Multivariate Analysis of Schizophrenic Dimensions

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Abstract

A number of symptom, historical, and behavioral variables were correlated and factor analyzed with schizophrenics in a private psychiatric hospital. The highest correlation was between the premorbid variables of years of education and age at first hospitalization. These two variables, and negative history of neurological problems, and magnitude of psychosocial stressors, loaded most highly on Factor 1. Paranoid versus Nonparanoid type had the highest factor loading and formed the label of Factor 2. The findings were related to both the process versus reactive dimension of schizophrenia and the Templer and Cappelletty (1986) primary-secondary conceptualization. The former was given more support by the findings than the latter. An accumulative biological risk model was proposed.

Hundreds of published studies have focused upon symptom, historical, behavioral, biological, demographic, and typology characteristics of schizophrenic patients. However, these individual studies have focused upon no more than a few of these variables. Furthermore, apparently none of these studies have involved multivariate statistics. In the present research a rather large number of variables were intercorrelated and factor analyzed.

Although the present study was intended to be to a large extent exploratory rather than hypothesis testing, the variables were chosen both because of their use in the previous literature and because of their relevance to two models of conceptualizing schizophrenia. One was the process-reactive formulation extensively researched in the 1950s and 1960s. The other was the primary-secondary conceptualization.

Process schizophrenia is characterized by onset at an early age, an insidious onset, evidence of less adequate premorbid psycho-social and psychosexual functioning, and greater genetic predisposition. Reactive schizophrenia is associated with briefer psychotic episodes and a relatively satisfactory adjustment earlier in life. The reactive schizophrenic typically becomes ill at a later age, with evidence of considerable precipitating environmental stressors. Reactive schizophrenia also has a better prognosis than process schizophrenia (Phillips, 1953;Cashdan, 1972; Kleinmuntz, 1974; Lahey&Cimenero, 1980; Coleman, 1984). Although the concept of process and reactive schizophrenia was originally viewed as a dichotomous typology, it became recognized as a continuum. The primary-secondary formulation of Templer and Cappelletty (1986) is basically a contemporary and more biologically based version of the primary-secondary formulation. The primary schizophrenic has more endogenous and possibly more genetic etiology and the unfavorable symptom and course characteristics of the process schizophrenic. Secondary schizophrenia is like reactive schizophrenia which was said to result from interpersonal and intrapsychic stress. Secondary schizophrenia, reflecting the extensive research in the last two decades suggesting biological etiology, is regarded as resulting from impact or chemical harm to the brain.

The variables included in the present research were age of first hospitalization, education, neurological problems, psychosocial stressors, paranoid vs non-paranoid subtype, drug/alcohol problems, presence or absence of epilepsy, presence or absence of affective disorders, gender, and number of schizophrenic relatives.

Research points to schizophrenics with an affective component having a better prognosis than those schizophrenics who exhibit affective flatness (Stephens, Astrup & Mangrum, 1966; McCabe, Fowler Cadoret & Winokur, 1972). Research indicates that a family history of affective disorder (depression, mania, or schizoaffective illness) is associated with a better prognosis (Kant, 1942;

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Valliant, 1962, 1964; Stephens, Astrup & Mangrum, 1966; Fowler, Tsuang, Cadoret, Monelly & McCabe, 1972; Procci, 1976; Tsuang, Dempsey & Fauscher, 1976; Fowler, 1978). Targum (1983) found that schizophrenics with neuroendocrine test findings similar to those of endogenously depressed patients had a better prognosis.

Research has shown education to be highly correlated with overall premorbid social functioning (Zigler & Levine, 1981). One would predict that the schizophrenics who function better are the brightest and the more likely to persevere in their educational pursuits. The re-hospitalization rate is also lower for brighter than duller schizophrenics (Heffner, Strauss & Grissell, 1975). The authors of that study viewed their findings as consistent with the literature that reports chronic schizophrenics to be less bright than acute schizophrenics.]

Female schizophrenics tend to have a later age of onset and fewer features of process schizophrenia (Lewine, Strauss & Gift, 1981; Loranger, 1984; DeLisi, Dauphinais & Hauser, 1989). Research indicates that women have a significantly less severe course to their illness than men. When subgrouped by age, the incidence ratios for men versus women is 2:1 at 15 to 25 years, approximately equal at 25 to 35 years, and at least 1:2 at 40 years (DeLisi, Dauphinais & Hauser, 1989).

It has long been recognized that a disproportionate number of head injured persons develop schizophrenia, or at least a schizophrenic like condition, some time after head injury. Davison and Bagley (1969) reviewed six studies and concluded "With an expectation of developing schizophrenia in the general population of .8% over a 25 year risk period (age 15 to 40 years) the observed incidence of over 10 to 20 year periods is 2 to 3 times the expected incidence." Research subsequent to the Davison and Bagley review supports their inferences. Davison (1983) found that of 291 persons who had been unconscious at least a week and followed up from 10 to 24 years later, schizophreniform psychosis developed in 2.4% of the cases. It does not appear that the bulk of these persons were already schizophrenic or pre-schizophrenic. One basis for arguing against this possibility is that such patients do not have a disproportionate number of schizophrenic relatives (Schultz, 1932; Hilbon, 1951; Davison & Bagley, 1969). There is an impressive array of literature to convincingly document that schizophrenics have a disproportionate number of perinatal complications and irregularities. Jacobsen and Kinney (1980) reported that 63 Danish schizophrenics had significantly more perinatal complications and significantly more severe complications than 63 Danish control persons. Woerner, Pollack and Klein (1973) found that schizophrenics had a significantly greater number of prenatal and perinatal birth complications than their siblings. Mednick reported that 39% of schizophrenic patients but only 13% of control persons were born after prolonged labor. Jacobsen and Kinney (1980) reported a prolonged labor birth for 40% of schizophrenics and 13% of normal control persons. Parnas, Schulsinger, Teasdale, Schulsinger, Feldman and Mednick (1982) reported prolonged labor for 33%> of schizophrenics and 19% of control persons. A stronger genetic component is assumed in primary schizophrenia. First-degree relatives have ten times the probability of being or becoming schizophrenic than first-degree relatives of non-schizophrenic persons (Schultz, 1932; Kallman, 1938; Zerbin-Rudin, 1967, 1972; Slater, 1968; Rosenthal, 1970; Slater & Cowie, 1971; Gottsman & Shields, 1972). Identical twins of schizophrenics have about five times the risk of schizophrenia than fraternal twins (Kringlen, 1967; Gottsman & Shields, 1972; Shields & Gottsman, 1972; Fischer, 1973). Fraternal twins have the same risk as ordinary siblings (Zerbin-Rudin, 1967; Blueler, 1972; Gottsman & Shields, 1972; Shields and Gottsman, 1972). Identical twins of schizophrenics reared and not reared with the proband to not differ in probability of becoming schizophrenic (Heston, 1966; Rosenthal, Wender, Kety, Schulsinger, Weiner & Ostergaard, 1968; Kety, Rosenthal, Wender & Schulsinger, 1968; Rosenthal, 1972, 1975; Wedner, Rosenthal, Kety, Schulsinger & Weiner, 1974).

In epilepsy, the frequency of schizophreniclike psychoses is many times higher than the chance expectation (Davison & Bagley, 1969). Schizophrenics with epilepsy have been shown to have more characteristics which are indicative of secondary schizophrenia such as less disturbed affect and less personality deterioration;. These schizophrenics also have less incidence of schizophrenia in family members

(Alstrom, 1950; Mitsuda, 1950; Slater, Beard & Glithero, 1963). It was proposed that schizophrenics with a history of epilepsy are more likely to have secondary schizophrenic features than those schizophrenics with no history of epilepsy.

The presence of the abuse of alcohol and drugs prior to onset of schizophrenia is proposed to be associated with secondary schizophrenia. In a review of the drug-induced psychoses and their relationship to schizophrenia, Davison (1975) made the following observations: (1) Most observers agree that a family history of schizophrenia is usually absent in alcohol and drug abusers (Benedetti, 1952; Victor & Hope, 1958; Alpert & Silver, 1970). (2) Pre-schizophrenic personalities are uncommon in such individuals. The personality usually encountered is the unstable sociopathic type (Benedetti, 1952; Victor & Hope, 1958; Connell, 1958; Evans, 1959; Mendels, 1964; Smart & Bateman, 1967; Hatrick & Dewhurst, 1970; Griffiths, Cavaqnagh & Oates, 1970). (3) Idiopathic schizophrenia requires a genetic predisposition whereas drugs and certain brain lesions act beyond this point in the etiological chain and produce directly the brain dysfunction which results in the schizophrenic experience.

The importance of paranoid vs non-paranoid subtype diagnosis distinction has been recognized in many studies. Paranoid schizophrenics tend to be brighter and functioning on a higher psychosocial level in addition to having better premorbid adjustment, lesser genetic determination, and having a more favorable prognosis (Freedman & Kaplan, 1967; Kaplan & Sodock, 1985; Yap, 1951; Lambo, 1965; Torrey, 1980; Templer & Veleber, 1982).

Method

All subjects for the present research met the Diagnostic and Statistical Manual of Mental Disorders (American Psychiatric Association, 1968, 1980, 1987) schizophrenia criteria or the schizophrenia criteria of an expanded version of the Present State Examination (Wing, Cooper, & Sartorius, 1974). These subjects were drawn from a private psychiatric hospital in California. The 323 patients, 170 males and 153 females, were composed of persons who had been inpatients at some time since 1980. Their mean age of first hospitalization was 20.69 with a standard deviation of 7.62. They ranged in formal education from 6 to 20 years, with a mean of 11.64 and a standard deviation of 2.44.

All information was obtained from the medical records at the private psychiatric hospital. There were a total of 66 neurological problems. Fourteen of these were head injuries. Thirty-two were perinatal and prenatal birth complications consisting of 5 forceps deliveries, 25 difficult births, and 2 premature births. The other kinds of neurological problems included one case of peripheral neuropathy, 2 cases of hyperactivity, 1 cerebral palsy, 2 minimal brain dysfunction, and 14 cases of coordination problems. The psychosocial stress assessment closely parallels DSM-III-R (1987) AXIS IV.

Results

Table 1 (see tables pp. 102, 103) displays the means and standard deviations for all of the relevant continuous variables. Table 2 displays the frequencies for the binary variables. Table 3 contains the product-moment correlational matrix for all of these continuous and binary variables. The highest correlation was .45 between education and age of first hospitalization. Age of first hospitalization was the variable that yielded the most significant and the highest correlations.

A principal components analysis was conducted on the 10 variables. A scree test (Cattell, 1966) suggested that only two of the factors were important enough to rotate for further anlaysis. Rotation of these two factors was accomplished with the varimax rotation procedure. These two factors accounted for a total of 20.50 percent of the variance. The factor loadings are contained in Table 4. Factor 1, having an Eigenvalue of 1.24 and accounting for 12.4 percent of the total variance, was called "process versus reactive". All 10 of the factor loadings could be viewed as in the direction of lesser process schizophrenia. It is here noted that most of the factor loadings are in the predicted direction for greater secondary as opposed to primary schizophrenia, but the neurological problem variable which is perhaps the most important variable in the Templer and Cappelletty (1986) conceptualization, loaded in the opposite direction. It is apparent that persons high on Factor 1 have a

history of more assets and fewer liabilities. If one wished to conjecture on the basis of the factor loadings for higher education and negative neurological history, Factor 1 might be thought of as tapping "neuropsychological integrity".

Factor 2 had an Eigenvalue of 0.81 and accounted for 8.1 percent of the total variance. The factor loadings in Table 7 show that schizophrenics high on Factor 2 are more likely to be female, to have a non-paranoid diagnosis, and to have an affective component. This factor could have been tentatively labeled "internal versus external overwhelm-ingness", but was given the more descriptive and more conservative name of "paranoid versus non-paranoid".

Multiple regression was carried out with age of first hospitalization as the dependent variable. This was to explore a model here proposed for integrating the biological variables. Table 5 contains the multiple regression summary.

Discussion

The findings point to the enduring merit of the process-reactive dimension of schizophrenia intensively researched in the 1950s and 1960s. Education and age of first hospitalization were regarded as important process-reactive variables at that time, and these variables had the highest loadings on Factor 1 in the present study.

The present findings, however, go beyond confirming the vitality of the process-reactiveconceptualization. They integrate the clinical history and course variables with the rapidly mounting evidence over the last two decades that schizophrenia is a brain disease. Table 4 shows that all four of the biological risk variables-number of schizophrenic relatives, neurological problems, epilepsy, and alcohol/drug abuse, have negative factor loadings. All of the other variables, those that previous research has shown to be associated with schizophrenia of lesser severity, have positive factor loadings.

The negative loading of neurological problems on Factor 1 was the opposite of what was tentatively expected on the basis of the Templer and Cappelletty (1986) conceptualization. However, in retrospect, some of the previous literature could be viewed as consistent with the obtained findings. Pollack and Greenberg (1966) reported that schizophrenics with obstetrical complications had a significantly younger age of first hospitalization and age of first treatment than schizophrenics without such complications. Pollack, Leven-stein and Klein (1968) found that schizophrenics who had a minimal brain damage childhood history had an apparently less favorable outcome at three year post-hospitalization follow-up.

It is apparent that the present research gives less support to the primary-secondary conceptualization than to the process-reactive conceptualization if both are viewed as having both etiological and clinical variables. However, Templer and Cappelletty (1986) maintained that their continuum is not one in which a greater role of primary or secondary characteristics mean less of the other. They went on to say that "Whether or not a person becomes schizophrenic is positively associated with *both* primary and secondary variables" (p. 258). Templer and Cappelletty were seemingly unclear, insofar as in some places such as the above quotation they implied it is a continuum of etiological variables, but in other places implied it may be a continuum of both etiological and clinical (including premorbid and course) variables. The present research findings suggest that if the Templer and Cappelletty notion of a continuum is viable it applies primarily to the etiological primary versus secondary (intrinsic versus extrinsic) variables, and/or to a temporal etiological continuum ranging from embryological development to mid-life.

Perhaps a more viable model at this time would be one in which biological etiological elements combine in an additive or multiplicative fashion. It must, of course, be acknowledged that numerous authors have suggested some sort of interaction of causal factors. These previous authors, however, have not subjected their contentions to multivariate analysis. In fact, typically, only two variables have been researched or discussed by these previous authors. It is here proposed that an *accumulative* model be considered in which a combination of some etiological variables are related to probability of becoming schizophrenic and/or to severity of the disorder in persons who are schizophrenic. To explore the second of these possibilities, an additional analysis was undertaken. Specifically, multiple regression was carried out with the chief

process variable of age at first hospitalization as the dependent variable. The independent variables were the four biological risk variables of neurological problems, epilepsy, positive drug/alcohol history and number of schizophrenic relatives.

Table 5 consists of the multiple regression summary table of biological risk variables with age of first hospitalization. The fact that the neurological problem variable provides an increment of prediction beyond that of alcohol and drugs should be viewed as providing some modest support for the above proposed accumulative risk model. Nevertheless, it should be borne in mind that the multiple regression was a post hoc analysis. On the other hand, it was an analysis decided upon with the full knowledge that the simple correlations of both epilepsy and number of schizophrenic relatives with age of hospitalization were of zero order. Also, only 8 (2.5%) of the 323 patients had epilepsy.

One advantage of such an accumulative model is that it is capable of incorporating the diversity of brain anomalies and other biological correlates that are found in a disproportionate number of, but not all schizophrenics. These include larger ventricle size, abnormal or atypical EEG, seasonality of births, prenatal and perinatal birth complications, minor physical anomalies, genetic disposition, epilepsy, head injury, corpus callosum thickness, sensory gaiting deficits, less cerebral blood flow, atypical distribution of blood flow, atypical glucose metabolism, limbic cellular disorganization, and increased dopamine receptors in the basal ganglia. If there are a large number of diverse biological etiological elements in schizophrenia, this could explain why many schizophrenics are normal on some variables in which a disproportionate number of schizophrenics are abnormal.

A negative relationship between neurological problems and schizophrenic relatives was predicted in view of the research such as that of Slater and Cowie (1971), who found more neurological problems in those schizophrenics who did not have schizophrenic relatives. And, previous research and reviews of this research indicate a low rate of schizophrenia in the relatives of persons who become schizophrenic after head injury (Davison & Bagley, 1969; Hillbom, 1951; Schulz, 1932). It would indeed seem that schizophrenics without schizophrenic relatives would need more neurological risk factors to push them to some sort of schizophrenia threshold. Nevertheless, the correlation was close to zero. Perhaps the present groups of schizophrenics is atypical. Also, the accuracy of information about the mental health of patients relatives should usually be regarded as questionable.

The rather high Factor 2 loading of paranoid subtype diagnosis is consistent with previous investigations that indicate the paranoid versus not paranoid schizophrenia diagnosis is a consistent and meaningful distinction with both clinical and research implications. It should be borne in mind, however, that the Eigenvalue for Factor 2 is lower than 1. Because of this and because its nature is unclear, the Factor 2 findings should be viewed as more hypothesis generating than permitting confident inferences.

In summary, the overall purpose of this investigation was successful in demonstrating the feasibility of a dimensional multivariate approach to the understanding of the individual differences of schizophrenia. A meaningful factor structure was found. Future multivariate research is suggested, especially in regard to the proposed accumulative biological risk model.

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Table 1
Means and Standard Deviations for Relevant Continuous Variables

Variable	Mean	SD
Composite Stressor	21.28	6.15
Neurological Problems	.20	.48
Years Education	11.64	2.44
Age First Hospitalized	20.69	7.62
# Schizophrenic Relatives	.28	.64

Table 2
Frequencies of Relevant Binary Variables

Variable	No	(%)	Yes
Affective Disorder	234	(72.4%)	89
Epilepsy	315	(97.5%)	8
Alcohol/Drug Abuse	198	(61.3%)	125
Gender (Female = Yes)	170	(52.6%)	153
Paranoid Diagnosis	232	(71.8%)	91

Table 3
Variable Intercorrelations

Vble	1	2	3	4	5	6	7	8	9
1. AF									
2. CS	.05								
3.NEU	-.08	-.07							
4. EP	.05	-.17	.14"						
5. A/D	-.09[a]	-.15	.09	-.09					
6. ED	.12[a]	.01	-.22[c]	.09	.00				
7. HO	.12[a]	.26"	-.17[c]	-.08	-.21°	.45°			
8. GE	.22[c]	.00	-.09[a]	.01	-.14"	.07	.12[a]		
9. PN	-.23[c]	.11	-.1l[a]	-.01	.12[a]	.05	.12[a]	-.25°	
10. RL	.13[b]	-.12	.02	-.01	.03	-.02	-.01	.05	-.04

a = p<.05 b = p<.01 c = p<.001

AF = Affective Diagnosis
CS = Composite Stressor
NEU = Neurological Problems
EP = Epilepsy
A/D = Alcohol & Drugs
ED = Years of Education
HO = Age First Hospitalized
GE = Gender
PN = Paranoid Diagnosis
RL = Biological Relatives with Schizophrenia

Table 4
Private Hospital Variable Factor Loadings

Variable	Factor 1	Factor 2
Age First Hospitalization	.80	.09
Education	.49	.07
Neurological Problems (#)	-.31	.03
Psychosocial Stressors (composite)	.30	.04
Paranoid (1 = No, 2 = Yes)	.21	-.58
Drug/Alcohol Problems (1 = no, 2 = Yes)	-.18	-.22
Epilepsy (1 = No, 2 = Yes)	-.17	.01
Affective Disorder (1 = No, 2 = Yes)	.14	.44
Gender (1 = M, 2 = F)	.10	.47
Schizophrenic Relatives (#)	-.05	.13
Eigenvalue	1.24	.81
Percentage of Variance	12.40	8.10

Table 5
Stepwise Multiple Regression of Biological Risk Variables with Age of First Hospitalization

Predictor Variable	r	R	R^2	Beta	F
Alcohol and Drugs	-.21	.21	.05	-.21	15.19*
Neurological Problems	-.17	.26	.07	-.15	11.41*

* $p < .001$