



HHS Public Access

Author manuscript

Schizophr Res. Author manuscript; available in PMC 2016 December 01.

Published in final edited form as:

Schizophr Res. 2015 December ; 169(1-3): 209–213. doi:10.1016/j.schres.2015.11.004.

Patterns of premorbid functioning in individuals at clinical high risk of psychosis

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Abstract

In schizophrenia, four typical patterns of premorbid functioning have been observed: stable-good, stable-intermediate, poor-deteriorating and deteriorating. However, it is unknown whether similar patterns exist in those who are at clinical high risk (CHR) of psychosis. The aim of this study was to examine patterns of premorbid functioning in a large sample of individuals at CHR of psychosis and its association with symptoms, functioning, and conversion to psychosis. One-hundred sixty people at CHR of psychosis were assessed on premorbid functioning using the Premorbid Adjustment Scale. Poorer premorbid functioning was significantly correlated with worse negative symptom severity and lower social functioning. Cluster analysis was used to identify patterns of premorbid functioning. Results indicated three patterns of premorbid functioning in our CHR sample: stable-intermediate, stable-good, and deteriorating. The deteriorating group had more severe disorganization, worse negative symptoms and poorer social functioning than the other groups. Participants who made the conversion to psychosis had significantly poorer premorbid functioning during adolescence compared to those who did not convert. These results suggest that those at a clinical high risk for psychosis display similar patterns in premorbid functioning as have been observed in those with a psychotic illness and that poor premorbid functioning may be a predictor of psychosis.

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Conflict of Interest

All authors declare no conflict of interest.

Contributors

The first author wrote the first version of the manuscript and performed data analysis. The second assisted with writing the manuscript. The third author performed data analysis. The fourth author assisted in data organization and data analysis. The fifth author assisted in data organization and data analysis. The sixth author assisted in data collection, data analysis, provided laboratory space and funding, and assisted in the writing of the final version of the manuscript.

Keywords

clinical high risk; conversion; premorbid functioning; schizophrenia; social functioning; symptoms

1. Introduction

It has been well-documented in many studies over the past fifty years that poor premorbid functioning in people with schizophrenia is associated with an earlier age at psychosis onset, increased negative symptom severity, neurocognitive deficits, and poor clinical outcome and psychosocial functioning (Silverstein et al., 2003; Addington and Addington, 2005; Haim et al., 2006). Furthermore, males with schizophrenia tend to show poorer premorbid functioning relative to female patients (Bailer et al., 1996). In studies of individuals experiencing a first episode of psychosis, premorbid functioning has been examined by using cluster analysis (Addington et al., 2003). This method identified specific courses of premorbid functioning across four developmental stages, namely childhood, early and late adolescence and adulthood. Typical patterns were stable-good, stable-intermediate, poor-deteriorating and deteriorating. Those with a deteriorating pattern were significantly younger at onset of psychosis, had increased levels of positive and negative symptoms and poorer quality of life than the two stable groups (Addington and Addington, 2005). This suggests that in schizophrenia different patterns of premorbid functioning developmentally may have different outcomes.

Premorbid functioning has also been examined in those at clinical high risk (CHR) of psychosis, that is, people who experience attenuated positive symptoms, brief intermittent psychotic symptoms, or have a genetic risk for the disorder and a recent decline in functioning (McGlashan et al., 2010). CHR individuals show poorer premorbid functioning compared to healthy controls (Tikka et al., 2013; Morcillo et al., 2014; Tarbox et al., 2013) and similar premorbid functioning to patients with psychosis (Addington et al., 2008). Consistent with findings in schizophrenia, males at CHR of psychosis tend to show poorer premorbid functioning than CHR females (Salokangas et al., 2014; Tarbox et al., 2013). Furthermore, poor premorbid functioning has been shown to be associated with later poorer outcome such as increased disorganization and negative symptom severity (Quijada et al., 2012) and low functional outcome (Salokangas et al., 2014).

Poor premorbid functioning has been associated with conversion to psychosis (Dragt et al., 2011) or in conjunction with reduced P300 parietal amplitude (Nieman et al., 2014). However, poor premorbid social functioning in early adolescence significantly predicted conversion to psychosis but not poor academic and overall premorbid functioning (Tarbox et al. 2013).

The CHR studies reported above typically used overall premorbid functioning scores or mean scores at different developmental periods, however with these at-risk young people it may be more useful to evaluate whether there are different patterns of premorbid functioning developmentally, and whether these patterns associate differentially with clinical characteristics as has been observed in schizophrenia (Haas et al., 2001; Addington et al., 2003).

The aim of this study was to examine premorbid functioning in a large sample of individuals at CHR including the association with conversion to psychosis. More specifically our hypotheses were that, first, poorer premorbid functioning would be associated with worse symptom severity and poorer social functioning. Secondly, using cluster analysis to determine specific courses of premorbid functioning across the developmental stages: childhood, early and late adolescence, we hypothesized that four groups representing distinct patterns of premorbid functioning would emerge, namely stable-good, stable-intermediate, poor-deteriorating and deteriorating. Third, we hypothesized that those with a deteriorating course or those with a poor-deteriorating course would present with worse negative symptoms and lower functioning. The fourth hypothesis was that those who made the transition to psychosis would show poorer premorbid functioning compared to those who did not.

2. Method

2.1 Participants

One-hundred and sixty (88 males, 72 females) individuals at CHR of psychosis participated as part of a multi-site NIMH funded study “Enhancing the Prospective Prediction of Psychosis” (PREDICT). This was a 4-year longitudinal observational study to determine predictors of conversion to psychosis in individuals at CHR of developing psychosis with participants having follow-ups of 6 months to 4 years. Approximately 30% were lost to follow-up by year one. The study was conducted at the Universities of Toronto, North Carolina, and Yale. All CHR individuals met the Criteria of Psychosis-risk Syndromes (COPS) based on the Structured Interview for Psychosis-risk Syndromes (SIPS). 154 CHR participants met attenuated positive symptom syndrome (APSS) criteria, which includes the emergence or worsening of a non-psychotic level disturbance in thought content, thought process or perceptual abnormality over the past year, four participants met criteria for genetic risk and deterioration (GRD), which required either a first degree relative with a psychotic disorder or the subject having schizotypal personality disorder plus at least a 30% drop in functioning on the General Assessment of Functioning (GAF) scale in the past 12 months, and 2 participants met both APSS and GRD.

Participants were excluded if they met criteria for any current or lifetime axis I psychotic disorder, prior history of treatment with an antipsychotic, IQ<70, or past or current history of a clinically significant central nervous system disorder that may confound or contribute to clinical high risk symptoms, or using antipsychotics at baseline. DSM-IV diagnoses of substance dependence and abuse were exclusion criteria. Antipsychotics were not used at any later points in this study.

2.2 Measures

Criteria for a psychosis-risk syndrome and for conversion to psychosis is were determined using the SIPS (McGlashan et al., 2010). Conversion meant that at least one of the five attenuated positive symptoms reached a psychotic level of intensity (rated 6) for a frequency of 1 hour/day for 4 days/week during the past month or that attenuated psychotic symptoms that reached a psychotic level of intensity that seriously impacted functioning

(e.g. severely disorganized or dangerous to self or others). Symptoms were assessed with the Scale of Prodromal Symptoms (SOPS), which consists of 19 items in four symptom domains: positive, negative, general, and disorganized.

Participants were rated on premorbid functioning with the Premorbid Adjustment Scale (PAS) (Cannon-Spoor et al., 1982). The PAS measures premorbid functioning in four areas of development: (i) sociability/withdrawal, (ii) peer relationships, (iii) ability to function outside the nuclear family and (iv) capacity to form intimate socio-sexual ties, at each of four developmental stages, namely, childhood (up to age 11), early adolescence (12-15 years), late adolescence (16-18 years), and adulthood (19 and up) (van Mastrigt and Addington, 2002). Premorbid functioning was defined as the period up until the onset of the first attenuated psychotic symptom that contributed to the participant meeting COPS criteria.

Social functioning was assessed using the Social Functioning Scale (SFS), a self-report questionnaire developed for outpatients with schizophrenia and has excellent psychometric properties (Birchwood et al., 1990). The SFS has a total score and seven sub-scores: Withdrawal/social engagement, Interpersonal communication, Independence-performance, Independence-competence, Recreation, Prosocial, and Employment/occupation. Only the total score was used in this study.

2.3 Procedures

All three sites involved in this longitudinal study of predictors of conversion to psychosis recruited CHR individuals. Raters were experienced research clinicians who demonstrated adequate reliability at routine reliability checks. Gold standard post-training agreement on the critical threshold for determining initial eligibility and subsequent conversion status based on the SIPS was excellent ($\kappa=.90$). The PI or clinical psychiatrist or psychologist at each site conducted a comprehensive clinical assessment to determine if entry criteria were met. Dr. Jean Addington chaired weekly conference calls to review criteria for all individuals admitted to the study. The study protocols and informed consents were reviewed and approved by the ethical review boards of all three study sites. All ratings were acquired at the baseline assessment.

2.4 Statistical Analysis

Due to the young age of the sample and the fact that only a small proportion completed the adult subscale for the cluster analysis we only used three developmental stages, childhood, early adolescence and late adolescence in all of our statistical analyses.

T-tests were used to compare CHR males and females on premorbid functioning variables and those who converted and those who did not on premorbid functioning. Spearman's correlations were used to measure associations between premorbid functioning and symptoms and social functioning. Cluster analysis was used to identify distinct patterns of premorbid functioning. *K*-means cluster analysis was used to assign cases to a fixed number of groups (clusters). This procedure attempts to identify relatively homogeneous groups of cases based on selected characteristics using an algorithm that can handle a large number of cases. The algorithm requires pre-specification of the number of clusters. To classify cases, we updated cluster centers iteratively. We used the PAS developmental subscale scores for

all 160 subjects in the analysis. The decision to use a *k*-means cluster method was based on past research done in the area and the potential of this analysis to handle missing cases. This was of particular importance as given the developmental nature of the PAS and the variation in the time frame for determining an individuals' premorbid functioning.

One-way analysis of variance (ANOVA) was used to compare the clusters on symptoms and social functioning. Chi-squared was used to compare clusters on conversion status.

3. Results

The sample consisted of 88 males and 72 females. The average age was 19.8 years (SD=4.6, range 12-31 years). The majority were white (76.9%), single (94.4%), and had completed high school (56%).

Mean scores for premorbid functioning, social functioning and SOPS ratings are presented in Table 1. Males and females did not differ in premorbid functioning at any of the developmental stages nor in the overall score.

Good social functioning was significantly associated with good premorbid functioning at early ($r=-0.35$) and late adolescence ($r=-0.36$), both at the $p<0.001$ level. There was no significant association between premorbid functioning at childhood and later social functioning ($r=-0.14$, $p=0.10$). Premorbid functioning was not associated with attenuated psychotic symptoms. After application of a Bonferroni correction only negative symptoms remained significantly associated with late adolescence. See Table 2.

Results of the cluster analyses demonstrated that the best model was by pre-selecting 3 clusters versus 4 or 5 clusters. As a result of our *k*-means cluster analyses we labeled our three groups stable-intermediate ($n=33$), stable-good ($n=92$), and deteriorating ($n=35$). These results are presented in Figure 1. The greatest distances (Euclidean) were first between stable-good and deteriorating (0.49), and secondly between stable-good and stable-intermediate (0.41) and stable-intermediate and deteriorating (0.38). The remaining analyses used the three clusters described above. See Figure 1.

Results of the ANOVA comparing the three clusters on symptoms and functioning were that the clusters differed significantly in negative symptoms, disorganized symptoms, and overall social functioning. Post hoc analyses revealed that the deteriorating group had more severe negative symptoms, disorganized symptoms, and poorer social functioning than the stable good group. In addition, the deteriorating group had more severe disorganized symptoms than the stable-intermediate group. The groups did not differ in attenuated positive symptoms. See Table 3.

Twenty-nine CHR participants in the sample converted to psychosis. Those who converted had significantly poorer scores on premorbid functioning at late adolescence. See Table 4.

There were five converters in the stable-intermediate cluster, 17 in the stable-good cluster and seven in the deteriorating cluster. However, chi-squared analysis demonstrated that there were no differences among the clusters in terms of number of converters ($\chi^2=0.87$).

4. Discussion

The aim of this study was to examine premorbid functioning in a large sample of individuals at CHR of psychosis. With respect to our first hypothesis results indicated that poorer premorbid functioning during late adolescence was significantly correlated with worse negative symptom severity. In addition, high social functioning was significantly correlated with good premorbid functioning during both periods of adolescence. These findings are consistent with what has been observed in schizophrenia (Addington and Addington, 2005) and in other CHR samples (Salokangas et al., 2014; Quijada et al., 2012). However, we did not observe the expected gender difference in premorbid functioning, i.e. males having lower premorbid functioning (Tarbox et al., 2013; Salokangas et al., 2014). Although no differences have been reported in another CHR sample (Walder et al., 2013).

The cluster analysis indicated three clusters of premorbid functioning characterized by stable-good, stable-intermediate, and deteriorating patterns of premorbid functioning, suggesting that trajectories of premorbid functioning in CHR are similar to those with schizophrenia (Haas and Sweeney, 1992; Larsen et al., 1996; ; Addington and Addington, 2005). Similar to Horton et al. (2015), we did not observe in this CHR sample the poor-deteriorating pattern where individuals start out with poor premorbid functioning that continues to deteriorate over time. Our third hypothesis was supported in that these three groups differed in their clinical and functional profiles such that the deteriorating group had more severe disorganization than the other two groups, and worse negative symptoms and poorer social functioning than the stable-intermediate group. These results suggest that patterns of premorbid functioning are tightly linked to symptom severity and functional outcome prior to the onset of psychosis. These findings support the research of Häfner et al. (1992) which suggests that an individual's level of social development at the end of the prodromal phase is an important determinant of the further course of the disorder. Early identification of CHR with a deteriorating pattern of premorbid functioning may lead to better treatment options to potentially improve functional outcome.

Our final analysis revealed that individuals at CHR who converted to psychosis had significantly poorer premorbid functioning during adolescence compared to those who did not convert. Overall, our results add to previous reports suggesting that poor premorbid functioning may be predictive of later conversion (Tarbox et al., 2013; Dragt et al., 2011).

The strengths of this study include the large sample size and that, to the best of our knowledge, this is the first study to use cluster analysis to explore premorbid functioning in a CHR sample. In addition, we were able to compare CHR individuals who converted to psychosis to those who did not through longitudinal data. However, there are several limitations. First, our CHR sample had a wide age range, which meant that a few participants did not receive ratings on early adolescence or late adolescence premorbid functioning. Secondly, there may be biases with retrospective data collection, for example CHR may not have remembered correctly early premorbid functioning or their current symptoms may have obscured their recall. In addition, we did not have any collateral input from family members to corroborate individuals' recall of their premorbid functioning; however, it should be noted that there is evidence that people with schizophrenia are as

reliable as people with no psychiatric symptoms when reporting on premorbid functioning (Brill et al., 2007). Fourthly, this was a four year naturalistic study and participants were recruited throughout the course of the study, thus follow-ups ranged from one to four years with the majority being followed for about one year. Therefore there may have been participants who would have converted if they had had longer follow-up. However, typically most conversion occur within the first year of follow-up and our conversion rates are similar to those reported elsewhere. Finally, in this study we only examined the relationship of premorbid functioning with later conversion. It may be that multivariate prediction algorithms incorporating combinations of other variables known to be associated with conversion could provide further information about the role of premorbid functioning in relation to conversion in CHR youth.

In summary, our results indicate that in a CHR sample we are seeing similar patterns in premorbid functioning as have been observed in those with a psychotic illness despite the fact that we know that less than one third will go onto develop a full blown illness. Furthermore, CHR individuals with poor premorbid functioning from adolescence have poorer outcome in terms of social functioning and negative symptoms especially if their premorbid functioning has been declining over time. Previous studies have identified that poor social functioning is related to later conversion (Carrión et al., 2013) and our results add that this poor social functioning may have been ongoing for several years prior to the onset of the first attenuated psychotic symptoms. In this sample we did not observe an association between childhood premorbid functioning and later social functioning. However, the clusters suggested that childhood premorbid functioning for most was in the moderate to good range.

In terms of clinical implications results of this study support the need for psychosocial treatment particularly treatments that would address social functioning and possibly social skills. Such interventions would be necessary not just to prevent conversion to psychosis but because as we have seen here and in other studies (Addington et al., 2011) many CHR individuals who may never go on to develop have poor social functioning.

Acknowledgement

This work was supported by National Institute of Mental Health grants to J. Addington (grant number U01MH066134), to D. Perkins (grant number U01MH066069), and to S. Woods (grant number U01MH066160).

Role of the funding source

The National Institute of Mental Health had no further role in study design; in the collection, analysis and interpretation of data; in the writing of the report; and in the decision to submit the paper for publication.

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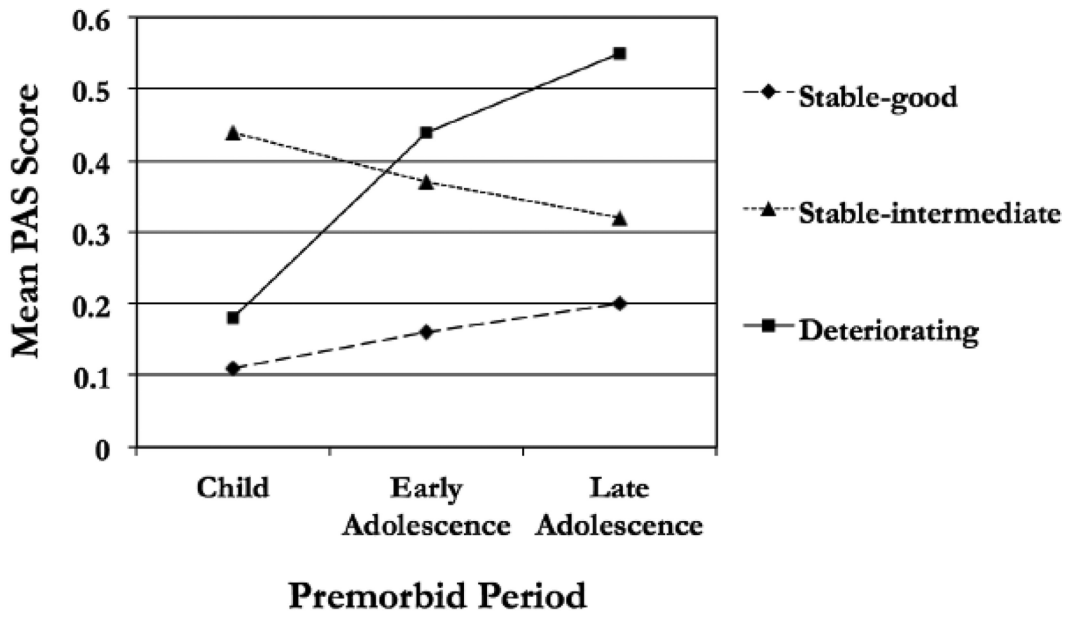
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Note: For PAS scores higher score means poorer premorbid functioning

Figure 1.
Clusters for Premorbid Functioning

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Table 1

Premorbid functioning, social functioning, and symptom scores

	Mean (SD)	Range
<i>Social Functioning Scale</i>	125.8 (20.9)	45.0—175.0
<i>Premorbid Functioning</i>		
Childhood	0.19 (0.17)	0.0—1.0
Early Adolescence ¹	0.26 (0.18)	0.0—0.8
Late Adolescence ²	0.28 (0.19)	0.0—1.0
<i>SOPS subscales</i>		
Positive	11.0 (3.2)	4.0—22.0
Negative	8.7 (5.8)	0.0—22.0
Disorganization	4.1 (2.8)	0.0—13.0
General	7.1 (3.9)	0.0—18.0

¹ Only 157 participants had reached early adolescence

² Only 119 participants had reached late adolescence

Table 2

Correlations between premorbid functioning and symptoms.

	Childhood	Early Adolescence	Late Adolescence
Total attenuated positive symptoms	0.06	0.05	0.15
Total negative symptoms	0.14	0.19 *	0.33 ***
Total disorganization symptoms	0.21 **	0.22 **	0.17
Total general symptoms	0.07	0.03	0.30 **

Bolded results are those that remained significant after Bonferroni correction for $p < 0.004$ (0.05/12).

*
p<0.05

**
p<0.01

p<0.001

Table 3

Significant associations between clusters, symptoms, and social functioning

Measure	Stable-intermediate (N=33)	Stable-good (N=92)	Deteriorating (N=35)	F Value
	Mean (SD)	Mean (SD)	Mean (SD)	
Baseline SOPS Scores				
Total positive	11.36 (3.22)	10.76 (3.21)	11.09 (3.12)	0.47
Total negative	9.00 (5.71)	7.47 (5.22)	11.54 (6.34) ^{a***}	6.83 ^{**}
Total disorganization	4.88 (2.85) ^{a*}	3.52 (2.32)	4.89 (3.41) ^{a*}	4.96 ^{**}
Total general	7.88 (3.98)	6.57 (3.67)	7.63 (4.35)	1.85

	Stable-intermediate (N=29)	Stable-good (N=86)	Deteriorating (N=32)	
Social Functioning	121.55 (21.03)	131.41 (18.99)	114.34 (20.85) ^{a***}	9.47 ^{***}

*
p<0.05**
p<0.01***
p<0.001^a Means were significantly different from stable-good group

Table 4

Comparison of the non-converters to the converters on premorbid scores

Premorbid scores	Non-converters Mean (SD)	Converters Mean (SD)	t-value
Childhood	0.19 (0.18)	0.19 (0.14)	0.26
Early Adolescence	0.26 (0.18)	0.27 (0.18)	-0.27
Late Adolescence	0.26 (0.16)	0.37 (0.25)	-2.70 *

*
p<0.05

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