldberg and Morrison, Turner and Wagonfeld and Dunham above (p. 62 f). In these studies, the individual is one with marginal role in his family and society, one who is liable to become a liability to others.

Haney et al's study is important because it illustrates that adjudication may rest on this factor, that is the medico-legal definition of illness may be dependent on factors other than actual illness. Thus rather than asking questions as to whether

the marginal status causes the illness, which has been the next step to such a finding in epidemiological studies, one is directed towards different questions not of etiology but, outside the medical realm, of examining the human context of the marginal individual.

Thus marginal is not used in the same sense as Stonequist's usage, which would predict it as a possible cause of mental disorder, but in terms of interaction and the individual's ability to resist definitions of his reality. (Here Haney et al's use of "liability" is interesting. They might well have used dangerousness which would have fit well the standard commitment criteria. "Liability" does not, however, connote the idea of the labelled individual threatening the concerted reality of the group as a whole.)

The conception of power drawn from Haney et al's study is not one of raw status opposition; but a much more complex process occurring in the interaction between the petitioner and the incompetent prior to the initiation of commitment proceedings. (Interaction here is used to refer to social, and not statistical interaction.) It must be emphasised that this statement is tentative, based on the tentative evidence which Haney et al present; but it does suggest a different line of enquiry, one which is consistent with Scheff's approach; but with different emphases.

There is nothing to suggest, in the studies reviewed, that it is only those who have severe disturbance who come to the attention of psychiatric screening agencies, which is Gove's conclusion after reviewing similar material. (1970:879) Indeed it is difficult to see how he reached this conclusion in a paragraph following an acknowledgement that Miller and Schwartz (1966:34) found that "the

judge reversed the medical recommendations for commitment ... in nearly one fourth of the cases". This suggests that factors other than diagnosed disorder influence decision to commit.

c/ Accommodation and articulation -- the normalised family

This last section may have seemed like a running dialogue with Gove (1970) and to a certain extent this was intentional. In his two papers Gove has presented one of the most systematic critiques of labelling theory, one, indeed, which has drawn support from Mechanic (1970) and Dunham (1970). However much of Gove's attack seems misplaced because of a misunderstanding or misinterpretation of the focus of enquiry.

The important feature of the societal reaction approach is that it explicitly recognises that factors other than the presence or absence of disorder are involved in the risk of an individual becoming a case; and it is in their discussion of the various dimensions of the 'labelling power-game' that they provide a useful explanatory adjunct to the studies we referred to earlier. However, the critique goes far deeper and questions the very concept of the disease itself when it is argued that the societal reaction may be the important factor in the stabilisation of secondary deviance. Thus, the focus of the societal reaction approach is shifted away from questions as to why one individual commits the initial breach of "residual rules" to the significance of the societal reaction to that breach, and its consequences for the individual, in terms of secondary deviance.

Much of the criticism of the approach has stemmed from a misinterpretation of this focus, specifically when it is interpreted as concentrating on the official labelling of 'residual rule-breakers' on the occasion of their commitment or treatment. Much of this criticism is legitimate given the concentration of the relatively little empirical research. As Gove (1970) argues, there has been little systematic empirical testing of the labelling model, and that which has been done has concentrated almost exclusively on the official act of labelling and its consequences, the effects of institutionalisation and possible stigma associated with the ascribed deviant role. ("Ascribed" because it is not a property of the individual.)

If interpreted in this way, the focus has little to say in questions of etiology, the question we were asking of the epidemiological studies. Such a question is not asked, and is illegitimate given the focus. The labelling approach does have important implications for epidemiological studies, by questioning the validity of the psychiatric decisions the epidemiologist takes as his cases; but it is perplexing to attempt to argue from studies concentrating on the official labelling process in which the behaviour which is defined as a problem is to all intents and purposes ignored. Given this perplexity it is relatively simple to reach the conclusion Gove does that:

the societal reaction does not explain why people initially commit the deviant act: it deals mainly with the secondary processes that may not always be of crucial importance. Just as focusing only on the processes involved in producing primary deviance may lead to an unrealistic image of deviant

behaviour, so, also will an exclusive focus on the societal reaction to an act of primary deviation. (1970:882)

This, of course, is a misinterpretation of Scheff's position. As we saw earlier, the critical variable is the audience rather than a property or act of the individual (Erikson, 1963:11); however, Scheff (1966) in his book, and earlier paper (1963) stresses that the theory tends to overemphasise the social processes by holding constant individual differences, and individual dynamics. Indeed, the stated purpose of Scheff's book is to stimulate discussion of alternative approaches:

In the present discussion of mental illness, the social system model is prepared not as an end in itself, but as the antithesis to the individual system model. By allowing for explicit consideration of these antithetical models, the way may be cleared for a synthesis, a model which has the advantages of both the individual and social system models; but the disadvantages of neither.
(1966:27)

Scheff does not ignore 'primary deviance', or the reasons for the initial rule breaking; but his concern is with the reaction to it and stabilisation of the "deviant career" (Becker, 1958). One may not agree with Scheff's explanation of the initial act (1966:32ff) but it is a misinterpretation to argue as does Gove, that he does not take account of primary deviation.

The ideas expressed by Gove (above) represent a fundamental misinterpretation of the labelling approach: individuals do not "commit deviant acts", there is no such thing as a "deviant act" within the perspective -- they break "residual rules" (behave bizarrely, shatter the taken for granted); and their rule-breaking may be labelled as deviant by another. The concern is with factors associated with the labelling of residual rule-breaking; not with

arguing that certain factors cause residual rule breaking. In this sense Gove's criticism is misplaced -- he does, however, raise (at least obliquely) a problem which is inherent in the approach. That is, it does not give sufficient attention to the stabilisation of residual-rule breaking prior to the official labelling of an individual's actions as evidence (symptoms) of psychiatric disorder. This statement needs clarification.

It is important to distinguish between the official act of labelling, which confers the social role of mental illness on an individual, and the more insidious process Scheff refers to as the "articulation" of role performance. In this latter process the "labelling" is far more subtle, and consists not in arraigning an individual before a psychiatric agent; but of acting towards him in a uniform, concerted manner, thus limiting the range of alternatives open to him and leading him to perform an unwanted role. Scheff adopts Szasz' use of type-casting here (1966:57) and argues that while an individual may not agree with the type he is cast into, he may, nevertheless incorporate elements of the type cast role into his own self-conception and ultimately his own behaviour.

The "baby of the family" may come to find his role obnoxious, but the uniform patterns of cues and actions which confront him in the family may lock in with his own vocabulary of responses so that it is inconvenient and difficult for him not to play the part expected of him. To the degree that alternative roles are closed off, the proffered role may come to be the only way the individual can cope with the situation.

It is absolutely crucial to recognise, at this point, that this

stabilised pattern is not secondary deviance. There has been no official labelling, and the interaction described represents a stable accommodation of behaviour to the expectations of others. It becomes deviance if it is labelled as such by a social agent. It is important to make this point clear because it serves as the basis of one of the writer's criticisms of the labelling approach (particularly with the notion of deviance). This type of accommodation will be called here 'normalising', in a sense which is different than that use of the term by Sampson et al (1962) and Yarrow et al (1967:33). In the sense used by these writers 'normalising' is essentially coping behaviour, for example Sampson et al comment on the extraordinary ability of the family to cope with the deviant behaviour of a member who is destined to become a mental patient, and Yarrow et al analyse the "cognitive and emotional problems encountered by the wife in coping with the mental illness of the husband".

By contrast 'normalising', will be used here to refer to the stable articulation of roles within the family referred to by Scheff. The distinctive feature of a 'normalised' family pattern, which distinguishes it from a normal family pattern is that if the actions of one of the members are taken out of the context of the interactions within the family, they may be thought of as unintelligible or meaningless and thus deserving of a psychiatric label.

In a certain sense we are taking "normalising" to mean

something similar to Lidz' concept of folie à famille:

In some (families), the dissatisfaction and unhappiness of one spouse is apparent to the other and to the children, but husband and wife complement or support each other sufficiently to permit a degree of harmony. In others, the distorted ideation of one partner was accepted or shared by the other, creating an atmosphere of folie à deux, or even of folie à famille when the entire family shared the aberrant conceptualisations.

(1968:658 - Bell & Vogel)

We mention it here to avoid confusion of the two terms. Normalising differs fundamentally from Lidz' use of folie à famille in that there is no assumption of psychopathology in one of the members of the normalised family. While it is not excluded, it is not taken as an assumption, and for our purposes we take normalising to mean the concerted actions of the family (or any other significant group -- in which there is emotional involvement) to deny the experiences or meaningfulness of the actions of one of its members. We will go into this in more detail in the following chapter.

'Normalising' is considered as an act of 'mystification' in the sense discussed above (p. 109 ff), and within this context, the "unmanageable emergency" of which Sampson et al (1962) speak which precipitates commitment, will be considered as an act of realisation and an attempt by the "patient" to radically reorganise his relationships to the others, and consequently to retain (or regain) his identity.

The application of a label (diagnosis) and the subsequent act of commitment are further acts of 'mystification' denying the individual's ability to act for himself and determine his individual

identity and integrity, by attributing his actions to the process of a disease.

d/ Mental illness and labelling

This model is considerably more complex than those we have been dealing with so far. The significant feature is that different responses to the same action may have entirely different consequences for the individual and for those with whom he interacts. It is necessary to make three distinctions here which will aid the reader in understanding this approach:

- (a) normalcy refers to the customary patterns of action by an individual or within a family (or significant group) which are not considered as abnormal by those with whom they interact outside the family group:
- (b) normalising (literally the act of making normal, Lemert, 1967) refers to the patterns of accommodation amongst family members which if taken out of this context seem bizarre and irrational; but which are understandable and normal as accommodations within that group:
- (c) deviant which refers to the official labelling of an action and its attendant consequences in terms of secondary deviance. (Lemert, 1967)

Within Scheff's schema, normalising may occur in response to an act of residual-rule-breaking which is responded to in a uniform manner by members of a significant group. Although from his

argument (1966:56ff) it will be apparent that residual rule-breaking is only a peripheral issue, in this instance Scheff is concerned with isolating the 'causes' of the mental illness role ('causes' even though they do differ significantly from causes within a medical model) and hence the stress he places on "residual rule-breaking". The concept of "residual rule-breaking" is essential if mental illness is to be fitted into the societal reaction framework, for as we argued above, one problem in the application of the approach in this particular instance is the lack of a systematic 'code' of rules whose infraction would constitute deviance -- hence residual-rules whose breach constitutes psychiatric symptoms (see Scheff's discussion of Goffman's "away" and Hebb; 1966:34ff).

The most important aspect of the 'societal reaction approach' is the societal reaction itself. The mental illness role is not so much a response to residual-rule breaking; but a situated accomplishment by which members of a family (or other significant group) explain the actions of another. What seems to be important are not so much the rules which are broken; but the societal reaction to what members define as rule breaking and their imposition of an explanation on another's actions.

This reaction may occur at any number of levels. As Scheff uses the term, he refers to the creation of deviance by an official societal reaction which confers membership of a socially deviant role. This role attribution, as Scheff argues has important consequences

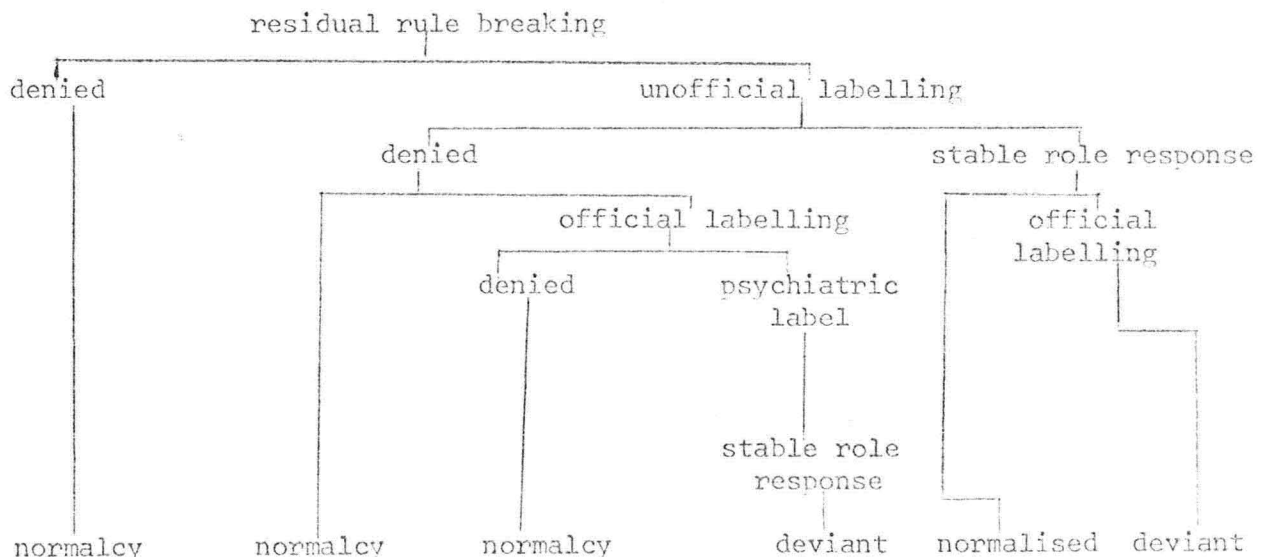
for individual identity and subsequent behaviour.

Thus, the social role of the mentally ill has a different significance at different phases of residual deviance. When labelling first occurs, it merely gives a name to rule-breaking which has other roots. When (and if) the rule-breaking becomes an issue, and is not ignored or rationalised away, labelling may create a social type or pattern of "symptomatic" behaviour in conformity with the stereotyped expectations of others. Finally, to the extent that the deviant role becomes part of the deviant's self conception, his ability to control his own behaviour may be impaired under stress, resulting in episodes of compulsive behaviour.

(1966:92)

However, it is not necessary to limit this process to the societal reaction; at another level, it is possible to examine the stabilisation of potentially deviant patterns of action in terms of the concerted reaction of a group to one of its members. While Scheff mentions this point he does not explore it fully, and hence the misinterpretations by Gove. While reaction on this level may produce behaviour which may be labelled as deviant by a psychiatric functionary, until it is so labelled it remains a "normal" pattern of response within that particular family i.e. "normalised".

Thus we may represent the model schematically:



This model differs somewhat from that given by Scheff, although derived from it, in the emphasis it places on normalised patterns of role response. As interpreted here, the societal reaction is taken to refer to the concerted actions of others in structuring the social world of an individual; in this instance of denying the rationality of his actions and attributing them to the disease process. While it is given paramount position in the above diagram, the breaking of residual rules will be treated as a peripheral issue, for what is important is not this initial act, if it ever occurs, but the concerted actions of others to what they define as acts of "residual rule-breaking". We noted in our discussion of cultural stereotypes that it is important to examine members' actions in referral decisions; here we will consider them as situated accomplishments by examining the human context of the labelled individual, and the manner in which members of such a significant group achieve definitions of disorder.

e/ Conclusions

We began this chapter by examining the conceptions of schizophrenia and mental illness used in the epidemiological and field studies. This discussion was precipitated by the analysis of the sociopsychological explanations of rate differentials. It was argued that questions about the rate of certain social or cultural factors which precipitate or predispose the "illness" are better asked on a sociopsychological level at which the experience and behaviour of the individual in question could be examined, and the significance of the various 'etiological factors'

identified in the epidemiological studies be determined for the individual. However, the solution of such questions was hampered by an uncritical acceptance of a medical model of illness reflected in the use of reported rates in the hospital studies, or the psychiatric diagnosis of a protocol in the field studies.

It was necessary to make a distinction between factors associated with the presentation of 'psychiatric symptoms' and factors associated with commitment and treatment. The discussion of the empirical work associated with the labelling approach legitimated such a distinction by suggesting that factors other than the presence or severity of disorder are associated with commitment or referral, and hence the possibility of becoming a case. This finding casts severe doubt on the utility of treatment statistics as indices of actual disorder, and restricts studies using such data (as in Hospital records) to explanations at the level of rates (for example in terms of the modified drift hypothesis, (p. 65) and precludes questions of etiology.

It was in the discussion of the field studies (p. 85) that the question of labelling first arose. Here it was argued that the response to stress may be either adaptive or maladaptive depending upon the inconvenience felt by the individual, or the response of others to his coping behaviour. Mental illness is thus that which is labelled mental illness, and within Leighton's perspective, many individuals function successfully in society with impairment which

if brought to the attention of a psychiatric agent would be treated as evidence (symptoms) of a mental disease.

This approach still accepts the concept of mental illness as a disease entity which affects the behaviour and experience of an individual. It was in the discussion of the 'societal' reaction approach that we questioned specifically this disease entity concept. After Laing, it was argued that in the absence of any definitive evidence of the existence of such a disease its status should be treated as problematic. The particular methodology employed in the epidemiological studies is admirably suited to the analysis of irrational behaviour in which the individual is regarded as subjected to external pressures over which he has no control. With the status of schizophrenia treated as problematic the methodology employed should be capable of distinguishing between rational and irrational behaviour. The quotation from Lemert (p. 115) highlighted the importance of considering the individual labelled as a case as the member of an interacting group. In this way, what may be interpreted out of context as irrational, paranoid delusions may be thought of as quite rational interpretations of the individual's social experience.

Here the concern is not with the factors associated with commitment, but with the presentation of labelled psychiatric symptoms. The distinction between these two levels thus seems legitimate particularly when the societal reaction approach is extended to refer to the stabilisation of patterns of action prior to commitment. Thus, while Scheff, Goffman, Lemert and others are

concerned with examining the stabilisation of secondary deviance after the application of a deviant label, the concern in the remainder of this work is not with the official labelling process; but in examining and exploring the accommodation an individual may be obliged to make to the concerted actions of others in a family or other significant primary group; particularly in the stabilisation of customary patterns of action which while 'normal' to the group may come to be labelled as psychiatric symptoms if the actions of one of the members is taken out of the context of the interacting group.

With this in mind, attention now turns to the family studies whose importance we mentioned earlier.

...Before I treat a patient... I need to know a good deal more about him than the patient himself can always tell me. Indeed, it is often the case that my patients give only pieces of a total situation which I have to explore. The single patient who is ill by himself, is rather the exception.

T. S. Eliot, The Cocktail Party. Harcourt, p. 114.

The criticisms made of the epidemiological literature and the ideas developed in the previous chapter suggest a radically different approach to the sociology of mental illness. If we accept the status of schizophrenia as problematic, then the task of sociological research becomes that of relating an individual's labelled 'pathological' experience to his social experience. That is, we are not concerned with establishing the causes of an illness which in turn cause behaviour and experience to be distorted; but rather we are concerned with placing the individual's actions within an interactive framework to establish the reasonableness of those actions in much the same manner as Lemert quoted above (p. 115). Thus our concern is not with etiology or causes or why a particular event occurred, but with that event as part of a sequence of interaction and with examining its intelligibility as such. This can only be achieved by foregoing judgements as to the rationality or accuracy of an individual's perceptions and experiences until it is established whether or not this experience is a reasonable interpretation of the manner in which

relevant others of his social context are acting, and have acted towards him.

This point we raised earlier, and it does seem to highlight the critical distinction within the labelling approach which has been obscured by its empirical "spin-off", and open avenues for an understanding of the stable patterns of action which may draw a psychiatric label. As we shall see, it does not make sense to ask questions of etiology within this focus unless one enquires into the process of stabilisation of the accommodation between individuals; and then questions of etiology may be entirely inappropriate. Similarly, it does not make sense to localise psychiatric disorder as an illness within a particular individual, for if the patterned actions can be shown to be an accommodation to the actions of others then the "problem" is with the system itself, and as such this latter should be the focus of treatment and research. Only by examining this interacting system as a whole is it possible to understand the experience of any one of its members, and thus obviate the objectification of the actions of an individual occasioned by the attribution of a disease label.

This perspective does not, it must be emphasised, preclude the possibility that there are individuals who are 'mad'; but it does preclude using as indices of the disorder hospital statistics, or accepting uncritically a psychiatric diagnosis. The critical question appears to be that raised also by Esterson (1970:230-231) of distinguishing between individuals who are labelled as 'mad', and

those who are mad by any criteria (whether labelled or not).

There is, of course, considerable difference between being labelled mad, and being mad. Some labelled schizophrenics are mad by any criterion that I know. While some in my experience are not, but have been mystified into believing they are. And some have been driven frantic as if they were mad.

(1970:231)

Esteron does not go into the question of what it is to be really mad (although one suspects the treatment would not be unlike Laing's Divided Self). He does however place it squarely within the framework developed here:

And even the mad ones are not necessarily mad in the way they are said to be by those who label them. People are often labelled mad for what their families and/or medical and nursing staff see as not to be uttered abroad ... Very often what these persons utter and do is deliberately highly provocative and is experienced by others as scandalous, their sin being to wash other people's dirty linen in public. But the live scandal is that persons are formally labelled mad or ill because they are scandalising others.

(1970:231-232)

Madness in these terms, is not considered as an illness in the medical sense of the term, but in terms of a personal perceptual and conceptual incongruity of the individual occasioned by the systematic distortion of his experience by others. The viewpoint offered is very similar to that of Kelley (1955) and Bannister (1960) who speak of the "serial invalidation of constructs", and is akin to Laing's use of "mystification" and Mueller's "distorted communication" referred to above (p.109ff). Thus, this sociological approach to mental illness operates on two levels: first, in terms of the invalidation of the individual as a person by the systematic distortion of his experience by others; and second, in terms of the

application of a disease label to the ensuing individual actions. The approach is conspiratorial not inasmuch as there is an active conspiracy to distort and label, although this may occur and is not precluded; but inasmuch as the label may be applied without an examination of the reasonableness of the individual's actions. This can be achieved only by examining the context of their occurrence. These comments suggest a radically different approach to doing the sociology of mental illness than that which has been current in the epidemiological literature.

a/ An alternative approach

It is suggested that in order to make theoretical statements about an individual's actions it is first necessary to make statements about how the individual makes sense of his social and physical environment. In contrast to the epidemiological studies reviewed which examined the extent to which social experience impinges on, or modifies an individual's behaviour, our concern is with how the individual perceives, defines and reacts to these processes.

There are at least two sorts of concepts in use in sociological research. On the one hand, there are first order constructs, what Schutz (1954) has called "common sense constructs", those concepts used in social action by participants in that action. On the other hand are those concepts used by the sociologist in order to classify and explain social action -- second order constructs. It is my contention that it is impossible to explain social interaction

theoretically by developing sets of causal laws about such interaction until we have first understood the interaction in terms in which the actors themselves understand it, that is in terms of the first order concepts. It follows from this that the constructs the sociologist uses, the second order constructs are, in effect, constructs of constructs made by actors in social interactions. (MacIntyre, 1967; Schutz, 1955; Cicourel, 1964; 1968:2) (There is, of course, a third order of constructs, used in stating formal theory, which are constructs of the second order constructs expressed in terms of the symbols used in mathematics or symbolic logic; but these need not concern us at this point.)

Given these two sets of constructs there are then two sets of rules of procedure. First, those used by the actor in applying his first order constructs in his construction of models of reality; and second rules of correspondence by which the sociologist categorises the latter in terms of second order constructs.

Thus, in order to make statements about an individual's actions it is necessary to make statements about the rules of procedure he uses in categorising his social reality. This involves two related assumptions: first, it is assumed that social interaction is structured, and that this structure tends to vary between different groups. Second, that individuals develop an awareness of themselves through their interactions with other people. This latter involves three subsidiary assumptions, that individuals construct

a model of themselves by taking others' perspectives of their behaviour; that the responses of others become an integral part in the development of a conception of the self; and that an individual's responses to the actions of others (and their reactions to him) are mediated by the structures of the groups within which they interact. There is nothing particularly startling or contentious about these assumptions which, to be sure, would be readily accepted by those involved in the epidemiological field research. They underlie the theorising about social isolation as a set of sociopsychological postulates to explain the rate differentials, and the implication of socialisation practices in predisposing (or acting on genetic factors) individuals to illness. What is different is the suggestion that they should serve as the resource of enquiry in their own right rather than remaining as unanalysed topics.

This structuring of interaction involves the communication between individuals. By communication I understand, not simply a system of verbal exchange, but the whole pattern of shared meanings and behaviours within a group of interacting individuals made intelligible to the participants (and accessible to the sociologist) by the sharing of implicitly or explicitly formulated rules (and rules about rules, Laing, 1969). By this I am not attempting to imply that man is a "rule governed animal" (see the discussion by Winch, 1958: 25-39; and Peters, 1958) but that he governs his interaction with others in terms of a shared system of mutually recognisable

linguistic and para-linguistic symbols (see Argyle, 1967) and a shared system of rules by which he translates the semantic content of his actions (his meaning or intention) into a patterned, syntactic, communicable form.

Thus, an adequate explanation of human action cannot rely simply on a description of its sequential or syntactic form (see Chomsky, 1968; for a similar point in linguistics) but must make reference to the shared rules and symbols presupposed in interaction, and to certain taken-for-granted 'background expectancies'. (Schutz, 1962) These consist of an accumulation of shared experience and expectations (a system of shared meanings) derived from previous interactions, which become the 'sanctioned properties of common discourse' (Garfinkel, 1967) whose use and presence is demanded by others in interaction. (See McLugh, 1968; for a discussion of the modes of response to situations in which the background expectancies are not met -- particularly the psychiatric episode.)

It should be apparent that breach of these rules, or background expectancies, is analagous to Scheff's discussion of breaking residual rules, except here, rather than attempting to classify the residual rules in terms of cultural stereotypes (as Nunally, 1967) or the like, the researcher is directed to examine the formation and application of such rules as peculiar to particular interacting groups . In this way it is possible to examine the manner in which definitions of illness are arrived at prior to the enactment of the formal labelling process. Further, without raising the question of mental illness it

is possible to examine the interaction within a group as 'normal' with reference to the rules invoked to make actions accountable within that group, when such actions if taken out of this context would be candidates for an illness label; that is, as breaking residual rules in Scheff's terms.

This approach to the problem of understanding human behaviour in relation to the groups with whom the individual has meaningful interaction raises four basic issues which have been raised by Cicourel in a different context (1968:6):

1. How is an individual's behaviour recognised as meaningful by the group with whom he interacts?
2. How do members of the group decide that a sequence of behaviour is "adequate" for the understanding of what is being communicated so that they in turn can present an adequate response?
3. How can we show meaningful communication between individuals even though there is no overt evidence that such a meaning exists?
4. How is it possible to establish antimonies in meaning even though overt agreement exists?

It is suggested that it is only by facing these issues and seeking explanations in terms of the shared rules (particularly the unstated rules) and symbols which structure a group of interacting individuals that an adequate explanation of an individual's social behaviour will be approached.

b/ Schizophrenia and the Family

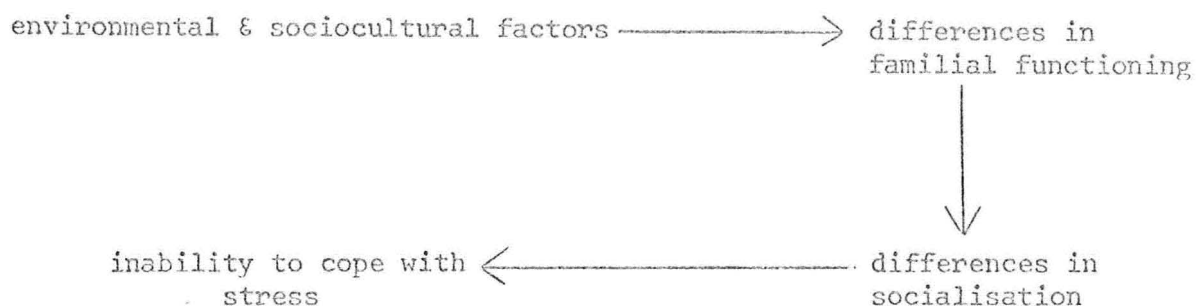
(i) The epidemiological research

It should be apparent that there is nothing in this approach which is incompatible with epidemiological research, and there is no reason at all (apart possibly from logistics) why epidemiological research should not concern itself with examining the relationship between patterns of family interaction and reported disorder.

Indeed much of the epidemiological research has suggested a need for a consideration of the interpersonal forces acting on the individual and associated with the disorder. Mishler and Scotch (1965:285) have suggested that epidemiologists should attempt to incorporate family process variables into their research. While this has not been done to any significant extent, it is not unusual for the researcher to appeal to such variables in explaining their results -- without, however, having the necessary data to support any inferences made on this basis. Srole's work is a case in point.

It is assumed in the above discussion that social interaction is structured, and that the structure will tend to vary between different groups. One feature of the epidemiological approach is that it suggests that socio-cultural factors may influence differences in intrafamilial functioning and structure. Throughout the discussion of the epidemiological literature constant reference has been made to the possible implication of the family in the etiological process. In part this has stemmed from an awareness on the part of the individual researchers of the contemporary clinical interest in the family, and

in part by the realisation of the need for some sort of intermediary structure intervening between the environmental and socio-cultural processes and the individual response. Characteristically the importance of the family has been stressed as an intervening variable. Srole (1962) terms it a component variable, which performs this buffer function. We discussed Srole's position earlier (p. 88) and it may be represented by the following diagram:



Here the causes of disorder are not located in the external factors, or within the family per se,; but the breakdown is seen to be a composite of the external stress, and the ability of the family to deal with such stress. Different group (social, geographical) contexts produce variations in family functioning which have attendant consequences on the interpersonal environment and experiences of the individual members. The adaptation the family makes to such circumstances, for Srole, may affect the frequency with which the family generates internal noxious situations as well as its ability to deal with external crises. Unfortunately Srole does not exploit this line of reasoning in his monograph, and little attempt is made to specify different types of family patterns and

Processes and their attendant risks for individual pathology.

Consequently, while the key finding of the research suggests that the:

offspring of low social class origin families at all adult age levels reflect maximum vulnerability to mental morbidity and minimum fulfillment of wellness...

Srole admits that the methods used to identify such groups cannot provide the data necessary to identify the processes involved:

to circumscribe such a deviant group is to delineate a socio-cultural habitat in which there is a probable imbalance of pathogenic and eugenic life conditions but is not to specify the specific chain of conditions that have such seemingly weighty consequences for the mental health of its inhabitants. To be isolated from its tangled group context, this chain of component factors requires pin-pointed research tailored to a formulation of the specific nature of that group, its processes and its problems.

(1962:354)

(This same criticism must hold for the Langer and Michael volume which analyses essentially the same data.)

This is not to detract from the importance of Srole's study, for it does provide a useful set of demographic indices for circumscribing such deviant groups (as Srole puts it). What it does do is highlight the problem we have been dealing with all through, that of examining the interpersonal environment of the impaired individual. Srole's theoretical approach is similar to that of Clausen and Kohn. Indeed, much of Srole's discussion of the impact of social processes on the family owes much to a similar discussion by Clausen who suggests that such factors effect "different response tendencies to stress, different ways of coping with the environment, differences in self conception and different modes of defense!" (quoted in Srole, 1962:20). While

it is assumed that any external pressure or group influence is likely to result in different patterns of socialisation, those most usually considered are those associated with social class. The explanation favoured by Kohn in Class and Conformity is that because of differences in socialisation techniques which are themselves grounded in the experience of social classes (1969:200), the lower class individual is less able to deal with the stress he encounters because he is equipped with an 'orientational system' that may be too rigid for 'critical circumstances that require subtlety and flexibility'¹.

This type of explanation is not incompatible with E. Becker's (1965) discussion of the implications of C. Wright Mill's work for psychiatry. In Mill's case it is limitations in available 'vocabularies of motive' which hamper the individual in dealing with the complexities and impersonality of contemporary urban life; for Kohn the problem is located in deficiencies in lower class socialisation which fails to equip the child to adequately perceive, assess, and deal with reality.

Kohn suggests an analogy between the conformist orientational system imparted by the lower class socialisation and the thought disorder of the schizophrenic. Unfortunately the scope and implications of this analogy must remain unexplored. The Class and Conformity study was concerned with gathering comparative data on 'normal socialisation' techniques in Italy and the United States and no data were collected on the family life of schizophrenics. The study (1969:vii) grew out of his earlier research with Clausen (1956) into social factors associated with the development of

schizophrenia. In this earlier paper, it was suggested that while social class was an important variable in determining the type of family experience of their 'normal' sample, schizophrenics from all social classes experienced family life characteristic of working class families. It was not clear what it was about lower-class life style which precipitated the disorder, and while Class and Conformity fills out some of the details of 'normal' lower-class socialisation, in the absence of detailed comparative data on the family experience of schizophrenics, the analogy between the conformist orientational system of the lower-class socialisation and the schizophrenic's thought disorder must necessarily remain speculative.

The original Clausen and Kohn study was important not for any definitive findings it unearthed, but for the stress it placed on considering social and demographic factors prior to drawing firm conclusions from any apparent relationships which emerged from the data. Thus while on the one hand their data would seem to support previous studies which have shown a relationship between maternal dominance and schizophrenia, when the data were analysed more closely such a conclusion was not found warranted. While there was a difference between middle-class schizophrenics and their controls there was little difference between the lower-class schizophrenic and his control in terms of maternal dominance. The authors conclude:

One fact we feel is abundantly clear: comparison of parent-child relationships of schizophrenics and normals cannot ignore the factor of social class, as has so often been done in the past. These studies which compared

schizophrenics, largely from lower status levels, with normal middle-class students or with groups of professionals have quite possibly documented a social class difference in maternal dominance rather than disease specific differences. Only if such differences are found between schizophrenics and normals drawn from the same class levels will we have real reason to assume that more intensive research in this area will prove fruitful.

(1956)

What the authors call for is a little methodological sophistication on the part of the researcher to consider both the impact of socio-cultural forces on the family and the internal family dynamics; for an exclusive concentration on one of these factors to the exclusion of the other must necessarily limit the accuracy and generality of any theoretical statement. It is worth leaving this point to one side for the moment while specific epidemiological studies are discussed and returning to it when we examine some of the clinical literature.

Much of the specifically epidemiological research on the family has concentrated on such structural variables as early parental or sibling deaths, or social disorganisation as a result of divorce, desertion, etcetera. (Rosenzweig & Bray, 1943: Blum & Rosenzweig, 1944) Here the concern is with the relationship between rates of disorder and socio-environmental factors. When a significant relationship is found, the explanation of that relationship runs into the same problems we discussed earlier. Thus Rosenzweig and Bray, who found that 36% of the male patients at Worcester State Hospital (n=356) had experienced a sibling death (half as much again as their control group), feel that the development of the disorder may be associated with the excessive

guilt their patients felt on the death of a sibling to whom they felt extreme hostility. Such an interpretation may have some basis in psychoanalytic theory; but is hardly derivable from a study which relied completely on hospital records. This same criticism may be directed to the more recent study of Hilgard and Newman (1963) who found an excess of parental deaths (compared with a random sample of 1096 persons from San Jose in the same age range) for females but not for males. Their suggestion is that the association is not so much a matter of the actual loss of the parent but of the poor quality of the step-parent. Again the study is limited to hospital records which would be insufficient to support such an explanation.

What would seem to be important is not the stark fact that there has been a death in the family but the adjustment the family and the child make to the loss. We must understand how they make sense of the loss. For example, before regarding the death of a parent or sibling as stressful to an individual (or weaving the tangled skein of guilt and hostility) it is crucial to establish whether or not it was defined as such. Laing (1969:2) discusses the example of a girl whose mother was severely impaired by a stroke. Although she recovered sufficiently to live on two more years, she was little more than a vegetable. So much so that the girl did not recognise her as her mother. As far as she was concerned her mother had died at the time of the stroke; and when death eventually came she felt only relief not grief. While it

might be possible to randomise similar differences in the perception of stressful events by appropriate sampling techniques the derived correlation would be a dubious basis on which to make etiological statements. The necessary data on adjustments are simply not available from the stark hospital records, and it is not accurate to consider a death as stressful until it can be shown to be so.

A more useful study is that of Lucas (1964) who found that poor family climate was highly correlated with parental absence or history of mental illness in one of the parents or sibs. Rather than relying on hospital records Lucas obtained information by interview of the families of 100 schizophrenic cases from public mental hospitals in Detroit. (The data collection and formation of a control group were essentially the same as Clausen and Kohn 1959.) Schizophrenia was found to be significantly associated with poor family climate. While this study does indicate that there is a significant association between disturbed parent-child relationships, and the subsequent development of schizophrenia, it is not clear what the significance of this association is, nor what processes are involved. There has, as yet, been no significant epidemiological concern with conceptualising and assessing the intra-familial experience of their cases, and in general epidemiologists have relied on the clinical researcher to fill out these details.

(ii) The clinical literature

The clinical literature on the family may be divided chronologically into two broad areas: the 'trait studies' which attempted to isolate some aspect of parental personality or

functioning which is seen as the key pathogenic element in parent-child relationships (Fromm-Reichman, Fleck, McCord, Myers & Roberts); and those studies which focus on the total family and attempt to specify and describe certain pathological patterns of interaction within that group (particularly the work of the Lidz, Bateson, and Wynne groups). The recent work on the total family experience has in part been stimulated by the shortcomings of the earlier trait studies.

(iii) The trait studies

Despite the lack of any definitive statement in the area of trait studies, it has been an almost universal observation that the relationship between the parents of schizophrenics are grossly unsatisfactory according to various criteria. The earlier studies tended to concentrate on the relationship between one parent and the schizophrenic child. The vogue created by Fromm-Reichmann's (1948) discovery of the 'schizophrenogenic mother' precipitated a rash of studies of the maternal personality. A bewildering array of traits were attributed to her; she was usually described as emotionally manipulative, dominating, over-protective while at the same time a rejecting person (Fleck et al, 1963; Garnezy et al, 1961; see however Wahl, 1956). Alanen (1958) after an intensive study of the mothers of schizophrenics described her as having frequently occurring anxiety and inward insecurity, unrealistic behaviour and thought patterns, aggressiveness and coldness in emotional life, and a proneness to dominating rather than submissive behaviour in interpersonal relations. (See also Lidz et al, 1957; McCord et al, 1962;

Kasanin et al, 1934; Weakland et al, 1962; Fromm-Reichmann, 1948.)

What is striking about these studies is the remarkable frequency with which such patterns are found. What, by contrast, is important is the lack of any convincing evidence that such traits are not responses to the child's actions or a consequence of the mother's relationship to the father, or an accumulation of all three.

The literature on the father does not exhibit the same uniformity. At times he is described as weak and immature, passive and unable to perform a paternal role (Myers & Roberts, 1955) while Lidz et al (1957) note cruel, sadistic and domineering characteristics. Lidz and his co-workers have attempted to explain these discrepant findings by attributing them to a sex-related pattern in which the fathers of schizophrenic boys tend to be weak and ineffectual, and those of the schizophrenic girls dominant and narcissistic. This explanation reflects Lidz' concern with the provision of appropriate role models in 'normal' development, a concern stemming from a Parsonian conception of the family. The significance of this compromise explanation is that it is made necessary by the indifference of many of the clinical researchers to the generality of their findings. To examine the personality of a male schizophrenic and attribute these traits to be of etiological significance for all schizophrenics (as has been done) is illegitimate as Lidz suggests. Similarly to attribute etiological significance to the personality of the mother without examining the occurrence of such

traits in the mothers of 'normals' or other persons with identified social 'pathologies' is also clearly illegitimate.

Despite the plethora of research in this area it is not possible to delineate a particular pattern of parental traits which is peculiar to the development of schizophrenic offspring.

Meissner's discussion of this point is illuminating (1970:4-5) for he illustrates the similarity in parental background of a variety of differently disturbed children. Overall, it would seem that there is no significant correlation between a particular type of parental characteristics and a particular form of pathology in the child. This conclusion is compatible with that of Spiegel and Bell (1959) who after reviewing a considerable literature purporting to demonstrate the relationship between parental traits and a variety of individual pathologies conclude that:

On the whole, the review of trait studies produced the impression that none of the parental traits held up for investigation can be correlated with a distinct or predictable pathological outcome, and that, while they may constitute a necessary condition, they certainly do not constitute a sufficient condition for the appearance of a specific psychological disorder in the child.

(1959:124)

This difficulty in specifying a pattern peculiar to the etiology of schizophrenia stems in part from a number of very basic methodological and conceptual problems (aside, of course, from the possibility that there is no such relationship). In Spiegel and Bell's sample of studies only 17 employed a control group (n = 85) and in general there was an indifference to establishing whether

similar parental patterns hold for different pathologies. In many cases the researcher exhibits a theoretical myopia by concentrating on a partial relationship within the family group (i.e. concentrating on the relationship between mother and child and ignoring the role of the father) and thus limits the generality and applicability of his statements by not considering or ignoring alternative explanations or other possible etiological variables.

Much of the inconsistency in the clinical research can be largely attributed to a partial or total neglect of several important variables which we have identified in the epidemiological studies, but which have not been systematically controlled in the clinical literature. Thus Sanua seems to be reiterating Clausen and Kohn's comments (p. 153) when he urges that: "future research should deal with such variables as social class, ethnicity or religious affiliation, age, sex, diagnostic categories, influence of the father as well as the mother, and so forth." (1961:265)

Sanua's comments on the discrepancies between the findings of Gerard and Siegel (1950) and Tietze (1949) are particularly pertinent. In their study Gerard and Siegel comment on the extreme attachment of the mother marked by excessive babying, spoiling and over-protectiveness, while Tietze found the reverse. Ten of his sample of mothers of schizophrenics overtly rejected their children, the remaining fifteen exhibited a more subtle rejection. Sanua (1965:249) suggests that these findings may be explicable in terms of the ethnic background of the parents sampled. While 70% of

Gerard and Siegel's sample was composed of patients from lower or lower-middle class often Jewish or Italian families; Tietze's sample was largely drawn (64%) from patients of Protestant professional and business classes. Sanua's contention is that the differences in rejection and over-protection may be largely explicable in cultural terms, thus questioning the use of such variables as etiological factors in these studies.

This same criticism may be made of Farina's work (1960) which examines the authority roles of parents of schizophrenics with 'good' and 'poor' premorbid adjustment. While he showed that mother dominance was more characteristic of the 'poor' group, and father dominance the characteristic of the 'good' group, the work of Baxter and Arthur suggests (1964) that such differences are largely attributable to certain social class biases in the selection of patients.

These comments on methodology would all be by the way, if it were not that they are equally applicable to the more recent research (see Rabkin, 1965; Meissener, 1970; Mishler and Waxler, 1966). Thus, despite their use of small group techniques the experimental research of Cheek (1965) and Farina (1960) among others must fall under this same criticism. It is Opler's (1957, Opler & Singer, 1956) awareness of these extra-familial or cultural influences on parental interaction which makes his work so important. He found that the Italian family of schizophrenics is typically father dominated, and the Irish family typically mother dominated

and these differences are reflected in respective symptomatology. Incautious extrapolation from a sample of families of one particular ethnic group could on this basis constitute a severe distortion.

It is ironical that this position is reiterated here when throughout it has been suggested that epidemiologists should take more cognizance of the family studies. It indicates the dangers of an uncritical acceptance of the clinical findings to explain statistical relationships in the epidemiological data. The irony comes when we find Srole developing a very cogent argument for the influence of socio-cultural forces on family processes, and then explaining some of his findings by referring to a study which ignores these forces! (1962:356, f.n. 30)

(iv) The total family studies

If we turn attention now to the total family studies, it is relatively easy to dismiss them in similar terms. With Lidz' work, for example, it is difficult to avoid the temptation of dismissing "schism" and "skew" as characteristics of middle and upper-middle class families of schizophrenics. Despite the voluminous research reports Lidz' sample was composed of 16 families selected for intensive interview over a period of years. The only datum we have on their background is that they were all able to afford prolonged private psychiatric treatment (1965). As with much of the clinical literature Lidz avoids the very groups, particularly lower class groups, which have been identified in the

epidemiological studies as having high incidence of disorder. It is situations like this which emphasise the importance of Kohn's and Opler's work, which seeks to establish the prevalence of a particular pattern of intrafamilial behaviour in the general population.

To dismiss them in this manner would be somewhat cavalier and in many instances to claim generality for theories which the authors themselves admit are limited and speculative. Bateson (1956; see also Rabkin, 1965: 118 who mentions a personal communication from Bateson) is modest in his claims for the importance of the "double-bind" for these very reasons. It is not unlikely, he argues, that there may be ethnic variations in the prevalence of the "double-bind" considering the frequency of this mode of communication in middle-class Jewish families. The most interesting feature of the more recent research in this area has been the attempt to establish how far this mode of communication is characteristic of families of children with different social pathologies.

On the other hand, to accept the findings and concepts of such studies uncritically undermines the utility of any theoretical statement which might be made. An alternative approach is to examine these studies for their methodological and conceptual implications in order to derive a set of workable hypotheses which, unfortunately, it will not be possible to test in these pages. To attempt to synthesise these theories, which is tempting given the similarity of the theoretical explanations, would be to distort the individual

research efforts. To do so would force closure on the developing theories which is not necessarily the claim of their authors.

In what follows I want to tease out the main ideas of these total family studies, and use these as the basis of a model of schizophrenia and family processes. The treatment of the various theories in this area will not be exhaustive; rather our concern is with picking out the similarities of emphasis which will enable us to construct such a model.

As argued earlier, what hampered the earlier "trait studies" was a limited and restricted conception of intra-familial relationships. Thus while the relationship between mother and child (or father and child) was considered important it was not examined as an interaction between mother and child such that the child's actions might conceivably cause, or influence, the mother's (or father's) actions. Further the conception of the child was unduly plastic and passive. That is, while the possibility of the child influencing the mother's behaviour was ignored, her influence upon him was so great to the point that it affected his adolescent and adult personality.

Taking this latter point first, one of the significant features of Kohn's approach (above p. 152) is that it focuses on the orientational system, and does not attempt to establish an isomorphism between parental personality or traits or child care practices, and the risk of pathology.

Stevenson (1957) in an interesting paper criticises the assumption in many of the clinical studies that the child is

considerably more plastic than the adult, and that the moulding a child receives in its early years places an indelible mark on the developing personality. The work of Orlansky (1949), Thurston and Mussen (1950), Sewell (1952) indicates that the idea of childhood training and experience have specific effects on an adult personality are acceptable. (See however Thurstone, 1957; who indicates that at least some patterns are learned.) What these studies show, and what is implicit in Kohn's ideas, is that the ability of the child to act on his world, and influence his social relationships should not be ignored.

The model of the child in the trait studies is that of a passive creature being molded by his social environment; by focusing on the orientational system, it is possible to examine the manner in which the child structures his world, and defines his relationships (and hence in Kohn's schema his ability to cope with stressful events). Because the trait studies are necessarily retrospective, that is, the mother of an already identified patient is interviewed, this denial of the individuality of the child is yet another element of mystification denying his ability to act on his world. The work of Chess et al (1959) is illuminating in this respect. In the eighty-five children studied specific individual reaction patterns occurred during the first few months of life which significantly affected the child's response to its social and physical environment, and in particular the parents' socialisation techniques. They found it possible to

distinguish children along a number of dimensions (activity/passivity; approach/withdrawal; regularity/irregularity) which significantly affected that child's response to sleeping, feeding, toilet training, etcetera. Coupled with our examination of the orientation system this line of reasoning suggests that rather than treating the child as a passive recipient of stimuli, or accepting other's descriptions of his home environment, it is important to examine his perception of parental actions towards him, and examine the manner in which he acts upon his interpretation of that experience.

Utilising this perspective, Ausubel et al (1954) find it important to consider the child's perception of his parents' attitudes and actions towards him as a significant variable in mediating his response to those actions and attitudes. In a similar vein Rabkin (1964) twists the 'trait studies' about and is examining the manner in which the labelled schizophrenic perceives his mother's relationship to him.

Lest it be misunderstood, it is not meant to imply that the family may not influence or teach the child an orientation system which is inadequate; but that this orientation system is not isomorphic with his subsequent pathology. What does seem to be important is the manner in which the child is equipped to define his relationship to other family members, and to define their relationship to him. The importance of the orientation system may be in the accuracy it permits the child in his interpretation of his parents' actions towards each other, and towards him.

One implication of treating the family as a whole is that it is now possible to consider the accuracy of such perceptions, and, more importantly to examine the effects of the members' actions upon each other, and upon each's experience of the family and intra-familial relationships. It is interesting that the approach we are suggesting corresponds to both Becker's (1964) and Laing's (1967:119) criticisms of psychoanalytic theory in that there is no systematic psychoanalytic theory which can deal with the attempts by one person to act on the experience of another, nor of the other's attempts to deal with such transpersonal "attacks". Goffman in his Encounters (1961) argues, in a similar vein, that "there seems to be no agent more effective than another person in bringing about a world for oneself alive, or by a glance, a gesture or a remark, shrivelling up the reality in which one is lodged."; and it is this feature which is beginning to emerge from the studies of the family as an interacting unit. What seems to be important is not the methodological nicety of examining the family as a whole, but the necessity of examining the integrity of the unit, and the manner in which family members perceive and define the family as a whole and actively seek to maintain this definition. It is this point which seems to be so critical in understanding both the actions which come to be labelled, and the decisions of family members to initiate labelling procedures.

One of the interesting findings to emerge from the studies of families of schizophrenics is that the onset of disorder may precipitate significant changes in the other family members. While

we might expect as happens in some cases that the commitment of the individual may lead to improvement in intrafamilial relationships, or that the onset of symptoms may be accompanied by disorder in other family members (Fleck et al 1957), what is surprising is that evidence of the patient's improvement during therapy may be accompanied by severe disturbances in other family members (Ackerman, 1954; Haley, 1962; Jackson, 1957; Jackson & Weakland, 1959: 1965). The family seems in many cases to offer an extreme resistance to any amelioration of the patient's symptoms (Esterson, 1971) which it seems will effect changes in the customary pattern of interaction within the family, so much so that the patient may relapse to his original level of functioning (Bowen et al, 1959).

The impression drawn from this literature is that the labelling of an individual is essential for the maintenance of the status quo within the family, and that the individual may aid in maintaining the status quo by becoming or remaining "ill".

These two ideas permeate the work of both Wynne and Bateson and represent a radical departure from the conception of mental illness we were dealing within both the epidemiological studies and trait studies.

(v) On causes again

It is not unnatural that epidemiological and trait studies which focus on etiology should attempt to isolate antecedent conditions which increase the risk of the development of a mental disorder. In the trait studies, the attempt was made to isolate

something in the personality of the parents which would serve as an antecedent to the development of schizophrenia in the child; however, the problem becomes considerably more complex when it is suggested that the relationship between parents and child be analysed in interactional terms with particular reference to the child's perception of that relationship. This involves considering the family as an interacting whole, and not isolating discrete dyads for analysis to the exclusion of other family members. Thus, for example, while the study of parental interaction as in Bowen's work (1960) on 'emotional divorce' was a step in this direction it still sought to isolate antecedent conditions to the subsequent development of pathology in the child. This is much the position of Lidz' work.

The general theme of Lidz' research is that problems in the family are brought about by basic psychological difficulties the parents bring to the relationship and it is the parents' failure to observe both age and sex differences in their relations with the children which results in the child learning inappropriate behaviour -- particularly sex-role related behaviour. While his work shares many similarities with that of the Bateson and Wynne groups it is nevertheless an attempt to establish antecedent conditions rather than examining the labelled's actions as elements in ongoing intrafamilial relationships.

Lidz dispenses with control groups in developing his theories; but does not dispense with a conception of the requisites of normal family life which he derives explicitly from Parsons'

work. The normal family is one in which the

spouses need to form a coalition as members of the parental generation maintaining their respective gender linked roles and be capable of transmitting instrumentally useful ways of adaptation suited to the society in which they live.

(1963:53)

Deviations from this pattern may thus be one important influence (Lidz, 1957:242 is careful to avoid attaching specific etiological significance to them) in the risk of disorder. Lidz et al identify two main patterns, 'schism' and 'skew', which show gross deviations from the 'normal' (ideal?) pattern. (1957) In 'schismatic' families reciprocity in role performance and in mutual goals is absent. These families are marked by chronic hostility and the mutual withdrawal of parents whose communication "consists primarily of coercive efforts and defiance or of efforts to mask the defiance to avoid fighting". In the 'skew' marriage, the marriage exists in a state of equilibrium, but the family life is distorted by a severe imbalance, or skew in the family relationship. These families were marked by severe psychopathology in one of the marital partners, to which the other spouse may react either by supporting the other, or alternatively by sharing his/her distorted ideation. Both these patterns represent considerable departures from the normal pattern, and consequently do not provide appropriate environments for proper age and sex role development of the children and foster the development of irrational and distorted patterns of thinking. (1963:101)

The greatest failing with this formulation, (as Mishler & Waxler 1966 have also noted) is the reliance it places on an assumed model of intra-familial functioning, and contrary to what we have argued thus far does not examine the development of specific intra-familial modes of functioning peculiar to a particular family. In the absence of comparative data on 'normal' families (however this is defined) there is no convincing argument that a skewed pattern may not be normative in particular instances, nor that the schismatic relationship is not associated with normal sex role development and absence of psychopathology. By assuming that deviations from a hypothetical normative pattern, (without examining the prevalence of such deviation in families of individuals not labelled as schizophrenic) have relevance in the risk of psychopathology and ignoring the child's perceptions of intrafamilial relationships and his part in them, Lidz' work reflects a continuing concern with establishing the etiology of an illness. Parental characteristics or relationships are taken as antecedent steps in causal chain and, with assumptions as to the plasticity of the child, as in some way causing the disorder.

Now Blalock (1964:8-9) in his discussion of causes includes the notion of "forcing", or variable producing a change in Y rather than simple covariation as a criterion of causality. If X is the cause of Y, Y cannot cause X. This asymmetry between cause and effect is the asymmetry between the parental actions and the child's response. In order for this conception to work there should be no,

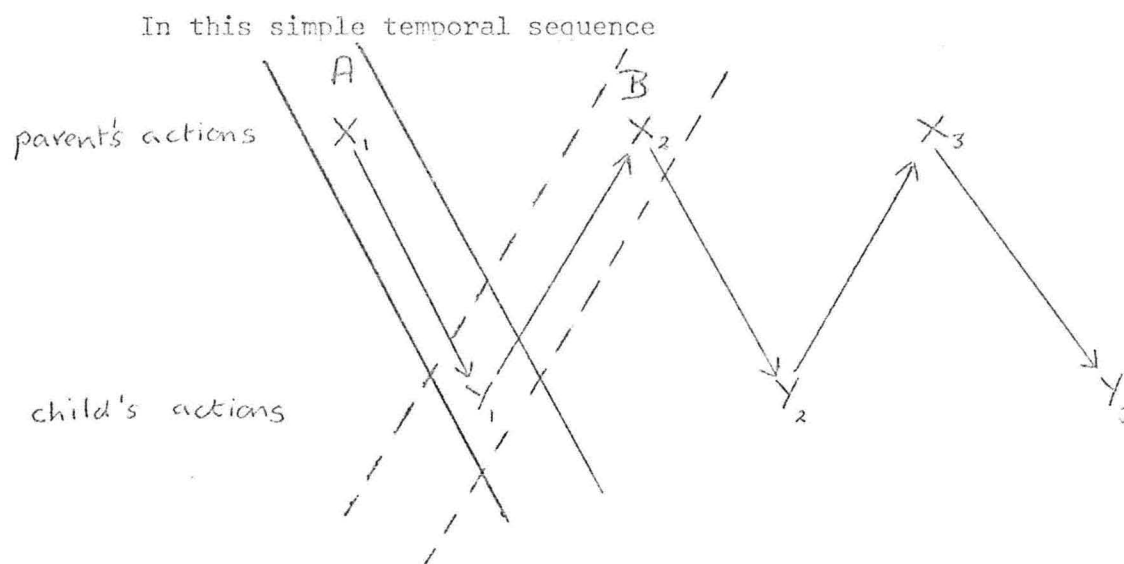
or minimal influence from child to parent while the parents' actions produce changes in the child, in this case influence development in particular ways. The temporal sequence between X and Y is guaranteed by the assumption of the plasticity of the child who reacts to the parents actions.

To introduce the child's perceptions and actions renders the schema considerably more complex. In this case it is necessary to consider the parents' actions as "caused", influenced by the child's actions -- or more realistically that the interaction between parents and child is a reciprocating pattern of accommodation. To do such, we must necessarily introduce a different conception of the "pathological" behaviour as an accommodation to the actions of others, who in turn are accommodating to those actions

So long as the range of observation is limited to an insulated aspect of this reciprocating system, the observer is confronted with something that is unintelligible which may induce him to attribute to it properties which it may not possess. If the researcher attributes a schizophrenic label to an individual's actions without examining their intelligibility in terms of the reciprocating system he invalidates the individual's actions by attributing them to a disease.

It is only possible to arrive at a causal chain like that described by Blalock if individual actions are isolated from this reciprocating system. If X causes Y, then Y cannot cause X. By contrast, in terms of a reciprocating system the direction of

causality (although such a term is inappropriate) depends upon the point at which the sequence is broken. Thus rather than considering X's effect on Y and vice versa, we are interested in the interaction between the two and the modification of X by the actions of Y and vice versa.



very different conceptions of causality arise depending upon where one breaks the sequence. If at A, the parents' actions are taken to 'cause' the child's actions. If at B, the reverse is taken to be the case. If instead we look at the whole sequence neither is the case, and the interaction between the parent and child is seen as an accommodation between the two. More importantly, the actions of any one member of this sequence may be unintelligible without reference to the actions of the other.

Now in terms of schizophrenia the 'presented symptoms' which are diagnosed as disorder may represent the end point of such a sequence of interaction and while unintelligible (or formally undecidable) when isolated, are understandable with reference to the previous interaction. This is not incompatible with the argument in the previous chapter that the schizophrenic's actions may be the

consequences of the concerted actions of others towards him. In both cases the disease label is inappropriate. This is very much the position taken by Bateson when he talks of "schismogenesis" and Singer and Wynne (1964:13-20) when they describe their 'transactional and epigenetic view' of the development of schizophrenia. These authors argue that the interactions between parents and parent and child depends at each phase on what occurred in previous phases, and rather than tracing a direct line from some aspect of parental functioning in early childhood to the subsequent development of disorder the authors emphasise the transactional development of such patterns of action which come to be labelled as disorders.

(vi) Rubber fences

Wynne and his coworkers have been concerned with the quality and structure of role relationships within the family, and rather than examining particular dyads or triads they have attempted to develop a conception of schizophrenia which takes into account the structure of the family as a whole. (1958:205) When you read through Laing and Esteron's book Sanity Madness and the Family it is surprising how often the delusional structures of the patients are recognisable in their patterns of relationships within their families. The identified patient appears, in his diagnosed symptoms to be giving expression to his intra-familial experience. Following this line of reasoning, Wynne's guiding hypothesis (1958) is that

fragmentation of experience, the identity diffusion, the disturbed modes of perception and communication and certain other characteristics of the acute reactive schizophrenic structure, are to a significant extent derived by a process of internalisation from the characteristics of the family social organisation.

Wynne and his colleagues found that the characteristic pattern of interaction in the families of the schizophrenics they studied was that of "pseudo-mutuality". In order to understand the importance of pseudo-mutuality it is necessary to understand certain of their basic assumptions, principally that human beings 'need' to move into relation with other human beings, and strive constantly to develop a sense of personal identity. Identity:

consists of those self representations, explicit and implicit which give continuity and coherence to experience despite a constant flux of inner and outer stimuli.

(1958:206)

The writers argue that the individual may meet these two needs of relation and identity either in mutuality, non-mutuality, or, in the one that concerns us here, pseudo-mutuality. To quote Wynne et al

Pseudo-mutuality refers to a quality of relatedness with several ingredients. Each person brings into the relation a primary investment in maintaining the sense of relation ... The past experience of each person and the current circumstances of the relation lead to an effort to maintain the idea or feeling, even though this may be illusory, that ones own behaviour and expectations meet with the expectations and behaviour of other persons in the relation.

(1958:208)

The feature which distinguishes pseudo-mutuality from the other forms of mutuality is the pre-occupation with maintaining the

relationship even if it means sacrificing individuality and personal identity in the process. When situations change, or alternative questions arise, rather than redefining the relationship and developing personal identity through the solution of the problems which occur, (see Cumming & Cumming, 1969) in the pseudo-mutuality the parties seek to maintain "the sense of reciprocal fulfillment" rather than accurately perceiving changes in expectations and reacting accordingly.

Consequently the delineation of roles within the family is rigid and inflexible, and infractions of role performance are either denied or re-interpreted in order to maintain the sense of relationship. Members are preoccupied with the performance of their roles to the detriment of the development of individuality and Wynne argues that the family members develop a strong concern in maintaining things as they are. The family is paramount, and its role structure is considered as an all encompassing, "a truly self-sufficient social system" able to fulfill all the needs of its members. The emerging child is forced to conform to the system and aid in maintaining the sense of mutuality. By being restricted to the rigid performance of presented roles he is prevented from meaningful participation in the wider society which Wynne considers is essential to the healthy development of individual personality. Wynne describes the family as being surrounded by a "rubber fence" which prescribes the range beyond which member's experience may not wander. Thus while members do interact with people other than family members and may

be physically removed from the family, they are nonetheless subjected to family expectations on their behaviour, and interpretation of experiences.

In seeking to maintain the 'sense of relation' role behaviour within the family becomes divorced from experience and Wynne (1963:14) argues that the thought disorders of the schizophrenic are a consequence of the disjunctive, fragmented and poorly integrated communication within the family necessary to maintain the semblance of relationship.

A number of points are interesting about Wynne's work, not least of which is that the schizophrenic is conceived not as a passive participant, a victim of pseudo-mutuality, but is thought of as actively involved in creating and maintaining the system. His actions are not seen as disfunctional but rather as necessary to maintain the sense of relationship; and it seems, though Wynne does not argue this, that the label is applied when he attempts to generalise his intra-familial experience and modes of functioning to situations outside the family in which they would not, of course, be appropriate. A second feature of Wynne's approach is that the stabilisation of a pseudo-mutual relationship can only occur in groups in which there is a high degree of emotional involvement and commitment and from which exit is difficult. In situations without this commitment, i.e. in which it is not important to maintain the relationship, non-mutuality is more characteristic.

If we return for a moment to Lidz' work there are elements which are similar to this formulation. Lidz, like Wynne, stresses that the families he studied (1963:101) tended to be insulated from

social contacts thus restricting exposure to conflicting meanings and expectations than those experienced in the family. The family could thus exist "normally" with its disordered patterns of interaction which were reinforced by the lack of external contact. Secondly, although it is not an important element in his work, Lidz does indicate that family members may think it necessary to maintain the status quo. Thus in his discussion of the 'skew' family one parent may seek to support the other or all family members may share a parent's delusions, and in the schismatic family Lidz suggests that parents strive to avoid conflict by distorting their communications, and maintaining the semblance of a relationship.

(vii) Bateson and Communication

If we turn now to Bateson and his colleagues' research we will find similar themes recurring. Although phrased in a different language, and with different foci, the work stimulated by Bateson and his co-workers has similar emphases. Bateson is best known for his identification of the "double-bind" (Bateson et al, 1956) but before discussing this particular relationship it is worth stressing a number of important features of his work which underly the importance of the "double-bind".

The distinguishing feature of the Bateson group is a general model of action in which communication is considered as equivalent to action, rather than as one of its many facets. This aspect of the work has been brought to fruition in Watzlawick et al's Pragmatics of Human Communication. (See also Watzlawick, 1964) Amongst the

axioms proposed by Watzlawick is that "one cannot not communicate", that is, all action in an interactional system has a pragmatic or meaning value despite the wishes of the communicator, and that communication will take place whether mutual understanding occurs or not. The very act of not speaking or acting in a situation from which one wishes to abstain may be interpreted by others as a form of communication where none was in fact intended.

Communication itself has two aspects: (a) an informative aspect; and (b) a relationship aspect (see Bateson and Ruesch, 1961). That is, apart from simply conveying information, a communication defines a relationship between the interactants. The relationship aspect defines the manner in which the data conveyed in the informative aspect is to be interpreted, and may be explicit or implicit, verbal or non-verbal. Watzlawick et al define an interactional system as 'two or more communicants in the process of, or at the level of defining the nature of their relationship'.(1968:121) Thus in interaction participants offer each other definitions and negotiate a common definition of the situation in which they are acting. This latter statement may be stated more forcibly as each seek to determine the nature of the relationship. It is this aspect which Haley has taken up in his more recent research. (See especially 1962, for his discussion of psychotherapy; and 1963) A second important theme is the by now familiar concern with the manner in which the family members seek to maintain the equilibrium of the family system. This has received its most complete examination in Haley's work. Haley considers interaction within the family as being

governed by a system of rules which is 'error-activated' (Haley, 1959(a): 373) so that if one of the family members breaks the rules the others will either seek to make him conform, or redefine the rule in view of the infraction. It is immaterial whether these rules are inside or outside the awareness of the interactants -- that is whether they are stated or unstated. They represent the taken-for-granted knowledge of the participants as members of the group which may be understood, but not communicated in sequential action. (They are in this sense analogous to the generative grammars of Chomsky, 1962)

The researcher, (or participant for that matter) arrives at these rules by examining the sequences of interaction and postulating a meta-communicational statement about the rules which govern the sequences of individual action. These rules set limits to the range of acceptable actions within each family (one could draw an analogy here to Wynne's 'rubber fence') and are phenomenologically unique for each family. Here the difference between this work and that of Lidz becomes apparent. Rather than postulating a normative pattern, as does Lidz, we are directed to attempt to isolate the unique normative pattern for a particular family.

(viii) Recipe for a double-bind

Bateson's interest in the double-bind was generated by the original research concern to establish what sequences of action, or communication within the family would induce (rather than cause) actions which would justify the diagnosis of schizophrenia. Bateson

and his co-workers reached the conclusion that the schizophrenic must live in an environment in which sequences of events are such that his disorder communication is in some sense appropriate. (Watzlawick, 1968:211) The double-bind is not postulated as a cause of schizophrenia, because it specifies a relationship between individuals. The schizophrenic label is applied when diagnostic attention is limited to one of the participants whose communicative actions are meaningless unless considered as normal responses to the binding communications. The double-bind is then a reciprocal pattern of communication which binds all of the participants.

The double-bind may be thought of very simply as a confusion of the informative and relationship aspects of communication referred to above, (p. 178-179) with the important addition that the individual is forbidden to meta-communicate, or comment on the incongruity. (Watzlawick draws out the influences of Russell's work on logical paradox where solution lies in recognising the incongruity between various levels of the problem.) While the message may be logically meaningless, if comment is prescribed, it remains a pragmatic reality which demands action on the part of the communicant. One cannot not communicate.

The double-bind has a number of necessary ingredients which Bateson et al (1956) outlined in their original publication:

(a) two or more persons involved in an intense relationship which has a high degree of survival value for one or all of them;

(b) in such a context a message is introduced which is incongruous, it asserts something and contains as its second element an assertion which contradicts the original assertion (For example: "Be spontaneous!");

(c) the recipient is forbidden to meta-communicate, or comment on the incongruity in the message, to withdraw from the situation, or to show any awareness of the incongruity. In such situations, while the message may be illogical, it is a pragmatic reality forcing the individual to react in a situation in which he is "faced with the dilemma of either being wrong in the primary context, or of being right for the wrong reason or in the wrong way." (1960:477)

In this sort of situation it is impossible to discriminate or correct the discrimination of what order of message the individual is to respond to, and consequently he may avoid responding by inaction or silence (which are not effective strategies because one cannot not communicate) or he may seek to disqualify his actions by negating the content of his messages. The solution of this dilemma lies in the use of undecodable messages which by their meaninglessness say they are saying nothing, and it is these messages which may come to be labelled as schizophrenia. The irony is that this form of disqualification may itself be a double-bind. Bateson gives a very brief example of the double bind in the original paper:

A young man who had fairly well recovered from an acute schizophrenic episode was visited by his mother. He was glad to see her and impulsively put his arm around her shoulders, whereupon she stiffened. He withdraw his arm, and she asked, "Don't you love me anymore?" He then blushed, and she said, "Dear, you must not be so easily embarrassed and afraid of your feelings."

(1956:259)

In this example the conflicting levels of communication are easily seen. The mother embarrasses her son when he shows affection and thereupon criticises him when he does not. The son is placed in an impossible dilemma. He can respond to neither message and is forbidden to comment on the ambiguity.

The interesting feature of the double-bind mode of communication in the family of the schizophrenic is that it may become the customary mode of communication. That is, while in all families double-binds may occur the schizophrenic's family is marked by a recurrence of this mode of communication. The obvious question is why this should be so.

In Bateson's work this revolves about the members' attempts to avoid the destruction of their 'selves'. Bateson considers the family of the schizophrenic as being unable to form stable coalitions which he considers important for the solution of the problems which face it. As a consequence, the family is unable to provide its members with adequate solutions to these problems and members are continually subjected to self-negating experiences which hamper the development of stable identities. (Like Wynne; Bateson shares Erickson's belief that the self develops by the individual resolving the crises which confront him. If the individual is denied this experience, or presented with pseudo-solutions the self is stunted in its development. See Erickson, 1950: 1957: and the discussion by Cummings and Cummings, 1962: 32-46) Because of this self negating experience the individual family members believe that the self can in fact be destroyed and consequently the family members adopt a

form of interaction or communication which is at once an attempt to establish whose self shall be destroyed, and to shield themselves from attempts to destroy their selves. This pattern which he labels, the 'double bind', becomes a persistent pattern in these families and an adaptive strategy by which members are able to maintain the stability (or sense of stability in Wynne's terms) of the family.

On this basis, not all contradictory messages would come to be labelled as schizophrenia, even though the interaction or communication in the family is 'deviant from the cultural environment'. Bateson terms this "normal" mode of communication "covert" schizophrenia (It is 'normal' because it is the endemic form of communication within these families) and distinguishes it from the 'overt' schizophrenia of the identified patient. Bateson is not particularly clear on what the distinction is between these two types; but the impression is that the overt phase is an exaggerated form of the typical intra-familial form of communication. But it is not clear why individuals should come to be labelled as schizophrenic; particularly when it would seem that all of the family members are candidates for the disease label.

One major problem which hampers the solution of this particular dilemma is the scant attention Bateson pays to the provision of control groups or of data on the prevalence of the double bind in families which do not have a labelled schizophrenic. It seems, (Bateson, 1959: 133-134) that the individual is labelled as schizophrenic when he adopts this form of communication with non-family members. What is 'normal' in the family would be conspicuously inappropriate with

non-members and it is this generalisation of mode of communication which leads to the recognition and diagnosis of schizophrenia.

Bateson is not clear however, on why the individual who comes to be labelled should act in the conspicuous fashion while the other members of the family are more selective and restrained in their use of this mode of communication. Haley's work offers a possible solution.

Haley, maintains this distinction between the overt and covert phases of schizophrenia when he extends the concept of the double-bind to include the family as a whole. As with Bateson, the idea of self-disconfirmation figures prominently in Haley's research. Haley emphasises two aspects of the family, (a) a system of rules which govern interaction within the family and (b) the power struggle within the family about who is to set these rules. The primary issue in all interaction, argues Haley, concerns who is to set the rules for the relationship. We commented above that Haley adopts the more forcible approach to the necessity of establishing a definition of a situation when he interprets it in terms of the attempts by each member to set limits on the behaviour of the others. This, for Haley is a characteristic of all families, and indeed all relationships; but what distinguishes the family of the schizophrenic is the collective denial that anyone is in fact setting the rules. While these families do, indeed, go about setting and following rules, they adhere to a meta-rule which prevents them recognising these procedures. This they achieve by using the double bind to disqualify their communications. For example, if my wife asks me to

take out the garbage and I refuse saying "I'd really like to help, but my ingrown toenail is bothering me!" I disqualify my refusal which rejects her attempts to govern my action, by blaming it on forces other than myself -- "it's not me; but the pesky toenail which prevents me from doing what you say!". Now, while this may seem a trivial example, Haley has found that the schizophrenic's family is characterised by chronic attempts by the members to deny that they are being influenced by others, or that they in turn are influencing others' actions. In this sense Haley views the double-bind as an adaptive strategy which the whole family may adopt to maintain its stability, while simultaneously denying the rule setting activity.

In terms of the individual the double-bind may be used as an active strategy to avoid committing himself to any particular definition of a situation. Haley makes the same distinction Bateson does in distinguishing between the overt and covert phases of schizophrenia, but is more definite in describing the situations in which individuals may be expected to exhibit the overt phase. While the double-bind may be an adaptive strategy in these families, there may be situations in which the individual is forced to break the family rules while being simultaneously subjected to the injunctions to remain within the rules. Haley outlines three such circumstances: (a) when two family prohibitions contradict each other and the individual must respond to both; (b) when forces outside the family, or maturational forces within the individual require him to infringe the rules; or (c) when prohibitions peculiar to him conflict

with the prohibitions common to all family members.

The individual's dilemma is that he cannot not communicate in such situations and he may seek to escape from a decision or deny that he is making a decision by a variety of different strategies. Thus, Watzlawick et al (1968:50-51) have observed that if a schizophrenic's behaviour is observed with 'etiological considerations in abeyance' it appears that the individual is trying desperately not to communicate. He may withdraw into himself, remain silent or immobile but in each case his actions may be misinterpreted by those around him. Consequently he may attempt to adopt a mode of communication which while conveying information denies that it is doing so (Haley, 1960:89-99); or which by the manner in which it is presented denies the commitment which is inherent in all communication. (Watzlawick et al, 1968:73) The individual places his listeners in a 'bind' by asking them to interpret his actions from a variety of different meanings which may be incompatible with each other. In this way he is able to deny all or any aspect of the communication. An example is in order at this point. Watzlawick et al (1968:73) describe the case of a patient who 'bounced' into her therapist's office and announced: "My mother had to get married and now I am here." The writers continue:

It took weeks to elucidate some of the many meanings she had condensed into this statement, meanings that were at the same time disqualified both by their cryptic format and by her display of apparent humor and zestfulness. Her gambit, as it turned out, was supposed to inform the therapist that

- (1) she was the result of an illegitimate pregnancy;
- (2) this fact had somehow caused her psychosis;

(3) "had to get married," referring to the shotgun nature of the mother's wedding, could either mean that Mother was not to be blamed because social pressure had forced her into the marriage, or that Mother resented the forced nature of the situation and blamed the patient's existence for it;

(4) "here" meant both the psychiatrist's office and the patient's existence on earth, and thus implied that on the one hand Mother had driven her crazy while on the other hand she had to be eternally indebted to her mother who had sinned and suffered to bring her into the world.

(1968:73)

By disqualifying his actions in this manner the individual is able to aid in maintaining the status quo and remain within the family rules.

c/ Implications

One must be careful to avoid drawing grand conclusions from this and the previously reviewed research. The temptation here is to argue that the attribution of a disease label is associated with situations in which the individual poses a threat to the family status quo. In such a situation he adopts a mode of functioning which permits him to remain within the family rules, and the family confirms this disqualification of the threatening actions by enacting psychiatric procedures. While this is suggested by Haley's research, it is not supported by his data base. When he argues that the onset of the 'overt phase' is associated with the infringement of family rules he is describing families in therapy and is not presenting us with data on the processes by which individuals come to be labelled. What he does do is explain why the family resists any amelioration of the patient's condition during therapy: but any comments on the labelling process must remain an inference.

While he does not discuss the labelling process he does locate the situations in which the individual may be obliged to infringe the family rules at a time when he is subjected to forces outside the family, and to maturational forces within himself. One theme which runs through the work of all of these writers is that the stresses associated with adolescence may be significant factors in the onset of the disorder. The impression given is that the individual's intra-familial experience has not prepared him adequately to deal with the stresses he experienced when faced with the necessity of assuming adult responsibilities. At the same time he is forced into contact with the world from which he has been shielded by his family. In such situations his behaviour may be inappropriate and bizarre, and consequently draw the disease label.

Haley's work gives us a different perspective on this problem. (Although what I am arguing may not necessarily be Haley's approach to this problem.) He similarly stresses the period of adolescence as a time which is associated with the 'overt' phase of schizophrenia in families in therapy. I want to suggest that it is this threat to family stability which occurs during adolescence, particularly in the child's attempts to seek independence from the family, which is associated not so much with the onset of the disorder; but with the decision by family members to seek psychiatric aid in invalidating the actions of one of its members and thus maintaining the stability. This is an extrapolation from Haley's work,

and is not backed up by any data on the processes of commitment, or on the normal family, and consequently must remain speculative.

It is, however, consistent with the continual stress in all of the writers reviewed on what the members perceive in the necessity of maintaining the stability of the family. (This aspect does not figure to such a great extent in Lidz' work; but is nonetheless there.) The family is characterised as being isolated from its social milieu functioning as a semi-autonomous unit which seeks to define and limit the experience of its members. Each theorist stresses, albeit in different ways, the necessity of interaction in the development of the selves of individual family members and in both Wynne and Bateson (and by inference Lidz) the family is considered as a group which the members recognise as absolutely essential for their survival and development. The members have an intense emotional commitment to the group which simultaneously prevents them from leaving it, and commits them to maintaining its stability.

If we accept Kelly's (1955) view that man is continually trying to make sense of the world around him and continuously checking the sense he has made by testing its predictive capacity then it will become apparent that the integrity of his self depends upon the integrity of the model he forms of the world. In a system of relationships, that integrity will also depend upon his experience of others' models of shared experience. In the family, to be in the family means sharing the same model of the family as the other

members. This model of the family, as Laing has argued (1967:119) may be the medium that each person uses to link their experiences together. Any attempt to restructure that model by one member results in a crisis, and a necessary restructuring of the models internalized by the other members. In certain families such restructuring may be considered a threat to the family, to each individual's conception of his experience of what the family relations are. In Laing's terms (1967:120):

Acts of spontaneity may be defined by the others as acts of destruction, of sickness and illness, because they entail the breakup of the internal family structures of the others. Each must sacrifice himself, therefore to preserve the (model) of the family.

In order to restructure his model the individual must seek to induce a restructuring of others' experience. Such restructuring may be resisted by what Laing terms "mystification" (1965), essentially, an action performed upon another to defend one's own person. A common form of "mystification" may be to deny that a person is responsible for his actions (or praxis) and attributes them to some disease (or process). In this respect, "madness" is the substituting of an impersonal series of events for what one person does and may constitute a systematic denial of another's experience. Bannister, (1960) using Kelly's terminology, describes this process as "serial invalidation of constructs".

The symptoms of such a "disease" may be anything that makes the family anxious about the independent behaviour of one of its members. Laing and Esterson (1964:31-49) in their discussion of the girl Maya describe how the customary needs of adolescence --

sexuality, aggression, self assertion, involvement outside the family -- may be considered a threat to the family status quo, and labelled a disease. (See Hughes & Marshall, 1971, for an earlier statement of their ideas.)

The individual who is placed in a situation in which he must break the family rules may actively deny his actions in order to remain within the family and maintain its stability. The family for its part confirms him in that invalidation by enacting psychiatric procedures which serve to disqualify his future actions.

This line of reasoning if followed up in actual research may enable us to breach the gap between these family studies and the labelling approach. What is really significant is that the ideas developed in the previous chapter on normalising are entirely consistent with the contemporary clinical research on the total family.

One problem with the labelling approach is that it has too often been interpreted exclusively in terms of the conspiratorial model. In part this is a direct consequence of the emphasis on the conspiracy to commit which is a major theme of Szasz' (1970) work on the institution of psychiatry, and of Laing (1967, see also 1971 and Laing and Esterson Sanity, Madness and the Family) and Cooper's (1969) on the family. These latter authors write of the violence which occurs in the family and interpret this violence as the denial of individuality by the attribution of the disease label.

While conspiracy and violence undoubtedly occur in some cases, it certainly is not true of all cases, and the overemphasis of the two processes has often led to a rejection of the societal reaction approach for the wrong reasons. In Chapter Three we emphasised the societal reaction over any conception of conspiracy or violence, and developed the notion of disorder as an accommodation between family members which may come to be labelled as schizophrenia. If this is treated as an hypothesis, we find it confirmed to a limited extent (given the methodological difficulties) in the clinical family studies, which suggests that this may be a fruitful line of enquiry.

d/ In summary

In this chapter an attempt was made to bridge the gap between the epidemiological studies and the clinical studies of the family. The necessity of a mediatory structure has long been recognised in the epidemiological research and it was suggested that the family could perform this function. The results of the examination of the clinical studies must necessarily remain incomplete for, while epidemiology has so often failed to conceptualise this interpersonal dimension, the clinical studies have themselves been plagued by a myopia which prevents them considering the importance of socio-cultural variables and their impact on the structure and functioning of the family.

We examined the trait studies and found them lacking because they failed to consider this socio-cultural dimension.

Further any significant progress in this area was hampered by a failure to conceptualise the family as an interacting whole, and by the undue and inaccurate emphasis that was placed on the plasticity of the child. When these two factors were considered a rather different conception of schizophrenia emerged and one which was consistent with the ideas developed in the previous chapter. Here schizophrenia is considered to be a "normal" pattern of response to particular types of family interaction. It was not clear in the clinical research why, if it is a normal pattern individuals should come to be labelled, and it was suggested that this problem could be resolved by examining the processes by which families seek to maintain the status quo and deal with threats to the status quo.

While this chapter dealt with the family it is not meant to imply that the family is the only group in which the schizophrenic's response may represent a 'normal' mode of action or communication. On the contrary attention was limited to the family because of the availability of information and what seems to be fruitful clinical concern with this particular group. The possibility is left open that any significant social group may develop patterns in which the schizophrenic's actions may be a 'normal' response. Having said this, our discussion has unearthed four elements which do seem to be essential characteristics of such groups:

(a) they are marked by intense emotional involvements of the members to the group as the defined reality, and by intense emotional

involvements amongst the individual members of the group;

(b) exit from the group is circumscribed by both internal and external pressures.

(c) the ability of the group to restructure the internal desires for change is severely limited;

(d) the ability of the group to deal with external crises and pressures for change is severely limited.

I want to emphasise these four points for they do seem to be common elements in the work of these three groups of writers (and in the work of Laing, 1967; and Esterson, 1971) reviewed. Rather than stressing pseudo-mutuality, or schisms and skews, or double-binds or attempting to synthesise all three (which would be illegitimate because they deal with essentially different levels of interaction within the family) I want to suggest that the impact of these theorists is, in the stress they place on first the attempts by members to maintain the status quo within the family and the implications this has for individuality; second, that the intrafamilial experience of the labelled schizophrenic provides a normal environment for his symptomatic behaviour; third, that the schizophrenic label defines a relationship amongst people, in the sense that while all of their actions may be bizarre by some objective criterion (cultural norms, or whatever) only one of them is labelled as ill, and this label serves to define and stabilise the relationship amongst them.

It would be a mistake to go further than this and formulate a theory of the process by which individuals come to be labelled as

schizophrenic because of the limitations of the data we have been dealing with. As noted above, very little attention has been paid to the provision of control groups or in establishing how prevalent these patterns are in the general population of families without labelled schizophrenics. Here the work of Cheek (1965) Farina (1960) and others is important for it is an attempt to establish the significance of these different patterns of disordered interaction and communication in the families of persons with other labelled social pathologies (delinquency) or psychosomatic disorders; but again these studies do not pay sufficient attention to isolating the possible affect of sociocultural variables, or of the impact of external crises on the structure and functioning of the family.

On this basis, future research must concern itself (a) with examining differences in the rigidity of family patterns and their association with different types of social pathology and (b) in examining the relationship between this variable and the significant variables which have been unearthed in the epidemiological studies. While it may be useful, for analytical purposes to deal with the family as a closed system as has been done in the clinical studies, a complete picture of these processes must examine the significance of extra-familial influences on the structure and functioning of the family.

IN CONCLUSION

It would be really satisfying in a work of this nature to attempt the grand synthesis -- a comprehensive theory which would take what is best from the mountains of clinical and epidemiological research reviewed and explain why people come to be labelled as schizophrenic. This idea is tempting but beyond the scope of this research; for while the ideas are there, the requisite data are absent. To arrive at definitive solutions would require the planning and execution of primary research and in the absence of such primary data we must avoid reanalysing the secondary data in the light of the insights unearthed, for to do so would leave us open to the same criticisms of 'missing data' and 'fallacy of the wrong level' which we made of the epidemiologists.

This is one reason we avoided reinterpreting Hollingshead and Redlich's findings in labelling terms; for while such an explanation is compatible with their results we do not have the requisite data in the original publication to support such an argument. A similar situation occurred in the previous chapter when adolescence was implicated in the process by which individuals come to be labelled. Here the temptation to synthesise is particularly great. One of the interesting features to emerge from the discussion of the epidemiological theories was that the onset of disorder seemed to be associated with that period in which the individual is

attempting to assert, or is customarily expected to assert his independence from his family. (see pp's 62 - 65)

When you examine Goldberg and Morrison's study (1963) carefully you find that while individuals who come to be labelled had no trouble in their school work, they failed to secure employment which was commensurate with their abilities. The same picture emerges in Rogler and Hollingshead's monograph in which they argue that while there seemed to be no difference between the family life of their identified patients and the controls, the disorder manifested itself on entry to the job market, or again at a point when the individual is expected to assume adult responsibilities. When we discussed these two findings and some others it was not clear whether the disorder was a result of the failure, or the failure a result of the disorder. In the previous chapter we opened up the possibility of re-interpreting these results in different terms. Here we are no longer concerned specifically with the presence or absence of a disorder but in examining the extent to which threats to the internal family stability may be met with the attribution of a disease label. The epidemiological data would seem to lend support to this hypothesis; but is clearly of the wrong order to be of more than suggestive value.

What I have tried to do in these pages is suggest that sociologists should go about doing the sociology of mental illness in a manner radically different than that employed in the epidemiological research. To this end sociologists must cast off the journeyman role they have so often assumed in psychiatric research and not content

themselves with advising the psychiatrist on the methodological requirements of large-scale survey research. This latter has so often meant an uncritical acceptance of the disease entity model when sociologists should be actively engaged in critical examination of current psychiatric models, for only in this way can significant progress be achieved. Thus, as we argued here, the recurrent difficulties which faced the sociologists as he undertook his epidemiological study: differences between prevalence and incidence rates; differential rates of admission to psychiatric facilities in different areas, and for different social classes; unreliability of diagnoses; and many more should not be treated as methodological difficulties to be overcome by more refined measurement techniques (as Dunham, 1965, suggests) but as investigable problems in themselves. This task was undertaken in these pages and was only achieved by critically examining the medical model of disorder and by seeking a viable alternative.

MacIver, in his much neglected book Social Causation, advises us that 'when a social phenomenon is defined by law, convention, or any institutional procedure, we should not assume that it can be referred to any one set of causes lying outside of the institutional system itself'. (1964:88) The examples he uses to illustrate his point are about crime; but the structure applies equally to the field of mental illness. "Schizophrenia" is an institutional definition. If it could be shown that the definition referred to an illness of entity independent of the definitional procedure the problem would not arise. If the presence or absence of this entity

were the only criterion for the diagnosis, or for the commitment to an institution there would be no qualms about seeking its causes in processes other than the definitional procedures. This is not the case. As Laing (1964), Leifer, (1971), Szasz (1970) and so many others have argued there are no objective, reliable, quantifiable criteria to refer to when making a psychiatric diagnosis. The existence of the entity is unproven and in the light of the community studies, and especially the work of Hollingshead and Redlich (1958) it appears that factors other than the presence or absence of the disorder are associated with the diagnosis and decision to commit. This is specifically the focus of the labelling approach which has produced considerable evidence to support this viewpoint and suggest that a schizophrenic diagnosis is not so much the recognition of an entity; but a social definition. To reiterate Schatzman's point -- the only thing certain about schizophrenia is that some people say that other people have it.

In the absence of objective criteria for the existence of the disorder, sociologists should not be concerned with accepting the institutional definition as the recognition of a fact for which they must seek causes outside this institutional framework. The proper focus of enquiry is with the social process by which individuals come to be diagnosed, or labelled, and part of this task was undertaken here.

The labelling approach, which has up until now been the only systematic critique of traditional psychiatric models, has

tended to overemphasise the institutional procedures associated with commitment and diagnosis. In so doing it has underemphasised the interpersonal processes prior to the actual labelling process. From Scheff's work we developed the idea of normalising which suggests that the actions which come to be labelled may be quite normal responses within the individual's family; and found this to be entirely consistent with the contemporary clinical interest in the family. The residual-rules of which Scheff speaks may be thought of as analogous to the rules which are invoked to establish membership within the family group (and do not refer to the cultural stereotypes as he suggests with his references to Nunally's work). These rules are phenomenologically unique for each family and it was suggested that the family's decision to seek psychiatric referral is associated with situations in which the individual is forced to breach these ground rules. The labelled actions were considered as an adaptive response on the part of the individual to invalidate his actions, in order to maintain the family stability.

The approach suggested here is not strictly a deviance approach to schizophrenia. The diagnosed patterns are considered as normal for particular families. They become deviance only when the label is sought, and then applied by a psychiatric functionary.

The approach to schizophrenia adopted here is thus radically different than that employed by the epidemiologists. The concern is not with the causes of an illness; but with the process by which individuals come to be labelled and as such the question of causes

or etiology is inappropriate.

This research can only serve as the starting point for a much fuller enquiry. It is difficult to estimate how much credence to give to the clinical studies which have an almost total neglect of the representativeness of their theories and findings. The theorists are in the process of deriving grounded theory but lack the systematic and constant comparative analysis which Glaser and Strauss (1967) stress is essential to the development of a formal or even a substantive theory. Consequently, in our analysis we specifically avoided emphasising any one of the specific intra-familial processes isolated by each group of theorists -- schisms and skews, double-binds, etcetera. Instead only those factors which seemed to be important for all of the writers were emphasised -- the concern of family members with maintaining homeostasis, the rigidity of the shared definition and so on (p. 194-195) -- and it was suggested that it is the examination of these factors which will provide a useful basis for future research. It is this interpersonal dimension which has been neglected in the epidemiological research and it is suggested that future research using this method should concentrate on the examination, delineation, and estimation of the prevalence of such patterns in the general population and their association with the attribution of not solely mental illness labels; but of deviance labels in general. Such research will provide a useful adjunct to the clinical research by specifying whether these processes are peculiar to the families of identified schizophrenics.

FOOTNOTES

Chapter One

INTRODUCTION

1. Returning to Wardle's suggestion in the light of Dohrenwend's comments it is a moot point whether sociologists will be able to perform this educative function if they cannot agree on what it is they are talking about.
2. The first chapter of Braginsky et al. (1969) gives an excellent discussion of this point, and relies heavily on the work of Kuhn (1970) in this area.
3. The Medical model has been described by Goffman (1968) and Scheff (1966). A useful summary is to be found in Hughes & Marshall (1971).
4. It must be emphasised at this point that there will be no attempt to argue that the identified patient does not act bizarrely (in Scheff's terminology "breaks residual rules"; 1966:31-32) but that such behaviour is intelligible to the observer by reference to the stated and unstated rules and meta-rules which are invoked to make actions mutually accountable those actions within the patient's family or other significant group.
5. Readers will note the similarity in approach and aims of this work and that of Douglas in his analysis of the literature on Suicide. (1967)
6. The method of data collection and analysis is analogous to that suggested by Glaser and Strauss in their Discovery of Grounded Theory (1967)

Chapter Two

1. His work confirmed Nolan's (1917) original research using different indices.
2. See Petras & Curtis (1968) who list a total of 350 studies dealing with social class and mental illness.
3. See McIver's discussion of a similar point with reference to explanations of crime and suicide statistics in his Social Causation.
4. Not however if we accept Srole's findings (1962) that the large majority of people in the urban environment are not well adjusted.
5. Homans and Inkeles both make this point that sociological theories involve implicit models of man.
See Homans, G.C. "Bringing men back in" American Sociological Review, 29:809-818
and Inkeles, A. "Personality and Social Structure" in Merton, R. K. et al (eds) Sociology Today
N.Y. Basic Books pp 249-276
6. Readers are asked to note the differences in emphases between this study and Dunham's pioneering research with Faris (1939). Dunham's research in this field is impressive because of his receptiveness to criticism and willingness to abandon or modify theories and models in the light of this criticism. The influence of Mary Bess Owen's (1941) critique of the Chicago study is apparent.
7. There is another fallacy involved here which involves the use of hospital statistics as indices of true disorder. We will deal with this problem on page 43 ff and in Chapter Three.
8. Robinson has been criticised for using "ecological" in a sense which has no generic relationship to "ecological theory". While the ecological fallacy is not a characteristic of all work in human ecology, I think Robinson's use of the term had much to do with the ecological research he reviewed which tended to commit the fallacy he described. As Robinson uses it, the ecological fallacy

refers to the problem of inferring individual from aggregate characteristics and is not meant as a criticism of ecological theory.

9. The type of hospital reporting procedure exemplified by Sainsbury and Grad (1962) is something of an exception.

Chapter Three

1. Gove has presented a detailed critique of Scheff's work in two recent papers (1970a, 1970b). We will be dealing with Gove's critique of Scheff in more detail as the chapter proceeds.

Chapter Four

1. See also the work of Bernstein (1958, 1960, 1962, 1964) which may prove a useful adjunct to Kohn's research.
2. This same criticism may be made of the papers edited by Mishler and Waxler in their Family Processes and Schizophrenia. 1968, N.Y. Science House.
3. This discussion relies heavily on Watzlawick et al (1968:56-58)

5. A particularly concise example of the double-bind is given by Laing in his book The Self and Others (1961, London, Tavistock). This particular piece reminds one of a stand-up comic's act; and would be amusing if it were not so tragic. This quotation is taken from Watzlawick's Anthology of Human Communication. (1964:44-45) Watzlawick's work is particularly useful for those readers unfamiliar with the concept of the double-bind for the piece is recorded on a tape which accompanies the work.

Identified patient: Well, when my mother sometimes makes me a big meal and I won't eat it if I don't feel like it.

Father: But he wasn't always like that, you know. He's always been a good boy.

Mother: That's his illness, isn't it doctor? He was never ungrateful. He was always polite and well brought up. We've done our best for him.

Identified patient: No, I've always been selfish and ungrateful. I've no self-respect.

Father: But you have.

Identified patient: I could have, if you respected me. No one respects me. Everyone laughs at me. I'm the joke of the whole world. I'm the joker all right.

Father: But, Son, I respect you, because I respect a man who respects himself.

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