

Confirmation of a Two-Factor Model of Premorbid Adjustment in Males with Schizophrenia

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Abstract

Because schizophrenia is considered to be a neurodevelopmental disorder, premorbid adjustment is of particular interest. Premorbid adjustment is probably not a unitary construct but rather is expressed across a number of developmental domains. The current investigation examined the validity of a two-factor model that differentiated premorbid adjustment across social and academic domains and evaluated relationships between these premorbid adjustment domains and other variables of interest. Participants with schizophrenia ($n = 141$) underwent evaluation of premorbid adjustment (using the Premorbid Adjustment Scale), intellectual functioning, and psychiatric symptoms. Using confirmatory factor analysis, a two-factor model of premorbid adjustment was identified that included an academic domain and a social domain. The social domain was associated with symptom variables, while the academic domain was associated with measures of intelligence. Results provide evidence for at least two domains of premorbid adjustment in schizophrenia. Distinguishing between these two premorbid domains may be theoretically important because of potential differences in incidence rates and deterioration courses; some individuals with schizophrenia may exhibit adequate academic adjustment but poor social adjustment, while others may exhibit the opposite pattern.

Keywords: Schizophrenia, premorbid, symptoms, intelligence, factor analysis

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Premorbid psychosocial adjustment has received considerable attention in schizophrenia research because of the proposed neurodevelopmental nature of the disorder (Weinberger 1995). Poor premorbid adjustment is related to numerous clinical variables of interest, including age of onset (Gittelman-Klein and Klein 1969), negative symptoms (Kelley et al. 1992), academic attainment (Erel et al.

1991), social skills (Mueser et al. 1990), neurobiological measures (van Kammen et al. 1994), and premorbid IQ (Aylward et al. 1984; Bilder et al. 1992; Goldberg et al. 1993). However, premorbid adjustment is probably not a unitary construct. In schizophrenia, two premorbid domains that have been examined are premorbid intellectual functioning and premorbid psychosocial functioning. Estimates of premorbid intellectual functioning are often based on current performance on subtests of intelligence tests (Bilder et al. 1988), such as the Wechsler Adult Intelligence Scale–Revised (WAIS–R, Wechsler 1981); on achievement test performance (Goldberg et al. 1993; Kremen et al. 1996); or on regression analyses of demographic variables (Vanderploeg et al. 1996). Premorbid psychosocial functioning is typically determined using scales based on information derived from records and from patient and family member reports (Cannon-Spoor et al. 1982).

Of the scales currently available to assess premorbid adjustment in schizophrenia, the Premorbid Adjustment Scale (PAS, Cannon-Spoor et al. 1982) is one of the most widely used. The PAS yields age-level scores reflecting childhood, early adolescence, late adolescence, and adulthood premorbid functioning, as well as a number of psychosocial domain scores that are rated at each age level (e.g., Social Functioning, Peer Relationships, School Performance, School Adaptation). Mukherjee et al. (1991) suggested that the PAS psychosocial domain scores actually represent two underlying domains of premorbid adjustment, an academic domain and a social domain. More recently, two groups of investigators have identified academic and social PAS psychosocial domains using exploratory factor analysis (van Kammen et al. 1994; Cannon et al. 1997). In 58 patients with schizophrenia, van Kammen et al. (1994) found that the academic factor was negatively correlated with cerebrospinal fluid (CSF)

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dopamine β -hydroxylase levels ($r = -0.31$, $df = 56$, $p = 0.02$), whereas the social factor was positively correlated with dopamine β -hydroxylase ($r = 0.24$, $df = 56$, $p = 0.08$). Decreased levels of dopamine β -hydroxylase are consistent with either perinatal traumatic lesions to the brain (van Kammen et al. 1983) or genetic influences (Wei et al. 1997; Cubells et al. 1998). Cannon et al. (1997) also found the academic and social PAS factors in a heterogeneous sample composed of comparison subjects ($n = 100$) and individuals with schizophrenia ($n = 28$) or bipolar disorder ($n = 70$). On both factors, patients with schizophrenia had significantly worse premorbid adjustment than controls.

Differences in relationships between dopamine β -hydroxylase and the PAS academic and social factors (van Kammen et al. 1994), as well as the ability of the two PAS factors to distinguish between normal controls and patients with schizophrenia (Cannon et al. 1997), provide some theoretical support for at least two domains of premorbid adjustment in schizophrenia. However, at present, the distinction between these two premorbid adjustment domains has not been directly tested. Also, the manner in which these domains relate to other clinical variables of interest in schizophrenia is not clear. In the current investigation, we directly tested the validity of a theoretical premorbid adjustment model consisting of social and academic premorbid adjustment domains using confirmatory factor analysis (CFA) and by examining relationships between the two domains and other clinical variables of interest. We hypothesized that the theoretical model consisting of two premorbid adjustment domains would provide the best explanation of the data and that the two premorbid adjustment domains would exhibit a differential pattern of associations to clinical variables.

Methods

Subjects. The sample consisted of 141 male veterans who were admitted to our schizophrenia research unit. They had *DSM-III-R* diagnoses (American Psychiatric Association 1987) of schizophrenia or schizoaffective disorder. Diagnoses were established using the Structured Clinical Interview for *DSM-III-R* (Spitzer et al. 1989) or the Schedule for Affective Disorders and Schizophrenia–Lifetime Version (Spitzer and Endicott 1979). Diagnostic information was reviewed in a multidisciplinary case conference, and a consensus diagnosis was also established. Patients had no other psychiatric diagnoses, were physically healthy (i.e., required no medications for medical disorders other than schizophrenia), and did not have any coexisting neurological disorders. None of the patients had abused substances for at least 4 months prior to testing. Subjects had an average age of

36.2 years (standard deviation [SD] = 7.8 years) and an average of 12.2 years of education (SD = 1.8 years). Average age of disease onset was 23.5 years (SD = 5.5 years). Average WAIS–R Full Scale IQ (FSIQ, Wechsler 1981) was 89.9 (SD = 9.6). The sample included 122 whites, 17 African-Americans, and 2 Hispanics. No significant differences ($p > 0.05$) were present between racial groups on the demographic, premorbid, or clinical variables. Haloperidol was the antipsychotic treatment for most patients ($n = 117$; mean dosage = 11.9 mg; SD = 7.1 mg). Of those patients taking haloperidol, 108 were taking either haloperidol only ($n = 59$) or a combination of haloperidol and benztropine ($n = 49$) with no other psychotropic medications. Twelve patients were medication-free when evaluated, and the remaining 21 were taking combinations of antipsychotic and other psychotropic medications. Written informed consent was obtained from each subject prior to participation in the study.

Measures. Variables assessed included premorbid psychosocial functioning, age at disease onset, current intellectual functioning, premorbid intellectual functioning, and current psychiatric symptoms. All data were obtained while subjects were inpatients on our schizophrenia research unit and participating in a clinical research protocol examining relapse prediction and treatment response to haloperidol. Premorbid functioning was assessed with the PAS (Cannon-Spoor et al. 1982). The PAS was completed with extensive information obtained from interviews with the patients, from family members, and from previous records. The PAS was completed by staff members on the schizophrenia research unit (psychologist or social worker) who were trained to reliably complete the subscale ratings.

On the PAS, premorbid adjustment is rated by age level and according to five psychosocial domains: Sociability, Peer Relationships, School Performance, School Adaptation, and Social-Sexual Functioning. Each domain is rated on a 7-point scale (0–6; higher scores indicate poorer adjustment). Because the current investigation was concerned with evaluating different domains of premorbid functioning (e.g., academic and social), the five psychosocial domain scores were calculated separately by summing the scores for each psychosocial domain across age levels and then dividing by the total number of scores. For example, the School Adaptation domain score represented the sum of the childhood, early adolescence, and late adolescence School Adaptation scores, divided by three. For five patients, data were not available for the late adolescent period because onset of schizophrenia occurred before they reached this age level. These five patients' psychosocial domain scores are the averages of the childhood and early adolescence periods only. PAS adulthood scores (age 19 and above) were not

included because most patients were already exhibiting significant features of the disorder by adulthood.

Age at disease onset was specified as the first time period during which subjects exhibited symptoms of schizophrenia for a minimum of 6 months. This 6-month time period was established through treatment team consensus following extensive interviews with the subjects, family member reports, and review of all available records. The WAIS-R was used to assess intellectual functioning, and the FSIQ score served as the index of current intellectual functioning. We also examined two estimates of premorbid cognitive functioning. The first was a WAIS-R index proposed by Bilder and coworkers (1988, 1992) that consisted of the mean of the WAIS-R Information and Vocabulary subtest scores. These WAIS-R subtests are considered "hold tests" because they are not strongly influenced by cognitive deterioration resulting from acquired brain damage. The second estimate of premorbid cognitive function was years of formal education. The total score from the Brief Psychiatric Rating Scale (BPRS, Overall and Gorham 1962) was used as the measure of current symptom severity. BPRS ratings were based on data derived from a standardized interview with patients, observations of ward behavior, and patient-staff interactions. The BPRS and WAIS-R were completed on average within 3 days of each other (mean = 2.6 days; SD = 2.2; $n = 137$). Interrater reliability was maintained through reliability rating meetings. All measures were completed according to standardized procedures by trained staff members.

Data Analysis. Confirmatory factor analysis (CFA) (Jöreskog and Sörbom 1993) was used to examine whether academic premorbid adjustment and social premorbid adjustment were distinct domains on the PAS, or whether they represented a more global unitary premorbid adjustment domain. To accomplish this, we evaluated three models that made a priori specifications regarding the relationships among the five PAS subdomain scores. Models included a null model, a one-factor model, and a two-factor model. The null model tested the hypothesis that the five PAS premorbid domain scores were not related. The one-factor model examined the hypothesis that premorbid functioning was expressed as a unitary domain; all five PAS domain scores were specified to load on a single factor. The two-factor model dichotomized premorbid functioning according to an academic domain and a social domain. The academic domain was composed of the School Performance and School Adaptation subdomains, while the social domain was composed of the Sociability, Peer Relationships, and Social-Sexual Functioning subdomains. Based on previous work (van Kammen et al. 1994; Cannon et al. 1997), we hypothesized that the theoretical model distinguishing between

academic and social domains was the superior model. Relative adequacy of hypothesized models was evaluated using a number of standard statistics, the most common of which include the chi-square statistic, the goodness of fit index (GFI), and the adjusted goodness of fit index (AGFI). The chi-square statistic was used to evaluate the fit between the hypothesized statistical models and the unrestricted actual data set. The GFI and AGFI have ranges of 0.00 to 1.00 and provided estimates of the amount of variance and covariance explained by the models. For these statistics, good fit is indicated by a non-significant chi-square statistic and a GFI and AGFI that approximate 1.00.

After the best theoretical model was determined using CFA, Pearson correlations were used to evaluate relationships between the premorbid domains and clinical variables of interest (age at disease onset, symptom severity, and current and premorbid intellectual functioning). To control for the number of correlations, an alpha level of 0.01 was used to determine the significance of correlations. Also, partial correlations were used to evaluate any systematic effects that disease chronicity (defined as years since disease onset) had on the relationships between the PAS scores and the clinical variables.

Results

As hypothesized, initial examination of the CFA results indicated the presence of academic and social premorbid domains. The social premorbid domain was composed of the PAS Sociability, Peer Relationships, and Social-Sexual Functioning scores, while the academic premorbid domain consisted of the PAS School Performance and School Adaptation scores. However, a factor loading of 1.09 was present for the School Adaptation subdomain on the academic factor, and it also had a negative error variance ($\theta_{\delta} = -0.19$). Because these values exceeded theoretical limits, the initial two-factor model was rejected as the best theoretical model. Based on prior investigations of the PAS factor structure (van Kammen et al. 1994; Cannon et al. 1997) and the CFA modification indexes for the current two-factor model, the two-factor model was respecified so that School Adaptation loaded on both the academic and social PAS factors (two-factor model with doublet specified). Theoretically, the loading of the School Adaptation domain score across both academic and social domains is reasonable because this domain evaluates behaviors associated with both academic and social functioning (e.g., getting along in school, discipline problems, participation in extracurricular activities, friends at school). Following respecification of the two-factor model, all values were within theoretical limits.

Table 1 contains fit index results of the CFAs for the null model (M_n), the one-factor model (M_1), and the two-factor model with School Adaptation specified to load on both factors (M_{2D}). The chi-square statistic and the GFI statistics indicated that the two-factor model was the superior model. The normed fit indexes, which provide estimates of incremental improvement in model fit compared to the null model, again supported the two-factor model as the optimal solution. Table 2 presents the maximum likelihood two-factor solution. Each of the five psychosocial domains had excellent loadings on their respective factors with the exception of the School Adaptation domain, which loaded on both factors. The correlation between the two factors was 0.31.

Correlations between validation variables and the PAS academic factor, social factor, and total average scores are contained in table 3. Inspection of box plots and distribution statistics indicated that all PAS and clinical variables were normally distributed. Means and SDs for the PAS academic, social, and total average scores were 2.53 (SD = 1.33), 2.21 (SD = 1.21), and 2.32 (SD = 1.04), respectively. The PAS social factor was significantly correlated with variables reflecting symptomatology, including age at disease onset and BPRS total score, while the academic factor was significantly correlated with variables reflecting intellectual functioning, including the WAIS-R FSIQ, premorbid IQ, and years of formal education. The total

Table 1. CFA results for premorbid functioning models

Model	χ^2	df	χ^2/df	GFI	AGFI	RMSR	NFI
Null model (M_n)	421.16*	10	42.12	0.47	0.20	0.44	0.00
One-factor model (M_1)	104.26*	5	20.85	0.82	0.47	0.15	0.75
Two-factor model (M_{2D}) ¹	3.04	3	1.01	0.99	0.96	0.01	0.99

Notes.—AGFI = adjusted goodness of fit index; CFA = confirmatory factor analysis; GFI = goodness of fit index; NFI = normed fit index; RMSR = root mean square residual.

¹ Two-factor model with School Adaptation specified to load on both factors.

* $p < 0.001$

Table 2. PAS two-factor maximum likelihood solution¹

Subdomain	Social	Academic
Social functioning	0.94	—
Peer relationships	0.90	—
School performance	—	0.99
School adaptation	0.28	0.67
Social-Sexual functioning	0.71	—

Notes.—PAS = Premorbid Adjustment Scale.

¹ For male patients with schizophrenia.

Table 3. Relationship of clinical variables to PAS social factor, academic factor, and total average scores

Variables	Social Factor		Academic Factor		Total Average	
	r	r ¹	r	r ¹	r	r ¹
Age at onset ($n = 135$)	-0.29**	-0.30**	-0.10	0.17	-0.25*	-0.29**
BPRS total score ($n = 120$)	0.23*	0.23*	0.15	0.16	0.22	0.23*
Education ² ($n = 140$)	-0.16	-0.17	-0.55**	-0.56**	-0.39**	-0.39**
Premorbid IQ ³ ($n = 137$)	0.01	0.01	-0.31**	-0.29**	-0.16	-0.14
WAIS-R FSIQ ($n = 141$)	0.05	0.05	-0.28**	-0.29**	-0.12	-0.12

Notes.—BPRS = Brief Psychiatric Rating Scale; PAS = Premorbid Adjustment Scale; WAIS-R FSIQ = Wechsler Adult Intelligence Scale-Revised, Full Scale IQ.

¹ Partial correlation controlling for chronicity of schizophrenia (years since disease onset).

² Years of formal education completed.

³ Index of premorbid intellectual functioning from Bilder et al. 1988.

* $p = 0.01$; ** $p = 0.001$

average score was significantly correlated with age at disease onset and years of formal education. Because the current sample was evaluated approximately 12.8 years after disease onset, we also examined potential systematic measurement bias on the PAS scores resulting from disease chronicity, using partial correlations. These partial correlations are also presented in table 3. With the exception of a significant partial correlation between the PAS total score and the BPRS total score, no significant changes were noted after controlling for disease chronicity.

Discussion

Two domains of premorbid adjustment were identified in the current study, including academic and social premorbid domains. These results have both methodological and theoretical implications. From a methodological standpoint, results provide support for use of two PAS domain scores rather than five separate subdomain scores or a total average score, at least in male patients. This suggestion is based on a number of considerations. Using the social and academic factor scores allowed relationships between premorbid adjustment and clinical variables to be detected that were not evident when the total average score was used, particularly when examining premorbid and current IQ. Similarly, when the total average score was correlated with one of the clinical variables of interest, either the academic or social factor was also correlated with that variable, so that no relevant information was lost by using the PAS factors. In addition, because the two domain scores represent a broader sampling of behaviors in the academic and social domains compared to the five individual scores, they probably provide more stable and reliable measures of these areas. Finally, use of two rather than five domain scores reduces the number of variables analyzed, thereby decreasing error associated with making multiple comparisons. In these ways, use of academic and social domain scores may increase the utility of the PAS in future studies.

The results also provide theoretical support for at least two general domains of premorbid adjustment. They extend findings of prior investigations that used exploratory factor analysis (van Kammen et al. 1994; Cannon et al. 1997) by confirming the presence of two premorbid adjustment domains that are relatively distinct. The correlation of 0.31 between the academic and social premorbid adjustment domains indicates that they share relatively little common variance. Additionally, the relative distinctiveness of the two premorbid domains is demonstrated by the consistent differential pattern of correlations present between clinical variables and the PAS academic and social factor scores.

These correlations also provide information regarding the underlying constructs measured by the factors. For the

academic factor, the magnitude of the correlations indicate that it is more strongly associated with years of formal education than it is with premorbid IQ or current IQ. This is probably because years of formal education reflects a combination of intellectual ability and adaptation to the academic environment (e.g., participation in extracurricular activities, discipline problems), while the WAIS-R premorbid IQ index is derived from measures of verbal abilities that do not directly evaluate academic adaptation. Cannon et al. (1997) found significant differences between patients with bipolar disorder and those with schizophrenia on the PAS academic factor, whereas Goldberg et al. (1993) did not find significant differences between bipolar disorder and schizophrenia using a premorbid IQ index based on verbal abilities, the Wide Range Achievement Test-Revised reading test (Jastak and Wilkinson 1984). Van Kammen et al. (1994) reported that low CSF dopamine β -hydroxylase activity was associated with poorer premorbid academic adjustment and fewer years of education obtained, findings consistent with studies reporting lower dopamine β -hydroxylase activity in alcoholic patients who exhibit poor learning (Kulcsar et al. 1986) and in demented patients with schizophrenia (Markianos and Tripodanakis 1985). The current results and the findings of these other studies (Goldberg et al. 1993; van Kammen et al. 1994; Cannon et al. 1995) suggest that the PAS academic factor is associated with premorbid IQ indexes that are based on tests of verbal abilities and with one biochemical index of cognitive impairment, but that the academic factor also accounts for unique variance associated with school adaptation and so is probably not simply another premorbid IQ estimate or general index of cognitive impairment.

The correlations also suggest that the PAS social factor evaluates a construct associated with premorbid symptoms (age at disease onset) and current symptom expression. Prospective longitudinal studies of children at high risk for schizophrenia indicate a relationship between increasing symptoms and poorer adjustment prior to the onset of schizophrenia (Dworkin et al. 1991), along with a stability in positive and negative symptom profile from late childhood to adulthood in some patients with schizophrenia (Cannon and Mednick 1993). The relationship noted here between the PAS social factor and current symptom expression may then simply reflect that symptoms present during the prodromal phase of the illness are expressed in a more severe form with the onset of the disorder, and that overall severity of these symptoms remains relatively stable following disease onset. The current findings of decreased symptoms and later age of onset associated with better premorbid social adjustment are also consistent with the reported association between better premorbid psychosocial adjustment and low dopamine β -hydroxylase activity (van Kammen et al. 1994). Low

dopamine β -hydroxylase activity that is under genetic control (Wei et al. 1997; Cubells et al. 1998) may have a protective effect on those individuals at risk for schizophrenia and be indicative of a less severe familial variant of the disorder (for review, see van Kammen et al. 1994). However, the association between current symptoms and premorbid social adjustment should be interpreted tentatively because symptoms fluctuate over time and we assessed symptoms at only one point in time.

A number of factors may limit generalizability of the results. Findings were constrained by the range of functioning assessed by the PAS. Additional domains of premorbid function may be present that were not evaluated by the PAS. Walker et al. (1996) recently reported that abnormalities in neuromotor function and negative affect during childhood are associated with enlarged ventricles in adulthood. Presence of premorbid behaviors analogous to formal positive and negative symptoms have also been reported in children who later develop schizophrenia (Cannon and Mednick 1993). Further research is necessary to determine if premorbid affective, neuromotor, and symptom manifestations should be conceptualized as separate premorbid domains or as subdomains of the academic and social domains. Also, the current sample had a chronic disease course and was evaluated an average of 12.8 years after disease onset. Partial correlations used to examine potential measurement bias introduced by disease chronicity did not support the presence of any such bias on the PAS. In addition, the significant correlation between premorbid adjustment and age at disease onset found in the current study has also been reported in first-break and more chronic samples (Gittelman-Klein and Klein 1969; Haas and Sweeney 1992; Gupta et al. 1995). The results of the partial correlations and the consistency of current results with those of first-break studies support the utility of the PAS when used to retrospectively evaluate premorbid adjustment, even in chronic samples. Finally, compared to females, males have earlier age of disease onset, exhibit worse overall premorbid adjustment, and exhibit more severe and rapid deterioration prior to onset of the disorder (Gittelman-Klein and Klein 1969; Childers and Harding 1990; Larsen et al. 1996). These differences have been found for some but not all of the PAS subscales (Childers and Harding 1990), making examination of the PAS factor structure in a female sample necessary. Despite these sex differences, the two-factor model may have relevance for females with schizophrenia. This is because differences between groups in severity of impairment on a given scale does not preclude similarity in the underlying constructs evaluated by that scale. In fact, Cannon et al. (1997) found the same two PAS factors as reported here, but in a mixed group of male and female patients, suggesting that the PAS aca-

demic and social domains may be present for both males and females.

Thus, the current results provide considerable support for a theoretical model consisting of at least two premorbid adjustment domains in schizophrenia. Distinguishing between these domains may be of some theoretical importance because of potential differences in incidence rates of impairment and deterioration courses. Some individuals with schizophrenia may exhibit adequate academic adjustment but poor social adjustment, while others may exhibit the opposite pattern. Given its relationship to symptoms, the social domain may be sensitive to expression of the schizophrenia prodrome that is characterized by persisting behavioral disturbances or general psychopathology. In contrast, the academic domain score may be more sensitive to the cognitive deficits associated with the schizophrenia prodrome. Individual examination of the academic and social premorbid domains in future investigations may help clarify potential developmental behavioral markers for schizophrenia risk and provide more sensitive indexes of later disease severity and course.

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