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Psychosis in a Developmental Psychopathology Context: A Factor Analytic Study of Schizophrenia in Adolescent Psychiatric Inpatients

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PSYCHOSIS IN A DEVELOPMENTAL PSYCHOPATHOLOGY CONTEXT: A FACTOR ANALYTIC STUDY OF SCHIZOPHRENIA IN ADOLESCENT PSYCHIATRIC INPATIENTS

by

Paul R. Adams

A dissertation submitted in partial fulfillment
of the requirements for the degree

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DOCTOR OF PHILOSOPHY

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DEDICATION

I cannot repay
what they gave
so freely,

Tangible help
and unseen
gifts of
love;

Sometimes simply
being there for me
and those I cherish.

I cannot repay,
except to give
as they have given,

And that I vow to do.

But though I can't
repay their help
I can express
my heartfelt
"Thanks".

In token of love
and gratitude,
this work is dedicated
to our parents

F. Dale Needham
Norrine Williams Needham

Jack R. Adams
Laurel Spaulding Adams

Paul R. Adams

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GARY H. KIGER, Associate Professor, Sociology
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To my wife, MARCIA ANNE NEEDHAM ADAMS, I owe a debt of gratitude that I cannot repay except by striving to love her as she loves me. I am grateful to her as companion, friend, lover, and mother of our children. Without her support, much of what I have accomplished personally and professionally would not have been possible. The security of knowing her love for me has been the foundation from which I could grow.

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Paul R. Adams

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ABSTRACT

Psychosis in a
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by

Paul R. Adams, Doctor of Philosophy

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Major Professor: Gerald R. Adams

Departments: Psychology
Family & Human Development

Demographic, historical, psychometric, and clinical data were obtained from the psychiatric files of all patients manifesting schizophrenic symptomatology who were hospitalized in an adolescent psychiatric facility during a five year period (N= 71). Factor analysis of the usable data resulted in three interpretable factors, which included: (1) aggressive behavior; (2) disturbed family functioning; and, (3) thought disorder. Age of first hospitalization correlated positively with factor three.

The results provide support for concerns expressed by a number of scientists and clinicians that schizophrenia may not be a discrete, unitary disorder; and that uncritical downward extension of adult diagnoses to adolescents and prepubescent children may be questionable. The results further suggest that current DSM-III and DSM-III-R subtypes of schizophrenia (which are clinically derived and symptom based), are not validated by empirically derived subtypes that include objective indices of behavior along with clinical symptoms. The correlation of "age of first hospitalization" with one of the three factors suggests that developmental level at the onset of illness may represent an important mediating variable in the severity and prognosis of certain subtypes of schizophrenia.

(238 pages)

CHAPTER I

INTRODUCTION

Developmental Psychopathology

"Developmental psychopathology" is a relatively new field of study within psychology. As a discipline it takes the insights of developmental psychology and those of abnormal psychology, or psychopathology, and attempts a synthesis between the two (Cicchetti, 1984). The rationale for such a synthesis is manifold.

Theorists studying psychopathology contend that many disorders have their antecedents in childhood or adolescence (Kolb & Brodie, 1982; Sroufe & Rutter, 1984). Personality disorders, to use one example, are generally thought to be psychogenic disorders etiologically linked to arrested or deviated development of personality (Kernberg, 1975; Kolb & Brodie, 1982; Manning, 1982; Masterson, 1981; Millon, 1981, 1983). Adult psychopathology is not seen as blossoming suddenly, in the absence of a prior context, but is often viewed as the result of an interaction between genetic, biochemical, environmental, interpersonal, and intrapsychic factors over a prolonged period of time hypothesized to have begun in the early formative years (though not always directly traceable to those early years) (Sroufe & Rutter, 1984).

A separate, but related issue, has to do with how disorders diagnosed in adulthood manifest themselves

during childhood or adolescence (Achenbach, 1982). Are they evidenced in any fashion that can be consistently and meaningfully differentiated from "normal" development? Are there behaviors or problems evident in childhood that have predictive value for diagnosing adult disorders? Does childhood psychopathology inevitably result in adult dysfunction? Do all adult disorders ultimately stem from childhood experience, and if not, which disorders do and do not? Another critical issue has to do with the effect of psychopathology that occurs during childhood or adolescence on the subsequent course of development (Achenbach, 1982; Sroufe & Rutter, 1984). How might a child/victim compensate for the effects of the pathology? Do normal developmental issues and tasks still occur with the same sequence and timing? What distortions of development are directly or indirectly linked to the illness? Are the effects of such "derailment" of normal developmental processes permanent, or are there compensatory mechanisms that restore normal functioning once the illness has abated or decreased in intensity? Does age of onset of a disorder correlate with prognosis? If adult dysfunction is linked to childhood psychopathology, how much of that dysfunction can be attributed to the continued manifestation of the original disorder, how much to the derailment of normal

developmental processes, and how much to the interaction of the two?

Psychodiagnosis in Children and Adolescents

A number of disorders seen both in children and adults share common symptoms and have the same diagnostic label; depression and schizophrenia are two major disorders that fall into this category (American Psychiatric Association, 1980, 1987). But whether or not such disorders are even the same entity in adults, children, and adolescents has yet to be established (Garber, 1984). For instance, little is known about the similarities and the critical differences that may exist between depression as evidenced in a prepubescent child, and depression in an adult of the same gender (Malmquist, 1983). There may exist profound etiological differences, differing responsiveness to various treatment modalities, different mediating variables, and critical differences in eventual outcome (Rutter, 1985b).

In a discussion of the broad category of "adolescent psychopathology", Miller (1980) suggests that current diagnostic nomenclature, as applied to adolescents, has come about by default. Current diagnostic categories, except for those disorders historically seen as limited to childhood (e.g., Attention Deficit Disorder), have been

generated from clinical observation and research with adult populations. Few empirical studies have been undertaken to determine the similarities or differences between adult and adolescent disorders. Noting that diagnoses for adolescents tend to be "downward extensions" of adult disorders based on similarity of symptoms, Miller (1980) states:

> It may be that these similarities are more apparent than real and that phenotypical similarities obscure more fundamental differences in genotypes. (p. 162)

Substantially different disorders that happen to share a few symptoms in common may have been labeled as the same disorder and be perceived as the same in all essentials. However, schizophrenia in children (to use another example) may be an entirely different disorder (or class of disorders) than schizophrenia in adults (Achenbach, 1982). The medical model that prevails in psychiatry unfortunately lends itself all too readily to the assumption of equivalence of disorder, due to similarity of symptoms, regardless of the age of the patient.

A final major area of consideration for developmental psychopathology has to do with the effect of the continuing development and growth of the child on any

existing psychiatric disorders (Achenbach, 1982). This is a significantly different question than the one posed previously regarding the effect of psychopathology on the course of development. Will a child or adolescent's continued development increase their effectiveness in coping with the disorder? Will some disorders become less debilitating as a function of age and maturity? Does the onset of puberty alter the course or change the nature of a disorder?

Questions might even be raised about how ongoing development affects the efficacy of different therapeutic interventions. Some forms of therapy may be more or less effective, depending on the client's developmental level (Kendall, Lerner, & Craighead, 1984). Clearly those that depend to any degree on the child's intellectual functioning are likely to be affected by the stage of cognitive development.

None of the questions posed above have been satisfactorily answered, but the attempt to explore these and related issues forms the core of developmental psychopathology. Of particular concern for the purposes of this study is the issue of the equivalence of a given psychiatric disorder that may carry the same diagnostic label for adults, adolescents, and children. The major diagnostic category of "schizophrenia" is the primary focus of attention in the present investigation.

CHAPTER II

LITERATURE REVIEW

Introduction

Schizophrenia is a disorder that commonly makes its first appearance in adolescence or young adulthood (American Psychiatric Association, 1980, 1987). Having an adolescent diagnosed as "schizophrenic" may have enormous impact on parents, siblings, and other relatives (Arieti, 1979). While "mental illness" of any sort tends to be difficult for patients and their families (Bernheim, Lewine, & Beale, 1982), a level of dysfunction severe enough to require psychiatric hospitalization brings in its wake a host of potential psychoemotional and social consequences (Rabkin, Gelb, & Lazar, 1980). Schizophrenia may be particularly difficult because it can so completely disrupt an individual's functioning, and may lead to drastic alterations in personality while the person is actively psychotic.

Current treatment for schizophrenia tends to be largely palliative; recurrent episodes of illness are likely; the course of the illness is variable; and prognosis is uncertain. This combination of factors compounds the difficulty of coping with schizophrenia for families, and also for the adolescents who suffer from behaviors they can neither understand nor control.

Definition of Schizophrenia

In this study the definition of schizophrenia will be that used in the Diagnostic and Statistical Manual of Mental Disorders, Third Edition (American Psychiatric Association, 1980), hereafter referred to as the DSM-III. (However, the definition of schizophrenia found in the Revised Third Edition, or DSM-III-R [American Psychiatric Association, 1987], is virtually the same; so the results of this study may be seen as equally applicable using the DSM-III-R criteria).

Regarding the diagnostic validity of the DSM-III as compared with six other current diagnostic systems, Endicott, Nee, Cohen, Fleiss, and Simon (1986) conclude the following:

> A research outcome of our findings is that investigators who wish to study samples of subjects with schizophrenia will probably do as well (or as poorly) using the DSM-III criteria as any of the others we have studied. (p. 19)

Fenton, Mosher, and Matthews (1981), after completing a similar study, stated that choosing one diagnostic system over another cannot be data-based, at this point in our knowledge. They contended that none of the existing systems have established construct validity, and caution

must be used to avoid uncritical adoption of any particular diagnostic system. However, despite the fact that no single system has proven itself to be superior, the interrater reliability for diagnosis of schizophrenia in children was found to be .79 in a mean weighted kappa combining results of several studies (although interrater reliability was much lower for subcategories of schizophrenia) [Werry, Methven, Fitzpatrick, & Dixon, 1983].

There is a time criterion that is important to note in the diagnosis of schizophrenia, as specified in DSM-III. If symptoms of the illness have persisted for at least six months (in either prodromal, active, or residual phases), then the diagnosis of schizophrenia can be used. However, if schizophrenic symptoms have been present for less than six months, another diagnostic code must be used (i.e., if symptoms have been present for less than six months, but more than two weeks, the diagnosis of "schizophreniform disorder" is used; if symptoms have been present for less than two weeks, the diagnosis of "atypical psychosis" is used).

One point should be noted regarding the six-month duration of illness (in either prodromal, active, or residual phases). At the psychiatric hospital from which

the data were obtained, the diagnosis of schizophrenia was used with caution for adolescents. The clinical staff were concerned about the possible stigmatization and adverse social consequences that might result from such a diagnosis. Because of this concern, there was strict adherence to the six month criterion. Patients who displayed schizophrenic symptomatology, but who failed to meet the time criterion, were given the alternate diagnoses recommended.

However, this practice by clinical staff resulted in relatively few youth who were given a formal diagnosis of schizophrenia. It also resulted in a sample size that the researcher judged smaller than was desirable for the present study, particulary since it was anticipated that a large number of variables would be entered into the factor analysis. The scarcity of adolescents in the sample necessitated inclusion of all subjects who displayed schizophrenic symptomatology, regardless of whether or not they met the time criterion. This decision was made only after consulting with the psychiatrist who had worked in this unit for most of the five years during which these patients were admitted, and verifying that she had adhered rigorously to the time criterion specified. Including all patients with schizophrenic symptomatology, even when they

do not meet the time criterion for schizophrenia, is supported by Kolb and Brodie (1982), in the tenth edition of their text on psychiatry, where they state that:

> Long clinical experience does not bear out the narrow time-limited concept of schizophrenia presented by the American classifiers, which is not accepted internationally. (p. 345)

There appears to be some question about the real difference between schizophrenia and schizophreniform disorders. The American researchers who participated in the cross-cultural International Pilot Study of Schizophrenia, reported no difference in outcome between those subjects diagnosed as "true" schizophrenics and those diagnosed as schizophreniform (Sartorius, Jablensky, Stromgren, & Shapiro, 1978). While this study antedates the 1980 advent of DSM-III, the sole current criterion (both in DSM-III and DSM-III-R) for differential diagnosis is the duration of illness rather than distinctive patterns of presenting symptoms. Randels, Villeponteaux, Marco, Shaw, and McCurdy (1982) state explicitly that:

> Based on a cross-sectional evaluation of a patient, Schizophreniform Disorder and Schizophrenia are indistinguishable. (p.346)

Additional support for combining subjects (for purposes of data analysis) regardless of whether they carry a diagnosis of "schizophreniform disorder" or "schizophrenia" comes from Kolb and Brodie (1982), who declare emphatically:

> .... there is no question that the majority of the schizophreniform disorders eventually will be reclassified as schizophrenia. (p. 459)

Questions About Schizophrenic Diagnoses in Children and Adolescents

Attempting a comprehensive review of the literature on schizophrenia would be a task beyond the scope and purpose of this study. The sheer volume of research being done in this area is enormous. As an illustration, in the 1986 edition of Psychological Abstracts (Vol. 73), there are 771 references to current papers on some aspect of schizophrenia. For the purposes of this study, only two major areas of schizophrenia were examined: (a) the continuity of the disorder, especially child/adolescent versus adult onset schizophrenia; and, (b) the homogeneity of schizophrenia as a discrete diagnostic entity.

Schizophrenia is one disorder that has typically been seen as equivalent in children, adolescents, and adults.

Indeed, the current psychiatric nosology in the Diagnostic and Statistical Manual of Mental Disorders (3rd ed.) states the following:

> Because the essential [italics in the original] features of Affective Disorders and Schizophrenia are the same in children and adults, there are no special categories corresponding to these disorders in this section of the classification [the section pertaining to childhood and adolescent disorders]. For example, if a child or adolescent has an illness that meets the criteria for Major Depression, Dysthymic Disorder, or Schizophrenia, these diagnoses should be given, regardless of the age of the individual. (American Psychiatric Association, 1980, p. 35)

This statement presupposes a virtual equivalence of the disorder labeled schizophrenia, regardless of the age of the patient or the age of onset. The Revised Third Edition, or DSM-III-R (American Psychiatric Association, 1987), contains an almost verbatim repetition of this statement (p.27). Schwartz and Johnson (1985) state, even more emphatically:

> It is generally agreed by virtually everyone working in the field today that schizophrenia in childhood is little different from schizophrenia in adults. (p. 143)

In reality, however, such emphatic statements belie the fact that little is known about the similarities and differences in the course and prognosis of a psychiatric disorder occurring in childhood and the "same" disorder occurring in adulthood (Achenbach, 1982; Garber,1984; Gelfand & Peterson, 1985). The disorders in most psychiatric nosology systems are classified by symptoms rather than etiology. The statement regarding the "essential features" of schizophrenia mentioned in the DSM-III (and later in the DSM-III-R) is clearly a reference to the shared symptoms between the disorder as manifested in children and as manifested in adults. It may be logical to classify according to symptoms, but the logic of classification does not inevitably mean that the disorder is precisely the same in different age groups, especially in a disorder as complex and multifaceted in its presentation as schizophrenia.

"Discrete Disorder" versus "Syndrome" Conceptualizations of Schizophrenia

Carpenter, Heinrichs, and Wagman (1985) offer the following thought provoking insights on the heterogeneity of schizophrenia:

> Schizophrenia may be medicine's prime example of a diversity of clinical manifestations within a

> single diagnostic class. Remarkable differences occur between cases in factors such as age of onset, constellation of psychotic features, course of illness, premorbid personality, prognostic features, presence of deficit symptoms, neurologic dysfunction, response to treatments, insight into illness, and the extent to which the personality is torn asunder. (p. 25)

Questions regarding the equivalence of the disorder in children, adolescents, and adults are confounded by the very real possibility that the label "schizophrenia" may be descriptive of a class of disorders rather than a discrete entity (Bellak, 1980; Randels et al., 1982; Strauss & Bellak, 1979; Strauss & Docherty, 1979). While there are accepted nosological subtypes of schizophrenia, the observation has been made that even those subtypes may reflect classes rather than single disorders (Bashina, 1980; Bellak, 1979, 1980; Carpenter et al., 1985; Carpenter & Stephens, 1979; Gur'yeva, Gindikin, & Isachenkova, 1980; Harding & Strauss,1985; Houlihan, 1977; Randels et al., 1982; Strauss & Bellak, 1979; Strauss & Docherty, 1979).

In other words, what is now currently diagnosed as "paranoid schizophrenia" may well consist of several disorders differing in etiology, course of illness, responsiveness to treatment, and eventual prognosis.

Indeed, even the DSM-III refers to schizophrenia as a "group of disorders" and states in a footnote that, "Schizophrenia is most likely a group of disorders of differing etiologies" (American Psychiatric Association, 1980, p. 181).

The DSM-III-R does not contain this statement; rather, it talks of schizophrenia as if it were a discrete disorder. However, it does state, in a glossary definition of "syndrome," that a syndrome is:

> A group of symptoms that occur together and that constitute a recognizable condition. "Syndrome" is less specific than "disorder" or "disease." The term disease generally implies a specific etiology or pathophysiologic process. In DSM-III-R most of the disorders are, in fact, syndromes. (p. 405, italics added)

It seems plausible that if there are indeed verifiable subtypes within the schizophrenia syndrome, there may be one or more subtypes within which age of onset is a significant variable. Several researchers have attempted to isolate specific subtypes of schizophrenia in children and adolescents (Bashina, 1980; Gur'yeva et al., 1980; Lewine, 1980; Loranger, 1984). These clinical studies appear to suggest promising leads and generate interesting hypotheses. However, as yet there is no

theoretical consensus between researchers, nor is there any consistency in the subtypes being identified.

Lewine (1980) found that age of onset of schizophrenia differed in males and females, with males having an earlier age of onset than females. Lewine (1980) also noted that males are also hospitalized earlier than females, although there were no differences in the time difference between age of onset and age of hospitalization (i.e., dysfunctional symptoms were not "tolerated" longer for either males or females). Similar results were obtained by Loranger (1984), who found that males displayed earlier age of onset whether measured by first treatment, first hospitalization, or by the immediate family's first awareness of psychotic symptoms and signs. This difference in age of onset between males and females caused both Lewine (1980) and Loranger (1984) to speculate about possible gender related differences in the etiology of schizophrenia. Beitchman (1985) found that regardless of gender, earlier age of onset of schizophrenia correlated with poorer prognosis, a finding supported by Shmaonova, Liberman, and Vrono (1980).

As noted previously, many researchers and theorists have challenged the idea that schizophrenia is a discrete disorder. Medical literature furnishes us with examples

of diagnostically "discrete" entities that later research has shown actually to be diverse disorders linked only by common symptomatology:

> In other branches of medicine, the sorting, defining, and regrouping of syndromes has often changed radically as new information became available. The generalized swelling known as dropsy was, over the years, found actually to represent several different disorders primarily involving entirely different systems. Certain blood dyscrasias, such as anemia, have been found actually to reflect an extremely large number of diverse disorders with a variety of different complex mechanisms. (Strauss & Bellak, 1979, p. 508)

The supposition that schizophrenia may be a syndrome representing several disorders has not yet been empirically substantiated. Nevertheless, there is persuasive evidence that schizophrenia as we presently define it represents a heterogeneous rather than a homogeneous classification. Carpenter et al. (1985) suggest three possible models that may account for such heterogeneity.

The "single disease-multiple site" model is likened to syphilis. A single disease entity affects different anatomical sites in different victims, resulting in differing symptoms and pathophysiology. This model

assumes a single causative factor and a specific treatment that could be applied to all schizophrenic patients.

A second model postulates a single disease, with a common pathogenic process, that is attributable to the interaction of multiple etiological factors. The factors are assumed to make different contributions to each individual case, thus resulting in the observed variability between cases. This model assumes multiple etiologic factors and combinations of factors interacting to produce the final outcome of schizophrenia. Treatment strategies would focus on identifying the individual components and targeting the treatment to specific components. However, this model also implies that:

> If the multiple disease manifestations result from the perturbations of a final common path, then a unitary treatment approach is plausible. (p.26)

Carpenter et al. (1985) indicate that these two models both assume a single disease entity, with the second model being merely a more complex extension of the first. They offer a third model that is described as a "multiple disease" conceptualization of schizophrenia. This model suggests that schizophrenia represents a

syndrome rather than a discrete entity. This model assumes multiple disorders with different etiologic factors, pathogenesis, clinical manifestations (with some similarity, but with significant differences for each discrete disorder), and a different prognosis for each "type." As each disease entity is identified, investigators may be guided by the principles of the first two models as they search for curative treatments. But Carpenter et al. (1985) caution, "Premature application of a disease-entity model to a heterogeneous syndrome is perilous" (p. 26).

The third model, the "multiple disease" hypothesis, suggests approaches to treatment quite different from the first two models:

> No single treatment approach is expected to be optimal for all schizophrenic-syndrome patients, but a series of therapeutic strategies is anticipated. Finding the appropriate treatment for each patient rather than the blanket application of a standard treatment is critical to the clinical task. (p. 26)

Strauss and Docherty (1979), argue that the multiple disease model, with its concept of discrete subtypes within a syndrome, increases the likelihood of finding effective treatment methods specific to individual

subtypes. They use the medical analogy of "infectious diseases":

> If all such illnesses had been grouped together under one diagnosis, the efficacy of penicillin-- limited as it is to certain infectious agents-- might have been completely overlooked. (p. 447)

While noting that there is, as yet, no conclusive evidence favoring any one model over another, Carpenter et al. (1985) point out that the first two models assume that schizophrenia is a proper disease class. This assumption suggests that schizophrenia is a disease entity with enough core features to differentiate it reliably from other disease entities. The syndrome model is somewhat more conservative in suggesting that until actual disease entities are identified, classification simply outlines the boundaries of the syndrome and "would not be expected to reflect within-class homogeneity" (Carpenter et al., 1985, p. 26).

Variables Correlated with the Onset of Schizophrenia

Contributing to the notion of schizophrenia as a syndrome are the diverse lines of research on possible

causal or contributory factors associated with schizophrenia. These include genetic studies (Cazzullo & Invernizzi, 1985; Gottesman & Shields, 1982; see Stone, 1980, for an integrative review of genetic research on schizophrenia and other major disorders); neurochemical and nueroanatomical research (Black, Yates, & Andreasen, 1988; Feinsilver, 1986; Johnson, 1989; Strauss & Carpenter, 1981); other biological factors (e.g., metabolic abnormalities, brain lesions, viral diseases, congenital defects) [Black et al., 1988; Kaplan, Freedman, & Sadock, 1980; Kety, 1975; Marcus, Hans, Byhouwer, & Norem, 1985; National Institute of Mental Health, 1984; Strauss & Carpenter, 1981]; dysfunctional family systems (Anderson, Reiss, & Hogarty, 1986; Lidz, 1978; Lidz & Fleck, 1985); distorted communication systems within the family, particularly the "double-bind" (Bateson, Jackson, Haley, & Weakland, 1956; Lidz & Fleck, 1985); and environmental stressors (Nuechterlein & Dawson, 1984; Strauss & Carpenter, 1981; and see Stone, 1980, pp. 90-95, for a remarkably lucid discussion of the interaction between environmental stressors and genetic vulnerability for schizophrenia). In addition, developmental psychologists have attempted to integrate theory and research findings into a developmental framework

(Achenbach, 1982, 1985; Cicchetti, 1984, 1988; Garber, 1984; Gelfand & Peterson, 1985; Lidz, 1978; Miller, 1980; Neale & Oltmanns, 1980; Rutter, 1985a, 1985b; Sroufe & Rutter, 1984).

If we identify the factors that might conceivably contribute to the onset of a schizophrenic episode, they could be clustered into five major areas: genetic factors, environmental stressors, familial patterns of interpersonal communication and interaction, a broad range of physiological factors, and what might be thought of as cognitive factors (including intrapsychic phenomena). At present it is not known which of these are salient causal factors, which are just correlated factors, and which are merely blind alleys. It appears entirely plausible that one or all may be implicated in the multifaceted syndrome we currently designate "schizophrenia."

Variables Affecting the Severity of Schizophrenia

In addition to the factors mentioned above, one other issue must be considered when discussing schizophrenia. This disorder, or group of disorders, varies not only in symptom manifestation, but in the severity and course of the disorder in different individuals. Even when

monozygotic twins are studied, and any potential genetic variation thereby controlled for, the concordance rate for schizophrenia is only 50% (Randels et al., 1982). If genetic influences were the sole determinants of schizophrenia, one would anticipate a concordance rate of 100% for monozygotic twins, rather than only 50%. This suggests the possibility that there is an interaction effect between two or more of the factors involved in schizophrenia. Supporting the notion of possible interaction effects is the evidence for cultural variations in the rate of recovery in schizophrenia, which Randels et al. (1982) link to cultural conceptions about the relative permanence and "curability" of mental illness.

Studies of the long-term outcome of schizophrenia, cited in Randels et al. (1982), indicate that roughly 25% of all persons diagnosed as schizophrenic make a full remission, 50% make a partial social recovery, and only 25% become chronically ill. This much variation in outcome suggests several explanatory hypotheses, two of which have been previously mentioned: etiologically distinct disorders may be involved; or there are interaction effects (as mentioned above). A third hypothesis is that there may exist differences in coping mechanisms and adaptive responses that serve to mitigate the impact of the disorder.

A meta-analysis by Aylward, Walker, and Bettes (1984) raises the possibility that intelligence may be one such mitigating factor, though certainly not the only possible mediating variable. Aylward et al. (1984) indicate that, "higher premorbid and postmorbid IQs (obtained during hospitalization) are related to better clinical outcome for schizophrenic patients" (pp. 447-48). The same authors also found that patients with lower IQs tended to be hospitalized at a substantially earlier age than patients with higher IQs. Aylward et al. (1984) conclude that:

> The studies reviewed here suggest a fairly consistent relationship between higher IQ and more positive outcome for schizophrenic patients. (p. 449)

One conclusion that might reasonably be drawn from the above discussion is that there may indeed exist subtypes of schizophrenia, with different etiologies, and different responsiveness to potential mediating variables. The interactive effect of differing types of schizophrenia and these potential mediating factors may account for the wide variation in symptom manifestation and course of illness that has been documented for schizophrenia.

Scott and Carran (1987), in a discussion of mental retardation, include some observations that seem equally germane to the study of schizophrenia:

> The single agent medical model has given way to multiple risk factor models in modern epidemiology. A primary contribution of these models has been recognition of the interactive nature by which risk variables can be viewed as predictors of a disorder.... Such a multivariate and sophisticated assessment of mental retardation [or schizophrenia] is directly needed to project causes beyond the simple medical pathogen-disease model. (p. 803, italics in the original)

Scott and Carran (1987) also indicate that well-conceptualized, descriptive epidemiological studies are vital if we are to understand disorders as complex as mental retardation (or schizophrenia). They suggest that such studies:

> .... should begin with a view of mental retardation [or schizophrenia] as a complex set of disorders, with multiple etiological factors that collectively contribute to... [the observed outcomes]. (p. 803)

Differential Diagnosis Through Psychological Testing

Efforts at accurate diagnosis of schizophrenia are many (American Psychiatric Association, 1952, 1968, 1980, 1987; Bleuler, 1950; Endicott et al., 1986; Feighner,

Robins, Guze, Woodruff, Winokur, & Munoz, 1972; Schneider, 1959; Spitzer, Endicott, & Robins, 1975; World Health Organization, 1973, 1975). One method that has been attempted in diagnosing schizophrenia is through the use of psychological tests (Anastasi, 1982; Lezak, 1976; Rapaport, Gill, & Schafer, 1968; Shaw & Holmstrom, 1982; Wolman, 1978). The assumption underlying this method is that schizophrenia will affect the victim's cognitive functioning, personality, and behavior in ways that are measurably different from the responses found in people not afflicted with this disorder. Such a notion has a strong appeal to those who have worked closely with schizophrenic patients, especially in hospital settings. In such settings, one can witness firsthand the profound changes that occur in the acute phase of this disorder, and the massive deterioration that may occur in the wake of schizophrenia that becomes chronic.

In the attempt to understand and differentiate disorders, researchers and clinicians have used psychological tests of many varieties. In their ability to differentiate broad nosological categories such as "depression" versus "schizophrenia," psychological test batteries have proved useful (Anastasi, 1982; Lezak, 1976; Rapaport et al., 1968; Shaw & Holmstrom, 1982; Wolman, 1978). However, the use of a <u>single</u> instrument, even one as relatively sophisticated as the Minnesota Multiphasic

Personality Inventory (MMPI), has had far less success than expected in differential diagnosis. In a recent review of the literature on the use of the MMPI as a diagnostic tool, Walters (1983) concludes that when used alone the MMPI cannot reliably differentiate schizophrenic versus non-schizophrenic patients.

> Despite over 100 MMPI research studies on schizophrenia, it is somewhat surprising, although revealing, that little is known about the MMPI correlates of schizophrenia. (Walters, 1983, p. 240)

Berg (1986) makes a similar observation regarding the limitations of the Rorschach; indicating that it can be a useful diagnostic tool, but that Rorschach results alone are not conclusive. Walters (1983) suggests that one possibility for future research is to attempt classification of schizophrenics into subgroups on the basis of their MMPI profiles, though he acknowledges that more diagnostic accuracy and reliability could be obtained by combining MMPI data with other psychometric, behavioral, clinical, and demographic data (Walters, 1983).

Summary

In summary, the research and clinical literature both indicate that schizophrenia often makes its first

appearance in adolescence. However, diagnostic confusion may arise when uncritically applying adult-oriented, symptom-based nosological systems to non-adult patient populations. It has not yet been empirically verified whether schizophrenia is the same disorder (or syndrome) in children/adolescents as in adults, or whether it represents a different disorder (or syndrome) in children/adolescents that only happens to share common symptoms with the adult disorder (or syndrome).

Further, there is reason to question the homogeneity of patient populations, regardless of age, to whom the diagnostic label "schizophrenia" is applied. The present diagnostic criteria used in DSM-III (and DSM-III-R) may be imprecise. In fact, it is frankly stated in DSM-III that the term "schizophrenia," while commonly used as if describing a discrete entity, will likely prove to be a group of disorders. There is reason to suspect that several factors may be etiologically linked to the different disorders presumably clustered in the schizophrenic syndrome. These include such things as genetic, neurological, and biochemical variables, and also include non-biological variables such as dysfunctional family systems and distorted intra-familial communications. At present, it is not known whether any of these possible etiological variables is "necessary and

sufficient", or if the interaction of two or more variables is required for any given "subtype" of schizophrenia. It appears probable that there are interactive effects between the etiologic variables. There also exists evidence supporting the notion of mediating variables such as IQ that affect the course and severity of schizophrenia.

CHAPTER III

THEORETICAL CONCEPTUALIZATIONS

The Role of Theory

It is a truism in the social sciences that research should be theory based. Indeed, research is typically seen as the testing and extension of theory. While there exist a number of theories pertaining to various aspects of schizophrenia, no single published theory seems broad enough in scope to encompass the numerous variables that appear essential for understanding schizophrenia. Textbooks on schizophrenia may include sections on genetics, family dynamics, and intrapsychic factors often associated with schizophrenia, but the theoretical discussions tend to be comparatively sparse. Typically, discussions of theory are limited to historical treatises on early theoretical notions of schizophrenia, or to explications of "mini-theories" that address only a limited aspect of schizophrenia (e.g., the double-bind "theory" of Bateson et al. 1956).

No single theory yet published persuasively synthesizes knowledge about genetic factors, family dynamics, neurological and biochemical factors, environmental stressors, intrapsychic and cognitive processes, developmental stages and tasks, coping mechanisms or adaptive strategies, and cultural factors

(and the interactive effect of all the above) into an integrated and testable framework. One might question whether the present state of psychology and psychiatry as scientific disciplines is sufficiently advanced for such a "grand theory" of schizophrenia to be really possible. Indeed, Kazdin (1989) contends that in the whole broad field of developmental psychopathology, there are currently only "mini-theories" available to guide research. Johnson (1989), referring specifically to schizophrenia, states:

> Most of the major etiological models of mental illness are too incomplete to cover adequately the complexities of schizophrenia, and they do not provide a good fit with current data. (p. 553)

Given that no "grand theory" exists, and none of the available "mini-theories" appear to offer a framework sufficient to encompass the intent of the current study, a blending of concepts from several "mini-theories" into a coherent framework for guiding the research was undertaken. The result of this synthesis was a series of assumptions about developmental psychopathology in general, about schizophrenia in particular, and some assumptions that challenge current theoretical conceptualizations of schizophrenia.

Theoretical Concepts About Schizophrenia Underlying this Study

The mini-theories and concepts underlying this study are embryonic at best, and are offered as theoretical assumptions rather than formal hypotheses. In the best interests of scientific objectivity, it was deemed appropriate to identify, as explicitly as possible, the major theoretical assumptions underlying this study, as derived from the literature review.

Clinical and research literature both offer increasing evidence suggesting that schizophrenia is a syndrome that includes several disorders that will eventually be identified as etiologically distinct. It seems likely that those disorders are dependent upon multiple interacting factors. The interactive effects can result in greater or lesser degrees of "severity" of schizophrenia as changes occur in the nature and extent of the factors involved. It appears that there may be mitigating factors that affect the degree to which an individual's life is disrupted by schizophrenia.

If Lidz's (1978) developmentally oriented speculations are correct about the relationship between schizophrenia and "schismatic" and "skewed" families, then one would expect to find a substantial degree of family disturbance among the youth in this study. A number of

potential measures of disturbed family functioning are included in the data (e.g., incidence of foster care, abuse by parents and other caretakers, parental visitation while a youth was hospitalized).

Schizophrenia is a disorder that typically makes its first appearance in adolescence or young adulthood. That single fact in isolation would suggest that developmental variables may be critical in the onset of this particular disorder. One might anticipate that age of first hospitalization, even though a crude measure of developmental level, may correlate with at least one of the "subtypes" of schizophrenia.

Figure 1 contains a conceptual model that attempts to summarize the major points enumerated. This model posits a conceptualization of schizophrenia as a disorder that is complex, multifaceted, and extremely difficult to study. The more complex conceptualization suggested in the model seems to reflect with appreciably greater accuracy the reality of schizophrenia as portrayed by an increasing number of scholars in both clinical and research literature. These concepts challenge the theory, based on the "medical model" of schizophrenia as a unitary disorder, which is the same in children, adolescents, and adults. Indeed, the single disease model begins to appear

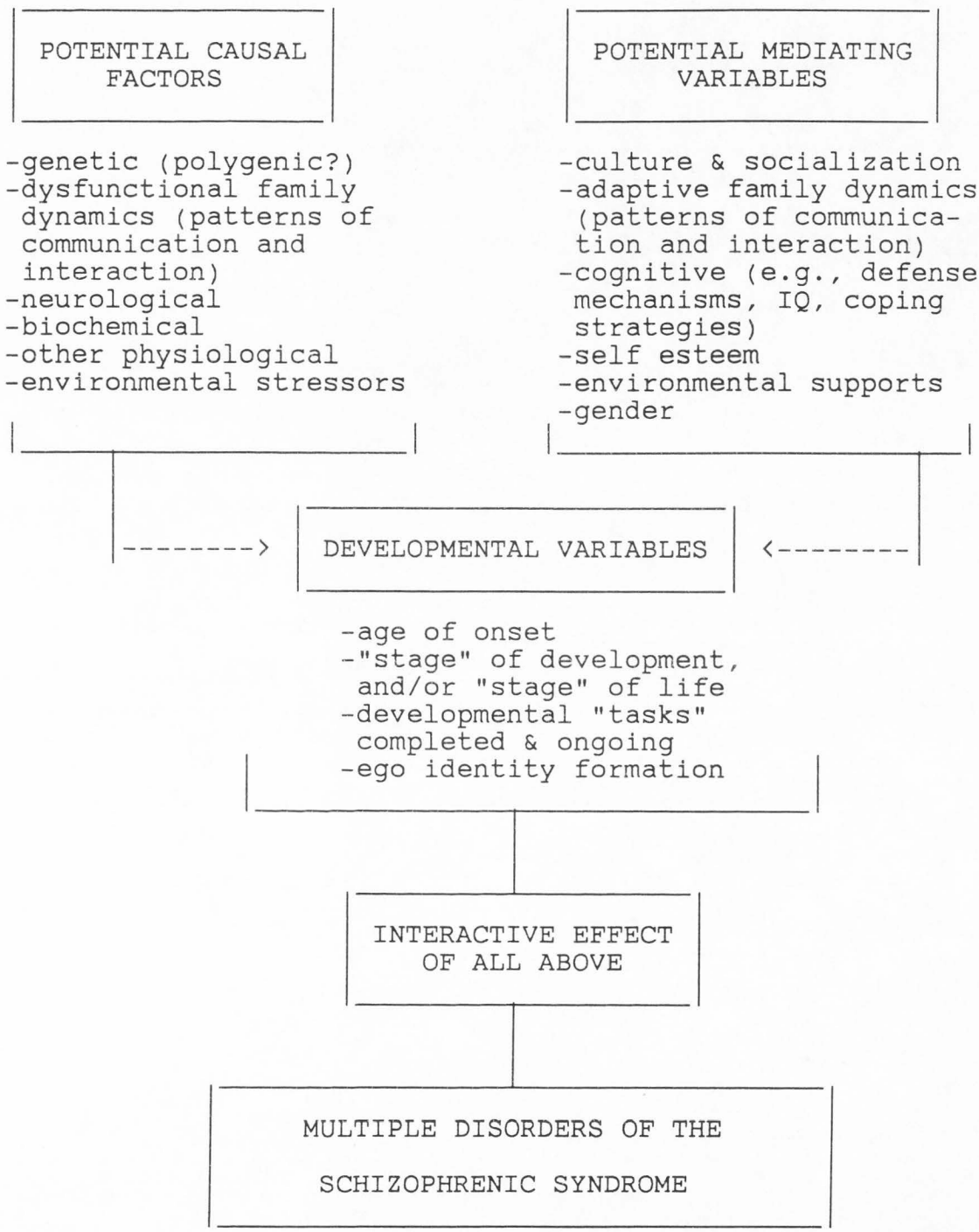

Figure 1: Interactive model of schizophrenia

increasingly unlikely in light of contemporary research findings.

A Developmental Framework for the Study of Schizophrenia

Lidz (1978) formulated a developmental theory of schizophrenia that appears useful in understanding the interaction between family environment and schizophrenia. He clearly acknowledges that there are, in all likelihood, biological and genetic factors (as yet undetermined) that are etiologically linked to schizophrenia. But Lidz contends that there is a substantial body of evidence that biology and genetics cannot, in and of themselves, account for the onset of schizophrenia.

Lidz (1978) identifies a number of developmental issues related to family functioning and interaction that he sees as contributory to the onset of schizophrenia. Families are thought by Lidz (1978) to serve four major functions: (1) parental nurturing, (2) structuring the personality, (3) teaching the basic social roles and about the basic institutions of the society, and (4) teaching the culture's language, mores, and ethos.

Lidz contends that families in which schizophrenics grow up fail to provide these prerequisites for integrated

personality development. This failure to provide is not limited to specific traumatic events to which the child is subjected, nor to specific developmental periods or events. Lidz contends that the difficulties and deprivations are "panphasic" in nature, extending across all the child's formative years.

Two types of dysfunctional families are identified by Lidz. "Schismatic" families are those split by gross parental conflict. The intensity of conflict is sustained at a high level, and is relatively constant. "Skewed" families are characterized by one psychologically aberrant parent whose behavior is tolerated as "normal" by the other parent. However, the healthier parent fails to provide a psychoemotional "buffer" for the child. In both types of families, the child's normal development is derailed by the pathology within the family.

Lidz (1978) also stresses the importance (in the pathogenesis of schizophrenia) of "egocentrism" as defined by Piaget (1926, 1929). Egocentrism refers to an overestimation of the power of thought, distortion of reality to the point of view and needs of the individual, and failure to distinguish between subjective and objective.

> Egocentrism is thought to increase each time that normal developmental processes bring the child into a new stage of life and concomitantly into a new and untried field of cognitive

> action, and slowly subsides as the child masters the new field, only to reappear in a new form as the child moves into the next stage of development. (Lidz, 1978, p. 83)

Lidz suggests that the common characteristic of egocentrism, regardless of the stage of development, is reliance on the "omnipotence of thought" (Lidz, 1978; p.87). This can lead, in extreme instances, to giving precedence to wishes over reality, especially if that reality is harsh, confusing, or painful. This process may contribute to the development of delusional ideation and schizophrenic thought disorder.

The first critical indicators of schizophrenia may come, according to Lidz, when goals fail to jell, but instead become increasingly diffuse. The youth may remain in the realm of fantasied achievement and equally fantasied gratification, made more appealing by contrast with a non-gratifying and bleak reality. The panphasic impediments to personality and cognitive integration that resulted from growing up in a disturbed and confusing family may then block the development of gender identity, ego identity formation, social skills, and appropriate defense mechanisms and coping strategies.

Cicchetti, Toth, and Bush (1988) make equally cogent arguments for conceptualizing pathogenesis in a

developmental framework. They suggest that "any psychopathology can be conceived as a distortion in the normal ontogenetic process" (Cicchetti et al., 1988, p. 1). In what they describe as a transactional developmental psychopathology model, Cicchetti et al. (1988) attempt to integrate genetic, biochemical and environmental influences within the normal developmental process. This model clearly has advantages over studying, in relative isolation, those factors thought to contribute to pathology. The interactional aspects of the model are particularly important:

> The multiple transactions among parental, child, and environmental characteristics contribute to the outcomes of child development in a reciprocal, dynamic fashion. Accordingly, if a child manifests pathological development over time, it is presumed that the child has been involved in a continuous maladaptive transactional process. (Cicchetti et al., 1988, p. 3; italics in the original)

If one subscribes to the medical model of schizophrenia, then there is a tendency to look for a "disease" entity, or a single causal agent (Carpenter et al., 1985). Assuming that the genetic variables that appear to contribute to schizophrenia will someday be identified, environmental influences must still be taken into account as etiologically significant, as was noted in

the literature review. Cicchetti et al. (1988) advocate for understanding those influences as interactions occurring over time, rather than a single landmark event that can be isolated as the "cause" of a disorder. The interactions may be parent-child, child-environment, and the reciprocal of both. Indeed, the characteristics of the child are seen as shaping the nature of the child's environment, with far-reaching effect:

> The longstanding manifestation of child maladaptation is shaped by parental and environmental support, while the child's characteristics help to determine the nature of the "environment." Because the child and the environment are seen as reciprocally influencing each other, it follows that development at a later point reflects not only the quality of earlier adaptation, but also the intervening environmental inputs. (Cicchetti et al., 1988, p. 3)

Development is not seen as the unfolding of tasks which are accomplished and then become of little significance. Rather, developmental tasks are seen as critical to continual adaptation. As new tasks emerge, issues that have been resolved may decrease in salience, but do not cease to affect the individual's behavior. As a consequence of this, Cicchetti et al. (1988) state that:

> Each issue represents a life-span developmental task that requires ongoing coordination and

> integration in the individual's adaptation to the environment and to the stage-salient developmental issue of the period. (p. 8)

Schizophrenia is a disorder that is profound and pervasive in the extent to which it impacts an individual's life. While the onset of psychotic symptoms can be acute, the general course of pathogenesis is an insidious one. The hypothesis propounded by Lidz (1978) regarding the "panphasic" effects of family dysfunction would seem equally appropos in discussing the influence of child/parent/environment reciprocal interactions affected by gradually unfolding psychopathology.

As a child or adolescent grapples with the demands of a particular stage of development, their effectiveness may be substantially impaired if they are simultaneously affected by schizophrenic symptoms which alter (however insidiously and gradually) their thought processes, judgment, and grasp of reality. If the youth is growing up in a hostile, rejecting, extremely ambivalent or inconsistent environment, then this will compound the interactive effect and the outcome may have profound longterm consequences.

> Severe childhood psychopathology could prevent the development of skills necessary for a normal adult life, so it could be argued that early

> problems necessarily are more potentially damaging than are ones that arise later in life. (Gelfand & Peterson, 1985, pp. 48-9)

Schizophrenia may affect the course of development by interfering with the specific stage-salient task, and may also affect the broader issues of development encompassed in the notion of socialization. The prodromal stage of schizophrenia is characterized by a gradual deterioration of functioning (American Psychiatric Association, 1980, 1987), which seems consistent with panphasic interference in the ongoing socialization of adolescents and children.

This interference is not limited to a single sphere, such as cognitive development, but may encompass all aspects of the individual's life. Social skills development may be affected by the deterioration of thought processes, especially as behavior becomes more influenced by the disorder. This seems particularly likely in the skewed and schismatic families described by Lidz (1978), or the double-bind families identified by Bateson et al. (1956).

The limitations in social skills will affect performance in social roles, especially outside the family. Behaviors that may serve an adaptive function within a pathological family system will often be

maladaptive outside the narrow confines of the family. The adverse effect on peer relationships may be critical to socialization (Hartup, 1979). Socially rejected children are deficient in socially competent behavior (Ladd & Mize, 1983) and appear more vulnerable to later psychological problems as a consequence. As youth struggle to cope with the normal demands of adolescence, with potentially dysfunctional family systems, and with the effects of a psychiatric disorder that is often not recognized in the prodromal stage, they may develop coping strategies that are dysfunctional. The observed behavior of people who have schizophrenia that is not controlled by medications ranges from flamboyant to reclusive, with nothing that stands out as "typical" schizophrenic behavior (American Psychiatric Association, 1980, 1987). One explanation for this broad spectrum of behaviors is that people have made individual adaptations to their particular situations, and developed unique coping strategies that enable them to function with some degree of comfort. However, those coping strategies may prove maladaptive in comparatively "normal" contexts.

Youth who suffer from schizophrenia may not be sufficiently advanced developmentally to have mastered a repertoire of effective coping strategies. This will not

only impair their ability to deal with the impact of illness, but decreased effectiveness in handling the impact of the schizophrenic disorder can further interfere with the acquisition of coping skills.

The disruptiveness of schizophrenia may be especially problematic to the extent that it interferes with the critical stage-related tasks of later childhood and adolescence. Erikson (1968) identifies the task of later childhood as development of a sense of mastery and personal competence. In their discussion of developmental psychopathology, Cicchetti et al. (1988) note that, "dysfunctions in the negotiation of stage-salient issues can affect the acquisition of competence" (p. 58). The task of adolescence is the development of a stable sense of one's personal identity (Adams & Montemayor, 1983; Erikson, 1968; Marcia, 1966). If this developmental task is not completed, the outcome is likely to be "identity diffusion" (Adams, Shea, & Fitch, 1979).

While a certain degree of identity diffusion is a normal part of adolescent life experience (Adams & Montemayor, 1983), psychological health requires eventual resolution of the "identity crisis" (Erikson, 1968). Failure to achieve such a resolution is currently being linked with a number of psychiatric disorders (Akhtar,

1984). An adolescent suffering from schizophrenia is thus caught in a cruel dilemma. The disorder may interfere with identity formation, and to the extent that it does so, the youth becomes increasingly vulnerable to further psychiatric dysfunction.

For those individuals who do not develop the full schizophrenic syndrome until well into their 20's or 30's, the severity of the disorder may be mitigated somewhat by the extent to which critical developmental tasks have been achieved. If such individuals have achieved some degree of ego identity, mastered effective coping strategies, developed an acceptable range of social skills, and demonstrated some degree of competence in earlier life, this may be advantageous in coping with the demands of the illness. This point remains speculative, since there has been so little research regarding similarities and differences of schizophrenia in adults and adolescents. It could be the case that substantial deterioration of functioning, after having attained a reasonable degree of competence, is devastating in it's impact on a person. However, there exists some research indicating that the prognosis for early onset schizophrenia is significantly more bleak than for later onset of illness (Beitchman, 1985; Shmaonova et al., 1980).

Summary

Current theories abut schizophrenia tend to be "mini-theories" about limited aspects of the disorder. There does not yet exist a "grand theory" that encompasses all the probable factors that contribute to the disorder as we currently understand it. This study was guided by several theoretical assumptions about schizophrenia: (1) schizophrenia is a syndrome that very likely includes two or more etiologically distinct disorders; (2) those disorders probably result from multiple factors interacting over time; (3) there seem to exist mitigating factors that affect the severity, course, and prognosis of schizophrenia.

Developmental variables may well be contributory to the pathogenesis of schizophrenia. The "panphasic" impediments to personality and cognitive integration resulting from pathological family functioning will contribute heavily to poor prognosis. Schizophrenia in child and adolescent populations may contribute to deficits in social skills and decreased social competence, ineffectual or maladaptive coping strategies, and a decreased sense of personal competence. These factors may increase the psychological vulnerability of the individual to further psychiatric dysfunction.

The panphasic disruption of normal developmental processes can result in identity diffusion beyond that considered "normal" for adolescents, and may contribute to the onset of schizophrenia. Developmental variables, if not actually causal, appear to be likely candidates as mediating variables in understanding the diversity of course and prognosis for schizophrenia.

CHAPTER IV

METHODOLOGY

An Empirical Approach to the Study of Schizophrenia

One can reasonably assume that regardless of the ultimate "causes" of schizophrenia, once a person has been afflicted with this disorder there are significant changes in the behavior and functioning of that individual. If the changes are of sufficient magnitude that psychiatric hospitalization is deemed necessary, then one would expect that at least some of those changes in behavior and functioning would be observable, measurable by both formal and informal methods, and potentially quantifiable. Indeed, this assumption lies at the very heart of any nosological or diagnostic system. Assuming that schizophrenia consists of one or more discrete disorders, it should be possible to identify empirically those "subtypes" of schizophrenia.

Previous attempts have been undertaken based on this assumption. There are, for example, the diagnostic subtypes of schizophrenia identified in the DSM-III and DSM-III-R (e.g., catatonic, paranoid). However, these subtypes have been derived largely from clinical

observation and experience. While recognizing the invaluable role of such clinical wisdom, the current study approached the problem of identifying subtypes by combining clinical data with somewhat more objective measures of behavior and functioning.

The purpose of this study was not to attempt validating a particular "mini-theory," nor was it an attempt to verify existing theories or concepts about possible subtypes of schizophrenia. Rather, the study was undertaken to identify possible "subtypes" of schizophrenia that are empirically derived, within certain guiding theoretical assumptions. This approach may lack some of the potential benefits of a more solidly theory-based study, such as generation and testing of hypotheses. But it has the strength of providing an empirical foundation for any conclusions that may be derived through post hoc analysis of the data. Given the limited nature of current theories regarding schizophrenia, this empirical approach was judged as being a defensible research strategy that might produce useful results for follow-up research activities.

Additional support for the idea of taking an empirical approach to this research seems to come from Powers, Hauser, and Kilner (1989):

> Current perspectives on adolescence are no longer grounded exclusively in theoretical

> formulations of what should occur in adolescence, but rather are based on empirically derived profiles of adolescent adaptation and growth. (p. 201, italics in the original)

Powers et al. (1989) contend that the study of adolescent mental health is at such a basic and tentative level that even "normal" or "healthy" adolescence cannot yet be precisely defined by psychologists. This being the case, one might contend that speculations about "abnormal" behaviors such as schizophrenia must be equally tentative.

Source of Data

In the current study, the clinical files from the adolescent unit of a psychiatric hospital and community mental health center in the northwestern United States were used as the data base. This Center is an accredited facility that serves a large catchment area, since it houses one of the few adolescent inpatient treatment units in the entire region. These files have been maintained according to standards for medical records as specified by the Joint Commission on Accreditation of Hospitals, and are audited several times a year by different accrediting agencies to ensure compliance with legal and medical guidelines.

The 71 subjects in this study ranged in age from 9 to 18, of which 52 were male, and 19 were female. Most of the children or adolescents admitted to this facility

undergo psychological testing (if possible), are given a physical examination, and have a developmental history taken from a parent or guardian. Detailed clinical notes are kept of each formal staff contact with the patient, and relevant informal observations are also noted in the file.

Since 1980, psychiatric diagnoses have been made in accordance with the guidelines in DSM-III, and the medical chart of each patient includes a "discharge summary" that contains a discharge diagnosis (based on observation of behavior while hospitalized). Psychological testing has been done by, or under the supervision of, the same psychologist during that same time period. As a result, there was some degree of consistency in the records available, which hopefully decreased potential errors in the data collected.

It should be noted that the data utilized included objective facts, as well as the more subjective data typical of clinical research (e.g., number of known suicide attempts, current legal custody of the child, whether or not involuntary hospitalization was recommended by the clinical staff). None of the subjective data called for a conclusion on the part of the researcher, and none of them required the researcher to make a judgment about the intended meaning of the person who actually recorded the information on the clinical forms. The

researcher simply recorded the information as it existed. Such data would seem relatively impervious to experimenter bias. Table 1 provides a summary of the data originally collected and the measures included in the preliminary study.

One criticism that must be addressed at this point is the cogent objection that even if the subjective data are apparently impervious to experimenter bias, they do not, in many instances, represent objective facts, but are judgments made by clinical staff members. These staff members undoubtedly differed in knowledge, training, theoretical stance, and experience. The time and effort expended on any given intake, history, or mental status examination no doubt varied according to the training and experience of staff members, as well as the other demands on their time when they were completing the forms.

These objections are unquestionably valid, and the limitations that they impose on the use and interpretation of the data are considerable. But such limitations would seem inherent in a study that relies on clinical data, especially when relying on extant records that were not developed for research use. Given adequate funding and personnel, it would be possible to design a much "cleaner" study that greatly decreased the error variance that plagues clinical research; but in the absence of those resources, research can only be carried on despite the

Table 1

Summary of Objective Measurements

Assessed in Preliminary Analysis

DEMOGRAPHIC INFORMATION FOR CHILD AND FAMILY
-standard information as available

MENTAL HEALTH HISTORY
-prior dysfunctional episodes, prior treatment
-past suicide/homicide ideation & attempts
-family history of mental illness & problem behaviors
-drug & alcohol history

CURRENT MENTAL HEALTH FUNCTIONING
-mental status examination, including current delusional ideation and/or hallucinations
-medication status during hospital stay & at discharge
-current suicide/homicide ideation & attempts
-drug & alcohol usage
-disruptive behavior/acting out while on unit (e.g., "incident reports" filed, seclusion in "quiet room")
-duration of current hospitalization
-recommendations for followup treatment

FAMILY ENVIRONMENT AND FUNCTIONING
-marital status bio-parents & current family structure
-number of residential changes, current housing status
-parental involvement while child in hospital
-history of foster care, current custody of child

MAJOR STRESSORS WHICH HAVE IMPACTED CHILD
-physical/sexual abuse
-death of significant others
-recurrent or serious illnesses/injuries

MEDICAL HISTORY AND CURRENT STATUS
-current physical exam & lab tests
-medical history checklist
-indices of neurological disorder

PSYCHOLOGICAL TEST RESULTS
-MMPI (all standard subscales)
-WRAT-R (reading, spelling, arithmetic)
-WISC-R or WAIS-R (verbal, performance, full scale, all standard subscales)
-Rorschach (raw scores on each card; i.e., presence or absence of salient responses)

limitations involved. One must simply acknowledge the limitations, do as methodologically sound a study as possible, interpret the data with caution, and focus on the potential heuristic value of the findings.

Data Collection Procedures

The mental health center from which data were collected maintained a record of the diagnosis for each individual who was hospitalized. Thus, every patient with a diagnosis of schizophrenia was identified by chart number, age, and date of admission. It was therefore possible to pull only those files that met the necessary age and diagnostic criteria. Only those charts on patients who were admitted in 1982 or later were used. The reason for this cutoff date was the fact that psychological testing of adolescents did not occur consistently at the Center until late in 1981.

When all the available charts with the diagnosis of schizophrenia were identified, the total number of such charts (N=39) was judged less than needed for the proposed statistical analysis. To this total number of charts was therefore added all those patients with a diagnosis of "Schizophreniform Disorder" (N=31) and "Atypical Psychosis" (N=3). These diagnostic categories differ from schizophrenia primarily in the duration of symptoms. If schizophrenic symptoms, in the prodromal and active stages

of psychosis, have existed less than six months, the diagnosis using DSM-III criteria must be "Schizophreniform Disorder." If symptoms have been evident for two weeks or less, the diagnosis given must be "Atypical Psychosis." However, as noted previously, these time criteria did not become vogue until the 1980 advent of DSM-III, and they are still not accepted by many professionals outside the United States (Kolb & Brodie, 1982).

The sum total of all three diagnostic categories (N= 73) did not result in a large data pool, but this limitation is one of the realities of attempting clinical research. One can only work with the data actually available, regardless of what might be "ideal." The catchment area of this adolescent psychiatric inpatient facility numbered several hundred thousand. The data collected represent every adolescent hospitalized in the facility over a five year period who had a psychotic disorder that was not clearly manic, chemically induced, or due to organic causes. If the numbers are somewhat less than might be desirable from a research standpoint, they reflect the reality of the incidence of disorders with schizophrenic symptomatology in the adolescent population within this catchment area.

If the patient had been hospitalized more than once, as occurred frequently in this population, data from the most recent hospitalization were recorded. Records from

the earlier admissions were reviewed for historical information such as sexual abuse, suicide attempts, and family history of mental illness. Such data were included even though they came from earlier admissions. However, the mental status examination, and the data reflecting patient functioning, were always taken from the most recent admission.

Once the relevant charts were identified, the researcher went through each chart individually to extract the data specified on the data collection form developed for this research. The researcher read the relevant data to an assistant who then entered data onto an individual "data collection form" for each patient. The researcher carefully read through the standard intake and admission forms, the medical history form, the treating psychiatrist's discharge summary, psychological evaluation reports, formal reports by social workers, the report of the physical examination, and any formal reports by other agencies (e.g., child protective services). The pages of nursing notes and the notes of staff contacts (often voluminous and always handwritten) were only skimmed. As per the original agreement with the Center, only the researcher handled and read the documents; in no case did the researcher's assistant actually handle or read from any patient files.

The psychological test protocols and profiles from the psychological evaluations were stored separately from the regular inpatient files. For those patients who had received such testing, the researcher extracted the relevant information directly from the protocols or profiles to maximize the information available. For instance, the scaled scores from the WAIS-R were recorded in addition to the Verbal, Performance, and Full Scale IQ scores normally reported in the psychological evaluation reports. Appendix 1 contains a detailed account of the data extracted from the clinical files, and of the data collection procedures. In this appendix are also notes and comments regarding how the data were actually used in the statistical analysis, since many data were synthesized or combined with others for summary scores (and some data were not used at all, for reasons detailed in Appendix 1).

Missing Data

In a number of instances, there were data gaps of anywhere from one missing "bit" to a great many missing "bits" of information. There appeared to be several factors that accounted for these missing data: (a) poor record completion by admitting staff [e.g., the back side of a double-sided form was completely blank, and left unsigned]; (b) the information was not recalled by the patients [e.g., their own history of psychiatric

hospitalizations], or possibly not known to the patients [e.g., history of their own birth and early development], and there was no other informant available; (c) the patients refused to give the requested information [e.g., history of criminal behaviors], and there was no other informant available; (d) the patients were so severely disturbed that they could not respond appropriately to questions [e.g., patients who were floridly psychotic, hallucinating, and non-responsive to verbal stimuli]; (e) not all patients were tested due to: shortness of stay, refusal to cooperate with the testing procedure, or they were too psychotic to participate in the testing process; (f) patients were too violent or aggressive to be questioned thoroughly or tested properly; (g) there were a few patients who were monolingual, non-English speaking, and no adequate translators could be located; (h) testing was not completed due to cultural factors that would have invalidated the test results [e.g., adolescents from other countries who had adequate fluency in English, but who had been in the United States for too brief a time to be assimilated].

As with the limitations in the size of the data pool, these gaps in data collection appear to be virtually unavoidable in clinical research. It is simply not possible to collect substantial amounts of extant data from clinical files and not find such data gaps. This

would seem especially likely when many of the facts sought were originally obtained from adolescents whose functioning was so profoundly disturbed as to require psychiatric hospitalization due to psychosis (see Appendix 1 for details regarding data collection procedures).

The files of two adolescents who were profoundly disturbed, had limited fluency in English, and had little or no family history available, were dropped from the data pool (one was diagnosed schizophrenic, and the other schizophreniform). In both cases there was such a paucity of information that it appeared to give an invalid profile of the youth involved. For instance, it was impossible to determine if delusional ideation was present, partly because of the language problems. In addition, these two youth were so severely disturbed that they were described in the clinical files as being virtually non-responsive to verbal stimuli.

Missing data were a critical determinant in reducing the number of variables initially collected (612) down to a more appropriate number for the factor analysis. For example, although comprehensive psychological testing was required (by informal agency policy) for most of the adolescents admitted to the Adolescent Unit, fewer than half of the subjects in this study completed testing of any sort. Since there was such a poor "test-rate" for

these subjects, the test data were dropped (with great regret) from the data pool.

On two variables retained for use in the factor analysis missing data were compensated for by substituting the median score obtained by the other subjects on the same variable (i.e., the Global Assessment Score [missing n=4] and the total number of days spent in psychiatric hospitals prior to the current admission [missing n=10]). The median score was used rather than the mean, because in both instances it resulted in a more conservative estimate that reflected a "healthier" level of functioning. ("Outliers" had skewed the mean scores in more pathological directions. Using median scores decreased the skewing, and more accurately reflected the group profile.)

When the final variables were identified for the initial factor analysis, all other missing data (within each of those variables) were treated in a uniform way. The data were obtained from clinical records that were largely narrative rather than "checklist" format. In the original data file compiled by the researcher, items were scored "present" only if identified somewhere in the clinical file as being present (e.g., reports of parental alcohol abuse). If they were clearly identified as "absent" (e.g., a statement that there was no known history of physical or sexual abuse), then the item was

scored as "absent." However, if there was no statement either way, the variable was scored as "missing."

When preparing for the factor analysis, the researcher was forced to make a decision based upon personal judgment. If even one subject had missing data, the computer would not accept that variable for statistical analysis (using the SPSSX statistical package). To avoid losing nearly all variables of interest, the researcher decided that missing data would be treated in a way that consistently erred in the direction of healthy functioning. For instance, if "judgment" was not marked in the original mental status form completed by the clinician at the Center, it was scored "fair" rather than "poor." If there was no indication whether or not hallucinations were present, it was assumed that they were absent. If nothing was entered in the file regarding sexual abuse, it was assumed (for purposes of statistical analysis only) that none had occurred. Clearly this procedure resulted in possible errors in the data, but the researcher could see no other way to avoid discarding large quantities of usable information because of a relatively few bits of missing data.

Data Collapsing and Synthesis

All data collected came from records maintained by the personnel at the mental health center. For each client who is admitted to the center, a series of standard forms are completed as part of the admission process. The researcher extracted selected data from those admission forms, from psychological test results (when it was available), and from clinical case notes. This data collection procedure resulted in over six hundred discrete bits of data for each subject. To reduce this data to a more manageable amount for statistical analysis, a number of discrete bits of information were synthesized, wherever feasible, into a single "index number" that was then entered into the data analysis in lieu of multiple smaller bits of data.

For example, consider the mental status examination section of the data collection form in Appendix 1. As it stands, there are 90 separate bits of data that are each coded individually. However, for the primary analysis this was reduced to 12 bits of data by synthesizing data into cluster scores. For instance, the section labeled "Mood/Affect" includes the following items:

MOOD/AFFECT- appropriate, elated, apathetic, calm, anxious, labile, fearful, depressed,

worried, angry, blunted, flattened, euphoria, excited, inappropriate (specify)

One might argue that if "appropriate" was circled as being applicable for a particular client, that the feeling states that are underlined above might be seen as tending toward psychological health, while those that are not underlined could be seen as tending towards psychopathology. However, if "inappropriate" is circled (or if appropriate is not circled), one might suggest that any of the feeling states identified would likely tend towards psychopathology.

For the primary analysis, which was a factor analysis, two scores were entered for "Mood/Affect." The first was a "healthy" score consisting of the sum of feeling states exhibited by this client that would tend towards health. The second score was the sum of feeling states that tended towards "psychopathology."

In a similar manner, the section labelled "Sensorium" was coded and scored as follows:

SENSORIUM- clouded consciousness, disorientation (time, place, person), oriented, memory loss (none, remote, recent, immediate), judgment (good, fair, poor), insight (good, fair, poor)

Those items that are underlined were seen as tending towards psychological health, and were summed for a single "healthy" score. Those items that are not underlined were seen as tending towards psychopathology, and were summed for a single "pathology" score. Only these summary scores were entered into the data for the primary factor analysis. Wherever feasible, data were synthesized to such "index scores" to reduce the sheer volume of information, and to reduce the number of variables being entered into the factor analysis.

Reliability of Additive Indices

A number of the additive indices just mentioned were entered into the final factor analysis. With any such index the question of reliability is raised. There may be logical justification for adding scores on discrete items to obtain a single index number, but the fact that a particular grouping of items may be logical does not mean the grouping is methodologically sound. However, statistical procedures for assessing reliability can give a valid measure of the extent to which the individual items comprising the index are correlated with one another. The higher the statistical correlation, the greater the likelihood that individual measures are meaningfully related to one another (and hopefully to the

theoretical construct that is assumed to underlie the index).

The individual items that comprised each of the indices entered into the final factor analysis were dichotomous variables with values of 1 or 0. Cronbach's Alpha is a measure of internal reliability that is recommended as appropriate for such data. This procedure is particularly useful in constructing scale scores such as those on the MMPI.

However, the use of additive indices in the present study was necessitated solely by the pressing need to reduce the number of variables to a manageable and statistically more appropriate level. There was no intention of developing psychometrically validated scales, nor should the index scores be thought of as being scales in that particular sense. The reliability coefficients for each of the additive indices are reported in Table 2. The name of each index is the code by which the index appears in the factor analysis pattern matrix, and the variables are listed in the same order they loaded on that matrix. Descriptive statistics for each variable entered into the factor analysis may be found in Appendix 2.

The two lowest alphas were both variables that combine only two discrete items. The variable F.ETCRIM was obtained by a factor analysis of several variables

Table 2

Reliability Coefficients for Additive Indices

	Alpha
COMMIT	.8673
ASSALTOT	.1513
P.MANNER	.4666
F.ETCRIM	.3353
PRNTSUPP	.8795
P.THPROC	.5487
P.SENSOR	.7591
P.THCONT	.3868

related to family pathology. A phi coefficient was computed on this variable as an additional measure of reliability. The obtained value of .28504 was statistically significant (p= .033). However, the variable ASSALTOT, which was derived by adding dichotomous responses on the presence or absence of assaultive behavior toward peers and staff, was not significant (phi= .09407, p= .428).

What this indicates is that there was not a signigicant correlation between assaultive behavior directed at peers and that directed toward clinical staff. The reason for this appears to be that most assaultive behavior was directed toward staff, and there were only 3 subjects who were physically assaultive toward staff and peers both. Despite the limited reliability of the

additive index, this measure was retained in the study as the best available indicator of aggressive behaviors occurring during hospitalization.

Data Reduction

The original data file included 612 discrete data for each subject, which is clearly too large a number to include in a factor analysis. A simple frequencies count for each item indicated that some of the items had far too many missing data to be usable (e.g., "family income" was available for fewer than half the subjects). All variables for which more than more than 25% of the subjects had missing data were deleted from the study.

From this substantially reduced variable list, the effort was then made to identify discrete items which might be closely enough related to combine in additive index scores. Comrey (1973) advocates such additive scores as preferable to dichotomous variables in factor analytic studies. For example, the additive score for "parental support" was derived by adding two dichotomous variables: (1) Did parents visit the patient while hospitalized? (2) Did parents participate in family counseling while the patient was hospitalized? This resulted in a single additive index score ranging from 0 to 2 rather than necessitating two separate 1/0 scores.

Other data were reduced by the stark reality that attempts to quantify qualitative information proved impossible. For instance, several of the youth admitted to the Center had appalling histories of extensive physical and sexual abuse. In some instances, there was repeated, brutal abuse by multiple abusers, including parents and other caretakers. Some of the youth had been subjected to abuse for virtually their entire lives. Some had also been injured severely enough to require medical hospitalization. However, there was simply no method by which the researcher could quantify the data to reflect the variety, complexity, and potential psychological impact of the abusive experiences to which many of these youth had been subjected. In the end, simply entering the fact of abuse or apparent non-abuse appeared to be the only feasible alternative for statistical analysis.

There was a fair amount of information on items thought to reflect family pathology and dysfunctional patterns of family dynamics. These included any biological relative or step-parent mentioned in the clinical file who had a history of: (1) mental illness [i.e., prior psychiatric treatment for any reason], (2) suicide [i.e., suicidal death rather than mere attempts], (3) alcohol abuse [i.e., treatment for alcoholism, attendance at AA, or references in the patient's file about serious drinking problems], (4) drug abuse [i.e.,

treatment for drug abuse, attendance at Narcotics Anonymous, or references in the patient's clinical file about serious drug abuse problems-- including misuse of prescription medications], and, (5) criminal history [i.e., mention in the patient's clinical file of any felony-level criminal activities, past prison record, current jail sentences, or pending criminal charges-- including child abuse].

Each variable was merely the additive total of biological relatives identified who displayed the requsite behavior. No attempt was made to quantify the degree of biological relationship with the subject. It was also impossible to quantify the potential impact the behavior may have had on the subject.

A factor analysis of these five variables was completed, with three factors being identified (see Table 3). All three factors identified were re-coded, and entered into the primary factor analysis as discrete variables. As all the variables were subjected to more refined analysis, only one of these three derived "family history" variables retained a factor loading that was statistically significant in the final factor analysis (i.e., that which reflected alcohol abuse and criminal behaviors).

Table 3

Factor Analysis of Family Pathology Variables:

Pattern Matrix

	Factor 1	Factor 2	Factor 3
Suicidal Death	.90090	.07917	-.17904
Mental Illness	.78030	-.11391	.22625
Alcohol Abuse	.02196	.82574	.16061
Criminal Behavior	-.02719	.72923	-.13028
Drug Abuse	-.00734	.03240	.97181

Factor	Eigenvalue	% of var
1	1.58703	31.7
2	1.11350	22.3
3	1.02862	20.6

Factor Correlation Matrix

	Factor 1	Factor 2	Factor 3
Factor 1	1.00000		
Factor 2	-.13287	1.00000	
Factor 3	.09122	-.08443	1.00000

Study 1

The purpose of Study 1 was to confirm that the data available did indeed differentiate between two discrete diagnostic groups (schizophrenia and depression). The rationale for choosing "depression" as the diagnostic contrast group was that this disorder is of sufficient magnitude that the resulting psychiatric impairment can require hospitalization, yet depression is also judged to be substantially different in its presentation from schizophrenia. If the data could not successfully differentiate between these two diagnostically and symptomatically discrete categories of hospitalized youth, then more refined statistical analysis (for "subtypes" within the schizophrenic data) would be pointless (i.e., if the data cannot differentiate between apples and bananas, then the same data are useless in identifying different varieties of apples).

Study 1 used as a data source the clinical files of 25 adolescents diagnosed as depressed, and the files of the youth with a schizophrenic diagnosis. The medical records department in the facility housing the files maintained a "master list" of all patients, listed by diagnostic category. The researcher identified all the patients who had the appropriate depressive diagnosis, and who met the age criterion used (i.e., first hospitalization at age 18 or under). The files were

selected in such fashion that they initially represented a randomized sample of the records available. However, when a file chosen by random sampling did not have the MMPI completed, the next file on the list was used that did have an MMPI. From these individual psychiatric files, data on two measures were recorded: the mental status examination, and the scores from the subscales of the MMPI. These sections both contain types of information thought to discriminate depression from schizophrenia, as those diagnoses are outlined in the DSM-III. A simple statistical comparison, detailed below, was used to verify that the two groups did differ, and that the diagnoses made were consistent with the criteria specified in DSM-III. (See Table 4 for a summary of the essential questions asked, and the statistical procedures used for Study 1.)

Study 2

Assuming in advance that analysis of the data from Study 1 would result in the successful differentiation of schizophrenia and depression, data were simultaneously collected for the larger primary study. This study used data collected from every file in the Center that met the diagnostic and age criteria. For a file to be included in the proposed study, the adolescent must have had a primary diagnosis of schizophrenia (or any subtype of

Table 4

Statistics for Preliminary Analysis

Question 1: Are there statistically significant differences in the "Schizophrenic" and "Depressed" patient data, which differentiate between those two diagnostic categories?

Statistic 1: t-test (selected MMPI subscales and the mental status data)

Question 2: Are there statistically significant differences between those patients who received an MMPI, and those who did not?

Statistic 2: t-test (mental status data)

Question 3: Are there statistically significant differences between those patients who meet the six month time criterion for schizophrenia, and those who do not meet the six month criterion?

Statistic 3: t-test (all 22 of the items which were eventually entered into the final factor analysis) [This was judged as a far more critical issue than question 2 {testability}, since this issue is at the core of construct validity. For this reason, a more comprehensive statistical analysis was completed.]

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schizophrenia), schizophreniform disorder, or atypical psychosis.

Some of the data collected included measures deemed potentially useful in discriminating subtypes of schizophrenia (e.g., MMPI subscales). Other data were included, not on theoretical grounds or because they were considered clinically relevant, but simply because they were readily available to the researcher (e.g., foster care). It was not known a priori whether any individual datum might eventually prove useful in empirically differentiating subtypes of schizophrenia, so all data that could be collected at little additional "cost" to the researcher were included.

From the data collected, statistical analyses (detailed below) were completed to attempt answering certain questions. These questions are not stated as hypotheses, but they did focus and direct the research in much the same way that formal hypotheses would have. That is, the goal of the research was to obtain defensible answers to those questions.

1. In adolescent psychiatric inpatient populations, are there diagnostic subtypes, within the broader diagnostic category of "schizophrenia," which can be empirically

differentiated on the basis of psychometric, demographic, historical, and clinical data?

2. If such subtypes can be identified, how do they correlate with the age of onset of schizophrenia (operationally defined as the age of first hospitalization)? That is: Can the age of onset be used to predict inclusion in a particular subtype of schizophrenia? Are there "age by gender" correlations with any subtype?

Although the developmental history information was collected, those data were deleted from the data set prior to factor analysis. It was hoped that a statistical correlation could be completed for each of the factors (meaning those factors that appeared to represent subtypes of schizophrenia) in order to determine the direction and magnitude of correlation they had with selected developmental markers. Those markers included such things as age of first hospitalization; age by gender; birthweight (and weight by gender); maternal ingestion of drugs or alcohol prenatally; whether or not the infant was fullterm or premature; and developmental milestones such as age of weaning, toilet training, and walking. However, there were so many missing data in the developmental histories, that no single marker was sufficiently "strong"

to be used in the statistical analysis. (See Table 5 for a summary of the essential questions asked and the statistical procedures used for Study 2.)

General Issues Involved in the Use of Factor Analysis

Factor analysis is a complex statistical technique for achieving what may be superficially seen as a relatively simple objective. A large body of data is reduced to a smaller number of factors which are thought to be related in a way that reflects underlying continuity of the variables within a given factor. For instance, variables such as fear of public speaking, preference for solitary activities, and few social contacts with friends may all be related to an underlying factor identified as "introversion." In the present study, the use of factor analysis is to determine if there are discrete factors which may identify "subgroups" of adolescents within a psychiatric population meeting certain diagnostic criteria.

The issue of sample size is important in factor analysis, as it is in other statistical procedures. The current study was based upon a smaller sample size than is ideally desirable. However, it represents the entire population of subjects meeting the diagnostic critereia who were hospitalized at this facility over a nearly five

Table 5

Statistics for Primary Analysis

Question 1: In adolescent psychiatric inpatient populations, are there diagnostic subtypes, within the broader diagnostic category of "schizophrenia," which can be empirically differentiated on the basis of psychometric, demographic, historical, and clinical data?

Task 1: Reduce data to relevant variables only; factors need not (at this point) reach convergence, or be clinically interpretable.

Statistic 1: Factor analysis (principal components, oblique rotation).

Task 2: Identify possible subtypes of schizophrenia from the reduced data obtained previously in Task 1. Factors must reach convergence, and be interpretable.

Statistic 2: Factor analysis (principal components, oblique rotation).

Question 2: How do the factors obtained correlate with the "age of onset" of schizophrenia (operationally defined as the "age of first hospitalization")?

Statistic 2a: Multiple stepwise regression, with age as the dependent variable

Statistic 2b: ANOVA (for each factor) age by gender (age = "older" and "younger" group for this analysis only)

Statistic 2c: Partial Correlation of each factor with age(controlling for gender)

Statistic 2d: Zero-order correlation of each factor with age (not controlling for gender)

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year span. Comrey (1973) states that with a small sample size there is less reliability of correlation coefficients. Since the factor analysis is based upon a correlation matrix, lower reliability of the correlation coefficients raises concern about the stability of the resultant factor structure. With small samples the random error of less reliable coefficients increases the absolute size of correlations in the matrix. This results in an increased common factor variance that is spurious and can produce distortions. With smaller samples there is also the risk that sampling errors have a greater influence and can decrease the clarity with which factors are identifiable. These facts mandate caution in methodology and a conservative interpretation of the factors.

There are questions raised in the statistical literature about the use of dichotomous variables in factor analysis. For instance, Comrey (1973) advocates avoiding dichotomous vairables whenever possible, while Kim and Mueller (1978) eschew their use altogether. As with small sample size, the use of dichotomous variables decreases the reliabililty of correlation coefficients and weakens confidence in the stability of the factor structure. In actual practice, however, the use of such variables is sometimes unavoidable. There are important research questions that do not permit a range of responses, as would be ideal for factor analysis, but are

limited to "yes/no" responses (e.g., gender of subjects). While Kim and Mueller (1978) decry the use of dichotomous data, they also acknowledge that many researchers continue to use such data, and data sets which are less than ideal cannot always be avoided.

When using dichotomous data, it is recommended that variables be coded "1" or "0." This then results in a phi coefficient which is the same as the Pearson product-moment correlation coefficient, which Comrey (1973) sees as preferable to other correlational procedures. Miller (1980) advocates that the frequency distribution split be no more than 20/80%, or in extreme cases, a 10/90% split. Anything more extreme than this results in potential instability in the factor stucture. There is also a tendency for correlation coefficients to be low due to mathematical constraints that result from use of dichotomous variables, expecially in the presence of skewed distribution. Comrey (1973) points out that frequency distribution splits more extreme than 20/80% result in decreased predictive power for the extreme variable. It might be said that prediction is at the heart of correlational analysis, so anything that weakens predictive power is to be avoided whenever possible. Since the sample size of the present study was already an issue, those dichotomous variables which did not result in

a frequency distribution ratio of 20/80% or better were deleted from the factor analysis.

The presence of mutually dependent variables is another issue that Comrey (1973) cautions about. For example, if one includes in a factor analysis such variables as verbal IQ score, performance IQ score, and full scale IQ score, the results will be distorted. The fact that full scale IQ scores include both verbal and performance IQ scores creates an artificial correlation. This could result in identification of a factor that is statistically significant and logically interpretable, but which is in reality a mere statistical artifact. Such in fact was the case during the preliminary analysis of the present study (discussed further in the "Data Reduction" section below). All variables that could be identified as mutually dependent were deleted from the study.

There are two primary methods of rotating axes in a factor analysis (Kim & Mueller, 1978). Orthogonal rotation assumes that the factors are not correlated. Use of this procedure results in maximum differentiation of factors. By contrast, oblique rotation assumes that the factors are correlated with one another. The subjects in this study were all in the same general age-range, they lived in the same geographic area, and they were all patients in a psychiatric facility. They all met similar diagnostic criteria for the psychiatric disorder which

resulted in their hospitalization, and the diagnoses were based upon commonality of behaviors and symptomatology. Since the subjects shared a common diagnosis, any "subtypes" should be part of a broader syndrome. There should, by definition, be shared features that justify diagnostic classification within that syndrome. Given these facts, it seemed appropriate to assume that any factors identified would be correlated, and the use of oblique rotation appropriate.

When applying an oblique factor rotation procedure, one is liable to obtain "complex data variables" that load significantly on more than one factor (Comrey, 1973). Such variables are acceptable, but should not be relied upon as heavily in defining the factor as the "pure-factor variables" that are unique to a particular factor. It is those pure-factor variables that more clearly define and differentiate factors. The complex data variables may load significantly, but they may also obfuscate as much as they clarify. The more complex data variables there are that load significantly on two or more factors, the more unstable the factor structure becomes, and the greater the risk of ambiguity.

Identification and Refinement of Final Factors

The variable list was reduced to 38 variables for the preliminary factor analysis. The total number of subjects (after eliminating those with virtually unusable data) was 71. The ratio of 38 variables to 71 subjects represents somewhat of an imbalance. However, the intent of the preliminary analysis was simply to eliminate additional variables that did not appear to contribute anything of substance.

A principal components factor analysis, using an oblique rotation, resulted in fifteen factors that failed to converge even after 50 iterations. Oblique rotation was chosen because it was judged that a shared diagnosis (with common symptomatology underlying that diagnosis), resulted in a high probability of correlation between the factors. Such correlation is assumed and acceptable with an oblique rotation, but not with an orthogonal rotation (Kim & Mueller, 1978).

Several variables were subsequently eliminated from the variable list, and the number of factors was limited to seven (each of which had an eigenvalue of 2.0 or greater). After 64 iterations, these seven factors converged. However, the last three factors appeared to be uninterpretable, and contributed little additional explanatory power.

Using the same variables (but limiting the number of factors to four) convergence was reached, again using an oblique rotation, after 23 iterations. However, several of the variables did not load significantly on any of the four factors, which resulted in a total of 31 variables thus making up the factors identified. Each of the factors had an eigenvalue of 2.0 or greater. This is appreciably higher than the 1.0 value used in many studies, and meets the guidelines advocated by Miller (1980) for factor analytic studies.

However, it was pointed out that several of the additive variables appeared to violate one of the mathematical assumptions which underlies factor analysis. If a subject's scores on one variable are dependent upon, or are substantially influenced by scores on another variable, the correlations may be artificially inflated. The resultant factor structure is affected, and the validity of the factors becomes suspect.

In the original factor analysis, there were twelve additive indices of "healthy" and "pathological" mental status information. A Pearson product-moment correlation coefficient was computed to determine if there was indeed a correlation. The results in Table 6 indicated conclusively that such a correlation did exist. Since the primary focus of the study was on pathological functioning, the "healthy" variables were dropped from the

Table 6

Pearson Correlation Coefficients for "Healthy" and "Pathological" Indices of the Mental Status Examination

Sensorium	-.9127 (71) p=.001	Thought Processes	-.3645 (71) p=.001
Mood/Affect	-.2138 (71) p=.037	Thought Content	-.3752 (71) p=.001
Motor Behavior	-.6821 (71) p=.001	Manner/Attitude	-.5159 (71) p=.001

analysis. Recomputing the factor analysis resulted in elimination of one of the factors, but a rather substantial "strengthening" of the other three. It turned out that the original "factor one" was a mere statistical artifact caused by the previously unrecognized correlation between the above mentioned variables. This incident illustrates rather convincingly the necessity of understanding clearly the assumptions underlying such complex statistical procedures as factor analysis.

Further refinement of the factor structures occurred by eliminating all variables that did not load at .40 or better, and recomputing the factor analysis. The final

analysis resulted in 22 variables, each with a factor loading of .40 or better. The three factors identified each had an eigenvalue greater than 2.0, which is quite conservative given that a 1.0 eigenvalue is often used as a cutoff criterion.

Entering 22 variables with a sample size of 71 readily meets the 2 to 1 ratio of sample size to variables suggested and used by Miller (1980) in his factor analytic studies of adolescent pathology. Kim and Mueller (1978) suggest that the sample size minus the number of variables minus one should be greater than or equal to fifty (N- # vars - 1 $\geq$ 50). This study does not quite meet that criterion (71- 22- 1= 48). However, the resultant factors were judged to be interpretable and clinically meaningful. The larger number of variables was also judged as more accurately reflecting the complex, multi-faceted clinical reality of schizophrenia than would be seen in a study with fewer variables. Appendix 2 contains a number of tables that delineate the descriptive statistics for each of the 22 variables that were included in the final factor analysis.

CHAPTER V

RESULTS

Construct Validity

Data analysis was initiated by addressing the construct validity of the classification of the schizophrenic group. The first step in this process was the contrasting of two known criterion groups (i.e., schizophrenic and depressed adolescents) to verify that the data appropriately differentiate two groups of patients classified as having distinct psychiatric disorders. Mental status and MMPI data were selected to assess theoretically appropriate differences between the two diagnostic categories.

It was anticipated that statistically significant differences might be obtained for such things as "thought processes," or the "schizophrenia" subscale of the MMPI. No significant differences were anticipated for other items such as the "masculinity-femininity" scale of the MMPI. An alpha of .05 was used in determining the significance. The schizphrenic group numbered 71, and the depressed group numbered 25.

In the statistical computation, the following variables were used: (a) additive index scores from the mental status examination (H= healthy; P= pathological); and, (b) raw scores from the Schizophrenia (scale 8) and

Depression (scale 2) subscales of the MMPI. The group mean for MMPI scores was substituted for missing data in the schizophrenic group. The "mean substitution" command was used in the statistical program in order to avoid discarding large amounts of data due to a few missing bits of information. MMPI raw scores, rather than scaled T-scores, were recorded on the profile sheet by the staff psychologist, who then interpreted the tests using adolescent norms. Since the MMPI scores were used for statistical analysis rather than clinical interpretation, the raw scores were just as useful as the standardized T-scores would have been.

A series of standard *t*-tests were computed, of which 9 successfully differentiated between the two groups at the .05 level or greater. (In point of fact, all but one of those *t*-tests were significant at an alpha level of .001 or greater.) The results of the analysis (see Table 7) indicate that the variables differentiate between the subjects diagnosed as depressed and those diagnosed as schizophrenic. Significant differences were obtained on the MMPI scales for depression and schizophrenia, and on the mental status data for motor behavior, thought processes, healthy thought content, and sensorium (e.g., memory loss, orientation, judgment). In each instance where significant differences existed, the depressed subjects scored in a "healthier" direction than did the

schizophrenic subjects. Such results are not surprising and serve to confirm that the data clearly differentiate between these two known criterion groups.

The data in Table 7 failed to differentiate between the two groups on five of the dependent measures. Using the mental status data available, no significant differences were obtained in healthy or pathological mood, which may seem somewhat unusual given that a mood disorder was the primary diagnosis for the contrast group. However, the summary score for each domain was arrived at by simply totaling the number of "healthy" or "pathological" responses identified as relevant for each subject under each domain. Virtually every depressed subject had "depressed" identified as one item in the mood domain. Only one third of the schizophrenic subjects (32.4%) had "depressed" identified as relevant, but they had other items marked in the mood domain. The summary score for each domain simply noted the total number marked; it did not identify qualitative distinctions for the items marked. While the use of summary scores obscured potentially significant differences, it was necessitated by the constraints of the statistical procedures involved in the primary analyses that were to follow (most notably the factor analysis).

Similar reasoning seems pertinent in understanding why no significant differences were found in either

Table 7

Statistical Comparison Between the Schizophrenic and Depressed Subjects

	F value	2-tail prob	t value	degrees of freedom	2-tail prob
MMPI-SC	4.34	.001	-5.80	85.20 (sep)	.001
MMPI-D	2.82	.006	-8.15	71.18 (sep)	.001
H-MOTOR	1.03	.888	-3.69	94 (pooled)	.001
P-MOTOR	2.17	.037	4.96	62.19 (sep)	.001
H-MOOD	2.87	.001	-1.16	30.10 (sep)	.255
P-MOOD	1.40	.356	-0.24	94 (pooled)	.810
H-MANNER	1.14	.737	-1.94	94 (pooled)	.055
P-MANNER	2.09	.046	1.81	60.97 (sep)	.076
H-THOUGHT PROCESS	1.38	.302	-4.42	94 (pooled)	.001
P-THOUGHT PROCESS	6.27	.001	7.25	92.45 (sep)	.001
H-THOUGHT CONTENT	2.39	.005	-2.17	31.37 (sep)	.038
P-THOUGHT CONTENT	1.75	.126	-0.46	94 (pooled)	.645
H-SENSORIUM	1.50	.270	-4.03	94 (pooled)	.001
P-SENSORIUM	3.47	.001	4.90	78.24 (sep)	.001

Note: sep= separate variance estimate
pooled= pooled variance estimate

healthy or pathological "manner" (i.e., manner toward staff members during the intake), and "pathological thought content." There may well have been qualitative differences, but there were no significant quantitative differences between the two groups on these particular mental status domains.

A second issue explored in establishing the construct validity was related to the fact that the majority of subjects in the schizophrenic group did not have MMPI scores. The question was raised whether those schizophrenic subjects who completed MMPI's represented a significantly different subgroup from those who did not. Comparisons were made between the two groups using the subjects' scores on the clinical variables from the mental status examination.

A series of *t*-tests between the two subgroups are summarized in Table 8. The two subgroups did not differ significantly on any of the twelve indices assessed. Since the two groups did not differ significantly on the *t*-tests computed, they were not treated separately in subsequent statistical analyses.

An additional issue that was addressed regarding construct validity was the fact that the subjects identified in this study as "schizophrenic" actually came from two different subgroups: (a) those with schizophrenic symptoms who met the six month time

Table 8

Statistical Comparison of Subjects

With and Without MMPI's

	F value	2-tail prob	t value	degrees of freedom	2-tail prob
P.Manner	1.30	.479	.84	69 (pooled)	.404
H.Manner	1.02	.926	-1.00	69 (pooled)	.320
P.Mood	1.13	.710	.49	69 (pooled)	.624
H.Mood	6.63	.001	-1.96	32.36 (sep)	.105
P.Motor	1.04	.899	.67	69 (pooled)	.508
H.Motor	1.22	.560	-1.05	69 (pooled)	.299
P.Sensor	1.33	.401	-.56	69 (pooled)	.575
H.Sensor	1.09	.824	.54	69 (pooled)	.593
P.Thproc	1.29	.451	-.95	69 (pooled)	.344
H.Thproc	1.36	.367	-.79	69 (pooled)	.434
P.Thcont	1.50	.234	.10	69 (pooled)	.917
H.Thcont	1.22	.590	.47	69 (pooled)	.641

pooled= pooled variance estimate
sep= separate variance estimate

criterion for a formal diagnosis of schizophrenia; and, (b) those displaying schizophrenic symptoms who did not meet the time criterion. These latter youth were given the diagnosis of either "schizophreniform disorder" or "atypical psychosis," depending on the amount of time during which symptoms had been evident. Since the "atypical" subgroup was very small (N= 3), they were added to the "schizophreniform" subgroup and a t-test was completed using all 22 of the variables subsequently entered into the final factor analysis.

The two subgroups differed significantly (alpha= .05) on 3 of the 22 variables; where one would expect only 5 of 100 tests to be significant at chance level. However, these differences appear to be consistent with the longer duration of illness that is the primary determinant for differential diagnosis between the two groups. The schizophrenic subgroup, who had been identified as psychiatrically disturbed for a longer period of time than the schizophreniform subgroup, scored in a more pathological direction on each variable. The schizophrenic subgroup tended to require more restrictive post discharge followup, had a greater frequency of suicidal behavior (twice as many attempts per subject), and evidenced more extensive delusional ideation.

This initial series of analyses established the following: (a) the data successfully differentiated

between two known criterion groups (schizophrenia and depression); (b) those subjects in the schizophrenia group who completed the MMPI, and those who did not, were not found to be significantly different; (c) those subjects who met the six month time criterion for schizophrenia did not differ significantly, in most areas, from those who failed to meet the time criterion (where they did differ, it appeared to be directly attributable to longer duration of illness rather than reflecting major differences between samples). These results are judged as demonstrating at least a minimal degree of construct validity in group classification.

It may initially seem a rather trivial outcome to demonstrate that one can successfully differentiate schizophrenia from depression, especially since clinicians have been doing it for years. However, there is much information available to the clinician that is unavailable to a researcher, particularly without direct access to the subjects. The attempt to establish some degree of construct validity demonstrated that <u>using the data available</u> one could successfully differentiate between adolescents with a diagnosis of schizophrenia and those with a diagnosis of depression.

It was further demonstrated that despite differences in formal diagnosis (i.e., Schizophrenia, Schizophreniform Disorder, and Atypical Psychosis), with duration of

illness being the determinant of those differences, the subjects did not differ sufficiently to necessitate treating them as distinct groups. Having at least tentatively established these things, one may then be somewhat more confident when those same data are used to identify "subgroups" among schizophrenic adolescents.

Descriptive Statistics for Subject Population

The total number of usable subjects in the final data pool was 71, of which 52 (73.2 %) were male and 19 (26.8 %) were female. The 5 subjects below age fourteen were all male, and only 4 out of 18 subjects aged fourteen and fifteen were female. However, the overall ratio of males and females in the fourteen to fifteen year old group is proportionate for the sample size (14 of 52 males= 26.9%, and 4 of 19 females= 21.1%).

About the same percentage of males and females were admitted involuntarily (nearly 70%), and slightly over half of each gender group had a legal hearing for additional involuntary treatment after their initial 72 hour detention had expired. The average length of stay for males was almost six days less than for females (male= 22.37 days; female= 28.05 days). Roughly 60% of the males and 70% of the females had experienced prior psychiatric hospitalizations with a median length of stay for males

being 11 days, and 14 for females (mean length of stay was skewed due to outliers). Approximately 60% of males and half of the female subjects had received prior outpatient psychiatric treatment. The mean score for staff ratings on the "Global Assessment Scale" was nearly the same for males and females, although the range of scores (both higher and lower) was greater for males. Continued post-discharge psychiatric hospitalization was recommended for about half the subjects, regardless of gender.

Somewhat unexpectedly, the proportion of males and females who had a history of violent behavior prior to the current hospitalization was roughly equivalent (males= 52%, females= 42%). These findings were initially viewed as somewhat suspect, given that aggressive behaviors are generally found to be higher for males than females. However, the gender ratio for assaultive behavior while in the hospital was similar (males= 40%, females= 37%). In addition, a roughly equivalent proportion of males and females spent time in the "quiet room" (a locked seclusion room) for out of control behavior; with both the mean and median number of hours in the "quiet room" being slightly higher for females (see Table 9 for specifics).

There was little difference in the proportion of males and females who evidenced two of the "classic" symptoms of schizophrenia; delusions and hallucinations occurred in over half of the subjects, regardless of

gender. An equivalent number of subjects required medication during their hospital stay and at discharge. A high proportion of both females (90%) and males (70%), had a documented family history of dysfunctional behavior. At least 65% of males, and nearly 80% of females were known to have made at least one suicidal gesture or attempt.

A third of all the subjects, both males and females, had documented histories of physical abuse. However, only 14% of males were known to have been sexually abused, as opposed to 41% of the females. In addition, 3 females (16%) reported having been raped, while no males reported having been raped.

Three fourths of the subjects were in the custody of the court (usually through the county social services agency) at the time of their hospitalization. However, only 27% of males, versus 42% of females, reported living in families that were intact (i.e., biological parents were living together). Nearly 20% of males and 10% of females reported that they did not live with either of their biological parents, but had been placed in longterm foster care settings. At some time during their lives, 42% of the females and 29% of the males had required at least a brief period of foster care. A large percentage of these youth (27% of males, and 47% of females) had no parental visits documented in their medical charts

(although there may have been non-documented visits or phone contact in some instances).

Forty percent of males and over 50% of females denied any form of drug usage. Those who acknowledged drug usage ranged from occasional experimentation to prolonged and repeated poly-drug abuse. Approximately 45% of males, and almost 60% of females, stated that they had no criminal history, and no current or pending legal charges. Not unexpectedly, males had more extensive, as well as more frequent, involvement with the juvenile justice system.

This discussion gives an overview of the youth involved in the study. Table 9 summarizes the descriptive statistics of the demographic and clinical data obtained on the schizophrenic subjects in this sample. Only those statistics that were judged as contributing in meaningful fashion were included in the table. For instance, the median scores on most variables generally added little useful insight or information, and were generally deleted in the interest of readability. In some instances, the median score was used rather than the mean. This was done when "outliers" skewed the mean scores in more pathological directions. Using median scores decreased the skewing, and more accurately reflected the group profile.

Table 9

Descriptive Statistics for Clinical Variables of the Schizophrenic Sample

		MALE	FEMALE
# OF SUBJECTS	(N= 71)	52 (73.2%)	19 (26.8%)
AGE (See Appendix 3, Table 39, for "age by gender" information)	range	9-18 yrs.	14-18 yrs.
	mean	15.64	16.16
	SD	1.96	1.07
	mode	17	16 & 17
	median	15.6	15.8
EDUCATION LEVEL	mean	8.5	7.8
	SD	3.1	4.2
ADMISSION STATUS	voluntary	16 (30.8%)	6 (31.6%)
	involuntary	36 (69.2%)	13 (68.4%)
DURATION OF THIS HOSPITALIZATION	range	3-81 days	2-76 days
	mean	22.37	28.05
	SD	18.33	22.07
	median	16.8	21.5
TOTAL # PRIOR PSYCHIATRIC HOSPITALIZATIONS	none	22 (42.3%)	6 (31.6%)
	1 hosp'n	14 (26.9%)	6 (31.6%)
	2 "	11 (21.2%)	4 (21.1%)
	3 "	2 (3.8%)	0 ---
	4 "	3 (5.8%)	3 (15.8%)
TOTAL # DAYS PRIOR PSYCH HOSPITALIZATIONS	no prior	22 (42.3%)	6 (31.6%)
	range	0-596 days	0-89 days
	mean	44.77	27.41
	SD	124.21	32.68
	median	11	14
PRIOR OUTPATIENT TREATMENT	no	31 (59.6%)	11 (52.6%)
	yes	21 (40.4%)	9 (47.4%)
SCORE: GLOBAL ASSESSMENT SCALE, (RANGE = 1- 100)	range	5-45	10-35
	mean	20.90	22.62
	SD	7.90	7.52
ADDITIONAL INVOL HOSP'N NEEDED AFTER INITIAL ADMISSION	no	27 (51.9%)	11 (57.9%)
	yes	25 (48.1%)	8 (42.1%)

Table 9 (continued)

Descriptive Statistics for Clinical Variables of the Schizophrenic Sample

		MALE	FEMALE
FOLLOWUP TREATMENT RECOMMENDED	none	2 (3.8%)	1 (5.3%)
	meds only	1 (1.9%)	0 ---
	outpatient	13 (25.0%)	7 (36.8%)
	day treatment	8 (15.4%)	2 (10.5%)
	ETOH/drug-(inpatient)	1 (1.9%)	0 ---
	psych hosp'n	27 (51.9%)	9 (47.49%)
HOURS SPENT IN QUIET ROOM	none	19 (36.5%)	6 (31.6%)
	range	0-737 hrs.	0-184 hrs.
	mean	28.33	30.95
	SD	102.20	54.42
	median	4.0	4.5
TOTAL # SUICIDE GESTURES/ATTEMPTS	none	18 (34.6%)	4 (21.1%)
	one or more	34 (65.4%)	15 (78.9%)
	range	0-9	0-8
MEDICATIONS NEEDED	(in hosp)		
	no	7(13.5%)	1 (5.3%)
	yes	45 (86.5%)	18 (94.7%)
	(discharge)		
	no	24 (46.2%)	8 (42.1%)
	yes	28 (53.8%)	11 (57.9%)
DELUSIONAL	no	20 (38.5%)	7 (36.8%)
	yes	32 (61.5%)	12 (63.2%)
HALLUCINATORY	no	25 (48.1%)	7 (36.8%)
	yes	27 (51.9%)	12 (63.2%)
FAMILY PATHOLOGY (mental illness, suicidal deaths, criminal behaviors, drug abuse, alcohol abuse--in any biological relative or step-parent)	no	15 (28.8%)	2 (10.5%)
	yes	37 (71.2%)	17 (89.5%)
ASSAULTIVE PRIOR TO ADMISSION	no	25 (48.1%)	11 (57.9%)
	yes	27 (51.9%)	8 (42.1%)

Table 9 (continued)

Descriptive Statistics for Clinical Variables of the Schizophrenic Sample

		MALE	FEMALE
ASSAULTIVE WHILE HOSPITALIZED	no	31 (59.6%)	12 (63.2%)
	yes	21 (40.4%)	7 (36.8%)
PHYSICAL ABUSE DOCUMENTED	no	34 (65.4%)	13 (68.4%)
	yes	18 (34.6%)	6 (31.6%)
SEXUAL ABUSE DOCUMENTED	no	45 (86.5%)	11 (57.9%)
	yes	7 (13.5%)	8 (42.1%)
RAPE DOCUMENTED	no	52 (100 %)	16 (84.2%)
	yes	-- ---	3 (15.8%)
# ITEMS MARKED ON MEDICAL PROBLEMS CHECKLIST	none	11 (21.2%)	3 (15.8%)
	range	0-8	0-5
	mean	2.39	2.16
	SD	2.08	1.54
PATIENT LIVES W/ BIO PARENT(S)	two par	14 (26.9%)	8 (42.1%)
	one par	28 (53.9%)	9 (47.4%)
	no par	10 (19.2%)	2 (10.5%)
WHO HAS LEGAL CUSTODY OF PT.	parent	12 (23.1%)	5 (26.3%)
	court	40 (76.9%)	14 (73.7%)
HAS PATIENT EVER BEEN IN FOSTER CARE	no	37 (71.2%)	11 (57.9%)
	yes	15 (28.8%)	8 (42.1%)
PARENTS VISIT PT. IN HOSPITAL	no	14 (26.9%)	9 (47.4%)
	yes	37 (73.1%)	10 (52.6%)
DRUG USAGE	no	21 (40.4%)	10 (52.6%)
	yes	31 (59.6%(	9 (47.4%)
CRIMINAL HISTORY	no	23 (44.2%)	11 (57.9%)
	yes	28 (45.8%)	8 (42.1%)
PARENTAL DEATH	mother	0	0
	father	4 (7.7%)	2 (10.5%)

Statistical Description of Factors Identified in this Study

In the aforementioned study by Miller (1980), which was on the broad and general issue of adolescent psychopathology, several specific criteria for factor analytic studies were offered. Miller (1980) advocated the following "rules" for the conservative use of factor analysis. Individual items must load .32 or higher in the factor loadings, and each factor must have an eigenvalue of 2 or higher. For rotation, each principal component factor must have a minimum of seven significant items (individual items can load on more than one factor, but are only counted once on the "rule of seven"). However, this particular "rule" is intended to guide the construction of scales, and was not adhered to with rigor in this study, since scale construction was not the objective. Third, to determine which of the rotated factors represent the basic dimensions of pathology, each factor should include at least seven significant items (each represented on only one factor). This statement also applies primarily to scale construction, and was not rigorously adhered to. Each factor should contain no more than one contradictory statement representing mutually contradictory concepts. Finally, each factor must describe a discrete and interpretable clinical dimension that is stable through various combinations of rotations. Miller

(1980) states that the number of subjects should be at least twice as many as the number of variables entered into the factor analysis.

An additional item should be mentioned here regarding the ratio of sample size to number of variables. Child (1970) indicates that as the number of factors increase, the factor loadings should also increase if they are to be judged significant, especially with small samples. He advocates use of the "Burt-Banks Formula," which takes into account the number of variables, the number of factors, and the sample size. Child (1970) states that use of this formula results in values that are "exceedingly stringent, especially for small samples" (p. 46).

Reference to a prepared table in Child's text (p. 99) gives the following information. For a sample size of 50 (which is smaller than the present study, and the criteria thereby more rigorous), the factor loadings on the third factor must reach .364 or higher to be statistically significant at the .01 level if one enters 20 variables, and a less stringent .357 if one enters 30 variables. Since no factor loadings under .40 were retained in this analysis, the criterion for statistical significance has readily been met.

In the present study, all three factors meet Miller's (1980) guidelines except in the number of items needed for

each scale; i.e. factor three has only five variables which load significantly instead of the seven recommended. However, the factor loading of .40 used in the present study is more stringent than the .32 criterion level Miller advocated. The factors also meet Child's (1970) "exceedingly stringent" criteria for statistical significance for this sample size and number of variables. As previously noted, the factors each have an eigenvalue greater than 2.0, which is more conservative than the 1.0 cutoff level often used in factor analytic studies (see Table 10). These three factors explain 40.4% of the observed variance in the sample.

One point should be noted regarding Miller's (1980) requirement for "discrete and interpretable clinical dimension[s]" previously mentioned. The subjects all came from the same hospital population with clinically similar symptoms. An oblique rotation was used in the factor analysis rather than an orthogonal rotation. Given these two facts, one might expect there to be a substantial correlation between the factors. However, the factor correlation matrix (see Table 11) shows no significant degree of correlation between any two of the factors. This suggests that even allowing for the "overlap" that results from using subjects who meet virtually equivalent diagnostic criteria, the factors identify relatively

Table 10

Eigenvalues of Each Factor

Factor	Eigenvalue	% of Variance	Cumulative %
1	3.58714	16.3	16.3
2	2.95467	13.4	29.7
3	2.33663	10.6	40.4

Table 11

Factor Correlation Matrix

	Factor 1	Factor 2	Factor 3
Factor 1	1.00000		
Factor 2	.02607	1.00000	
Factor 3	.03587	-.02736	1.00000

discrete subgroups within this particular sample. Entering the same variables in a factor analysis using an orthogonal rotation instead of an oblique rotation amply confirmed that the factors are indeed quite discrete and

independent of one another. The variables loaded in the same order except that "Custody" and "Foster Care" reversed their adjacent positions; and in most instances, the factor loadings were virtually identical (see Appendix 3, Table 38).

It should also be noted that the factors are not comprised of any "complex data variables" that load significantly on more than one factor. Rather, the three factors identified contain only "pure-factor variables" that are unique to each factor. This is judged by Comrey (1973) as increasing the ability to clearly discern the underlying factor structure.

The number of subjects available was lower than the researcher had originally anticipated, and certainly lower than optimally desirable, but represents the total population accessible to the researcher for study. However, the present study nonetheless meets the guidelines that have been used to guide similar factor analytic studies completed by other researchers. This may serve to strengthen confidence in the following interpretations, despite the relatively small sample size. The results of the final factor analysis are presented in Table 12.

Table 12

Factor Analysis Pattern Matrix

(Oblique Rotation with 22 Variables)

	(AGGRESS) FACTOR 1	(FAMILY) FACTOR 2	(THOUGHT) FACTOR 3
COMMIT	.74298	.01385	.24622
ASLTHRET	.71345	.05139	-.28002
ADMSTAT	-.64747	.28200	-.13998
ASSALTOT	.63788	.29813	-.09575
PRIORHOS	.54769	.08564	.13922
FOLLOWUP	.49813	.18317	.24770
QRHOURS	.48174	.07705	-.08821
PRTX.OP	-.45319	.34313	.10534
P.MANNER	.43038	-.24455	-.00497
SUICIDE	.40278	.38487	.01709
F.ETCRIM	.08017	.62504	-.07452
PA.YES	-.06301	.61710	-.14160
BIOPAR	.03431	-.59995	.08381
FOSTCARE	.06192	.58466	-.08330
CUSTODY	-.24766	-.58403	.04541
PRNTSUPP	-.01951	-.55927	-.24736
SA.YES	-.27654	.44062	.12667
P.THPROC	-.00582	.03780	.76442
P.SENSOR	.04025	-.02533	.75152
DELUSTOT	.01635	.01589	.67796
GAS	-.21691	.11407	-.58002
P.THCONT	-.11615	-.04412	.44631

Narrative Description of Factors

Factor one identified a group of youth who seemed to be more "aggressive" and acting out. This factor was characterized by higher rates of assaultive threats and behavior prior to hospitalization, and more frequent assaultive behavior to staff and peers while hospitalized. The nature and quality of interaction with staff was generally seen as more negative; even at the time of intake youth in this group were more likely to be hostile and uncooperative. There was a positive correlation with time spent in the "quiet room" for out of control behavior. Those youth scoring high on this particular factor were somewhat more likely to have been hospitalized involuntarily, and also more likely to have been committed beyond the original 72 hour detention.

There was a negative correlation with prior outpatient treatment, but a positive correlation with prior hospitalizations. This group demonstrated behaviors that resulted in a greater likelihood of more restrictive followup treatment being recommended (e.g., further hospitalization rather than outpatient treatment). This factor was, somewhat surprisingly, the only one to load significantly for suicidal behavior, which can be seen as aggression directed at the self.

Factor two appears to reflect a group with "disturbed family functioning" and dysfunctional patterns of intra-

familial interaction. This factor loaded positively for both physical and sexual abuse (the only one of the three factors to do so). Family history was more likely to be characterized by the presence of such potential indicators of dysfunction as alcohol abuse and criminal behavior. Those in this group were more likely to have been in foster care, and less likely to have been in the legal custody of their parents at the time of hospitalization. They were also less likely to have both biological parents in the home; in fact, this was the only factor to show a significant correlation with the presence (or, in this case, the absence) of biological parents. Factor two also included a negative correlation with those measures thought to indicate parental support during hospitalization (i.e., visitation while the subject was hospitalized, and participation in family therapy during the course of inpatient treatment).

Factor three identified the group most likely to demonstrate "delusional ideation." They were, in fact, the only group to achieve a significant positive correlation with this hallmark of schizophrenia. These youth were more likely to receive lower ratings by staff on the "global assessment scale" of overall functioning, even though their interaction with staff was not generally characterized by aggression. As a group they were less likely to have engaged in assaultive behavior prior to

hospitalization. During their hospital stay, there was less problem with aggressive behavior. This group was seen by clinical staff as demonstrating substantial difficulties with thought processes (e.g., loosening of associations), and with thought content (e.g., delusional ideation). More so than their hospitalized peers, youth in this group evidenced disturbances in the sensorium (e.g., disorientation, impaired insight and judgment).

Age of Hospitalization as a Developmental Correlate of Factors

Upon completion of the factor analysis, the next step in the statistical analysis was to determine if the "age of onset" of schizophrenia correlated with any of the factors. The "age of onset" for schizophrenia was defined, for the purpose of this study, as the age of first hospitalization, or (when age of first hospitalization was not available) the earliest known age for hospitalization. This is clearly less than ideal as a measure of developmental level, especially since there are many contingencies that can affect when a person is hospitalized (e.g., family finances, availability of bed space, intervention by social agencies, informal social sanctions for deviant behavior that originate in extra-familial sources). However, it was the only estimate of

"age of onset" of schizophrenia available to the researcher, and was used despite the obvious limitations.

A Pearson Product-moment correlation coefficient was computed to determine if there was a statistically significant relationship for age of hospitalization and any of the factors, without controlling for gender (see Table 13). In addition, a partial correlation coefficient was calculated for which the effect of gender was controlled (see Table 14).

The correlation coefficients are substantially the same with both procedures. The results indicate that while gender was not a significant mediating variable, there was a postive correlation with the operationally defined age of onset and factor three (thought disordered).

In the next phase of the analysis regarding age of hospitalization, gender was entered as the first variable in a multiple regression equation with age as the dependent variable. When it was determined that gender was not a significant variable, at least in this particular statistical analysis, each of the three factors identified in the factor analysis was regressed onto age. Factor three (thought disordered) again correlated significantly with age when using this regression procedure ($p = .041$), although neither of the other factors reached statistical significance.

Table 13

Age by Factor Correlations,
Without Controlling for Gender

	(AGGRESS) FACTOR 1	(FAMILY) FACTOR 2	(THOUGHT) FACTOR 3
AGE	.0376	-.0957	.2394
	(71)	(71)	(71)
	p= .378	p= .214	p= .022

Table 14

Age by Factor Partial Correlations,
Controlling for Gender

	(AGGRESS) FACTOR 1	(FAMILY) FACTOR 2	(THOUGHT) FACTOR 3
AGE	.0258	-.0529	.2446
	(71)	(71)	(71)
	p= .416	p= .332	p= .021

One additional attempt was made to explore the extent to which age might be correlated with each of the factors identified. The subjects were clustered into a "younger" group aged 9-15 (n=30, male=21, female=9), and an "older" group aged 16-18 (n=41, male=31, female=10). These particular age groupings were made less on theoretical grounds than by the statistical necessity of attempting to get approximately equal sized groups for each cell. A 2 x 2 analysis of variance was completed using the two age groups compared by gender (male= 52; female= 19).

Factor three (thought disorder) was statistically significant in the "age by gender" interaction effect (see Table 15). Examination of the interaction effect indicated that younger age group females obtained higher mean scores on this factor than did males. For the older group the opposite held true, with males obtaining higher scores than females.

Summary

The statistical analyses completed resulted in the identification of three discrete factors. These factors all had an eigenvalue of 2.0 or greater. The factor loadings for each of the 22 variables that were entered into the analysis met or exceeded a .40 cutoff score. The first factor related to "aggressive behavior," the second

Table 15

ANOVA: Factor Three by "Age of First Hospitalization" and Gender

	SS	df	MS	F	Signif
Age	2.046	1	2.046	2.300	.134
Gender	.651	1	.651	.732	.395
Interaction	7.982	1	7.982	8.972	.004

to "disturbed family functioning," and the third to "thought disorder." Age of first hospitalization was significantly correlated, through several statistical procedures, with factor three (thought disorder). A significant age by gender interaction for factor three was obtained when the subjects were grouped into younger (9-15) and older (16-18) age groups.

CHAPTER VI

DISCUSSION

Introduction

The purpose of this research was to ask two key questions (and challenge certain underlying assumptions related to those questions) about schizophrenia, especially in adolescents and prepubescent children. First, in adolescent psychiatric inpatient populations, are there diagnostic subtypes, within the broader diagnostic category of "schizophrenia," that can be empirically differentiated (by factor analysis) on the basis of psychometric, demographic, historical, and clinical data? Second, how do the factors obtained correlate with the age of onset of schizophrenia (operationally defined as the age of first psychiatric hospitalization)?

The three factors obtained after the final factor analysis each had an eigenvalue above 2.0, which is appreciably higher than the criterion level of 1.0 used in many studies. More importantly, the factors were judged to be interpretable and clinically meaningful, despite the limitations imposed by the small number of subjects available. Except for sample size, the present study meets criteria that have been used to guide similar factor analytic studies on adolescent psychopathology completed

by other researchers (Child, 1970; Miller, 1980), including criteria for statistical significance ($p < .01$) given the number of subjects, the number of variables entered into the analysis, and the number of factors extracted. This may serve to strengthen confidence in the results.

The three factors define relatively discrete groups of youth with some expected commonality, but who also manifest potentially critical differences. It seems possible that some of these factors may represent "subtypes" of schizophrenia. If such "subtypes" are empirically validated by subsequent research, the possiblilty of differing etiology for one or more of the "subtypes" then becomes an issue of primary interest in future research.

In the discussion that follows, frequent reference is made to genetic loading, genetic vulnerability, and so forth. The research on genetic factors in schizophrenia (see Stone, 1980, for a review) provides a body of convincing evidence indicating (persuasively, though not conclusively) that polygenic factors play a role in many (if not all) of the possible disorders subsumed under the rubric of schizophrenia (Gottesman & Shields, 1982). However, even in monozygotic twins, who share an identical genetic inheritance, the concordance rate for schizophrenia is not 100 per cent (Kendler & Robinette,

1983; Randels et al., 1982). Other non-genetic variables affect onset, or absence, of the disorder (Plomin, 1989). Nonetheless, despite the variability in genetic studies, the evidence for genetic influence is so convincing (see Plomin, 1989, for a recent review) that it simply must be taken into account in any speculations about schizophrenia.

Stone (1980) argues that the presumed "genetic loading" in schizophrenia can vary from relatively small to nearly overwhelming. Those with a high "genetic loading" for schizophrenia will develop the disorder no matter how nearly ideal their environmental support system, or how protected they may be from excessive levels of stress.

Other people, with a lesser degree of "genetic loading," will vary in whether or not the disorder actually develops. Such variation is attributable to variables in their environment. Some may have exceptionally good parents with supportive families and low-stress environments. Others with a similar "genetic loading" may be born to abusive or pathologically disturbed parents, and grow up in hostile and dangerous environments. The former may not develop active symptoms, while the latter may show a range of symptoms that fluctuates with the level of environmental stress. This hypothesis seems a persuasive one in explaining the 50%

(rather than 100%) concordance rate for schizophrenia observed in monozygotic twins (Randels et al., 1982). With this preface on genetics, the following discussion of the research findings is offered.

Factor One: Aggressive Behavior

Factor one identified a group of youth who seemed to be more "aggressive" and acting out in their behavior prior to hospitalization. It was also those in this group who appear to have been most physically aggressive toward staff and peers during the course of their hospital stay. Youth in this group required more frequent application of restrictive behavioral controls such as isolation in the "quiet room." Those youth scoring high on this particular factor were more likely to have been hospitalized involuntarily, and also more likely to have been involuntarily committed beyond the original 72 hour detention (which legally required evidence demonstrating a probability of harm to self or others if not hospitalized). The incidence of prior psychiatric hospitalization was higher for these adolescents. They more frequently demonstrated behaviors that resulted in staff recommendations for restrictive followup treatment (e.g., transfer to a long-term care facility rather than outpatient treatment). Aggression in this particular group also took the form of suicidal and self destructive

behavior, this being the only factor that obtained a positive correlation with suicide.

The aggressive/acting out subgroup may not represent an etiologically distinct group of schizophrenic youth as much as it describes a behavioral response characteristic of adolescents in many different contexts. Miller (1980) identified an aggressive subgroup in his study of adolescent psychopathology. Achenbach (1982) also discusses the frequent occurrence of aggressive behavior in his text on developmental psychopathology. Indeed, some measure of aggression is common to nearly all adolescent personality inventories or behavioral rating scales.

The adolescents in this subgroup may be simply responding to a bewildering and frightening situation by striking out aggressively. (This is discussed at greater length in the section below entitled "Developmental Implications".) Such responses will certainly be familiar to those who have worked with adolescents in treatment, correctional, and even academic settings. But even if it turns out that this subgroup does not reflect distinctive etiology, it still remains that the treatment and management of this group will clearly differ from that provided for youth in the other subgroups. Even medical management will need to differ (especially on an outpatient basis), since this will very likely be the

group that is most resistant to treatment regimens and least compliant about taking medication.

Factor Two: Disturbed Family Functioning

Youth identified by factor two showed evidence of dysfunctional patterns of intra-familial interaction. This factor was the only one to load positively for physical and sexual abuse. Family history was more likely to be positive for the presence of alcohol abuse and criminal behaviors. Those in this group were more likely to have been in foster care, and less likely to have been in the legal custody of their parents at the time of hospitalization. They were also less likely to come from intact homes. Youth in this group appeared to receive less parental support during hospitalization, as evidenced by the absence of parental visitation or participation in family counseling.

The factor that reflects "disturbed family functioning" seems to support the research findings of family studies theorists who have attempted to identify dysfunctional patterns within the families of schizophrenics that may contribute to the occurrence of schizophrenia (e.g., Anderson et al., 1986; Lidz & Fleck, 1985). This factor may also reflect the distorted communication systems within the families of

schizophrenics that have been documented by other researchers, and postulated by them as having etiological significance (Bateson et al., 1956; Lidz & Fleck, 1985).

Studies of family dynamics and patterns of internal communication have been dismissed by some as inadequate to explain the etiology of schizophrenia, and as ignoring the biochemical and genetic factors that seem so critical. However, it may be that in the subgroup of youth who load high on this particular factor, these family interactions do indeed play a critical role. Emphasizing the role of family dysfunction (e.g., Lidz, 1978) does not eliminate biochemical or genetic elements, but highlights an interaction effect in which family stress and severely disordered communications are integral components in precipitating the onset of illness where the biochemical "potential" or genetic "loading" already exists (Stone, 1980). In this theoretical context, the family dysfunction and disordered communications may represent variables that are "necessary" but not "sufficient" causal factors for the onset of schizophrenia.

An additional consideration in understanding the factor posited as describing disturbed family functioning is that this factor was the only one that loaded positively for any of the family pathology variables. This means that these families displayed higher than usual rates of alcohol abuse and criminal behaviors. Such

behaviors occurring within the family environment, especially serious alcohol abuse, may well be expected to contribute to an overall disturbance of functioning.

Factor Three: Thought Disorder

Factor three identifies the only group that obtained a significant correlation for "delusional ideation," which is often considered one of the hallmarks of schizophrenia (Randels et al., 1982; Schneider, 1959). These youth were generally rated lower by staff on the "global assessment scale" of overall functioning, despite the fact that their interaction with staff was generally less aggressive (in fact, there was a negative, although non-significant, correlation with both assaultive behavior and time spent in the quiet room). As a group they were somewhat less assaultive both prior to and during hospitalization.

Cognitive functioning was substantially impaired in this group of youth. They were the only group to load significantly on "pathological thought processes" and on "pathological thought content" as evidenced by the mental status examination. Certainly one would not assume that they were the only group to manifest such disturbance, but staff at the center appear to have seen a greater degree of impairment in the youth loading high on this factor. Judgment and insight were more often identified as poor in

this group than in others. There was a greater tendency for these youth to exhibit disturbance of cognitive functioning as reflected in orientation and memory impairment. It appears that factor three identifies more stereotypically schizophrenic youth than either of the other factors derived from the data analysis.

Being typified by more prominent delusional ideation and a greater degree of cognitive impairment, this factor may represent a subgroup in which biochemical and genetic variables play a dominant role. As noted in the literature review, there is substantial evidence implicating such variables in the onset of schizophrenia (e.g., Cazzullo & Invernizzi, 1985; Feinsilver, 1986; Kaplan et al., 1980; Kety, 1975; Marcus et al., 1985; Plomin, 1989; Stone, 1980; Strauss & Carpenter, 1981). This group of youth may well have developed schizophrenia even in optimal family environments simply because of a strong "genetic predisposition" (Stone, 1980) that operated relatively independently of environmental considerations to directly affect brain functioning. However, this speculation is weakened somewhat by the fact that family history of mental illness and other measures of family dysfunction did not load significantly on this factor. One might have expected a positive loading on family pathology variables if genetic influences were primary in this factor.

Developmental Implications of the Factors Identified

Data were collected on a number of variables that could be seen as developmental "markers" (e.g., low birth weight, maternal alcohol and drug usage prenatally, age of weaning, walking and toilet training). However, the data were missing for a majority of subjects due to inadequate developmental histories. In most cases, the lack of a reliable informant appeared to be the primary reason for poor developmental histories. Not surprisingly, most of the youth who did not have a parent available at the time of intake were unable to recall their own birthweight or the age at which they had been toilet trained. The paucity of developmental information resulted in "age of onset" of schizophrenia (operationally defined as age at first known hospitalization) as the only developmental marker that was consistently available. Several different statistical procedures were applied to the data in assessing this issue.

A Pearson product-moment correlation indicated that age of hospitalization was significantly correlated with one of the three factors identified. Two correlations were completed, one that controlled for gender ($p = .021$), and one that did not ($p = .022$). The results indicated that while gender was not a significant variable, there was a positive correlation with age of onset (as

operationally defined) and factor three (thought disorder). A multiple regression for each factor, with age entered as the dependent variable, again resulted in factor three (thought disorder) being the only factor to attain statistical significance (p= .041).

However, while a 2 x 2 ANOVA of gender and age (older vs. younger group) resulted in no main effect for either age or gender on any of the factors, there was a significant interaction effect (p=.004) obtained for factor three (thought disorder). The older group ranged from 16-18 (n=41, male=31, female=10). The younger group ranged from 9-15 (n=30, male=21, female=9). Examination of the data indicated that in the younger age group females obtained higher mean scores on this factor than did males. For the older group the opposite held true, with males obtaining higher scores than females.

The reasons for this interaction effect are not clear. Lewine (1980) found gender differences in the age of hospitalization, with boys being hospitalized at an earlier age. The same author notes, however, that there were no significant gender differences in the time between age of onset of illness and age of hospitalization (i.e., the symptoms were not "tolerated" longer for children of either sex). Loranger (1984) noted that regardless of the measure used to determine age of onset (e.g., first hospitalization, family's first awareness of psychiatric

symptoms) boys displayed earlier age of onset. Both authors raised the question of possible gender differences in the etiology of schizophrenia. It appears that both Lewine (1980) and Loranger (1984) were studying schizophrenia as a unitary disorder. The present study suggests that the observed age and gender differences may also be affected by "subtype" of schizophrenia.

"Age of onset" is not a particularly interesting datum per se, but it provokes interest as a potential "marker variable". Although it is only a crude indicator, age is commonly used as a marker for the onset of puberty. With puberty comes a host of attendant biological and social changes. The present study demonstrated a correlation between age and one of the factors identified (thought disorder). One is tempted to speculate about the relationship between this particular factor and puberty. It seems logical to suggest that genetically controlled mechanisms which actuate during puberty may contribute, in some fashion not as yet determined, to the onset of schizophrenia. If one assumes that schizophenic thought disorder is related, at least in part, to neurochemical dysfunction, it seems reasonable to ask how the onset of massive hormonal and biochemical changes during puberty may be related to schizophrenic thought disorder.

Despite the comparatively sparse epidemiological data about schizophrenia, there appears to be consensus that

age of onset is adolescence or early adulthood (American Psychiatric Association, 1980, 1987). This again raises questions, as yet unanswered, about the possible relationship between puberty and one or more possible "subtypes" of schizophrenia. The present study does not provide definitive answers, but suggests that further exploration in this area is clearly warranted.

If there are indeed age and gender differences that are associated with certain "subtypes" of schizophrenia, an obvious developmental question that immediately surfaces is, "How much of the difference is attributable to environment, and how much to inherited genetic endowment?" The issue of heritability is an important one pragmatically, as well as in terms of broadening our knowledge about schizophrenia.

This study does not contribute directly to the research on genetic factors related to schizophrenia. However, at least one issue raised by this research seems germane in the search for possible genetic influences contributing to schizophrenia. Of importance in this study is the fact that possible "subtypes" of schizophrenia could be empirically demonstrated. If these represent true "subtypes," in the sense of different etiologies, there may well be different combinations of genetic factors that are specific to a particular "subtype." Recognition of this possibility may assist in

making sense of the confusing and inconsistent results of research on genetic contributions to schizophrenia.

Clearly associated with this issue is the problem of differentiating genetic and environmental influences in the development of symptoms. Of particular concern to developmental psychologists is the nature and quality of the family environment. If it can be demonstrated that a "subtype" of schizophrenia is consistently correlated with certain types of family systems, such knowledge might have a critical influence on treatment and prevention strategies for that "subtype." It might become feasible to identify families "at risk" for schizophrenia, and develop genuinely preventive interventions. Indeed, preliminary studies are already being undertaken with children who have at least one biological parent with diagnosed schizophrenia (Mirsky & Silberman, 1985).

Since early onset of schizophrenia is thought to result in poorer prognosis, identification of a "subtype" of schizophrenia associated with family functioning could potentially result in "early intervention" strategies for upgrading the quality of family life. While this may not prevent the onset of schizophrenic symptoms, it may delay onset sufficiently to give the child time to acquire adaptive and coping behaviors that come only after attaining a certain level of cognitive and social development. Acquisition of these personal "assets" may

serve to mitigate the severity, or at least the impact, of symptoms which then occur.

This study identified one factor characterized by aggressive behaviors, and this seems to represent a developmental issue with treatment implications. It appears that the aggression may be accounted for by either one of two possible explanations.

Youth with previously effective coping skills may be reacting aggressively in direct response to their illness. The delusions and hallucinations that characterize schizophrenia can be terrifying. The reaction of other people to the illness, especially family members, can be confusing. An adolescent may be striking out aggressively as a direct result of the fear and confusion. It will also be remembered that schizophrenia is characterized by deterioration of functioning in many areas (American Psychiatric Association, 1980, 1987). It seems reasonable that this high level of aggression displayed by some adolescents may represent one manifestation of such deterioration.

In other youth, the insidious onset of illness may ~~have~~ interfere~~d~~ with normal development. The youth may not have learned appropriate adaptive strategies for handling their aggressive impulses. With the onset of puberty and its attendant changes, relatively primitive childhood coping mechanisms may become ineffectual. If a

particular youth was impacted by a prolonged prodromal stage of schizophrenia (probably unrecognized, except in hindsight), the illness may have interfered with the acquisition of skills necessary for coping with the normal demands of adolescence.

Early prodromal schizophrenia may also interfere with the developmental tasks for a particular stage. In the latest stage of childhood ("stage" as defined by Erikson, 1968), interference with normal developmental tasks is hypothesized to result in a sense of inferiority, or a diminished perception of one's personal competence. During adolescence, the developmental "task" relates to identity formation (Adams & Montemayor, 1983; Erikson, 1968; Marcia, 1966). Failure to complete this "task" results in identity diffusion (Adams et al., 1979; Grotevant & Adams, 1984). Prolonged or extreme identity diffusion is being increasingly linked with various forms of psychopathology (see Akhtar, 1984, for an overview of the clinical implications of identity diffusion). In either stage, interference with normal development is seen as resulting in decreased personal effectiveness. With the onset of acutely psychotic symptoms, the adolescent is caught unprepared to cope, and will likely regress to earlier (and more primitive) responses such as "tantruming" or other aggressive behavior.

The treatment response to these two hypothesized causes of aggressive behavior would have to be different. Adolescents who had previously developed effective coping strategies must learn how to use those strategies in coping with the new demands of their illness. This is clearly a much different task than confronts those youth whose insidious course of illness impaired the acquisition of effective coping skills, or those who failed to successfully complete earlier "developmental tasks."

It was noted in the "Theory" section of this paper that family environment is thought to be a critical issue in the etiology of schizophrenia. Several theorists were cited who may disagree in detail, but who concur that the family is the crucible wherein are forged many of the critical components for psychological health and for such debilitating disorders as schizophrenia. It seems that those assumptions are reflected in the results of this research. Factor two was specifically noted as being related to disturbed family functioning. The variables that loaded on this particular factor are suggestive of substantial levels of dysfunctional behavior within the families of origin of these adolescents.

A significant number of youth reported family members with alcohol problems, and with behaviors that would be seen as criminal (including child abuse). The frequency of these reported variables is even more notable when one

realizes that these relationships were deemed important enough by busy clinical staff to record in the form of narrative comments in the patient's chart.

The family dysfunction included elements of abuse, both physical and sexual. Not surprisingly, foster care had been a relatively frequent occurrence for these adolescents. However, one must use caution in drawing causal conclusions about the relationship between abuse and pathology. It may well be the case that children who are abused are more vulnerable to later pathology (Gelfand & Peterson, 1985). However, one could also wonder about the effect on parents of caring for a child who displays increasing levels of deviant and sometimes glaringly aberrant behaviors. It may be that parents become angry and frustrated by the unremitting demands and possibly overwhelming struggle of caring for a child whose behavior they can neither understand nor control. Abusive behaviors may, in some instances, be the direct outcome of parental frustration.

There were several other indicators of family dysfunction that loaded on this particular factor. Many of these youth were currently in the legal custody of the courts rather than of their families. It is most often the case that legal and social agencies become involved with a family as a result of behaviors displayed by either the parents or the youth that are judged unacceptable.

Even when the youth were in the legal custody of a parent, families in this group were more likely to be disrupted by parental separation or divorce.

This factor also loaded negatively with the only two measures of "parental support" that were available. Treatment staff in the adolescent unit where these youth were hospitalized encouraged visitation by parents and immediate family members. There were some youth who did not have a single notation in their chart regarding a visit by parents. Family counseling was offered as part of the treatment approach at this center. The clinical staff were keenly aware of the critical importance of family dynamics in both contributing to and ameliorating problems for their adolescent patients. Some of the families did not, or possibly could not, participate in even a single one of the family sessions offered to them. For some of these youth, parental support appears to have been extremely limited.

This research did not, and could not, assess qualitative differences in the nature of parent-child interactions or family functioning. Recording the bare facts about the presence or absence of child abuse, or of parental participation in family counseling, may seem a rather stark way of attempting to measure the impact of family dysfunction. However, if such crude indicators support the contention of theorists regarding the

importance of family dynamics, what might be revealed by more sensitive indicators? This is clearly an area requiring more extensive and more refined research.

Family dysfunction is identified by Lidz (1978) as a contributory element in exacerbating to pathological extremes the normal egocentrism of childhood. Lidz (1978) refers to "egocentrism" as an overestimation of the power of thought, distortion of reality to the point of view and needs of the individual, and failure to distinguish between subjective and objective. A pathological form of egocentrism may be reflected in factor three (thought disorder).

It is true that there was no significant correlation between the factor identified in this study related to family dysfunction and that related to "thought disorder". However, the factor related to "disturbed family functioning" assessed only rough measures of family dysfunction. The "skewed" and "schismatic" families discussed by Lidz (1978), and the distorted intrafamilial communications studied by Bateson et al. (1956), are far too subtle to be tapped by the measures available for the current research study. Factor three (thought disorder) might indeed be correlated with disturbed family functioning when assessed by more refined measures.

It may be seen as premature to even discuss psychodynamic explanations of thought disorder until

physiological and neurological bases for such disorders can be conclusively ruled out. Given the effectiveness of neuroleptic medications in clearing delusional thought processes, and the promising research being done on neurotransmitter substances, there appears to be ample reason for expecting neurological substrates in one or more "subtypes" of schizophrenia (Randels et al., 1982).

However, as a number of clinicians and researchers have noted, the research on family dynamics and intrafamilial communication helps explain why a disorder that may well have biological underpinnings takes such a diversity of paths in its behavioral manifestations (Anderson et al., 1986; Lidz, 1978; Bateson et al., 1956; Lidz & Fleck, 1985). This research also helps in understanding some of the anomalies of behavior found amongst patients who have suffered from this disorder for many years.

Schizophrenia is by definition a syndrome that distorts sensory perceptions, clouds judgment, alters normal associational patterns in thinking, and even affects the content of thought. If the onset of schizophrenia for a particular adolescent is insidious rather than acute, the disorder (assuming physiological underpinnings) may also affect cognitive development during the critical transition from concrete operational thought to formal operational thought. What effect this

might have on the individual is likely to vary drastically due to the influence of many potential mediating variables (e.g., IQ, coping skills previously mastered, parental support). That it will affect cognitive development in some fashion seems highly probable. This, too, is another area needing further research.

In conclusion, to understand schizophenia in adolsecents and children, one must study it firmly embedded in a developmental context rather than artificially isolated from the living realities of adolescence. This study linked age as significantly correlated with of one of three factors identified. But even those factors for which age was not a statistically significant correlate are better understood by attempting to delineate developmental issues that affect the course of illness. This will hold true regardless of what may eventually be discovered about the etiologies of various "subtypes" of schizophrenia. In reciprocal manner, efforts to clearly specify how the disorder may alter the normal path of development will enhance understanding of aberrant behaviors that occur later in life in response to stress or to a recurrence of schizophrenia.

Limitations of the Research Design

This study clearly suffers from certain methodological limitations, some of which seem difficult

to avoid in clinical research. The ratio of variables (N= 22), to subjects (N= 71), is judged acceptable (Miller, 1980), but the sample size is smaller than would have been desirable. The limited sample size forces one to be more than usually cautious and tentative in the interpretation of data, and in evaluating the results.

A second limitation, and clearly a considerable one, was the fact that data were obtained from extant sources that were developed to meet clinical needs rather than research objectives. The researcher could not control the nature of the information obtained, with the result that available data rather than optimally desirable data had to be used.

This is obviously one of the drawbacks in many clinical studies, since the researcher may not be able to control the variables and data collection as precisely as in laboratory studies. It would have been desirable to have detailed accounts of family functioning and interaction for each subject, along with personality profiles of biological and step parents. But such a research objective would have required vast resources of money and staff time. Thorough investigation of drug and alcohol history, collected on standardized data collection forms, with corroborative evidence from parents, police, medical records, and current drug screenings would have been desirable; but here, too, the cost and time factors

are prohibitive. The extant data may have been less than optimal, but they still represented a rich source of information that had hitherto gone largely untapped by researchers.

A third limitation, and a corollary of the second, is the regrettable paucity of available test data. Over half of the patients in the study had no test data at all, while barely a handful received a full battery (i.e., WISC-R or WAIS-R, WRAT-R, MMPI, 16PF, and Rorschach). The rationale for complete, partial, or no testing was not noted in the chart as far as could be determined. The t-test between those with and without the MMPI revealed few significant differences between the two groups on the variables measured. Still, it would have been invaluable to have complete test data on each subject, both pre- and post-morbid. Data from widely used, standardized psychometric instruments could have been a fruitful source of information in attempting to identify possible subtypes of schizophrenia.

Another limitation of the present design reflects (once more) the difficulties involved in clinical studies, especially those using extant data sources. Only a mere handful of files were so complete that they did not have at least one missing datum. Most files had several small gaps in available data. In two instances, the data were sufficiently incomplete that entire files were dropped

from the study (both subjects were so profoundly dysfunctional that no information could be obtained, and there were no other informants available). These missing data affected the study in several ways, enumerated below.

Some items were simply dropped from the study because too few subjects had the data to be useful. For instance, due to the lack of information about family income and parental occupations, not even a rough estimate of socio-economic status could be obtained. To use another example, the lack of consistency in the intellectual testing made it impossible to determine if IQ correlated with age of hospitalization, or with any of the factors identified.

In other instances, missing data were identified (for statistical analysis only) as "zero" data. Unless an event or incident was recorded in the file as definitely having occurred, or definitely having not occurred (e.g., rape, sexual abuse, death of parents) it was entered as "missing" on the data collection form. However, in the statistical analysis, such missing data were treated as if the event had not occurred (e.g., the subject had never been raped).

Clearly, this allowed errors to creep into the subsequent analysis. For example, not a single one of the 71 subjects is known to have lost their biological mother through death, although six reported the death of their

biological fathers. This seems statistically unlikely, and must be considered suspect since at least a few of the "non-deaths" represent "non-information" rather than careful and detailed data accurately collected for research purposes.

Strengths of the Research Design

The present study used as data certain facts about the subjects that are relatively objective (e.g., previous hospitalizations, occurrence of assaultive/aggressive behavior while hospitalized). In most instances, the composite scores represent the presence or absence of discrete events or behaviors. Even the mental status data, which are based on interviewer judgment, were obtained from a checklist-type form on which the interviewer indicated the presence or absence of specific behaviors or symptoms. Many of the data (including composite scores) were based on completely objective facts about the subject (e.g., admission status, recommended followup treatment, amount of time spent in the quiet room, whether or not parents visited the client during the hospital stay). Use of such objective data decreased (though did not eliminate) reliance on self-reported information obtained from the patient.

There is also the possibility that objective correlates (e.g., death of a parent, drug usage), if found

by further research to be consistently associated with certain "subtypes" of schizophrenia, could have diagnostic significance. This would seem an advantageous supplement to current diagnostic criteria, which are based largely on symptoms (many of which are self-reported). One might also find objective criteria with predictive value in identifying "at-risk" populations or individuals (e.g., scores on validated psychometric instruments).

This study tapped a hitherto unused data pool that was remarkably rich, despite being partially fragmented. The range and diversity of information included in the factor analysis would have been practically impossible to obtain in any other way (at least any way that is financially feasible, even for a major research center). The diversity of the data gave the study a breadth not typically seen in research on schizophrenia. It allowed the researcher to go beyond simple correlations, such as IQ and post-morbid adjustment, and address (though only tentatively) issues of greater range and scope. While correlational studies offer useful information, the magnitude of this disorder is too great to be adequately comprehended and understood by studies which are intended to be extremely narrow in scope.

The breadth of this study allowed for a conceptual complexity not attainable in experimental studies that are designed to test one or two precise, and carefully

limited, hypotheses. While the study suffers from a corollary lack of depth, the identification of interpretable and meaningful factors seems to outweigh that limitation. Such factors appear to have considerable potential in making sense out of a complex and multi-faceted phenomenon.

One of the major deficits in our current understanding of schizophrenia, especially in adolescents, is the lack of a conceptual framework within which to integrate the isolated facts and the fragments of knowledge which are now amassing. While this study does not pretend to offer such a framework, it may be that only those with a similar design and scope will offer any realistic probability of formulating even a minimally adequate conceptual framework.

Another strength of this study lies in the attempt herein to address issues of interest in the broad area of developmental psychopathology. Although "age of first hospitalization" is admittedly a crude indicator of developmental level, the study was designed to ascertain if even such a primitive measure would suggest links between age (and developmental "stage") and any "subtype" of schizophrenia. The fact that such linkages appeared in the data analysis encourages the search for more sophisticated and precise measures to facilitate more

thorough exploration of a poorly understood area of psychology.

Another strength of this study is the use of factor analysis to determine "subtypes" within the schizophrenic population studied. These subtypes were not derived solely from clinical observation, nor from client self reports. The subtypes identified in this study were empirically derived from a combination of clinical, self-report, and relatively objective data. This makes the findings somewhat more reliable, and the study potentially more replicable, than would be the case relying on clinical observations alone. Such studies may also, as pointed out by Powers et al. (1989), contribute to theory development. Although one normally looks to theories for the generation of research hypotheses; in this instance, empirically derived research findings may reciprocate by contributing to the generation of theoretical concepts.

Future Research Needs

The results of this study lend support to the idea that schizophrenia is not a unitary disorder, but may represent different disorders with common symptomatology. The factors empirically derived in this study do not conform to the clinically derived subtypes of

schizophrenia current in psychiatric nomenclature (e.g., paranoid).

Such a finding should not be surprising, given the nature of the data from which these two differing conceptualizations of schizophrenia originated. The clinically derived subtypes currently in vogue are based primarily on the observable and reported symptoms manifested during the flagrantly psychotic phase of the disorder. The subtypes identified in the current study incorporate much more than symptom patterns.

It may eventually be those other components, the "non-symptom" objective facts, that ultimately give critical clues to the etiological combinations resulting in the observed (but widely variable) symptoms, pathogenesis, course of illness, outcome, and prognosis currently observed in schizophrenia. The search for empirically validated "subtypes" appears to be essential to understanding the variability of this disorder.

The present study merely tantalized one regarding the relationship between "subtypes" of schizophrenia and the developmental level of the subject. The sole developmental index (age of first hospitalization) is too crude a measure on which to base solid conclusions. However, the fact that even such an unsophisticated indicator showed linkages between developmental level and pathology, suggests that additional research in this area

would be appropriate. More sophisticated indices of developmental level, the use of validated psychometric instruments, incorporation of a wide variety of objective correlates (and potential correlates), and the use of factor analytic statistical techniques, may all combine for a greater understanding of schizophrenia in particular, and developmental psychopathology in general. The multitude of questions posed in the literature review appear to be answerable only by developing research strategies consonant with the complexity of the research topic.

Several other developmental issues appear to be significant enough to require further research. Premature birth and low birthweight for infants (National Center for Health Statistics, 1986; Winick, 1979), have both been implicated in adverse outcomes for children, including later neurological anomalies. One has to wonder to what extent such factors may contribute to the onset of psychopathology in children and adolescents. In and of themselves, they may be relatively limited in their contribution to the onset of schizophrenia; but the interactive effect with other variables could well be significant.

One has to also wonder how maternal drug abuse prenatally affects the developing fetus, and to what extent (if any) this may contribute to the development of

schizophrenic symptomatology later in life. Since prenatal neurological damage may be irreversible (Dhopeshwarkar, 1983), maternal ingestion (during critical stages of neurological development) of chemicals known to affect the brain could potentially result in irreversible changes. Even if such changes were relatively minor, in an organ as complex as the human brain the outcomes could be critical. The increasing concern about "fetal alcohol syndrome" (Clarren & Smith, 1978; Rossett & Weiner, 1984), and evidence that excessive prenatal alcohol consumption results in long term effects on the child (Shaywitz, Choen, & Shaywitz, 1980), combine to suggest that continued research about the relationship between maternal drug abuse and the later development of child or adolescent psychopathology is clearly needed.

Given the uncertainty about the relative contributions of heredity and environment that has plagued developmental psychology since its inception as a discipline (Lerner, 1976), it comes as little surprise to see the same issues raised regarding schizophrenia and other psychiatric disorders (Plomin, 1989; Stone, 1980). The literature review mentioned some of the research studying various environmental factors and the onset of schizophrenia. But another broad area of concern for future research is the relationship between schizophrenia and psychological trauma.

A number of the youth in this study had experienced life events which are traumatic by any definition (e.g., prolonged and repeated abuse by parents, witnessing a parent's suicide, gang rape). If Stone's (1980) hypothesis about the relationship between genetic vulnerability and environmental stress is correct, then one would expect that such traumatic experiences may well contribute to the onset of schizophrenia in certain youth.

Even if schizophrenic symptomatology does not inevitably result from trauma, the psychological impact may well vary as a direct result of the youth's developmental level. In one study on the stress-related effects of a natural disaster, it was shown that even a relatively benign natural disaster had a significant impact on victim populations (Adams, 1981; Adams & Adams, 1984). When victims who are both physically and psychologically immature are subjected to extreme levels of stress (especially prolonged or repeated stress), the impact is likely to be considerable (Erikson, 1976; Lifton & Olson, 1976). Since burgeoning populations virtually assure that greater numbers of youth will be impacted by trauma of one sort or another, understanding the possible effects of such experiences appears critical, especially if there are causal links to the development of serious psychopathology.

In reviewing the clinical and research literature pertinent to this study, one issue overshadowed everything else: knowledge about schizophrenia appears to be mushrooming far more rapidly than it can possibly be assimilated. The resultant body of knowledge is fragmented, coming from geneticists, biochemists, neurologists, pathologists, and other scientists, as well as from more "traditional" sources such as psychologists, sociologists, and psychiatrists.

Environmental stressors, neurotransmitters, and "schizophrenogenic mothers" (Fromm-Reichman, 1948) have all been studied; claims have been made that each contributes, in a causal way, to the development of schizophrenia. But the data are fragmented, the facts mostly an unorganized and indigestible mass of information, and the streams of knowledge originate from a broad array of mutually incomprehensible specialties. The result is an overwhelming flood that inundates without enlightening.

There is a tremendous need for evaluation, critical analysis, and synthesis of the currently unmanageable data-mass into a more intelligible and usable body of information and theory. This would seem attainable only by the combined (and no doubt prolonged) efforts of an interdisciplinary team that blends data from individual specialties into a cross-fertilized, integrated core of

knowledge. This information must then be "translated" from the jargon of particular specialties into educated English, intelligible across disciplines and national boundaries. Without such an integrative effort, attempts to understand and treat the disorders of the schizophrenic syndrome will be impaired.

Conclusion

The present study does not provide any incontestable or incontrovertible answers to the multitude of questions surrounding the syndrome of schizophrenia. It does support certain concepts that guided the study, although they were not presented as formal hypotheses.

The diagnostic label of "schizophrenia" is given to disorders that share common symptoms, but which may represent groups of disorders rather than a discrete entity. This study provides support for the argument that the syndrome currently labeled "schizophrenia" may include relatively discrete "subtypes". If future research provides support for the existence of "subtypes" of schizophrenia, the next question becomes one of assessing whether or not the "subtypes" (interacting with genetic and psychosocial factors) represent disorders that are entirely different in etiology, pathogenesis, course of illness, and prognosis.

The study also suggests that age of onset may be a critical variable for some "subtypes" of schizophrenia. Statistically significant relationships were obtained for "age" (i.e., age of first hospitalization) and one of the factors (thought disorder). These findings support the speculation that "schizophrenia" occurring at age thirteen may be a different entity than "schizophrenia" occurring at age twenty-five. Even if it turns out to be virtually the same disorder (for each subtype) for adults and adolescents, this study suggests that developmental level (at the onset of illness) may be an important mediating variable for severity, course of illness, and prognosis.

To a limited degree, this research supports the contention that the uncritical downward extension, to children and adolescents, of diagnostic systems based upon adult populations, may need extensive reexamination. For instance, the "aggressive/acting out" group identified in this study may be unique to adolescence. Clearly more research is needed to either validate or refute the assumption of equivalence of disorder, regardless of age of onset, which is currently guiding American psychiatric thought and diagnostic practices.

If there do indeed exist discrete subtypes of schizophrenia, they should be amenable to relatively empirical diagnosis. These subtypes may be identifiable by a combination of objective indices and clinically

observed symptoms and behaviors. The subtypes of schizophrenia suggested in this research, which combines at least a few objective indices with clinical data, differ radically from those currently identified in the DSM III (and DSM-III-R), which are based almost entirely upon clinical observation of presenting symptoms.

It seems entirely plausible that if an exploratory study such as the present one can achieve significant results with a limited sample size, better funded studies with a greatly restricted scope and a larger sample may well achieve clearly validated and reliable results. This would provide direction for the increasing number of clinicians and scientists who see current diagnostic categories as needing a validated empirical base to support clinical assessment that is limited to presenting symptoms.

If there are indeed empirically verifiable subtypes of schizophrenia (or any other psychiatric disorder), and if age of onset is a critical variable in any of the subtypes, that is important information in its own right. However, of even greater importance, effective treatment is more probable when accurate diagnosis is made of disorders that differ radically in etiology. For instance, if one subtype of schizophrenia is neurochemical in origin, but another represents polygenic "vulnerability" factors interacting with environmental

stressors and the physiological changes of puberty, the treatment and/or prevention of those two subtypes will likely be substantially different. The results of this research are one small step in the direction of developing an optimum combination of clinical symptoms and empirical data, which may someday lead to validated and reliable diagnostic criteria (and more effective intervention and treatment strategies) for a major psychiatric disorder in adolescents and adults.

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APPENDICES

Appendix 1: Data Collection Procedures

Following is a detailed account of data collection procedures used in this study. The items in CAPITAL letters are those that were on the original "Data Collection Form" used in collecting the raw data from the clinical files at the Center studied. These data were then entered onto a floppy disk for later statistical analysis. The indented comments enclosed in brackets [ ] are to explain how the data were actually used in the data analysis, since much of it had to be adapted or combined with other data for synthesized scores. Starred items * represent data that were collected on the data collection form, but were not entered into the data pool for statistical analysis.

Some of the data were recorded in "weighted" scores, rather than simple "presence" or "absence" of a particular phenomenon. This was done to reflect differences that were judged as important by the researcher. For example, if a youth physically assaults a parent, that seems a quite different experience (psychologically) than getting in a fight with a peer at school. Both would be counted, for the purposes of this study, as "assault." But in the initial stages of statistical analysis the violence directed toward a parent was given a "heavier" weighting to reflect what appears to be a more serious issue. (See

below for discussion of why "weightings" were eventually dropped from the analysis.)

In no case were there unarguable theoretical reasons for the weightings chosen. Weighting the assault of a parent "3," and that of a peer "1," was simply to reflect the researcher's assumption of the different psychological meaning of these experiences for a youth. No presumption is made that the weightings have any empirical basis, nor was there any attempt to assess the relative psychological "impact" of the experiences (i.e., it is not assumed that assaulting a parent has "three times as great" an impact as assaulting a peer would have).

The weightings are merely chosen to reflect the fact that differences exist, and to provide at least a crude means of quantifying those differences. To use another example, it seemed important to reflect numerically that there is a psychological difference for an adolescent between losing a parent by suicide (Cain & Fast, 1972; Warren, 1972), versus losing a parent due to cancer, even if one cannot fully assess the impact of those differences.

However, in the final data analysis, the weighted scores were not used. In most cases, the simple presence or absence of a particular phenomenon or symptom was entered into the analysis (e.g., the fact that a child either had or had not lost a parent through death was

entered, but the nature of the parental death [suicide, homicide, accidental, illness] was not entered in as a "weighting"). It simply proved too complex a task to quantify even major differences (let alone nuances) in the enormously varied life experiences of the subjects in this sample. In addition, the weightings assigned were not as readily defensible, when vigorously challenged, as had originally been anticipated by the researcher.

Wherever possible, data were recorded directly from the clinical file without any change. In the initial data collection, weighted scores were used for a number of measures, but those were ultlimately recoded for the data analysis. The numbers next to some of the items which follow reflect the weighted scores. However, the recoded scores were in the form of "presence"= 1 and "absence"= 0, or else "yes"= 1 and "no"= 0 for each item (e.g., legal custody of child was changed from weighted scores to a simple "yes/no" about whether or not parents had custody).

ID # *

GENDER

DATE OF BIRTH *

AGE:

DIAGNOSIS * Schizophrenia, Schizophreniform Disorder, Atypical Psychosis

DATE OF ADMISSION *

DATE OF DISCHARGE *

DURATION OF CURRENT HOSPITALIZATION Number of days ___

(The actual number of days the adolescent was a patient at this facility for this particular admission. This total does not include the days of continued hospitalization at any other facility, even if the youth was transferred there directly.)

CHILD IN LEGAL CUSTODY OF: 3.parent 2.guardian or foster parent 1.court

[This was entered into the data analysis as "yes/no, child is in custody of parents."]

ADMISSION STATUS: Voluntary? yes no

FOLLOW-UP FACILITY: 5.psychiatric hospitalization 4.short term drug-alcohol inpatient treatment 3.day treatment 2.outpatient 1.medication only 0.none no/info

(How restrictive was the level of post-discharge followup recommended? This ranged from no recommendations at all for followup treatment, to recommendations for transfer to a longterm care facility.)

GROSS MONTHLY INCOME (from all sources)

PARENT OCCUPATION: * (Mother Father)

HOLLINGSHEAD INDEX *

[The researcher had hoped to get a rough indicator of socioeconomic status from parental occupations and family income. However, there were too many missing data to permit this.]

CURRENT FUNCTIONING

SUICIDE: total # gestures/attempt ___ no/info

[This measure included all known gestures or attempts at suicide. It also included threats of suicide that were taken seriously enough to warrant hospitalization, even if the client had not yet acted upon those threats.]

ASSAULT:

total # threats ___

[This includes all known threats of homicide; and all non-homicidal threats that were taken seriously enough to warrant hospitalization, even if the adolescent did not act upon those threats.]

total # attempts ___ no/info

[Assaultive incidents were initially weighted by taking the total number of such attempts and multiplying times three. This was done to avoid

having threats and actual assaultive behavior equated in the summary score, which was a single numerical "total" for assault. However, weightings were ultimately deleted for purposes of analysis, and the total is simply a count of the number of recorded incidents.]

TARGET: 3/0.parent 2/0.sibling 1/0.(other family, friend, stranger) no/info

[This has reference to the "target" of the assaultive behavior. The weightings, as previously explained, are simply to reflect the psychological difference between violence directed toward a parent, and violence directed toward others. There is no theoretical basis for the weightings chosen; nor is there any attempt to assess the relative psychological "impact" via the weightings. Ultimately, however, this item was deleted from the study.]

MENTAL HEALTH HISTORY

PRIOR DYSFUNCTIONAL EPISODES: none no/info

onset (age): #1____ #2____ #3____

duration (weeks): #1____ #2____ #3____

[This measure was not assessed consistently by the staff at the center. There resulted in so

much missing data that it was virtually useless as a measure. Part of the reason it was so poorly assessed may be due to the fact that there were no guidelines for intake staff regarding what constituted a "dysfunctional episode."]

PRIOR PSYCHIATRIC TREATMENT (Outpatient or non-hospital):
none no/info

age: #1____ #2____ #3____

(Outpatient treatment at any time prior to admission. Duration of such treatment was not generally recorded, and is not taken into account in the score. In the actual analysis, only the mere fact of whether or not a subject had received any outpatient therapy was utilized.)

PRIOR PSYCHIATRIC HOSPITALIZATIONS (At this Center):
none no/info

age: #1____ #2____

duration(days): #1____ #2____

PRIOR PSYCHIATRIC HOSPITALIZATIONS (At any other Center):
none no/info

age: #1____ #2____

duration (days): #1____ #2____

[In the final statistical analysis, the above

two measures were added into a single score that reflected simply the presence or absence of any prior psychiatric hospitalization at this Center or any other Center. Only the number of hospitalizations was entered as a variable, since the duration of those at other facilities was not consistently available.]

MENTAL STATUS EXAMINATION (at intake)

[Those items that are underlined are seen as tending toward psychological "health" rather than "pathology," except as noted below. Each subject had a "Healthy" score and a "Pathology" score figured for each of the mental status domains. The scores were determined by simply adding the total number of items marked for each subject. Such "collapsing" of data was mandated by the statistical necessity of reducing the number of variables to be entered into the factor analysis. The mental status data were thereby reduced from 90 discrete variables to only 12 summary scores, which were the data entered into the subsequent statistical analyses.]

MOTOR ACTIVITY- WNL, relaxed, hyperactive, hypoactive, restless, tremors, tics, posturing, pacing, paralysis (specify), eye contact (4.good, 3.intermittent, 2.fair, 1.poor, 0.none), pressured speech....

[For the statistical analysis, "eye contact" scores of 4, 3, or 2 were rated as equivalent, and included in the "Healthy" score. Scores of 1 or 0 were rated as equivalent, and included in the "Pathology" score.]

MOOD/AFFECT- appropriate, elated, apathetic, calm, anxious, labile, fearful, depressed, worried, angry, blunted, flattened, euphoria, excited, inappropriate (specify)

[If "appropriate" was marked for a given subject, then the items underlined counted toward the "Healthy" total; otherwise they were counted toward the "Pathology" total.]

MANNER/ATTITUDE- critical, suspicious, disinterested, irritable, threatens violence, assaultive, destructive, withdrawn, impulsive, argumentative, cooperative, positive, constructive, receptive

THOUGHT PROCESSES- appropriate, concrete, blocking, confused, incoherent, response latency, irrelevant, tangential responses, neologism, perseveration, flight of ideas, dissociated, coherent, organized

THOUGHT CONTENT- appropriate, helplessness, worthlessness, delusions, phobias, obsessions, compulsions, guilt, ideas of reference, hallucinations (auditory, visual, other), suicide (idea, plan), homicide (idea, plan)

[If "appropriate" was marked for a given

subject, then the items underlined counted toward the "Healthy" total; otherwise they were counted toward the "Pathology" total.]

SENSORIUM- clouded consciousness, disorientation (time, place, person), oriented, memory loss (none, remote, recent, immediate), judgment (3.good, 2.fair, 1.poor), insight (3.good, 2.fair, 1.poor)

[For the statistical analysis, "judgment" and "insight" scores of 3 or 2 were rated as equivalent, and included in the "Healthy" total; while scores of 1 were included in the "Pathology" total.]

LEGAL STATUS

PENDING LEGAL ACTIONS OR PROBS: 2/0.crim 1/0.non-crim none no/info

PAST LEGAL ACTIONS/RESULTS: 2/0.criminal 1/0.non-crim none no/info

Was child-- offender: yes no prosecuted: yes no
"jail": yes no

1.one time only 2.repeat offender no/info

[The scores pertaining to criminal behavior were summed for a single index score of criminal involvement.]

FAMILY HISTORY (Environment)

PARENTS (Current):

FATHER 3.Bio 2.(Adopt Step) 1.Live-in 0.None Age * No/info

MOTHER 3.Bio 2.(Adopt Step) 1.Live-in 0.None Age * No/info

RESIDENTIAL CHANGES OR MOVES: # in last five years ____

SOCIOECONOMIC FACTORS AND LIVING CONDITIONS

Family Housing arrangement: 4.own/home 3.renting 2.(live/w/relatives OR live/w/others) 1.temp/shelter 0.none no/info

SIGNIFICANT FAMILY OR OTHER LOSSES:

Child age at mother death ___

3.suicide 2.homicide 1.(illness accident)

Child age at father death ___

3.suicide 2.homicide 1.(illness accident)

Child age at sibling death ___

3.suicide 2.homicide 1.(illness accident)

Child age significant other death

3.suicide 2.homicide 1.(illness accident)

[The weightings simply reflect the fact that different causes of death may have differing psychological impact on the child. No other meaning should be imputed to, or inferred from, the weightings. There was no theoretical basis for the weights selected.]

[One thing that the weightings do not reflect was how closely any given subject was involved in the death of the parent. A few subjects were the first to find the body of a

parent who had died. In at least one instance, the child witnessed the suicidal death of a parent. Clearly, such experiences are going to profoundly affect the child involved; but attempting to quantify such experiences for statistical analysis proved beyond the capacity of this researcher. Ultimately, the weighted scores were dropped, and only the simple fact of whether or not a child had lost a parent through death was entered into the analysis.]

FAMILY HISTORY:

1-mother 2-father
Criminal Hx: 3-sibling
4-mtrnl gr-mo 5-mtrnl gr-fa
Mental Illness: 6-ptrnl gr-mo 7-Mtrnl gr-fa
8-mtrnl other 9-ptrnl other
Suicide:
0-none no/info
Drug abuse:
ETOH abuse:

[In preparing the above for statistical analysis, the categories of:

"Mental Illness" and "Suicide" were both given a weighting of 3;

"Drug Abuse" and "Alcohol Abuse" were both given a weighting of 2;

"Criminal History" was given a weighting of 1.

[Within each of those categories, any single datum identified by:

1 or 2 was changed to a 3
3,4,5,6,7 was changed to a 2
8 or 9 was changed to a 1

[Each separate datum thus changed was multiplied by the weighting for its category. The sum of all these multiplied data (within a category) is the "weighted score" for that category. The sum of the weighted scores for all of the categories was computed to determine a single "Family History" index score, which was then entered into the statistical analysis.]

[This index score was intended as a rough measure of the extent of family pathology or dysfunction, which has theoretically been defined as related to both genetic and environmental factors within the family. Reduction of the data into a single index score was forced by the necessity of reducing the number of variables entered into the factor analysis. Clearly such intangible variables as the "seriousness" of a family member's pathology are not reflected in the summary score.]

[As with the other weighted scores, this one was ultlimately dropped from the final

analysis. The family history score finally used is simply an additive total of the biological relatives who were identified in the clinical record as suffering from mental illness, alcoholism, etc.]

DEVELOPMENTAL HISTORY

PREGNANCY: If "preemie": _____days

[No decision was made by the researcher as to what constituted a premature birth. If the parent (or whoever provided the developmental information) listed the birth as "premature," the information was entered into the statistical analysis. As it turned out, this variable did not attain statistical significance for this study. However, given the fact that low birth weight has been implicated as a contributory factor in research on a variety of disorders, further study of this variable appears warranted.]

BIRTHWEIGHT:_____oz.

[See comment immediately above on "pregnancy."]

DURING PREGNANCY:

ALCOHOL: Ounces/week _____ no/info

[It turned out that even an approximate measure of prenatal alcohol consumption was impossible to determine. In the statistical analysis, only a "yes/no" measure of maternal alcohol usage was entered.]

DRUGS: heroin___ LSD___ no/info
cocaine___ other halluc___
amphetamines___ PCP___
barbiturates___ marijuana

[A simple additive summary of drug usage was finally entered into the statistical analysis. It was not possible to determine the frequency or extent of maternal drug usage.]

WEANING: age(mos)___

TOILET TRAINING: completed at age ____(mos.)

WALKING: age____(mos.)

[Due to the extent of missing data, not a single one of the three developmental milestones above was available for statistical analysis.]

PHYSICAL ABUSE:

Abuse: duration/months___ age began:____ no/info

who: 3/0.parent 2/0.other/caretaker 1/0.non/caretaker

frequency: 1.one/time/only 2.repeated

child injured: yes no Hospitalization? yes no

SEXUAL ABUSE:

duration/months_____ age began____ no/info

who: 3/0.parent 2/0.other/caretaker 1/0.non/caretaker

frequency: 1.one/time/only 2.repeated

child injured: yes no Hospitalization? yes no

[The information regarding physical and sexual abuse was generally recorded by staff in the clinical notes as it was learned from the client; although sometimes the information was available at intake, and appeared on the intake form. However, there was no consistency in the nature and amount of information recorded. In the final statistical analysis, only the bare fact of "yes/no" regarding the occurrence of abuse was entered.]

[This resulted in a serious over-simplification of the data. Several of the youth admitted to the Center had appalling histories of extensive abuse. In some instances, there was repeated, brutal abuse by multiple abusers, including parents and other caretakers. Some of the youth had been subjected to abuse for virtually their entire lives. Some had also been injured severely enough to require medical hospitalization.]

[However, there was simply no way the researcher could quantify the data to reflect the variety, complexity, and potential psychological impact of the abusive experiences to which many of these youth had been subjected. In the end, simply entering the fact of abuse or (apparent) non-abuse appeared to be the only feasible alternative for statistical analysis. The amount of missing data was another major factor in this decision.]

[But as the researcher read through the files, and came across incident after incident of physical and sexual abuse (not to speak of the verbal and emotional "battering" that some of the youth reported), it seemed evident that this is an area needing much further research. It seems entirely plausible that the cumulative trauma of severe, repeated abuse could contribute in a causal way to the development of serious psychopathology.]

Was child EVER RAPED? yes no How many times? ___

RAPIST: 4/0 parent 3/0 other adult relative

2/0 other adult 1/0 peer

[A response limited to "yes/no" for ever having been raped was finally the only item entered for statistical analysis. However, the same concerns that were expressed above regarding physical and sexual abuse apply equally to the data for rape. It is important to note that some of these youth had been sexually abused, and had also experienced forcible rape.]

FOSTER HOME PLACEMENT OR INSTITUTIONALIZATION: no/info

Age first foster care: Total foster care (mos.):

[In the final statistical analysis, only the fact of "yes/no" to ever having required foster care was entered. The reasons for the foster care, the number of times the child was placed in foster care, and the total duration of foster care are not reflected in the score.]

CHILD'S STRENGTHS:

total # listed hobbies, talents, skills: ____

[This was an area not recorded consistently by the Center staff. Some of the youth appeared to have a number of interests and skills; while others appeared to be extremely limited. However, there was no way to use the data due to the frequency with which this item was <u>not</u>

recorded by staff, despite being identified as one of the areas to be explored with all patients.]

CURRENT GRADE OR HIGHEST GRADE COMPLETED: ___

SPECIAL SERVICES EVER NEEDED?

total #: age/began: no/info

[This was entered in the statistical analysis as a simple "yes/no" regarding special services usage. The reason for special services, the extent of impairment or disability, and the duration of special services are not reflected in the score.]

ADMISSION "GAS": ___

[The "Global Assessment Scale" was used by clinical staff to determine a single numerical rating of current functioning at the time of intake. The word "scale" may be seen as something of a misnomer by those with a background in psychometrics, especially since this scale has no validity or reliability information. It is not a scale constructed from a variety of discrete items, such as the scales on the MMPI. This scale did include written guidelines for determining the individual's

level of functioning. No comparable assessment was completed upon discharge.]

MEDICAL HISTORY CHECKLIST

[All the items in the medical history marked with ^ were added to obtain a single "Illness" summary score for the factor analysis.]

[All the items in the medical history marked with + were added to obtain a single "neurological" summary score for the factor analysis.]

RATE PRESENT HEALTH: 3.good 2.fair 1.poor

^CURRENT MEDICAL PROBLEMS: yes no

^CURRENT MEDICATIONS: yes no

CURRENT DRUG/ALCOHOL: Recent: 1.decrease 2.same 3.increase No/info

marijuana	00. none	07. once/2-3wk
cocaine	01. < 1/yr	08. 1/wk
amphetamines	02. once 6mo-1yr	09. 2-3/wk
heroin	03. once/6mo	10. 4-5/wk
barbiturates	04. once/4-5mo	11. 1/day
alcohol	05. once/2-3mo	12. 2-3/day
hallucinogens	06. 1/mo	
PCP		
crack		
other		

[A "Drug Abuse" summary score was obtained by adding the weighted scores (weighted for

frequency of use) for each drug that the youth is known to have used. The scores reflect all drug usage reported in the clinical file, even if the youth denied current usage. As with previous issues, there is no theoretical basis for the weightings chosen. It was assumed that more frequent usage poses a more serious problem than less frequent usage. It was not possible to consistently determine the full extent and duration of drug abuse for each subject, so these weighted data were not included in the study.]

[Since drug data were reliant on youth self-report, they are clearly suspect. However, this information was judged too important to be deleted from the study because of that limitation. It may seem inappropriate to equate marijuana usage with PCP or crack usage; but the necessity of reducing the number of variables for the factor analysis mandated this decision.]

[Ultimately, this variable did not prove statistically significant, and was deleted. However, the DSM-III and DSM-III-R both comment on the similarity of schizophrenic symptoms and the behavioral manifestations of certain forms of drug abuse. The possibility of drug induced

schizophrenia is one that requires further research.]

HAS HE/SHE HAD:	NO	NOW	PAST
^ heart disease			
^ high blood pressure			
^ rheumatic fever			
^ kidney problems			
^ eczema			
^ cancer			
^ asthma			
^ diabetes			
^ thyroid problems			
^ frequent colds/sore throat			
+ exposure to solvents or pesticides for a long time			
+ epilepsy/convulsions seizures			
^ back pain			
^ ulcers			
^ hepatitis/jaundice/yellow skin			
^ blood disorders (severe anemia, leukemia, Sickle cell anemia)			
+ severe headaches			
+ dizziness/fainting			

^ANY OTHER HEALTH PROBLEMS OR DISEASES? yes no

^ANY KNOWN ALLERGIES: yes no

^IS S/HE ON A SPECIAL DIET? yes no

^HAS S/HE EVER HAD ANY SERIOUS INJURIES? yes no

+HAS S/HE EVER BEEN KNOCKED UNCONSCIOUS OR SUFFERED A HEAD INJURY? yes no

^HAS S/HE EVER HAD ANY SURGERIES? yes no

HAS SHE EVER BEEN PREGNANT? no/info Number of:

livebirths; miscarriages; abortions; stillbirths

^HAS S/HE EVER BEEN TREATED FOR A DRUG OVERDOSE OR ACCIDENTAL POISONING? yes no no/info

^EVER BEEN HOSP'D FOR ANY OTHER REASON? total #; no/info

DOES S/HE SMOKE? 0. none 1. < 1/2 pk/day 2. 1/2-1 pk/day 3. 1-1&1/2 pk/day 4. 2 pk/day 5. > 2 pk/day

^RECOMMENDATION FOR MEDICAL CONSULTATION? yes no no/info

PHYSICAL EXAMINATION (In-house)

Blood Pressure _____/_____

(Are systems normal?):

HEENT	yes	no	extremities	yes	no
lungs	yes	no	neuro	yes	no
abdomen	yes	no	GU	yes	no
CVA	yes	no			

[These were eventually combined (except for blood pressure) for a single summary score.]

PSYCHIATRIC CLINICAL DATA
(during current hospitalization)

HALLUCINATIONS: none no/info

auditory: sound (1/0) voice (1/0) refer/to or/about/patient (1/0)

visual: formed (1/0) nonformed (1/0)

olfactory (1/0) tactile (1/0) gustatory (1/0)

chemical/organic/basis?: yes no (1/0)

[A summary score for "Hallucinations" was obtained by simply adding the total number of "yes/no" "presence/absence" responses regarding hallucinations (each response scoring 1 or 0). This was recognized by the researcher as being a gross over-simplification, but there did not

appear to be any way of measuring the subjective "intensity" of the hallucinatory experiences, nor the degree to which they interfered with functioning.]

DELUSIONS: none no/info
paranoid (1/0) grandiose (1/0) somatic (1/0)
nihilistic (1/0) reference (1/0) being/influenced (1/0)
thought/broadcast (1/0) thought/insertion (1/0)
thought/withdrawal (1/0) other:_____ (1/0)
bizarre: yes no (1/0)

[A summary score for "Delusions" was obtained in the same way as was done with "Hallucinations," by simply adding the total number of "yes/no" responses regarding delusions (yes= 1, no= 0). This once again resulted in over-simplification of the clinical reality.]

[The researcher could not find a logically defensible method for quantifying the impact of the delusions, how complete or extensive the delusional systems might be, nor the extent to which they were debilitating. Whatever errors may have crept in due to using this admittedly simplistic method for quantifying delusional ideation, it appears less erroneous than

deleting the information altogether would have been.]

ASSAULTIVE while on unit: none staff peers

[The score for "ASSAULT" (while in the hospital) was determined by adding the "yes/no" responses for assault toward staff or peers. The total number of assaultive incidents was not searched out in the clinical notes, but is reflected in the time spent in the "quiet room" (see below).]

Use of "QUIET ROOM" necessary? hours duration_____
total frequency of use____ no/info
Was client "runner"? total/run #_____ no/info

[The total number of hours in the "quiet room" was the only one of these measures that turned out to be significant at a high enough level for inclusion in the final statistical analysis. The quiet room was used sparingly for adolescents manifesting assaultive or otherwise "out of control" behavior, including actual or attempted runaway from the facility.]

MEDICATION required:
during hospitalization yes no
at discharge yes no

[These were added for a "Medication" summary score for the statistical analysis. The summary score clearly does not reflect dosage, type of medication, or extent of use.]

Did PARENTS VISIT at all in hospital: yes no

Did PARENTS PARTICIPATE IN FAMILY COUNSELING while in hospital: yes no

["Parents Visit" and "Parents Participate in Family Counseling" were added to obtain a summary score of "Parental Support." This is merely a simple "yes/no" count; no attempt was made to assess "quality" of parental support.]

14-DAY INVOLUNTARY sought? present admission: yes no

prior admit #1 THIS CENTER yes no

prior admit #2 THIS CENTER yes no

180 DAY INVOLUNTARY sought, present admission: yes no

[Was the client recommended (even if not actually committed) for additional involuntary treatment, at this or any other center, in the course of this hospitalization? The responses were eventually combined into a single non-additive index score that merely reflected the fact of recommendation for involuntary hospitalization beyond the original 72-hour

detention. No measure of the duration of subsequent hospitalization was possible from the data available to the researcher.]

PSYCHOMETRIC DATA

[The Center had an informal policy that most of the patients who were admitted to the Adolescent Unit were referred for psychological testing. However, the youth whose files were used in this study did not, as a group, receive the full battery of tests. In fact, many of them were not tested at all. There were no consistent notations in the chart as to why a particular youth was or was not tested. Due to the extent of missing data (i.e., subjects who were not tested), this entire body of data was deleted, with great regret, from the statistical analyses.]

WISC-R or WAIS-R ? *

verbal score____ performance score____ full scale____

WISC-R/WAIS-R SCALE SCORES:

information___; similarities___; arithmetic___; vocabulary___; comprehension___; digit span___; picture completion___; picture arrangement___; block design___; object assembly___; coding___; mazes___.

MMPI SCALE SCORES

?___; L___; F___; K___; Hs___; D___; Hy___; Pd___;
Mf___; Pa___; Pt___; Sc___; Ma___; Si___.

WRAT or WRAT-R ? * Level I or Level II ? *

reading___ spelling___ arithmetic___

16 PF

Form:___ *

Factor scores:

A___ B___ C___ E___ F___ G___ H___ I___ L___ M___
N___ O___ Q1___ Q2___ Q3___ Q4___

RORSCHACH

[The scoring system used by the psychologist at the Center was a blend of the Exner system and the psychologist's own system. The coding of data for this study consisted of a simple count of the various types of responses (as recorded by the psychologist administering the test), to each of the 10 stimulus cards. If this seems excessively simplistic, the reader is reminded that a good many competent psychologists have attempted, without success, to quantify the Rorschach in an effort to make it a somewhat more reliable instrument. As it turned out, there were not enough subjects who completed the Rorschach to include these data in the study's statistical analysis.]

Appendix 2: Descriptive Statistics for Variables Used in the Analyses

Table 16

Descriptive Statistics for Variables Used in the Analyses: Involuntary Commitment

---

PATTERN MATRIX VARIABLE NAME: Commit

APPENDIX 1 VARIABLE LABEL: Involuntary commitment sought?

MEANING: Was involuntary psychiatric commitment sought (even if not granted by the court) at any time during the patient's hospitalization? This does not include those patients who were originally hospitalized on a 72 hour hold order, unless additional commitment was sought after the original 72 hour emergency hospitalization.

VALUE LABEL: 1 = yes (psychiatric commitment was sought)
0 = no (psychiatric commitment was not sought)

HOW MEASURED: (self explanatory)

DESCRIPTIVE STATISTICS: 46.5 % scored 1 n= 33
53.5 % scored 0 n= 38

Table 17

Descriptive Statistics for Variables Used in the Analyses: Assaultive Behavior Prior to Hospitalization

---

PATTERN MATRIX VARIABLE NAME: Aslthret

APPENDIX 1 VARIABLE LABEL: Attempts or serious threats of assault

MEANING: Any assaultive behavior occurring prior to hospitalization; or threats of assaultive behavior which were serious enough to warrant hospitalization; or threats to kill another person that were made prior to hospitalization.

VALUE LABEL: The actual number of assaultive behaviors or serious threats occurring prior to hospitalization, each incident scored as "1" in determining total.

HOW MEASURED: Actual count of incidents recorded in file

DESCRIPTIVE STATISTICS: mean= 2.338

SD= 3.573

range= 0 thru 17

"zero" scores= 50.7% n=36

Table 18

Descriptive Statistics for Variables Used in the Analyses: Admission Status

---

PATTERN MATRIX VARIABLE NAME: Admstat

APPENDIX 1 VARIABLE LABEL: Admission Status

MEANING: Was the patient admitted to the hospital on a voluntary basis?

VALUE LABEL: 1 = yes (admission was voluntary)

0 = no (admission was not voluntary)

HOW MEASURED: Actual count of incidents recorded in file

DESCRIPTIVE STATISTICS: 69.0 % scored 1 n= 49

31.0 % scored 0 n= 22

Table 19

Descriptive Statistics for Variables Used in the Analyses: Assualtive Behavior While Hospitalized

---

PATTERN MATRIX VARIABLE NAME: Assaltot

APPENDIX 1 VARIABLE LABEL: Total assaultive behavior

MEANING: The total number of physically assaultive or aggressive behaviors occurring while the patient was hospitalized, including aggressive behavior toward staff or other patients.

VALUE LABEL: The number of recorded incidents, each incident scored as "1" in determining total.

HOW MEASURED: Actual count of incidents recorded in file

DESCRIPTIVE STATISTICS: mean= .437

SD= .579

range= 0 thru 2

"zero" scores= 60.6% n=43

Table 20

Descriptive Statistics for Variables Used in the Analyses: Prior Hospitalizations

---

PATTERN MATRIX VARIABLE NAME: Priorhos

APPENDIX 1 VARIABLE LABEL: Prior hospitalizations

MEANING: Did the patient have a record of prior psychiatric hospitalizations at this or any other facility?

VALUE LABEL: Number of prior psychiatric hospitalizations known to have occurred at this or any other facility.

HOW MEASURED: Actual count as recorded in clinical file

DESCRIPTIVE STATISTICS: mean= 1.127

SD= 1.218

range= 0 thru 4

"zero" scores= 39.4% n=28

Table 21

Descriptive Statistics for Variables Used in the Analyses: Post-Discharge Treatment Recommendations

---

PATTERN MATRIX VARIABLE NAME: Followup

APPENDIX 1 VARIABLE LABEL: Followup facility

MEANING: How restrictive was the level of post-discharge treatment recommended? This ranged from no followup treatment, to recommendations for transfer to a longterm care facility. Higher numbers reflect increasingly restrictive treatment recommendations.

VALUE LABEL: 5= longterm psychiatric hospitalization
4= drug/alcohol inpatient treatment
3= day treatment
2= outpatient treatment
1= medication only
0= no treatment necessary

5= 50.7% (n= 36)
4= 1.4% (n= 1)
3= 14.1% (n= 10)
2= 28.2% (n= 20)
1= 1.4% (n= 1)
0= 4.2% (n= 3)

Table 22

Descriptive Statistics for Variables Used in the Analyses: Length of Time Youth was Confined to "Quiet Room"

PATTERN MATRIX VARIABLE NAME: QRhours

APPENDIX 1 VARIABLE LABEL: "Quiet room" hours

MEANING: The total number of hours that the patient spent in the "quiet room" for assaultive or out of control behavior.

VALUE LABEL: Actual number of hours

HOW MEASURED: By totaling the number of hours on all "quiet room" observation reports for each subject.

DESCRIPTIVE STATISTICS: mean= 29.028

SD= 91.498

range= 0 thru 737

median= 4.0

"zero" scores= 35.2% n=25

Table 23

Descriptive Statistics for Variables Used in the Analyses: Prior Outpatient Psychiatric Treatment

---

PATTERN MATRIX VARIABLE NAME: Prtx.op

APPENDIX 1 VARIABLE LABEL: Prior outpatient treatment

MEANING: Has the patient ever had outpatient psychiatric treatment prior to the current hospitalization?

VALUE LABEL: 1= yes

0= no

HOW MEASURED: (self explanatory)

DESCRIPTIVE STATISTICS: 57.7% scored 1 n= 41

42.3% scored 0 n= 30

Table 24

Descriptive Statistics for Variables Used in the Analyses: Pathological Manner/ Attitude

---

PATTERN MATRIX VARIABLE NAME: P.Manner

APPENDIX 1 VARIABLE LABEL: Mental status examination: Pathological manner/attitude

MEANING: Those items in the "Manner/Attitude" section of the mental status examination that tend toward pschopathology, specifically in reference to interaction with clinical staff during the intake.

VALUE LABEL: Number of items identified by clinical staff member who completed the mental status examination, with each item scored "1" or "0" in determining total (see Appendix 1 for further details).

HOW MEASURED: Actual count of items

DESCRIPTIVE STATISTICS: mean= 1.268

SD= 1.298

range= 0 thru 7

"zero" scores= 26.8% n=19

Table 25

Descriptive Statistics for Variables Used in the Analyses: Suicidal Behaviors

---

PATTERN MATRIX VARIABLE NAME: Suicide

APPENDIX 1 VARIABLE LABEL: Suicide attempts or serious threats

MEANING: Any recorded reports of suicide attempts or gestures, and any suicide threats taken seriously enough to warrant hospitalization.

VALUE LABEL: Actual number of attempts or serious threats, each scored as "1" in determining total.

HOW MEASURED: Actual count of incidents recorded in file

DESCRIPTIVE STATISTICS: mean= 1.789

SD= 2.242

range= 0 thru 9

"zero" scores= 31.0% n=22

Table 26

Descriptive Statistics for Variables Used in the Analyses: Family Pathology

---

PATTERN MATRIX VARIABLE NAME: F.Etcrim

APPENDIX 1 VARIABLE LABEL: Family history of alcohol abuse, and family history of criminal behaviors.

MEANING: Five measures of possible pathological functioning within the family were assessed. These included any biological relative or step-parent mentioned in the clinical file who had a history of: (1) mental illness [i.e., prior psychiatric treatment for any reason], (2) suicide [i.e., suicidal death rather than mere attempts], (3) alcohol abuse [i.e., treatment for alcoholism, attendance at AA, or references in the patient's file about serious drinking problems], (4) drug abuse [i.e., treatment for drug abuse, attendance at Narcotics Anonymous, or references in the patient's clinical file about serious drug abuse problems-- including misuse of prescription medications], and, (5) criminal history [i.e., mention in the patient's clinical file of any felony-level criminal activities, past prison record, current jail sentences, or pending criminal charges-- including child abuse]. Each variable was merely the additive total of biological relatives

---

Table 26 (continued)

Descriptive Statistics for Variables Used in the Analyses: Family Pathology

identified who displayed the requsite behavior. No attempt was made to quantify the degree of biological relationship with the subject.

A factor analysis of these five variables was completed, with three factors being identified (see Table ****). All three factors identified were re-coded, and entered into the primary factor analysis as discrete variables. As all the variables were subjected to more refined analysis, only one of these three derived "family history" variables retained a factor loading that was statistically significant in the final factor analysis.

VALUE LABEL: Each biological relative or (current) step-parent who was identified as displaying the requisite behaviors was coded "1".

HOW MEASURED: Additive total of relatives manifesting the target behavior.

DESCRIPTIVE STATISTICS: mean= .789

SD= 1.094

range= 0 thru 6

"zero" scores= 54.9% n=39

Table 27

Descriptive Statistics for Variables Used in the Analyses: Physical Abuse

PATTERN MATRIX VARIABLE NAME:	PA.Yes
APPENDIX 1 VARIABLE LABEL:	Physical Abuse
MEANING:	Has the subject ever been physically abused?
VALUE LABEL:	10=yes
	0=no
HOW MEASURED:	(self explanatory)
DESCRIPTIVE STATISTICS:	33.8% scored 10 n=24
	66.2% scored 0 n=47

Table 28

Descriptive Statistics for Variables Used in the Analyses: Parental Marital Status

---

PATTERN MATRIX VARIABLE NAME: Biopar

APPENDIX 1 VARIABLE LABEL: Biological parents

MEANING: Are biological parents reported as currently married and living together?

VALUE LABEL: 1= yes

0= no

HOW MEASURED: (self explanatory)

DESCRIPTIVE STATISTICS: 31.0% scored 1 n= 22

69.0% scored 0 n= 49

Table 29

Descriptive Statistics for Variables Used in the Analyses: Foster Care

PATTERN MATRIX VARIABLE NAME:	Fostcare
APPENDIX 1 VARIABLE LABEL:	Foster care
MEANING:	Has the child ever been in foster care?
VALUE LABEL:	1= yes
	0= no
HOW MEASURED:	(self explanatory)
DESCRIPTIVE STATISTICS:	67.8% scored 1 n= 48
	32.4% scored 0 n= 23

Table 30

Descriptive Statistics for Variables Used in the Analyses: Legal Custody of Youth

PATTERN MATRIX VARIABLE NAME:	Custody
APPENDIX 1 VARIABLE LABEL:	Legal custody of child
MEANING:	Is child in legal custody of parents?
VALUE LABEL:	1= yes
	0= no
HOW MEASURED:	(self explanatory)
DESCRIPTIVE STATISTICS:	23.9% scored 1 n= 17
	76.1% scored 0 n= 54

Table 31

Descriptive Statistics for Variables Used in the Analyses: Parental Support During Hospitalization

---

PATTERN MATRIX VARIABLE NAME: Prntsupp

APPENDIX 1 VARIABLE LABEL: Parental support

MEANING: Did parents visit child while in the hospital? Did parents participate in the family counseling offered?

VALUE LABEL: 1= yes

0= no

HOW MEASURED: Additive total of "yes/no" response to both questions.

DESCRIPTIVE STATISTICS: mean= 1.239

SD= .918

range= 0 thru 2

"zero" scores= 32.4% n=23

Table 32

Descriptive Statistics for Variables Used in the Analyses: Sexual Abuse

---

PATTERN MATRIX VARIABLE NAME: SA.Yes

APPENDIX 1 VARIABLE LABEL: Sexual Abuse

MEANING: Has the subject ever been sexually abused?

VALUE LABEL: 10=yes

0=no

HOW MEASURED: (self explanatory)

DESCRIPTIVE STATISTICS: 21.1 % scored 10 n=15

78.9 % scored 0 n=56

Table 33

Descriptive Statistics for Variables Used in the Analyses: Pathological Thought Processes

---

PATTERN MATRIX VARIABLE NAME: P.Thproc

APPENDIX 1 VARIABLE LABEL: Mental status examination: Pathological thought processes

MEANING: Those items in the "Thought Processes" section of the mental status examination that tend toward pschopathology

VALUE LABEL: Number of items identified by clinical staff member who completed the mental status examination, with each item scored "1" or "0" in determining total (see Appendix 1 for further details).

HOW MEASURED: Actual count of items

DESCRIPTIVE STATISTICS: mean= 2.634

SD= 1.846

range= 0 thru 7

"zero" scores=12.7% n=9

Table 34

Descriptive Statistics for Variables Used in the Analyses: Pathological Sensorium

---

PATTERN MATRIX VARIABLE NAME: P.Sensor

APPENDIX 1 VARIABLE LABEL: Mental status examination: Pathological sensorium

MEANING: Those items in the "Sensorium" section of the mental status examination that tend toward pschopathology

VALUE LABEL: Number of items identified by clinical staff member who completed the mental status examination, with each item scored "1" or "0" in determining total (see Appendix 1 for further details).

HOW MEASURED: Actual count of items

DESCRIPTIVE STATISTICS: mean= 2.831

SD= 2.208

range= 0 thru 8

"zero" scores= 12.7% n=9

Table 35

Descriptive Statistics for Variables Used in the Analyses: Delusional Ideation

---

PATTERN MATRIX VARIABLE NAME: Delustot

APPENDIX 1 VARIABLE LABEL: Total of delusions

MEANING: The number of different types of delusional ideation noted during the hospital stay (e.g., paranoid delusions, delusions of grandeur, nihilistic delusions)

VALUE LABEL: Number of types of delusions noted, with each type scored "1" in determining total (see Appendix 1 for further details).

HOW MEASURED: Actual count from clinical file

DESCRIPTIVE STATISTICS: mean= .930

SD= .931

range= 0 thru 3

"zero" scores= 38.0% n=27

Table 36

Descriptive Statistics for Variables Used in the Analyses: Global Assessment Scale

---

PATTERN MATRIX VARIABLE NAME: GAS

APPENDIX 1 VARIABLE LABEL: Global Assessment Scale

MEANING: Overall assessment of functioning at the time of intake. The GAS score is a single numeric score that was determined by the intake worker, and is based on a scale that ranges from 1-100. The scale includes written descriptions and examples to help determine the range of functioning.

VALUE LABEL: Any number from 1 to 100, with higher numbers indicating a higher level of functioning.

HOW MEASURED: Score determined by intake clinician

DESCRIPTIVE STATISTICS: mean= 21.388

SD= 7.544

range= 5 thru 45

"zero" scores= 0 % n=0

Table 37

Descriptive Statistics for Variables Used in the Analyses: Pathological Thought Content

---

PATTERN MATRIX VARIABLE NAME: P.Thcont

APPENDIX 1 VARIABLE LABEL: Mental status examination: Pathological thought content

MEANING: Those items in the "Thought Content" section of the mental status examination that tend toward pschopathology

VALUE LABEL: Number of items identified by clinical staff member who completed the mental status examination, with each item scored "1" or "0" in determining total (see Appendix 1 for further details).

HOW MEASURED: Actual count of items

DESCRIPTIVE STATISTICS: mean= 1.845

SD= 1.527

range= 0 thru 6

Appendix 3: Additional Statistical Tables

Table 38

Factor Analysis Rotated Factor Matrix

(Orthogonal Rotation with 22 Variables)

	(AGGRESS) FACTOR 1	(FAMILY) FACTOR 2	(THOUGHT) FACTOR 3
COMMIT	.74758	.02140	.25881
ASLTHRET	.70852	.06644	-.26826
ADMSTAT	-.64680	.27425	-.15463
ASSALTOT	.63922	.30922	-.08843
PRIORHOS	.55112	.09814	.14757
FOLLOWUP	.50470	.18696	.25395
QRHOURS	.48081	.08568	-.08083
PRTX.OP	-.44727	.33462	.09321
P.MANNER	.42745	-.23791	-.00551
SUICIDE	.40728	.39067	.01925
F.ETCRIM	.08569	.62729	-.08090
PA.YES	-.05880	.61820	-.15034
BIOPAR	.02921	-.60062	.09185
CUSTODY	-.25323	-.58840	.04839
FOSTCARE	.06683	.58678	-.08949
PRNTSUPP	-.03039	-.55575	-.24066
SA.YES	-.26918	.43445	.11637
P.THPROC	-.00900	.02615	.76363
P.SENSOR	.05412	-.03608	.75231
DELUSTOT	.02930	.00589	.67784
GAS	-.22651	.11953	-.58501
P.THCONT	-.10820	-.05262	.44472

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	Eigenvalue	Percent of Var
Factor 1	3.58714	16.3
Factor 2	2.95467	13.4
Factor 3	2.33663	10.6

Table 39

Ages of Subjects by Gender

AGE	MALE	FEMALE
9	1 (1.9%)	0
10	1 (1.9%)	0
11	1 (1.9%)	0
12	1 (1.9%)	0
13	1 (1.9%)	0
14	6 (11.5%)	2 (10.5%)
15	8 (15.4%)	2 (10.5%)
16	12 (23.1%)	7 (36.8%)
17	16 (30.8%)	7 (36.8%)
18	5 (9/6%)	1 (5.3%)

VITA

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