

**PRE-ONSET ABNORMALITIES, PSYCHOSOCIAL STRESSORS, AND THE  
DEVELOPMENT OF PSYCHOSIS: A PROSPECTIVE, POPULATION-BASED STUDY**

by

Judy Lorraine Thompson

B.A., Stetson University, 1992

M.A., University of Richmond, 1998

Submitted to the Graduate Faculty of  
Arts and Sciences in partial fulfillment  
of the requirements for the degree of  
Doctor of Philosophy

University of Pittsburgh

2006

UNIVERSITY OF PITTSBURGH  
FACULTY OF ARTS AND SCIENCES

This dissertation was presented

by

\_\_\_\_\_  
Judy Lorraine Thompson

It was defended on

\_\_\_\_\_  
July 13, 2006

and approved by

\_\_\_\_\_  
Michael F. Pogue-Geile, Ph.D.  
Committee Chair

\_\_\_\_\_  
Susan B. Campbell, Ph.D.

\_\_\_\_\_  
Gretchen L. Haas, Ph.D.

\_\_\_\_\_  
Michael A. Sayette, Ph.D.

\_\_\_\_\_  
Magda Stouthamer-Loeber, Ph.D.

PRE-ONSET ABNORMALITIES, PSYCHOSOCIAL STRESSORS, AND THE  
DEVELOPMENT OF PSYCHOSIS: A PROSPECTIVE, POPULATION-BASED STUDY

Judy L. Thompson, Ph.D.

University of Pittsburgh, 2006

ABSTRACT

There is now considerable evidence that at least some children who later develop schizophrenia differ from those who do not across a variety of behavioral domains. Further clarifying the nature and specificity of such antecedents will inform models of etiology and pre-onset pathophysiology, as well as efforts to develop preventative strategies for psychotic disorders. Thus, a prospective study of potential predictors of psychosis was conducted. Data from 737 male participants of the population-based longitudinal Pittsburgh Youth Study were examined to determine whether psychotic-like experiences and behavior, social withdrawal, peer rejection, and problematic parent-child relationships as assessed annually from ages 13 to 17 predict early adulthood psychotic symptoms as assessed by the Diagnostic Interview Schedule at a mean age of 22. Sixteen boys reported at least one psychotic symptom that persisted for at least one month (psychosis group), 52 met criteria for antisocial personality disorder (APD), and 22 for a depressive and/or anxiety disorder. These groups were compared to the 647 boys not reporting psychotic symptoms nor meeting criteria for APD or an anxiety or depressive disorder (controls). Schizophrenia-like positive symptoms, social withdrawal, peer rejection, and problematic parent-child relationships at ages 13 to 17 were associated with the development of

early adulthood psychotic symptoms, but were not specifically predictive of psychosis relative to APD or depressive and/or anxiety disorders. Further, the psychosis group increased significantly more on indices of positive symptoms and peer rejection across adolescence compared to controls, and such patterns of change over time were generally specific to psychosis relative to APD and depressive and/or anxiety disorders. The current study adds to the existing literature by being among the few to use a representative sample to address such questions, and underscores the utility of assessing both level of and patterns of change over time on indices of functioning when attempting to identify and characterize the functioning of individuals at risk for psychosis development.

## TABLE OF CONTENTS

	Page
<a href="#">LIST OF TABLES</a> .....	ix
<a href="#">LIST OF FIGURES</a> .....	xii
<a href="#">ACKNOWLEDGMENTS</a> .....	xiv
<a href="#">1.0 INTRODUCTION</a> .....	1
<a href="#">1.1 Theoretical Importance: Models of Etiology and Pathophysiology of Schizophrenia</a> .....	1
<a href="#">1.2 Clinical Importance: Early Intervention and Prevention</a> .....	4
<a href="#">1.3 Rationale for the Domains Assessed by the Current Study</a> .....	5
<a href="#">1.3.1 Positive and Negative Schizophrenia-like Features</a> .....	5
<a href="#">1.3.2 Peer Rejection</a> .....	6
<a href="#">1.3.3 Parent-Child Relationships</a> .....	7
<a href="#">1.4 Antecedents and Causal Inferences</a> .....	8
<a href="#">1.5 Strategies used to Assess Antecedents</a> .....	9
<a href="#">1.6 Studies of Antecedents to Schizophrenia</a> .....	12
<a href="#">1.7 Studies of Schizophrenia-like Behaviors and Symptoms</a> .....	14
<a href="#">1.7.1 Positive and Disorganized Features</a> .....	14
<a href="#">1.7.1.1 3 to 5 years of age</a> .....	15
<a href="#">1.7.1.2 6 to 10 years of age</a> .....	15
<a href="#">1.7.1.3 11 to 16 years of age</a> .....	24
<a href="#">1.7.1.4 17 years of age and older</a> .....	25
<a href="#">1.7.1.5 Summary of findings regarding positive and disorganized features</a> .....	26
<a href="#">1.7.2 Negative Features</a> .....	27
<a href="#">1.7.2.1 Birth to 2 years of age</a> .....	28
<a href="#">1.7.2.2 3 to 5 years of age</a> .....	29
<a href="#">1.7.2.3 6 to 10 years of age</a> .....	29
<a href="#">1.7.2.4 11 to 16 years of age</a> .....	30
<a href="#">1.7.2.5 17 years of age and older</a> .....	40
<a href="#">1.7.2.6 Summary of findings regarding negative features</a> .....	40
<a href="#">1.8 Studies of Peer Rejection</a> .....	42
<a href="#">1.8.1 6 to 10 Years of Age</a> .....	43
<a href="#">1.8.2 11 to 16 Years of Age</a> .....	46
<a href="#">1.8.3 17 Years of Age and Up</a> .....	46
<a href="#">1.8.4 Summary of Findings Regarding Indices of Peer Rejection</a> .....	47

	Page
<a href="#">1.9 Studies of Parent-Child Relationships</a>	47
<a href="#">1.9.1 3 to 5 Years of Age</a>	48
<a href="#">1.9.2 6 to 10 Years of Age</a>	52
<a href="#">1.9.3 11 to 16 Years of Age</a>	52
<a href="#">1.9.4 Summary of Findings Regarding Parent-Child Relationships</a>	54
<a href="#">1.10 Summary of Literature Review</a>	55
<a href="#">1.11 The Current Study</a>	55
<a href="#">1.11.1 Hypothesis 1 – Schizophrenia-like Positive Symptoms</a>	56
<a href="#">1.11.2 Hypothesis 2 – Schizophrenia-like Negative Symptoms</a>	57
<a href="#">1.11.3 Hypothesis 3 – Peer Rejection</a>	58
<a href="#">1.11.4 Hypothesis 4 – Problematic Parent-Child Relationship</a>	59
<a href="#">1.11.5 Hypothesis 5 – The Moderating Effect of Schizophrenia-like Symptoms</a>	59
<a href="#">2.0 METHOD</a>	60
<a href="#">2.1 Participants and Overview of the Pittsburgh Youth Study</a>	60
<a href="#">2.1.1 Current Sample</a>	61
<a href="#">2.1.2 Outcome Groups</a>	62
<a href="#">2.1.2.1 Psychosis</a>	62
<a href="#">2.1.2.2 Antisocial personality disorder</a>	63
<a href="#">2.1.2.3 Depressive and/or anxiety disorders</a>	63
<a href="#">2.1.2.4 Well controls</a>	65
<a href="#">2.2 Measures</a>	65
<a href="#">2.2.1 Creating Constructs for Age Periods of Interest</a>	66
<a href="#">2.2.2 Constructs of the Primary Antecedents of Interest</a>	67
<a href="#">2.2.2.1 Schizophrenia-like Positive Symptoms</a>	68
<a href="#">2.2.2.2 Schizophrenia-like Negative Symptoms</a>	68
<a href="#">2.2.2.3 Peer Rejection</a>	72
<a href="#">2.2.2.4 Parent-Child Relationship</a>	73
<a href="#">3.0 RESULTS</a>	74
<a href="#">3.1 Sample Characteristics</a>	74
<a href="#">3.1.1 Selective Attrition Analyses</a>	77
<a href="#">3.1.2 Demographic Characteristics</a>	77
<a href="#">3.1.2.1 Psychosis versus controls</a>	77
<a href="#">3.1.2.2 Psychosis versus APD</a>	79
<a href="#">3.1.2.3 Psychosis versus depression/anxiety</a>	79
<a href="#">3.1.2.4 APD versus controls</a>	79
<a href="#">3.1.2.5 Depression/Anxiety versus controls</a>	79
<a href="#">3.1.2.6 Covariates for primary analyses</a>	79
<a href="#">3.1.3 Clinical Characteristics</a>	82
<a href="#">3.1.3.1 Psychosis group</a>	82
<a href="#">3.1.3.2 Psychosis versus controls</a>	83
<a href="#">3.1.3.3 Psychosis versus APD</a>	83

	Page
3.1.3.4 Psychosis versus depression/anxiety.....	83
3.1.3.5 APD versus controls .....	83
3.1.3.6 Depression/Anxiety versus controls .....	90
3.2 Preliminary Analyses.....	90
3.2.1 Data Inspection of the Primary Constructs.....	90
3.2.2 Internal Consistency and Inter-rater Correlations of the Primary Constructs.....	91
3.2.3 Descriptive Overview of Raw Scores of Primary Constructs by Age.....	93
3.2.4 Overview of HLM-derived Intercept and Slope Coefficients.....	93
3.2.5 Associations between Demographic Characteristics and HLM-derived Intercept and Slope Coefficients.....	100
3.2.5.1 Additional covariates for primary analyses .....	108
3.2.6 Associations Among HLM-derived Intercept and Slope Coefficients by Domain and by Age.....	108
3.3 Primary Analyses.....	109
3.3.1 Hypothesis 1 – Schizophrenia-like Positive Symptoms .....	113
3.3.1.1 Hypothesis 1a – Psychosis versus controls.....	113
3.3.1.2 Hypothesis 1b – Psychosis versus APD.....	119
3.3.1.3 Hypothesis 1b – Psychosis versus depression/anxiety.....	119
3.3.1.4 Hypothesis 1b – APD versus controls .....	120
3.3.1.5 Hypothesis 1b – Depression/anxiety versus controls.....	120
3.3.2 Hypothesis 2 – Schizophrenia-like Negative Symptoms.....	121
3.3.2.1 Hypothesis 2a – Psychosis versus controls.....	121
3.3.2.2 Hypothesis 2b – Psychosis versus APD.....	125
3.3.2.3 Hypothesis 2b – Psychosis versus depression/anxiety.....	125
3.3.2.4 Hypothesis 2b – APD versus controls .....	125
3.3.2.5 Hypothesis 2b – Depression/anxiety versus controls.....	126
3.3.3 Hypothesis 3 – Peer Rejection .....	126
3.3.3.1 Hypothesis 3a – Psychosis versus controls.....	126
3.3.3.2 Hypothesis 3b – Psychosis versus APD.....	127
3.3.3.3 Hypothesis 3b – Psychosis versus depression/anxiety.....	127
3.3.3.4 Hypothesis 3b – APD versus controls .....	131
3.3.3.5 Hypothesis 3b – Depression/anxiety versus controls.....	131
3.3.4 Hypothesis 4 – Problematic Parent-Child Relationship.....	131
3.3.4.1 Hypothesis 4a – Psychosis versus controls.....	135
3.3.4.2 Hypothesis 4b – Psychosis versus APD.....	135
3.3.4.3 Hypothesis 4b – Psychosis versus depression/anxiety.....	136
3.3.4.4 Hypothesis 4b – APD versus controls .....	136
3.3.4.5 Hypothesis 4b – Depression/anxiety versus controls.....	137
3.3.5 Hypothesis 5 – The Moderating Effect of Schizophrenia-like Symptoms .....	137
3.4 Secondary Analyses.....	138
3.4.1 Controlling for Substance Dependence .....	138

	Page
<a href="#">3.4.2 Psychosis Versus All Other Groups Combined</a> .....	144
<a href="#">3.4.3 Controlling for Age of Onset</a> .....	145
<a href="#">4.0 DISCUSSION</a> .....	160
<a href="#">4.1 Summary of Primary Findings</a> .....	160
<a href="#">4.2 Integration of Findings and Comparison with Other Studies by Domain</a> ....	163
<a href="#">4.2.1 Schizophrenia-like Positive Symptoms</a> .....	163
<a href="#">4.2.2 Schizophrenia-like Negative Symptoms</a> .....	166
<a href="#">4.2.3 Peer Rejection</a> .....	169
<a href="#">4.2.4 Parent-Child Relationship</a> .....	170
<a href="#">4.3 Methodological Considerations</a> .....	172
<a href="#">4.4 Clinical Implications</a> .....	177
<a href="#">4.5 Implications for Models of Etiology and Pre-onset Pathophysiology</a> .....	180
<a href="#">4.6 Future Directions</a> .....	183
<a href="#">4.7 Conclusions</a> .....	184
<a href="#">APPENDIX A. Internal Consistency (Cronbach’s alpha) for Schizophrenia-like Positive Symptom, Schizophrenia-like Negative Symptom, and Peer Rejection Informant-specific Constructs</a> .....	186
<a href="#">APPENDIX B. Inter-rater Correlations of Informant-specific Constructs for Schizophrenia-like Positive Symptoms, Schizophrenia-like Negative Symptoms, Peer Rejection, and Parent-Child Relationship</a> ...	188
<a href="#">APPENDIX C. Descriptive Statistics of Primary Constructs (raw scores) Using Total Sample</a> .....	190
<a href="#">APPENDIX D. Descriptive Statistics of HLM Coefficients by Demographic Groups for Schizophrenia-like Positive Symptoms, Schizophrenia-like Negative Symptoms, Peer Rejection, and Parent-Child Relationship</a> ...	192
<a href="#">APPENDIX E. Logistic Regression Results of Pairwise Diagnostic Group Comparisons when including Covariates for Schizophrenia-like Positive Symptoms, Schizophrenia-like Negative Symptoms, Peer Rejection, and Parent-Child Relationship</a> .....	195
<a href="#">APPENDIX F. APD versus Controls: Mean Estimated Quadratic Growth Trajectories for Schizophrenia-like Positive Symptoms, Schizophrenia-like Negative Symptoms, and Problematic Parent-Child Relationship Based on Age-13 Intercept and Quadratic Slope Coefficients</a> .....	204
<a href="#">BIBLIOGRAPHY</a> .....	207



## LIST OF TABLES

Table	Page
<a href="#"><u>1a. Studies of Schizophrenia-like Behaviors and Symptoms: Positive and Disorganized Features</u></a> .....	16
<a href="#"><u>1b. Studies of Schizophrenia-like Behaviors and Symptoms: Negative Features</u></a> .....	31
<a href="#"><u>2. Studies of Indices of Peer Rejection</u></a> .....	44
<a href="#"><u>3. Studies of Parent-Child Relationships</u></a> .....	49
<a href="#"><u>4. Psychotic Symptom Items of the Diagnostic Interview Schedule for DSM-IV (DIS)</u></a> .....	64
<a href="#"><u>5. List of Variables by Construct</u></a> .....	69
<a href="#"><u>6. Sample Attrition</u></a> .....	75
<a href="#"><u>7. Demographic Characteristics and Diagnostic Outcome of Attrited Versus Final Sample</u></a> .....	78
<a href="#"><u>8. Demographic Characteristics of Diagnostic Outcome Groups</u></a> .....	80
<a href="#"><u>9. Demographic Characteristics: Diagnostic Group Comparisons</u></a> .....	81
<a href="#"><u>10. Current Functioning and Clinical Characteristics of Diagnostic Outcome Groups</u></a> .....	84
<a href="#"><u>11. Clinical Characteristics and Functioning: Diagnostic Group Comparisons</u></a> .....	87
<a href="#"><u>12. Associations between Demographic Characteristics and HLM-derived Intercept and Slope Coefficients for Schizophrenia-like Positive Symptoms, Schizophrenia-like Negative Symptoms, Peer Rejection, and Parent-Child Relationship</u></a> .....	101
<a href="#"><u>13. Spearman Correlations Among HLM-derived Intercept and Slope Coefficients by Domain for Schizophrenia-like Positive Symptoms, Schizophrenia-like Negative Symptoms, Peer Rejection, and Parent-Child Relationship</u></a> .....	110

Table	Page
<a href="#"><u>14. Spearman Correlations Among HLM-derived Intercept Coefficients by Age and Among Slope Coefficients for Schizophrenia-like Positive Symptoms, Schizophrenia-like Negative Symptoms, Peer Rejection, and Parent-Child Relationship</u></a> .....	111
<a href="#"><u>15. Descriptive Statistics of HLM-derived Coefficients for Primary Constructs by Diagnostic Outcome Group</u></a> .....	114
<a href="#"><u>16. Schizophrenia-like Positive Symptoms: Logistic Regression Results of Pairwise Diagnostic Group Comparisons</u></a> .....	117
<a href="#"><u>17. Schizophrenia-like Negative Symptoms: Logistic Regression Results of Pairwise Diagnostic Group Comparisons</u></a> .....	123
<a href="#"><u>18. Peer Rejection: Logistic Regression Results of Pairwise Diagnostic Group Comparisons</u></a> .....	129
<a href="#"><u>19. Parent-Child Relationship: Logistic Regression Results of Pairwise Diagnostic Group Comparisons</u></a> .....	133
<a href="#"><u>20. Schizophrenia-like Positive Symptoms when including Substance Dependence as a Covariate: Logistic Regression Results of Pairwise Group Comparisons</u></a> ...	140
<a href="#"><u>21. Schizophrenia-like Negative Symptoms when including Substance Dependence as a Covariate: Logistic Regression Results of Pairwise Group Comparisons</u></a> ...	141
<a href="#"><u>22. Peer Rejection when including Substance Dependence as a Covariate: Logistic Regression Results of Pairwise Group Comparisons</u></a> .....	142
<a href="#"><u>23. Parent-Child Relationship when including Substance Dependence as a Covariate: Logistic Regression Results of Pairwise Group Comparisons</u></a> .....	143
<a href="#"><u>24. Descriptive Statistics of HLM-derived Coefficients and Logistic Regression Results for Comparison of Psychosis versus Other Groups Combined on Primary Constructs</u></a> .....	146
<a href="#"><u>25. Controlling for Age of Onset: Descriptive Statistics of HLM-derived Coefficients for Primary Constructs by Diagnostic Outcome Group</u></a> .....	155
<a href="#"><u>26. Schizophrenia-like Positive Symptoms When Controlling for Age of Onset: Logistic Regression Results of Pairwise Group Comparisons</u></a> .....	156
<a href="#"><u>27. Schizophrenia-like Negative Symptoms When Controlling for Age of Onset: Logistic Regression Results of Pairwise Group Comparisons</u></a> .....	157

Table	Page
<a href="#"><u>28. Peer Rejection When Controlling for Age of Onset: Logistic Regression Results of Pairwise Group Comparisons</u></a> .....	158
<a href="#"><u>29. Parent-Child Relationship When Controlling for Age of Onset: Logistic Regression Results of Pairwise Group Comparisons</u></a> .....	159
<a href="#"><u>E1. Schizophrenia-like Positive Symptoms: Pairwise Group Comparisons when including Covariates</u></a> .....	195
<a href="#"><u>E2. Schizophrenia-like Negative Symptoms: Pairwise Group Comparisons when including Covariates</u></a> .....	198
<a href="#"><u>E3. Peer Rejection: Pairwise Group Comparisons when including Covariates</u></a> .....	200
<a href="#"><u>E4. Parent-Child Relationship: Pairwise Group Comparisons when including Covariates</u></a> .....	202

## LIST OF FIGURES

Figure	Page
<a href="#"><u>1a. Mean raw scores plus or minus one standard deviation on Schizophrenia-like Positive Symptoms from ages 13 to 17 for total sample (range of possible scores=0-36)</u></a> .....	94
<a href="#"><u>1b. Mean raw scores plus or minus one standard deviation on Schizophrenia-like Negative Symptoms from ages 13 to 17 for total sample (range of possible scores=0-24)</u></a> .....	95
<a href="#"><u>1c. Mean raw scores plus or minus one standard deviation on Peer Rejection from ages 13 to 17 for total sample (range of possible scores=0-18)</u></a> .....	96
<a href="#"><u>1d. Mean raw scores plus or minus one standard deviation on Parent-Child Relationship from ages 13 to 17 for total sample (range of possible scores=26-78)</u></a> .....	97
<a href="#"><u>2. Mean estimated growth trajectories for Schizophrenia-like Positive Symptoms by group based on age-13 intercept and linear slope coefficients</u></a> .....	116
<a href="#"><u>3. Mean estimated growth trajectories for Schizophrenia-like Negative Symptoms by group based on age-13 intercept and linear slope coefficients</u></a> .....	122
<a href="#"><u>4. Mean estimated growth trajectories for Peer Rejection by group based on age-13 intercept and linear slope coefficients</u></a> .....	128
<a href="#"><u>5. Mean estimated growth trajectories for Parent-Child Relationship by group based on age-13 intercept and linear slope coefficients</u></a> .....	132
<a href="#"><u>6. Mean estimated growth trajectories for Schizophrenia-like Positive Symptoms by group based on age-13 intercept and linear slope coefficients when controlling for age of onset</u></a> .....	151
<a href="#"><u>7. Mean estimated growth trajectories for Schizophrenia-like Negative Symptoms by group based on age-13 intercept and linear slope coefficients when controlling for age of onset</u></a> .....	152

Figure	Page
<u>8. Mean estimated growth trajectories for Peer Rejection by group based on age-13 intercept and linear slope coefficients when controlling for age of onset</u> .....	153
<u>9. Mean estimated growth trajectories for Parent-Child Relationship by group based on age-13 intercept and linear slope coefficients when controlling for age of onset</u> .....	154
<u>F1. Mean estimated quadratic growth trajectories for Schizophrenia-like Positive Symptoms</u> .....	204
<u>F2. Mean estimated quadratic growth trajectories for Schizophrenia-like Negative Symptoms</u> .....	205
<u>F3. Mean estimated quadratic growth trajectories for Schizophrenia-like Parent-Child Relationship</u> .....	206

## ACKNOWLEDGMENTS

I would first like to thank my advisor Dr. Michael Pogue-Geile for his guidance, encouragement, and support throughout my work on this project as well as my graduate training more generally. He has provided me with many rich experiences and intellectual exchanges that have greatly contributed to my professional development. I want to extend my appreciation to Rolf Loeber and Magda Stouthamer-Loeber for their generosity in allowing me use data from the Pittsburgh Youth Study for this project, as well as for their unwavering encouragement and guidance that made this project possible. I also want to thank Rebecca Stallings for the many hours and expert knowledge of the PYS data she shared with me as this project developed. I would like to thank Drs. Sue Campbell, Gretchen Haas, and Michael Sayette for their insights, guidance, and contributions to this project and my development more generally. I express gratitude to Kathryn Weaver and David Henry of the University of Illinois-Chicago, who provided invaluable assistance regarding the statistical aspects of this project. I would also like to recognize Steve Silverstein and Ellen Herbener for their insights and support regarding my completion of this project over the last year. More generally, I am very thankful for the numerous professional opportunities provided to me by the clinical psychology program over the last several years.

I am very grateful for the ongoing guidance, support and understanding of my friends, especially Alison and Andy Gilbert, Sarah Tarbox, Larry Kalb, Maria Bleil, and Kathryn Weaver. Finally, I especially thank my parents Lois and William Thompson, and siblings Leslie Wells, Laura and Roger Betts, and William and Veronica Thompson for their unconditional encouragement and support.

## 1.0 INTRODUCTION

There is now considerable evidence that at least a subset of children who later develop schizophrenia differs from those who do not across a variety of domains, including cognitive, interpersonal, emotional, and motor functioning (Cannon et al., 2002; Erlenmeyer-Kimling et al., 2000; for review, see Isohanni et al., 2004; Jones, 1997; Kremen et al., 1998; Poulton et al., 2000). Important questions remain regarding the nature, specificity, and timing of these antecedent abnormalities, however. Clarifying the precursors of schizophrenia and other psychotic illnesses is an important task because such information critically informs models of etiology and pre-onset pathophysiology, as well as the efforts to develop preventative strategies for these often devastating disorders. Thus in an effort to contribute to these aims, the current study prospectively examined potential antecedents of psychosis.

### *1.1 Theoretical Importance: Models of Etiology and Pathophysiology of Schizophrenia*

Based on the well-grounded findings that point to important roles for both genetic and environmental factors in the etiology of schizophrenia (Gottesman, 1991), the vulnerability-stress, or diathesis-stress model of schizophrenia (e.g., Asarnow & Goldstein, 1986; Meehl, 1962) has become widely accepted as a useful framework in attempting to elucidate how such factors work together in determining who develops the full syndrome (Norman & Malla, 1993; Rakfeldt & McGlashan, 2004; Wahlberg & Wynne, 2001; Walker et al., 1996). The general thesis of this model is that individuals with a susceptibility to schizophrenia, which may derive

from both genetic and environmental factors, are especially sensitive to additional risk-increasing effects of certain environmental experiences or stressors. Thus susceptibility and stressor exposure interact such that when a susceptible individual is exposed to certain environmental stressors, her or his risk is increased even further -- and when/if a certain risk threshold is crossed, the full syndrome eventually develops. It should be noted that many researchers incorporate bi-directional influences of susceptibility factors and stressor exposure into their conceptualization of this model (e.g., social skill deficits resulting largely from genetic susceptibility may drive peer rejection, which in turn acts upon the child as a stressor to further increase susceptibility; Asarnow & Goldstein, 1986; Murray & Fearon, 1999). Thus susceptibility is viewed as interacting dynamically with both risk and protective factors rather than as a static condition that is just acted upon (Wahlberg & Wynne, 2001). Within this framework, it becomes clear that identifying and carefully characterizing pre-onset abnormalities of schizophrenia is critical to understanding the factors that contribute to illness development.

Developmental models of schizophrenia seek to identify what pathological processes lead to illness onset and when such processes begin. Numerous models have proposed that early (i.e. originating in the pre- or perinatal period) schizophrenia-specific brain abnormalities interact with or impair later maturational processes of the central nervous system in such a way that results in the manifestation of schizophrenia symptoms during young adulthood (e.g., Murray & Lewis, 1987; Walker, 1994; Weinberger, 1987). The observation of early behavioral abnormalities in pre-schizophrenia children (for reviews, see Waddington, Lane, Scully, Larkin, & O'Callaghan, 1998; Walker, 1994) and the association between obstetric complications and minor physical anomalies with the disorder (for reviews, see Harrison & Eastwood, 2001; Turner, Fedtsova, & Jeste, 1997; Waddington et al., 1998) have influenced the formulation and



been cited in support of such early neurodevelopmental theories. In contrast, “late” developmental models (e.g., Feinberg, 1982, 1982/83; Pogue-Geile, 1991) posit that schizophrenia-specific brain abnormalities that arise closer to the age of onset (e.g., during the substantial maturational brain changes of adolescence), perhaps in conjunction with earlier-occurring non-specific insults, result in the onset of the disorder. Other models have emphasized the potential importance of both early and later developmental abnormalities in their attempts to elucidate the developing pathophysiology of this disorder (Keshavan, Anderson, & Pettegrew, 1994; McGlashan & Hoffman, 2000; Woods, 1998).

Although observable behavior is of course only a fallible index of the integrity of the brain systems underlying such functioning, clarifying pre-onset behavioral abnormalities and risk factors and how they relate to each other and to normal brain maturational processes (e.g., adolescent-associated changes in the mesocortical dopamine system and the HPA axis) provides vital clues for models that seek to characterize the genesis and development of the pathophysiological processes that result in onset of schizophrenia symptoms (e.g., Murray & Fearon, 1999; Walker, Lewis, Loewy, & Palyo, 1999; Walker & Diforio, 1997). For example, based on the existing literature regarding pre-onset abnormalities of schizophrenia, Murray and Fearon (1999) have proposed that behavioral deficits (e.g., cognitive impairment and schizotypal tendencies) that are present early in life and secondary to susceptibility genes and/or early brain insults drive the development of further abnormalities (e.g., social isolation, drug abuse) that stress already vulnerable brain systems and thus increase risk to the point that the full syndrome develops. It is clear that the further characterization of the nature, timing, and specificity of pre-onset abnormalities will importantly aid in the continued generation, refinement, and rejection as appropriate, of hypotheses regarding the pathological processes contributing to syndrome onset.

## *1.2 Clinical Importance: Early Intervention and Prevention*

Interest in initiating treatment before full schizophrenia or psychosis onset with the goal of attenuating or even preventing the development of the disorder has recently increased (Cornblatt, 2002; McGorry, 1998; Yung et al., 1998; Yung et al., 2003), in part due to research suggesting that hastening the initiation of antipsychotic drug treatment after illness onset is associated with better prognosis (Perkins, Gu, Boteva, & Lieberman, 2005). Such findings support the position that a given course of illness or even full psychosis in those showing pre-onset attenuated clinical symptoms is not inevitable but can at least be somewhat ameliorated or perhaps even prevented by timely and appropriate intervention (Cornblatt, 2002; Yung et al., 1998). Prevention efforts of course crucially depend on the identification of individuals who are indeed at imminent risk of developing schizophrenia before active psychosis takes hold. This task has been a difficult one for schizophrenia researchers and clinicians because of the limited knowledge of the specific predictors of schizophrenia development and the pathological processes underlying symptom development and maintenance (for discussion, see Cornblatt, 2002; McGorry, Edwards, Mihalopoulos, Harrigan, & Jackson, 1996). Thus the advancement of prevention efforts depends critically on the further elucidation of the nature and specificity of pre-onset abnormalities and risk factors. Further, identifying common experiences that are not specifically related to psychosis development but may increase risk in susceptible individuals (e.g., psychosocial stressors) may still be helpful in identifying those at increased risk when such factors are viewed in conjunction with other antecedents (Dazzan, Kravariti, Fearon, & Murray, 2004). Moreover, the identification of such risk factors informs potential targets for intervention once susceptible individuals are identified. For example, in light of findings implicating

disturbed parent-child relationships as a risk-increasing factor for schizophrenia-spectrum disorders among those at heightened genetic risk for schizophrenia (Tienari et al., 1994; Tienari et al., 2004), Wahlberg and Wynne (2001) discuss the potential benefits of more explicitly incorporating family interventions in efforts at primary prevention.

### *1.3 Rationale for the Domains Assessed by the Current Study*

Based on the literature suggesting that children who later develop psychotic illness show functional impairment across a variety of domains, it appears that there are a number of potential areas that would be fruitful points of focus when attempting to clarify abnormalities and experiences associated with the development of such illnesses (e.g., motor development, cognitive functioning, interpersonal functioning). The current project focused on four such domains. The rationale for selecting these domains is provided next.

#### *1.3.1 Positive and Negative Schizophrenia-like Features*

Out of the vast range of behavioral indicators that may be studied as potentially related to later psychosis, a focus on childhood and adolescent experiences and behavioral features that resemble the characteristic symptoms (i.e., delusions, hallucinations, disorganized features, and negative symptoms) of schizophrenia is viewed as particularly useful. Firstly, with regard to positive symptoms specifically, if it were determined that transient psychotic experiences occur in at least a subset of children who later develop full psychosis, this could have implications for efforts to identify those at increased risk for schizophrenia and other psychotic illnesses. This is because it is possible that such experiences would be less common among those who do not go on to develop psychotic illness (so have better specificity) compared to some of the other antecedents identified so far (e.g., cognitive impairment). Of course, the usefulness of such

experiences as a predictor would also depend on their sensitivity and positive predictive value (Dazzan et al., 2004). Furthermore, sporadic or attenuated psychotic experiences are typically conceptualized as part of the prodromal phase of psychotic illness and are thus thought to signal the early stages of the active illness process. Thus findings suggesting that transient psychotic experiences occur in a relatively substantial number of children who later develop full schizophrenia would have implications for developmental models of schizophrenia pathophysiology that specifically attempt to address the emergence of psychotic experiences. Information regarding the degree to which the negative symptoms of schizophrenia are present among children who later develop the illness would also inform developmental models of pathophysiology. Thus based on these considerations, behavioral features resembling the positive and negative symptoms of schizophrenia were assessed by the current study.

### *1.3.2 Peer Rejection*

Guided by the diathesis-stress model and models of pathophysiology that implicate stressor exposure in the development of psychotic symptoms (e.g., Corcoran et al., 2003; Thompson, Pogue-Geile, & Grace, 2004; Walker & Diforio, 1997), psychosocial stressors in general were selected as another useful point of focus for the present study. Peer rejection was selected specifically based on an interest in exploring stressors related to peer relationships. This aspect of peer interactions was chosen over a more general focus (e.g., problems with peers) with the goal of examining a factor that seems to be clearly stressful. For example, it is possible that in some contexts, other aspects of problematic peer relationships, while stressful, may serve to buffer the child from the full brunt of peer problems, as may be the case for peer conflict, which in some instances may serve to elevate a child's status within a subset of children. Further, because siblings within families often do not share peer relationships, the selection of peer

rejection as a possible antecedent to later psychosis is congruent with the findings from twin and adoption studies that support an important role for nonshared, but not shared, environmental influences in the development of schizophrenia (e.g., Cannon, Kaprio, Lonnqvist, Huttunen, & Koskenvuo, 1998).

It is acknowledged that there is an inherent difficulty in measuring peer rejection as an interpersonal stressor independent of child variables that are likely correlated with peer rejection (e.g., social isolation or withdrawal). Regardless of whether such peer treatment is largely initiated as a reaction to the potential behavioral differences of a child at increased risk for psychopathology, however, peer rejection is still experienced by the child and thus viewed as potentially informative regarding stressors that may serve to further increase risk among individuals susceptible to psychosis.

### *1.3.3 Parent-Child Relationships*

A large body of work has shown that family factors, such as traits considered to reflect expressed emotion (e.g., criticism, hostility, emotional overinvolvement), reliably predict relapse in schizophrenia (for meta-analysis, see Butzlaff & Hooley, 1998). In light of this literature, it appears that examining family factors as a source of psychosocial stress that may contribute to the development of psychosis is worthwhile. Based on the findings related to expressed emotion among relatives of patients with schizophrenia, the current study focused specifically on negative aspects of the parent-child relationship.

At first consideration, the focus on family environment may seem incongruent with findings that point to minimal influences of the role of shared environmental experiences in the development of schizophrenia (Cannon et al., 1998). However, research by Reiss and colleagues (1995) supports the notion that differential parenting across siblings (including both twin and

non-twin siblings) may act as a source of nonshared environmental experiences on the development of psychopathology. It can also be hypothesized that shared family experiences (such as parent traits that are stable across interactions with all children of the family) may be experienced differentially by children who vary on psychopathology risk due to other nonshared environmental influences, which would represent a shared by nonshared environment interaction. Thus due to these considerations, a focus on family factors is not seen as incongruent with behavioral genetic findings related to psychopathology development.

It should be further acknowledged that when examining family factors among families in which the children are raised by their biological parents, genetic and environmental influences cannot be disentangled. For example, if an association between problematic parent-child relationships and later psychosis is found, there is no way to rule out the possibility that genetic factors related to both problematic parenting styles and psychosis are driving the association (among other possibilities), rather than such a relation reflecting a contributory influence of family factors on psychosis development; such issues related to making causal inferences is discussed in more detail next.

#### *1.4 Antecedents and Causal Inferences*

To prepare the inferential framework of the following review and present study findings, it is useful to discuss briefly the scope of the conclusions that can be drawn from the identification of abnormalities that precede the onset of an outcome of interest. Such antecedents may arise as a consequence of increased risk for the later development of the outcome, contribute causally to the likelihood of the outcome developing, or both. An example relevant to the present context is the potential association between peer rejection and psychosis. If such a

relation is demonstrated, it cannot be concluded that indeed peer rejection is increasing risk for later psychosis, because it may be that the “3<sup>rd</sup> variable” of pre-onset susceptibility is increasing both peer rejection (e.g., by way of social cognition deficits) and later psychosis. Although the identification of antecedents informs hypotheses regarding causal influences, pathological processes related to outcome, and the consequences of such, only empirical work that demonstrates that manipulation of a given antecedent affects the likelihood of the outcome developing can establish that the antecedent is actually causally related to the outcome (for discussion, see Kraemer et al., 1997). Thus the terms “antecedent,” “risk factor” (Kraemer et al., 1997), and “precursor” are conceptualized as describing a correlate of the outcome of interest that precedes the onset of this outcome and are not intended to denote causal implications. These terms will be used interchangeably throughout this paper. The limitations of the inferences that can be drawn based on antecedent identification will guide, as appropriate, the conclusions drawn from the review and empirical aspect of the current project.

### *1.5 Strategies used to Assess Antecedents*

When attempting to tackle the task of clearly identifying schizophrenia antecedents, researchers are unfortunately faced with the methodological difficulties that arise when studying the pre-onset period of a relatively low-incidence phenotype that onsets in late adolescence or early adulthood. Thus such researchers must rely on less than ideal research strategies (for discussion, see Davidson et al., 1999). The research designs used to address questions regarding antecedents are necessarily longitudinal and may be either retrospective or prospective. Retrospective studies that rely on self report about prior events and functioning are vulnerable to recall bias and thus will not be considered in the literature review that follows. Retrospective

archival studies circumvent this problem but are at the mercy of previously collected measures or records (e.g., school reports), which may not provide information regarding the specific phenomena of interest. Further, the reliability of retrospectively collected data may be less than ideal (Pogue-Geile & Harrow, 1984).

Prospective studies offer the considerable strengths of allowing for the selection of reliable measures and the assessment of the development of precursors across a relatively wide range of age periods within the same group of individuals. The primary disadvantages of such designs include their greater expense and the fact that conclusions regarding pre-onset abnormalities cannot be fully drawn until participants pass through the risk period of the phenomenon of interest. Further, issues of selective attrition and statistical power are often of concern with such studies.

Among the prospective designs, those that involve selecting a cohort of individuals based on some characteristic (Pogue-Geile & Harrow, 1984) include the “high-risk” design. This type of study involves assessing individuals who presumably are at increased risk for developing the disorder of interest and thus, by capitalizing on the larger number of individuals who eventually develop the disorder of study, offers a more efficient means of examining antecedents of interest than do unselected cohort or population-based prospective studies. Familial high-risk studies of schizophrenia (e.g., Erlenmeyer-Kimling et al., 1993) usually involve studying first degree relatives, typically offspring, of patients with schizophrenia longitudinally from birth or some point in childhood ideally through the risk period of illness onset with the goal of identifying antecedents of the disorder. Behavioral high-risk studies typically select adolescents or young adults who are considered to be at especially high risk for developing psychosis within the relatively near future based on behavioral characteristics (e.g., attenuated or transient psychotic



symptoms). This type of high-risk study includes those of the prodrome, which is the period preceding the development of full psychotic symptoms during which nonspecific and attenuated positive symptoms are often present (an der Heiden & Häfner, 2000; Cornblatt, 2002; Parnas, 1999; Yung et al., 1998). Prodromal studies seek to identify and characterize individuals in the prodromal period and then follow them as they develop (or not) the full syndrome of schizophrenia or other psychotic disorder. Although such studies cannot address questions regarding potential impairment before the prodrome, they can provide critical information regarding changes that occur immediately before and during the transition to active psychosis. However, because high-risk individuals (as defined either by parental diagnosis or behavioral abnormalities) who develop schizophrenia may differ in important ways from individuals who do not meet the high-risk selection criteria used, generalizability from these selected cohort studies may be limited.

The unselected cohort or population-based prospective study involves following a cohort selected to represent the general population. Of course when studying a low base-rate phenomenon such as schizophrenia, such a design becomes less tenable due to the large number of participants needed to yield even a small number of individuals who eventually develop the disorder (Pogue-Geile & Harrow, 1984). However, because this design offers all of the strengths of the longitudinal prospective design without the hindrance of generalizability limitations, it is considered the most powerful method to assess antecedents of a phenomenon of interest, albeit one of the most costly in terms of both time and money. Thus in the literature review that follows, more weight will be given to results yielded by studies employing unselected rather than selected (e.g. high risk) samples, with the most weight being given to the results derived from those using population-based samples.

### *1.6 Studies of Antecedents to Schizophrenia*

To provide a framework for the present study, a literature review of the domains of interest are presented below. Specifically, retrospective archival, high-risk, and population-based longitudinal studies that examined the relation between either schizophrenia or psychosis and schizophrenia-like positive features, schizophrenia-like negative features, peer rejection, or parent-child relationships are presented and discussed. When viewing the results of studies investigating antecedents of psychotic illness, it is important to note that the differences obtained between those who later become ill and those who do not are often quite small. However, as noted above, often subtle but measurable differences between such groups have been detected in a number of functional domains (e.g., Erlenmeyer-Kimling et al., 2000; Malmberg et al., 1998).

In addition, research has suggested that some of the abnormalities observed in pre-schizophrenia children are also present in children who later develop mood and/or anxiety disorders (e.g., see Cannon et al., 2002; Cannon et al., 1997), although results from such studies have been mixed (e.g., Done, Crow, Jonestone, & Sacker, 1994; Erlenmeyer-Kimling et al., 2000; Reichenberg et al., 2002). Accordingly, the review that follows attempts to draw conclusions regarding the specificity, in addition to the nature and timing, of the antecedents of interest. It should be noted, however, that specificity, which is typically conceptualized as the degree to which the trait in question is related to the disorder of interest but not to other disorders, is a quantitative concept rather than a categorical one. Further, the specificity of a given trait for the disorder of interest is relative in the sense that it of course is defined by what other disorders were examined with respect to that trait. Questions of the specificity of abnormalities observed in pre-schizophrenia children have often been considered via the

inclusion of other diagnostic groups that are, like the schizophrenia group, compared to normal controls. If the pre-schizophrenia children diverge from controls on the trait of interest but the other-disorder children do not, then that trait is often considered specific to schizophrenia. It seems, however, that to fully address whether pre-schizophrenia individuals are distinguishable on the trait in question from those who go on to develop other disorders, other-disorder children should be compared both to controls and to pre-schizophrenia children. Most studies that attend to questions of specificity include the former but not latter comparison; thus the consideration of specificity issues in this literature review will for the most part focus on differences observed in pre-schizophrenia but not other-disorder children when compared to controls. Because such comparisons do not allow one to draw conclusions regarding the potential differences or lack thereof between the schizophrenia and other diagnostic groups of interest, however, conclusions regarding specificity are limited under such conditions.

It will be noted that across the investigations reviewed, there is a great deal of variability with regard to the psychosis-related diagnostic outcome groups used. Specifically, they range from schizophrenia and schizoaffective disorder, to schizophreniform disorder and delusional disorder, to brief psychosis with duration greater than one week and psychosis not otherwise specified. This in part reflects the practical difficulties of attempting to include only those individuals who meet full diagnostic criteria when studying relatively young adults who are still within the risk period for developing the full syndrome (e.g., Yung et al., 2003). It further reflects the assumption that examining factors related to the development of full psychotic symptoms that do not appear to be transient is informative to the study of schizophrenia in general both because such symptoms are a major feature of the illness and because a subset of individuals experiencing such symptoms will go on to meet full criteria for schizophrenia.

As noted above, the review that follows does not include retrospective studies that relied on self report about prior events and functioning due to concerns regarding recall bias. Further, all studies included were required to have used an adequate control group, and to have examined the antecedents as related to a group comprised of at least eight participants who later developed schizophrenia or other psychotic illness.

### *1.7 Studies of Schizophrenia-like Behaviors and Symptoms*

The search for behavioral precursors of schizophrenia has long included attempts to identify subtler forms of the striking psychotic and negative symptoms that characterize the disorder after full onset (e.g., Bower & Shellhamer, 1960; Michael, Morris, & Soroker, 1957). As reflected by Tables 1a and b, the various behaviors and symptoms assessed in individuals who later develop schizophrenia have included those thought to reflect the positive and disorganized features of the disorder (i.e., hallucinations, delusions, and disorganized speech and behavior), as well as its negative symptoms, such as social withdrawal, anhedonia, and flat affect. It can be seen that the means by which such behavioral disturbances have been assessed, the age of children studied, and the specific schizophrenia-like features examined have varied considerably across investigations. The following review attempts to draw general conclusions across these studies as appropriate. To facilitate the consideration of the timing of such abnormalities, findings will be considered for each age group as noted in Tables 1a and b.

#### *1.7.1 Positive and Disorganized Features*

The symptoms considered here include behaviors and experiences thought to reflect either attenuated or transient forms of full psychotic or disorganized features of schizophrenia, which include hallucinations, delusions, disorganized speech, and disorganized behavior. As can

be seen by reviewing Table 1a, a relatively limited number of studies have either prospectively assessed such features or used contemporaneous data of childhood to examine how these traits may be present to a detectable degree in children who later develop schizophrenia or psychosis. Due to the limited numbers of applicable investigations, studies that used composite scales that included other behavioral traits (e.g., flat affect, seclusiveness, nail biting; see Table 1a) in addition to at least several of the features of interest were included with the awareness that findings based on such heterogeneous measures are more difficult to interpret with regard to the specific traits in question here.

*1.7.1.1 3 to 5 years of age.* One study of Table 1a assessed children during this age period (Bearden et al., 2000) and found that “deviant behaviors” at age 4, which included meaningless hand motions and laughter, stereotyped behavior, among other behaviors (see Table 1a for a complete list) were increased among those who later developed schizophrenia or schizoaffective disorder compared to well controls. Further, they reported that the unaffected siblings of these pre-schizophrenia/-schizoaffective disorder children were also rated as displaying more deviant behaviors at age 4 than control children. As the authors of this large population-based study noted, these results suggest that such deviant behaviors not only are increased as early as age 4 in those who later develop schizophrenia, but that they may also be associated with an increased genetic or at least familial risk for the disorder.

*1.7.1.2 6 to 10 years of age.* Overall, the studies of Table 1a suggest that aspects of disorganized speech (Ott, Allen, & Erlenmeyer-Kimling, 2001; Roff, 2001; Roff & Fultz, 2003) and various aspects of odd, bizarre, or unusual behavior (Bearden et al., 2000; Roff, 2001; Roff & Fultz, 2003) may be elevated to a detectable degree in children who later develop schizophrenia compared to controls during the age period of 6 to 10 years. As discussed, the

Table 1a  
*Studies of Schizophrenia-like Behaviors and Symptoms: Positive and Disorganized Features*

Study	Sample	Behaviors/symptoms	3 to 5 yrs old	6 to 10 yrs old	11 to 16 yrs old	17 yrs and up
<i>Studies of psychotic symptoms</i>						
<i>Unselected samples: Population-based:</i>						
Poulton et al., 2000; Dunedin Multi-disciplinary Health and Development Study	-schfm=25 -mania=14 -depr <sup>1</sup> =119 -anxiety <sup>2</sup> =180 -ctrls=423	Assessed at age 11:  -psychotic symptoms <sup>3</sup>	---	---	-pre-schfm > ctrls	---
<i>Studies of Schizophrenia-like positive symptoms and behaviors, including composite scales and general ratings</i>						
<i>Selected samples: Child guidance clinic or child/adolescent treatment unit:</i>						
O'Neal & Robins, 1958	-sch=28 -ctrls=57	Noted at median age of 13-14 (range=1.5 to 17 yrs old):  -odd ideas, paranoid ideas	---	---	-pre-sch > ctrls	---

Table 1a (continued)

Study	Sample	Behaviors/symptoms	3 to 5 yrs old	6 to 10 yrs old	11 to 16 yrs old	17 yrs and up
Cannon, M. et al., 2001	-sch=59 -affective psychosis <sup>4</sup> =27 -ctrls=86	Per clinician ratings made regarding prior 12 months at mean age of 13-14:  -abnormal suspiciousness or sensitivity	---	---	-pre-sch > ctrls; <i>NS</i> for affective psychosis	---
Roff, 2001 <sup>5</sup>	-male poor outcome sch=23 -male good outcome sch=21 -male "antisocial" <sup>6</sup> =50 -male ctrls <sup>7</sup> =50	Per review of child guidance center files; assessed at mean age of 9.3  -scale based on thought disorder, flat affect, bizarre behavior, teacher considers child disturbed, seclusiveness	---	-poor outcome sch > all other groups	---	---
Roff & Fultz, 2003 <sup>5</sup>	-male sch=49 -male "antisocial" <sup>8</sup> =50 -male ctrls <sup>7</sup> =49	Per review of child guidance center files; assessed at mean age of 8.7:  -scale based on bizarre behavior, teacher considers child disturbed, seclusiveness	---	-pre-sch > ctrls	---	---
<i>Selected samples: Behavioral high risk:</i>						
Chapman et al., 1994	Among psychosis-prone <sup>9</sup> and ctrls: -psychosis <sup>10</sup> =13 -ctrls <sup>11</sup> =494	Assessed at mean age of approx. 20:  -psychotic and psychotic-like experiences rating <sup>12</sup>  -Perceptual Aberration-Magical Ideation Scale <sup>13</sup>	---	---	---	-pre-psychosis > ctrls  -pre-psychosis > ctrls

Table 1a (continued)

Study	Sample	Behaviors/symptoms	3 to 5 yrs old	6 to 10 yrs old	11 to 16 yrs old	17 yrs and up
Klosterkötter et al., 1997	Among outpatients referred to psychiatric clinics <sup>14</sup> : -sch=56 -non-sch=40	Assessed by self-report BSABS <sup>15</sup> at median age of 28 (range=17-54):				
		-blocking of thoughts	---	---	---	-pre-sch > ctrls
		-disturbances of discrimination between ideas and perceptions	---	---	---	-pre-sch > ctrls
		-tendency to delusion of reference	---	---	---	-pre-sch > ctrls
		-perceiving optical stimuli as larger or smaller than they are	---	---	---	-pre-sch > ctrls
		-changes in perception of the face/body of others	---	---	---	-pre-sch > ctrls
		-changes in perception of one's own face	---	---	---	-pre-sch > ctrls
-acoasms: hearing sounds such as clapping, knocking, humming in absence of external stimuli and experiencing as acoustic annoyance	---	---	---	-pre-sch > ctrls		



Table 1a (continued)

Study	Sample	Behaviors/symptoms	3 to 5 yrs old	6 to 10 yrs old	11 to 16 yrs old	17 yrs and up
Miller et al., 2002, 2003; PRIME clinic	Among patients referred for suspected prodromal syndrome: -sch psychosis=8 -non-sch psychosis=15	Assessed by SOPS <sup>16</sup> at mean age of 17.9:  - <u>prodromal syndrome</u> as based on presence of attenuated positive symptoms OR brief intermittent psychotic symptoms as defined by ratings on: unusual thought content/delusional ideas, suspiciousness/persecutory ideas, grandiosity, perceptual abnormalities/hallucinations, or disorganized communication	---	---	---	-pre-sch psychosis > non-sch psychosis
Lencz et al., 2003; Cornblatt et al., 2003; RAP clinic	Among patients meeting criteria for attenuated positive symptoms <sup>17</sup> : -psychosis <sup>18</sup> =9 -nonpsychosis=25	SOPS <sup>16</sup> ratings of following positive symptoms at mean age of approx. 16:  -unusual thought, content/delusional ideas, suspiciousness/persecutory ideas, grandiosity, perceptual abnormalities/hallucinations, disorganized communication	---	---	-pre-psychosis > nonpsychosis	---
Yung et al., 2003; McGorry et al., 2002; PACE clinic	Among UHR <sup>19</sup> patients: -psychosis <sup>20</sup> =20 -nonpsychosis=29	Assessed monthly over period up to 12 mo at mean age of 19 on:  -BPRS <sup>21</sup> -psychotic subscales (unusual thought content, suspiciousness, hallucinations, conceptual disorganization)	---	---	---	-pre-psychosis > nonpsychosis

Table 1a (continued)

Study	Sample	Behaviors/symptoms	3 to 5 yrs old	6 to 10 yrs old	11 to 16 yrs old	17 yrs and up
<i>Selected samples: Familial high risk:</i>						
Parnas et al., 1982; Copenhagen HR Project <sup>22</sup>	Among HR-sch: -sch=13 -borderline sch <sup>23</sup> =29 -ctrls=55	Per clinician assessment at mean age of 15:  -incoherent thought structure (formal thought disorder) <sup>24</sup>  -pathological associations (formal thought disorder) <sup>24</sup>	---	---	-NS <sup>25</sup>  -NS <sup>25</sup>	---
Olin et al., 1995; Copenhagen HR Project <sup>22</sup>	Among HR-sch: -sch=30 -SPD <sup>26</sup> =39 -nonpsychotic disorders=32 <sup>27</sup> -ctrls=66	Teacher rated at mean age of 15.1 (range=9 to 20 yrs old):  “future psychotic or emotional problem”	---	---	-pre-sch > ctrls <sup>25</sup> for males and females	---
Carter et al., 2002; Copenhagen HR Project <sup>22</sup>	Among HR-sch and ctrls: -sch=33 -other mental illness <sup>28</sup> =132 -ctrls=128	Assessed at mean age of 15:  -MMPI index (unusual beliefs, thoughts, perceptions) <sup>29</sup>	---	---	-NS (trend for pre-sch > ctrls) <sup>25</sup>	---
Ott et al., 2001; NYHRP	Among HR-sch, HR-mood, and ctrls: -sch-related psychosis <sup>30</sup> =9 -mood disorder or ctrls=113	Videotaped interviews at mean age of 9 rated for:  -global thought disorder <sup>31</sup>  -positive thought disorder <sup>31,32</sup>	---	-pre-sch-related psychosis > other groups <sup>33</sup>  -pre-sch-related psychosis > other groups <sup>33</sup>	---	---

Table 1a (continued)

Study	Sample	Behaviors/symptoms	3 to 5 yrs old	6 to 10 yrs old	11 to 16 yrs old	17 yrs and up
<i>Unselected samples: Population-based:</i>						
Jones et al., 1994; British 1946 birth cohort (NSHD)	-sch=30 -ctrls=4716	Self report measure at age 13:  -negative attitude to others <sup>34</sup>	---	---	-NS <sup>35</sup>	---
Bearden et al., 2000; Philadelphia Collaborative Perinatal Project (NCP)	-sch/schaff=49 and 65 <sup>36</sup> -unaff sibs of sch/schaff=26 and 32 <sup>36</sup> -ctrls=4492 and 4922 <sup>36</sup>	Clinician-rated at ages 4 and 7:  -“deviant behaviors” (e.g. echolalia, stereotyped behavior) <sup>37</sup>	-pre-sch>ctrls <sup>38</sup>	-pre-sch > ctrls <sup>38</sup>	---	---

*Note.* Table presents results for schizophrenia and/or psychosis-related diagnostic groups only; see text for discussion of results of other diagnostic groups in context of specificity questions. Significant group differences noted if  $p \leq .05$  as reported by authors; trend noted if  $p \leq .09$  (as able based on provided information).

approx=approximately; BPRS=Brief Psychiatric Rating Scale; BSABS=Bonn Scale for the Assessment of Basic Symptoms; ctrls=controls; depr=depression; HR= high risk; mo=months; NCP=National Collaborative Perinatal Project; NSHD=National Survey of Health and Development; NS=nonsignificant; NYHRP=New York High-Risk Project; sch=schizophrenia; schaff=schizoaffective; schfm=schizophreniform; sibs=siblings; SOPS=Scale of Prodromal Symptoms; UHR=ultra-high-risk; unaff=unaffected; yrs=years

<sup>1</sup>included major depressive episode and dysthymia.

<sup>2</sup>included generalized anxiety disorder, panic disorder, agoraphobia, social phobia, obsessive-compulsive disorder, posttraumatic stress disorder, and specific phobia.

<sup>3</sup>as measured by these items from the Diagnostic Interview Schedule for Children (DISC-C), which was administered by a child psychiatrist: 1) “Some people believe in mind reading or being psychic. Have other people ever read your mind?” 2) Have you ever had messages sent just to you through television or radio?” 3) “Have you ever thought that people are following you or spying on you?” 4) “Have you heard voices that other people can’t hear?” 5) “Has something ever gotten inside your body or has your body changed in some strange way?”

<sup>4</sup>defined as bipolar affective disorder or severe depression with psychotic features per ICD-10 criteria.

<sup>5</sup>Note that the reports by Roff, 2001, and Roff & Fultz, 2003, used partially overlapping samples.

<sup>6</sup>defined by a record of severe bad conduct while in service, serious enough to lead to other than an honorable discharge or to a number of days Absent Without Official Leave or days of confinement totaling at least 60; it was noted that many were diagnosed by service psychiatrists as having a personality disorder.

<sup>7</sup>defined by promotion in rank and no significant disciplinary or psychiatric records.

<sup>8</sup>defined by a record of severe bad conduct with a disciplinary record while in the service.

<sup>9</sup>as defined by high scores on the Revised Physical Anhedonia Scale (Chapman et al., 1976), Perceptual Aberration Scale (Chapman et al., 1978), Magical Ideation Scale (Eckblad & Chapman, 1983), and/or the Impulsive Nonconformity Scale (Chapman et al., 1984).

Table 1a (continued)

<sup>10</sup>included schizophrenia, psychosis NOS, delusional disorder, and bipolar disorder with psychotic features

<sup>11</sup>included all participants who did not meet criteria for psychosis as defined above but who may have met criteria for other disorders (e.g., personality and mood disorders)

<sup>12</sup>an interview-based rating of these six broad classes of psychotic and psychotic-like experiences: transmission of thoughts, passivity experiences, voice experiences and other auditory hallucinations, thought withdrawal, other personally relevant aberrant beliefs, and aberrant visual experiences

<sup>13</sup>combining scales assessing “schizophreniclike” distortions in the perception of one’s own body, other perceptual distortions, and beliefs in forms of causation that are considered magical or “invalid” by conventional standards

<sup>14</sup>patients referred were considered to have responded insufficiently to treatment and thus were considered problematic cases; diagnoses at initial assessment included schizoid personality disorder, schizotypal personality disorder, borderline/histrionic/narcissistic personality disorder, dependent/obsessive-compulsive personality disorder, major depression, dysthymia, hypochondriasis, somatization disorder, panic disorder, obsessive-compulsive disorder, and generalized anxiety disorder

<sup>15</sup>Bonn Scale for the Assessment of Basic Symptoms; Gross, Huber, Klosterkötter, & Linz, 1987

<sup>16</sup>Scale of Prodromal Symptoms; Miller et al., 1999

<sup>17</sup>defined as presence of attenuated positive symptoms based on SOPS ratings of the following symptoms: unusual thought content/delusional ideas, suspiciousness/persecutory ideas, grandiosity, perceptual abnormalities/hallucinations, or disorganized communication

<sup>18</sup>included schizophrenia (4), schizoaffective disorder (2), delusional disorder (1), and psychotic disorder NOS (2)

<sup>19</sup>criteria for UHR: attenuated positive symptoms (presence of at least one of these symptoms: ideas of reference, odd beliefs or magical thinking, perceptual disturbance, paranoid ideation, odd thinking and speech, odd behavior and appearance; held with a reasonable degree of conviction and occurring at least several times a week) OR transient psychotic symptoms (presence of at least one of these symptoms but with duration of less than one week: hallucinations, delusions, formal thought disorder) OR first degree relative with psychotic disorder or schizotypal personality disorder and significant decline in mental state or functioning occurring within past year

<sup>20</sup>included schizophrenia, schizophreniform disorder, schizoaffective disorder-depressed type, bipolar disorder with psychotic features, major depression with psychotic features, brief psychosis with duration greater than one week, and psychotic disorder NOS

<sup>21</sup>modified 24-item version of the Brief Psychiatric Rating Scale (Overall & Gorham, 1962; Lukoff et al., 1986)

<sup>22</sup>note that the three reports of Copenhagen HR Project (Carter et al., Olin et al., and Parnas et al.) used overlapping samples.

<sup>23</sup>this diagnosis corresponds to DSM-III diagnosis of schizotypal personality disorder

<sup>24</sup>items selected from psychiatric interview

<sup>25</sup>schizophrenia vs. controls

<sup>26</sup>included schizotypal and paranoid personality disorder

<sup>27</sup>included nonpsychotic depression, substance abuse, and other nonpsychotic Axis I and II disorders

<sup>28</sup>included atypical psychosis (8), schizoaffective disorder (1), schizophreniform psychosis (1), schizotypal personality disorder (41), paranoid personality (5), schizoid personality (1), depression (17), substance abuse (17), other Axis I disorders, and other Axis II disorders

<sup>29</sup>this scale designed to reflect unusual beliefs, perceptions, and thoughts; comprised sum of weighted raw scores from Infrequency (F), schizophrenia (8), and psychoticism (PSY) scales of a partial MMPI.

<sup>30</sup>included schizophrenia, schizoaffective disorder-mainly schizophrenic, and unspecified psychosis

<sup>31</sup>based on the Scale for Thought, Language, and Communication (Andreasen, 1986)

<sup>32</sup>included derailment and tangentiality

<sup>33</sup>reported that pre-sch-related psychosis group elevated on scale but unclear if compared to other groups combined or separately

<sup>34</sup>a measure from Pinter aspects of personality inventory; example items are “I find that very few people can be trusted,” and “I often get blamed for things I didn’t do”

Table 1a (continued)

<sup>35</sup>when controlling for sex and SES

<sup>36</sup>*n* for the age 4 and 7 assessments, respectively

<sup>37</sup>consisted of these items: thumbsucking, nail biting, meaningless hand motions, meaningless laughter, excessive crying, echolalia or other speech difficulties, stereotyped behavior, and/or other deviant behaviors

<sup>38</sup>when controlling for sex, race, age at examination, parental SES, and parental education

report that deviant behaviors were significantly increased in unaffected siblings of pre-schizophrenia children (Bearden et al.) as well suggests that such behaviors may be reflective of increased genetic risk for the disorder. In addition, the findings of Roff's research group (Roff, 2001; Roff & Fultz, 2003) suggest that some of these abnormalities (e.g., bizarre behavior, thought disorder, seclusiveness, etc.) may be specific to those who later develop schizophrenia compared to antisocial personality tendencies, although such findings may be limited to boys. Due to the limited number of studies assessing such traits during this age period and the possible generalizability limitations of the studies of Ott et al. and Roff's group (both used selected samples; see Table 1a), however, further studies examining various aspects of odd behavior and disorganized speech at these early ages would be useful.

*1.7.1.3 11 to 16 years of age.* The findings of Table 1a are mixed but suggestive regarding whether various aspects of suspiciousness, odd behavior, and paranoid, or unusual ideas are increased in children who later develop schizophrenia compared to controls during the age period of 11 to 16 years. The three studies reporting that such features were increased among pre-schizophrenia children (Cannon et al., 2001; Olin, John, & Mednick, 1995; O'Neal & Robins, 1958) all have generalizability limitations due to their sample ascertainment methods, which lessens somewhat the weight of their findings. At the same time, it is interesting to note that these three investigations, in contrast to the two studies that found that such behavioral features were not associated with later schizophrenia (Carter, Schulsinger, Parnas, Cannon, & Mednick, 2002; Jones, Rodgers, Murray, & Marmot, 1994), examined these traits as rated by either clinicians or teachers rather than relying on self-report measures. Thus it may that these traits (e.g., increased suspiciousness and sensitivity, unusual thoughts) are subtly increased

among those who later develop the disorder compared to other children during this age period, but that due to their nature, they are less amenable to detection via self-report measures during these early adolescent years.

The findings of Poulton et al.'s (2000) prospective population-based investigation indicated that psychotic experiences as assessed by a psychiatrist at age 11 were increased among those who developed schizophreniform disorder by age 26, and that such an association was maintained when controlling for sex, SES, and age 11-IQ scores. Further, their results suggest that such symptoms are specifically predictive of this disorder compared to mania and depressive disorders, although not compared to anxiety disorders. These findings are compelling regarding possible increased rates of full psychotic experiences among pre-adolescent children who later develop schizophreniform disorder and thus strongly warrant attempts to replicate them. Lencz et al. (2003) also showed that attenuated psychotic symptoms as assessed by interview predicted who later developed full psychosis, although their sample consisted of older children on average (mean of 16 years) who were all considered at high risk for impending psychosis, the latter of which renders their findings less generalizable than those of Poulton and colleagues.

*1.7.1.4 17 years of age and older.* The three behavioral high-risk studies of young adults considered to be at varying degrees of risk for impending psychosis listed in Table 1a showed that frequency or degree of attenuated and/or brief psychotic symptoms predicted the development of full psychosis among these high-risk individuals (Klosterkötter, Schultze-Lutter, Gross, Huber, & Steinmeyer, 1997; Miller et al., 2002; Yung et al., 2003). The findings of Chapman and colleagues (1994), who studied college students rather than a clinical sample, were congruent with the above findings in that among their putatively psychosis-prone and control

young adult participants, the degree of both self-endorsed and interview-based ratings of psychotic and psychotic-like experiences predicted who later developed full psychosis. Thus these findings strongly point to increased rates of psychotic-like experiences among young adults who later develop full psychosis. However, all of these studies have generalizability limitations because they assessed such experiences and traits either only among those considered to be at high risk for psychosis or over-selected for individuals at increased risk; thus additional studies of such behavioral features among samples of young adults that are more representative of the general population are needed.

*1.7.1.5 Summary of findings regarding positive and disorganized features.* To summarize, several studies as reviewed above suggest that behavioral features and traits that resemble the positive and/or disorganized features of schizophrenia may be increased as early as age 4 and are perhaps detectable through at least early adolescence among those who go on to develop schizophrenia, including deviant or odd behavior or ideas (Bearden et al., 2000; Cannon et al., 2001; Olin et al., 1995; O'Neal & Robins, 1958; Roff, 2001; Roff & Fultz, 2003) and aspects of disorganized speech (Ott et al., 2001; Roff, 2001; Roff & Fultz, 2003). Further, these abnormalities may be specific to children who later develop schizophrenia compared to those who later display antisocial traits (Roff; Roff & Fultz) or are later diagnosed with mood disorder with psychotic features (Cannon et al., 2001). However, due to mixed findings (e.g., see Carter et al., 2002; Jones et al., 1994; Parnas et al., 1982), the design limitations of several of these investigations, and the limited number of studies examining the same traits across several age periods of childhood, additional research employing representative samples and systematically-collected data across childhood is needed to corroborate the interesting suggestions of the reviewed studies.



As noted above, several behavioral high-risk studies (Chapman et al., 1994; Klosterkötter et al., 1997; Lencz et al., 2003; Miller et al., 2002; Yung et al., 2003) have shown that among adolescents and/or young adults among whom at least a subset were considered to be at increased risk for impending psychosis, psychotic-like features or attenuated and/or brief psychotic symptoms predicted who later developed full psychosis. Only one study of psychotic symptoms during late childhood (age 11) used a large population-based sample and prospectively-collected data (Poulton et al., 2000). These investigators reported that the level of psychotic experiences was predictive of later schizophreniform disorder in a linear fashion, and that such an association appeared to be specific to schizophreniform disorder compared to mood disorders but not anxiety disorders. Thus this latter study provides corroboration for behavioral high risk studies that suggest that psychotic experiences are present before full-blown psychosis develops, but importantly points to the possibility that such experiences may be detectable as early as age 11 among children selected from the general population who later develop schizophreniform disorder. As noted, additional studies employing this type of design that assess psychotic experiences in childhood and adolescence are needed to replicate this finding and to help further elucidate questions of the timing and specificity of such symptoms.

### *1.7.2 Negative Features*

The negative symptoms of schizophrenia include avolition, alogia, anhedonia, flat affect, and social isolation or withdrawal. Thus the following review includes studies that measured such symptoms directly or assessed behaviors that were judged to most likely reflect the presence of such disturbances (e.g., “fewer than two close friends” as measured by Malmberg, Lewis, David, & Allebeck, 1998, included as an index of social isolation). Because the purpose of this portion of the review is to examine findings relating specifically to pre-onset negative

symptoms, studies that assessed only the more non-specific disturbances associated with schizophrenia (e.g., dysphoric mood, anxiety, global deterioration in functioning) were not included. The task of determining whether a particular measure or trait was appropriate for inclusion based on these guidelines was especially challenging for social isolation or withdrawal. This is an important symptom for pre-onset study, as it has long been noted as part of the “personality” of children who later develop schizophrenia (Bower & Shellhamer, 1960; Offord & Cross, 1969). However, social functioning in pre-schizophrenia children has often been assessed quite generally, with measures used incorporating traits such as social cognition, social anxiety, and popularity, in addition to behaviors possibly reflective of social isolation (e.g., Rabinowitz et al., 2000). Further, traits such as shyness and introversion are likely to result in some degree of social withdrawal at times; thus it is unclear whether measures of these and similar traits should be considered to reflect social isolation. Guided by the purpose of the review as stated above and the descriptions of the measures provided by the authors of the studies considered, investigations that included composite measures that contained several items judged to reflect aspects of social functioning other than social isolation or measures that appeared to primarily assess social anxiety, social rejection, shyness, or timidity, were not included. It is acknowledged that in some instances such decisions required more subjective judgment than is ideal; however, efforts were made to apply these guidelines evenly across the studies considered.

*1.7.2.1 Birth to 2 years of age.* As noted in Table 1b, Walker and colleagues (1993) rated the home movies of participants from birth to 2 years for the occurrence and duration of various facial expressions and found that children who later developed schizophrenia displayed less facial expressions of joy compared to their unaffected same-sex siblings, although not

consistently throughout this age period (see Table 1b). The only other investigation listed in Table 1b to assess possible abnormalities during this age period was that of Parnas and colleagues (1982), who examined data from the Copenhagen High-Risk Project. Their results suggest that high-risk offspring who later develop schizophrenia may be more passive as babies compared to high-risk offspring who remain well, and that such increased passivity during infancy may not be specific to babies who later develop full schizophrenia, but may also characterize babies who go on to develop schizotypal traits in the absence of full-blown symptoms. Overall, due to the mixed findings for this age period obtained by Walker et al., the possible generalizability limitations of Parnas et al.'s familial high-risk study, and because such findings are derived from single studies, firm conclusions cannot be drawn from them.

*1.7.2.2 3 to 5 years of age.* As can be seen in Table 1b, Walker and colleagues (1993) again obtained mixed results when examining facial expressions via home movies for this age period, but overall their findings suggest that during these years, pre-schizophrenia boys tend to show higher rates of negative affect and pre-schizophrenia girls tend to show a reduced rate of positive affect (joy) compared to same-sex sibling controls. Further, the results from the British 1946 birth cohort study reported by Jones and colleagues (1994) suggest that during this age period, children who later develop schizophrenia may tend toward social isolation per mother ratings (see Table 1b).

*1.7.2.3 6 to 10 years of age.* The findings summarized in Table 1b for this age period suggest that alogia-like features, ratings of negative symptoms in general (Ott et al., 2001), and negative affect at least among boys (Walker et al., 1993), may be increased among children who later develop schizophrenia compared to controls. Four of the five studies of Table 1b that assessed aspects of social withdrawal during this age period found that pre-schizophrenia or -

schizophreniform disorder children did not differ significantly from control children (Cannon et al., 2002; Crow, Done, & Sacker, 1995; Michael et al., 1957; Watt, 1978). Thus these findings suggest that this feature may not be increased from ages 6 to 10 years among those who later develop schizophrenia, although the positive findings regarding isolative behavior during this age period of Jones and colleagues (1994) counter this conclusion. Two of the studies that reported negative results regarding social withdrawal used large samples representative of the general population (Cannon et al., 2002; Crow et al., 1995), which strengthen their findings. However, a similar design was employed by Jones and colleagues, who used the large British 1946 birth cohort for their investigation and also statistically controlled for sex and SES. Thus based on the reports available at this point, it seems unlikely that social isolation during the age period of 6 to 10 years is significantly elevated among children who later develop schizophrenia. However, due to the mixed findings combined with possible methodological issues noted, further research is needed to strengthen this conclusion.

*1.7.2.4 11 to 16 years of age.* Overall, the studies of Table 1b regarding this age period suggest that several types of behavioral features similar to the negative symptoms of schizophrenia may be detectable in pre-schizophrenia children when compared to controls, including aspects of passivity (Carter et al., 2002), decreased interest in the environment (Bower & Shellhamer, 1960), decreased facial expressions of joy and increased expressions of negative emotions among girls (Walker et al., 1993), and reduced interpersonal rapport (O'Neal & Robins, 1958; Parnas et al., 1982). Further, the results of Parnas et al. suggest that disturbed emotional rapport during this age period may also characterize children who later develop schizotypal personality traits.

Table 1b  
*Studies of Schizophrenia-like Behaviors and Symptoms: Negative Features*

Study	Sample	Behaviors/symptoms	Birth to 2 yrs old	3 to 5 yrs old	6 to 10 yrs old	11 to 16 yrs old	17 yrs and up old
<i>Selected samples: Child guidance clinic or child/adolescent treatment unit:</i>							
Michael et al., 1957	-male sch=10 -male nonsch diagnosis=14 -male ctrls=582	Per ratings of files from mean age of 9 (range=2-18 yrs old):  -introversion	---	---	-NS	---	---
O'Neal & Robins, 1958	-sch=28 -ctrls=57	Noted at median age of 13-14 (range=1.5 to 17 yrs old):  -cold, unaffectionate	---	---	---	-pre-sch > ctrls	---
Cannon, M. et al., 2001	-sch=59 -affective psychosis <sup>1</sup> =27 -ctrls=86	Per clinician ratings made regarding prior 12 months at mean age of 13-14:  -autism/social withdrawal	---	---	---	-pre-sch > ctrls; NS for affective psychosis	---
<i>Selected samples: Behavioral high risk:</i>							
Chapman et al., 1994	Among psychosis-prone <sup>2</sup> and ctrls: -psychosis <sup>3</sup> =13 -ctrls <sup>4</sup> =494	Assessed at mean age of approx. 20:  -Physical Anhedonia Scale <sup>5</sup>	---	---	---	---	-NS

Table 1b (continued)

Study	Sample	Behaviors/symptoms	Birth to 2 yrs old	3 to 5 yrs old	6 to 10 yrs old	11 to 16 yrs old	17 yrs and up
Yung et al., 2003; McGorry et al., 2002; PACE clinic	Among UHR <sup>6</sup> patients: -psychosis <sup>7</sup> =20 -nonpsychosis=29	Assessed monthly over period up to 12 mo at mean age of 19 on SANS <sup>8</sup> :					
		-SANS-total	---	---	---	---	-NS
		-SANS-affective flattening	---	---	---	---	-NS
		-SANS-alogia	---	---	---	---	-NS
		-SANS-avolition- apathy	---	---	---	---	-pre- psychosis> nonpsychosis
		-SANS-anhedonia- asociality	---	---	---	---	-trend for pre- psychosis> nonpsychosis

Table 1b (continued)

Study	Sample	Behaviors/symptoms	Birth to 2 yrs old	3 to 5 yrs old	6 to 10 yrs old	11 to 16 yrs old	17 yrs and up old
<i>Selected samples: Familial high risk:</i>							
Parnas et al., 1982; Copenhagen HR Project <sup>9</sup>	Among HR-sch: -sch=13 -borderline sch <sup>10</sup> =29 -ctrls=55	Parent describing child as baby when child mean age of 15:  -passive baby  Per clinician assessment at mean age of 15:  -incongruent facial expression <sup>11</sup>  -abnormal emotional rapport <sup>11</sup>  -“schizoid” <sup>11, 12</sup>  -negative contact scale (e.g., suspicious, withdrawn) <sup>13</sup>	-pre-sch > ctrls <sup>14</sup>	---	---	---	---
			---	---	---	-NS <sup>14</sup>	---
			---	---	---	-pre-sch > ctrls <sup>14</sup>	---
			---	---	---	-NS <sup>14</sup>	---
			---	---	---	-NS <sup>14</sup>	---
Carter et al., 2002; Copenhagen HR Project <sup>9</sup>	Among HR-sch and ctrls: -sch=33 -other mental illness <sup>15</sup> =132 -ctrls=128	Per teacher ratings at mean age of 15:  -passive behavior <sup>16</sup>	---	---	---	-pre-sch > ctrls <sup>14</sup>	---

Table 1b (continued)

Study	Sample	Behaviors/symptoms	Birth to 2 yrs old	3 to 5 yrs old	6 to 10 yrs old	11 to 16 yrs old	17 yrs and up
Ott et al., 2001; NYHRP	Among HR-sch, HR-mood, and ctrls: -sch-related psychosis <sup>17=9</sup> -mood disorder or ctrls=113	Videotaped interviews at mean age of 9 rated for:  -negative thought disorder <sup>18</sup>  -negative symptoms <sup>19</sup>	---	---	-pre-sch-related psychosis > other groups <sup>20</sup>  -pre-sch-related psychosis > other groups <sup>20</sup>	---	---
<i>Unselected samples: Samples of convenience:</i>							
Bower et al., 1960	-male sch=44 -male ctrls=44	High school teacher and counselor comments <sup>21</sup> on:  -lack of interest in environment  -apathy	---	---	---	-pre-sch > ctrls  -pre-sch > ctrls	---
Watt, 1978 <sup>22</sup>	-39 sch -101 ctrls	Per coded teacher comments in annual reports on students:  Introversion factor <sup>23</sup>	---	---	-NS for grades K-6	-boys: NS for grades 7-12; girls: pre-sch > ctrls for grades 7-12	---



Table 1b (continued)

Study	Sample	Behaviors/symptoms	Birth to 2 yrs old	3 to 5 yrs old	6 to 10 yrs old	11 to 16 yrs old	17 yrs and up
Walker et al., 1993	-sch=32 -unaff. sibs of sch=31	Childhood home movies rated for facial expressions of:					
		-joy	-boys: pre-sch < unaff sibs at 0-4 mo, <i>NS</i> at 4-12 mo; girls: <i>NS</i> at 0-4 mo, pre-sch < unaff sibs at 4-8 mo, <i>NS</i> at 8-12 mo	-boys: <i>NS</i> at 1-7 yrs; girls: pre-sch < unaff sibs at 1-4 yrs, <i>NS</i> at 4-7 yrs	- <i>NS</i> at 7-10 yrs	-boys: <i>NS</i> at 10-13 yrs, trend for pre-sch > unaff sibs at 13-16 yrs; girls: pre-sch < unaff sibs at 10-16 yrs	---
		-interest	---	-boys: <i>NS</i> at 0-4 yrs; girls: pre-sch > unaff at 0-4 yrs	- <i>NS</i> at 4-12 yrs	- <i>NS</i> at 12-16 yrs	---
		-negative affect <sup>24</sup>	---	-boys: pre-sch > unaff sibs at 0-4 yrs; girls: <i>NS</i> at 0-4 yrs	-boys: <i>NS</i> at 4-8 yrs, pre-sch > unaff sibs at 8-12 yrs; girls: <i>NS</i> at 4-12 yrs	-boys: <i>NS</i> at 12-16 yrs; girls: pre-sch > unaff sibs at 12-16 yrs	---
<i>Unselected samples: Population-based:</i>							
Hartmann et al., 1984	-male sch=24 <sup>25</sup> -male ctrls=48 <sup>25</sup>	Per ratings from multiple sources (e.g. child, teacher, parent interviews, records) when child age 10-17:					
		-flat affect/anhedonia	---	---	---	- <i>NS</i> <sup>26</sup>	---

Table 1b (continued)

Study	Sample	Behaviors/symptoms	Birth to 2 yrs old	3 to 5 yrs old	6 to 10 yrs old	11 to 16 yrs old	17 yrs and up
Jones et al., 1994; British 1946 birth cohort (NSHD)	-sch=30 -ctrls=4716	Per mother comments on child at ages 4 and 6:  -prefers solitary play	---	-pre-sch > ctrls <sup>27</sup>	-pre-sch > ctrls <sup>27</sup>	---	---
Crow et al., 1995, Done et al., 1994; British 1958 birth cohort (NCDS)	-sch=30-33 <sup>28</sup> -affective psychosis <sup>29</sup> =31 <sup>28</sup> -neurosis=67-70 -ctrls=1378-1385 <sup>28</sup>	Per teacher-rated Bristol Social Adjustment Guide <sup>30</sup> when child age 7 and 11:  -withdrawal <sup>31</sup>	---	---	-NS	-boys: NS; girls: presch > ctrls; NS for affective psychosis	---
Malmberg et al., 1998	-male sch =approx. 195 <sup>28</sup> -male other psychoses= approx. 193 <sup>28</sup> -male ctrls =approx. 48598-48714 <sup>28</sup>	Self report as part of assessment at conscription between age 18 and 20:  -fewer than two close friends  -never talked about personal things with other people	---	---	---	---	-pre-sch > ctrls; NS for other psychoses  -pre-sch > ctrls; NS for other psychoses

Table 1b (continued)

Study	Sample	Behaviors/symptoms	Birth to 2 yrs old	3 to 5 yrs old	6 to 10 yrs old	11 to 16 yrs old	17 yrs and up old
Cannon, M. et al., 2002; Dunedin Multi-disciplinary Health and Development Study	-schfm=36 -mania=20 -nonpsychotic anxiety or depr=278 -ctrls=642	Parent and teacher ratings made at ages 5 (parent only), 7, 9, 11 averaged:  -index of social isolation <sup>32</sup>	---	---	-NS <sup>27</sup>	---	---

*Note.* Table presents results for schizophrenia and/or psychosis-related diagnostic groups only; see text for discussion of results of other diagnostic groups in context of specificity questions. Significant group differences noted if  $p \leq .05$  as reported by authors; trend noted if  $p \leq .09$  (as able based on provided information).

approx=approximately; ctrls=controls; depr=depression; HR= high risk; mo=months; NCDS=National Child Development Study; nonsch=non-schizophrenia; NSHD=National Survey of Health and Development; NS=nonsignificant; NYHRP=New York High-Risk Project; SANS=Schedule for the Assessment of Negative Symptoms; sch=schizophrenia; schfm=schizophreniform; sibs=siblings; UHR=ultra-high-risk; unaff=unaffected; yrs=years

<sup>1</sup>included bipolar affective disorder and severe depression with psychotic symptoms.

<sup>2</sup>as defined by high scores on the Revised Physical Anhedonia Scale (Chapman et al., 1976), Perceptual Aberration Scale (Chapman et al., 1978), Magical Ideation Scale (Eckblad & Chapman, 1983), and/or the Impulsive Nonconformity Scale (Chapman et al., 1984).

<sup>3</sup>included schizophrenia, psychosis NOS, delusional disorder, and bipolar disorder with psychotic features.

<sup>4</sup>included all participants who did not meet criteria for psychosis as defined above but who may have met criteria for other disorders (e.g., personality and mood disorders).

<sup>5</sup>assesses experiences of sensory and aesthetic pleasures of eating, touching, feeling, sex, temperature, movement, smell, sight, sound.

<sup>6</sup>criteria for UHR: attenuated positive symptoms (presence of at least one of these symptoms: ideas of reference, odd beliefs or magical thinking, perceptual disturbance, paranoid ideation, odd thinking and speech, odd behavior and appearance; held with a reasonable degree of conviction and occurring at least several times a week) OR transient psychotic symptoms (presence of at least one of these symptoms but with duration of less than one week: hallucinations, delusions, formal thought disorder) OR first degree relative with psychotic disorder or schizotypal personality disorder and significant decline in mental state or functioning occurring within past year.

<sup>7</sup>included schizophrenia, schizophreniform disorder, schizoaffective disorder-depressed type, bipolar disorder with psychotic features, major depression with psychotic features, brief psychosis with duration greater than one week, and psychotic disorder NOS.

<sup>8</sup>Schedule for the Assessment of Negative Symptoms (Andreasen, 1982).

<sup>9</sup>Note that the two reports of Copenhagen HR Project (Carter et al. and Parnas et al.) used overlapping samples.

<sup>10</sup>This diagnosis corresponds to DSM-III diagnosis of schizotypal personality disorder.

<sup>11</sup>items selected from psychiatric interview.

<sup>12</sup>interviewer's global impression regarding participant being withdrawn or having "shut in" personalities.

<sup>13</sup>derived from following items of Adjective Checklist, rated by interviewer of psychiatric interview: tense, egoistic, peculiar, cautious, fearful, inhibited, introverted, shut in, awkward, distant, suspicious, reserved, self-insecure, shy, ambivalent, withdrawn, guarded.

<sup>14</sup>schizophrenia vs. controls.

<sup>15</sup>included atypical psychosis (8), schizoaffective disorder (1), schizophreniform psychosis (1), schizotypal personality disorder (41), paranoid personality (5), schizoid personality (1), depression (17), substance abuse (17), other Axis I disorders, and other Axis II disorders.

Table 1b (continued)

<sup>16</sup>This scale is based on teacher ratings of classroom behavior; comprised of these items: very shy, reserved, and silent; does not react to teacher's praise or criticism; normal activity level (-), low level of activity (i.e., passive), minimal emotional reactivity, rarely takes initiative/passively waits for teacher's instructions; rarely asks questions or participates in discussions/remains quiet and unengaged; rarely takes part in spontaneous activities despite encouragement from peers; seldom laughs and smiles with peers/remains serious.

<sup>17</sup>included schizophrenia, schizoaffective disorder-mainly schizophrenic, and unspecified psychosis.

<sup>18</sup>based on the Scale for Thought, Language, and Communication (Andreasen, 1986); characterized by poverty of linguistic production and expression.

<sup>19</sup>one of the two factors based on items from sections regarding motor behavior, speech, relatedness during the interview, and affect from the Mental Health Assessment Behavior Form (a psychiatric interview designed for the NYHRP).

<sup>20</sup>reported that pre-sch-related psychosis group elevated on scale but unclear if compared to other groups combined or separately; analyses controlled for age.

<sup>21</sup>retrospective interview in which interviewees unaware of purpose of study and both interviewees and interviewers blind to patient status.

<sup>22</sup>Results reported here are based on combined sample of this report.

<sup>23</sup>included these bipolar items: much group participation-little, popular-unpopular, sociable-unsociable, and talkative-quiet.

<sup>24</sup>expressions of anger, sadness, disgust, contempt, and fear combined.

<sup>25</sup>half of sample adjudicated delinquent.

<sup>26</sup>Cases and controls were matched on age at interview, IQ within 10 points, ethnicity, and delinquent-nondelinquent status.

<sup>27</sup>when controlling for sex and SES.

<sup>28</sup>*n* varied across variables or ages assessed.

<sup>29</sup>included mania, depressive psychosis, and "retarded" depression.

<sup>30</sup>Stott, 1987.

<sup>31</sup>included the traits distant, cut off from people, and avoids communication.

<sup>32</sup>parents and teachers rated child on statement "child is a loner" using 3-point scale; mean parent and teacher ratings averaged.

The above findings also more strongly suggest that social withdrawal is increased among pre-schizophrenia children during this age period compared to the prior age period discussed. Specifically, three of five studies found that their index of social withdrawal was significantly increased among pre-schizophrenia children (Cannon et al., 2001; Crow et al., 1995; Watt, 1978). Further, two of these reports (Cannon et al.; Crow et al.) suggest that this feature may be specific to pre-schizophrenia children compared to those who later develop mood disorder with psychotic features, although the findings of Crow et al. also suggest that girls who later develop a non-psychotic illness that is severe enough to require inpatient treatment may also display increased social withdrawal during this age period. Two of the studies discussed here used unselected and thus more representative samples (Crow et al.; Watt), and it is clear that two of the investigations (Cannon; Watt) used a control group that was matched on age, sex, and SES, which strengthen the weight of these positive findings regarding social withdrawal. Interestingly, the two investigations that used the more representative samples (Crow et al.; Watt) reported that social withdrawal was increased among the girls but not boys who later developed schizophrenia. The control group used by Watt (1978) was matched on sex, which diminishes the likelihood that such findings reflect that girls are more socially withdrawn than boys at this age, regardless of whether they later develop schizophrenia or not. Such findings, then, suggest that during this age period either this feature is more noticeable in pre-schizophrenia girls than boys, or that it characterizes girls but not boys, who later develop the disorder. Further, the one investigation of this age period that failed to detect significantly increased social withdrawal during this age period among children who later developed schizophrenia (Parnas, 1982) was a familial high-risk study and therefore may have

generalizability limitations. In light of these issues and the strengths of the studies reporting positive findings, it appears that social withdrawal behaviors may be increased during this age period in children, or at least in girls, who later develop schizophrenia.

In contrast, no evidence emerged that inappropriate or incongruent affect (Parnas et al., 1982) or ratings of flat affect and anhedonia combined, as measured by Hartmann and colleagues (1984), were significantly increased during this age period among children (boys only for Hartmann et al.) who later developed schizophrenia. However, because these findings come from single studies and the Parnas et al. study has possible generalizability issues, firm conclusions regarding these behavioral traits during this age period cannot be drawn here.

*1.7.2.5 17 years of age and older.* Two behavioral high-risk investigations (Chapman & Chapman, 1994; Yung et al., 2003) and one population-based study (Malmberg et al., 1998) listed in Table 1b assessed possible pre-onset negative symptoms during young adulthood. Yung and colleagues (2003) found that among young adults considered at ultra high risk for developing psychosis in the near future, aspects of avolition and apathy were associated with the development of psychosis within several months whereas flat affect and alogia were not (Yung et al., 2003). The findings of Malmberg et al.'s (1998) large-scale population-based study of Swedish males suggest that aspects of social isolation may be increased in young adults, or at least young adult males, who later develop schizophrenia, but not among those who develop other psychotic illnesses. In contrast, Chapman and colleagues (1994) found no support for a relation between aspects of physical anhedonia (see Table 1b) and later psychosis development.

*1.7.2.6 Summary of findings regarding negative features.* Although it is difficult to draw broad conclusions regarding several of the behavioral features assessed by the studies reviewed above due to the limited number of investigations examining these specific behaviors (e.g., rates

of negative- and positive-emotion facial expression, flat affect, alogia), some generalities can be noted. For example, the findings of Walker and colleagues (1993) in general suggest that pre-schizophrenia boys and girls may display higher rates of negative affect and lower rates of positive affect, respectively, compared to sibling controls, and that facial expression differences may be present as early as infancy and extend to varying degrees at least through age 16. In addition, although limited, the findings of Parnas and colleagues (1982) suggest that babies who go on to develop schizophrenia or schizotypal features may be more passive compared to controls. Further, although limited in number and design, a few studies point to possible increases in alogia-like symptoms (Ott et al., 2001), interpersonal rapport difficulties (O'Neal & Robins, 1958; Parnas et al., 1982), and passivity-like features (Bower et al., 1960; Carter et al., 2002) during middle childhood and/or early adolescence among pre-schizophrenia children. The behavioral high risk study of Yung and colleagues (2003) also suggest that features of avolition and apathy may be increased among ultra-high risk young adults who are within a few months of transitioning to full psychosis compared to those who do not develop full psychosis.

Aspects of social withdrawal were examined by a number of studies reviewed above. Overall, the findings point more clearly to increased social withdrawal behaviors among pre-schizophrenia children during early adolescence (Cannon et al., 2001; Crow et al., 1995; Watt, 1978) and young adulthood (Malmberg et al., 1998) compared to early or mid-childhood (Cannon et al., 2002; Crow et al.; Jones et al., 1994). However, the type and number of studies that examined such features varied by age group, which makes it more difficult to draw conclusions across these age periods. As noted, the findings of three studies suggest that social withdrawal features may be specific to pre-schizophrenia children or young adults relative to those who later develop other psychoses (e.g., mood disorder with psychotic features; Cannon et

al., 2001; Crow et al.; Malmberg et al.). Results also suggest that social withdrawal may be especially increased among girls who later develop schizophrenia, at least during the age period of 11 to 16 years (Crow et al.; Watt).

In conclusion, a number of studies suggest that various behavioral features similar to the negative symptoms of schizophrenia may be increased to a detectable degree across childhood in those who later develop schizophrenia. However, for most of these features (e.g., alogia, disturbed affect, passivity), the number of studies and their designs are limited, which underscores the need for additional investigations that examine such traits across childhood among representative samples. The results reviewed here most strongly point to possible increases in social withdrawal behaviors during adolescence and young adulthood. Again, however, the number of studies using large, representative samples and systematically-collected data are limited. Thus additional research will be useful in helping to determine more clearly if and when such abnormalities emerge among children who later develop schizophrenia, and to further investigate potential sex differences and questions of specificity regarding these traits.

### *1.8 Studies of Peer Rejection*

Various aspects of social functioning of children who later develop schizophrenia have long been of interest to researchers attempting to describe pre-onset features and behaviors of the disorder; however, such functioning has typically been assessed in a broad way, with measures employed rarely focusing exclusively on aspects of peer rejection. As acknowledged earlier, it is difficult to disentangle the interpersonal variable of peer rejection from person characteristics that might drive or be a reaction to such treatment by others. Despite this difficulty, the current study sought to focus specifically on peer rejection as a potential stressor that is happening “to”



the child rather than, or at least in addition to, being driven by the child. Thus this portion of the review only includes studies that clearly measured aspects of peer rejection. Thus it does not include investigations that assessed either vague or general aspects of peer relationships (e.g., relationship problems with other children as measured by Ambelas, 1992; Cannon et al., 2001) or behaviors that may have been associated with peer rejection (e.g., the sociability index of Jones et al., 1994) but did not include any exclusive measures of peer rejection or aspects thereof. Only four retrospective archival or prospective investigations (with the exception of Bower & Shellhamer, 1960, which used retrospectively but blindly collected information) were found that met these criteria; these studies are presented in Table 2 and reviewed below by age period.

#### *1.8.1 6 to 10 Years of Age*

As displayed in Table 2, Cannon and colleagues (2002) found that the children who were later diagnosed with schizophreniform disorder were significantly more likely than controls to be described by their parents and teachers as not much liked by other children when parent and teacher ratings at ages 5 (parent only), 7, 9, and 11 were averaged. Unfortunately, the authors did not report the results for each age separately, which obscures when among these early years their pre-schizophreniform disorder children were perceived as significantly less liked than control children. They further reported that those who were later diagnosed with either mania or a depressive or anxiety disorder were rated more highly than control children on this index of peer rejection. Such results suggest that on average from age 5 to 11 years, children who later develop schizophreniform disorder are more rejected by their peers than are control children as observed by parents and teachers, but that this difference is not specific to schizophreniform disorder compared to mania or anxiety/depressive disorders.

Table 2  
*Studies of Indices of Peer Rejection*

Study	Sample	Behaviors/symptoms	6 to 10 yrs old	11 to 16 yrs old	17 yrs and up
<i>Selected samples: Familial high risk</i>					
Olin et al., 1995; Copenhagen HR Project	Among HR-sch: -sch=30 -SPD <sup>1</sup> =39 -nonpsychotic disorders <sup>2</sup> =32 -ctrls=66	Teacher rated at mean age of 15.1 (range=9 to 20 yrs old):  -lonely and rejected by others	---	-pre-sch > ctrls <sup>3</sup> for males; NS for females	---
<i>Unselected samples: Samples of convenience:</i>					
Bower & Shellhamer, 1960	-male sch=44 -male ctrls=44	High school teacher and counselor comments <sup>4</sup> on:  -degree to which not liked or disliked by others	---	-pre-sch > ctrls	---
<i>Unselected samples: Population-based:</i>					
Malmberg et al., 1998	-male sch=approx. 195 -male other psychoses=approx. 193 -male ctrls=approx. 49,261	Self report as part of assessment at conscriptioin between age 18 and 20:  -unpopular with peers	---	---	-pre-sch > ctrls; NS for other psychoses

Table 2 (continued)

Study	Sample	Behaviors/symptoms	6 to 10 yrs old	11 to 16 yrs old	17 yrs and up
Cannon, M. et al., 2002; Dunedin Multi-disciplinary Health and Development Study	-schfm=36 -mania=20 -nonpsychotic anxiety or depr=278 -ctrls=642	Parent and teacher ratings made at ages 5 (parent only), 7, 9, 11 averaged: -“child is not liked by other children”	-pre-schfm > ctrls <sup>5</sup>	---	---

*Note.* Table presents results for schizophrenia and/or psychosis-related diagnostic groups only; see text for discussion of results of other diagnostic groups in context of specificity questions. Significant group differences noted if  $p \leq .05$  as reported by authors; trend noted if  $p \leq .09$  (as able based on provided information).

approx=approximately; ctrls=controls; depr=depression; HR= high risk; NS=nonsignificant; sch=schizophrenia; schfm=schizophreniform; SPD=schizotypal personality disorder; yrs=years

<sup>1</sup>included schizotypal and paranoid personality disorder.

<sup>2</sup>included nonpsychotic depression, substance abuse, and other nonpsychotic Axis I and II disorders.

<sup>3</sup>schizophrenia vs. controls.

<sup>4</sup>retrospective interview in which interviewees unaware of purpose of study and both interviewees and interviewers blind to patient status.

<sup>5</sup>when controlling for sex and SES.

### *1.8.2 11 to 16 Years of Age*

Two studies of Table 2 examined aspects of peer rejection during adolescence (Bower & Shellhamer, 1960; Olin et al., 1995). Overall, their findings suggest that as perceived by teachers, boys who later develop schizophrenia are less liked by their peers during adolescence compared to those who do not develop schizophrenia. Regarding the specificity of this difference, the results of Olin and colleagues (1995) suggest that at least among high-risk offspring, boys who later develop schizophrenia experience more peer rejection than those who develop schizotypal or paranoid personality disorders or a nonpsychotic psychiatric disorder. Due to the possible generalizability limitations of this study, however, as well as the limited number of investigations specifically examining the relation between peer rejection during adolescence and later schizophrenia or psychosis, additional research using samples more representative of the general population and further addressing specificity questions is needed before firm conclusions regarding this association can be drawn.

### *1.8.3 17 Years of Age and Up*

The only investigation of Table 2 to examine peer rejection in early adulthood is the population-based Swedish conscript study of Malmberg and colleagues (1998). Among their large all-male sample of army conscripts, they found that those who later developed schizophrenia endorsed the self-report item, “unpopular with peers” when assessed between the ages 18 and 20 more often than controls, whereas those who developed other psychoses did not. These findings suggest that young adults who later develop schizophrenia specifically compared to other (possibly less severe) psychotic syndromes more often feel disliked by their peers than those who do not develop psychosis.

#### *1.8.4 Summary of Findings Regarding Indices of Peer Rejection*

Although the number of studies reviewed here is limited, when viewed in combination, they suggest that increased peer rejection is perhaps experienced by at least boys who later develop schizophrenia or schizophreniform disorder compared to controls across childhood and into late adolescence/early adulthood. The results of Cannon et al.'s (2002) investigation, which examined girls and boys together, suggest that peer rejection in early and middle childhood is not restricted to pre-schizophreniform disorder boys, although it appeared that they did not test for sex differences on this feature specifically. Their study further suggests that increased peer rejection may also be experienced by children who later develop APD or depressive and/or anxiety disorders and thus may not be specific to pre-schizophreniform disorder children. Interestingly, the findings of Malmberg et al. (1998) indicate that at least by early adulthood, increased peer rejection before illness onset may be specific to schizophrenia compared to other psychotic disorders, such as mood disorder with psychosis. It is clear, however, that additional research using representative samples of multiple outcome groups followed across childhood and adolescence is needed to bolster these suggestion that peer rejection is increased among those who later develop schizophrenia, and to further explore questions of specificity regarding and possible sex differences in this association.

#### *1.9 Studies of Parent-Child Relationships*

Researchers have long looked to various aspects of parent-child interactions and parental characteristics to which individuals with schizophrenia were exposed as children for clues regarding when and how pre-schizophrenia children differ from their peers in the search for environmental factors that may increase risk for developing the illness, especially among those

thought to be at increased susceptibility for doing so (e.g., Goldstein, 1987; Tienari et al., 1989). As was the case for peer rejection, the present study was specifically concerned with characteristics of parent-child relationships that likely result in a degree of stress for the child and do not simply appear to reflect aspects of the child's functioning. Thus the review that follows does not include studies that appear to have measured only attitudes or behaviors of the child toward the parent (e.g., "overly dependent on mother" as measured by O'Neal & Robins, 1958; hostile or indifferent toward parents as measured by Hartmann et al., 1984). Further, because the focus of the present study is specifically on overly critical, intrusive, unaffectionate, or distant parenting styles specific to the child under study, the review does not include investigations that instead focused on parental diagnoses or psychopathology, early maternal separation (e.g., Mäki et al., 2003), parenting skills (Jones et al., 1994), or physical aspects of the environment (e.g., crowding, cleanliness, Jones et al., 1994) in relation to the later development of schizophrenia, although it is acknowledged that such factors likely influence parent-child relationships and/or the stress level of the children in the home. Six retrospective archival or prospective studies were found that met these criteria; they are presented in Table 3 and reviewed by age period below.

### *1.9.1 3 to 5 Years of Age*

As can be seen in Table 3, the only population-based study listed (Cannon et al., 2002) assessed early aspects of problematic mother-child interactions by examining ratings made on features of the mother's attitude or behavior toward her child during the child's age-3 assessment, (e.g., critical or negative evaluation of the child; see Table 3 for a complete list of items). Cannon et al. reported that the mothers of children who were later diagnosed with schizophreniform disorder were more likely to be rated as atypical on at least one of these

Table 3  
*Studies of Parent-Child Relationships*

Study	Sample	Behaviors/symptoms	3-5 yrs old	6 to 10 yrs old	11 to 16 yrs old
<i>Selected samples: Child guidance clinic or child/adolescent treatment unit:</i>					
Ricks & Berry, 1970	-sch=100 -ctrls=100	Family environment classifications via child guidance center files; initially assessed at median age of 14:			
		-parent overly intrusive and dominant, child isolated from non parental relationship (“symbiotic union”) <sup>1</sup>	---	---	-pre-sch > ctrls <sup>2</sup>
		-parent withdrawn and indifferent to child or vacillates from indifference to harshness often after loss of spouse or another child (“depressed environments”) <sup>1</sup>	---	---	-pre-sch > ctrls <sup>2</sup>
		-child openly rejected, not trusted, and asked or forced to leave home (“family sacrifice”) <sup>1</sup>	---	---	-pre-sch > ctrls <sup>2</sup>
		-evidence that child loved, cared for, and supported within normal limits (“mildly disturbed environment”) <sup>1</sup>	---	---	-pre-sch < ctrls <sup>2</sup>
Ambelas, 1992	-male sch=18 -male ctrls=18	Per child guidance center files, assessed mean age of 10 (range=6-15 yrs old):			
		-relationship problems with mother	---	-NS <sup>3</sup>	---
		-relationship problems with father	---	-NS <sup>3</sup>	---
		-relationship problems with stepfather	---	-NS <sup>3</sup>	---

Table 3 (continued)

Study	Sample	Behaviors/symptoms	3-5 yrs old	6 to 10 yrs old	11 to 16 yrs old
Cannon, M. et al., 2001	-sch=59 -affective psychosis=27 -ctrls=86	Per clinician ratings made regarding prior 12 months at mean age of 13-14:  -disturbed child-mother relationship  -disturbed child-father relationships		---	-NS  -NS
<i>Selected samples: Familial high risk:</i>					
Carter et al., 2002; Copenhagen HR Project <sup>4</sup>	Among HR-sch: -sch=32 -other mental illness <sup>5</sup> =94 -ctrls=70	Based on social worker and psychiatric interviews with parent and child respectively and mother's psychiatric records; child mean age of 15:  -maternal conflict scale <sup>6</sup>  -paternal conflict scale <sup>7</sup>	---	---	-NS <sup>8</sup>  -pre-sch > ctrls <sup>8</sup>
Schiffman et al., 2002; Copenhagen HR Project <sup>4</sup>	Among HR-sch: -sch=31 -SPD=36 -ctrls=68	Based on social worker and psychiatric interviews with parent and child respectively when child mean age of 15:  -mother-child and father-child relationships both classified as poor <sup>9</sup>	---	---	-pre-sch > ctrls <sup>8</sup>
<i>Unselected samples: Population-based:</i>					
Cannon, M. et al., 2002; Dunedin Multi-disciplinary Health and Development Study	-schfm=36 -mania=20 -nonpsychotic anxiety or depr=278 -ctrls=642	Per mother ratings by psychologist or doctor on attitude and behavior regarding child when child age 3:  -atypical mother-child interaction <sup>10</sup>	-pre-schfm > ctrls <sup>11</sup>	---	---



Table 3 (continued)

*Note.* Table presents results for schizophrenia and/or psychosis-related diagnostic groups only; see text for discussion of results of other diagnostic groups in context of specificity questions. Significant group differences noted if  $p \leq .05$  as reported by authors; trend noted if  $p \leq .09$  (as able based on provided information).

ctrls=controls; depr=depression; HR= high risk; NS=nonsignificant; sch=schizophrenia; schfm=schizophreniform; SPD=schizotypal personality disorder; yrs=years

<sup>1</sup> see Waring & Ricks (1965) for full descriptions of these “family environments.”

<sup>2</sup>cases and controls matched on age, sex, IQ, SES, ethnicity, and period seen at guidance center, and when possible, presenting symptoms.

<sup>3</sup>cases and controls matched on age, sex, time of referral within two months, and reason for referral.

<sup>4</sup>Note that the two reports of Copenhagen HR Project (Carter et al. and Schiffman et al.) used overlapping samples and measures.

<sup>5</sup>included atypical psychosis (8), schizoaffective disorder (1), schizophreniform psychosis (1), schizotypal personality disorder (41), paranoid personality (5), schizoid personality (1), depression (17), substance abuse (17), other Axis I disorders, and other Axis II disorders.

<sup>6</sup>comprised of these items from social worker interview with mother: extent to which child and mother get along; mother often scolds child; child tells mother about problems; child prefers to talk to father; extent to which family gets along; and these from psychiatric interview with child: child’s attitude toward mother; mother is high strung and easily upset; mother frequently shouts at and scolds the child or other members of the family; unusual amount of parental conflict.

<sup>7</sup>comprised of these items from social worker interview with mother: father’s stability at work; father has frequent job changes due to personal problems; child’s relationship with father; and these from psychiatric interview with child: child’s attitude toward father; frequency of contact with father; father is high strung and easily upset; unusual amount of parental conflict.

<sup>8</sup>schizophrenia vs. controls.

<sup>9</sup>used median split for Mother Scale (comprised of these items: attitude toward mother is not positive; child and mother do not get along well; mother often scolds child; child does not tell mother his/her troubles; frequency of contact with mother is not regular) and Father Scale (comprised of these items: attitude toward father is not positive; effect of contact with father is not positive; frequency of contact with father is not regular; father is described as weak, ineffective, reserved) to classify child’s relationship with mother and father as good or poor.

<sup>10</sup>mothers rated on 8 features: harshness toward child; critical or negative evaluation of child; rough, awkward handling of child; no effort to help child; unaware or unresponsive to child’s needs; indifference to child’s performance; demanding of the child’s attention; soiled, unkempt appearance of the child. Scores on ratings summed and a score of 1 or more used to indicate “atypical” mother-child interaction.

<sup>11</sup>when controlling for sex and SES.

behavioral features compared to those of the control children. In contrast, the mothers of children who were later diagnosed with either mania or a depressive or anxiety disorder did not significantly differ from those of control children. Such results suggest that early unfavorable mother-child interactions may be related to later schizophreniform disorder, and that this association may be specific to this disorder relative to mania and depressive/anxiety disorders.

### *1.9.2 6 to 10 Years of Age*

Using a selected sample of boys treated at a child guidance center at a mean age of 10 years, Ambelas (1992) found that according to guidance center records, those who were later hospitalized for schizophrenia were not significantly more likely than a matched group of control boys to have a documented relationship problem with their mother, father, or stepfather. The generalizability limitations of this study, the large age range during which these children were assessed (range=6-15 years), and the study's reliance on small samples and guidance center records lessen the weight of these negative findings, however.

### *1.9.3 11 to 16 Years of Age*

Three of the four studies listed in Table 3 that examined parent-child relationships during early to mid-adolescence reported an association between problematic aspects of these relationships and later schizophrenia (Carter et al., 2002; Ricks & Berry, 1970; Schiffman et al., 2002). Thus overall, it appears that these results by and large support the notion that problematic relationships with both mother and father during adolescence are associated with the later development of schizophrenia, with the results of Carter et al. only supporting a relation for poor father-child interactions and later schizophrenia among high-risk offspring. However, when considering the types of samples used in the three studies that reported this association, it

becomes less clear what types of conclusions can be drawn. Specifically, according to the report of Ricks and Berry (1970), over half of the individuals in their schizophrenia sample and 16% of their control sample had mothers who were considered either psychotic, “schizoid,” or “borderline psychotic” character disordered based on the child guidance center records of their children participants. This suggests that as a group their sample may have been at increased genetic risk for schizophrenia compared to individuals from the general population. As noted in Table 3, the reports of Carter et al. and Schiffman et al. are based on familial high-risk samples. Based on these considerations, it seems that the above results suggest that among individuals who are as a group at increased genetic risk for schizophrenia, problematic relationships with parents is associated with later schizophrenia. As discussed by Schiffman et al. (2001) and others, such findings are consistent with the hypothesis that nonoptimal rearing conditions may further increase risk for schizophrenia among those who are at increased susceptibility. Based on the nature of the above findings and the lack of studies employing representative samples when examining parent-child interactions during adolescence in relation to later schizophrenia, no conclusions can be drawn here regarding whether association between adverse parent-child relationships and later schizophrenia is reliably detectable among the general population.

An intriguing set of findings has emerged from the Finnish Adoptive Family Study of Schizophrenia (Tienari et al., 1994; Tienari et al., 2004). This set of studies is not listed in Table 3 because they report on schizophrenia-spectrum disorders more generally, and thus although their outcome group of interest included several individuals who developed schizophrenia, it also comprised a large number who met criteria for only Axis II spectrum disorders. However, their results are unique and relevant to the discussion at hand and thus will be considered briefly. Tienari and colleagues (2004) reported that among genetically high-risk but not low-risk

adoptees, problematic parent-child relationships within the adoptive family were predictive of the later development of schizophrenia-spectrum disorders. Importantly, pre-onset family ratings (mean ratings; see Tienari et al., 2004) did not discriminate the adoptive families who were rearing high- and low-risk children, which weakens the argument that such differential relations between family stressor exposure and psychopathology development are being driven solely by abnormalities of high-risk adoptees (a genotype-environment correlation). As the authors discuss, such results are consistent with the diathesis-stressor model of schizophrenia, and specifically implicate suboptimal rearing conditions as a deleterious environmental experience for those who are at increased susceptibility. These results are congruent with the findings of the Copenhagen High-Risk Project, reviewed above (e.g., Carter et al., 2002; Schiffman et al., 2002).

#### *1.9.4 Summary of Findings Regarding Parent-Child Relationships*

Although the conclusions that can be drawn from the above review are quite limited due to the small number of studies overall and especially those studies using representative samples, a few points can be made. First of all, the findings from the methodologically-strong population-based Dunedin study of Cannon and colleagues (2002) suggest that children who experience early (age 3) atypical or dysfunctional attitudes or behaviors directed toward them by their mother may be more likely to develop schizophreniform disorder but not mania or depressive/anxiety disorders. Further, as noted above, the findings from the Copenhagen High-Risk Project (Carter et al., 2002; Schiffman et al., 2002) suggest that among individuals at increased genetic or at least familial risk for schizophrenia, disturbed parent-child relationships are associated with the later development of the disorder. As noted, no evidence emerged from this limited review that a problematic relationship with a parent beyond the age of 3 is associated with later schizophrenia in the general population. It is clear that additional research examining

this question across childhood and adolescence among population-based samples is needed to further elucidate the nature of the relation between such family factors and the later development of schizophrenia.

### *1.10 Summary of Literature Review*

Overall then, the available literature regarding the proposed domains of focus is suggestive but generally limited with regard to the number of studies using representative samples. As noted, the paucity of research is especially striking with regard to possible relations between peer rejection or family factors and later psychosis, especially in light of recent research strongly implicating the role of psychosocial stressors in increasing risk among susceptible individuals (Tienari et al., 2004). Further, the specific findings reported by Poulton et al. (2000) regarding the relation of psychotic symptoms at age 11 and later schizophreniform disorder have important implications and thus are in need of replication. Moreover, relatively few of the existing studies of these domains have clearly examined questions of specificity, especially with regard to nonpsychotic diagnoses such as depressive and anxiety disorders.

### *1.11 The Current Study*

Thus in light of both the theoretical interests outlined earlier and the limited literature on schizophrenia-like abnormalities and psychosocial stressors as possibly predictive of later psychosis, the present study sought to characterize the associations between these potential antecedents and the later development of psychosis using a population-based, prospectively-followed sample. Its primary aims were to determine 1) if schizophrenia-like behavioral features and psychosocial stressor exposure observed during adolescence predict full psychotic symptoms

in early adulthood; 2) if such relations are specific to psychosis development relative to the development of antisocial personality disorder and depressive and/or anxiety disorders; and 3) if adolescent schizophrenia-like behavioral features moderate the relation between adolescent stressor exposure and early-adulthood psychotic symptoms.

Specifically, the current study used data from the Pittsburgh Youth Study, a prospective, population-based study of boys. Because of the limited number of individuals in the study sample who met criteria for schizophrenia, individuals who endorsed at least one psychotic symptom of at least one month's duration comprised the psychosis group; only a subset of these individuals met diagnostic criteria for schizophrenia or schizophreniform disorder, as described more fully below. Boys who did not endorse psychotic symptoms but met criteria for antisocial personality disorder (APD) comprised the APD group, whereas those meeting criteria for a nonpsychotic depressive and/or anxiety disorder made up the depression/anxiety group. As noted, these latter clinical groups were included so that questions of specificity could be addressed. The control group used for all analyses comprised boys who did not endorse any psychotic symptoms and did not meet criteria for APD or a depressive or anxiety disorder. Group differences were examined on the four primary antecedents of interest (i.e., schizophrenia-like positive symptoms, schizophrenia-like negative symptoms, peer rejection, and parent-child relationship) at each age from 13 to 17 years for a total of five age periods, which allowed for investigation of the longitudinal questions of interest.

The following primary hypotheses were tested:

*1.11.1 Hypothesis 1 – Schizophrenia-like Positive Symptoms:*

*1a. Relation to early adulthood psychosis.* The level of schizophrenia-like positive symptoms will be increased across the ages assessed among those of the psychosis group compared to the

well control group. Examination across age will reveal that the psychosis group increases significantly more across adolescence compared to controls (i.e. has a steeper positive slope and thus becomes increasingly deviant across adolescence relative to controls) on schizophrenia-like positive symptoms.

*1b. Specificity to early adulthood psychosis.*

-The level of schizophrenia-like positive symptoms will be increased across the ages assessed among those of the psychosis group compared to both the APD and depressive and/or anxiety disorder groups. Further, the psychosis group will increase significantly more across adolescence compared to both the APD and depressive and/or anxiety disorder groups on schizophrenia-like positive symptoms.

-The level of schizophrenia-like positive symptoms will not be increased across the ages assessed among those who developed APD or those of the depressive and/or anxiety group compared to controls.

*1.11.2 Hypothesis 2 – Schizophrenia-like Negative Symptoms:*

*2a. Relation to early adulthood psychosis.* The level of schizophrenia-like negative symptoms will be increased across the ages assessed among those of the psychosis group compared to the control group. Examination across age will reveal that the psychosis group increases significantly more across adolescence compared to controls on schizophrenia-like negative symptoms.

*2b. Specificity to early adulthood psychosis.*

-The level of schizophrenia-like negative symptoms will be increased across the ages assessed among those of the psychosis group compared to both the APD and depressive and/or anxiety

disorder groups. The psychosis group will increase significantly more across adolescence on this construct compared to both the APD and depressive and/or anxiety disorder groups.

-The level of schizophrenia-like negative symptoms will not be increased across the ages assessed among those who developed APD relative to the control group.

-The level of schizophrenia-like negative symptoms will be increased across the ages assessed among the boys of the depressive and/or anxiety group compared to controls. Additionally, the boys of the depressive and/or anxiety disorder group will increase significantly more across adolescence (i.e. will have a steeper positive slope) compared to the controls on schizophrenia-like negative symptoms.

### *1.11.3 Hypothesis 3 – Peer Rejection:*

*3a. Relation to early adulthood psychosis.* The level of peer rejection will be increased across the ages assessed among those of the psychosis group compared to controls, and the psychosis group will increase significantly more across adolescence on peer rejection compared to controls.

#### *3b. Specificity to early adulthood psychosis.*

-The level of peer rejection will be increased across the ages assessed among those of the psychosis group compared to both the APD and depressive and/or anxiety disorder groups.

Further, the psychosis group will increase significantly more across adolescence compared to both the APD and depressive and/or anxiety disorder groups on peer rejection.

-The level of peer rejection will be increased across the ages assessed among the boys of the APD and depressive and/or anxiety disorder groups compared to controls. Also, the boys of the APD and depressive and/or anxiety disorder groups will increase significantly more across adolescence compared to the controls on peer rejection.



*1.11.4 Hypothesis 4 – Problematic Parent-Child Relationship:*

*4a. Relation to early adulthood psychosis.* The level on the index of problematic parent-child relationship will be increased across the ages assessed among those of the psychosis group compared to controls, and the psychosis group will increase significantly more than controls across adolescence on the index of problematic parent-child relationship.

*4b. Specificity to early adulthood psychosis.*

-The level of problematic parent-child relationship will be increased across the ages assessed among those of the psychosis group compared to both the APD and depressive and/or anxiety disorder groups, and the psychosis group will increase significantly more across adolescence compared to the APD and depressive and/or anxiety disorder groups on this construct.

-The level of problematic parent-child relationship will be increased across the ages assessed among the boys of the APD and depressive and/or anxiety disorder groups compared to controls, and the APD and depressive and/or anxiety disorder groups will increase significantly more across adolescence compared to the controls on the index of problematic parent-child relationship.

*1.11.5 Hypothesis 5 – The Moderating Effect of Schizophrenia-like Symptoms:*

Schizophrenia-like symptoms will moderate the relation between stressor exposure and later psychosis. Specifically, there will be a significant interaction between psychosocial stressors (peer rejection and problematic parent-child relationship combined across ages) and schizophrenia-like symptoms (positive and negative combined across ages) in predicting psychosis.

## 2.0 METHOD

### *2.1 Participants and Overview of the Pittsburgh Youth Study*

The participants in this study were boys of the Pittsburgh Youth Study. The Pittsburgh Youth Study (PYS) is an ongoing prospective investigation designed to assess the development of and risk factors for delinquency, psychopathology, and drug use by studying a sample of boys through childhood, adolescence, and early adulthood (Loeber, Farrington, Stouthamer-Loeber, & Van Kammen, 1998). Its principal investigator is Rolf Loeber, and its co-investigators are Magda Stouthamer-Loeber, David P. Farrington, and Helene Raskin White (Loeber et al., 2002). The PYS began in 1987, at which time three samples of boys were randomly selected from the first, fourth, and seventh grades of the City of Pittsburgh public school system; approximately 1,100 boys were selected from each grade. Of the 3,436 selected, approximately 85% of the boys and their parents agreed to participate in the screening assessment. The goal of this initial screening was to identify boys who appeared to be at especially high risk for the development of delinquent and disruptive behaviors, and thus included an assessment of antisocial and other problem behaviors conducted with the boys, as well as their primary caretakers and teachers. Based on this screening, a risk score of antisocial and delinquent-like acts was created, and approximately 250 boys from each sample were randomly selected from those who scored in the top 30th% on this risk score (Loeber et al., 1998). An equal number of boys were then randomly selected from the remaining 70% of each sample. This yielded a total of approximately 500 boys each for the youngest, middle, and oldest samples. Thus half the boys of each sample is

considered to be at high risk for the development of delinquent behavior, and half at low risk. A data-weighting procedure based on this sampling strategy can be performed so that the results obtained are generalizable to the population from which this sample was drawn.

Slightly over half the boys in the follow-up samples were African American, and just under half were European American, a racial composition similar to that of the Pittsburgh public schools at the time. At initial follow-up, most of the boys lived with their biological mothers (92.6%). However, only 37.8% lived with their biological fathers, and 43.6% did not have a father figure in the household (Loeber et al., 1998). These demographic features of the follow-up samples did not differ significantly from the original screening samples. Further, the boys of the follow-up samples had standardized test reading scores that did not differ significantly from those obtained by children in the Pittsburgh public school system (Loeber et al., 1998).

Additional information on participant selection and sample characteristics are provided in Loeber et al. (1998).

### *2.1.1 Current Sample*

The participants of the current study were selected from the youngest and oldest samples of the PYS. Because the middle sample was regularly assessed only until age 13, its participants were not included in the present investigation. At the screening phase, the boys were a mean age of 6.5 and 12.8 years for the youngest and oldest samples respectively. The boys, their primary caretakers, and their teachers, were initially re-assessed every six months (9 six-month assessments for the youngest and 6 for the oldest), and then yearly thereafter (Loeber et al., 2002). The PYS investigators took great lengths to minimize attrition over the years and thus were able to maintain relatively high participation rates across phases. Approximately 83% of the youngest and oldest samples participated during the most recent assessment phase (Loeber et

al., 2002). Due to the low base rate of the outcome of interest (psychosis), the data for the two samples were combined by age; thus only data for ages at which both samples were assessed were included for the present study. Specifically, data on the antecedents of interest were examined from ages 13 to 17 for both samples.

### *2.1.2 Outcome Groups*

Outcome groups were formed based on diagnostic information obtained from the most recent administration (age 19 for the youngest and age 25 for the oldest samples) of the Diagnostic Interview Schedule for DSM-IV (DIS; Robins, Cottler, Bucholz, & Compton, 1998; DSM-IV, Diagnostic and Statistical Manual of Mental Disorders, 4<sup>th</sup> edition; American Psychiatric Association), a psychiatric interview designed to be administered by lay interviewers to facilitate its use in large, epidemiological studies. The DIS was administered by trained interviewers of the PYS. Symptoms that were deemed to be due to medical conditions, medications, or substance use did not count toward diagnostic criteria. Based on available data, boys with a history of epilepsy or convulsions without fever or who were mentally retarded were excluded from this study. The remaining boys were assigned into one of four groups based on their responses on the DIS, as described below.

*2.1.2.1 Psychosis.* The psychosis group comprised boys who endorsed at least one psychotic symptom (i.e. delusion or hallucination) that persisted for at least one month and was unexplained (i.e., there appeared to be no plausible explanation for the participant's experience or belief, and apparently it was not due to medication, a physical condition, or substance use). Table 4 lists the DIS items used to assess psychotic symptoms. The boys who endorsed at least one unexplained psychotic symptom but reported that they never experienced such symptoms most of the time for at least a month-long period were excluded from this study. Although this

group is theoretically interesting, based on power limitations, it was decided that it was best to exclude them rather than consider them as a fifth group. Further, because some of these boys may go on to develop more persistent psychotic symptoms, it was determined that it was best to exclude them rather than include them in the control group.

Interviewer ratings of flat affect, alogia, aspects of avolition, and aspects of disorganized speech were considered, along with the endorsement of psychotic symptoms, in determining whether any of these boys met criteria for schizophrenia, schizoaffective disorder, schizophreniform disorder, or other DSM-IV psychotic disorder. To further characterize the functional status of this group more generally, the symptom-related impairment and treatment information provided by the DIS was supplemented by various indices of functioning obtained from other sources collected by the PYS, including data regarding history of medical treatment and educational and occupational status.

*2.1.2.2 Antisocial personality disorder.* As noted, to address specificity questions, two other-disorder groups were created. The APD group included boys who as young adults met DSM-IV criteria for APD but did not endorse any psychotic symptoms. Boys meeting criteria for both APD and any other DSM-IV diagnosis for which they were assessed (excluding psychotic disorders) were included in the APD group.

*2.1.2.3 Depressive and/or anxiety disorders.* The “depression/anxiety” group comprised boys who met DSM-IV criteria for a depressive and/or anxiety disorder but did not endorse psychotic symptoms nor met criteria for APD. The specific diagnoses included were major depressive disorder without psychosis, dysthymia, panic disorder, agoraphobia, generalized anxiety disorder, social phobia, specific phobia, post-traumatic stress disorder, and obsessive-compulsive disorder.

Table 4

*Psychotic Symptom Items of the Diagnostic Interview Schedule for DSM-IV (DIS)*

---

*I. Delusional Beliefs:*

- H1. Have you ever believed that you were being secretly tested or experimented on?
- H2. Have you ever believed that someone was plotting against you or trying to hurt you or poison you?
- H3. Have you ever believed that somebody was spying on you? (if yes: How did you know it was happening?)
- H4. Was there ever a time when you believed somebody was following you?
- H5. Have you ever seen people you didn't know talking to each other and thought they were talking about you or laughing at you?
- H6. Have you ever believed that someone was reading your mind?
- H7. Have you ever believed that you could actually hear what another person was thinking, even though that person was not speaking?
- H8. Have you ever believed that others could hear your thoughts?
- H9. Have you ever believed that some person, power or force could control your movements or thoughts against your will?
- H10. Have you ever believed that someone or something could put thoughts that were not your own directly into your mind?
- H11. Have you ever felt that someone or something took or stole your thoughts from your mind?
- H12. Have you ever been convinced that someone you had not met was in love with you?
- H13. Have you ever believed that you were being sent special messages through the television or radio, or that a program had been arranged just for you alone?
- H13b. Did you ever believe that a newspaper, magazine, or song was meant only for you and no one else?
- H14. Have you ever felt strange forces working on you, as if you were being hypnotized or magic was being performed on you, or you were being hit by x-rays or laser beams?
- H15. Have you ever believed that you had done something terrible for which you should have been punished?

Table 4 (continued)

*II. Hallucination Experiences:*

H18. Have you ever had the experience of seeing things or a person that others who were present could not see -- that is, had a vision when you were completely awake? *Note:* this item does not count as psychotic symptom if only vision (s) is of recently deceased family member.

H19. Have you more than once had the experience of hearing things or voices other people couldn't hear? *Note:* this item does not count as psychotic symptom if only experience(s) is of occasionally hearing the voice of recently deceased loved one.

*If yes:*

H19d. Did you ever hear voices that others could not hear?

*If yes:*

H19e. Did you ever hear voices that other people couldn't hear that were commenting on what you were doing or thinking?

H19f. Did you ever hear voices telling you what to do?

H19g. Did you ever hear two or more voices talking to each other that other people couldn't hear?

H19h. Did you ever carry on a conversation with the voices -- when you spoke to them and they spoke to you?

H20. Have you ever been bothered by strange smells around you that nobody else seemed to be able to smell, perhaps even odors coming from your own body?

H21. Have you ever had unusual feelings inside or on your body -- like being touched when nothing was there or feeling something moving in your body?

H22. Have you ever tasted strange tastes in your mouth that were not from anything you had eaten?

---

*2.1.2.4 Well controls.* This group was made up of the boys who did not endorse any psychotic symptoms and did not meet criteria for the other-disorder groups as defined above.

## *2.2 Measures*

The current study used data collected from assessments completed by the boys, as well as their primary caretakers and teachers. The primary caretaker refers to the adult of the household

who claimed to have primary responsibility for the boy. In most cases, this was the biological, step, or adoptive mother. For the sake of simplicity, the current paper uses the term parent, mother, and primary caretaker interchangeably when discussing the data under study.

As noted, data on the four primary antecedents of interest were examined for each age from 13 to 17 years for a total of five age periods. Only participants who had data for at least three of the five age periods for all four primary constructs were included in the study.

### *2.2.1 Creating Constructs for Age Periods of Interest*

Because there was variation in the age of the participants at each assessment phase that at some phases spanned five years, constructs for the primary antecedents were based on the age at which participants were assessed rather than the actual phases of assessment to minimize the age variance of participants within each age period examined. Specifically, for each age of interest (i.e., age 13, age 14, etc., through age 17), the values of the relevant variables from the phase(s) at which the participant was the corresponding age were used. For example, for boys who were age 13 at the first and second assessment phases, values of the relevant variables from the first and second phases were used to create the age 13 constructs; for boys who were age 13 at the second and third phases, however, values from these phases were used to create the age 13 constructs. The “effective age” of each participant across assessment phases was used as the basis for these constructs. This age construct was created by using the participant’s actual age at the first follow-up assessment and then subtracting three months to yield the effective age value for this follow-up; 6 (and then later 12) months were then added to this value for each subsequent phase to obtain the effective age of the participants for later phases. Effective age



rather than actual age was used because the effective age construct corrects for unevenly-spaced assessment dates from phase to phase and thus ensures that participants have data for each age for which they were assessed.

### 2.2.2 *Constructs of the Primary Antecedents of Interest*

Three of the four constructs used to assess the antecedents of interest were created using items from the Child Behavior Checklist (CBCL; Achenbach & Edelbrock, 1983), the Teacher's Report Form (TRF; Achenbach & Edelbrock, 1986), and the Youth Self Report (YSR; Achenbach & Edelbrock, 1987). The CBCL, TRF, and YSR are generally parallel scales that are administered to parents, teachers, and children, respectively. Respondents are asked to indicate whether each behavior is *not true (0)*, *somewhat or sometimes true (1)*, or *very true or often true (2)* of the child. The versions both of the CBCL and TRF used by the PYS contained several supplemental items concerning delinquent behaviors. Research has indicated that the three informants of these scales (i.e., parents, teachers, and children themselves) often do not agree on the degree of problematic behavior experienced by the child (e.g., Youngstrom, Loeber, & Stouthamer-Loeber, 2000), which suggests that the different informants may provide information on behavioral difficulties that are more evident in certain contexts. Therefore, responses from all three informants (child, parent, and teacher) were summed when possible to create constructs based on the CBCL and related scales. As discussed by Youngstrom and colleagues (2000), however, various informants may differ with regard to how they approach such rating scales (e.g., teachers may tend to use a wider range of the responses available based on their exposure to a wider range of child functioning than do parents). To address this issue, for each scale used, responses to items for a given construct were standardized (using the entire study sample) within scale before summing across informants to create the construct. Specifically, to create age-

period data, informant-specific responses were summed across the items comprising the given construct for each age and then standardized. The values obtained using this procedure for child, parent, and teacher informants were then summed. Because this procedure may result in a greater degree of variance for constructs for which inter-rater responses are particularly correlated, the construct sum was also standardized to facilitate group comparisons across constructs and ages. Because no TRF (teacher) data were available for use after the age of 16, age 17 values for the CBCL-derived constructs were created from parent and child ratings only.

*2.2.2.1 Schizophrenia-like Positive Symptoms.* The six corresponding items from the CBCL, TRF, and YSR that assessed possible hallucination-like experiences, oddity, or suspiciousness were chosen for the schizophrenia-like positive symptoms construct. These items are listed in Table 5. Participants were considered missing at a given age for this construct if they did not have data for at least four of the six items from the parent, teacher, or child informant. Beginning at the phase at which the boys were a mean age of 16, teacher items were unavailable for this construct; thus beginning at this phase, participants were considered missing if they did not have data for at least four of the six items from the parent or child informant.

*2.2.2.2 Schizophrenia-like Negative Symptoms.* The CBCL and related-scale items chosen for this construct included those that for the most part assessed aspects of social withdrawal; see Table 5 for a complete list of items. Participants were considered missing at a given age for this construct if they did not have data for at least four of the five items from the parent or at least three of the four items from the teacher or child. Beginning at the phase at which the boys were a mean age of 16, participants were considered missing if they did not have data for at least four of the five items from the parent or at least two of the three items from the teacher or at least three of the four items from the child. Teacher items were unavailable for this

Table 5  
*List of Variables by Construct*

---

***Schizophrenia-like Positive Symptoms***

Made from the following corresponding items from YSR, CBCL, and TRF (0=not true; 1=somewhat or sometimes true; 2=very true or often true):

- 34. You feel that others are out to get you/Feels others are out to get him
- 40. You hear things that no one else seems to hear (at later phases worded as: You hear sounds or voices that other people think aren't there)/Hears things that aren't there
- 70. You see things that nobody else seems able to see/Sees things that aren't there
- 84. You do things other people think are strange/Strange behavior
- 85. You have thoughts that other people would think are strange/Strange ideas
- 89. You are suspicious/Suspicious

*Note.* Beginning at the phase at which the boys were a mean age of 16, teacher responses for these items became unavailable. Thus this construct did not include teacher items for ages 16 or 17.

---

***Schizophrenia-like Negative Symptoms***

Made from the following corresponding items from YSR, CBCL, and TRF (0=not true; 1=somewhat or sometimes true; 2=very true or often true):

- \*42. You like to be alone/Likes to be alone
- 65. You refuse to talk/Refuses to talk
- \*\*88. You enjoy being with others (boy only)
- 111. You keep from getting involved with others/Withdrawn, doesn't get involved with others
- 80. Stares blankly (parent and teacher only)
- 184. Spends long periods of time alone (parent only)

*Note.* Beginning at the phase at which the boys were a mean age of 17, teacher responses for these items became unavailable. Thus this construct did not include teacher items for age 17.

\*Teacher responses for this item became unavailable beginning at the phase at which the boys were a mean age of 16.

\*\*All items were scored so that higher scores reflected more potentially problematic behavior

---

Table 5 (continued)

***Peer Rejection***

Made from the following corresponding items from YSR, CBCL, and TRF (0=not true; 1=somewhat or sometimes true; 2=very true or often true):

- 12. You feel lonely/Complains of loneliness
- 38. You get teased a lot/Gets teased a lot
- 48. You are not liked by other kids/Not liked by other children/pupils

*Note.* Beginning at the phase at which the boys were a mean age of 16, teacher items for this construct were not included, as most became unavailable at this phase. Thus this construct did not include teacher items for ages 16 or 17.

---

***Parent-Child Relationship***

Bad Relationship with Primary Caretaker – Parent items (1=almost never; 2=sometimes; 3=often)

- Thought your child was a good kid
- Felt proud of him
- Felt like you needed a vacation from him
- Wished you had never had him
- Got along with him
- Thought he was a difficult child
- Thought he was good company
- Felt he was an easy child
- Felt he was an affectionate child
- Felt he was a troublemaker
- Enjoyed spending time with him
- Wished he would just leave you alone
- Lost patience with him
- Enjoyed being his parent
- Felt he needed too much attention
- Felt he was a happy child

Table 5 (continued)

Bad Relationship with Primary Caretaker – Child items (1=almost never; 2=sometimes; 3=often)

\*Items for Child Informant Scale, Version A:

- Felt primary caretaker was really good
  - Felt primary caretaker bugged you
  - \*\* -Felt proud of primary caretaker
  - \*\* -Wished you had a different primary caretaker
  - Felt primary caretaker gave you problems
  - Felt primary caretaker was easy to get along with
  - Felt primary caretaker loved you
  - Felt primary caretaker was too strict or hard on you
  - Wished primary caretaker would just leave you alone
  - Liked being kid of primary caretaker
  - Felt primary caretaker was happy
  - Felt primary caretaker meant what he/she said
  - Felt primary caretaker gave you punishment you deserved
- 

\*Items for Child Informant Scale, Version B:

- Felt you really liked your mother/father
- Felt that your mother/father loved you
- Felt angry at your mother/father
- Felt that you could go to your mother/father in an emergency
- Felt close to your mother/father
- Felt that if you were in trouble, you could tell your mother/father
- Thought that your mother/father bugged you a lot
- Felt that your mother/father was easy to get along with

Table 5 (continued)

- Felt that your mother/father was too demanding
- Felt that your mother/father did not understand you
- Felt that it was easy to talk to your mother/father
- Felt that it was better to avoid your mother/father
- Felt that your mother/father belittled you or put you down
- Felt that you could discuss your problems with your mother/father
- Had a fight with your mother/father

*Note.* All items were scored so that higher scores reflected more problematic parent-child relationships.

\*Because constructs were based on the age at which participants were assessed rather than the actual phases of assessment, for a subset of participants data from Version B of the child informant scale contributed to the Parent-Child Relationship construct for ages 15, 16, and/or 17 rather than items from Version A.

\*\*For a subset of participants, the items from Version B of the child informant scale plus these two additional items contributed to the Parent-Child Relationship construct for ages 16 and/or 17 rather than items from Version A; this set of items is referred to as Version C of the child informant scale.

---

*Note.* CBCL=Child Behavior Checklist; TRF=Teacher's Report Form; YSR=Youth Self Report

construct beginning at the phase at which the boys were a mean age of 17; thus beginning at this phase, participants were considered missing if they did not have data for at least four of the five items of this construct from the parent or least three of the four items from the child.

*2.2.2.3 Peer Rejection.* Because peer rejection that is happening to the child and self-imposed social isolation or withdrawal, which was measured as an aspect of schizophrenia-like negative features, may in some cases be difficult to distinguish by external raters (e.g., teachers and parents), the items used for this domain were very carefully selected in an effort to minimize its potential blurring with aspects of social isolation. Table 5 notes the three items selected from

the CBCL, TRF, and YSR that were thought to most clearly assess this construct. Participants were considered missing at a given age for this construct if they did not have data for at least two of the three items from the parent, teacher, or child. Beginning at the phase at which the boys were a mean age of 16, teacher items for this construct were not included, as most became unavailable at this phase; thus beginning at this phase, participants were considered missing if they did not have data for at least two of the three items from the parent or child.

*2.2.2.4 Parent-Child Relationship.* For this domain, an established construct developed by the PYS was used (e.g., Loeber et al., 2000; Stouthamer-Loeber, Loeber, Wei, Farrington, & Wikström, 2002). This construct is called “bad relationship with primary caretaker,” and was available for both child and parent informants. The child construct consists of 13 to 17 items asking about the boy’s perception of his primary caretaker and their relationship, and the parent version is made up of 16 items about her perception of her child and their relationship. The response format for both parent and child was: *almost never (1), sometimes (2), or often (3)*. See Table 5 for a full list of items for both informants. Following the rationale above regarding the benefits and cautions of using multiple informants, both parent and child versions of this construct were combined for the present study using the same strategy described above to obtain standardized scores for the age periods of interest. Participants were considered missing at a given age for this construct if they did not have data for at least 12 of the 16 items from the parent or at least 10 of the 13 items from the child when Version A of the child informant scale was used, 11 of the 15 items from the child when Version B of the child informant scale was used, and at least 12 of the 17 items from the child when Version C of the child informant scale was used (see Table 5).

## 3.0 RESULTS

### *3.1 Sample Characteristics*

As displayed in Table 6, diagnostic information from the most recent DIS administration of the PYS (approximately age 19 for the youngest and age 25 for the oldest samples) was available for 832 boys; thus these boys were considered for inclusion in the present study. However, 99 of these boys were excluded because 1) data were missing or incomplete regarding duration or some other aspect of psychotic symptoms (5 boys), 2) they endorsed at least one psychotic symptom but reported that such symptom(s) never persisted for at least one month (15 boys), 3) available data suggested a history of epilepsy, convulsions without fever, or mental retardation (29 boys), or 4) data were missing for any one of the primary constructs at more than two age periods (50 boys); see Table 6 for detailed information regarding sample attrition. These exclusions resulted in a final unweighted sample of 733 boys. As noted earlier, a data-weighting procedure based on the initial sampling strategy of the PYS can be performed so that the sample more closely resembles the population from which the sample was originally drawn. This procedure was performed for all analyses of the present study except when noted otherwise and resulted in a final weighted sample of 737 boys. See Table 6 for the unweighted and weighted *ns* of the sample by outcome group.



Table 6  
Sample Attrition

	Youngest sample	Oldest sample	Total	
<i>N</i> of full follow-up sample	503 ↓	506 ↓	1009 ↓	
DIS data at age 19 for youngest sample and age 25 for oldest?	424 = yes (79 = no) ↓	408 = yes (98 = no) ↓	832 ↓	
DIS data regarding duration of psychotic symptoms (if applicable) complete?	420 = yes (4 <sup>1</sup> = no) ↓	407 <sup>2</sup> = yes (1 = no) ↓	827 ↓	
Either did not endorse psychotic symptom(s) on DIS or reported that such symptom(s) persisted for at least one month?	414 <sup>3</sup> = yes (6 = no) ↓	398 = yes (9 = no) ↓	812 ↓	
Absence of a history of epilepsy, convulsions without fever, or mental retardation?	396 = yes (18 = no) ↓	387 = yes (11 = no) ↓	783 ↓	
Data for at least 3 of the 5 ages examined <sup>4</sup> for Schizophrenia-like Positive Symptoms <sup>5</sup> , Schizophrenia-like Negative Symptoms <sup>6</sup> , Peer Rejection <sup>7</sup> , and Parent-Child Relationship <sup>8</sup> ?	376 = yes (20 = no) ↓	357 = yes (30 = no) ↓	733 ↓	
<b>Final sample</b>	<b>376</b>	<b>357</b>	Unweighted: <b>733</b>	Weighted: <b>737</b>
-Psychosis group	9	8	17	16 (2.1%)
-APD group	22	39	61	52 (7.1%)
-Depression/anxiety group	8	12	20	22 (3.0%)
-Control group	337	298	635	647 (87.8%)

*Note.* APD= antisocial personality disorder; DIS= Diagnostic Interview Schedule for DSM-IV; DSM-IV=The Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition

<sup>1</sup>Two of these participants did not endorse any history of psychotic symptoms on the DIS; however, for one of these cases, another source of information suggested that he had a diagnosis of schizophrenia, and for the second case, another source of information indicated that he had been hospitalized for schizophrenia; thus both of these cases were excluded altogether due to this conflicting information.

Table 6 (continued)

<sup>2</sup>One of these participants endorsed psychotic symptoms on DIS but did not have duration of psychotic symptoms data available; however, based on interviewer comments noting that participant reported command auditory hallucinations and delusional beliefs and that he was on antipsychotic medication, this participant was included in the psychosis group rather than excluded.

<sup>3</sup>One of these participants reported psychotic symptoms on DIS but did not endorse persistence of psychotic symptoms for at least one month; however, based on interviewer comments based on age 20 interview noting that participant was on antipsychotic medication and had been hospitalized multiple times for psychiatric reasons, he was included in the psychosis group rather than excluded.

<sup>4</sup>Participant was required to have data for 3 of the 5 ages examined (i.e., 13, 14, 15, 16, and 17) for all four of the primary antecedents (i.e., schizophrenia-like positive symptoms, schizophrenia-like negative symptoms, peer rejection, and parent-child relationship) to be included.

<sup>5</sup>Participant was considered missing at a given age for Schizophrenia-like Positive Symptoms if he did not have data for at least 4 of the 6 items of this construct for parent or teacher or child. Beginning at the phase at which the boys were a mean age of 16, teacher items were unavailable for this construct; thus beginning at this phase, participant was considered missing if he did not have data for at least 4 of the 6 items for parent or child. This construct did not include teacher items for ages 16 or 17.

<sup>6</sup>Participant was considered missing at a given age for Schizophrenia-like Negative Symptoms if he did not have data for at least 4 of the 5 items of this construct for parent or at least 3 of the 4 items for teacher or child. Beginning at the phase at which the boys were a mean age of 16, participant was considered missing if he did not have data for at least 4 of the 5 items for parent or at least 2 of the 3 items for teacher or at least 3 of the 4 items for child. Teacher items were unavailable for this construct beginning at the phase at which the boys were a mean age of 17; thus beginning at this phase, participant was considered missing if he did not have data for at least 4 of the 5 items for parent or at least 3 of the 4 items for child. This construct did not include teacher items for age 17.

<sup>7</sup>Participant was considered missing at a given age for Peer Rejection if he did not have data for at least 2 of the 3 items of this construct for parent or teacher or child. Beginning at the phase at which the boys were a mean age of 16, teacher items for this construct were not included, as most became unavailable at this phase; thus beginning at this phase, participant was considered missing if he did not have data for at least 2 of the 3 items for parent or child. This construct did not include teacher items for ages 16 or 17.

<sup>8</sup>Participant was considered missing at a given age for Parent-Child Relationship if he did not have data for at least 12 of the 16 items of this construct for parent or at least 10 of the 13 items for child when Version A of the child informant scale was used, 11 of the 15 items of this construct for child when Version B of the child informant scale was used, and at least 12 of the 17 items of this construct for child when Version C of the child informant scale was used (see Table 5).

### *3.1.1 Selective Attrition Analyses*

To assess whether the boys who were excluded due to missing data differed from the boys comprising the final sample, the attrited and final samples were compared on several demographic characteristics and on diagnostic outcome. As displayed in Table 7, the attrited and final samples did not differ significantly on the number of boys classified as high risk according to the initial screening of the PYS nor on the proportion of boys from the youngest (versus oldest) sample. However, the attrited boys were less likely to be of European-American descent than were those of the final sample, and the parents of the attrited had significantly fewer years of education and lower socioeconomic status (SES) compared to the parents of the final sample boys. As shown in Table 7, the subset of attrited boys for whom there was diagnostic information from the most recent DIS administration did not differ from the final sample on diagnostic group composition.

### *3.1.2 Demographic Characteristics*

Table 8 presents demographic characteristics of the diagnostic outcome groups, and Table 9 displays the results of the pairwise group comparisons on these characteristics. Given the questions of this study, the following presentation of results focuses on the pairwise group comparisons of theoretical interest.

*3.1.2.1 Psychosis versus controls.* As displayed in the second column of Table 9, the psychosis group did not differ significantly from controls on the proportion of boys from the youngest sample, the proportion from the high risk group, age at which the DIS was administered, ethnicity, years of parental education, or parental SES.

Table 7  
*Demographic Characteristics and Diagnostic Outcome of Attrited Versus Final Sample*

Characteristic	Final sample ( <i>n</i> =737) <sup>1</sup>	Attrited sample ( <i>n</i> =223) <sup>1</sup>	$\chi^2$	<i>p</i> <sup>2</sup>
Sample (% from youngest)	51.0%	44.2%	3.00	.083
Risk group (% from high risk)	36.2%	38.6%	.402	.526
Ethnicity (% European American <sup>3</sup> )	44.1%	35.9%	4.75	.029
Parental education <sup>4</sup> (years; <i>M</i> ( <i>SD</i> ))	12.88 (2.02)	12.36 (1.79)	10.62 <sup>5</sup>	≤.001
Parental SES <sup>6</sup> ( <i>M</i> ( <i>SD</i> ))	37.57 (12.23)	34.10 (11.16)	12.77 <sup>5</sup>	<.001
	Final sample ( <i>n</i> =737) <sup>1</sup>	Attrited with DIS ( <i>n</i> =51) <sup>1</sup>		
Diagnostic Outcome <sup>1</sup>			2.70	.440
Psychosis	16 (2.1%)	2 (3.7%)		
APD	52 (7.1%)	5 (9.4%)		
Depression/Anxiety	22 (3.0%)	3 (5.3%)		
Controls	647 (87.8%)	41 (81.7%)		

*Note.* APD= antisocial personality disorder; DIS= Diagnostic Interview Schedule for DSM-IV; DSM-IV=The Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition; M=mean; SD=standard deviation; SES=socioeconomic status

<sup>1</sup>weighted *n*.

<sup>2</sup>two-tailed *p* values for  $\chi^2$ .

<sup>3</sup>remainder predominantly African American; specifically for final: 52.9% African American, 1.7% biracial, 1.0% Asian, 0.3% Hispanic, and 0.1% American Indian; for attrited: 63.2% African American, 0.5% Asian, and 0.3% biracial.

<sup>4</sup>caretaker or partner education, whichever higher, as assessed when boy was approximately age 13.

<sup>5</sup> $\chi^2$  from logistic regression, which was used to assess group differences because the data for this variable were non-normally distributed.

<sup>6</sup>based on Hollingshead's Factor (1975; 6=lowest, 66=highest); value reflects caretaker or partner SES, whichever higher, as assessed when boy was approximately age 13.

3.1.2.2 *Psychosis versus APD.* There were no significant differences between the psychosis and APD groups on the demographic characteristics displayed in Table 9.

3.1.2.3 *Psychosis versus depression/anxiety.* Likewise, no significant differences were found between the psychosis and depression/anxiety groups on the characteristics examined.

3.1.2.4 *APD versus controls.* The boys of the APD group were significantly older than those of the control group when the DIS was administered, and their parents had fewer years of education and lower SES than the control parents. Further and not surprisingly, those of the APD group were more likely to come from the high risk group than were controls.

3.1.2.5 *Depression/Anxiety versus controls.* The depression/anxiety group did not differ from controls on any of the demographic characteristics examined.

3.1.2.6 *Covariates for primary analyses.* Because the characteristics listed in Table 9 could theoretically explain variance in the primary constructs examined in this study, those that differed significantly by diagnostic group were considered for use as covariates in the primary analyses, with the exception of risk group. As high risk group status was in part determined by the presence of childhood behaviors that are included in the diagnostic criteria of APD and thus in part reflects important features of APD, it was deemed inappropriate to use this characteristic as a covariate in comparisons involving the APD group. Likewise, it is possible that years of parental education and/or parental SES may influence risk for the disorders of this study and/or share a causal influence(s) with the disorders. Similarly, because for some boys, being older when the DIS was administered (and thus older at the initiation of the study) resulted from being held back a grade(s) in school, age at DIS may in some cases reflect difficulties associated with increased risk for these disorders. Thus these three characteristics were not used as covariates in the primary group comparisons. However, to further our understanding of the possible

Table 8  
*Demographic Characteristics of Diagnostic Outcome Groups*

Characteristic	Psychosis ( <i>n</i> =16) <sup>1</sup>	APD ( <i>n</i> =52) <sup>1</sup>	Depression/ Anxiety ( <i>n</i> =22) <sup>1</sup>	Controls ( <i>n</i> =647) <sup>1</sup>
Sample (% from youngest)	53.4%	40.7%	36.1%	52.2%
Age at DIS ( <i>M</i> ( <i>SD</i> ))	21.86 (3.76)	22.77 (3.56)	22.89 (3.43)	21.77 (3.46)
Risk group (% from high risk)	51.0%	58.3%	23.1%	34.5%
Ethnicity				
% European American	53.6%	42.7%	53.2%	43.7%
% African American	41.3%	51.1%	37.1%	53.8%
% Hispanic	0%	0%	0%	0.3%
% Asian	0%	0%	3.6%	1.0%
% biracial	5.1%	6.2%	6.1%	1.1%
% American Indian	0%	0%	0%	0.1%
Parental education – academic degree <sup>2</sup>				
% no high school diploma or degree	14.3%	19.8%	13.0%	10.2%
% GED or high school diploma	64.3%	63.8%	31.7%	59.0%
% Associate’s degree	17.3%	4.2%	22.5%	13.3%
% Bachelor’s degree	0%	4.1%	9.6%	6.3%
% advanced degree	0%	1.3%	16.8%	6.3%
% other	4.1%	6.8%	6.5%	5.0%
Years of parental education <sup>2</sup>	12.23 (2.03)	12.15 (1.92)	13.59 (2.28)	12.93 (2.01)
Parental SES <sup>3</sup> ( <i>M</i> ( <i>SD</i> ))	39.33 (10.67)	33.20 (11.75)	40.62 (18.53)	37.77 (12.00)

*Note.* APD= antisocial personality disorder; DIS= Diagnostic Interview Schedule for DSM-IV; DSM-IV=The Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition; M=mean; SD=standard deviation; SES=socioeconomic status

<sup>1</sup>weighted *n*.

<sup>2</sup>caretaker or partner education, whichever higher, as assessed when boy was approximately age 13.

<sup>3</sup>based on Hollingshead’s Factor (1975; 6=lowest, 66=highest); value reflects caretaker or partner SES, whichever higher, as assessed when boy was approximately age 13.

Table 9  
*Demographic Characteristics: Diagnostic Group Comparisons*

Characteristic	Psychosis v. Ctrls	Psychosis v. APD	Psychosis v. Depression/Anxiety	APD v. Ctrls	Depression/Anxiety v. Ctrls
	$\chi^2$ ; <i>p</i>	$\chi^2$ ; <i>p</i>	$\chi^2$ ; <i>p</i>	$\chi^2$ ; <i>p</i>	$\chi^2$ ; <i>p</i>
Sample (youngest or oldest)	.007; .993	.795; .373	1.05; .306	2.71; .100	2.15; .143
Age at DIS <sup>1</sup>	.010; .922	.782; .376	.793; .373	<b>4.00; .046</b> (APD > Ctrls)	2.25; .134
Risk group (low or high)	1.66; .198	.294; .588	3.06; .080	<b>11.24; .001</b> (APD > Ctrls)	1.31; .253
Ethnicity (European American or other <sup>2</sup> )	.548; .459	.572; .449	.005; .942	.040; .841	1.01; .315
Years of parental education <sup>1,3</sup>	1.90; .168	.019; .890	3.55; .060	<b>7.06; .008</b> (APD < Ctrls)	2.12; .146
Parental SES <sup>1,4</sup>	.259; .611	3.58; .059	.065; .799	<b>6.80; .009</b> (APD < Ctrls)	1.10; .294

*Note.* APD= antisocial personality disorder; Ctrls=controls; DIS= Diagnostic Interview Schedule for DSM-IV; DSM-IV=The Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition; SES=socioeconomic status; v=versus

All *p* values are two tailed.

<sup>1</sup> $\chi^2$  from logistic regression was used to assess group differences on continuous demographic variables because the data from several of these variables were non-normally distributed.

<sup>2</sup>the “other” group was predominantly African American.

<sup>3</sup>caretaker or partner education, whichever higher, as assessed when boy was approximately age 13.

<sup>4</sup>based on Hollingshead’s Factor (1975; 6=lowest, 66=highest); caretaker or partner SES, whichever higher, as assessed when boy was approximately age 13.

associations between diagnostic group status and the primary constructs, analyses indicating significant differences on the primary constructs between the APD and control groups were repeated using years of parental education, parental SES, and age at DIS as covariates.

### *3.1.3 Clinical Characteristics*

Clinical characteristics and indices of functioning of the diagnostic outcome groups were examined next in order to obtain a more complete description of the functional status of these groups. This was especially helpful in more fully describing the psychosis group, which was not defined using DSM-IV criteria, as detailed earlier. These characteristics are presented in Table 10 for each group, and the results of the pairwise group comparisons on a subset of these characteristics are summarized in Table 11. Because the characteristics of Table 11 are either related to how the diagnostic groups were defined or reflect aspects of functioning that are likely to be influenced by and thus reflective of difficulties often associated with the diagnostic status of the outcome groups, these traits were not considered as possible covariates for the primary analyses.

*3.1.3.1 Psychosis group.* As displayed in Table 10, of the 16 boys endorsing full psychotic symptoms of at least a month's duration, one boy met criteria for schizophrenia, one for schizophreniform disorder, and three for delusional disorder. As can be seen, several also met criteria for a mood and/or anxiety disorder, and/or APD. Further, seven out of the 16 boys met criteria for substance dependence; see Table 10. Specifically, four of these seven boys met criteria for alcohol dependence and four for cannabis dependence. As can be seen, the majority of the psychosis group boys denied psychotic symptom-related impairment in occupational and interpersonal functioning, and only approximately one-third reported seeking help for



psychological problems during the year preceding the DIS. Further, most of the boys of the psychosis group were students and/or employed at least part time when the DIS was administered.

*3.1.3.2 Psychosis versus controls.* As displayed in the second column of Table 11, the boys of the psychosis group were significantly more likely than controls to meet criteria for substance dependence. In addition, these boys were more likely than controls to seek help for a psychological problem and to be hospitalized for psychiatric reasons during the year preceding the DIS than were controls. The psychosis group boys also had significantly lower SES than did the control boys.

*3.1.3.3 Psychosis versus APD.* The boys of the psychosis group were significantly more likely than the APD boys to meet criteria for a mood disorder, as well as for an anxiety disorder. The psychosis group boys were also more likely to seek help for a psychological problem in the year preceding the DIS than were the boys of the APD group.

*3.1.3.4 Psychosis versus depression/anxiety.* The boys of the psychosis group had significantly fewer years of education and lower SES at the time of the DIS than did the boys of the depression/anxiety group. Not surprisingly, the depression/anxiety group boys were more likely than those of the psychosis group to be diagnosed with an anxiety disorder.

*3.1.3.5 APD versus controls.* Compared to controls, the boys of the APD group were more likely to meet criteria for substance abuse and dependence. They were also more likely than controls to be hospitalized for psychiatric reasons during the year preceding the DIS. In addition, they had significantly fewer years of education, had lower SES, and were less likely to be students and/or employed when the DIS was administered compared to controls.

Table 10  
*Current I Functioning and Clinical Characteristics of Diagnostic Outcome Groups*

Characteristic	Psychosis (n=16) <sup>2</sup>	APD (n=52) <sup>2</sup>	Depression/Anxiety (n=22) <sup>2</sup>	Controls (n=647) <sup>2</sup>
Psychotic disorder <sup>3</sup>				
schizophrenia	4.1% <sup>4</sup> (1)	---	---	---
schizophreniform disorder	5.1% <sup>4</sup> (1)	---	---	---
delusional disorder	17.8% <sup>4</sup> (3)	---	---	---
mood disorder with psychosis	13.7% <sup>4</sup> (2 <sup>5</sup> )	---	---	---
psychosis NOS	59.3% <sup>4</sup> (9)	---	---	---
Reported on DIS impairment at job, school, home, self care, or interpersonal life since psychotic symptoms began <sup>6</sup>	20.5% <sup>4</sup> (3/16)	---	---	---
Reported on DIS that psychotic symptoms have caused problems with family, friends, work, or in other situations <sup>7</sup>	30.6% <sup>4</sup> (5/16)	---	---	---
Any mood disorder <sup>3, 8</sup>	25.5% <sup>4</sup>	6.6% <sup>4</sup>	42.9% <sup>4</sup>	---
major depressive disorder without psychosis	0%	5.4% <sup>4</sup>	42.9% <sup>4</sup>	---
bipolar disorder without psychosis	0%	1.4% <sup>4</sup>	---	---
dysthymia	7.7% <sup>4</sup>	1.2% <sup>4</sup>	0%	---

Table 10 (continued)

Characteristic	Psychosis ( <i>n</i> =16) <sup>2</sup>	APD ( <i>n</i> =52) <sup>2</sup>	Depression/ Anxiety ( <i>n</i> =22) <sup>2</sup>	Controls ( <i>n</i> =647) <sup>2</sup>
Any anxiety disorder <sup>3</sup>	17.8% <sup>4</sup>	3.6% <sup>4</sup>	68.3% <sup>4</sup>	---
panic disorder	4.1% <sup>4</sup>	0%	11.9% <sup>4</sup>	---
agoraphobia	0%	2.3% <sup>4</sup>	9.0% <sup>4</sup>	---
generalized anxiety disorder	0%	0%	6.1% <sup>4</sup>	---
social phobia	13.8% <sup>4</sup>	0%	17.7% <sup>4</sup>	---
specific phobia	0%	1.2% <sup>4</sup>	0%	---
post-traumatic stress disorder	0%	0%	5.4% <sup>4</sup>	---
obsessive-compulsive disorder	0%	0%	27.1% <sup>4</sup>	---
APD <sup>3</sup>	27.2% <sup>4</sup>	100%	---	---
Substance use disorder <sup>3</sup>				
substance abuse but not dependence <sup>9</sup>	4.1% <sup>4</sup>	43.8% <sup>4</sup>	3.6% <sup>4</sup>	11.9% <sup>4</sup>
substance dependence <sup>10</sup>	43.3% <sup>4</sup>	22.7% <sup>4</sup>	20.6% <sup>4</sup>	4.0% <sup>4</sup>
Sought help for a psychological problem(s) from a professional or other source during past year <sup>11</sup>	29.6% <sup>4</sup>	1.6% <sup>4</sup>	46.1% <sup>4</sup>	2.7% <sup>4</sup>
Psychiatric hospitalization(s) during past year	4.1% <sup>4</sup>	5.0% <sup>4</sup>	0%	0.6% <sup>4</sup>
Currently a student and/or employed <sup>12</sup>	82.2% <sup>4</sup>	62.8% <sup>4</sup>	78.2% <sup>4</sup>	83.3% <sup>4</sup>
Current education (years; <i>M</i> ( <i>SD</i> ))	11.78 (2.04)	11.23 (1.72)	13.25 (1.99)	12.04 (1.73)
Current personal SES <sup>13</sup>	23.25 (14.32)	25.86 (12.03)	37.05 (13.03)	31.21 (13.01)

Table 10 (continued)

*Note.* APD= antisocial personality disorder; DIS= Diagnostic Interview Schedule for DSM-IV; DSM-IV=The Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition; M=mean; NOS=not otherwise specified; SD=standard deviation; SES=socioeconomic status

<sup>1</sup>“Current” refers to assessment period during which DIS administered (at approximately age 19 for youngest sample and 25 for oldest).

<sup>2</sup>weighted *n*.

<sup>3</sup>DSM-IV diagnoses per DIS interview (at approximately age 19 for youngest sample and 25 for oldest).

<sup>4</sup>weighted percentage.

<sup>5</sup>Specifically one participant met criteria for major depressive disorder with psychosis and one for bipolar disorder with psychosis.

<sup>6</sup>per following items of DIS: “After these beliefs or experiences began, did you find that you were less able to do your work at a job, at school, or at home?”; “After these beliefs or experiences began, were less able to make friends or enjoy social relationships?”; “After these beliefs or experiences began, did you go through a period when you would not bathe or wash your clothes?”

<sup>7</sup>per following items of DIS: “Have these beliefs or experiences caused problems with family, friends, or work in the past year?”; “Have these beliefs or experiences ever caused problems with family, friends, work, or in other situations?”

<sup>8</sup>includes major depressive disorder with psychosis and bipolar disorder with psychosis.

<sup>9</sup>Specific diagnoses included alcohol abuse, cannabis abuse, cocaine abuse, and hallucinogen abuse.

<sup>10</sup>Specific diagnoses included alcohol dependence, cannabis dependence, stimulant dependence, sedative dependence, cocaine dependence, opiate dependence, hallucinogen dependence, and other drug dependence.

<sup>11</sup>included helpseeking from a psychologist, psychiatrist, counselor, social worker, mental health center, psychiatric outpatient clinic, family service, counseling or social service agency, community program (e.g., crisis center or hot line), minister, priest, rabbi, teacher, school counselor, school social worker, family doctor, or primary care physician.

<sup>12</sup>at least part time.

<sup>13</sup>based on Hollingshead’s Factor (1975; 6=lowest, 66=highest).

Table 11  
*Clinical Characteristics and Functioning: Diagnostic Group Comparisons*

Characteristic	Psychosis v. Ctrl	Psychosis v. APD	Psychosis v. Depression/Anxiety	APD v. Ctrl	Depression/Anxiety v. Ctrl
	$\chi^2; p$	$\chi^2; p$	$\chi^2; p$	$\chi^2; p$	$\chi^2; p$
Any mood disorder <sup>1,2</sup>	---	<b>4.90; .027</b> (Psy > APD)	1.04; .307	---	---
Any anxiety disorder <sup>1,3</sup>	---	<b>3.99; .046</b> (Psy > APD)	<b>9.08; .003</b> (Psy < Dep/Anx)	---	---
Substance abuse but not dependence <sup>1,4</sup>	.480; .488	<b>7.73; .005</b> (Psy < APD)	.054; .816	<b>41.04; &lt;.001</b> (APD > Ctrl)	1.12; .290
Substance dependence <sup>1,5</sup>	<b>52.11; &lt;.001</b> (Psy > Ctrl)	2.60; .107	2.15; .143	<b>34.01; &lt;.001</b> (APD > Ctrl)	<b>15.81; &lt;.001</b> (Dep/Anx > Ctrl)
Sought help for a psychological problem(s) from a professional or other source during past year <sup>6</sup>	<b>39.23; &lt;.001</b> (Psy > Ctrl)	<b>12.81; &lt;.001</b> (Psy > APD)	1.01; .315	.092; .762	<b>104.57; &lt;.001</b> (Dep/Anx > Ctrl)

Table 11 (continued)

Characteristic	Psychosis v. Ctrl	Psychosis v. APD	Psychosis v. Depression/Anxiety	APD v. Ctrl	Depression/Anxiety v. Ctrl
	$\chi^2; p$	$\chi^2; p$	$\chi^2; p$	$\chi^2; p$	$\chi^2; p$
Psychiatric hospitalization(s) during past year	<b>6.52; .011</b> (Psy > Ctrl)	.003; .957	1.35; .245	<b>13.00; &lt;.001</b> (APD > Ctrl)	.132; .716
Currently a student and/or employed <sup>7</sup>	.045; .832	1.89; .169	.137; .711	<b>13.31; &lt;.001</b> (APD < Ctrl)	.720; .396
Current years of education <sup>8</sup>	.355; .551	1.17; .280	<b>4.80; .028</b> (Psy < Dep/Anx)	<b>11.33; .001</b> (APD < Ctrl)	<b>8.48; .004</b> (Dep/Anx > Ctrl)
Current personal SES <sup>8,9</sup>	<b>5.54; .019</b> (Psy < Ctrl)	.509; .476	<b>8.05; .005</b> (Psy < Dep/Anx)	<b>8.10; .004</b> (APD < Ctrl)	<b>4.10; .043</b> (Dep/Anx > Ctrl)

*Note.* APD= antisocial personality disorder; Ctrl=controls; Dep/Anx=depression/anxiety group; DIS= Diagnostic Interview Schedule for DSM-IV; DSM-IV=The Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition; Psy=psychosis group; SES=socioeconomic status; v=versus  
All *p* values are two tailed.

<sup>1</sup>DSM-IV diagnoses per DIS interview (at approximately age 19 for youngest sample and 25 for oldest).

<sup>2</sup>Specific diagnoses included major depressive disorder with psychosis, major depressive disorder without psychosis, bipolar disorder with psychosis, bipolar disorder without psychosis, and dysthymia.

<sup>3</sup>Specific diagnoses included panic disorder, agoraphobia, generalized anxiety disorder, social phobia, specific phobia, post-traumatic stress disorder, and obsessive-compulsive disorder.

<sup>4</sup>Specific diagnoses included alcohol abuse, cannabis abuse, cocaine abuse, and hallucinogen abuse.

<sup>5</sup>Specific diagnoses included alcohol dependence, cannabis dependence, stimulant dependence, sedative dependence, cocaine dependence, opiate dependence, hallucinogen dependence, and other drug dependence.

Table 11 (continued)

<sup>6</sup>included helpseeking from a psychologist, psychiatrist, counselor, social worker, mental health center, psychiatric outpatient clinic, family service, counseling or social service agency, community program (e.g., crisis center or hot line), minister, priest, rabbi, teacher, school counselor, school social worker, family doctor, or primary care physician.

<sup>7</sup>at least part time.

<sup>8</sup> $\chi^2$  from logistic regression was used to assess group differences on continuous demographic variables because the data from several of these variables were non-normally distributed.

<sup>9</sup>based on Hollingshead's Factor (1975; 6=lowest, 66=highest).

*3.1.3.6 Depression/Anxiety versus controls.* As displayed in Table 11, the boys of the depression/anxiety group were significantly more likely than controls to receive a diagnosis of substance dependence, as well as to seek help for psychological problems in the year preceding the DIS. Interestingly, these boys also were characterized by significantly more years of education and higher SES compared to controls.

## **3.2 Preliminary Analyses**

### *3.2.1 Data Inspection of the Primary Constructs*

The data distributions of the primary constructs for each age (i.e., Schizophrenia-like Positive Symptoms, Schizophrenia-like Negative Symptoms, Peer Rejection, and Parent-Child Relationship, each for ages 13, 14, 15, 16, and 17) were examined using the total sample for skewness and kurtosis. The values obtained when dividing skew by the standard error of skew, and likewise for kurtosis (Tabachnick & Fidell, 1996), indicated that all 20 of the primary variables were positively skewed (all  $ps < .001$ ) and that 18 of the 20 had significant kurtosis (all  $ps < .01$ ). Congruent with these findings, visual inspection of histograms clearly suggested that all 20 of these variables were positively skewed. This was not surprising, given the nature of the behavioral features measured by these constructs. Based on these findings, logistic regression was used for all primary analyses because it does not assume normally-distributed data, nor does it make assumptions about homogeneity of covariance matrices.

Data distributions of the primary constructs were inspected separately by group for the presence of outliers. Visual inspection of boxplots and frequency tables suggested several extreme values across the 20 variables, with the greatest number observed among the control



group. However, in considering the size of the control group (weighted  $n=647$ ) and the nature of the questions of the study (i.e., the interest in individuals who may have relatively extreme scores on the primary constructs), a conservative approach was taken when determining if relatively extreme values should be removed or altered. After examining the size of the breaks in the scores forming the positive tails of the variables, it was decided that none of the scores would be eliminated or transformed.

### *3.2.2 Internal Consistency and Inter-rater Correlations of the Primary Constructs*

As noted earlier, three of the four primary constructs (i.e., Schizophrenia-like Positive Symptoms, Schizophrenia-like Negative Symptoms, and Peer Rejection) were created for the present study. The items for each of these constructs (presented in Table 5) were selected because they appeared to conceptually reflect the theoretical construct of interest based on face validity. To assess the degree to which the items contributing to each of these constructs were related within informant, internal consistency was examined using Cronbach's alpha. These results for the 40 informant-specific constructs (i.e., parent-, teacher-, and child-informant versions of Schizophrenia-like Positive Symptoms, Schizophrenia-like Negative Symptoms, and Peer Rejection for ages 13 through 17) are presented in Appendix A. As can be seen, the alpha coefficients were generally lower than ideal, with most falling in the range of 0.5 to 0.7. However, the theoretical constructs that the primary constructs of this study were designed to measure are not unidimensional. For example, the items selected for the Schizophrenia-like Positive Symptoms construct included those that appeared to assess oddity, suspiciousness, and hallucination-like experiences, which are different aspects of this theoretical construct. Thus it was not surprising that the alpha coefficients were relatively low.

As noted, the fourth construct used, Parent-Child Relationship, was an existing construct developed by the PYS. Across the phases used in the present study, the alpha coefficients for the parent-informant-version of this construct for the full youngest and oldest samples of the PYS ranged from .37 to .44 ( $M=.40$ ) and .37 to .53 ( $M=.46$ ), respectively. The child-informant version of Parent-Child Relationship yielded alphas ranging from .84 to .90 ( $M=.87$ ) and .82 to .91 ( $M=.87$ ) for the full youngest and oldest PYS samples, respectively.

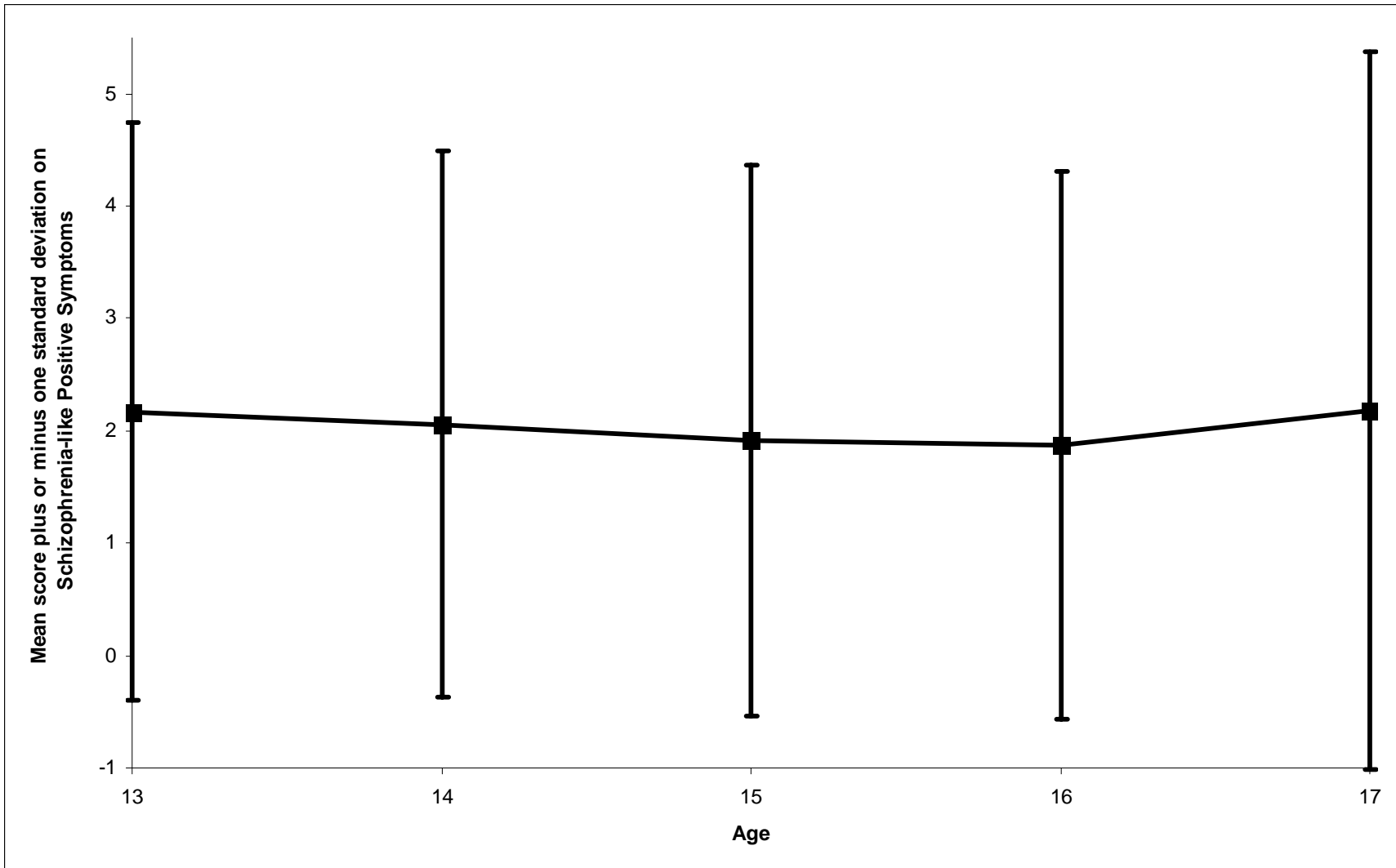
To assess the degree of agreement across raters, Spearman correlations of the informant-specific constructs were examined for the four primary constructs by age. It should be noted that these analyses were not weighted, as the weighting procedure cannot be implemented when calculating Spearman correlation coefficients. As detailed in Appendix B, all of the resulting pairwise correlations were positive and most (33 out of 40) were statistically significant, as expected. However, the actual correlation coefficients were generally low, ranging from .01 to .34. As noted earlier, the rationale for combining informant-specific constructs was that doing so would provide the best assessment of the behaviors in question because different informants could at times provide information on behavioral difficulties that are evident only in certain contexts (Loeber et al., 1998). The relatively low inter-rater correlations observed are consistent with the notion that the various informants of the PYS (i.e. parents, teachers, and the boys themselves) are at times providing distinct information on the functioning of the participant being assessed and thus were not surprising, and did not result in any deviations from the plan to combine the informant-specific constructs.

### 3.2.3 Descriptive Overview of Raw Scores of Primary Constructs by Age

Figures 1a-1d provide a visual overview of the mean raw scores and standard deviations of the primary constructs from ages 13 to 17 using the total sample. Please refer to Appendix C for more detailed information regarding the values depicted in the figures. For all constructs, higher scores indicate a higher level of problematic behaviors and/or experiences. As can be seen for all four of these constructs, when collapsed across group, there is relatively little change across the ages assessed. Further, the level of the mean scores obtained by the sample is relatively low compared to the range of possible scores; this is especially notable for Schizophrenia-like Positive Symptoms, which is not surprising given the nature of the behaviors assessed by this construct. As depicted by the standard deviation bars and the range of scores obtained, all of the constructs are characterized by notable variability in scores among the boys.

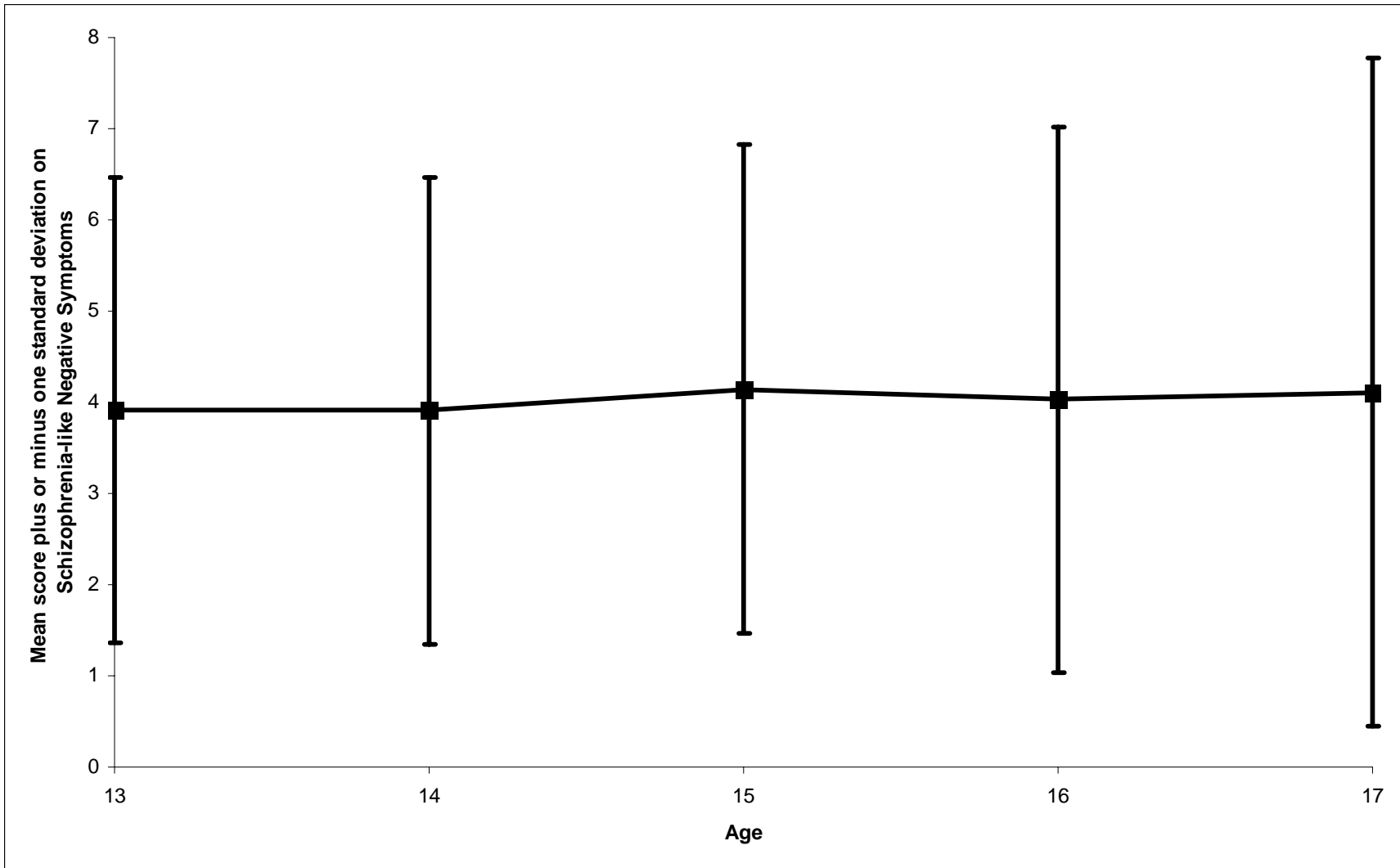
### 3.2.4 Overview of HLM-derived Intercept and Slope Coefficients

To allow for the examination of diagnostic outcome group differences in both the *level* of the primary constructs at each age and the *rate of change* on these constructs across the ages assessed, hierarchical linear modeling (HLM; Raudenbush & Bryk, 2002) was used to obtain participant-level intercept and slope estimates for each of the primary constructs. This approach, which assumes that longitudinal data are hierarchical because repeated observations are nested within individuals, allows for the calculation of individual growth trajectories for each participant (Raudenbush & Bryk, 2002). For the purposes of the present study, this permitted testing whether participant-level intercept and slope coefficients discriminated diagnostic groups, which allowed for questions regarding group differences both in level at each age and in change over time to be addressed. Another significant advantage of HLM is that it does not require participants to have an equal number of observations; thus participants with missing data can be



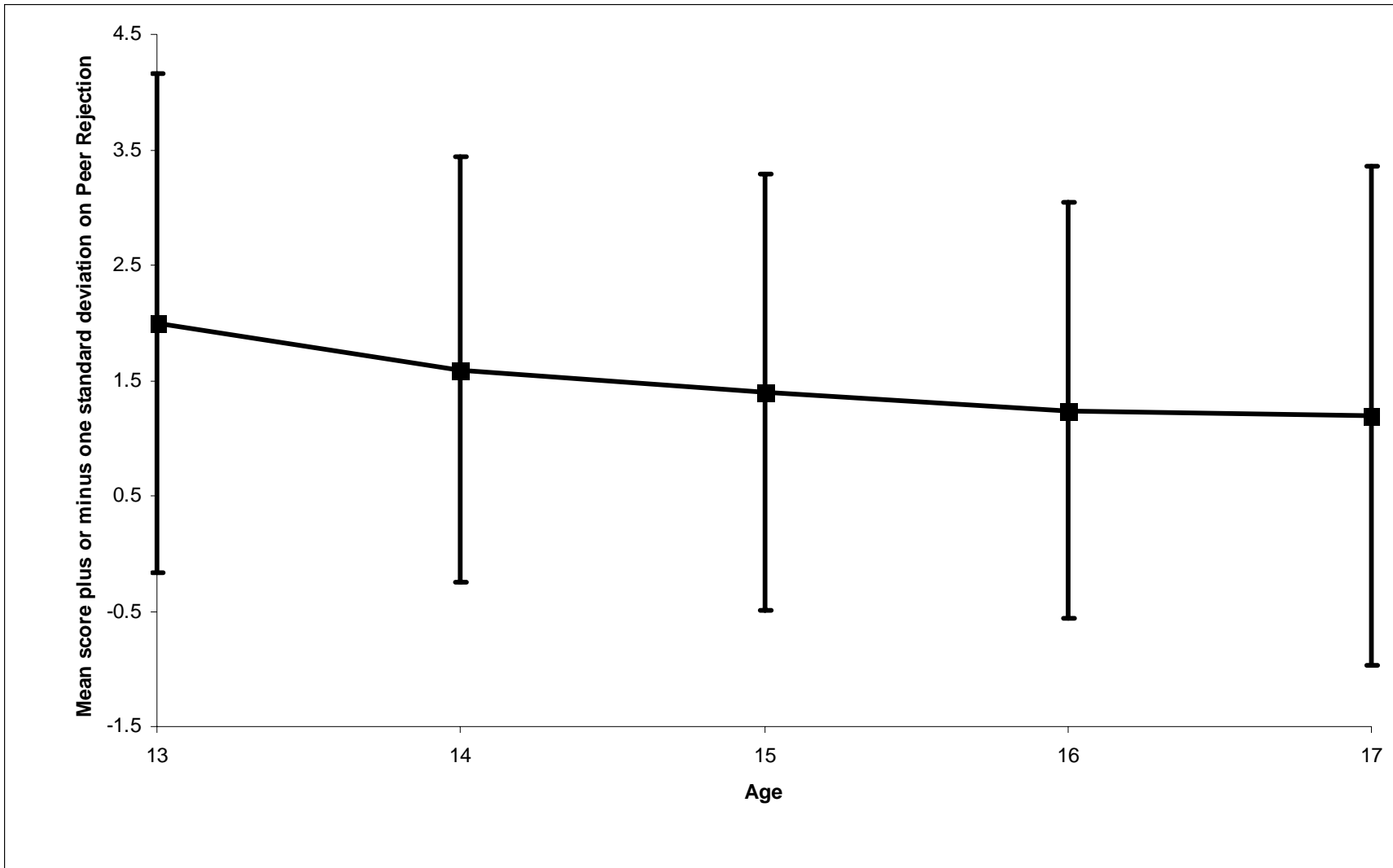
*Figure 1a.*

Mean raw scores plus or minus one standard deviation on Schizophrenia-like Positive Symptoms from ages 13 to 17 for total sample (range of possible scores=0-36).



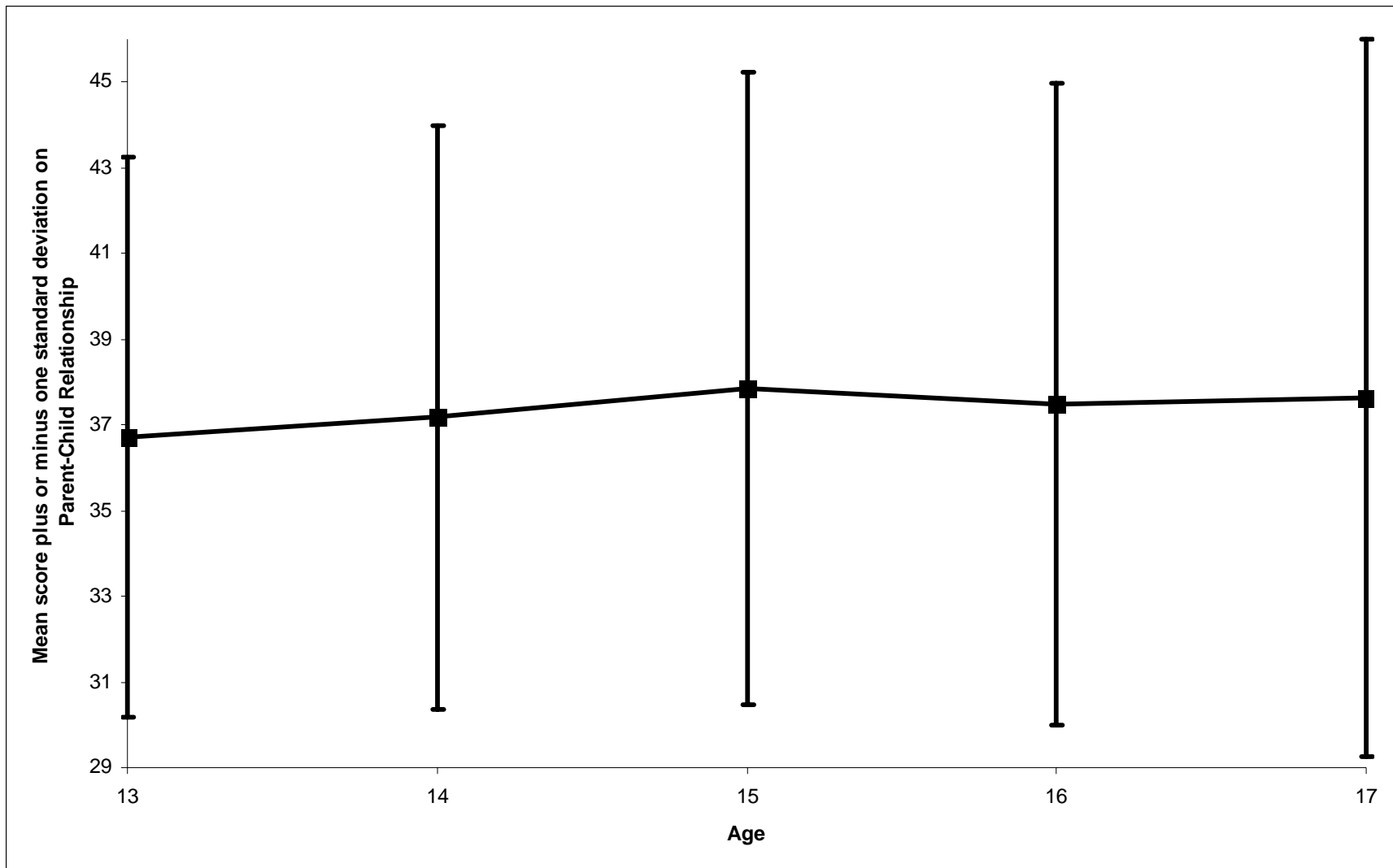
*Figure 1b.*

Mean raw scores plus or minus one standard deviation on Schizophrenia-like Negative Symptoms from ages 13 to 17 for total sample (range of possible scores=0-24).



*Figure 1c.*

Mean raw scores plus or minus one standard deviation on Peer Rejection from ages 13 to 17 for total sample (range of possible scores=0-18).



*Figure 1d.*

Mean raw scores plus or minus one standard deviation on Parent-Child Relationship from ages 13 to 17 for total sample (range of possible scores=26-78).

included (Raudenbush & Bryk, 2002), which, compared to listwise deletion procedures, allows for more efficient use of available data and yields parameter estimates that are less biased. As noted earlier, for the present study, participants missing up to two of the five observations for each construct were included, which reduced the number of participants excluded due to missing data.

As described earlier, due to a concern that the various informants of the present study may have differed in how they approached the rating scales on which the primary constructs were based, as well as to facilitate group comparisons across constructs and ages, both the informant-specific and combined-informant constructs were standardized for the primary analyses (see Method section for details regarding creation of primary constructs). The standardized combined-informant versions of the primary constructs were analyzed using the Hierarchical Linear and Nonlinear Modeling for Windows software program, version 6.02 (HLM 6.02; Raudenbush, Bryk, & Congdon, 2005). Specifically, using the total sample, for each construct a level-1 model was specified for which the repeated observations for each age served as the criterion variable and age served as the predictor variable. The intercept and slope terms were treated as random, which allowed them to vary by participant. For each construct, this model was estimated five times, with the construct values for each age serving as the intercept in turn. This yielded intercept estimates for each age, which allowed group differences to be assessed at each of the five ages. Level-2 residual files were created for each model in order to derive the participant-level intercept and slope coefficients. The empirical Bayes coefficients were used for the primary analyses because they tend to have smaller prediction errors and thus tend to be more accurate than ordinary least-squares estimates (Raudenbush & Bryk, 2002).



The models used to obtain the intercept and slope coefficients described above assume linear change across time. Thus additional HLM level-1 models that included both age and age squared as predictor variables and the repeated observations for each construct in turn as the criterion variable were estimated using the total sample and separately for each group to assess whether it was appropriate to use only linear slope terms rather than including both linear and quadratic terms in the models. For all of these models, results indicated that when entered along with the linear slope term (age), the quadratic slope term (age squared) was not significantly different from zero (all  $ps > .148$ ). Because the primary purpose of the present study was to assess diagnostic group differences, however, the quadratic coefficients derived using the total sample were also used within logistic regression separately for each construct to determine whether they significantly discriminated the diagnostic groups. These pairwise group comparisons indicated that the quadratic component significantly differed only between the APD and control groups for Schizophrenia-like Positive Symptoms, Schizophrenia-like Negative Symptoms, and Parent-Child Relationship. Thus, with the exception of these APD-control group comparisons, for all analyses the linear slope terms were used to examine group differences in change over time. Because the quadratic slope term more effectively discriminated the APD boys from controls than did the linear component for the three domains noted (all  $ps < .05$ ), the linear and quadratic slope coefficients were used to assess APD-control group differences in change over time for these three constructs.

As expected, preliminary analyses within HLM indicated that when using the total sample, all of the intercept and slope coefficients were characterized by significant variability among the boys ( $\chi^2$  ranged from 857.69 to 6460.76; all  $ps \leq .001$ ); these findings are consistent with the variability noted in the raw scores, described above, and supported further analyses to

examine whether the intercept and slope terms for these constructs varied by diagnostic outcome group. The normality assumption of HLM precluded using the obtained intercept and slope coefficients as criterion variables within a full HLM level-2 model. Thus for the analyses used to examine group differences on the primary constructs, the HLM-derived intercept and slope coefficients were used as predictor variables within logistic regression, which, as noted, does not assume normality.

### *3.2.5 Associations between Demographic Characteristics and HLM-derived Intercept and Slope Coefficients*

Because the HLM-derived intercept and slope coefficients were used for the primary analyses rather than the primary construct raw or standardized scores, associations between the demographic variables and intercept and slope coefficients were examined; these results are presented in Table 12. As displayed, logistic regression analyses indicated that when compared to the youngest sample of the PYS, the oldest sample boys obtained significantly higher mean levels (as reflected by the mean intercept values) of Schizophrenia-like Positive Symptoms, Schizophrenia-like Negative Symptoms, and Peer Rejection at ages 13 through 17, and of problematic Parent-Child Relationship at ages 14 through 17. Appendix D provides the corresponding descriptive statistics for the intercept and slope coefficients for each construct by demographic group. Comparison of the slope estimates further indicated that although boys from the oldest sample increased only slightly across age on Schizophrenia-like Positive Symptoms, Schizophrenia-like Negative Symptoms, and problematic Parent-Child Relationship, they increased significantly more than the youngest-sample boys, who obtained weak negative slopes on all four of the constructs (see Appendix D).

Table 12

*Associations between Demographic Characteristics and HLM-derived Intercept and Slope Coefficients for Schizophrenia-like Positive Symptoms, Schizophrenia-like Negative Symptoms, Peer Rejection, and Parent-Child Relationship*

Coefficient	Sample <sup>1,2</sup>	Risk group <sup>2,3</sup>	Ethnicity <sup>2,4</sup>	Age at DIS <sup>5</sup>	Years of parental education <sup>5,6</sup>	Parental SES <sup>5,7</sup>
	$\chi^2; p$	$\chi^2; p$	$\chi^2; p$	$r_s; p$	$r_s; p$	$r_s; p$
Schizophrenia-like Positive Symptoms:						
Age 13 intercept	<b>6.62; .010</b> (O>Y)	<b>55.03; &lt;.001</b> (H>L)	.020; .888	<b>.218; &lt;.001</b>	<b>-.084; .025</b>	<b>-.093; .012</b>
Age 14 intercept	<b>10.02; .002</b> (O>Y)	<b>50.98; &lt;.001</b> (H>L)	.002; .964	<b>.232; &lt;.001</b>	<b>-.075; .045</b>	<b>-.083; .025</b>
Age 15 intercept	<b>13.86; &lt;.001</b> (O>Y)	<b>44.41; &lt;.001</b> (H>L)	.003; .954	<b>.245; &lt;.001</b>	-.062; .095	-.071; .057
Age 16 intercept	<b>17.60; &lt;.001</b> (O>Y)	<b>36.12; &lt;.001</b> (H>L)	.026; .873	<b>.255; &lt;.001</b>	-.046; .220	-.056; .132
Age 17 intercept	<b>20.68; &lt;.001</b> (O>Y)	<b>27.40; &lt;.001</b> (H>L)	.065; .799	<b>.260; &lt;.001</b>	-.024; .527	-.036; .334
Slope	<b>8.48; .004</b> (O>Y)	<b>13.76; &lt;.001</b> (L>H)	.405; .524	.069; .064	<b>.073; .051</b>	<b>.088; .017</b>

Table 12 (continued)

Coefficient	Sample <sup>1,2</sup> $\chi^2; p$	Risk group <sup>2,3</sup> $\chi^2; p$	Ethnicity <sup>2,4</sup> $\chi^2; p$	Age at DIS <sup>5</sup> $r_s; p$	Years of parental education <sup>5,6</sup> $r_s; p$	Parental SES <sup>5,7</sup> $r_s; p$
Schizophrenia-like Negative Symptoms:						
Age 13 intercept	<b>14.61; &lt;.001</b> (O>Y)	<b>16.63; &lt;.001</b> (H>L)	<b>6.13; .013</b> (Ot>EA)	<b>.198; &lt;.001</b>	-.062; .095	<b>-.102; .006</b>
Age 14 intercept	<b>19.58; &lt;.001</b> (O>Y)	<b>17.12; &lt;.001</b> (H>L)	<b>8.61; .003</b> (Ot>EA)	<b>.224; &lt;.001</b>	-.069; .065	<b>-.109; .003</b>
Age 15 intercept	<b>24.99; &lt;.001</b> (O>Y)	<b>17.00; &lt;.001</b> (H>L)	<b>11.43; .001</b> (Ot>EA)	<b>.253; &lt;.001</b>	<b>-.073; .050</b>	<b>-.114; .002</b>
Age 16 intercept	<b>30.19; &lt;.001</b> (O>Y)	<b>16.17; &lt;.001</b> (H>L)	<b>14.26; &lt;.001</b> (Ot>EA)	<b>.278; &lt;.001</b>	<b>-.075; .043</b>	<b>-.119; .001</b>
Age 17 intercept	<b>34.50; &lt;.001</b> (O>Y)	<b>14.66; &lt;.001</b> (H>L)	<b>16.76; &lt;.001</b> (Ot>EA)	<b>.299; &lt;.001</b>	<b>-.076; .040</b>	<b>-.122; .001</b>
Slope	<b>7.88; .005</b> (O>Y)	1.14; .286	<b>5.35; .021</b> (Ot>EA)	<b>.106; .004</b>	-.025; .500	-.022; .563

Table 12 (continued)

Coefficient	Sample <sup>1,2</sup> $\chi^2; p$	Risk group <sup>2,3</sup> $\chi^2; p$	Ethnicity <sup>2,4</sup> $\chi^2; p$	Age at DIS <sup>5</sup> $r_s; p$	Years of parental education <sup>5,6</sup> $r_s; p$	Parental SES <sup>5,7</sup> $r_s; p$
Peer Rejection:						
Age 13 intercept	<b>14.30; &lt;.001</b> (O>Y)	<b>17.45; &lt;.001</b> (H>L)	.009; .923	<b>.209; &lt;.001</b>	<b>-.126; .001</b>	<b>-.132; &lt;.001</b>
Age 14 intercept	<b>16.75; &lt;.001</b> (O>Y)	<b>16.54; &lt;.001</b> (H>L)	.047; .829	<b>.213; &lt;.001</b>	<b>-.123; .001</b>	<b>-.131; &lt;.001</b>
Age 15 intercept	<b>18.98; &lt;.001</b> (O>Y)	<b>15.04; &lt;.001</b> (H>L)	.116; .733	<b>.217; &lt;.001</b>	<b>-.117; .002</b>	<b>-.127; .001</b>
Age 16 intercept	<b>20.69; &lt;.001</b> (O>Y)	<b>13.02; &lt;.001</b> (H>L)	.216; .642	<b>.220; &lt;.001</b>	<b>-.110; .003</b>	<b>-.120; .001</b>
Age 17 intercept	<b>21.63; &lt;.001</b> (O>Y)	<b>10.68; .001</b> (H>L)	.336; .562	<b>.230; &lt;.001</b>	<b>-.104; .005</b>	<b>-.116; .002</b>
Slope	.945; .331	<b>4.97; .026</b> (L>H)	.723; .395	-.029; .432	<b>.110; .003</b>	<b>.093; .012</b>

Table 12 (continued)

Coefficient	Sample <sup>1,2</sup> $\chi^2; p$	Risk group <sup>2,3</sup> $\chi^2; p$	Ethnicity <sup>2,4</sup> $\chi^2; p$	Age at DIS <sup>5</sup> $r_s; p$	Years of parental education <sup>5,6</sup> $r_s; p$	Parental SES <sup>5,7</sup> $r_s; p$
Parent-Child Relationship:						
Age 13 intercept	3.13; .077	<b>46.79; &lt;.001</b> (H>L)	.026; .872	<b>.114; .002</b>	-.029; .437	-.060; .105
Age 14 intercept	<b>5.33; .021</b> (O>Y)	<b>43.82; &lt;.001</b> (H>L)	.000; .989	<b>.121; .001</b>	-.023; .531	-.054; .144
Age 15 intercept	<b>8.09; .004</b> (O>Y)	<b>38.95; &lt;.001</b> (H>L)	.021; .885	<b>.128; .001</b>	-.016; .661	-.045; .225
Age 16 intercept	<b>11.13; .001</b> (O>Y)	<b>32.52; &lt;.001</b> (H>L)	.094; .759	<b>.134; &lt;.001</b>	-.015; .691	-.040; .284
Age 17 intercept	<b>14.05; &lt;.001</b> (O>Y)	<b>25.35; &lt;.001</b> (H>L)	.211; .646	<b>.138; &lt;.001</b>	-.010; .796	-.032; .393
Slope	<b>9.24; .002</b> (O>Y)	<b>14.44; &lt;.001</b> (L>H)	1.09; .296	.035; .341	.034; .360	.043; .246

*Note.* DIS= Diagnostic Interview Schedule for DSM-IV; DSM-IV=The Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition; EA=European American; H=high; L=low; O=oldest; Ot=other; PYS=Pittsburgh Youth Study; SES=socioeconomic status; Y=youngest

All *p* values are two tailed.

Table 12 (continued)

<sup>1</sup>youngest or oldest sample.

<sup>2</sup> $\chi^2$  from logistic regression was used to assess associations between dichotomous demographic variables and HLM coefficients.

<sup>3</sup>low or high risk based on initial assessment at screening phase of PYS.

<sup>4</sup>European American or other; the “other” group was predominantly African American.

<sup>5</sup>Spearman correlation coefficients were used to assess associations between continuous demographic variables and HLM coefficients.

Note that unweighted *N*s used for these analyses, as the weighting procedure cannot be implemented when calculating Spearman correlation coefficients.

<sup>6</sup>caretaker or partner education, whichever higher, as assessed when boy was approximately age 13.

<sup>7</sup>based on Hollingshead’s Factor (1975; 6=lowest, 66=highest); caretaker or partner SES, whichever higher, as assessed when boy was approximately age 13.

Similarly, boys classified as high risk based on the initial screening assessment of the PYS displayed higher levels of Schizophrenia-like Positive Symptoms, Schizophrenia-like Negative Symptoms, Peer Rejection, and problematic Parent-Child Relationship across all ages assessed compared to the low risk boys. Interestingly, slope comparisons indicated that the shallow but negative mean trajectories of the high-risk boys on Schizophrenia-like Positive Symptoms, Peer Rejection, and problematic Parent-Child Relationship differed significantly from the weak but positive slopes of the low risk boys on these three constructs.

Significant ethnic group differences emerged only for Schizophrenia-like Negative Symptoms. Specifically, boys not of European-American descent (who were predominantly African American) obtained significantly higher levels of negative symptoms across all ages compared to the European-American boys. Further, the shallow but positive slope for negative symptoms across age obtained by this group was significantly greater than the weak but negative slope of the European-American boys.

As presented in Table 12, Spearman correlational analyses (using unweighted *ns*) indicated that age at the time of DIS administration was significantly correlated with levels of Schizophrenia-like Positive Symptoms, Schizophrenia-like Negative Symptoms, Peer Rejection, and problematic Parent-Child Relationship at ages 13 through 17, such that the boys who were older at DIS administration obtained higher levels on these constructs at all ages assessed. These findings are consistent with those presented above concerning the youngest and oldest samples because, as noted, the youngest sample was a mean age of 19 at the time of DIS administration,



whereas the oldest sample was a mean age of 25. However, in contrast to the sample comparisons, older age at DIS was significantly associated with a more positive slope only for Schizophrenia-like Negative Symptoms.

Boys whose parents had fewer years of education and lower SES tended to display greater levels of Schizophrenia-like Positive Symptoms at ages 13 and 14, but not at ages 15 through 17; see Table 12. Lower parental education and SES were also significantly associated with a less positive slope across age for positive symptoms. Boys with lower parental education also tended to have higher levels of Schizophrenia-like Negative Symptoms at ages 15 through 17, whereas those with lower parental SES obtained a greater level of negative symptoms across all ages assessed. Neither years of parental education nor parental SES was related to the rate of change in negative symptoms across age. For Peer Rejection, both the level and rate of change across the ages assessed were significantly correlated with years of parental education and SES. Boys whose parents had fewer years of education and lower SES experienced greater levels of Peer Rejection from ages 13 to 17, but also tended to increase across age at a less positive rate on this construct compared to boys with higher parental education and SES. Parental education and SES were not associated with either the level of or change over time in problematic Parent-Child Relationship across the ages assessed.

Due to the associations observed between certain demographic characteristics and the levels and slopes of the primary constructs, additional analyses were conducted in an attempt to better understand these findings. Such analyses indicated that the significant associations observed between age at DIS and the construct coefficients remained significant even when examining such relations separately for the youngest and oldest samples for all constructs except problematic Parent-Child Relationship. Thus with the exception of this latter construct, boys

within both the youngest and oldest samples who were older when the DIS was administered tended to obtain higher scores on the primary constructs compared to boys who were younger at the time of the DIS.

*3.2.5.1 Additional covariates for primary analyses.* As reviewed earlier, for the most part, the diagnostic outcome groups did not differ significantly on the demographic variables. However, it is clear that these demographic characteristics explain some of the variance in the primary constructs. Thus in an effort to clarify relations between the primary constructs and diagnostic groups, the pairwise comparisons for each of the HLM coefficients (which initially involved no covariates) were repeated using the demographic characteristics that were significantly associated with the given coefficient (as indicated in Table 12) as covariates, with the following exceptions. Age at DIS was not used as a covariate for the pairwise comparisons for Parent-Child Relationship (with the exception of the age-13 intercept; see Table 12) because the associations between this characteristic and the HLM coefficients for this domain were not independent of sample, as noted above. Risk status was not used as a covariate for the pairwise comparisons involving the APD group, for reasons discussed earlier. Finally, the demographic characteristics that significantly interacted with the HLM coefficient it was associated with in predicting group was not used as a covariate because such an interaction indicates that the coefficient is related to diagnostic outcome group differentially for different levels of the demographic characteristic; in such circumstances, using the demographic characteristic as a covariate would violate the homogeneity of regression assumption of analysis of covariance.

### *3.2.6 Associations Among HLM-derived Intercept and Slope Coefficients by Domain and by Age*

As summarized in Table 13, Spearman correlational analyses (using unweighted *ns*) indicated that within each domain, the associations between the intercept coefficients for ages 13

through 17 were positive and highly significant (all  $ps < .001$ ), as expected. Interestingly, the associations between the intercept and slope coefficients (see the last column of Table 13) indicate that for all four domains, higher levels of problematic experiences and behaviors at age 13 were related to a greater decline in such behaviors (i.e., more negative slopes) across adolescence.

As can be seen in Table 14, at each age assessed, the intercept coefficients across domains were positively and significantly associated. Correlations were generally medium in size and were strikingly similar across the associations assessed, ranging between .39 and .60. The slope coefficients for each domain were also positively and significantly related to each other, although these associations were generally small in size. As displayed, the strongest association observed among the slopes was between Schizophrenia-like Positive Symptoms and Peer Rejection (.41), whereas the smallest was between Peer Rejection and problematic Parent-Child Relationship (.12).

### ***3.3 Primary Analyses***

As noted, logistic regression was used for all primary analyses. Due to the directional hypotheses as well as to increase power, one-tailed tests were used except when noted otherwise. The alpha level was not adjusted for the primary analyses due to the limited power of the study when comparisons involved the psychosis group due to its small size (weighted  $n=16$ ). However, it is certainly recognized that the multiple group comparisons conducted with the primary constructs inflated the likelihood that a true null hypothesis was rejected. Thus the false discovery rate procedure (Curran-Everett, 2000), which seeks to control the number of true null hypotheses that are rejected, was used to calculate adjusted alpha levels for the primary pairwise

Table 13

*Spearman Correlations Among HLM-derived Intercept and Slope Coefficients by Domain for Schizophrenia-like Positive Symptoms, Schizophrenia-like Negative Symptoms, Peer Rejection, and Parent-Child Relationship*

Schizophrenia-like Positive Symptoms						
Coefficient	13	14	15	16	17	Slope
Age 13	-	.989**	.956**	.900**	.823**	-.297**
Age 14		-	.989**	.954**	.895**	-.172**
Age 15			-	.988**	.950**	-.043
Age 16				-	.987**	.090*
Age 17					-	.223**
Slope						-
Schizophrenia-like Negative Symptoms						
Coefficient	13	14	15	16	17	Slope
Age 13	-	.990**	.960**	.907**	.835**	-.357**
Age 14		-	.989**	.956**	.901**	-.238**
Age 15			-	.988**	.953**	-.109**
Age 16				-	.988**	.027
Age 17					-	.162**
Slope						-
Peer Rejection						
Coefficient	13	14	15	16	17	Slope
Age 13	-	.992**	.967**	.925**	.864**	-.435**
Age 14		-	.991**	.965**	.918**	-.337**
Age 15			-	.991**	.961**	-.230**
Age 16				-	.989**	-.119**
Age 17					-	-.002
Slope						-

Table 13 (continued)

Parent-Child Relationship						
Coefficient	13	14	15	16	17	Slope
Age 13	-	.990**	.960**	.908**	.838**	-.362**
Age 14		-	.989**	.957**	.904**	-.243**
Age 15			-	.989**	.956**	-.114**
Age 16				-	.989**	.020
Age 17					-	.154**
Slope						-

*Note.* Analyses unweighted because the weighting procedure cannot be implemented when calculating Spearman correlation coefficients.

\* $p < .05$ ; \*\* $p < .01$ .

Table 14

*Spearman Correlations Among HLM-derived Intercept Coefficients by Age and Among Slope Coefficients for Schizophrenia-like Positive Symptoms, Schizophrenia-like Negative Symptoms, Peer Rejection, and Parent-Child Relationship*

Coefficient	SP-SN	SP-PR	SP-PC	SN-PR	SN-PC	PR-PC
Age 13	.496**	.592**	.493**	.509**	.439**	.429**
Age 14	.507**	.599**	.483**	.533**	.447**	.442**
Age 15	.511**	.597**	.461**	.551**	.449**	.446**
Age 16	.502**	.583**	.432**	.560**	.438**	.438**
Age 17	.485**	.566**	.394**	.559**	.417**	.418**
Slope	.267**	.406**	.204**	.263**	.164**	.119**

*Note.* PC=Parent-Child Relationship; PR=Peer Rejection; SN=Schizophrenia-like Negative Symptoms; SP=Schizophrenia-like Positive Symptoms.

Analyses unweighted because the weighting procedure cannot be implemented when calculating Spearman correlation coefficients.

\*\* $p < .01$

group comparisons undertaken to test Hypotheses 1 through 4. The adjusted alpha levels were calculated by considering the five pairwise group comparisons for a given intercept or slope coefficient a family of comparisons, and then sequentially adjusting the alpha level for each comparison in an order determined by the significance levels of the comparisons. The pairwise comparisons of Hypotheses 1 through 4 that remained statistically significant when using this multiple-comparison procedure are indicated as such in Tables 16 through 19.

Hypotheses 1 through 4 concerned the relation of the four proposed antecedents to psychosis and the specificity of these relations to psychosis. Thus the focus of these hypotheses was on the pairwise diagnostic group comparisons on the four primary constructs of interest. Group differences at age 13 and in the growth trajectories (slopes) across adolescence were of principal interest. Based on this, as well as to help control for inflation of the probability of making a Type-I error, 4-group omnibus tests using multinomial logistic regression were conducted for each construct in which the age 13 intercept and slope served as the predictor variable in turn and group (psychosis, APD, depression/anxiety, controls) served as the criterion variable. All omnibus tests were two-tailed. Pairwise group comparisons were then conducted for each age and slope for a given construct if at least one of the omnibus tests for that construct indicated significant group differences. For these pairwise tests, each intercept or slope term served as the predictor variable in turn, and group served as the criterion variable. As noted, all primary analyses were first conducted without using covariates, and then were repeated when adjusting for key demographic characteristics that were selected for covariate use based on criteria specified earlier.

### 3.3.1 Hypothesis 1 – Schizophrenia-like Positive Symptoms

Figure 2 presents the mean estimated growth trajectories by group based on the HLM-derived age 13 intercept and slope coefficients for Schizophrenia-like Positive Symptoms. Table 15 provides the descriptive statistics of the HLM-derived coefficients for each domain by group. Four-group omnibus logistic regression analyses indicated that there were significant group differences in both the level of Schizophrenia-like Positive Symptoms at age 13 (age 13 intercept),  $\chi^2(3, N = 737) = 40.46, p < .001$ , and the rate of change (slope) in this construct across adolescence,  $\chi^2(3, N = 737) = 11.25, p = .010$ . Thus pairwise group comparisons were conducted to assess group differences in level at each age, as well as on slope; these results are summarized in Table 16.

*3.3.1.1 Hypothesis 1a – Psychosis versus controls: The level of Schizophrenia-like Positive Symptoms will be increased across the ages assessed among those of the psychosis group compared to the well control group, and the psychosis group will increase significantly more on this construct across adolescence (i.e. have a steeper positive slope) compared to controls.* As displayed in Table 16, the psychosis group boys obtained significantly higher levels of Schizophrenia-like Positive Symptoms than did controls across ages 13 to 17. Furthermore, as predicted, the psychosis group increased on this construct at a significantly greater rate across adolescence than did controls; see Figure 2. The effect sizes of the psychosis-control group differences were in the medium to large range (Cohen's  $d = .60$  to  $1.12$ ). As summarized in Table E1 of Appendix E, all of these associations remained significant when including covariates in the logistic regression analyses; please see Appendix E for the specific covariates used for each of the pairwise group comparisons.

Table 15

*Descriptive Statistics of HLM-derived Coefficients for Primary Constructs by Diagnostic Outcome Group*

Coefficient	Psychosis ( <i>n</i> =16) <sup>1</sup>	APD ( <i>n</i> =52) <sup>1</sup>	Depression/Anxiety ( <i>n</i> =22) <sup>1</sup>	Controls ( <i>n</i> =647) <sup>1</sup>
<b>Schizophrenia-like Positive Symptoms</b>				
Age 13 intercept	.280 (.743)	.529 (1.08)	.215 (.744)	-.102 (.561)
Age 14 intercept	.353 (.749)	.510 (1.01)	.238 (.696)	-.105 (.538)
Age 15 intercept	.426 (.802)	.490 (.954)	.260 (.679)	-.107 (.527)
Age 16 intercept	.499 (.894)	.470 (.930)	.283 (.695)	-.109 (.530)
Age 17 intercept	.572 (1.01)	.450 (.936)	.305 (.742)	-.111 (.545)
Linear slope (age)	.073 (.192)	-.020 (.167)	.022 (.151)	-.002 (.084)
Quadratic slope (age squared)	---	-.015 (.050) <sup>2</sup>	---	.007 (.035) <sup>2</sup>
<b>Schizophrenia-like Negative Symptoms</b>				
Age 13 intercept	.453 (.908)	.246 (.749)	.438 (.883)	-.063 (.603)
Age 14 intercept	.451 (.869)	.240 (.689)	.426 (.803)	-.065 (.576)
Age 15 intercept	.449 (.842)	.234 (.645)	.414 (.737)	-.067 (.560)
Age 16 intercept	.448 (.829)	.228 (.622)	.402 (.688)	-.069 (.556)
Age 17 intercept	.446 (.831)	.222 (.622)	.390 (.661)	-.072 (.563)
Linear slope (age)	-.002 (.110)	-.006 (.120)	-.012 (.128)	-.002 (.081)
Quadratic slope (age squared)	---	-.012 (.058) <sup>2</sup>	---	.008 (.051) <sup>2</sup>



Table 15 (continued)

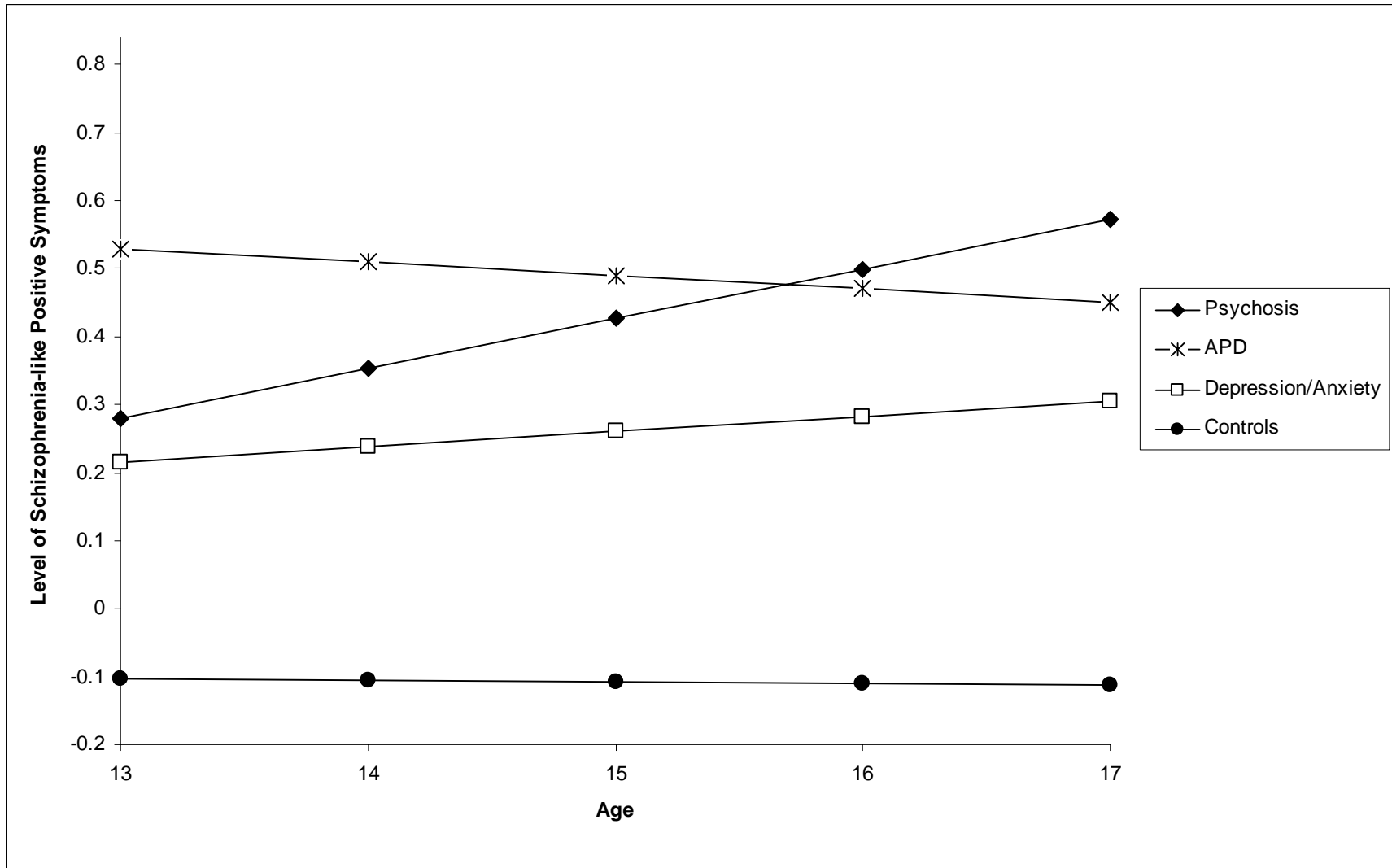
Coefficient	Psychosis ( <i>n</i> =16) <sup>1</sup>	APD ( <i>n</i> =52) <sup>1</sup>	Depression/Anxiety ( <i>n</i> =22) <sup>1</sup>	Controls ( <i>n</i> =647) <sup>1</sup>
<b>Peer Rejection</b>				
Age 13 intercept	.334 (.929)	.210 (.724)	.533 (.1.07)	-.058 (.659)
Age 14 intercept	.362 (.892)	.212 (.696)	.535 (1.01)	-.064 (.628)
Age 15 intercept	.390 (.871)	.214 (.685)	.536 (.956)	-.070 (.609)
Age 16 intercept	.418 (.865)	.215 (.692)	.537 (.914)	-.077 (.601)
Age 17 intercept	.446 (.875)	.217 (.717)	.539 (.885)	-.083 (.606)
Linear slope (age)	.028 (.118)	.002 (.112)	.001 (.116)	-.006 (.086)
Quadratic slope (age squared)	---	---	---	---
<b>Parent-Child Relationship</b>				
Age 13 intercept	.258 (.911)	.459 (.749)	.339 (.787)	-.107 (.752)
Age 14 intercept	.283 (.834)	.430 (.705)	.357 (.757)	-.105 (.717)
Age 15 intercept	.308 (.774)	.401 (.675)	.375 (.748)	-.102 (.697)
Age 16 intercept	.332 (.733)	.372 (.660)	.392 (.761)	-.099 (.693)
Age 17 intercept	.357 (.716)	.343 (.662)	.410 (.793)	-.097 (.706)
Linear slope (age)	.025 (.135)	-.029 (.105)	.018 (.127)	.003 (.108)
Quadratic slope (age squared)	---	-.015 (.064) <sup>2</sup>	---	.008 (.056) <sup>2</sup>

*Note.* Mean (standard deviation).

APD= antisocial personality disorder

<sup>1</sup>weighted *n*.

<sup>2</sup>The quadratic slope terms derived from models using both linear and quadratic components are presented here for the APD and control groups because the quadratic component of the slope significantly discriminated these two groups for Schizophrenia-like Positive Symptoms, Schizophrenia-like Negative Symptoms, and Parent-Child Relationship. Note, however, that the intercept and linear slope coefficients presented were derived from models using age as the only predictor.



*Figure 2.*

Mean estimated growth trajectories for Schizophrenia-like Positive Symptoms by group based on age-13 intercept and linear slope coefficients.

Table 16

*Schizophrenia-like Positive Symptoms: Logistic Regression Results of Pairwise Diagnostic Group Comparisons<sup>1</sup>*

Coefficient	Psychosis v. Ctrls <sup>2</sup>			Psychosis v. APD <sup>3</sup>			Psychosis v. Depression/Anxiety <sup>4</sup>			APD v. Ctrls <sup>5,6</sup>			Depression/Anxiety v. Ctrls <sup>7</sup>		
	<i>d</i> <sup>8</sup>	$\chi^2$	<i>p</i> <sup>9</sup>	<i>d</i> <sup>10</sup>	$\chi^2$	<i>p</i> <sup>9</sup>	<i>d</i> <sup>11</sup>	$\chi^2$	<i>p</i> <sup>9</sup>	<i>d</i> <sup>12</sup>	$\chi^2$	<i>p</i> <sup>9</sup>	<i>d</i> <sup>13</sup>	$\chi^2$	<i>p</i> <sup>9</sup>
Age 13 intercept	<b>.60</b>	<b>5.17</b>	<b>.012*</b>	-.39	.834	.181	.10	.073	.394	<b>.99</b>	<b>32.79</b>	<b>&lt;.001*</b>	<b>.50</b>	<b>5.16</b>	<b>.012*</b>
	<b>(Psychosis&gt;Ctrls)</b>									<b>(APD&gt;Ctrls)</b>			<b>(Dep/Anx&gt;Ctrls)</b>		
Age 14 intercept	<b>.75</b>	<b>7.62</b>	<b>.003*</b>	-.26	.357	.275	.19	.248	.310	<b>1.00</b>	<b>34.37</b>	<b>&lt;.001*</b>	<b>.56</b>	<b>6.41</b>	<b>.006*</b>
	<b>(Psychosis&gt;Ctrls)</b>									<b>(APD&gt;Ctrls)</b>			<b>(Dep/Anx &gt;Ctrls)</b>		
Age 15 intercept	<b>.89</b>	<b>10.21</b>	<b>&lt;.001*</b>	-.11	.061	.403	.28	.492	.242	<b>1.00</b>	<b>34.66</b>	<b>&lt;.001*</b>	<b>.62</b>	<b>7.55</b>	<b>.003*</b>
	<b>(Psychosis&gt;Ctrls)</b>									<b>(APD&gt;Ctrls)</b>			<b>(Dep/Anx &gt;Ctrls)</b>		
Age 16 intercept	<b>1.02</b>	<b>12.51</b>	<b>&lt;.001*</b>	.05	.012	.456	.36	.727	.197	<b>.97</b>	<b>33.23</b>	<b>&lt;.001*</b>	<b>.66</b>	<b>8.38</b>	<b>.002*</b>
	<b>(Psychosis&gt;Ctrls)</b>									<b>(APD&gt;Ctrls)</b>			<b>(Dep/Anx &gt;Ctrls)</b>		
Age 17 intercept	<b>1.12</b>	<b>14.21</b>	<b>&lt;.001*</b>	.20	.195	.330	.44	.904	.171	<b>.92</b>	<b>30.18</b>	<b>&lt;.001*</b>	<b>.68</b>	<b>8.79</b>	<b>.002*</b>
	<b>(Psychosis&gt;Ctrls)</b>									<b>(APD&gt;Ctrls)</b>			<b>(Dep/Anx &gt;Ctrls)</b>		
Linear slope (age)	<b>.79</b>	<b>9.05</b>	<b>.002*</b>	<b>.98</b>	<b>3.68</b>	<b>.028</b>	.53	.857	.178	-.18	1.64	.100	.26	1.72	.095
	<b>(Psychosis&gt;Ctrls)</b>			<b>(Psychosis&gt;APD)</b>											
Quadratic slope (age squared)	---			---			---			<b>-.59</b>	<b>13.88</b>	<b>&lt;.001</b>	---		
										<b>(Ctrls &gt;APD)</b>					

*Note.* APD= antisocial personality disorder; Ctrls=controls; Dep/Anx=Depression/Anxiety group; df=degrees of freedom; M=mean; N=number; Sd=standard deviation; v=versus

Table 16 (continued)

\* still considered significant after adjusting the alpha level for a given comparison using the false discovery rate procedure (Curran-Everett, 2000) when considering the five pairwise group comparisons for a given coefficient the family of comparisons on which the adjustments were based.

<sup>1</sup>Primary group comparisons, presented here, did not include use of covariates; please see text for report of results obtained when covariates used.

<sup>2</sup> $df = 1$ , and  $N = 663$

<sup>3</sup> $df = 1$ , and  $N = 68$

<sup>4</sup> $df = 1$ , and  $N = 38$

<sup>5</sup> $df = 1$ , and  $N = 699$

<sup>6</sup>For the APD-control group comparisons, the intercept and linear slope coefficients derived when using age as the only predictor were used. The quadratic slope results are also presented here because the quadratic component significantly discriminated these two groups on this domain. As expected, when the APD and control groups were compared on the intercept terms derived from the quadratic growth models for this domain, results were similar to those obtained when using the intercept terms of the linear growth models. Specifically,  $\chi^2$  ranged from 28.56 to 33.83, all  $ps < .001$ . Please see Figure E1 in Appendix E for a visual representation of the mean estimated quadratic growth trajectories for these two groups on this domain.

<sup>7</sup> $df = 1$ , and  $N = 669$

<sup>8</sup>Cohen's measure of effect size:  $(M_{\text{psychosis}} - M_{\text{control}}) / Sd$ .  $Sd$  of total sample excluding psychosis group used.

<sup>9</sup>one-tailed  $p$  values used for all comparisons due to a-priori hypotheses with exception of APD-control group comparison on quadratic slope.

<sup>10</sup> $(M_{\text{psychosis}} - M_{\text{APD}}) / Sd$ .  $Sd$  of total sample excluding psychosis group used.

<sup>11</sup> $(M_{\text{psychosis}} - M_{\text{depression/anxiety}}) / Sd$ .  $Sd$  of total sample excluding psychosis group used.

<sup>12</sup> $(M_{\text{APD}} - M_{\text{control}}) / Sd$ .  $Sd$  of total sample excluding psychosis group used.

<sup>13</sup> $(M_{\text{depression/anxiety}} - M_{\text{control}}) / Sd$ .  $Sd$  of total sample excluding psychosis group used.

3.3.1.2 *Hypothesis 1b – Psychosis versus APD: The level of Schizophrenia-like Positive Symptoms will be increased across the ages assessed among those of the psychosis group compared to the APD group, and the psychosis group will increase significantly more across adolescence (i.e. have a steeper positive slope) compared to the APD group.* The boys of the psychosis and APD groups did not differ significantly in their level of Schizophrenia-like Positive Symptoms for any of the ages assessed. However, as displayed in Figure 2, the pattern of change on this construct across adolescence discriminated the two groups. Specifically, as predicted, the psychosis group increased significantly more on positive symptoms than did the APD group boys, who obtained a weak but negative slope. Comparisons of the intercepts for ages 13 through 17 remained nonsignificant when including covariates in the analyses. As noted in Table E1, when including sample, parental education, and parental SES as covariates, the psychosis-APD linear slope difference was no longer significant. Additional analyses with each of these covariates separately suggested that this reduction in the association between slope and group was primarily due to adjusting for parental education and parental SES,  $\chi^2(1, N = 66) = 1.61, p = .10$ . As reviewed earlier, parental education and parental SES did not significantly differ between the psychosis and APD groups; however, there was a trend for the parents of the APD group to have significantly lower SES than the parents of the psychosis group boys,  $\chi^2(1, N = 66) = 3.58, p = .059$ ; see Table 9.

3.3.1.3 *Hypothesis 1b – Psychosis versus depression/anxiety: The level of Schizophrenia-like Positive Symptoms will be increased across the ages assessed among those of the psychosis group compared to the depression/anxiety group, and the psychosis group will increase significantly more across adolescence compared to the depression/anxiety group boys.* There were no significant differences between the psychosis and depression/anxiety groups in

level of Schizophrenia-like Positive Symptoms across the ages assessed. However, although the slopes for these two groups did not differ significantly, the effect size of the slope difference was in the medium range (Cohen's  $d = .53$ ), with the psychosis group increasing more than the depression/anxiety group across adolescence; see Figure 2. Consequently, although these two groups differed very little on positive symptoms at age 13 ( $d = .10$ ), the two groups became increasingly divergent across adolescence, resulting in a difference in the small-to-medium range ( $d = .44$ ) by age 17. All of these comparisons remained nonsignificant when including covariates in the analyses.

*3.3.1.4 Hypothesis 1b – APD versus controls: The level of Schizophrenia-like Positive Symptoms will not be increased across the ages assessed among those of the APD group relative to controls.* The APD group obtained significantly higher levels of Schizophrenia-like Positive Symptoms than did controls across ages 13 to 17. Further, although the linear slope estimates did not differ between the APD and control groups, the quadratic components of the group slopes significantly differed. Specifically, compared to controls, the APD group experienced a significant acceleration in their slight decrease in positive symptoms across adolescence; see Figure F1 of Appendix F. All of these associations remained significant when including covariates in the analyses.

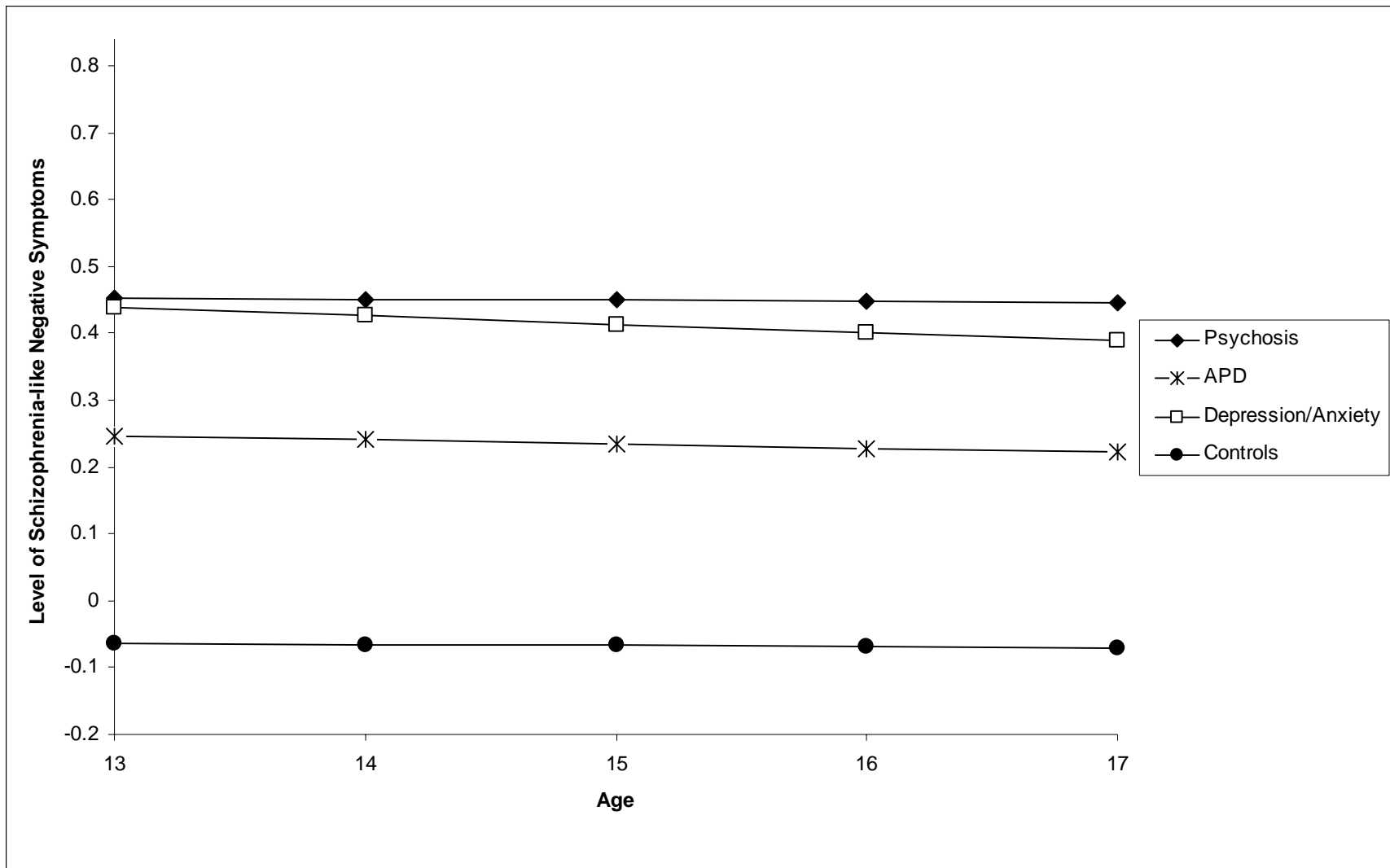
*3.3.1.5 Hypothesis 1b – Depression/anxiety versus controls: The level of Schizophrenia-like Positive Symptoms will not be increased across the ages assessed among those of the depression/anxiety group relative to controls.* The level of Schizophrenia-like Positive Symptoms was significantly higher among the boys of the depression/anxiety group than those

of the control group for all ages assessed. However, the rate of change on this construct across adolescence did not differ significantly between these two groups. This pattern of findings persisted when adjusting for demographic characteristics.

### **3.3.2 Hypothesis 2 – Schizophrenia-like Negative Symptoms**

Figure 3 displays the mean estimated growth trajectories by group for Schizophrenia-like Negative Symptoms. Omnibus logistic regression analyses indicated significant group differences in the level of Schizophrenia-like Negative Symptoms at age 13,  $\chi^2(3, N = 737) = 28.32, p < .001$ ); however, the rate of change in this construct across adolescence did not significantly differ by group,  $\chi^2(3, N = 737) = .36, p = .949$ . Pairwise group comparisons were conducted next to assess group differences in level of negative symptoms at each age, as well as on slope of this construct; these results are presented in Table 17.

*3.3.2.1 Hypothesis 2a – Psychosis versus controls: The level of Schizophrenia-like Negative Symptoms will be increased across the ages assessed among those of the psychosis group compared to the well control group, and the psychosis group will increase significantly more across adolescence (i.e. have a steeper positive slope) on this construct compared to controls.* As summarized in Table 17, the psychosis group obtained a significantly higher level of Schizophrenia-like Negative Symptoms than did controls across all ages assessed; Cohen's *d* indicated that the effect sizes of these differences were in the large range ( $d = .81$  to  $.89$ ). However, contrary to expectations, the flat slope obtained by the psychosis group for this construct was nearly identical to the slope of the controls; as can be seen, both groups experienced very little change in their level of negative symptoms across adolescence. This pattern of findings persisted when including covariates in the logistic regression analyses (see Table E2 of Appendix E).



*Figure 3.*

Mean estimated growth trajectories for Schizophrenia-like Negative Symptoms by group based on age-13 intercept and linear slope coefficients.



Table 17

*Schizophrenia-like Negative Symptoms: Logistic Regression Results of Pairwise Diagnostic Group Comparisons*

Coefficient	Psychosis v. Ctrls <sup>2</sup>			Psychosis v. APD <sup>3</sup>			Psychosis v. Depression/Anxiety <sup>4</sup>			APD v. Ctrls <sup>5,6</sup>			Depression/Anxiety v. Ctrls <sup>7</sup>		
	<i>d</i> <sup>8</sup>	$\chi^2$	<i>p</i> <sup>9</sup>	<i>d</i> <sup>10</sup>	$\chi^2$	<i>p</i> <sup>9</sup>	<i>d</i> <sup>11</sup>	$\chi^2$	<i>p</i> <sup>9</sup>	<i>d</i> <sup>12</sup>	$\chi^2$	<i>p</i> <sup>9</sup>	<i>d</i> <sup>13</sup>	$\chi^2$	<i>p</i> <sup>9</sup>
Age 13 intercept	<b>.81</b>	<b>9.11</b>	<b>.002*</b>	.33	.828	.182	.02	.003	.480	<b>.49</b>	<b>11.07</b>	<b>&lt;.001*</b>	<b>.79</b>	<b>11.85</b>	<b>&lt;.001*</b>
	<b>(Psychosis&gt;Ctrls)</b>									<b>(APD&gt;Ctrls)</b>			<b>(Dep/Anx&gt;Ctrls)</b>		
Age 14 intercept	<b>.86</b>	<b>9.90</b>	<b>.001*</b>	.35	.994	.160	.04	.009	.463	<b>.51</b>	<b>11.90</b>	<b>&lt;.001*</b>	<b>.81</b>	<b>12.58</b>	<b>&lt;.001*</b>
	<b>(Psychosis&gt;Ctrls)</b>									<b>(APD&gt;Ctrls)</b>			<b>(Dep/Anx &gt;Ctrls)</b>		
Age 15 intercept	<b>.89</b>	<b>10.45</b>	<b>&lt;.001*</b>	.37	1.15	.142	.06	.020	.444	<b>.52</b>	<b>12.38</b>	<b>&lt;.001*</b>	<b>.83</b>	<b>12.95</b>	<b>&lt;.001*</b>
	<b>(Psychosis&gt;Ctrls)</b>									<b>(APD&gt;Ctrls)</b>			<b>(Dep/Anx &gt;Ctrls)</b>		
Age 16 intercept	<b>.90</b>	<b>10.66</b>	<b>&lt;.001*</b>	.38	1.27	.130	.08	.036	.425	<b>.52</b>	<b>12.35</b>	<b>&lt;.001*</b>	<b>.82</b>	<b>12.82</b>	<b>&lt;.001*</b>
	<b>(Psychosis&gt;Ctrls)</b>									<b>(APD&gt;Ctrls)</b>			<b>(Dep/Anx &gt;Ctrls)</b>		
Age 17 intercept	<b>.89</b>	<b>10.46</b>	<b>&lt;.001*</b>	.39	1.32	.126	.10	.056	.406	<b>.51</b>	<b>11.78</b>	<b>&lt;.001*</b>	<b>.80</b>	<b>12.13</b>	<b>&lt;.001*</b>
	<b>(Psychosis&gt;Ctrls)</b>									<b>(APD&gt;Ctrls)</b>			<b>(Dep/Anx &gt;Ctrls)</b>		
Linear slope (age)	.004	.000	.493	.05	.016	.450	.12	.070	.340	-.04	.099	.377	-.11	.302	.292
Quadratic slope (age squared)		---			---			---		<b>-.46</b>	<b>6.35</b>	<b>.012</b>		---	
										<b>(Ctrls&gt;APD)</b>					

*Note.* APD= antisocial personality disorder; Ctrls=controls; Dep/Anx=Depression/Anxiety group; df=degrees of freedom; M=mean; N=number; Sd=standard deviation; v=versus

Table 17 (continued)

\* still considered significant after adjusting the alpha level for a given comparison using the false discovery rate procedure (Curran-Everett, 2000) when considering the five pairwise group comparisons for a given coefficient the family of comparisons on which the adjustments were based.

<sup>1</sup>Primary group comparisons, presented here, did not include use of covariates; please see text for report of results obtained when covariates used.

<sup>2</sup> $df = 1$ , and  $N = 663$

<sup>3</sup> $df = 1$ , and  $N = 68$

<sup>4</sup> $df = 1$ , and  $N = 38$

<sup>5</sup> $df = 1$ , and  $N = 699$

<sup>6</sup>For the APD-control group comparisons, the intercept and linear slope coefficients derived when using age as the only predictor were used. The quadratic slope results are also presented here because the quadratic component significantly discriminated these two groups on this domain. As expected, when the APD and control groups were compared on the intercept terms derived from the quadratic growth models for this domain, results were similar to those obtained when using the intercept terms of the linear growth models. Specifically,  $\chi^2$  ranged from 9.43 to 13.48, all  $ps < .05$ . Please see Figure E2 in Appendix E for a visual representation of the mean estimated quadratic growth trajectories for these two groups on this domain.

<sup>7</sup> $df = 1$ , and  $N = 669$

<sup>8</sup>Cohen's measure of effect size:  $(M_{\text{psychosis}} - M_{\text{control}}) / Sd$ .  $Sd$  of total sample excluding psychosis group used.

<sup>9</sup>one-tailed  $p$  values used for all comparisons due to a-priori hypotheses with exception of APD-control group comparison on quadratic slope.

<sup>10</sup> $(M_{\text{psychosis}} - M_{\text{APD}}) / Sd$ .  $Sd$  of total sample excluding psychosis group used.

<sup>11</sup> $(M_{\text{psychosis}} - M_{\text{depression/anxiety}}) / Sd$ .  $Sd$  of total sample excluding psychosis group used.

<sup>12</sup> $(M_{\text{APD}} - M_{\text{control}}) / Sd$ .  $Sd$  of total sample excluding psychosis group used.

<sup>13</sup> $(M_{\text{depression/anxiety}} - M_{\text{control}}) / Sd$ .  $Sd$  of total sample excluding psychosis group used.

3.3.2.2 *Hypothesis 2b – Psychosis versus APD: The level of Schizophrenia-like Negative Symptoms will be increased across the ages assessed among those of the psychosis group compared to the APD group, and the psychosis group will increase significantly more across adolescence on this construct compared to the APD boys.* There were no significant differences between the psychosis and APD groups in either the level of or rate of change in Schizophrenia-like Negative Symptoms across the ages assessed. All of these comparisons remained nonsignificant when including covariates in the analyses.

3.3.2.3 *Hypothesis 2b – Psychosis versus depression/anxiety: The level of Schizophrenia-like Negative Symptoms will be increased across the ages assessed among those of the psychosis group compared to the depression/anxiety group, and the psychosis group will increase significantly more across adolescence on this construct compared to the depression/anxiety group.* The boys of the psychosis and depression/anxiety groups did not differ significantly in either their level of Schizophrenia-like Negative Symptoms for any of the ages assessed or their rate of change in negative symptoms across adolescence. These comparisons remained nonsignificant when including covariates in the analyses.

3.3.2.4 *Hypothesis 2b – APD versus controls: The level of Schizophrenia-like Negative Symptoms will not be increased across the ages assessed among those of the APD group relative to controls.* Compared to controls, the boys of the APD group obtained significantly higher levels of Schizophrenia-like Negative Symptoms across all ages assessed. Further, as displayed in Figure F2 of Appendix F, although neither group showed significant change on negative symptoms across adolescence, the APD group experienced significantly more acceleration in their slight decrease on this construct over time than did the control group. All of these associations remained significant when including covariates in the analyses.

*3.3.2.5 Hypothesis 2b – Depression/anxiety versus controls: The level of Schizophrenia-like Negative Symptoms will be increased across the ages assessed among those of the depression/anxiety group compared to the well control group, and the depression/anxiety group will increase significantly more across adolescence on this construct compared to controls. As expected, the level of Schizophrenia-like Negative Symptoms was significantly higher among the boys of the depression/anxiety group than those of the control group at all ages assessed. However, these two groups did not differ significantly in their rate of change on this construct across adolescence. This pattern of findings remained unchanged when including covariates in the regression analyses.*

### **3.3.3 Hypothesis 3 – Peer Rejection**

Figure 4 presents the mean estimated growth trajectories by group for Peer Rejection. The 4-group omnibus logistic regression analyses indicated that there were significant group differences in level of Peer Rejection at age 13,  $\chi^2(3, N = 737) = 21.16, p < .001$ , although the growth trajectories of this construct did not differ by group,  $\chi^2(3, N = 737) = 2.68, p = .444$ . Thus pairwise group comparisons were conducted on this construct to assess group differences in level at each age, as well as on slope; these results are summarized in Table 18.

*3.3.3.1 Hypothesis 3a – Psychosis versus controls: The level of Peer Rejection will be increased across the ages assessed among those of the psychosis group compared to the well control group, and the psychosis group will increase significantly more across adolescence (i.e. have a steeper positive slope) on this construct compared to controls. As predicted, the boys of the psychosis group experienced significantly more Peer Rejection across all ages assessed than did controls; the effect sizes of these differences were in the medium to large range (Cohen's  $d = .57$  to  $.83$ ). However, although the psychosis group displayed a nonsignificant weak increase in*

peer rejection across adolescence, the slope of this group did not significantly differ from the slope of the controls. Comparisons of the intercepts for ages 13 through 17 remained significant when including covariates in the analyses (see Table E3 of Appendix E). Interestingly, when controlling for risk status, parental education, and parental SES, the weak increase of the psychosis group in peer rejection across adolescence became significantly greater than the near-zero slope of the control group. Recall that although the psychosis and control groups did not differ on risk status, parental education, or parental SES, these three demographic characteristics were associated with the slope of peer rejection among the total sample (see Table 12).

*3.3.3.2 Hypothesis 3b – Psychosis versus APD: The level of Peer Rejection will be increased across the ages assessed among those of the psychosis group compared to the APD group, and the psychosis group will increase significantly more across adolescence on this construct compared to the APD boys.* The psychosis and APD groups did not differ significantly in level of Peer Rejection for any of the ages assessed, nor did the groups display significantly different growth trajectories for this construct across adolescence. These comparisons remained nonsignificant when controlling for demographic covariates.

*3.3.3.3 Hypothesis 3b – Psychosis versus depression/anxiety: The level of Peer Rejection will be increased across the ages assessed among those of the psychosis group compared to the depression/anxiety group, and the psychosis group will increase significantly more across adolescence on this construct compared to the depression/anxiety group.* There were no significant differences in either the level of Peer Rejection at any of the ages assessed or the rate of change in Peer Rejection across adolescence between the psychosis and depression/anxiety groups. All of these associations remained nonsignificant when repeating the analyses with covariate adjustment.

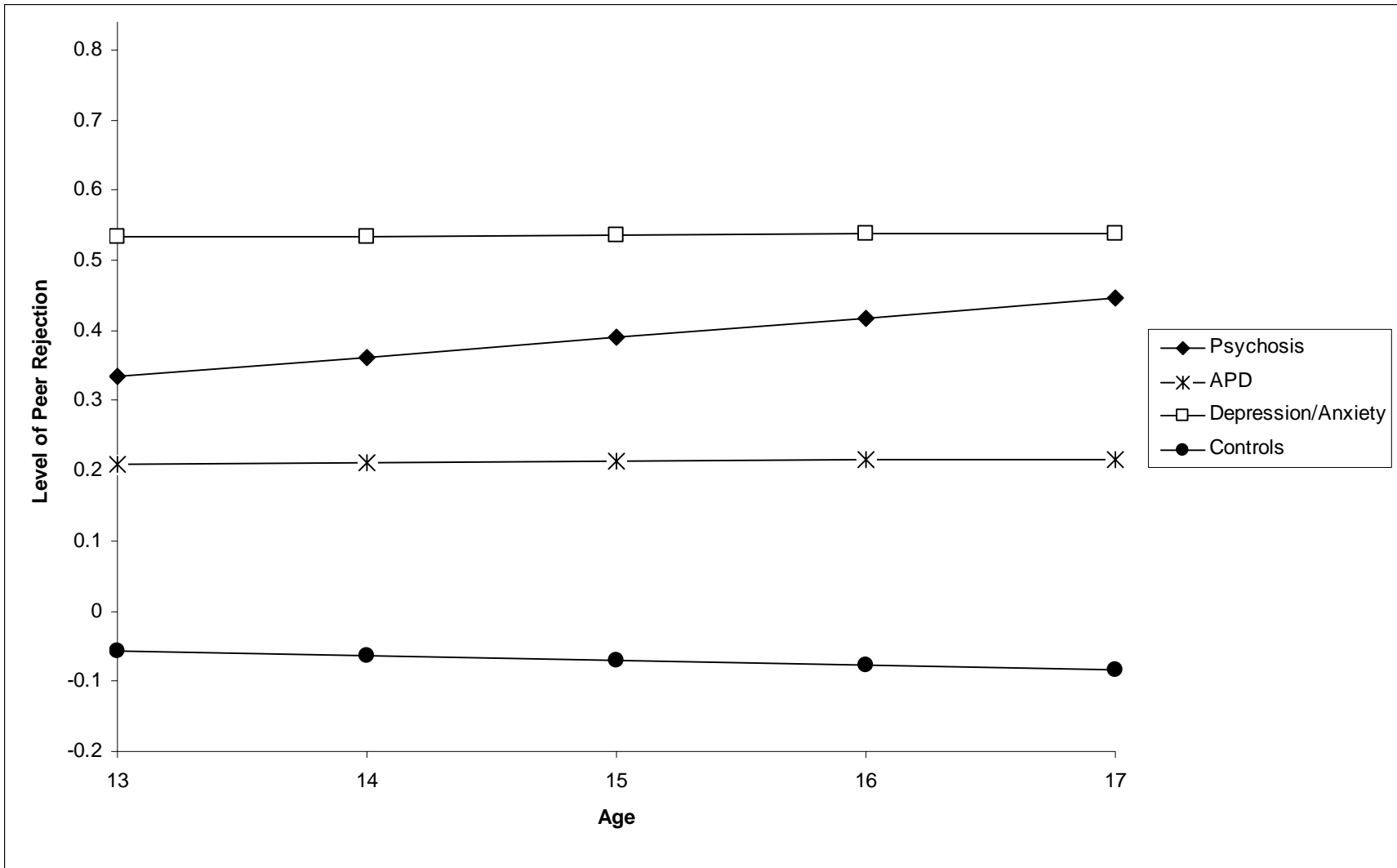


Figure 4.

Mean estimated growth trajectories for Peer Rejection by group based on age-13 intercept and linear slope coefficients

Table 18

Peer Rejection: Logistic Regression Results of Pairwise Diagnostic Group Comparisons<sup>1</sup>

Coefficient	Psychosis v. Ctrls <sup>2</sup>			Psychosis v. APD <sup>3</sup>			Psychosis v. Depression/Anxiety <sup>4</sup>			APD v. Ctrls <sup>5,6</sup>			Depression/Anxiety v. Ctrls <sup>7</sup>		
	<i>d</i> <sup>8</sup>	$\chi^2$	<i>p</i> <sup>9</sup>	<i>d</i> <sup>10</sup>	$\chi^2$	<i>p</i> <sup>9</sup>	<i>d</i> <sup>11</sup>	$\chi^2$	<i>p</i> <sup>9</sup>	<i>d</i> <sup>12</sup>	$\chi^2$	<i>p</i> <sup>9</sup>	<i>d</i> <sup>13</sup>	$\chi^2$	<i>p</i> <sup>9</sup>
Age 13 intercept	<b>.57</b>	<b>4.25</b>	<b>.020*</b>	.18	.307	.290	-.29	.381	.269	<b>.39</b>	<b>6.83</b>	<b>.005*</b>	<b>.86</b>	<b>11.79</b>	<b>&lt;.001*</b>
	<b>(Psychosis&gt;Ctrls)</b>									<b>(APD&gt;Ctrls)</b>			<b>(Dep/Anx&gt;Ctrls)</b>		
Age 14 intercept	<b>.65</b>	<b>5.34</b>	<b>.011*</b>	.23	.486	.243	-.26	.318	.287	<b>.42</b>	<b>7.83</b>	<b>.003*</b>	<b>.91</b>	<b>13.04</b>	<b>&lt;.001*</b>
	<b>(Psychosis&gt;Ctrls)</b>									<b>(APD&gt;Ctrls)</b>			<b>(Dep/Anx &gt;Ctrls)</b>		
Age 15 intercept	<b>.72</b>	<b>6.40</b>	<b>.006*</b>	.28	.693	.203	-.23	.247	.310	<b>.44</b>	<b>8.64</b>	<b>.002*</b>	<b>.95</b>	<b>14.01</b>	<b>&lt;.001*</b>
	<b>(Psychosis&gt;Ctrls)</b>									<b>(APD&gt;Ctrls)</b>			<b>(Dep/Anx &gt;Ctrls)</b>		
Age 16 intercept	<b>.78</b>	<b>7.31</b>	<b>.004*</b>	.32	.900	.172	-.19	.175	.338	<b>.46</b>	<b>9.17</b>	<b>.001*</b>	<b>.97</b>	<b>14.54</b>	<b>&lt;.001*</b>
	<b>(Psychosis&gt;Ctrls)</b>									<b>(APD&gt;Ctrls)</b>			<b>(Dep/Anx &gt;Ctrls)</b>		
Age 17 intercept	<b>.83</b>	<b>7.97</b>	<b>.003*</b>	.36	1.08	.150	-.15	.108	.371	<b>.47</b>	<b>9.33</b>	<b>.001*</b>	<b>.98</b>	<b>14.57</b>	<b>&lt;.001*</b>
	<b>(Psychosis&gt;Ctrls)</b>									<b>(APD&gt;Ctrls)</b>			<b>(Dep/Anx &gt;Ctrls)</b>		
Linear slope (age)	.38	2.44	.060	.30	.657	.209	.30	.502	.240	.09	.390	.266	.09	.165	.343

Note. APD= antisocial personality disorder; Ctrls=controls; Dep/Anx=Depression/Anxiety group; df=degrees of freedom; M=mean; N=number; Sd=standard deviation; v=versus

\* still considered significant after adjusting the alpha level for a given comparison using the false discovery rate procedure (Curran-Everett, 2000) when considering the five pairwise group comparisons for a given coefficient the family of comparisons on which the adjustments were based.

<sup>1</sup>Primary group comparisons, presented here, did not include use of covariates; please see text for report of results obtained when covariates used.

<sup>2</sup>df = 1, and N = 663

Table 18 (continued)

<sup>3</sup> $df = 1$ , and  $N = 68$

<sup>4</sup> $df = 1$ , and  $N = 38$

<sup>5</sup> $df = 1$ , and  $N = 699$

<sup>6</sup>Unlike other domains, APD-control group comparisons were not conducted for the quadratic slope term because, as noted, this term did not discriminate these two groups on Peer Rejection.

<sup>7</sup> $df = 1$ , and  $N = 669$

<sup>8</sup>Cohen's measure of effect size:  $(M_{\text{psychosis}} - M_{\text{control}}) / Sd$ .  $Sd$  of total sample excluding psychosis group used.

<sup>9</sup>one-tailed  $p$  values used for all comparisons due to a-priori hypotheses.

<sup>10</sup> $(M_{\text{psychosis}} - M_{\text{APD}}) / Sd$ .  $Sd$  of total sample excluding psychosis group used.

<sup>11</sup> $(M_{\text{psychosis}} - M_{\text{depression/anxiety}}) / Sd$ .  $Sd$  of total sample excluding psychosis group used.

<sup>12</sup> $(M_{\text{APD}} - M_{\text{control}}) / Sd$ .  $Sd$  of total sample excluding psychosis group used.

<sup>13</sup> $(M_{\text{depression/anxiety}} - M_{\text{control}}) / Sd$ .  $Sd$  of total sample excluding psychosis group used.



*3.3.3.4 Hypothesis 3b – APD versus controls: The level of Peer Rejection will be increased across the ages assessed among those of the APD group compared to the control group, and the APD group will increase significantly more on this construct across adolescence compared to controls.* As predicted, the boys of the APD group experienced significantly higher levels of Peer Rejection at all the ages assessed compared to controls. However, the near-flat growth trajectories the two groups obtained on this construct did not differ significantly. This pattern of findings remained unchanged when including covariates in the analyses.

*3.3.3.5 Hypothesis 3b – Depression/anxiety versus controls: The level of Peer Rejection will be increased across the ages assessed among those of the depression/anxiety group compared to the well control group, and the depression/anxiety group will increase significantly more across adolescence on this construct compared to controls.* As expected, boys of the depression/anxiety group obtained significantly higher levels of Peer Rejection across all ages assessed. However, both of these groups obtained near-zero slopes on this construct, which did not differ by group. This pattern of findings persisted when including covariates in the analyses.

#### **3.3.4 Hypothesis 4 – Problematic Parent-Child Relationship**

Figure 5 presents the mean estimated growth trajectories by group for problematic Parent-Child Relationship. Four-group omnibus logistic regression analyses indicated significant group differences in the level of this construct at age 13,  $\chi^2(3, N = 737) = 31.66, p < .001$ ; however, the rate of change on this construct across adolescence did not significantly differ by group,  $\chi^2(3, N = 737) = 5.28, p = .153$ . Pairwise group comparisons were conducted on this construct next to assess group differences in level at each age, as well as on slope; these results are presented in Table 19.

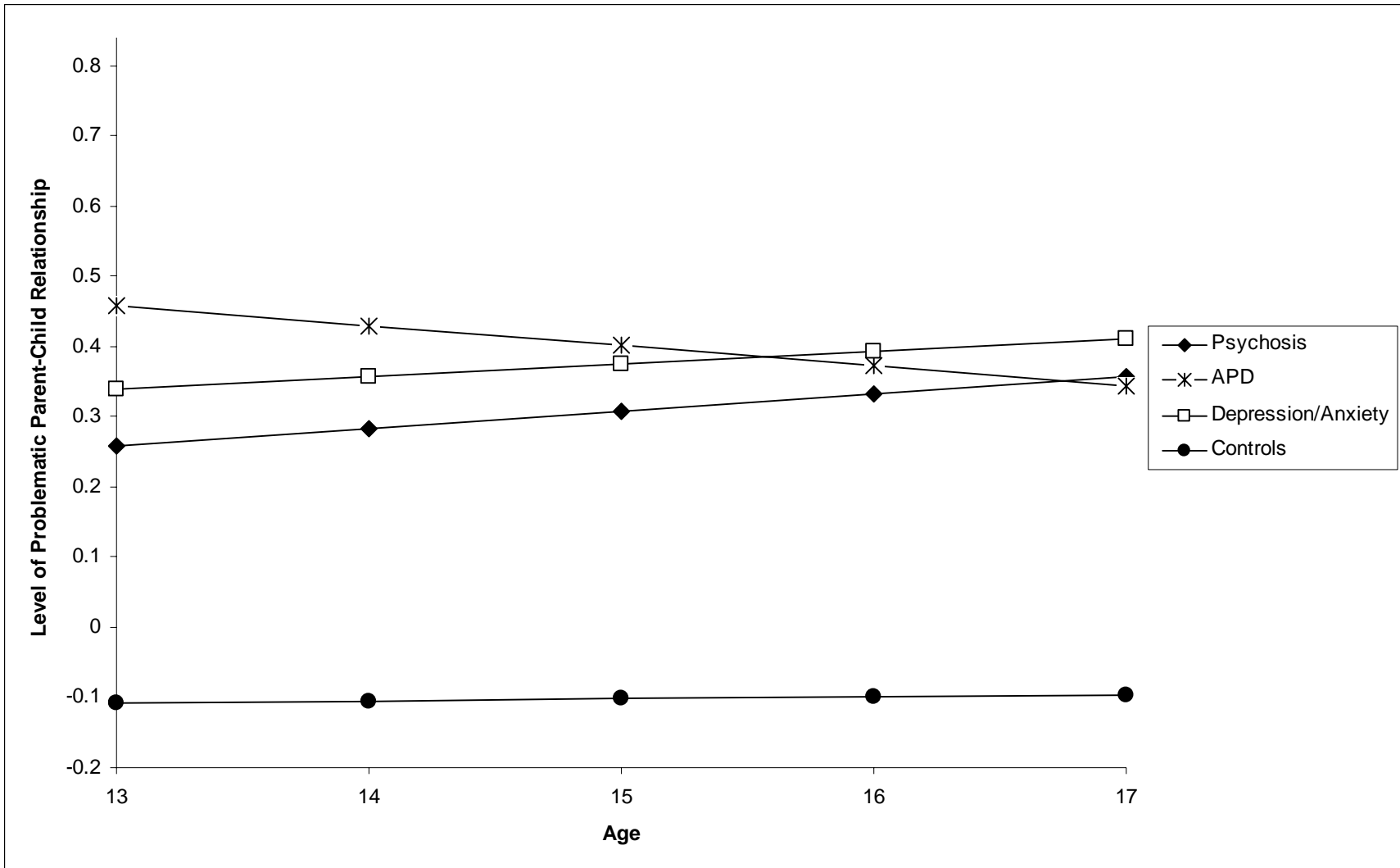


Figure 5.

Mean estimated growth trajectories for Parent-Child Relationship by group based on age-13 intercept and linear slope coefficients.

Table 19

*Parent-Child Relationship: Logistic Regression Results of Pairwise Diagnostic Group Comparisons*

Coefficient	Psychosis v. Ctrls <sup>2</sup>			Psychosis v. APD <sup>3</sup>			Psychosis v. Depression/Anxiety <sup>4</sup>			APD v. Ctrls <sup>5,6</sup>			Depression/Anxiety v. Ctrls <sup>7</sup>		
	<i>d</i> <sup>8</sup>	$\chi^2$	<i>p</i> <sup>9</sup>	<i>d</i> <sup>10</sup>	$\chi^2$	<i>p</i> <sup>9</sup>	<i>d</i> <sup>11</sup>	$\chi^2$	<i>p</i> <sup>9</sup>	<i>d</i> <sup>12</sup>	$\chi^2$	<i>p</i> <sup>9</sup>	<i>d</i> <sup>13</sup>	$\chi^2$	<i>p</i> <sup>9</sup>
Age 13 intercept	<b>.48</b>	<b>3.25</b>	<b>.036</b>	-.26	822.	.183	-.10	.089	.383	<b>.74</b>	<b>23.82</b>	<b>&lt;.001*</b>	<b>.58</b>	<b>6.70</b>	<b>.005*</b>
	<b>(Psychosis&gt;Ctrls)</b>									<b>(APD&gt;Ctrls)</b>			<b>(Dep/Anx&gt;Ctrls)</b>		
Age 14 intercept	<b>.53</b>	<b>4.03</b>	<b>.023*</b>	-.20	.502	.240	-.10	.085	.386	<b>.73</b>	<b>23.66</b>	<b>&lt;.001*</b>	<b>.63</b>	<b>7.87</b>	<b>.003*</b>
	<b>(Psychosis&gt;Ctrls)</b>									<b>(APD&gt;Ctrls)</b>			<b>(Dep/Anx &gt;Ctrls)</b>		
Age 15 intercept	<b>.58</b>	<b>4.77</b>	<b>.015*</b>	-.13	.223	.319	-.09	.075	.392	<b>.71</b>	<b>22.48</b>	<b>&lt;.001*</b>	<b>.67</b>	<b>8.87</b>	<b>.002*</b>
	<b>(Psychosis&gt;Ctrls)</b>									<b>(APD&gt;Ctrls)</b>			<b>(Dep/Anx &gt;Ctrls)</b>		
Age 16 intercept	<b>.61</b>	<b>5.37</b>	<b>.011*</b>	-.06	.042	.419	-.08	.062	.402	<b>.67</b>	<b>20.21</b>	<b>&lt;.001*</b>	<b>.70</b>	<b>9.50</b>	<b>.001*</b>
	<b>(Psychosis&gt;Ctrls)</b>									<b>(APD&gt;Ctrls)</b>			<b>(Dep/Anx &gt;Ctrls)</b>		
Age 17 intercept	<b>.63</b>	<b>5.71</b>	<b>.009*</b>	.02	.006	.471	-.07	.047	.414	<b>.61</b>	<b>17.13</b>	<b>&lt;.001*</b>	<b>.71</b>	<b>9.67</b>	<b>.001*</b>
	<b>(Psychosis&gt;Ctrls)</b>									<b>(APD&gt;Ctrls)</b>			<b>(Dep/Anx &gt;Ctrls)</b>		
Linear slope (age)	.20	.643	.212	<b>.49</b>	<b>2.77</b>	<b>.048</b>	.06	.027	.435	-.29	4.03	.023 <sup>14</sup>	.14	.424	.258
				<b>(Psychosis&gt;APD)</b>						<b>(Ctrls &gt;APD)</b>					
Quadratic slope (age squared)	---	---	---	---	---	---	---	---	---	<b>-.40</b>	<b>7.48</b>	<b>.006</b>	---	---	---
										<b>(Ctrls&gt;APD)</b>					

*Note.* APD= antisocial personality disorder; Ctrls=controls; Dep/Anx=Depression/Anxiety group; df=degrees of freedom; M=mean; N=number; Sd=standard deviation; v=versus

Table 19 (continued)

\* still considered significant after adjusting the alpha level for a given comparison using the false discovery rate procedure (Curran-Everett, 2000) when considering the five pairwise group comparisons for a given coefficient the family of comparisons on which the adjustments were based.

<sup>1</sup>Primary group comparisons, presented here, did not include use of covariates; please see text for report of results obtained when covariates used.

<sup>2</sup> $df = 1$ , and  $N = 663$

<sup>3</sup> $df = 1$ , and  $N = 68$

<sup>4</sup> $df = 1$ , and  $N = 38$

<sup>5</sup> $df = 1$ , and  $N = 699$

<sup>6</sup>For the APD-control group comparisons, the intercept and linear slope coefficients derived when using age as the only predictor were used. The quadratic slope results are also presented here because the quadratic component significantly discriminated these two groups on this domain. As expected, when the APD and control groups were compared on the intercept terms derived from the quadratic growth models for this domain, results were similar to those obtained when using the intercept terms of the linear growth models. Specifically,  $\chi^2$  ranged from 13.56 to 24.17, all  $ps < .001$ . Please see Figure E3 in Appendix E for a visual representation of the mean estimated quadratic growth trajectories for these two groups on this domain.

<sup>7</sup> $df = 1$ , and  $N = 669$

<sup>8</sup>Cohen's measure of effect size:  $(M_{\text{psychosis}} - M_{\text{control}}) / Sd$ .  $Sd$  of total sample excluding psychosis group used.

<sup>9</sup>one-tailed  $p$  values used for all comparisons due to a-priori hypotheses with exception of APD-control group comparison on quadratic slope.

<sup>10</sup> $(M_{\text{psychosis}} - M_{\text{APD}}) / Sd$ .  $Sd$  of total sample excluding psychosis group used.

<sup>11</sup> $(M_{\text{psychosis}} - M_{\text{depression/anxiety}}) / Sd$ .  $Sd$  of total sample excluding psychosis group used.

<sup>12</sup> $(M_{\text{APD}} - M_{\text{control}}) / Sd$ .  $Sd$  of total sample excluding psychosis group used.

<sup>13</sup> $(M_{\text{depression/anxiety}} - M_{\text{control}}) / Sd$ .  $Sd$  of total sample excluding psychosis group used.

<sup>14</sup> $p$  noted is one-tailed; note however that the direction of the difference is opposite of the one-tailed direction; thus this difference is not considered significant.

*3.3.4.1 Hypothesis 4a – Psychosis versus controls: The level of problematic Parent-Child Relationship will be increased across the ages assessed among those of the psychosis group compared to the well control group, and the psychosis group will increase significantly more across adolescence (i.e. have a steeper positive slope) on this construct compared to controls.* As predicted, the boys of the psychosis group obtained higher levels of problematic Parent-Child Relationship across all the ages assessed; Cohen's  $d$  indicated that the effect sizes of these differences were in the medium to large range ( $d = .48$  to  $.63$ ). However, contrary to expectations, the weak positive slope of the psychosis group boys did not differ significantly from the control group slope. As displayed in Table E4 of Appendix E, when including age at DIS and risk status as covariates, the psychosis-control group difference at age 13 was no longer significant, but remained a strong trend. Additional analyses with these covariates separately indicated that this reduction in the association between age 13 intercept and group was primarily due to adjusting for risk status,  $\chi^2(1, N = 663) = 2.33, p = .064$ . Although not significantly different on risk status, the psychosis group comprised 51% from the high risk group, whereas 34.5% of controls were classified as high risk (see Tables 8 and 9). The age 14 through 17 intercept comparisons remained significant when using demographic covariates.

*3.3.4.2 Hypothesis 4b – Psychosis versus APD: The level of problematic Parent-Child Relationship will be increased across the ages assessed among those of the psychosis group compared to the APD group, and the psychosis group will increase significantly more across adolescence on this construct compared to the APD boys.* Contrary to expectations, the psychosis and APD groups did not differ significantly in level of problematic Parent-Child Relationship at any of the ages assessed. However, as predicted, the pattern of change in this construct across adolescence significantly discriminated these two groups. Specifically, as

shown in Figure 5, the psychosis group boys displayed a shallow increase across age on the index of problematic Parent-Child Relationship, whereas the APD boys showed a weak decrease. This pattern of findings persisted when using covariate adjustment.

*3.3.4.3 Hypothesis 4b – Psychosis versus depression/anxiety: The level of problematic Parent-Child Relationship will be increased across the ages assessed among those of the psychosis group compared to the depression/anxiety group, and the psychosis group will increase significantly more across adolescence on this construct compared to the depression/anxiety group.* The psychosis and depression/anxiety groups did not differ significantly in level of problematic Parent-Child Relationship at any of the ages assessed. Furthermore, the two groups obtained very similar growth trajectories for this construct, which were characterized by a nonsignificant but mild increase across adolescence. These associations remained nonsignificant when including covariates in the analyses.

*3.3.4.4 Hypothesis 4b – APD versus controls: The level of problematic Parent-Child Relationship will be increased across the ages assessed among those of the APD group compared to the control group, and the APD group will increase significantly more on this construct across adolescence compared to controls.* As predicted, the boys of the APD group obtained significantly higher levels of problematic Parent-Child Relationship than did controls across all ages assessed. However, comparing the linear and quadratic components of the group slopes indicated that when compared to the controls, who showed very little change across age, the APD group decreased slightly on this construct but in an accelerating fashion over time (see Figure F3 of Appendix F). This pattern of findings persisted when repeating analyses using covariates.

*3.3.4.5 Hypothesis 4b – Depression/anxiety versus controls: The level of problematic Parent-Child Relationship will be increased across the ages assessed among those of the depression/anxiety group compared to the well control group, and the depression/anxiety group will increase significantly more across adolescence on this construct compared to controls. As expected, boys of the depression/anxiety group obtained significantly higher levels of problematic Parent-Child Relationship than did controls at all the ages assessed. However, the weak positive slope that the depression/anxiety group displayed on this construct did not differ significantly from the near-zero slope obtained by the controls. This pattern of findings did not change when adjusting for demographic characteristics*

***3.3.5 Hypothesis 5 – The Moderating Effect of Schizophrenia-like Symptoms:***

*Schizophrenia-like symptoms will moderate the relation between stressor exposure and later psychosis. Specifically, there will be a significant interaction between psychosocial stressors and schizophrenia-like symptoms in predicting psychosis.* For Hypothesis 5, the standardized combined-informant versions of the primary constructs were used to create higher-order composite constructs. Specifically, for each primary construct, the values of the construct for each age were averaged across all the ages assessed. The averaged values for Schizophrenia-like Positive Symptoms and Schizophrenia-like Negative Symptoms were then summed to create the new construct, Schizophrenia-like Symptoms. Similarly, the averaged values for Peer Rejection and Parent-Child Relationship were summed to create the Stressors construct. These new constructs, as well as their product (Schizophrenia-like Symptoms X Stressors), were used as terms in logistic regression to test Hypothesis 5. Specifically, after entering Schizophrenia-like Symptoms and Stressors as predictor variables at Step 1, their product term was entered at

Step 2 to test for a significant interaction in predicting group membership. Group (psychosis versus controls) served as the criterion variable. This analysis indicated that the interaction term did not significantly predict psychosis,  $\chi^2(1, N = 663) = .01, p = .485$ .

### ***3.4 Secondary Analyses***

Secondary analyses were conducted to address questions that were beyond the scope of the primary hypotheses but that importantly pertain to the implications of the findings presented thus far, as well as to further explore questions that were prompted by the preliminary and primary results. Although there were a number of interesting questions that could have been explored, this endeavor was limited to the analyses that were viewed as most critical to informing the primary results and their implications.

#### ***3.4.1 Controlling for Substance Dependence***

As presented in Tables 10 and 11 and reviewed earlier, almost half the boys of the psychosis group (43.3%) met criteria for substance dependence. Due to the striking difference between the psychosis and control groups in the percentage of boys receiving a diagnosis of substance dependence (4.0% among controls;  $\chi^2(1, N = 663) = 52.11, p < .001$ ), the question arose regarding the possible contribution of the diagnosis of substance dependence to the psychosis-control group differences observed on the primary constructs. Before directly addressing this question, the associations between substance dependence (represented by a dichotomous variable-yes/no) and the intercept and slope coefficients for each domain were examined among the total sample using logistic regression. These analyses indicated that the boys with a diagnosis of substance dependence obtained significantly higher levels of both



Schizophrenia-like Positive Symptoms and Peer Rejection than did boys without substance dependence across ages 13 to 17 ( $\chi^2$  ranged from 4.40 to 9.03, all  $ps < .01$ , two-tailed). Similarly, the boys with a diagnosis of substance dependence obtained significantly greater levels of problematic Parent-Child Relationship at ages 14 and 15 compared to those without such a diagnosis,  $\chi^2 (1, N = 737) = 3.80$  and  $3.75$ , respectively,  $ps < .053$ , two-tailed; further, trend differences were present between the two groups on this construct at ages 13, 14, and 17 (substance dependence greater than no substance dependence;  $ps$  ranged from  $.055$  to  $.064$ , two-tailed). Although a trend difference suggested that those with a substance dependence diagnosis obtained a higher level of Schizophrenia-like Negative Symptoms at age 17 than the boys without substance dependence ( $p=.083$ , two-tailed), no significant differences were observed between these two groups on negative symptoms for the ages assessed (for ages 13 to 16, all  $ps > .10$ , two-tailed). Further, these two groups did not differ in their rate of change on any of the four primary constructs across adolescence (all  $ps > .10$ , two-tailed).

Although the primary interest was in assessing the impact of controlling for substance dependence on psychosis-control group differences, the APD and depression/anxiety groups also comprised significantly more boys who met substance dependence criteria than did the control group (see Tables 10 and 11). Thus all of the primary group comparisons were repeated using substance dependence as a covariate; these results are presented in Tables 20 through 23. As can be seen by comparing these tables to the corresponding tables of the primary analyses (Tables 16 through 19), when controlling for substance dependence, there was a reduction in  $\chi^2$  for all of the psychosis-control group comparisons. However, even after this adjustment, compared to controls the psychosis group continued to be characterized by significantly higher levels of Schizophrenia-like Positive Symptoms, Schizophrenia-like Negative Symptoms, Peer Rejection,

Table 20

*Schizophrenia-like Positive Symptoms when including Substance Dependence as a Covariate: Logistic Regression Results of Pairwise Group Comparisons*

Coefficient	Psychosis v. Ctrls	Psychosis v. APD	Psychosis v. Depression/Anxiety	APD v. Ctrls <sup>1</sup>	Depression/Anxiety v. Ctrls
	$\chi^2; p^2$	$\chi^2; p^2$	$\chi^2; p^2$	$\chi^2; p^2$	$\chi^2; p^2$
Age 13 intercept	<b>3.53; .030</b> (Psychosis>Ctrls)	.923; .169	.368; .272	<b>27.14; &lt;.001</b> (APD>Ctrls)	<b>4.43; .018</b> (Dep/Anx>Ctrls)
Age 14 intercept	<b>5.59; .009</b> (Psychosis>Ctrls)	.464; .248	.664; .208	<b>28.47; &lt;.001</b> (APD>Ctrls)	<b>5.63; .009</b> (Dep/Anx >Ctrls)
Age 15 intercept	<b>7.72; .003</b> (Psychosis>Ctrls)	.138; .355	.960; .164	<b>28.67; &lt;.001</b> (APD>Ctrls)	<b>6.74; .005</b> (Dep/Anx >Ctrls)
Age 16 intercept	<b>9.51; .001</b> (Psychosis>Ctrls)	.003; .478	1.17; .140	<b>27.38; &lt;.001</b> (APD>Ctrls)	<b>7.56; .003</b> (Dep/Anx >Ctrls)
Age 17 intercept	<b>10.76; &lt;.001</b> (Psychosis>Ctrls)	.058; .405	1.28; .129	<b>24.75; &lt;.001</b> (APD>Ctrls)	<b>8.02; .003</b> (Dep/Anx >Ctrls)
Linear slope (age)	<b>6.93; .004</b> (Psychosis>Ctrls)	<b>3.04; .041</b> (Psychosis>APD)	.640; .212	1.06; .152	2.15; .072
Quadratic slope (age squared)	---	---	---	<b>11.91; .001</b> (Ctrls>APD)	---

*Note.* APD= antisocial personality disorder; Ctrls=controls; Dep/Anx=Depression/Anxiety group; v=versus

<sup>1</sup>For the APD-control group comparisons, the intercept and linear slope coefficients derived when using age as the only predictor were used. The quadratic slope results are also presented here because the quadratic component significantly discriminated these two groups on this domain.

<sup>2</sup>one-tailed *p* values used for all comparisons in direction of primary hypotheses with exception of APD-control group comparisons on quadratic slope.

Table 21

*Schizophrenia-like Negative Symptoms when including Substance Dependence as a Covariate: Logistic Regression Results of Pairwise Group Comparisons*

Coefficient	Psychosis v. Ctrls	Psychosis v. APD	Psychosis v. Depression/Anxiety	APD v. Ctrls <sup>1</sup>	Depression/Anxiety v. Ctrls
	$\chi^2; p^2$	$\chi^2; p^2$	$\chi^2; p^2$	$\chi^2; p^2$	$\chi^2; p^2$
Age 13 intercept	<b>8.74; .002</b> (Psychosis>Ctrls)	.930; .168	.432; .256	<b>9.10; .002</b> (APD>Ctrls)	--- <sup>3</sup>
Age 14 intercept	<b>9.21; .001</b> (Psychosis>Ctrls)	1.06; .152	.442; .253	<b>9.80; .001</b> (APD>Ctrls)	--- <sup>3</sup>
Age 15 intercept	<b>9.41; .001</b> (Psychosis>Ctrls)	1.16; .141	.436; .255	<b>10.21; &lt;.001</b> (APD>Ctrls)	--- <sup>3</sup>
Age 16 intercept	<b>9.27; .001</b> (Psychosis>Ctrls)	1.22; .135	.410; .261	<b>10.21; &lt;.001</b> (APD>Ctrls)	--- <sup>3</sup>
Age 17 intercept	<b>8.81; .002</b> (Psychosis>Ctrls)	1.20; .137	.364; .274	<b>9.77; .001</b> (APD>Ctrls)	<b>12.03; &lt;.001</b> (Dep/Anx >Ctrls)
Linear slope (age)	--- <sup>3</sup>	.001; .491	.063; .401	.072; .394	.393; .266
Quadratic slope (age squared)	---	---	---	<b>5.27; .022</b> (Ctrls>APD)	---

*Note.* APD= antisocial personality disorder; Ctrls=controls; Dep/Anx=Depression/Anxiety group; v=versus

<sup>1</sup>For the APD-control group comparisons, the intercept and linear slope coefficients derived when using age as the only predictor were used. The quadratic slope results are also presented here because the quadratic component significantly discriminated these two groups on this domain.

<sup>2</sup>one-tailed *p* values used for all comparisons in direction of primary hypotheses with exception of APD-control group comparisons on quadratic slope.

<sup>3</sup>substance dependence was not used as a covariate to test this comparison because it significantly interacted with this HLM coefficient in predicting group.

Table 22

*Peer Rejection when including Substance Dependence as a Covariate: Logistic Regression Results of Pairwise Group Comparisons*

Coefficient	Psychosis v. Ctrls	Psychosis v. APD	Psychosis v. Depression/Anxiety	APD v. Ctrls	Depression/Anxiety v. Ctrls
	$\chi^2; p^1$	$\chi^2; p^1$	$\chi^2; p^1$	$\chi^2; p^1$	$\chi^2; p^1$
Age 13 intercept	2.54; .056*	.349; .278	.085; .386	<b>4.54; .017</b> (APD>Ctrls)	<b>10.44; &lt;.001</b> (Dep/Anx>Ctrls)
Age 14 intercept	<b>3.37; .034</b> (Psychosis>Ctrls)	.468; .247	.070; .396	<b>5.22; .011</b> (APD>Ctrls)	<b>11.64; &lt;.001</b> (Dep/Anx >Ctrls)
Age 15 intercept	<b>4.20; .021</b> (Psychosis>Ctrls)	.590; .222	.054; .408	<b>5.78; .008</b> (APD>Ctrls)	<b>12.55; &lt;.001</b> (Dep/Anx >Ctrls)
Age 16 intercept	<b>4.90; .014</b> (Psychosis>Ctrls)	.698; .202	.038; .423	<b>6.12; .007</b> (APD>Ctrls)	<b>13.01; &lt;.001</b> (Dep/Anx >Ctrls)
Age 17 intercept	<b>5.39; .010</b> (Psychosis>Ctrls)	.780; .189	.023; .440	<b>6.21; .007</b> (APD>Ctrls)	<b>12.96; &lt;.001</b> (Dep/Anx >Ctrls)
Linear slope (age)	1.53; .108	.271; .302	.115; .368	.225; .318	.119; .365

*Note.* APD= antisocial personality disorder; Ctrls=controls; Dep/Anx=Depression/Anxiety group; v=versus

\* indicates a change from primary analyses regarding whether the difference between the groups was statistically significant.

<sup>1</sup>one-tailed *p* values used for all comparisons in direction of primary hypotheses.

Table 23

*Parent-Child Relationship when including Substance Dependence as a Covariate: Logistic Regression Results of Pairwise Group Comparisons*

Coefficient	Psychosis v. Ctrls	Psychosis v. APD	Psychosis v. Depression/Anxiety	APD v. Ctrls <sup>1</sup>	Depression/Anxiety v. Ctrls
	$\chi^2; p^2$	$\chi^2; p^2$	$\chi^2; p^2$	$\chi^2; p^2$	$\chi^2; p^2$
Age 13 intercept	2.48; .058*	.733; .196	.002; .481	<b>20.21; &lt;.001</b> (APD>Ctrls)	<b>6.13; .007</b> (Dep/Anx>Ctrls)
Age 14 intercept	<b>3.17; .038</b> (Psychosis>Ctrls)	.437; .254	.007; .467	<b>20.12; &lt;.001</b> (APD>Ctrls)	<b>7.28; .004</b> (Dep/Anx >Ctrls)
Age 15 intercept	<b>3.91; .024</b> (Psychosis>Ctrls)	.182; .335	.014; .453	<b>19.19; &lt;.001</b> (APD>Ctrls)	<b>8.32; .002</b> (Dep/Anx >Ctrls)
Age 16 intercept	<b>4.60; .016</b> (Psychosis>Ctrls)	.025; .437	.023; .440	<b>17.36; &lt;.001</b> (APD>Ctrls)	<b>9.08; .002</b> (Dep/Anx >Ctrls)
Age 17 intercept	<b>5.14; .012</b> (Psychosis>Ctrls)	.016; .451	.033; .428	<b>14.82; &lt;.001</b> (APD>Ctrls)	<b>9.42; .001</b> (Dep/Anx >Ctrls)
Linear slope (age)	630.; .214	<b>2.66; .052</b> (Psychosis>APD)	.032; .429	3.85; .025 <sup>3</sup> (Ctrls>APD)	.438; .254
Quadratic slope (age squared)	---	---	---	<b>5.16; .023</b> (Ctrls>APD)	---

*Note.* APD= antisocial personality disorder; Ctrls=controls; Dep/Anx=Depression/Anxiety group; v=versus

\* indicates a change from primary analyses regarding whether the difference between the groups was statistically significant.

<sup>1</sup>For the APD-control group comparisons, the intercept and linear slope coefficients derived when using age as the only predictor were used. The quadratic slope results are also presented here because the quadratic component significantly discriminated these two groups on this domain.

<sup>2</sup>one-tailed *p* values used for all comparisons in direction of primary hypotheses with exception of APD-control group comparisons on quadratic slope. <sup>3</sup>*p* noted is one-tailed; note however that the direction of the difference is opposite of the one-tailed direction; thus this difference is not considered significant.

and problematic Parent-Child Relationship across nearly all of the ages assessed, as well as by a significantly greater increase across adolescence on Schizophrenia-like Positive Symptoms. The exceptions to this were the significantly greater levels of Peer Rejection and problematic Parent-Child Relationship at age 13 among the psychosis group boys observed with the primary analyses: When controlling for substance dependence, the psychosis-control group differences on these intercepts were reduced to a strong trend (see Tables 22 and 23). It should be noted that several of the pairwise group comparisons on Schizophrenia-like Negative Symptoms could not be assessed with this covariate adjustment because substance dependence significantly interacted with the given HLM coefficient in predicting group (see Table 21 for the specific comparisons affected).

As can be seen, the pattern of findings yielded by the primary analyses persisted when controlling for substance dependence for all of the other pairwise group comparisons (i.e., for psychosis versus APD, psychosis versus depression/anxiety, APD versus controls, and depression/anxiety versus controls across all ages and slopes for all four domains).

#### *3.4.2 Psychosis Versus All Other Groups Combined*

The inclusion of the two clinical control groups (i.e., APD and depression/anxiety) in addition to the well control group was an important component of this study because it permitted questions of specificity to be addressed. At the same time, however, an argument could be made that pulling out the boys who met criteria for APD, depressive disorders, and/or anxiety disorders from the primary control group resulted in a “super-clean” control group that was not representative of the population-based sample of the PYS. This is an important consideration for the present study, because one of its more general aims was to contribute to efforts to identify

predictors of psychosis development among the general population. Thus to address questions regarding group differences between the boys who developed psychotic symptoms by early adulthood compared to those who did not, logistic regression analyses were used to compare the psychosis group to all other groups combined on the intercept and slope coefficients for the four domains; these results, along with the corresponding descriptive statistics, are presented in Table 24.

As can be seen by comparing the 3<sup>rd</sup> and 4<sup>th</sup> columns of Table 24 to the tables of the primary analyses (Tables 16 through 19), the pattern of differences between the psychosis and other groups combined across the four domains was very similar to differences observed between the psychosis and well control groups. Specifically, compared to the other groups combined, the psychosis group obtained significantly higher levels of Schizophrenia-like Positive Symptoms, Schizophrenia-like Negative Symptoms, Peer Rejection, and problematic Parent-Child Relationship across nearly all of the ages assessed; further, they increased significantly more on Schizophrenia-like Positive Symptoms across adolescence compared to the other groups combined. The exception was the level of problematic Parent-Child Relationship at age 13: The difference between the psychosis group and other groups combined on this intercept was at the trend level ( $p=.065$ ), whereas the psychosis group was significantly increased on this intercept compared to well controls.

### *3.4.3 Controlling for Age of Onset*

The current study sought to examine the adolescent functioning of boys who later developed full psychotic symptoms. However, it is possible that some of the psychosis group boys experienced full psychotic symptoms between the ages of 13 and 17. Likewise, it is possible that some of the boys in the depression/anxiety group met criteria for a depressive

Table 24

*Descriptive Statistics of HLM-derived Coefficients and Logistic Regression Results for Comparison of Psychosis versus Other Groups Combined on Primary Constructs*

Coefficient	Psychosis <sup>1</sup> (n=16) <sup>2</sup>	Other groups combined <sup>1,3</sup> (n=721) <sup>2</sup>	$\chi^2$	p <sup>4</sup>
<b>Schizophrenia-like Positive Symptoms</b>				
Age 13 intercept	.280 (.743)	-.047 (.641)	<b>2.94</b>	<b>.044</b>
Age 14 intercept	.353 (.749)	-.050 (.612)	<b>4.59</b>	<b>.016</b>
Age 15 intercept	.426 (.802)	-.052 (.596)	<b>6.41</b>	<b>.006</b>
Age 16 intercept	.499 (.894)	-.055 (.595)	<b>8.13</b>	<b>.002</b>
Age 17 intercept	.572 (1.01)	-.058 (.609)	<b>9.55</b>	<b>.001</b>
Linear slope	.073 (.192)	-.003 (.095)	<b>8.14</b>	<b>.002</b>
<b>Schizophrenia-like Negative Symptoms</b>				
Age 13 intercept	.453 (.908)	-.025 (.634)	<b>7.17</b>	<b>.004</b>
Age 14 intercept	.451 (.869)	-.028 (.603)	<b>7.96</b>	<b>.003</b>
Age 15 intercept	.449 (.842)	-.031 (.583)	<b>8.58</b>	<b>.002</b>
Age 16 intercept	.448 (.829)	-.033 (.575)	<b>8.91</b>	<b>.002</b>
Age 17 intercept	.446 (.831)	-.036 (.580)	<b>8.85</b>	<b>.002</b>
Linear slope	-.002 (.110)	-.003 (.086)	.002	.483



Table 24 (continued)

Coefficient	Psychosis <sup>1</sup> ( <i>n</i> =16) <sup>2</sup>	Other groups combined <sup>1,3</sup> ( <i>n</i> =721) <sup>2</sup>	$\chi^2$	<i>p</i> <sup>4</sup>
<b>Peer Rejection</b>				
Age 13 intercept	.334 (.929)	-.020 (.689)	<b>3.25</b>	<b>.036</b>
Age 14 intercept	.362 (.892)	-.025 (.659)	<b>4.14</b>	<b>.021</b>
Age 15 intercept	.390 (.871)	-.031 (.639)	<b>5.04</b>	<b>.013</b>
Age 16 intercept	.418 (.865)	-.037 (.632)	<b>5.85</b>	<b>.008</b>
Age 17 intercept	.446 (.875)	-.042 (.637)	<b>6.47</b>	<b>.006</b>
Linear slope	.028 (.118)	-.006 (.089)	2.17	.071
<b>Parent-Child Relationship</b>				
Age 13 intercept	.258 (.911)	-.052 (.770)	2.30	.065
Age 14 intercept	.283 (.834)	-.052 (.733)	<b>4.14</b>	<b>.021</b>
Age 15 intercept	.308 (.774)	-.051 (.712)	<b>3.58</b>	<b>.029</b>
Age 16 intercept	.332 (.733)	-.050 (.707)	<b>4.13</b>	<b>.021</b>
Age 17 intercept	.357 (.716)	-.049 (.719)	<b>4.50</b>	<b>.017</b>
Linear slope	.025 (.135)	.001 (.109)	.746	.194

*Note.*

<sup>1</sup>Mean (standard deviation).

<sup>2</sup>weighted *n*.

<sup>3</sup>APD, depression/anxiety, and healthy control groups combined.

<sup>4</sup>one-tailed *p* values used for all comparisons.

and/or anxiety disorder before age 17. Thus unfortunately the findings reviewed above do not permit the conclusion that the primary constructs reflect precursors, rather than corollaries, of the psychopathology of interest. In an effort to address this limitation, the primary pairwise comparisons for ages 13 through 15 were repeated while excluding both the psychosis group boys who reported first experiencing any psychotic symptom before the age of 16, and the depression/anxiety group boys who reported first experiencing anxiety or depressive symptoms characteristic of the specific disorder(s) for which they met criteria (e.g., panic attacks, fears characteristic of social phobia, compulsive behavior characteristic of OCD, depressive symptoms for two weeks or more) before the age of 16. These exclusions reduced the psychosis group from 16 boys to 10, and the depression/anxiety group from 22 to 9 boys (weighted *ns*). Such attempts to control for age of onset could not be made for the APD group because by definition all of the boys meeting APD criteria displayed behaviors characteristic of this disorder before the age of 15. The age of symptom onset was determined by using participant responses from the DIS. Using such DIS information for age of onset is an especially conservative and limited approach because 1) it dates not the onset of persisting psychotic symptoms or when boys first met criteria for a depressive and/or anxiety disorder, but the initial occurrence of symptoms (although as noted above, age of symptom onset for major depressive disorder signified the age at which the participant reported first experiencing depressive symptoms for two weeks or more); and 2) this information was retrospectively collected (the DIS was administered when the boys were a mean age of 22), and in some cases, the span of time between the age at which boys reported first having symptoms and DIS administration was over 10 years; this increases the likelihood that this information provided only a rough estimate of the onset of full symptoms. It

is also acknowledged that using this information to index age of onset may have resulted in a loss of information regarding the very phenomena of interest (e.g., transient psychotic experiences before full psychosis onset) in some instances. Further, as noted above, excluding boys based on symptom onset reduced the size of the psychosis and depression/anxiety groups considerably, which both reduced the power of the comparisons involving these groups and the likelihood that these groups were representative of boys who later develop full psychotic symptoms or depressive and/or anxiety disorders. However, because no other age of onset information is provided by the DIS or was available through other sources, it was deemed to be the best approach to address this issue.

Figures 6 through 9 present the mean estimated growth trajectories (for ages 13 through 15 only) by group for the four primary constructs based on the HLM age 13 intercept and slope coefficients that were calculated with the total sample adjusted for age of onset, as described above. Table 25 provides the corresponding descriptive statistics of the HLM coefficients for each domain by group. As displayed in Tables 26 through 29, logistic regression analyses indicated that in contrast to the primary results, when controlling for age of onset, there were no significant differences between the psychosis and control groups in level of Schizophrenia-like Positive Symptoms, Peer Rejection, or problematic Parent-Child Relationship from ages 13 to 15. Further, although the psychosis group obtained a very weak positive slope for Schizophrenia-like Positive Symptoms, it did not differ significantly from the near-zero negative slope of the controls. The psychosis group boys did continue to display significantly higher levels of Schizophrenia-like Negative Symptoms for ages 13 through 15. Interestingly, this reduced psychosis group also displayed a significantly greater increase on the index of

problematic Parent-Child Relationship from ages 13 to 15 compared to controls (see Figure 9), whereas the full psychosis group did not differ significantly from controls on this slope (from ages 13 through 17).

The pattern of findings obtained for other pairwise comparisons was generally similar to that yielded by the primary analyses. However, as can be seen by comparing Figures 6 through 9 with the corresponding primary figures (Figures 2 through 5), in general the reduced psychosis and depression/anxiety groups were more distinct from each other with regard to the level of problematic behaviors they displayed from ages 13 through 15, as well as in their pattern of change across these ages, than were the full groups. This is reflected in the significant differences in slope between the psychosis and depression/anxiety groups for Schizophrenia-like Positive Symptoms and Peer Rejection; as can be seen, the depression/anxiety group started out at a higher level and decreased significantly more from ages 13 to 15 than did the reduced psychosis group, which showed relatively little change on these constructs across early adolescence.

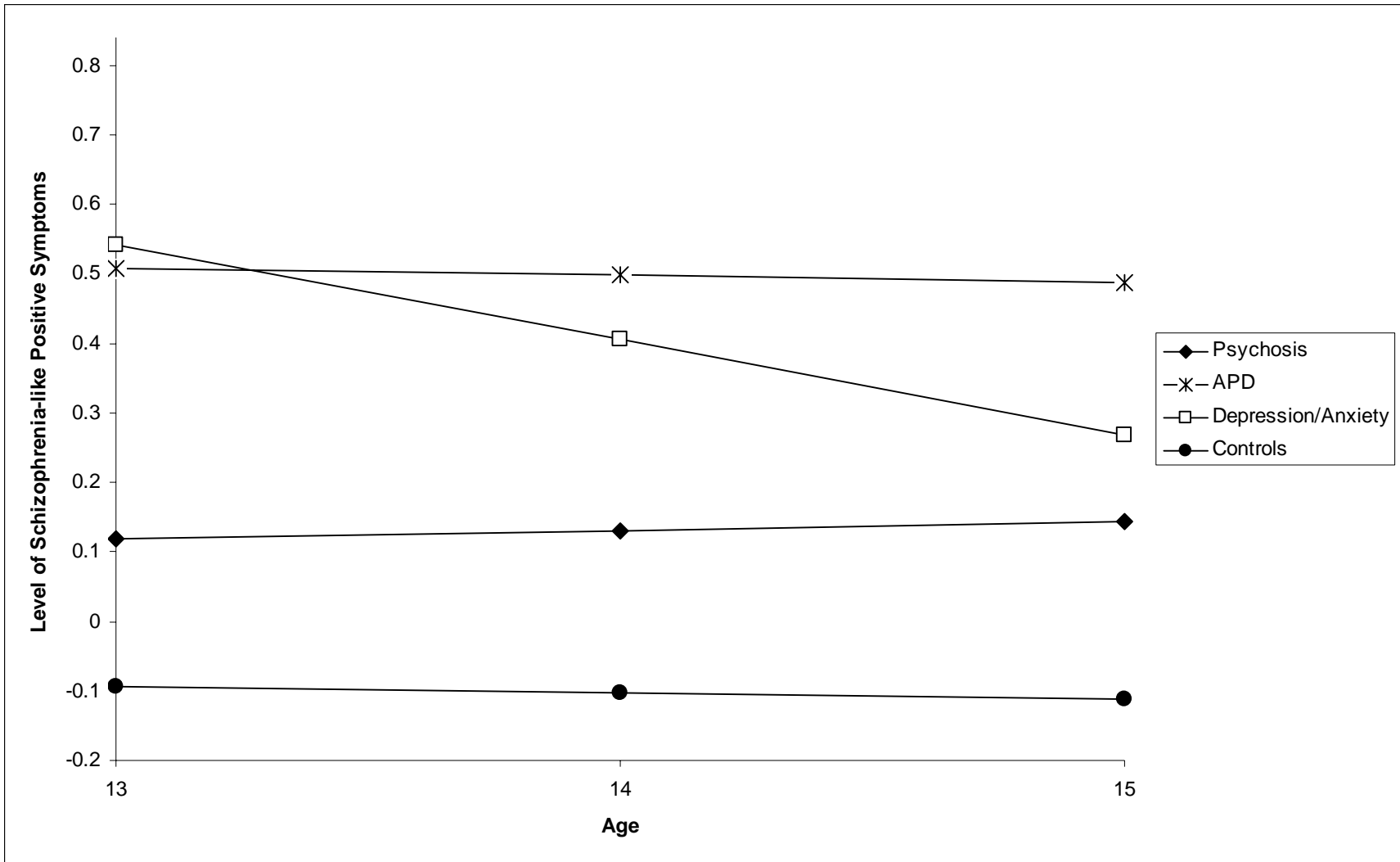


Figure 6.

Mean estimated growth trajectories for Schizophrenia-like Positive Symptoms by group based on age-13 intercept and linear slope coefficients when controlling for age of onset.

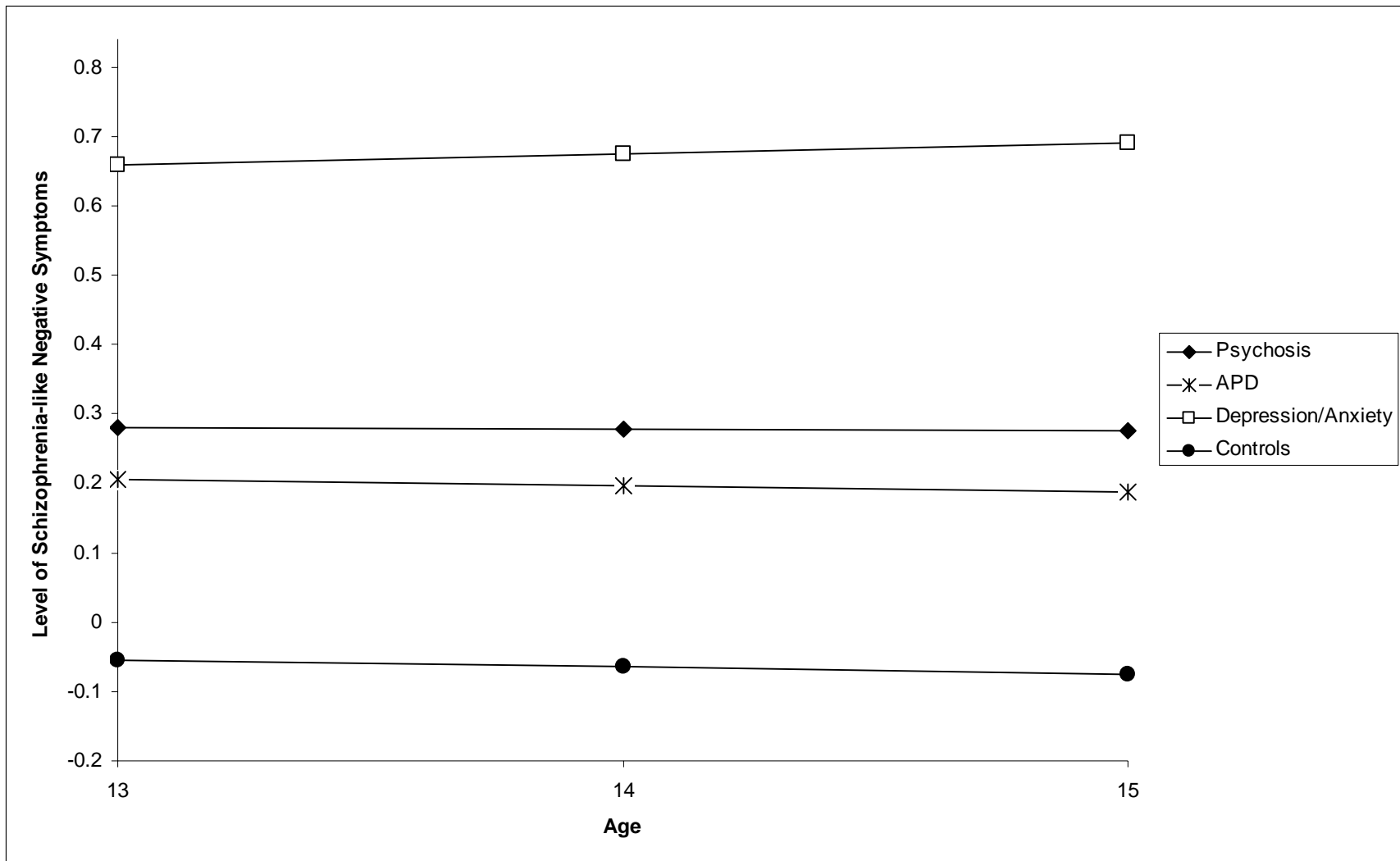


Figure 7.

Mean estimated growth trajectories for Schizophrenia-like Negative Symptoms by group based on age-13 intercept and linear slope coefficients when controlling for age of onset.

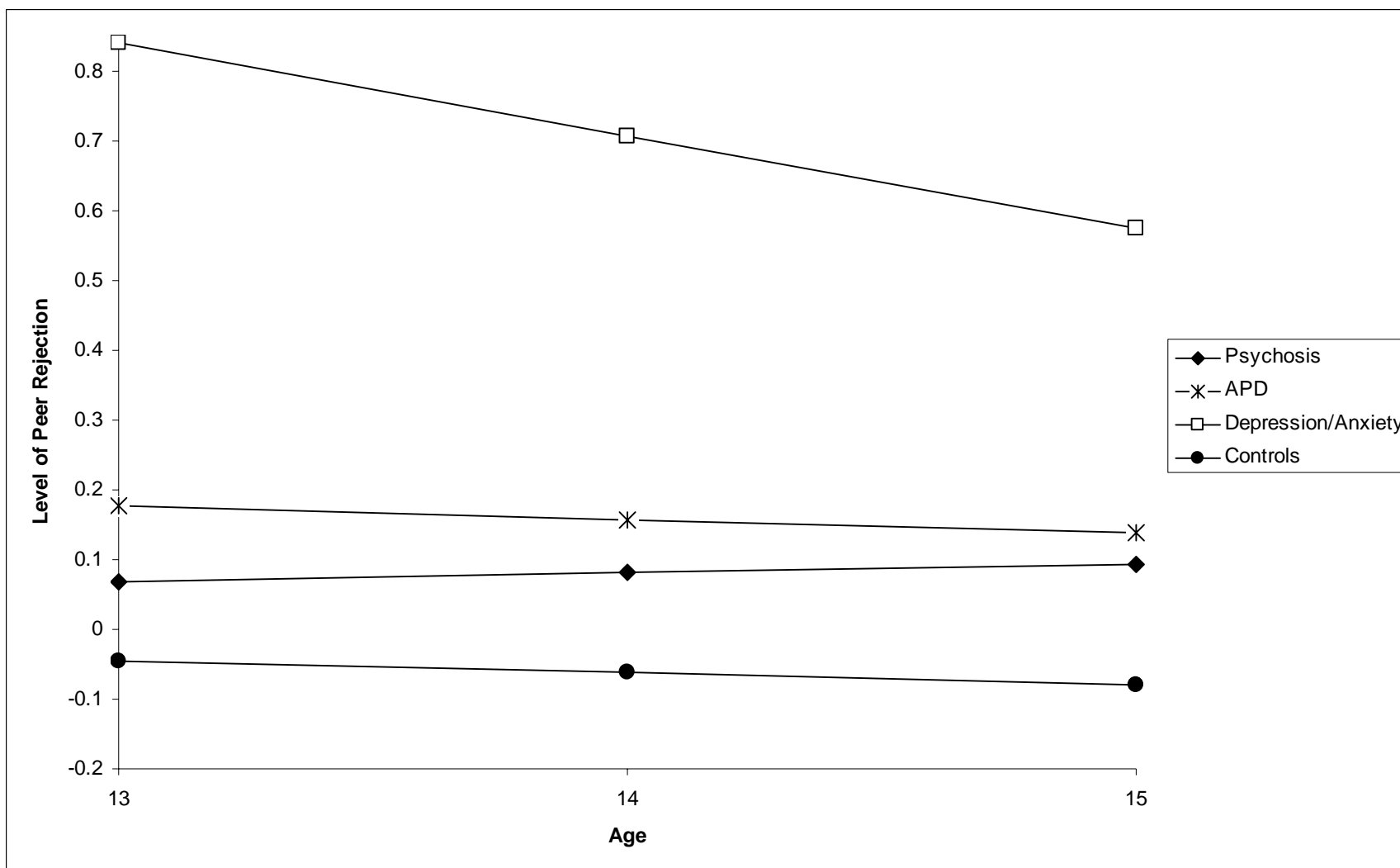


Figure 8.  
 Mean estimated growth trajectories for Peer Rejection by group based on age-13 intercept and linear slope coefficients when controlling for age of onset.

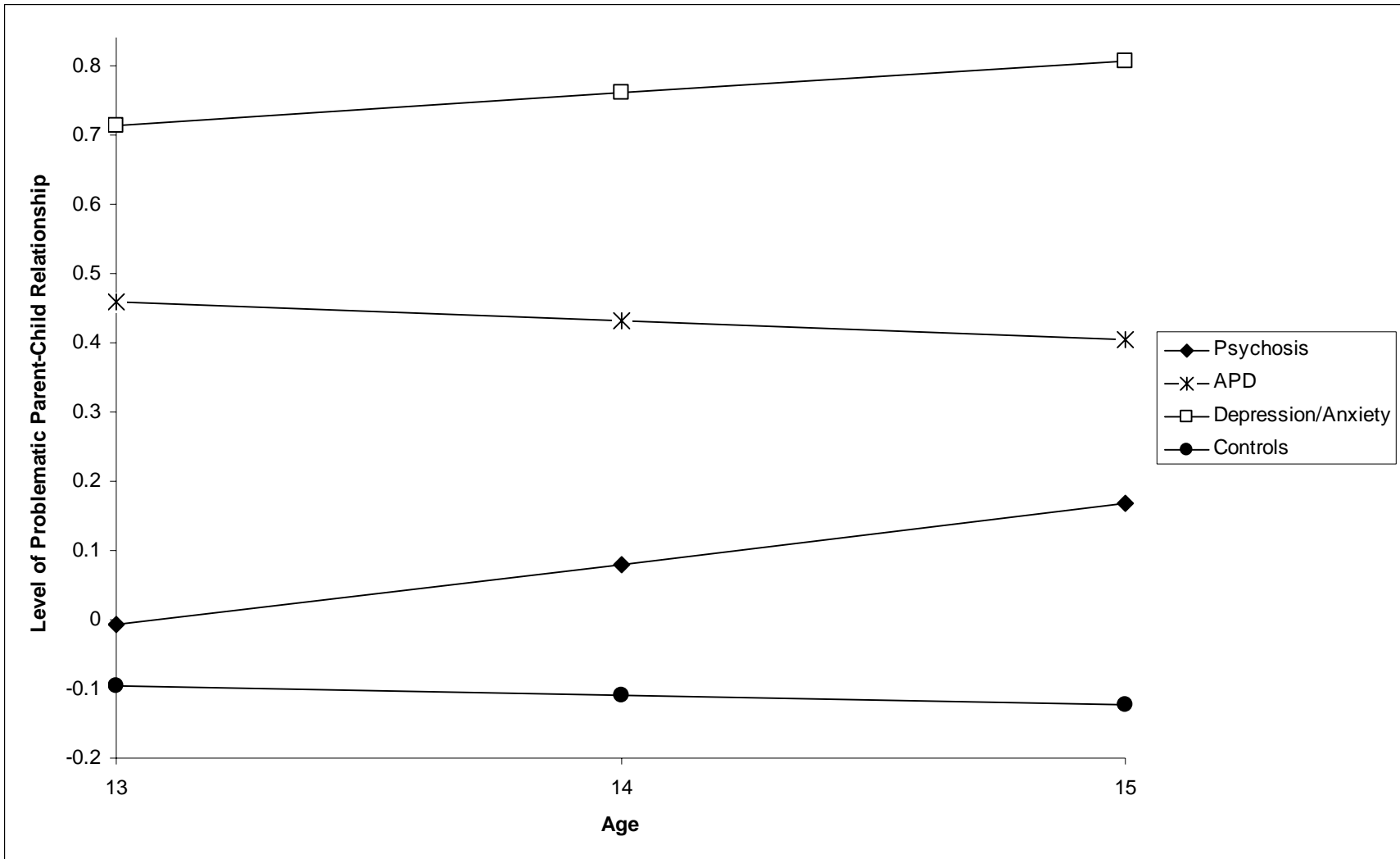


Figure 9.

Mean estimated growth trajectories for Parent-Child Relationship by group based on age-13 intercept and linear slope coefficients when controlling for age.



Table 25

*Controlling for Age of Onset: Descriptive Statistics of HLM-derived Coefficients for Primary Constructs by Diagnostic Outcome Group*

Coefficient	Psychosis ( <i>n</i> =10) <sup>1</sup>	APD ( <i>n</i> =52) <sup>1</sup>	Depression/Anxiety ( <i>n</i> =9) <sup>1</sup>	Controls ( <i>n</i> =647) <sup>1</sup>
<b>Schizophrenia-like Positive Symptoms</b>				
Age 13 intercept	.118 (.757)	.508 (1.10)	.541 (1.17)	-.093 (.551)
Age 14 intercept	.131 (.773)	.498 (1.05)	.405 (.939)	-.103 (.556)
Age 15 intercept	.143 (.806)	.488 (1.03)	.269 (.718)	-.113 (.585)
Linear slope (age)	.013 (.122)	-.010 (.196)	-.136 (.246)	-.010 (.116)
<b>Schizophrenia-like Negative Symptoms</b>				
Age 13 intercept	.280 (.817)	.206 (.769)	.658 (1.09)	-.055 (.588)
Age 14 intercept	.277 (.782)	.196 (.729)	.675 (1.03)	-.065 (.576)
Age 15 intercept	.275 (.782)	.187 (.704)	.692 (.992)	-.074 (.582)
Linear slope (age)	-.002 (.169)	-.010 (.113)	.016 (.139)	-.009 (.103)
<b>Peer Rejection</b>				
Age 13 intercept	.069 (.874)	.178 (.731)	.839 (1.44)	-.045 (.661)
Age 14 intercept	.081 (.824)	.157 (.695)	.707 (1.29)	-.062 (.635)
Age 15 intercept	.093 (.803)	.137 (.692)	.574 (1.14)	-.080 (.631)
Linear slope (age)	.012 (.162)	-.020 (.153)	-.132 (.179)	-.018 (.121)
<b>Parent-Child Relationship</b>				
Age 13 intercept	-.006 (.812)	.458 (.748)	.712 (.954)	-.096 (.751)
Age 14 intercept	.080 (.861)	.431 (.744)	.759 (.943)	-.109 (.739)
Age 15 intercept	.167 (.940)	.404 (.777)	.807 (.950)	-.122 (.753)
Linear slope (age)	.086 (.165)	-.027 (.169)	.047 (.131)	-.013 (.138)

*Note.* Mean (standard deviation).

APD= antisocial personality disorder

<sup>1</sup>weighted *n*.

Table 26

*Schizophrenia-like Positive Symptoms When Controlling for Age of Onset: Logistic Regression Results of Pairwise Group Comparisons*

Coefficient	Psychosis v. Ctrls	Psychosis v. APD	Psychosis v. Depression/Anxiety	APD v. Ctrls	Depression/Anxiety v. Ctrls
	$\chi^2; p^1$	$\chi^2; p^1$	$\chi^2; p^1$	$\chi^2; p^1$	$\chi^2; p^1$
Age 13 intercept	1.20; .137	1.50; .111	.977; .162	<b>30.41; &lt;.001</b> (APD>Ctrls)	<b>6.91; .005</b> (Dep/Anx>Ctrls)
Age 14 intercept	1.40.; .119	1.42; .117	.534; .233	<b>30.79; &lt;.001</b> (APD>Ctrls)	<b>4.71; .015</b> (Dep/Anx >Ctrls)
Age 15 intercept	1.47; .113	1.26; .131	.140; .354	<b>28.84; &lt;.001</b> (APD>Ctrls)	<b>2.59; .054</b> (Dep/Anx >Ctrls)
Linear slope (age)	.349; .278	.133 .358	<b>3.41 .033</b> (Psychosis > Dep/Anx)	.000; .500	8.38; .002 <sup>2</sup> (Ctrls > Dep/Anx)

*Note.* APD= antisocial personality disorder; Ctrls=controls; Dep/Anx=Depression/Anxiety group; v=versus  
<sup>1</sup>one-tailed *p* values used for all comparisons in direction of primary hypotheses.

<sup>2</sup>*p* noted is one-tailed; note however that the direction of the difference is opposite of the one-tailed direction; thus this difference is not considered significant.

Table 27

*Schizophrenia-like Negative Symptoms When Controlling for Age of Onset: Logistic Regression Results of Pairwise Group Comparisons*

Coefficient	Psychosis v. Ctrls	Psychosis v. APD	Psychosis v. Depression/Anxiety	APD v. Ctrls	Depression/Anxiety v. Ctrls
	$\chi^2; p^1$	$\chi^2; p^1$	$\chi^2; p^1$	$\chi^2; p^1$	$\chi^2; p^1$
Age 13 intercept	<b>2.83; .046</b> (Psychosis>Ctrls)	.078; .390	.810; .184	<b>8.29; .002</b> (APD>Ctrls)	<b>9.38; .001</b> (Dep/Anx>Ctrls)
Age 14 intercept	<b>3.09; .040</b> (Psychosis>Ctrls)	.105; .373	.982; .161	<b>8.75; .002</b> (APD>Ctrls)	<b>10.60; &lt;.001</b> (Dep/Anx >Ctrls)
Age 15 intercept	<b>3.17; .038</b> (Psychosis>Ctrls)	.132; .358	1.13; .145	<b>8.69; .002</b> (APD>Ctrls)	<b>11.30; &lt;.001</b> (Dep/Anx >Ctrls)
Linear slope (age)	.045; .416	.030 .432	.076; .392	.000; .500	.531; .233

*Note.* APD= antisocial personality disorder; Ctrls=controls; Dep/Anx=Depression/Anxiety group; v=versus  
<sup>1</sup>one-tailed *p* values used for all comparisons in direction of primary hypotheses.

Table 28

*Peer Rejection When Controlling for Age of Onset: Logistic Regression Results of Pairwise Group Comparisons*

Coefficient	Psychosis v. Ctrls	Psychosis v. APD	Psychosis v. Depression/Anxiety	APD v. Ctrls	Depression/Anxiety v. Ctrls
	$\chi^2; p^1$	$\chi^2; p^1$	$\chi^2; p^1$	$\chi^2; p^1$	$\chi^2; p^1$
Age 13 intercept	.272; .301	.187; .333	2.20; .069	<b>4.80; .015</b> (APD>Ctrls)	<b>9.43; .001</b> (Dep/Anx>Ctrls)
Age 14 intercept	.456; .250	.102; .375	1.76; .092	<b>5.03; .013</b> (APD>Ctrls)	<b>7.97; .003</b> (Dep/Anx >Ctrls)
Age 15 intercept	.651; .210	.035; .427	1.25; .132	<b>4.92; .014</b> (APD>Ctrls)	<b>6.03; .007</b> (Dep/Anx >Ctrls)
Linear slope (age)	.600; .220	.372 .271	<b>3.58; .030</b> (Psychosis > Dep/Anx)	.019; .446	6.12; .007 <sup>2</sup> (Ctrls > Dep/Anx)

*Note.* APD= antisocial personality disorder; Ctrls=controls; Dep/Anx=Depression/Anxiety group; v=versus

<sup>1</sup>one-tailed  $p$  values used for all comparisons in direction of primary hypotheses.

<sup>2</sup> $p$  noted is one-tailed; note however that the direction of the difference is opposite of the one-tailed direction; thus this difference is not considered significant.

Table 29

*Parent-Child Relationship When Controlling for Age of Onset: Logistic Regression Results of Pairwise Group Comparisons*

Coefficient	Psychosis v. Ctrls	Psychosis v. APD	Psychosis v. Depression/Anxiety	APD v. Ctrls	Depression/Anxiety v. Ctrls
	$\chi^2; p^1$	$\chi^2; p^1$	$\chi^2; p^1$	$\chi^2; p^1$	$\chi^2; p^1$
Age 13 intercept	.138; .356	3.50; .031 <sup>2</sup> (APD > Psychosis)	3.10; .039 <sup>2</sup> (Dep/Anx > Psychosis)	<b>22.69; &lt;.001</b> (APD>Ctrls)	<b>8.10; .002</b> (Dep/Anx>Ctrls)
Age 14 intercept	.616; .217	1.94; .082	2.70; .050 <sup>2</sup> (Dep/Anx > Psychosis)	<b>22.45; &lt;.001</b> (APD>Ctrls)	<b>9.70; .001</b> (Dep/Anx >Ctrls)
Age 15 intercept	1.35; .123	.787; .188	2.22; .068	<b>20.67; &lt;.001</b> (APD>Ctrls)	<b>10.76; &lt;.001</b> (Dep/Anx >Ctrls)
Linear slope (age)	<b>4.34; .019</b> (Psychosis>Ctrls)	<b>3.78 .026</b> (Psychosis>APD)	.358; .275	.463; .248	1.52; .109

*Note.* APD= antisocial personality disorder; Ctrls=controls; Dep/Anx=Depression/Anxiety group; v=versus

<sup>1</sup>one-tailed *p* values used for all comparisons in direction of primary hypotheses.

<sup>2</sup>*p* noted is one-tailed; note however that the direction of the difference is opposite of the one-tailed direction; thus this difference is not considered significant.

## 4.0 DISCUSSION

The overall goal of the present study was to contribute to efforts to clarify the nature, timing, and specificity of the precursors of psychotic disorders. Its specific aims were to determine 1) if schizophrenia-like behavioral features and psychosocial stressor exposure observed during adolescence predict full psychotic symptoms in early adulthood in a population-based, prospectively-followed sample; 2) if such relations are specific to psychosis development as compared to the development of APD and depressive and/or anxiety disorders; and 3) if adolescent schizophrenia-like behavioral features moderate the relation between adolescent stressor exposure and early-adulthood psychotic symptoms.

### *4.1 Summary of Primary Findings*

*1. Psychosis versus controls:* As predicted, compared to well controls, boys who endorsed full psychotic symptoms in early adulthood (the psychosis group) obtained significantly higher levels on indices of schizophrenia-like positive symptoms, schizophrenia-like negative symptoms, peer rejection, and problematic parent-child relationship across ages 13 through 17. Furthermore, the psychosis group boys increased significantly more on the indices of positive symptoms and peer rejection across adolescence than did controls, who showed very little change on these constructs over time (the peer rejection slope difference was significant when controlling for risk status, parental education, and parental SES, and was at the trend level

without covariate adjustment). In contrast, the psychosis and control groups did not differ in their rate of change on the index of negative symptoms or problematic parent-child relationship.

2. *The specificity of these relations to early-adulthood psychosis:* The psychosis-control group differences observed generally were not specific to psychosis. That is, the boys of the psychosis group did not differ significantly from those who developed either APD or depressive and/or anxiety disorders on the index of schizophrenia-like positive symptoms, schizophrenia-like negative symptoms, peer rejection, or problematic parent-child relationship at any of the ages assessed. Furthermore, when compared to well controls, both the APD and depression/anxiety groups showed significantly greater levels on all four of these constructs across ages 13 through 17, just as the psychosis group did.

However, the increase in positive symptoms over time displayed by the psychosis group was clearly distinct from and significantly more positive than the weak negative slope the APD group obtained on this construct. Further, although not statistically significant, the psychosis group displayed a greater increase in positive symptoms over time than did the depression/anxiety group, and the effect size of this difference was in the medium range. At the same time, neither the APD nor depression/anxiety group differed from well controls in their rate of change on this construct. Taken together, these results suggest that the significantly greater increase in positive symptoms displayed by the psychosis group across adolescence compared to well controls was specific to psychosis relative to APD and depressive and/or anxiety disorders. Additionally, although the psychosis group slope for peer rejection did not differ significantly from that of either the APD or depression/anxiety group, these latter groups did not differ from

well controls in their rate of change on this construct. Such findings suggest at least some degree of specificity in the mild but significantly greater increase in peer rejection the psychosis group experienced across adolescence compared to well controls.

3. *The moderating effect of schizophrenia-like symptoms:* Adolescent schizophrenia-like symptoms (positive and negative symptoms combined) did not significantly interact with the index of adolescent stressor exposure (peer rejection and problematic parent-child relationship combined) in predicting psychosis.

4. *Psychosis versus all other groups combined:* Comparing the boys who developed psychotic symptoms by early adulthood to the boys who did not yielded a pattern of findings very similar to that obtained when comparing the psychosis group to well controls. Specifically, compared to boys who did not develop psychotic symptoms, the psychosis group displayed significantly higher levels on indices of schizophrenia-like positive symptoms, schizophrenia-like negative symptoms, and peer rejection across ages 13 through 17, and on the index of problematic parent-child relationship from ages 14 to 17. Furthermore, the psychosis group boys increased significantly more across adolescence on the index of positive symptoms compared to the other groups combined.

5. *Controlling for age of onset:* When comparing only the boys of the psychosis group who reported first experiencing any psychotic symptom after the age of 15 to well controls on the behavioral indices of interest from ages 13 to 15, there were no significant group differences in the level of schizophrenia-like positive symptoms, peer rejection, or problematic parent-child relationship. Furthermore, this subset of psychosis group boys did not increase significantly



more than controls on schizophrenia-like positive symptoms across ages 13 to 15. However, these boys did obtain significantly higher levels on the index of negative symptoms from ages 13 to 15 compared to controls.

## *4.2 Integration of Findings and Comparison with Other Studies by Domain*

### *4.2.1 Schizophrenia-like Positive Symptoms*

As noted above, present findings indicated that boys who endorsed full psychotic symptoms in early adulthood displayed increased levels of schizophrenia-like positive symptoms (e.g., suspiciousness, psychotic-like experiences) from ages 13 to 17 compared to well controls; these group differences were maintained when controlling for demographic characteristics and substance dependence. However, such behavioral abnormalities were not specific to psychosis compared to APD or depressive and/or anxiety disorders. In addition to displaying greater levels at all ages assessed, the psychosis group increased significantly more on positive symptoms across adolescence compared to controls, and this increased slope was generally specific to psychosis relative to the other clinical groups of this study. It should be kept in mind, however, that despite displaying a more positive trajectory on this index compared to those who later developed APD and depressive and/or anxiety disorders, the psychosis group did not differ significantly from these other clinical groups in level of positive symptoms at any of the ages assessed, even at age 17. It should also be noted that when controlling for sample, parental education, and parental SES, the slope of the psychosis group for schizophrenia-like positive symptoms was no longer significantly greater than the APD group slope. Additional covariate analyses suggested that this reduction in the association between slope and group was primarily due to adjusting for parental education and parental SES. When considering the implications of

these findings, it should be noted that because it is possible that parental education and/or parental SES may influence the risk for APD and/or share a causal influence with this disorder, adjusting for these characteristics may have resulted in an unrepresentative APD group with regard to their pattern of change in positive symptoms across adolescence; the fact that this APD group was derived from a population-based sample strengthens this possibility. Thus with these points in mind, the current results are viewed as supporting the hypothesis that boys who develop full psychotic symptoms show a growth trajectory of progressively increasing schizophrenia-like positive symptoms across adolescence compared to controls that is unique to psychosis relative to APD and depressive and/or anxiety disorders.

As described above, when attempts were made to control for age of onset by excluding the psychosis group boys who reported first experiencing any psychotic symptom before the age of 16, psychosis-control group differences on this construct for ages 13 through 15 were not maintained. Such findings suggest the possibility that the differences observed were driven by abnormalities present in a subset of boys who had developed full, persisting psychotic symptoms during or before adolescence. Unfortunately, as discussed more fully below, due to the limitations of the age of onset measure used here, conclusions cannot be drawn regarding whether any of the psychosis group boys indeed developed persisting psychotic symptoms before age 18.

A number of studies have assessed the association between childhood or adolescent behavioral features that resemble the positive symptoms of schizophrenia and later psychosis or schizophrenia (e.g., Cannon et al., 2001; Chapman et al., 1994; Lencz et al., 2003; Ott et al., 2001; Roff & Fultz, 2003; see Table 1a). However, most of these investigations used selected samples (e.g., clinical samples of convenience, familial or behavioral high risk samples) and are

limited accordingly with regard to generalizability. This author is aware of only two population-based, prospective investigations that have examined whether mid to late childhood schizophrenia-like positive symptoms predict the development of later psychotic illness (Jones et al., 1994; Poulton et al., 2000). Thus the current study adds to this literature by being among the few to use a more representative sample to examine these associations, as well as possibly the only investigation to use such a sample to assess relations between both the level of and rate of change on such adolescent behavioral features and adulthood psychosis.

Poulton and colleagues (2000) found that psychotic experiences (i.e., delusional beliefs and/or hallucinatory experiences) endorsed at age 11 predicted the development of schizophreniform disorder as assessed at age 26, and that this association was specific to schizophreniform disorder relative to mania and depressive disorders, but not anxiety disorders. Although the present study differed in a number of ways from the Poulton et al. investigation (e.g., in addition to hallucinatory experiences, our multidimensional positive symptoms construct assessed for suspiciousness, strange and paranoid thoughts, and strange behavior), its findings are generally similar to theirs in suggesting that psychotic or psychotic-like experiences and related behaviors are increased by age 13 among those who develop full and persisting psychotic symptoms by early adulthood. Furthermore, the current finding that such behavioral features were also increased among those who developed significant internalizing symptoms by early adulthood (i.e., depressive and/or anxiety disorders) are similar to the report by Poulton et al. that psychotic experiences were increased in children who later developed anxiety disorders. However, as noted, the Poulton group did not find an association between childhood psychotic symptoms and adulthood depressive disorders.

The other population-based investigation to examine schizophrenia-like positive symptoms in mid to late childhood was the large British 1946 Birth Cohort study (Jones et al., 1994). Jones and colleagues found that pre-schizophrenia children did not endorse significantly higher levels of suspiciousness and increased sensitivity per a self report measure administered at age 13 compared to children who did not develop this disorder. Thus neither our primary findings, nor the results obtained when comparing boys who developed psychotic symptoms by early adulthood to those who did not, are consistent with this report. This discrepancy may be due to the fact that although the positive symptoms construct used by the current study included the trait of suspiciousness, it also consisted of other dimensions, as noted above. It may be that these other behaviors measured by our construct (e.g., psychotic-like experiences) contributed more heavily to psychosis-control group differences than did suspiciousness specifically. It is also possible that traits such as suspiciousness are subtly increased during adolescence among children who later develop psychotic illness but that due to their nature, they are difficult to detect by self report during this age period. If this is indeed the case, then the use of external informants by the present study may have increased its sensitivity to detect such behavioral differences between those who later develop psychosis and those who do not.

#### *4.2.2 Schizophrenia-like Negative Symptoms*

As is the case for positive symptoms of schizophrenia, the negative symptoms that characterize this disorder are multidimensional, and include traits and symptoms such social withdrawal, anhedonia, avolition, and flat affect. The index of negative symptoms used by the present study primarily assessed aspects of social isolation or withdrawal. Findings indicated that boys who endorsed full psychotic symptoms in early adulthood displayed increased levels on this measure from ages 13 to 17 compared to well controls, even when adjusting for

demographic covariates and substance dependence. However, as was the case for positive symptoms, this increase in negative symptoms was not specific to psychosis relative to APD or depressive and/or anxiety disorders. As noted, when excluding boys who reported first experiencing any psychotic symptom before the age of 16, the psychosis group continued to display significantly higher levels of negative symptoms compared to controls for ages 13 through 15. Although this attempt to control for age of onset is problematic, as noted, these findings strengthen the conclusion that boys who later develop psychosis show increased levels of the traits captured by our negative symptoms construct during early adolescence, before full psychotic symptoms are present. In contrast to their pattern of change over time on positive symptoms, the psychosis group displayed a flat, near-zero slope for negative symptoms across adolescence, just as the control group did.

As summarized in Table 1b, several studies have assessed the association between childhood or early adulthood social withdrawal or isolation and later schizophrenia (Cannon et al., 2001; Jones et al., 1994, Malmberg et al., 1998; Parnas et al., 1982). To date, the limited findings from among unselected and thus more representative samples have suggested that during mid to late childhood, social withdrawal may be increased to a detectable degree among girls but not boys who later develop schizophrenia (Crow et al., 1995; Watt, 1978). Specifically, Watt (1978) found that according to school records from 7<sup>th</sup> to 12<sup>th</sup> grade, teachers were more likely to describe pre-schizophrenia girls, but not boys, as more socially introverted (e.g., unsociable, quiet) than controls. Crow and colleagues (1995) examined data from the British 1958 Birth Cohort study and, in accordance with the results of Watt, found that girls but not boys of both the pre-schizophrenia and pre-neurosis groups were rated by their teachers as significantly more withdrawn at age 11 than were controls. It may be that differences in how

social withdrawal was measured by the present study and these investigations contributed to the divergence in results concerning boys who developed full psychosis. Specifically, in addition to using teacher observations to measure social withdrawal, our index employed ratings from primary caretakers and children themselves. It is possible that this incorporation of subjective information from the boys yielded a more sensitive measure of social isolation than those relying on external informants alone. Further, our index included two items that reflected preference (e.g., “you like to be alone,” “you enjoy being with others”), in addition to those assessing social behavior/tendencies, and thus differed from the purely behavioral measures used by Crow et al. and Watt.

Interestingly, results from the large-scale population-based Swedish army conscript study of Malmberg and colleagues (1998) suggest that self-reported social isolation is increased at least by young adulthood among males who later develop schizophrenia but not for those who develop other psychoses, such as mood disorder with psychosis, compared to those who do not develop a psychotic disorder. Thus the current results are somewhat consistent with the Malmberg et al. investigation, which was characterized by an especially large, representative sample, although by design its psychotic disorder groups did not include those meeting psychotic disorder criteria before age 18 nor those never hospitalized for psychotic illness. Due to both the small size of our psychosis group, as well as the young age of the boys of this group and the concomitant lack of clarity regarding their eventual diagnostic outcome as they move through the risk period of schizophrenia onset, the current study cannot address specificity questions with regard to psychotic disorders, as Malmberg and colleagues did.

### 4.2.3 *Peer Rejection*

As described above, current findings showed that compared to well controls, boys who developed full psychotic symptoms by early adulthood experienced increased levels of peer rejection from ages 13 to 17; these group differences were maintained when controlling for demographic characteristics and substance dependence, with exception of the psychosis-control group difference at age 13, which was reduced to a strong trend when controlling for substance dependence. As noted, these increased levels of peer rejection were not specific to psychosis relative to APD or depressive and/or anxiety disorders. Further, when excluding boys who reported first experiencing any psychotic symptom before the age of 16, psychosis-control group differences in peer rejection for ages 13 through 15 were not maintained. Regarding change over time in the level of peer rejection experienced, the mild slope of the psychosis group was significantly greater than that of the controls (when controlling for risk status, parental education, and parental SES), and this difference was somewhat specific to psychosis compared to APD and depressive and/or anxiety disorders, as the peer rejection slope did not discriminate these other clinical groups from controls.

Only a handful of retrospective archival or prospective studies have assessed the relation between childhood or early adulthood peer rejection specifically (rather than more general aspects of social functioning) and the development of psychosis or schizophrenia (Bower & Shellhamer, 1960; Cannon et al., 2002; Olin et al., 1995; Malmberg et al., 1998; see Table 2). Two of these investigations employed population-based, prospectively-followed samples. Specifically, Cannon and colleagues (2002) found that children who were later diagnosed with schizophreniform disorder were significantly more likely than controls to be described by parents and teachers as not much liked by other children when ratings from ages 5 to 11 were averaged.

Interestingly, this group further reported that children who later developed either mania or a depressive or anxiety disorder also were rated more highly on this index of peer rejection than were controls. Malmberg and colleagues (1998) reported that males who later developed schizophrenia rated themselves as unpopular with peers more frequently than did controls when assessed between the ages of 18 and 20, whereas those who developed other psychoses did not differ from controls. Although the specificity results of Malmberg et al. cannot be compared directly to those of Cannon et al. or the present study because non-psychotic clinical groups were not included, it is of interest that their findings suggest peer rejection may be specifically related to later schizophrenia compared to other psychoses. In contrast to both the investigation of Cannon et al. and the current study, Malmberg et al. relied on self report to assess peer rejection. Given this and the nature of the item used to assess peer rejection (“unpopular with peers”), response biases related to social desirability may have influenced their specificity findings. Overall then, the current report is generally congruent with the limited existing literature on this topic and extends it by using a population-based sample to examine the association between peer rejection measured across adolescence and later psychosis.

#### *4.2.4 Parent-Child Relationship*

Present findings indicated that boys who developed full psychotic symptoms by early adulthood obtained greater levels on the index of problematic parent-child relationship than did controls from ages 13 to 17. These group differences were generally maintained when using demographic characteristics or substance dependence as covariates, although the psychosis-control group difference at age 13 was reduced to a strong trend when controlling for risk status and age at DIS, or for substance dependence. As was the case for the other domains under study, this increase in problematic parent-child relationships was not specific to psychosis relative to



APD or depressive and/or anxiety disorders. When excluding boys who reported first experiencing any psychotic symptom before the age of 16, psychosis-control group differences on this construct for ages 13 through 15 were not maintained. Although the psychosis group boys displayed a weak positive increase on this index across adolescence, their slope did not differ significantly from that of the controls.

Although aspects of family functioning have long been of interest to schizophrenia researchers, relatively few retrospective archival or prospective studies have assessed problematic aspects of parent-child relationships as possibly predictive of later psychosis or schizophrenia. Among these investigations are reports from the Copenhagen High Risk Project (Carter et al., 2002; Schiffman et al., 2002), which suggest that among individuals at increased genetic (or at least familial) risk for schizophrenia, problematic parent-child relationships as experienced during adolescence are associated with the later development of schizophrenia. Results from the population-based Dunedin study (Cannon et al., 2002) indicated that children who experienced problematic mother-child interactions at age 3 were more likely to later develop schizophreniform disorder, but not mania or a depressive or anxiety disorder. This author is not aware of any population-based study to examine whether problematic parent-child relationships experienced in mid to late childhood predict the development of later psychosis; thus the current investigation informs efforts to identify whether this likely source of psychosocial stress as experienced during adolescence is predictive of later psychosis among representative samples, as it seems to be among high risk samples.

### *4.3 Methodological Considerations*

As noted above, several strengths characterize the present study, including its use of a population-based, prospectively-followed sample that was assessed annually across adolescence, the incorporation of multiple informants to measure adolescent functioning, inclusion of two clinical control groups that permitted questions of specificity to be addressed, and the use of an analytical strategy that allowed for a more efficient use of available data and examination of questions regarding group differences in change over time. However, there are number of limitations that must be considered when interpreting its findings. First, only males were included in our sample, which obviously limits the generalizability of results and does not allow for tests of possible sex effects (e.g., Olin et al., 1995; Crow et al., 1995; Watt, 1978).

Another limitation of the current study is its inability to determine whether any of the psychosis group boys developed persisting psychotic symptoms before age 18. This is a considerable shortcoming of this investigation because, as discussed above, the possibility that psychosis-control group differences were driven by abnormalities present in a subset of boys who had developed full, persisting psychotic symptoms during or before adolescence cannot be ruled out. As described, efforts to address this constraint were made by using age of first symptom information provided by the DIS. However, using this information to index age of onset was an especially conservative and limited approach that possibly resulted in a loss of information regarding the very phenomena of interest, as noted earlier. Thus this issue remains unresolved by the current study.

The participants of this study were drawn from weighted samples of the PYS, which are considered representative of the population of boys attending City of Pittsburgh public schools in 1987 (when the PYS was initiated). Although the use of this representative sample is a notable

strength of the present investigation, results of selective attrition analyses suggest that the generalizability of the final sample used here was attenuated somewhat when boys were excluded due to missing data. Specifically, compared to the final sample, the attrited boys were less likely to be of European-American descent, and their parents had significantly fewer years of education and lower SES. The relations between these demographic characteristics and the behavioral features assessed suggest that the levels of problematic behaviors and experiences observed in our final sample may underestimate the levels present in the population from which the PYS samples were drawn.

To allow for an adequate sample size of the primary outcome of interest (full psychosis), the youngest and oldest samples of the PYS were combined to create the current sample. Analyses indicated that the diagnostic outcome groups did not differ significantly from each other on sample composition. However, it should be noted that when compared to the youngest sample, the oldest sample boys obtained significantly higher mean levels on the four behavioral indices used in the present study at nearly all ages assessed. They also increased significantly more across adolescence on the indices of schizophrenia-like positive symptoms, negative symptoms, and problematic parent-child relationship compared to the youngest sample. These results clearly indicate that the two samples are not comparable with regard to the behaviors under study here. As noted, due to such findings, most of the primary analyses were repeated using sample, among other demographic characteristics, as covariates.

Another potential limitation of the current study was its reliance on DIS data to identify individuals experiencing full, persisting psychotic symptoms in the absence of corroboration from expert diagnosticians. The DIS is designed to be administered by lay interviewers and in this case was administered by trained interviewers of the PYS. It allows for the exclusion of

symptoms apparently due to substance use, medication, or medical conditions, and also provides specific probes designed to minimize the possibility that “explainable” experiences are characterized as psychotic symptoms. These features of the DIS strengthen its validity with regard to identifying individuals experiencing full psychotic symptoms. Moreover, to be included in the psychosis group, the present study required that boys endorse at least one psychotic symptom that persisted for at least one month, which further enhances confidence that boys of the psychosis group were actually experiencing full psychotic symptoms. At the same time, when considering the nature of psychotic symptoms, which are at times difficult to accurately identify, findings suggesting that psychotic-like experiences are endorsed relatively frequently among non-clinical samples (e.g., Poulton et al., 2000), and the fact that almost 70% of the psychosis group boys of the present study denied psychotic symptom-related impairment in occupational or interpersonal functioning, it seems likely that at least a few of the boys classified as psychosis were not experiencing full and persisting psychotic symptoms.

Another related limitation concerns the age at which boys were assessed for psychosis (age 19 for the youngest sample and age 25 for the oldest). Because participants had not yet passed through the risk period for developing psychotic disorders, it is possible that some participants who were not included in the psychosis group will go on to develop full psychotic symptoms. Based on findings suggesting that both internalizing and externalizing symptoms are increased in children who later develop schizophrenia (e.g., Crow et al., 1995), and that many individuals who develop schizophrenia experience non-specific symptoms such as depression and anxiety before developing psychotic symptoms (Häfner & an der Heiden, 1999), it is interesting to speculate that the APD and depression/anxiety groups may be especially likely to contain such “false negatives.” At the same time, because the psychosis group comprised

individuals who endorsed experiencing full psychotic symptoms by their early to mid 20s, it is possible that the current findings only apply to individuals with a relatively early onset of psychosis and thus a more severe course of illness.

Additionally, although the use of multiple informants to assess adolescent functioning is generally considered a strength of this study, it is possible that combining information from parents, teachers, and the participants themselves may have obscured specific group differences in some cases (e.g., for schizophrenia-like positive symptoms).

Furthermore, as is often the case for population-based longitudinal studies, three of the behavioral domains of interest (schizophrenia-like positive symptoms, schizophrenia-like negative symptoms, and peer rejection) were not among the aspects of functioning the PYS specifically sought to examine. Thus constructs were created to assess these domains using existing measures of the PYS and are somewhat limited (see Table 5 for the list of variables comprising each construct). For example, the schizophrenia-like negative symptoms construct primarily assessed aspects of social isolation and thus did not include other important features of this domain, such as anhedonia and avolition. Further, the peer rejection construct did not include specific items regarding the experience of feeling excluded by and/or as if one does not “fit in” with peers.

To provide a perspective on the pattern of significant and nonsignificant group differences to emerge from this study, results from power analyses (Cohen, 1977) are summarized here. As expected, the pairwise group comparisons characterized by the lowest power were those for which no significant differences emerged in level of the behavioral features assessed: psychosis versus depression/anxiety and psychosis versus APD. Specifically, for comparisons between the psychosis and depression/anxiety groups (harmonic mean=19) using

one-tailed tests, the power to detect medium effects was .45, and the power to detect small effects was .16. Power to detect a medium effect for the psychosis versus APD comparisons (harmonic mean=24) using one-tailed tests was .53, whereas it was .18 to detect small effects. The limited power of these comparisons likely contributed in some cases to the lack of significant discrimination between the psychosis and other clinical groups (e.g., psychosis-depression/anxiety group differences in positive symptoms at ages 16 and 17, psychosis-APD group differences in peer rejection at ages 16 and 17). At the same time, the other comparisons, for which significant differences consistently emerged, were characterized by more power. This was especially the case for the APD versus control comparisons (harmonic mean=96): sample sizes resulted in a .96 power to detect medium effects and a .40 power to detect small effects ( $d=.20$ ). Power to detect a medium effect for the depression/anxiety versus control comparisons (harmonic mean=43) was .74, whereas it was .24 to detect small effects for this comparison. The primary comparison of interest (psychosis versus controls; harmonic mean=31) using one-tailed tests had a .62 power to detect medium effects ( $d=.50$ ) and a .20 power to detect small effects ( $d=.20$ ).

Finally, the current investigation involved multiple group comparisons across a number of ages and domains. It is acknowledged that undertaking such a number of comparisons inflated the likelihood that a true null hypothesis was rejected. As described, omnibus tests and, albeit in a limited fashion, the false discovery rate procedure (Curran-Everett, 2000) were employed in attempts to control for the inflation of the probability of making a Type-I error. However, due to the limited power of this study when comparisons involved the psychosis

group, alpha levels were not adjusted for the primary analyses in efforts to reduce the probability of making a Type-II error, with the understanding that such an approach increased the probability of making a Type-I error.

#### *4.4 Clinical Implications*

Interest in identifying individuals who appear to be at increased risk for psychotic disorders before full syndrome onset has recently increased dramatically (Cornblatt, 2002), in part due to research suggesting that earlier initiation of antipsychotic drug treatment after illness onset is associated with a better prognosis (see Perkins et al., 2005). Such findings have fueled efforts to identify at-risk individuals before full illness onset with the goal of providing timely and appropriate intervention in hopes that full psychosis can be somewhat ameliorated or even prevented in some cases (Cornblatt, 2002; Yung et al., 2003). Identifying predictors of psychosis development, especially those that may have some degree of specificity, informs efforts to elucidate the factors or combination of factors that are most useful in accurately identifying such individuals (Dazzan et al., 2004).

Current findings suggest that adolescent schizophrenia-like positive symptoms, social isolation, peer rejection, and problematic parent-child relationships are indeed associated with early adulthood psychotic symptoms. However, results further indicated that, at least as measured by the current study, these behavioral features and experiences are not specifically predictive of psychosis. Although this lack of specificity reduces the usefulness of these factors in identifying individuals who will later develop psychotic illness, they may still be helpful in this regard when viewed in conjunction with other psychosis predictors. As reviewed by Dazzan and colleagues (2004), several researchers have found that combining antecedents of psychosis

(e.g., indices of social and cognitive functioning) substantially increased their ability to predict who later becomes ill. With regard to the behavioral domains under study here, future analyses using a multivariate approach with these predictors and/or other indices that are predictive of psychosis in this sample (e.g., tobacco use; Thompson et al., 2005) may prove useful in identifying a combination of factors that are related to psychosis specifically. Furthermore, although psychosis-control group differences on the behavioral domains of interest were not specific to psychosis at any of the ages assessed, current results indicated that boys who developed full psychotic symptoms became increasingly deviant relative to controls on indices of schizophrenia-like positive symptoms and peer rejection as they moved through adolescence, and that such trajectories were at least somewhat specific to psychosis relative to APD and depressive and/or anxiety disorders. Such findings suggest that a course of progressively problematic functioning and experiences as assessed by these two domains may uniquely characterize those who develop full psychotic symptoms. More generally, they suggest that in some cases, patterns of change on behavioral indices over time rather than level of pre-onset functioning may discriminate adolescents who later develop psychosis from those who experience other psychopathology outcomes. Thus our results underscore the utility of assessing individuals at multiple time points so that both the level of and patterns of change over time on indices of functioning can be examined when attempting to identify individuals at risk for psychosis development.

The lack of specificity with regard to psychosis-control group differences was somewhat striking in that it was observed across all four domains at every age assessed. Further, although it was predicted that the non-psychosis clinical groups would display increased levels of negative symptoms (hypothesized for depression/anxiety only), peer rejection, and problematic parent-



child relationships, the expectation that the psychosis group would emerge as the most deviant of the clinical groups on these constructs was not borne out. Such findings may reflect that the traits and experiences assessed by this study are associated with increased risk for psychopathology in general rather than for psychosis specifically. This lack of specificity was especially surprising for schizophrenia-like positive features, given the nature of the symptoms and experiences this construct was designed to assess, the expectation that such experiences would be less common among the total sample than the other behavioral features assessed (which was the case), and the generally specific findings regarding the association between childhood psychotic experiences and later schizophreniform disorder reported by Poulton and colleagues (2000). It is possible that including items on this construct that assessed for relatively vague experiences (e.g., “strange behavior,” “strange ideas”) contributed to its inability to discriminate the clinical groups of this study, and that the subset of items that more clearly described psychotic-like experiences is more specifically predictive of psychosis. Future analyses in which specific dimensions of this construct are examined separately would inform such speculations. It is also possible that a negative symptoms construct comprising items assessing traits and subjective experiences that more specifically reflect the negative-symptom syndrome of schizophrenia (e.g., aspects of anhedonia, flat affect) may have better discriminated the clinical groups of this study. Unfortunately such measures are not available through the PYS and thus this speculation cannot be explored.

In addition, it is possible that in some cases, combining ratings from caretakers, teachers, and the children themselves may have contributed to the lack of specificity observed with these measures. This concern seems especially relevant to the constructs designed to assess schizophrenia-like features. For example, psychotic-like symptoms, including hallucinatory

experiences, paranoid thoughts, and thoughts that are experienced as strange or alien, are likely best assessed by self report because they reflect internal, subjective experiences. Furthermore, it may be that external raters, such as teachers, tend to endorse such items for children who display unusual or maladaptive behaviors that are not specifically associated with later psychosis. If this is the case, then combining informant information for the positive symptoms construct may have obscured a specific association between self-reported psychotic-like experiences and adulthood psychosis. Again, additional analyses in which ratings from individual informants are examined separately would inform such speculations.

Additionally, in contrast to the boys of the APD and depression/anxiety groups, boys comprising the psychosis group were selected based on the endorsement of symptoms alone rather than meeting criteria for a syndrome that included symptom-related impairment; thus this group was quite heterogeneous with regard to clinical status (e.g., two met full criteria for schizophrenia or schizophreniform disorder whereas some endorsed only one persisting psychotic symptom and denied symptom-related functional impairment). It is possible that the subset of psychosis group boys who were more severely affected (e.g., those experiencing impairing psychotic symptoms) displayed levels of adolescent behavioral abnormalities that were significantly greater than those of the APD and depression/anxiety groups. Unfortunately due to the limited number of boys comprising the psychosis group, the current study cannot explore this possibility.

#### *4.5 Implications for Models of Etiology and Pre-onset Pathophysiology*

Clarifying the factors associated with the development of psychosis is critical for advancing models that attempt to characterize the pathological processes present before and

possibly contributing to the full onset of psychotic symptoms. Unfortunately the lack of specific findings to emerge from this study and the possible measurement issues noted above make it more difficult to speculate regarding how current results inform hypotheses regarding pre-onset pathophysiology. However, if it were determined that the subset of schizophrenia-like positive items that more clearly assessed psychotic-like experiences was specifically predictive of psychosis, such findings would suggest that as a group individuals who later develop full psychosis are characterized to a greater extent than those who do not develop such symptoms by some degree of the pathology thought to underlie active psychotic symptoms (e.g., compromised mesolimbic dopamine regulation; see Thompson et al., 2004, for review) as early as mid to late childhood. Alternatively, current findings could reflect that such experiences and related pathological processes characterize adolescents who are at increased risk for psychopathology more generally.

The current interest in examining the association between psychosocial stressor exposure (i.e. peer rejection and problematic parent-child relationships) and the development of psychotic symptoms largely derived from predictions of models of etiology and pathophysiology that have emerged from the diathesis-stress framework and implicate stressor exposure in the movement of at-risk individuals to full psychosis onset. For example, it has been proposed that brain pathology that may be present before full psychosis onset (e.g., prefrontal cortical abnormalities, compromised mesolimbic dopamine regulation) may result in a subcortical dopamine system that is hyperresponsive to stress and thus may especially increase risk for the development of psychotic symptoms in the face of stressor exposure (see Corcoran et al., 2003; Thompson et al., 2004; Walker & Diforio, 1997, for review and discussion). The current study attempted to use behavioral measures to evaluate such a model. Specifically, it was hypothesized that

schizophrenia-like symptoms (conceptualized as an index of increased risk for psychosis development) would moderate the relation between stressor exposure and later psychosis. As reviewed earlier, however, results did not support this hypothesis. If a behavioral index that appeared to reflect increased psychosis risk could be identified in this sample (e.g., psychotic-like experiences specifically, as described above), it would be of great interest to assess whether such traits interact with stressor exposure in predicting later psychosis. However, it is acknowledged that the current study is quite limited in its capacity to address such questions both because of the lack of relevant measures available and the small number of boys comprising the psychosis group.

The associations observed between adolescent peer rejection and problematic parent-child relationships with later psychosis, APD, and depressive and/or anxiety disorders may largely reflect that general interpersonal difficulties commonly characterize the functioning of adolescents who later experience adulthood psychopathology. Even if this is the case, however, the behavioral deficits contributing to such interpersonal difficulties may still be specific to each of these outcomes (e.g., cognitive impairment and schizotypal tendencies may drive such difficulties among pre-psychosis individuals, whereas social anxiety and self-defeating cognitions may contribute to such among those who later develop depressive and/or anxiety disorders). It is also possible that current results reflect that such experiences increase risk for a subset or all of the outcomes assessed. For example, it is possible that peer rejection works to increase psychosis risk among vulnerable individuals by both increasing distress and by decreasing normalizing and reassuring interactions with friends in the face of experiences associated with psychosis risk (e.g., increases in anxiety and paranoid ideas; Murray & Fearon,

1999). In a similar way, peer rejection experiences may increase risk for developing a full depressive and/or anxiety disorder in those at risk for doing so (e.g., by reinforcing fears and cognitions related to being inadequate and thus rejected by others).

#### *4.6 Future Directions*

As noted throughout this discussion, the current findings suggest a number of fruitful areas of focus for additional work, both for the current project as well as more generally. For example, multivariate analyses with the behavioral domains of this study and/or other indices shown by previous work to predict psychosis (e.g., school functioning) may be useful in identifying a combination of factors that may be related to psychosis specifically in this population-based sample. Furthermore, present results underscore the importance clarifying whether the addition of external informants enhances or possibly hinders the ability of measures such as those used here (e.g., the positive-symptoms index) to discriminate groups; although beyond the scope of the current study, this project more generally is in a unique position to address such questions. Moreover, given the current findings and those of others (i.e., Poulton et al., 2000), further work within this project and by other population-based studies assessing the association between childhood psychotic-like symptoms specifically and later psychosis would be useful. That peer rejection and problematic parent-child relationships were associated with full psychosis suggests that additional work attempting to determine if such experiences potentiate associations between indices of psychosis risk and full psychosis development may be fruitful. In addition to possibly informing models of etiology and pathophysiology, such work could importantly suggest potential targets for psychosocial interventions once susceptible individuals are identified. Such research may be especially productive with samples of high-risk

individuals, such as those identified as prodromal to psychosis. Finally, the current findings highlight the importance of incorporating trajectory analyses into efforts to characterize the pre-onset functioning of individuals who later develop psychosis.

#### *4.7 Conclusions*

The present findings indicated that among a population-based sample of males, adolescent schizophrenia-like positive symptoms, social isolation, peer rejection, and problematic parent-child relationships are associated with early adulthood psychotic symptoms, but that such behavioral features and experiences are not specifically predictive of psychosis relative to APD or depressive and/or anxiety disorders. Results further suggest that as a group, boys who develop full psychotic symptoms by early adulthood display a course of progressively increasing schizophrenia-like positive symptoms and greater levels of peer rejection as they move through adolescence compared to controls, and that such a trajectory is at least somewhat specific to psychosis relative to APD and depressive and/or anxiety disorders. However, it should be noted that the current study did not have the means to accurately date the onset of full psychosis among those endorsing such symptoms in early adulthood; thus the possibility that the group differences observed were driven by abnormalities present in a subset of boys who had developed full psychotic symptoms during or before adolescence cannot be ruled out. The specificity findings to emerge from this investigation suggest that the behavioral features and experiences assessed may be associated with increased risk for psychopathology in general rather than for psychosis specifically. Alternatively, it may be that various measurement issues of the current study obscured specific associations between the behavioral features assessed and early

adulthood psychosis; this concern is especially relevant to the index that was used to measure adolescent psychotic-like experiences. Future work is needed to clarify such issues.

The current investigation adds to the existing literature by being among the few to use a representative sample to assess the associations among these behavioral features as observed during adolescence and early adulthood psychosis, and to examine whether such associations are specific to psychosis. Furthermore, it extends such research by characterizing the adolescent trajectories of individuals who develop full psychotic symptoms compared to those who do not on such behavioral indices, and importantly underscores the utility of assessing both level of and patterns of change over time on indices of functioning when attempting to identify and characterize the functioning of individuals at risk for psychosis development.

APPENDIX A

INTERNAL CONSISTENCY (CRONBACH'S ALPHA) FOR SCHIZOPHRENIA-LIKE POSITIVE SYMPTOM, SCHIZOPHRENIA-LIKE NEGATIVE SYMPTOM, AND PEER REJECTION INFORMANT-SPECIFIC CONSTRUCTS

Construct	Parent	Teacher	Child
Schizophrenia-like Positive Symptoms at:			
Age13	.582 (N=666)	.688 (N=567)	.522 (N=673)
Age14	.570 (N=717)	.718 (N=440)	.574 (N=724)
Age15	.577 (N=713)	.677 (N=145)	.557 (N=720)
Age16	.474 (N=624)	---	.533 (N=718)
Age17	.604 (N=250)	---	.558 (N=708)
Schizophrenia-like Negative Symptoms at:			
Age13	.682 (N=666)	.755 (N=613)	.342 (N=673)
Age14	.701 (N=717)	.700 (N=475)	.346 (N=724)
Age15	.695 (N=713)	.803 (N=158)	.419 (N=721)
Age16	.716 (N=624)	.706 (N=291)	.455 (N=718)
Age17	.691 (N=250)	---	.515 (N=708)
Peer Rejection at:			
Age13	.541 (N=667)	.690 (N=618)	.530 (N=673)
Age14	.558 (N=716)	.596 (N=482)	.512 (N=724)
Age15	.529 (N=713)	.634 (N=157)	.570 (N=720)
Age16	.524 (N=624)	---	.437 (N=718)
Age17	.641 (N=250)	---	.516 (N=708)



## APPENDIX A (continued)

*Note.* All internal consistency analyses used weighted *Ns*. The *Ns* noted here reflect the unweighted number of cases available for reliability analyses for each construct, i.e. cases with data for all items contributing to that construct. These *Ns* do not reflect the number of cases used to create the informant-combined constructs used for primary analyses because, as detailed in Table 6, participants were not required to have all items of a given construct for inclusion.

APPENDIX B

INTER-RATER CORRELATIONS OF INFORMANT-SPECIFIC CONSTRUCTS FOR SCHIZOPHRENIA-LIKE POSITIVE SYMPTOMS, SCHIZOPHRENIA-LIKE NEGATIVE SYMPTOMS, PEER REJECTION, AND PARENT-CHILD RELATIONSHIP

Schizophrenia-like Positive Symptoms			
Age	Parent-teacher	Parent-child	Teacher-child
13	.152** (N=602)	.230** (N=666)	.117** (N=605)
14	.228** (N=469)	.229** (N=716)	.060 (N=475)
15	.029 (N=155)	.169** (N=708)	.152 (N=156)
16	---	.141** (N=618)	---
17	---	.098 (N=245)	---
Schizophrenia-like Negative Symptoms			
Age	Parent-teacher	Parent-child	Teacher-child
13	.111** (N=626)	.117** (N=666)	.095* (N=629)
14	.190** (N=636)	.154** (N=716)	.102* (N=642)
15	.107* (N=531)	.183** (N=708)	.012 (N=535)
16	.170** (N=289)	.198** (N=618)	.088 (N=297)
17	---	.187** (N=245)	---

(APPENDIX B continued on next page)

APPENDIX B (continued)

Peer Rejection			
Age	Parent-teacher	Parent-child	Teacher-child
13	.290** (N=624)	.335** (N=666)	.196** (N=627)
14	.196** (N=491)	.287** (N=716)	.198** (N=497)
15	.194* (N=163)	.189** (N=708)	.044 (N=164)
16	---	.243** (N=618)	---
17	---	.220** (N=245)	---

Parent-Child Relationship			
Age	Parent-teacher	Parent-child	Teacher-child
13	---	.213** (N=611)	---
14	---	.240** (N=693)	---
15	---	.278** (N=691)	---
16	---	.325** (N=600)	---
17	---	.273** (N=233)	---

*Note.* Unweighted *Ns* used in analyses, as the weighting procedure cannot be implemented when calculating Spearman correlation coefficients. The *Ns* provided here reflect the number of cases with data for both of the informant-specific constructs used in the given bivariate analysis. These *Ns* do not reflect the number of cases used to create the informant-combined constructs used for primary analyses because, as detailed in Table 6, participants were considered missing at a given age for the informant-combined version of this construct only if they were missing on all of the contributing informant-specific constructs.

\* $p < .05$ ; \*\* $p < .01$ .

APPENDIX C

DESCRIPTIVE STATISTICS OF PRIMARY CONSTRUCTS (RAW SCORES) USING  
TOTAL SAMPLE

Construct	<i>N</i>	Mean ( <i>Sd</i> )	Range
Schizophrenia-like Positive Symptoms (range of possible scores=0-36)			
Age13	689	2.17 (2.57)	0.00-27.00
Age14	730	2.06 (2.44)	0.00-19.50
Age15	731	1.91 (2.45)	0.00-18.75
Age16	727	1.87 (2.44)	0.00-16.50
Age17	719	2.18 (3.19)	0.00-24.00
Schizophrenia-like Negative Symptoms (range of possible scores=0-24)			
Age13	689	3.91 (2.56)	0.00-14.40
Age14	730	3.91 (2.56)	0.00-15.00
Age15	731	4.14 (2.68)	0.00-15.80
Age16	729	4.03 (2.99)	0.00-21.00
Age17	719	4.11 (3.66)	0.00-24.00
Peer Rejection (range of possible scores=0-18)			
Age13	689	1.99 (2.16)	0.00-15.00
Age14	730	1.59 (1.84)	0.00-10.50
Age15	731	1.40 (1.89)	0.00-13.50
Age16	727	1.24 (1.80)	0.00-10.50
Age17	719	1.20 (2.16)	0.00-12.00

APPENDIX C (continued)

Construct	<i>N</i>	Mean ( <i>Sd</i> )	Range
Parent-Child Relationship (range of possible scores=26-78)			
Age13	635	36.72 (6.53)	26.00-64.94
Age14	712	37.18 (6.80)	26.00-63.66
Age15	727	37.85 (7.39)	26.00-68.25
Age16	719	37.48 (7.49)	26.00-67.60
Age17	712	37.62 (8.37)	26.00-68.82

*Note.* *Sd*=standard deviation.

APPENDIX D

DESCRIPTIVE STATISTICS OF HLM COEFFICIENTS BY DEMOGRAPHIC GROUPS  
FOR SCHIZOPHRENIA-LIKE POSITIVE SYMPTOMS, SCHIZOPHRENIA-LIKE  
NEGATIVE SYMPTOMS, PEER REJECTION, AND PARENT-CHILD RELATIONSHIP

Coefficient	Sample		Risk group <sup>1</sup>		Ethnicity	
	Youngest (n=376)	Oldest (n=361)	Low (n=470)	High (n=267)	European American (n=325)	Other <sup>2</sup> (n=412)
<b>Schizophrenia-like Positive Symptoms:</b>						
Age 13 intercept	-.099 (.608)	.022 (.676)	-.171 (.509)	.192 (.781)	-.044 (.679)	-.037 (.617)
Age 14 intercept	-.111 (.565)	.032 (.659)	-.162 (.495)	.173 (.741)	-.042 (.656)	-.040 (.585)
Age 15 intercept	-.122 (.539)	.041 (.655)	-.153 (.497)	.154 (.717)	-.041 (.651)	-.043 (.565)
Age 16 intercept	-.134 (.533)	.051 (.664)	-.144 (.514)	.135 (.711)	-.039 (.663)	-.046 (.560)
Age 17 intercept	-.145 (.546)	.060 (.684)	-.136 (.544)	.116 (.722)	-.038 (.691)	-.050 (.570)
Slope	-.011 (.104)	.010 (.091)	.009 (.087)	-.019 (.114)	.001 (.107)	-.003 (.091)

(APPENDIX D continued on next page)

## APPENDIX D (continued)

Coefficient	Sample		Risk group <sup>1</sup>		Ethnicity	
	Youngest (n=376)	Oldest (n=361)	Low (n=470)	High (n=267)	European American (n=325)	Other <sup>2</sup> (n=412)
Schizophrenia-like Negative Symptoms:						
Age 13 intercept	-.103 (.599)	.077 (.676)	-.088 (.625)	.114 (.659)	-.081 (.662)	.037 (.626)
Age 14 intercept	-.115 (.573)	.083 (.637)	-.088 (.592)	.107 (.629)	-.092 (.626)	.041 (.596)
Age 15 intercept	-.126 (.561)	.090 (.605)	-.088 (.570)	.100 (.614)	-.103 (.601)	.045 (.578)
Age 16 intercept	-.138 (.565)	.096 (.582)	-.088 (.559)	.092 (.612)	-.114 (.588)	.049 (.573)
Age 17 intercept	-.149 (.585)	.103 (.567)	-.089 (.560)	.085 (.624)	-.125 (.587)	.052 (.581)
Slope	-.011 (.095)	.006 (.076)	-.000 (.083)	-.007 (.094)	-.011 (.087)	.004 (.086)
Peer Rejection:						
Age 13 intercept	-.107 (.615)	.086 (.760)	-.094 (.655)	.131 (.744)	-.015 (.734)	-.010 (.666)
Age 14 intercept	-.115 (.573)	.084 (.738)	-.093 (.626)	.117 (.713)	-.023 (.703)	-.013 (.636)
Age 15 intercept	-.123 (.543)	.083 (.726)	-.092 (.608)	.102 (.695)	-.031 (.682)	-.015 (.619)
Age 16 intercept	-.131 (.528)	.081 (.724)	-.092 (.601)	.087 (.690)	-.039 (.672)	-.017 (.614)
Age 17 intercept	-.139 (.528)	.080 (.734)	-.091 (.607)	.073 (.699)	-.047 (.674)	-.019 (.624)
Slope	-.008 (.091)	-.002 (.089)	.001 (.086)	-.015 (.097)	-.008 (.088)	-.002 (.092)

APPENDIX D (continued)

Coefficient	Youngest (n=376)	Oldest (n=361)	Low (n=470)	High (n=267)	European American (n=325)	Other <sup>2</sup> (n=412)
<b>Parent-Child Relationship:</b>						
Age 13 intercept	-.095 (.782)	.005 (.763)	-.192 (.716)	.213 (.804)	-.051 (.787)	-.042 (.763)
Age 14 intercept	-.106 (.731)	.019 (.738)	-.180 (.686)	.194 (.763)	-.045 (.753)	-.044 (.725)
Age 15 intercept	-.116 (.696)	.033 (.727)	-.167 (.670)	.175 (.739)	-.039 (.734)	-.047 (.700)
Age 16 intercept	-.127 (.677)	.047 (.731)	-.154 (.671)	.156 (.733)	-.033 (.734)	-.049 (.690)
Age 17 intercept	-.138 (.678)	.060 (.750)	-.141 (.688)	.137 (.744)	-.027 (.751)	-.051 (.696)
Slope	-.011 (.113)	.014 (.104)	.013 (.104)	-.019 (.115)	.006 (.115)	-.002 (.105)

*Note.* Mean (standard deviation).

<sup>1</sup>risk-group status based on initial assessment at screening phase of Pittsburgh Youth Study.

<sup>2</sup>the “other” group was predominantly African American.



APPENDIX E

LOGISTIC REGRESSION RESULTS OF PAIRWISE DIAGNOSTIC GROUP COMPARISONS WHEN INCLUDING COVARIATES FOR SCHIZOPHRENIA-LIKE POSITIVE SYMPTOMS, SCHIZOPHRENIA-LIKE NEGATIVE SYMPTOMS, PEER REJECTION, AND PARENT-CHILD RELATIONSHIP

Table E1.  
*Schizophrenia-like Positive Symptoms: Pairwise Group Comparisons when including Covariates*

Coefficient	Psychosis v. Ctrls	Psychosis v. APD	Psychosis v. Depression/Anxiety	APD v. Ctrls <sup>1</sup>	Depression/Anxiety v. Ctrls
	$\chi^2; p^2$	$\chi^2; p^2$	$\chi^2; p^2$	$\chi^2; p^2$	$\chi^2; p^2$
Age 13 intercept	<b>4.03<sup>3</sup>; .023</b> (Psychosis>Ctrls)	.146 <sup>4</sup> ; .352	.206 <sup>5,6</sup> ; .325	<b>26.55<sup>4</sup>; &lt;.001</b> (APD>Ctrls)	<b>4.63<sup>4,7</sup>; .016</b> (Dep/Anx>Ctrls)
Age 14 intercept	<b>6.45<sup>3</sup>; .006</b> (Psychosis>Ctrls)	.051 <sup>4</sup> ; .412	.098 <sup>8,9</sup> ; .377	<b>28.42<sup>4</sup>; &lt;.001</b> (APD>Ctrls)	<b>5.59<sup>10,11</sup>; .009</b> (Dep/Anx >Ctrls)
Age 15 intercept	<b>8.69<sup>8</sup>; .002</b> (Psychosis>Ctrls)	.035 <sup>10</sup> ; .426	.323 <sup>8</sup> ; .285	<b>29.36<sup>4</sup>; &lt;.001</b> (APD>Ctrls)	<b>8.49<sup>8</sup>; .002</b> (Dep/Anx >Ctrls)

(APPENDIX E continued on next page)

APPENDIX E, Table E1 (continued)

Coefficient	Psychosis v. Ctrls	Psychosis v. APD	Psychosis v. Depression/Anxiety	APD v. Ctrls <sup>1</sup>	Depression/Anxiety v. Ctrls
	$\chi^2; p^2$	$\chi^2; p^2$	$\chi^2; p^2$	$\chi^2; p^2$	$\chi^2; p^2$
Age 16 intercept	<b>11.15<sup>8</sup>; &lt;.001</b> (Psychosis>Ctrls)	.028 <sup>10</sup> ; .434	.593 <sup>8</sup> ; .221	<b>28.98<sup>4</sup>; &lt;.001</b> (APD>Ctrls)	<b>8.94<sup>8</sup>; .002</b> (Dep/Anx >Ctrls)
Age 17 intercept	<b>13.03<sup>12, 13</sup>; &lt;.001</b> (Psychosis>Ctrls)	.239 <sup>10</sup> ; .313	.837 <sup>8</sup> ; .180	<b>27.27<sup>4</sup>; &lt;.001</b> (APD>Ctrls)	<b>8.94<sup>8</sup>; .002</b> (Dep/Anx >Ctrls)
Linear slope (age)	<b>9.47<sup>12, 14</sup>; .001</b> (Psychosis>Ctrls)	1.04 <sup>15</sup> ; .155*	1.95 <sup>6, 16</sup> ; .082	.162 <sup>4</sup> ; .344	.507 <sup>6, 16</sup> ; .239
Quadratic slope (age squared)	---	---	---	<b>12.44<sup>17</sup>; &lt;.001</b> (Ctrls>APD)	---

Note. APD= antisocial personality disorder; Ctrls=controls; Dep/Anx=Depression/Anxiety group; DIS= Diagnostic Interview Schedule for DSM-IV; DSM-IV=The Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition; SES= socioeconomic status; v=versus

\* indicates a change from primary analyses regarding whether the difference between the groups was statistically significant.

<sup>1</sup>For the APD-control group comparisons, the intercept and linear slope coefficients derived when using age as the only predictor were used. The quadratic slope results are also presented here because the quadratic component significantly discriminated these two groups on this domain.

<sup>2</sup>one-tailed *p* values used for all comparisons due to a-priori hypotheses with exception of APD-control group comparison on quadratic slope.

<sup>3</sup>sample, risk status, age at DIS, years of parental education, and parental SES used as covariates.

<sup>4</sup>sample, age at DIS, years of parental education, and parental SES used as covariates.

<sup>5</sup>sample, risk status, age at DIS, and years of parental education used as covariates.

<sup>6</sup>parental SES was not used as a covariate because it interacted with this HLM coefficient in predicting group.

<sup>7</sup>risk status was not used as a covariate because it interacted with this HLM coefficient in predicting group.

<sup>8</sup>sample, risk status, and age at DIS used as covariates.

<sup>9</sup>years of parental education and parental SES were not used as covariates because they both interacted with this HLM coefficient in predicting group.

<sup>10</sup>sample and age at DIS used as covariates.

<sup>11</sup>risk status, years of parental education, and parental SES were not used as covariates because they interacted with this HLM coefficient in predicting group.

<sup>12</sup>sample was not used as a covariate because it interacted with this HLM coefficient in predicting group.

<sup>13</sup>risk status and age at DIS used as covariates.

APPENDIX E, Table E1 (continued)

<sup>14</sup>risk status, years of parental education, and parental SES used as covariates.

<sup>15</sup>sample, years of parental education, and parental SES used as covariates.

<sup>16</sup>sample, risk status, and years of parental education used as covariates.

<sup>17</sup>age at DIS, years of parental education, and parental SES used as covariates.

(APPENDIX E continued on next page)

APPENDIX E (continued)

Table E2.  
Schizophrenia-like Negative Symptoms: Pairwise Group Comparisons when including Covariates

Coefficient	Psychosis v. Ctrls	Psychosis v. APD	Psychosis v. Depression/Anxiety	APD v. Ctrls <sup>1</sup>	Depression/Anxiety v. Ctrls
	$\chi^2; p^2$	$\chi^2; p^2$	$\chi^2; p^2$	$\chi^2; p^2$	$\chi^2; p^2$
Age 13 intercept	<b>8.01<sup>3</sup>; .003</b> (Psychosis>Ctrls)	1.37 <sup>4</sup> ; .121	.314 <sup>3</sup> ; .288	<b>8.51<sup>5</sup>; .002</b> (APD>Ctrls)	<b>11.43<sup>3</sup>; &lt;.001</b> (Dep/Anx>Ctrls)
Age 14 intercept	<b>8.97<sup>3</sup>; .002</b> (Psychosis>Ctrls)	1.61 <sup>4</sup> ; .103	.429 <sup>3</sup> ; .256	<b>9.21<sup>5</sup>; .001</b> (APD>Ctrls)	<b>12.01<sup>3</sup>; &lt;.001</b> (Dep/Anx >Ctrls)
Age 15 intercept	<b>10.68<sup>6</sup>; &lt;.001</b> (Psychosis>Ctrls)	1.98 <sup>7,8</sup> ; .080	.557 <sup>6</sup> ; .228	<b>9.66<sup>5</sup>; .001</b> (APD>Ctrls)	<b>11.68<sup>6</sup>; &lt;.001</b> (Dep/Anx >Ctrls)
Age 16 intercept	<b>11.06<sup>6</sup>; &lt;.001</b> (Psychosis>Ctrls)	1.81 <sup>7,8</sup> ; .089	.634 <sup>6</sup> ; .213	<b>9.73<sup>5</sup>; .001</b> (APD>Ctrls)	<b>11.52<sup>6</sup>; &lt;.001</b> (Dep/Anx >Ctrls)
Age 17 intercept	<b>10.91<sup>6</sup>; &lt;.001</b> (Psychosis>Ctrls)	2.24 <sup>5</sup> ; .068	.679 <sup>6</sup> ; .205	<b>9.37<sup>5</sup>; .001</b> (APD>Ctrls)	<b>10.85<sup>6</sup>; &lt;.001</b> (Dep/Anx >Ctrls)
Linear slope (age)	.007 <sup>9</sup> ; .467	.076 <sup>9</sup> ; .392	.175 <sup>9</sup> ; .338	.209 <sup>9</sup> ; .324	.488 <sup>9</sup> ; .243
Quadratic slope (age squared)	---	---	---	<b>8.26<sup>10</sup>; .004</b> (Ctrls>APD)	---

Note. APD= antisocial personality disorder; Ctrls=controls; Dep/Anx=Depression/Anxiety group; DIS= Diagnostic Interview Schedule for DSM-IV; DSM-IV=The Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition; SES= socioeconomic status; v=versus

APPENDIX E, Table E2 (continued)

<sup>1</sup>For the APD-control group comparisons, the intercept and linear slope coefficients derived when using age as the only predictor were used. The quadratic slope results are also presented here because the quadratic component significantly discriminated these two groups on this domain.

<sup>2</sup>one-tailed *p* values used for all comparisons due to a-priori hypotheses with exception of APD-control group comparison on quadratic slope.

<sup>3</sup>sample, risk status, ethnicity, age at DIS, and parental SES used as covariates.

<sup>4</sup>sample, ethnicity, age at DIS, and parental SES used as covariates.

<sup>5</sup>sample, ethnicity, age at DIS, years of parental education, and parental SES used as covariates.

<sup>6</sup>sample, risk status, ethnicity, age at DIS, years of parental education, and parental SES used as covariates.

<sup>7</sup>sample, age at DIS, years of parental education, and parental SES used as covariates.

<sup>8</sup>ethnicity was not used as a covariate because it interacted with this HLM coefficient in predicting group.

<sup>9</sup>sample, ethnicity, and age at DIS used as covariates.

<sup>10</sup>age at DIS, years of parental education, and parental SES used as covariates.

(APPENDIX E continued on next page)

APPENDIX E (continued)

Table E3.  
Peer Rejection: Pairwise Group Comparisons when including Covariates

Coefficient	Psychosis v. Ctrls	Psychosis v. APD	Psychosis v. Depression/Anxiety	APD v. Ctrls	Depression/Anxiety v. Ctrls
	$\chi^2$ ; $p^1$	$\chi^2$ ; $p^1$	$\chi^2$ ; $p^1$	$\chi^2$ ; $p^1$	$\chi^2$ ; $p^1$
Age 13 intercept	<b>3.06<sup>2</sup>; .040</b> (Psychosis>Ctrls)	1.46 <sup>3</sup> ; .113	.051 <sup>3,4</sup> ; .411	<b>3.84<sup>3</sup>; .025</b> (APD>Ctrls)	<b>11.64<sup>3,4</sup>; &lt;.001</b> (Dep/Anx>Ctrls)
Age 14 intercept	<b>4.14<sup>2</sup>; .021</b> (Psychosis>Ctrls)	1.73 <sup>3</sup> ; .095	.023 <sup>3,4</sup> ; .440	<b>4.65<sup>3</sup>; .016</b> (APD>Ctrls)	<b>12.69<sup>3,4</sup>; &lt;.001</b> (Dep/Anx >Ctrls)
Age 15 intercept	<b>5.28<sup>2</sup>; .011</b> (Psychosis>Ctrls)	.779 <sup>5,6</sup> ; .189	.006 <sup>3,4</sup> ; .470	<b>5.42<sup>3</sup>; .010</b> (APD>Ctrls)	<b>13.46<sup>3,4</sup>; &lt;.001</b> (Dep/Anx >Ctrls)
Age 16 intercept	<b>6.31<sup>2</sup>; .006</b> (Psychosis>Ctrls)	.893 <sup>5,6</sup> ; .173	.000 <sup>3,4</sup> ; .500	<b>6.03<sup>3</sup>; .007</b> (APD>Ctrls)	<b>13.81<sup>3,4</sup>; &lt;.001</b> (Dep/Anx >Ctrls)
Age 17 intercept	<b>7.11<sup>2</sup>; .004</b> (Psychosis>Ctrls)	.965 <sup>5,6</sup> ; .163	.005 <sup>3,4</sup> ; .472	<b>6.42<sup>3</sup>; .006</b> (APD>Ctrls)	<b>14.12<sup>7,8</sup>; &lt;.001</b> (Dep/Anx >Ctrls)
Linear slope (age)	<b>2.90<sup>9</sup>; .044*</b> (Psychosis>Ctrls)	.414 <sup>10</sup> ; .260	1.53 <sup>6,11</sup> ; .108	.852 <sup>10</sup> ; .178	.117 <sup>6,11</sup> ; .366

*Note.* APD= antisocial personality disorder; Ctrls=controls; Dep/Anx=Depression/Anxiety group; DIS= Diagnostic Interview Schedule for DSM-IV; DSM-IV=The Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition; SES= socioeconomic status; v=versus  
\* indicates a change from primary analyses regarding whether the difference between the groups was statistically significant.  
<sup>1</sup>one-tailed  $p$  values used for all comparisons due to a-priori hypotheses.  
<sup>2</sup>sample, risk status, age at DIS, years of parental education, and parental SES used as covariates.  
<sup>3</sup>sample, age at DIS, years of parental education, and parental SES used as covariates.

APPENDIX E, Table E3 (continued)

<sup>4</sup>risk status was not used as a covariate because it interacted with this HLM coefficient in predicting group.

<sup>5</sup>sample, age at DIS, and years of parental education used as covariates.

<sup>6</sup>parental SES was not used as a covariate because it interacted with this HLM coefficient in predicting group.

<sup>7</sup>sample, age at DIS, and parental SES used as covariates.

<sup>8</sup>risk status and years of parental education were not used as covariates because they both interacted with this HLM coefficient in predicting group.

<sup>9</sup>risk status, years of parental education, and parental SES used as covariates.

<sup>10</sup>years of parental education and parental SES used as covariates.

<sup>11</sup>risk status and years of parental education used as covariates.

(APPENDIX E continued on next page)

APPENDIX E (continued)

Table E4.  
*Parent-Child Relationship: Pairwise Group Comparisons when including Covariates*

Coefficient	Psychosis v. Ctrls	Psychosis v. APD	Psychosis v. Depression/Anxiety	APD v. Ctrls <sup>1</sup>	Depression/Anxiety v. Ctrls
	$\chi^2$ ; $p^2$	$\chi^2$ ; $p^2$	$\chi^2$ ; $p^2$	$\chi^2$ ; $p^2$	$\chi^2$ ; $p^2$
Age 13 intercept	2.30 <sup>3</sup> ; .065*	.699 <sup>4</sup> ; .202	.008 <sup>3</sup> ; .464	<b>22.07<sup>5</sup>; &lt;.001</b> (APD>Ctrls)	<b>8.01<sup>3</sup>; .003</b> (Dep/Anx>Ctrls)
Age 14 intercept	<b>3.05<sup>6</sup>; .041</b> (Psychosis>Ctrls)	.359 <sup>7</sup> ; .275	.004 <sup>6</sup> ; .475	<b>21.29<sup>8</sup>; &lt;.001</b> (APD>Ctrls)	<b>9.07<sup>6</sup>; .002</b> (Dep/Anx >Ctrls)
Age 15 intercept	<b>3.78<sup>6</sup>; .026</b> (Psychosis>Ctrls)	.122 <sup>7</sup> ; .364	.032 <sup>6</sup> ; .429	<b>19.66<sup>8</sup>; &lt;.001</b> (APD>Ctrls)	<b>9.81<sup>6</sup>; .001</b> (Dep/Anx >Ctrls)
Age 16 intercept	<b>4.41<sup>6</sup>; .018</b> (Psychosis>Ctrls)	.004 <sup>7</sup> ; .474	.089 <sup>6</sup> ; .383	<b>17.16<sup>8</sup>; &lt;.001</b> (APD>Ctrls)	<b>10.11<sup>6</sup>; &lt;.001</b> (Dep/Anx >Ctrls)
Age 17 intercept	<b>4.85<sup>6</sup>; .014</b> (Psychosis>Ctrls)	.052 <sup>7</sup> ; .410	.165 <sup>6</sup> ; .342	<b>14.10<sup>8</sup>; &lt;.001</b> (APD>Ctrls)	<b>9.93<sup>6</sup>; .001</b> (Dep/Anx >Ctrls)
Linear slope (age)	.961 <sup>6</sup> ; .164	<b>2.84<sup>7</sup>; .046</b> (Psychosis>APD)	.425 <sup>6</sup> ; .258	<b>5.19<sup>8</sup>; .012</b> (Ctrls>APD)	.133 <sup>6</sup> ; .358
Quadratic slope (age squared)	---	---	---	<b>8.49<sup>5</sup>; .004</b> (Ctrls>APD)	---

*Note.* APD= antisocial personality disorder; Ctrls=controls; Dep/Anx=Depression/Anxiety group; DIS= Diagnostic Interview Schedule for DSM-IV; DSM-IV=The Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition; SES= socioeconomic status; v=versus



APPENDIX E, Table E4 (continued)

\* indicates a change from primary analyses regarding whether the difference between the groups was statistically significant.

<sup>1</sup>For the APD-control group comparisons, the intercept and linear slope coefficients derived when using age as the only predictor were used. The quadratic slope results are also presented here because the quadratic component significantly discriminated these two groups on this domain.

<sup>2</sup>one-tailed  $p$  values used for all comparisons due to a-priori hypotheses with exception of APD-control group comparison on quadratic slope.

<sup>3</sup>risk status and age at DIS used as covariates.

<sup>4</sup>age at DIS used as a covariate.

<sup>5</sup>age at DIS, years of parental education, and parental SES used as covariates.

<sup>6</sup>sample and risk status used as covariates.

<sup>7</sup>sample used as a covariate.

<sup>8</sup>sample, age at DIS, years of parental education, and parental SES used as covariates.

APPENDIX F

APD VERSUS CONTROLS: MEAN ESTIMATED QUADRATIC GROWTH TRAJECTORIES FOR SCHIZOPHRENIA-LIKE POSITIVE SYMPTOMS, SCHIZOPHRENIA-LIKE NEGATIVE SYMPTOMS, AND PROBLEMATIC PARENT-CHILD RELATIONSHIP BASED ON AGE-13 INTERCEPT AND QUADRATIC SLOPE COEFFICIENTS

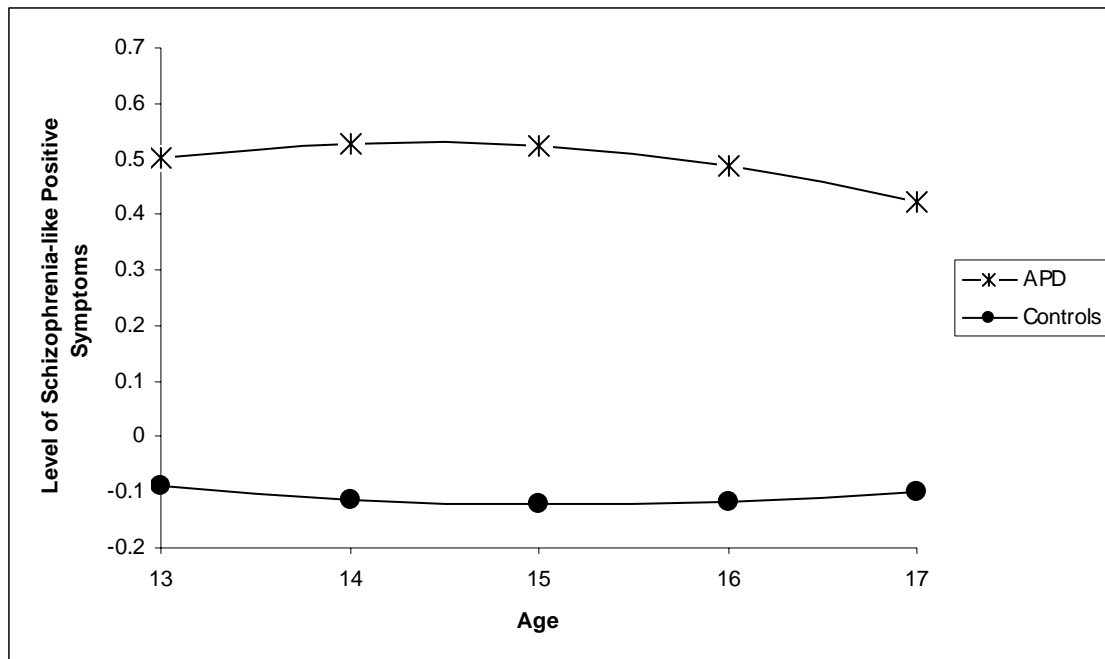


Figure F1.  
*Mean Estimated Quadratic Growth Trajectories for Schizophrenia-like Positive Symptoms*

(APPENDIX F continued on next page)

APPENDIX F (continued)

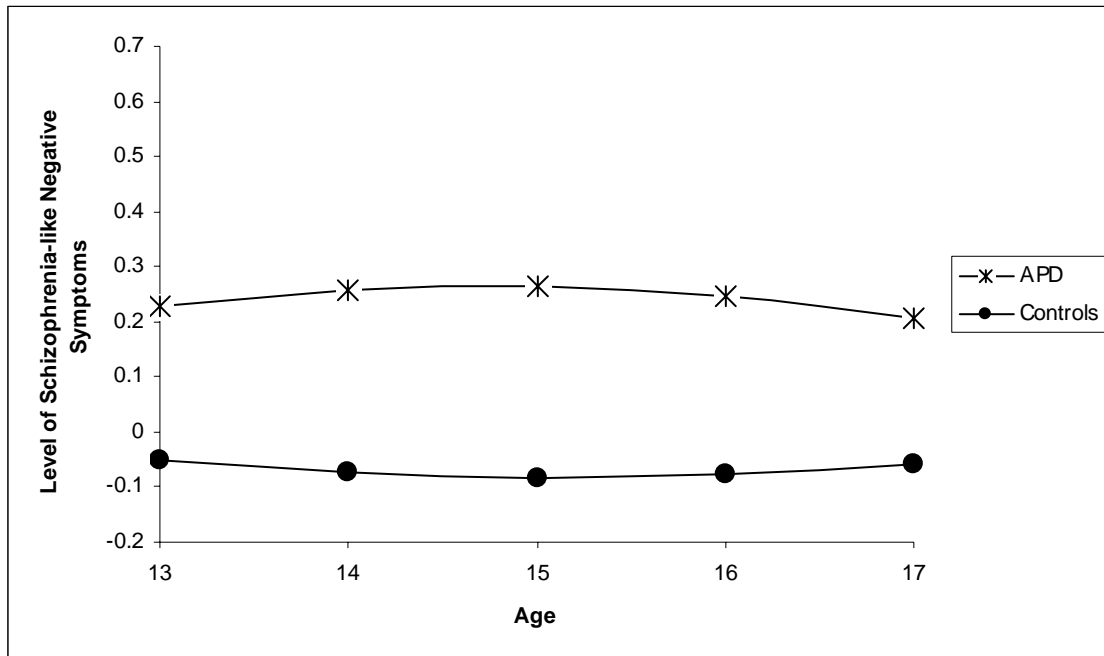


Figure F2.  
*Mean Estimated Quadratic Growth Trajectories for Schizophrenia-like Negative Symptoms*

(APPENDIX F continued on next page)

APPENDIX F (continued)

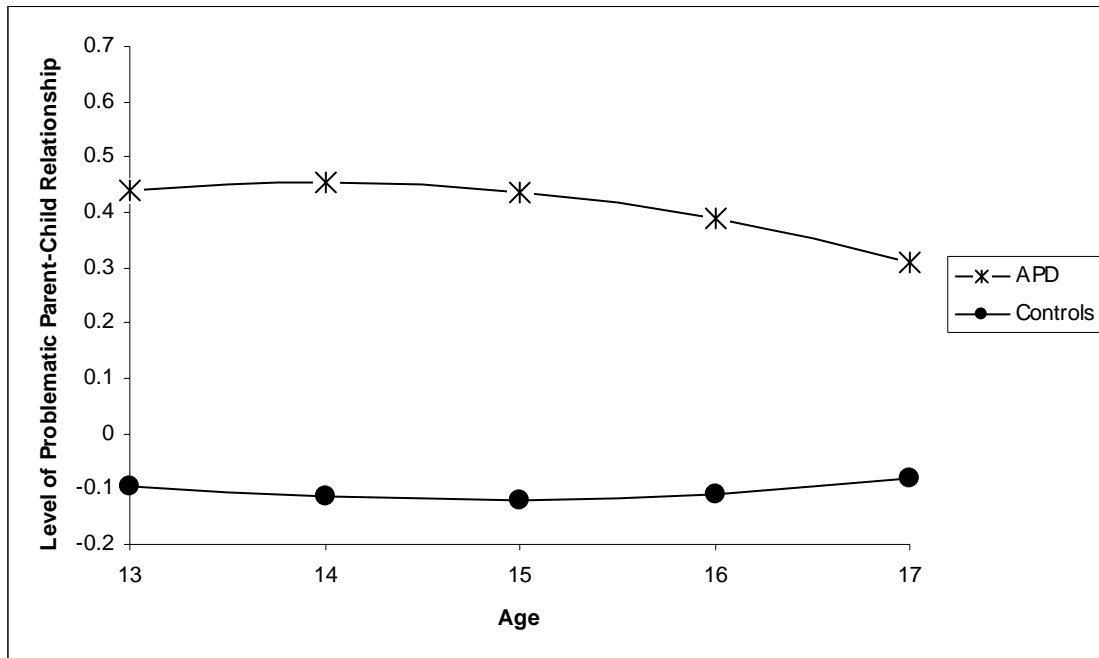


Figure F3.  
*Mean Estimated Quadratic Growth Trajectories for Parent-Child Relationship*

*Note.* APD=antisocial personality disorder

## BIBLIOGRAPHY

- Achenbach, T. M., & Edelbrock, C. S. (1983). *Manual for the Child Behavior Checklist and Revised Child Behavior Profile*. Burlington, VT: University of Vermont, Department of Psychiatry.
- Achenbach, T. M., & Edelbrock, C. S. (1986). *Manual for the Teacher's Report Form and Teacher Version of the Child Behavior Profile*. Burlington, VT: University of Vermont, Department of Psychiatry.
- Achenbach, T. M., & Edelbrock, C. S. (1987). *Manual for the Youth Self Report and Profile*. Burlington, VT: University of Vermont, Department of Psychiatry.
- Ambelas, A. (1992). Preschizophrenics: Adding to the evidence, sharpening the focus. *British Journal of Psychiatry*, *160*, 401-404.
- American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4<sup>th</sup> ed.). Washington, DC: American Psychiatric Association.
- an der Heiden, W., & Häfner, H. (2000). The epidemiology of onset and course of schizophrenia. *European Archives of Psychiatry*, *250*, 292-303.
- Andreasen, N. C. (1982). Negative symptoms in schizophrenia: Definition and reliability. *Archives of General Psychiatry*, *39*, 784-788.
- Andreasen, N. C. (1986). Scale for the assessment of thought, language and communication (TLC). *Schizophrenia Bulletin*, *12*, 473-482.
- Asarnow, J. R., & Goldstein, M. J. (1986). Schizophrenia during adolescence and early adulthood: A developmental perspective on risk research. *Clinical Psychology Review*, *6*, 211-235.
- Bearden, C. E., Rosso, I. M., Hollister, J. M., Sanchez, L. E., Hadley, T., & Cannon, T. D. (2000). A prospective cohort study of childhood behavioral deviance and language abnormalities as predictors of adult schizophrenia. *Schizophrenia Bulletin*, *26*(2), 395-410.
- Bower, E. M., & Shellhamer, T. A. (1960). School characteristics of male adolescents who later became schizophrenic. *American Journal of Orthopsychiatry*, *30*, 712-729.

- Butzlaff, R. L., & Hooley, J. M. (1998). Expressed emotion and psychiatric relapse: A meta-analysis. *Archives of General Psychiatry*, *55*(6), 547-552.
- Cannon, M., Caspi, A., Moffitt, T. E., Harrington, H., Taylor, A., Murray, R., & Poulton, R. (2002). Evidence of early-childhood pan-developmental impairment specific to schizophreniform disorder: Results from a longitudinal birth cohort. *Archives of General Psychiatry*, *59*(5), 449-456.
- Cannon, M., Jones, P., Gilvarry, C., Rifkin, L., McKenzie, K., Foerster, A., & Murray, R. M. (1997). Premorbid social functioning in schizophrenia and bipolar disorder: Similarities and differences. *American Journal of Psychiatry*, *154*(11), 1544-1550.
- Cannon, M., Walsh, E., Hollis, C., Kargin, M., Taylor, E., Murray, R. M., & Jones, P. B. (2001). Predictors of later schizophrenia and affective psychosis among attendees at a child psychiatry department. *British Journal of Psychiatry*, *178*, 420-426.
- Cannon, T. D., Kaprio, J., Lonqvist, J., Huttunen, M., & Koskenvuo, M. (1998). The genetic epidemiology of schizophrenia in a Finnish twin cohort: A population-based modeling study. *Archives of General Psychiatry*, *55*(1), 67-74.
- Carter, J. W., Schulsinger, F., Parnas, J., Cannon, T., & Mednick, S. A. (2002). A multivariate prediction model of schizophrenia. *Schizophrenia Bulletin*, *28*(4), 649-682.
- Chapman, L. J., & Chapman, J. P. (1994). Putatively psychosis-prone subjects 10 years later. *Journal of Abnormal Psychology*, *103*(2), 171-183.
- Chapman, L. J., Chapman, J. P., Numbers, J. S., Edell, W. S., Carpenter, B. N., & Beckfield, D. (1984). Impulsive nonconformity as a trait contributing to the prediction of psychotic-like schizotypal symptoms. *Journal of Nervous and Mental Disease*, *172*, 681-691.
- Chapman, L. J., Chapman, J. P., & Raulin, M. L. (1976). Scales for physical and social anhedonia. *Journal of Abnormal Psychology*, *85*, 374-382.
- Chapman, L. J., Chapman, J. P., & Raulin, M. L. (1978). Body-image aberration in schizophrenia. *Journal of Abnormal Psychology*, *87*, 399-407.
- Cohen, J. (1977). *Statistical power analysis for the behavioral sciences* (Revised ed.). NY: Academic Press.
- Corcoran, C., Walker, E., Huot, R., Mittal, V., Tessner, K., Kestler, L., & Malaspina, D. (2003). The stress cascade and schizophrenia: Etiology and onset. *Schizophrenia Bulletin*, *29*(4), 671-692.
- Cornblatt, B. A. (2002). The New York High Risk Project to the Hillside Recognition and Prevention (RAP) Program. *American Journal of Medical Genetics (Neuropsychiatric Genetics)*, *114*, 956-966.

- Cornblatt, B. A., Lencz, T., Smith, C. W., Correll, C. U., Auther, A. M., & Nakayama, E. (2003). The schizophrenia prodrome revisited: A neurodevelopmental perspective. *Schizophrenia Bulletin*, 29(4), 633-651.
- Crow, T. J., Done, D. J., & Sacker, A. (1995). Childhood precursors of psychosis as clues to its evolutionary origins. *European Archives of Psychiatry and Clinical Neuroscience*, 245, 61-69.
- Curran-Everett, D. (2000). Multiple comparisons: Philosophies and illustrations. *American Journal of Physiology: Regulatory, Integrative, and Comparative Physiology*, 279, R1-R8.
- Davidson, M., Reichenberg, A., Rabinowitz, J., Weiser, M., Kaplan, Z., & Mark, M. (1999). Behavioral and intellectual markers for schizophrenia in apparently healthy male adolescents. *American Journal of Psychiatry*, 156(9), 1328-1335.
- Dazzan, P., Kravariti, E., Fearon, P., & Murray, R. M. (2004). Is the development of schizophrenia predictable? In W. S. Stone & S. V. Faraone & M. T. Tsuang (Eds.), *Early Clinical Intervention and Prevention in Schizophrenia* (pp. 225-252). Totowa, NJ: Humana Press.
- Done, D. J., Crow, T. J., Jonestone, E. C., & Sacker, A. (1994). Childhood antecedents of schizophrenia and affective illness: Social adjustment at ages 7 and 11. *British Medical Journal*, 309(6956), 699-703.
- Eckblad, M., & Chapman, L. J. (1983). Magical ideation as an indicator of schizotypy. *Journal of Consulting and Clinical Psychology*, 51, 215-225.
- Erlenmeyer-Kimling, L., Cornblatt, B. A., Rock, D., Roberts, S., Bell, M., & West, A. (1993). The New York High Risk Project: Anhedonia, attentional deviance, and psychopathology. *Schizophrenia Bulletin*, 19, 141-153.
- Erlenmeyer-Kimling, L., Rock, D., Roberts, S., Janal, M., Kestenbaum, C., Cornblatt, B., Adamo, U. H., & Gottesman, I. I. (2000). Attention, memory, and motor skills as childhood predictors of schizophrenia-related psychoses: The New York High-Risk Project. *American Journal of Psychiatry*, 157(9), 1416-1422.
- Feinberg, I. (1982). Schizophrenia and late maturational brain changes in man. *Psychopharmacology Bulletin*, 18(3), 29-31.
- Feinberg, I. (1982/83). Schizophrenia: Caused by a fault in programmed synaptic elimination during adolescence? *Journal of Psychiatric Research*, 17(4), 319-334.
- Goldstein, M. J. (1987). The UCLA High-Risk Project. *Schizophrenia Bulletin*, 13(3), 505-514.

- Gottesman, I. I. (1991). *Schizophrenia Genesis: The Origins of Madness*. New York: W. H. Freeman.
- Gross, G., Huber, G., Klosterkötter, J., Linz, M. (1987). Bonner Skala für die Beurteilung von Basissymptomen (BSABS; Bonn Scale for the Assessment of Basic Symptoms). Berlin: Springer.
- Häfner, H., & an der Heiden, W. (1999). The course of schizophrenia in light of modern follow-up studies: the ABC and WHO studies. *European Archives of Psychiatry and Clinical Neuroscience*, 249 (suppl. 4), IV/14-26.
- Harrison, P. J., & Eastwood, S. L. (2001). Neuropathological studies of synaptic connectivity in the hippocampal formation in schizophrenia. *Hippocampus*, 11, 508-519.
- Hartmann, E., Milofsky, E., Vaillant, G., Oldfield, M., Falke, R., & Ducey, C. (1984). Vulnerability to schizophrenia: Prediction of adult schizophrenia using childhood information. *Archives of General Psychiatry*, 41, 1050-1056.
- Isohanni, M., Isohanni, I., Koponen, H., Koskinen, J., Laine, P., Lauronen, E., Miettunen, J., Mäki, P., Riala, K., Räsänen, S., Saari, K., Tienari, P., Veijola, J., & Murray, G. (2004). Developmental precursors of psychosis. *Current Psychiatry Reports*, 6, 168-175.
- Jones, P. (1997). The early origins of schizophrenia. *British Medical Bulletin*, 53(1), 135-155.
- Jones, P., Rodgers, B., Murray, R., & Marmot, M. (1994). Child developmental risk factors for adult schizophrenia in the British 1946 birth cohort study. *Lancet*, 344, 1398-1402.
- Keshavan, M. S., Anderson, S., & Pettegrew, J. W. (1994). Is schizophrenia due to excessive synaptic pruning in the prefrontal cortex? The Feinberg hypothesis revisited. *Journal of Psychiatric Research*, 28(3), 239-265.
- Klosterkötter, J., Schultze-Lutter, F., Gross, G., Huber, G., & Steinmeyer, E. M. (1997). Early self-experienced neuropsychological deficits and subsequent schizophrenic diseases: An 8-year average follow-up prospective study. *Acta Psychiatrica Scandinavica*, 95(5), 396-404.
- Kraemer, H. C., Kazdin, A. E., Offord, D. R., Kessler, R. C., Jensen, P. S., & Kupfer, D. J. (1997). Coming to terms with the terms of risk. *Archives of General Psychiatry*, 54(4), 337-343.
- Kremen, W. S., Buka, S., Seidman, L. J., Goldstein, J. M., Koren, D., & Tsuang, M. T. (1998). IQ decline during childhood and adult psychotic symptoms in a community sample: a 19-year longitudinal study. *American Journal of Psychiatry*, 155(5), 672-677.



- Lencz, T., Smith, C. W., Auther, A., Correll, C. U., & Cornblatt, B. A. (2003). The assessment of "prodromal schizophrenia": Unresolved issues and future directions. *Schizophrenia Bulletin*, 29(4), 717-728.
- Levin, J. R., Serlin, R. C., & Seaman, M. A. (1994). A controlled, powerful multiple-comparison strategy for several situations. *Psychological Bulletin*, 115(1), 153-159.
- Loeber, R., Drinkwater, M., Yanming, Y., Anderson, S. J., Schmidt, L. C., & Crawford, A. (2000). Stability of family interaction from ages 6 to 18. *Journal of Abnormal Child Psychology*, 28(4), 353-369.
- Loeber, R., Farrington, D. P., Stouthamer-Loeber, M., Moffitt, T. E., Caspi, A., White, H. R., Wei, E. H., & Beyers, J. M. (2002). The development of male offending: Key findings from fourteen years of the Pittsburgh Youth Study. In Thornberry & Krohn (Eds.), *Taking Stock of Delinquency: An Overview of Findings from Contemporary Longitudinal Studies* (pp. 93-136). New York: Kluwer Academic/Plenum Publishers.
- Loeber, R., Farrington, D. P., Stouthamer-Loeber, M., & Van Kammen, W. B. (1998). *Antisocial behavior and mental health problems*. Mahwah, NJ: Lawrence Erlbaum Associates.
- Lukoff, D., Liberman, R. P., & Nuechterlein, K. H. (1986). Symptom monitoring in the rehabilitation of schizophrenic patients. *Schizophrenia Bulletin*, 12, 578-602.
- Mäki, P., Veijola, J., Joukamaa, M., Läärä, E., Hakko, H., Jones, P. B., & Isohanni, M. (2003). Maternal separation at birth and schizophrenia - a long-term follow-up of the Finnish Christmas Seal Home Children. *Schizophrenia Research*, 60, 13-19.
- Malmberg, A., Lewis, G., David, A., & Allebeck, P. (1998). Premorbid adjustment and personality of people with schizophrenia. *British Journal of Psychiatry*, 172(4), 308-313.
- McGlashan, T. H., & Hoffman, R. E. (2000). Schizophrenia as a disorder of developmentally reduced synaptic connectivity. *Archives of General Psychiatry*, 57, 637-648.
- McGorry, P. (1998). Preventive strategies in early psychosis: Verging on reality. *British Journal of Psychiatry*, 172(Suppl. 33), 1-2.
- McGorry, P. D., Edwards, J., Mihalopoulos, C., Harrigan, S. M., & Jackson, H. J. (1996). EPPIC: An evolving system of early detection and optimal management. *Schizophrenia Bulletin*, 22(2), 305-326.
- McGorry, P. D., Yung, A. R., & Phillips, L. J. (2002). "Closing In": What features predict the onset of first-episode psychosis in an ultra high-risk group? In R. B. Zipursky & S. C. Schulz (Eds.), *The Early Stages of Schizophrenia* (pp. 3-31). D.C.: American Psychiatric Publishing, Inc.

- Meehl, P. E. (1962). Schizotaxia, schizotypy, and schizophrenia. *American Psychologist*, *17*, 827-838.
- Michael, C. M., Morris, D. P., & Soroker, E. (1957). Follow-up studies of shy, withdrawn children II: Relative incidence of schizophrenia. *American Journal of Orthopsychiatry*, *27*, 331-337.
- Miller, T. J., McGlashan, T. H., Rosen, J. L., Cadenhead, K., Ventura, J., McFarlane, W., et al. (2003). Prodromal assessment with the Structured Interview for Prodromal Syndromes and the Scale of Prodromal Symptoms: Predictive validity, inter-rater reliability, and training to reliability. *Schizophrenia Bulletin*, *29*(4), 703-715.
- Miller, T. J., McGlashan, T. H., Rosen, J. L., Somjee, L., Markovich, P. J., Stein, K., & Woods, S. W. (2002). Prospective diagnosis of the initial prodrome for schizophrenia based on the Structured Interview for Prodromal Syndromes: Preliminary evidence of interrater reliability and predictive validity. *American Journal of Psychiatry*, *159*(5), 863-865.
- Miller, T. J., McGlashan, T. H., Woods, S. W., Stein, K., Driesen, N., Corcoran, C. M., et al. (1999). Symptom assessment in schizophrenic prodromal states. *Psychiatric Quarterly*, *70*, 273-287.
- Murray, R. M., & Fearon, P. (1999). The developmental 'risk factor' model of schizophrenia. *Journal of Psychiatric Research*, *33*, 497-499.
- Murray, R. M., & Lewis, S. W. (1987). Is schizophrenia a neurodevelopmental disorder? *British Medical Journal*, *295*, 681-682.
- Norman, R. M. G., & Malla, A. K. (1993). Stressful life events and schizophrenia I: A review of research. *British Journal of Psychiatry*, *162*, 161-166.
- Offord, D. R., & Cross, L. A. (1969). Behavioral antecedents of adult schizophrenia. *Archives of General Psychiatry*, *21*, 267-283.
- Olin, S. S., John, R. S., & Mednick, S. A. (1995). Assessing the predictive value of teacher reports in a high risk sample for schizophrenia: a ROC analysis. *Schizophrenia Research*, *16*, 53-66.
- O'Neal, P., & Robins, L. N. (1958). Childhood patterns predictive of adult schizophrenia: A 30-year follow-up study. *American Journal of Psychiatry*, *November*, 385-391.
- Ott, S. L., Allen, J., & Erlenmeyer-Kimling, L. (2001). The New York High-Risk Project: Observations on the rating of early manifestations of schizophrenia. *American Journal of Medical Genetics (Neuropsychiatric Genetics)*, *105*, 25-27.
- Overall, J. E., & Gorham, D. R. (1962). The Brief Psychiatric Rating Scale. *Psychological Reports*, *10*, 799-812.

- Parnas, J. (1999). From predisposition to psychosis: Progression of symptoms in schizophrenia. *Acta Psychiatrica Scandinavica, Supplementum*, 99(395), 20-29.
- Parnas, J., Schulsinger, F., Schulsinger, H., Mednick, S. A., & Teasdale, T. W. (1982). Behavioral precursors of schizophrenia spectrum. *Archives of General Psychiatry*, 39, 658-664.
- Perkins, D. O., Gu, H., Boteva, K., & Lieberman, J. A. (2005). Relationship between duration of untreated psychosis and outcome in first-episode schizophrenia: A critical review and meta-analysis. *American Journal of Psychiatry*, 162(10), 1785-1804.
- Pogue-Geile, M. F. (1991). The development of liability to schizophrenia: Early and late developmental models. In E. F. Walker (Ed.), *Schizophrenia: A Life-course Developmental Perspective* (pp. 277-298).
- Pogue-Geile, M. F., & Harrow, M. (1984). Strategies for psychopathology research. In A. S. Bellack & M. Hersen (Eds.), *Research Methods in Clinical Psychology*. NY: Pergamon Press.
- Poulton, R., Caspi, A., Moffitt, T., Cannon, M., Murray, R., & Harrington, H. (2000). Children's self-reported psychotic symptoms and adult schizophreniform disorder: A 15-year longitudinal study. *Archives of General Psychiatry*, 57(11), 1053-1058.
- Rabinowitz, J., Reichenberg, A., Weiser, M., Mark, M., Kaplan, Z., & Davidson, M. (2000). Cognitive and behavioural functioning in men with schizophrenia both before and shortly after first admission to hospital: Cross-sectional analysis. *British Journal of Psychiatry*, 177, 26-32.
- Rakfeldt, J., & McGlashan, T. H. (2004). The nature of the prodrome in schizophrenia. In W. S. Stone & S. V. Faraone & M. T. Tsuang (Eds.), *Early Clinical Intervention and Prevention in Schizophrenia* (pp. 75-91). Totowa, NJ: Humana Press.
- Raudenbush, S. W., & Bryk, A. S. (2002). *Hierarchical linear models: Applications and data analysis methods* (2<sup>nd</sup> ed.). Thousand Oaks, CA: Sage.
- Raudenbush, S., Bryk, A., & Congdon, R. (2005). Hierarchical Linear and Nonlinear Modeling (Version 6.1). [Computer software]. Lincolnwood, IL: Scientific Software International, Inc.
- Reichenberg, A., Weiser, M., Rabinowitz, J., Caspi, A., Schmeidler, J., Mark, M., Kaplan, Z., & Davidson, M. (2002). A population-based cohort study of premorbid intellectual, language, and behavioral functioning in patients with schizophrenia, schizoaffective disorder, and nonpsychotic bipolar disorder. *American Journal of Psychiatry*, 159(12), 2027-2035.

- Reiss, D., Hetherington, M., Plomin, R., Howe, G. W., Simmens, S. J., Henderson, S. H., O'Conner, T. J., Bussell, D. A., Anderson, E. R., & Law, T. (1995). Genetic questions for environmental studies: Differential parenting and psychopathology in adulthood. *Archives of General Psychiatry*, *52*, 925-936.
- Ricks, D. F., & Berry, J. C. (1970). Family and symptom patterns that precede schizophrenia. In M. Roff & D. F. Ricks (Eds.), *Life History Research in Psychopathology* (Vol. 1, pp. 31-50). Minneapolis: The University of Minnesota Press.
- Robins, L., Cottler, L., Buchholz, K., & Compton, W. (1998). *Diagnostic Interview Schedule for DSM-IV*. St. Louis, MO: Washington University.
- Roff, J. D. (2001). Comparison of childhood problem behaviors in boys with subsequent schizophrenic, antisocial, and good adult outcomes. *Psychological Reports*, *89*, 633-640.
- Roff, J. D., & Fultz, J. M. (2003). Childhood antecedents of schizophrenia: Developmental sequencing and specificity of problem behavior. *Psychological Reports*, *92*, 793-803.
- Schiffman, J., Abrahamson, A., Cannon, T., LaBrie, J., Parnas, J., Schulsinger, F., & Mednick, S. (2001). Early rearing factors in schizophrenia. *International Journal of Mental Health*, *30*(1), 3-16.
- Schiffman, J., LaBrie, J., Carter, J., Cannon, T., Schulsinger, F., Parnas, J., & Mednick, S. (2002). Perception of parent-child relationships in high-risk families, and adult schizophrenia outcome of offspring. *Journal of Psychiatric Research*, *36*, 41-47.
- Stott, G. H. (1987). The social adjustment of children. Manual to the Bristol social adjustment guides. London: Hodder and Stoughton.
- Stouthamer-Loeber, M., Loeber, R., Wei, E., Farrington, D. P., & Wikström, P.-O. H. (2002). Risk and promotive effects in the explanation of persistent serious delinquency in boys. *Journal of Consulting and Clinical Psychology*, *70*(1), 111-123.
- Tabachnick, B. G., & Fidell, L. S. (1996). *Using multivariate statistics* (3<sup>rd</sup> ed.). New York: Harper Collins.
- Thompson, J. L., Pogue-Geile, M. F., & Grace, A. A. (2004). Developmental pathology, dopamine, and stress: A model for the age of onset of schizophrenia symptoms. *Schizophrenia Bulletin*, *30*(4), 875-900.
- Thompson, J. L., Pogue-Geile, M. F., Stouthamer-Loeber, M., Stallings, R., & Loeber, R. (2005). *A prospective study of adolescent cannabis use and early adulthood psychotic symptoms*. Presented at the 2005 International Congress on Schizophrenia Research, Savannah, GA.

- Tienari, P., Lahti, I., Sorri, A., Naarala, M., Moring, J., & Wahlberg, K.-E. (1989). The Finnish Adoptive Family Study of Schizophrenia: Possible joint effects of genetic vulnerability and family environment. *British Journal of Psychiatry*, *155*(Suppl. 5), 29-32.
- Tienari, P., Wynne, L. C., Moring, J., Lahti, I., Naarala, M., Sorri, A., Wahlberg, K.-E., Saarento, O., Seitamaa, M., Kaleva, M., & Läksy, K. (1994). The Finnish Adoptive Study of Schizophrenia: Implications for family research. *British Journal of Psychiatry*, *164*(Suppl. 23), 20-26.
- Tienari, P., Wynne, L. C., Sorri, A., Lahti, I., Läksy, K., Moring, J., Naarala, M., Nieminen, P., & Wahlberg, K.-E. (2004). Genotype-environment interaction in schizophrenia-spectrum disorder: Long-term follow-up study of Finnish adoptees. *British Journal of Psychiatry*, *184*, 216-222.
- Turner, E. E., Fedtsova, N., & Jeste, D. V. (1997). Cellular and molecular neuropathology of schizophrenia: New directions from developmental neurobiology. *Schizophrenia Research*, *27*, 169-180.
- Waddington, J. L., Lane, A., Scully, P. J., Larkin, C., & O'Callaghan, E. (1998). Neurodevelopmental and neuroprogressive processes in schizophrenia: Antithetical or complementary, over a lifetime trajectory of disease? *The Psychiatric Clinics of North America*, *21*(1), 123-149.
- Wahlberg, K.-E., & Wynne, L. C. (2001). Possibilities for prevention of schizophrenia: Suggestions from research on genotype-environment interaction. *International Journal of Mental Health*, *30*(1), 91-103.
- Walker, E. F. (1994). Developmentally moderated expressions of the neuropathology underlying schizophrenia. *Schizophrenia Bulletin*, *20*(3), 453-480.
- Walker, E. F., & Diforio, D. (1997). Schizophrenia: A neural diathesis-stress model. *Psychological Review*, *104*(4), 667-685.
- Walker, E. F., Grimes, K. E., Davis, D. M., & Smith, A. J. (1993). Childhood precursors of schizophrenia: Facial expressions of emotion. *American Journal of Psychiatry*, *150*(11), 1654-1660.
- Walker, E., Lewis, N., Loewy, R., & Palyo, S. (1999). Motor dysfunction and risk for schizophrenia. *Development and Psychopathology*, *11*, 509-523.
- Walker, E. F., Neumann, C. C., Baum, K., Davis, D. M., Diforio, D., & Bergman, A. (1996). The developmental pathways to schizophrenia: Potential moderating effects of stress. *Development and Psychopathology*, *8*, 647-665.
- Waring, M., & Ricks, D. (1965). Family patterns of children who became adult schizophrenics. *Journal of Nervous and Mental Disease* *140*(5), 351-364.

- Watt, N. F. (1978). Patterns of childhood social development in adult schizophrenics. *Archives of General Psychiatry*, 35, 160-165.
- Weinberger, D. R. (1987). Implications of normal brain development for the pathogenesis of schizophrenia. *Archives of General Psychiatry*, 44, 660-669.
- Woods, B. T. (1998). Is schizophrenia a progressive neurodevelopmental disorder? Toward a unitary pathogenetic mechanism. *American Journal of Psychiatry*, 155(12), 1661-1670.
- Youngstrom, E., Loeber, R., & Stouthamer-Loeber, M. (2000). Patterns and correlates of agreement between parent, teacher, and male adolescent ratings of externalizing and internalizing problems. *Journal of Consulting and Clinical Psychology*, 68(6), 1038-1050.
- Yung, A. R., Phillips, L. J., McGorry, P. D., McFarlane, C. A., Francey, S., Harrigan, S., Patton, G. C., & Jackson, H. J. (1998). Prediction of psychosis: A step towards indicated prevention of schizophrenia. *British Journal of Psychiatry*, 172(Suppl. 33), 14-20.
- Yung, A. R., Phillips, L. J., Yuen, H. P., Francey, S. M., McFarlane, C. A., Hallgren, M., & McGorry, P. D. (2003). Psychosis prediction: 12-month follow up of a high-risk ("prodromal") group. *Schizophrenia Research*, 60, 21-32.