Published in final edited form as:

Behav Genet. 2011 July ; 41(4): 499–511. doi:10.1007/s10519-010-9401-x.

Heritability and Longitudinal Stability of Schizotypal Traits During Adolescence

Marissa Ericson,

Department of Psychology (SGM 501), University of Southern California, 3620 S.McClintock Ave, Los Angeles, CA 90089-1061, USA

Catherine Tuvblad,

Department of Psychology (SGM 501), University of Southern California, 3620 S.McClintock Ave, Los Angeles, CA 90089-1061, USA

Adrian Raine,

Departments of Criminology, Psychiatry and Psychology, University of Pennsylvania, Philadelphia, PA, USA

Kelly Young-Wolff, and

Department of Psychology (SGM 501), University of Southern California, 3620 S.McClintock Ave, Los Angeles, CA 90089-1061, USA

Laura A. Baker

Department of Psychology (SGM 501), University of Southern California, 3620 S.McClintock Ave, Los Angeles, CA 90089-1061, USA

Marissa Ericson: marissae@usc.edu

Abstract

The study investigated the genetic and environmental etiology of schizotypal personality traits in a non-selected sample of adolescent twins, measured on two occasions between the ages of 11 and 16 years old. The 22-item Schizotypal Personality Questionnaire-Child version (SPQ-C) was found to be factorially similar to the adult version of this instrument, with three underlying factors (Cognitive-Perceptual, Interpersonal-Affective, and Disorganization). Each factor was heritable at age 11–13 years ($h^2 = 42\text{--}53\%$) and 14–16 years old ($h^2 = 38\text{--}57\%$). Additive genetic and unique environmental influences for these three dimensions of schizotypal personality acted in part through a single common latent factor, with additional genetic effects specific to both Interpersonal-Affective and Disorganization subscales at each occasion. The longitudinal correlation between the latent schizotypy factor was $r = 0.58$, and genetic influences explained most of the stability in this latent factor over time (81%). These longitudinal data demonstrate significant genetic variance in schizotypal traits, with moderate stability between early to middle adolescence. In addition to common influences between the two assessments, there were new genetic and non-shared environmental effects that played a role at the later assessment, indicating significant change in schizotypal traits and their etiologies throughout adolescence.

Keywords

Schizotypy; Schizotypal personality; Genetics; Heritability; Longitudinal

Schizotypal personality (schizotypy) has been considered an endophenotype for schizophrenia, in that it shares neurological, psychophysiological, and behavioral characteristics associated with disorders of the schizophrenia spectrum (Gottesman and Gould 2003; Lenzenweger 2006; Linney et al. 2003). Behavioral genetic studies have demonstrated heritable factors are important for schizotypal personality in adults (Linney et al. 2003). Despite the fact that self-report screening has been found to aid in the detection of liable individuals in the adult population (MacDonald et al. 2001), there is currently no instrument to assess schizotypal traits in child or adolescent samples. Additionally, no studies to date have investigated the influence of genetic and environmental factors that contribute to the stability of schizotypal traits in adolescence using a measure designed to assess traits during this developmental period. It has been argued that schizotypal personality traits are qualitatively similar to the characteristic symptoms of schizophrenia, albeit quantitatively less severe. As such, it behooves us to understand the development and etiology of schizotypal personality traits, in order to aid our understanding of the wider schizophrenia spectrum. The aim of the present study was thus to investigate the genetic and environmental influences and their stability from ages 11–16 years old in a longitudinal twin study.

Given that the age of onset for schizophrenia occurs in late adolescence or early adulthood [American Psychiatric Association Diagnostic and Statistical Manual of Mental Disorders (DSM-IV-TR), 2004], investigating schizotypal traits, especially during development, offers a promising way to identify individuals with schizophrenia latent liability (Claridge 1987; Meehl 1989). The validity of this approach is suggested by an increase in liability for categorically defined Schizotypal Personality Disorder (SPD) and schizotypal personality in the relatives of patients with schizophrenia (Kendler 1985), as well as elevated probability of psychotic episodes in individuals who present with high levels of schizotypy (Chapman et al. 1994; Linney et al. 2003). Psychometrically measured schizotypy has been found to be elevated among schizophrenia patients (Chapman et al. 1978) and their relatives (Chen et al. 1998), and those with higher schizotypal scores have more relatives with schizophrenia (Chapman et al. 1994). Moreover, twin studies have shown schizotypal personality disorder is genetically related to schizophrenia and therefore may reflect some facets of genetic predisposition to schizophrenia (Chang et al. 2002; Kendler et al. 1993, 1981).

Significant genetic influences have been found in studies of adult twins from the general population using many different self-report schizotypy scales (heritabilities ranging from 29 to 67%; for review, see Linney et al. 2003), although one study failed to report genetic influences on a specific scale measuring perceptual aberrations (Kendler and Hewitt 1992). In addition, the shared environment (i.e., non-genetic factors that contribute to twin similarity) has been found to influence both the positive symptoms (Kendler and Hewitt 1992; MacDonald et al. 2001), as well as the negative symptoms of schizotypy in adults (Miller 1993).

The dimensional structure of schizotypal traits has also been investigated, both phenotypically and genetically. Schizotypal personality has been shown to have a highly analogous dimensional structure in the general population, with latent factors resembling the classical schizophrenia positive, negative, and disorganization symptom dimensions (Raine et al. 1994; Raine 1991; Vollema and Van den Bosch 1995; Claridge et al. 1996; Mason et al. 2005). The majority of evidence supports three symptom clusters: (1) Cognitive-Perceptual deficits (comprised of Ideas of Reference, Magical Thinking, Unusual Perceptual Experiences, and Paranoid Ideation), (2) Interpersonal-Affective deficits (Social Anxiety, No Close Friends, Blunted Affect, Paranoid Ideation), and (3) Disorganized behavior (Odd Behavior and Odd Speech).

Factor analytic studies of adults have reported two or three phenotypic factors, depending on the number and item content of scales. For example, a three-factor model was found to fit the data better than a single or a two-factor model; these three schizotypal factors paralleled the three reported schizophrenia symptom dimensions (Mata et al. 2000, 2003). Further, using the Schizotypal Personality Questionnaire (SPQ; Raine 1991) a three-factor model, comprised of Cognitive–Perceptual deficits, Interpersonal Deficits, and Disorganization, was found to underlie individual differences in both schizophrenic and normal samples (Rossi and Daneluzzo 2002). This demonstrates some parallel aspects between schizotypal traits and schizophrenia symptoms, at least phenotypically.

There are only a few studies that have investigated genetic and environmental influences on the symptom dimensions of schizotypal traits. One adult twin study suggested a latent common schizotypy factor in both males and females (MacDonald et al. 2001). This latent factor—influenced by additive genetic (15%), shared-environmental (45%) and unique environmental effects (i.e., environmental factors not shared by twins, but which serve to make co-twins different from one another) (40%)—was mainly characterized by positive symptoms (e.g., perceptual aberration, magical ideation). Moreover, scores on the positive symptom dimension of SPQ administered to relatives of patients with psychotic disorders were found to relate most strongly to their genetic risk of psychosis (Vollema et al. 2002). However, in another study that only included female twins (Linney et al. 2003), one core dimension could not sufficiently account for the genetic covariation among the various schizotypy components of the Oxford-Liverpool Inventory of Feelings and Experiences (Mason et al. 1995) and the Peters Delusions Inventory (PDI; Peters et al. 1996). Two latent factors were required—each influenced by genetic and non-shared environmental effects—such that one factor was essentially comprised of the positive symptom scales and the second factor was based on negative dimension scales. Both latent factors had loadings from the cognitive scale, suggesting that while schizotypy traits may be phenotypically multidimensional, both positive and negative aspects may be genetically related to the cognitive disorganization dimension. Although phenotypic studies in adults consistently suggest as many as two or three underlying dimensions may characterize schizotypal traits, the dimensional structure of the genetic and environmental influences remains unclear.

Only one study to date has investigated the genetic and environmental etiology of schizotypal traits during adolescence (Lin et al. 2007). Using the SPQ (Raine 1991) and the Perceptual Aberration Scale (PAS; Chapman et al. 1978) in 12–16 year old Taiwanese twins, 41–49% of the variance of the PAS and the three SPQ subscales were accounted for by genetic factors. While genetic factors were additive for the PAS and the Disorganization subscale of the SPQ, they were non-additive (i.e., including dominance and/or epistasis) on the Cognitive–Perceptual and Interpersonal subscales. The remaining variance of each subscale was accounted for by non-shared environmental factors. Though significant genetic covariation was found among these scales (bivariate heritability amongst the various traits and scales ranged from 50 to 65%), neither a single common factor nor an independent pathway model fit the data, suggesting that genetic and environmental influences underling the various schizotypal traits may be multidimensional (Lin et al. 2007). Overall, findings regarding the genetic and environmental etiologies and their underlying factor structures have yet to be replicated across different age groups and in other populations.

It must be noted that the Lin et al. study utilized a measure (SPQ) that had been validated for use in adults, in spite of the fact that sample was comprised of children and adolescents; thus, these results must be interpreted with caution. The present study utilized a newly created SPQ-Child version developed specifically for use in child and adolescent samples, with wording changes from the original SPQ-B items in order to make them more age appropriate.

It is well known that an increase in the rate of psychotic symptoms (e.g., hallucinations and delusions) and adjustment problems occurs in adolescence (Erlenmeyer-Kimling 2001; Laurens et al. 2007; Walker et al. 1996; Walker 2001), and the initial manifestation of poor adjustment generally follows the onset of puberty (Walker and Bollini 2002). Additionally, hallucinatory experiences and delusional beliefs at age 11 are both strong and specific indices of a schizophrenia spectrum disorder (SSD) diagnosis by age 26 (Poulton et al. 2000). Despite these findings, there is currently no instrument to assess schizotypal traits in child or adolescent samples.

The present study attempted to clarify further the genetic and environmental etiology of schizotypy using an unselected sample of adolescent twins assessed on two occasions. The aims of the study were fourfold: (1) to investigate the phenotypic factor structure of the SPQ-C, with the expectation that a factor structure similar to that found for the SPQ-B in adults—three symptom dimensions—will emerge and that these dimensions will share a common genetic and environmental etiology; (2) to examine the relative contributions of genetic and environmental effects on self-reported schizotypal traits during adolescence; (3) to investigate whether a common latent factor accounts for genetic and environmental covariation among the three symptom dimensions of schizotypal traits; and (4) to examine the longitudinal genetic and environmental stability of schizotypal traits between the ages of 11 and 16 years old.

Methods

Participants

The subjects were participants in the Southern California Twin Project, an ongoing longitudinal twin study assessing risk factors for aggressive and antisocial behaviors [see (Baker et al. 2006, 2007)]. The total sample includes 724 sets of twins and triplets ($N = 1,457$ adolescents) and their primary caregivers, with assessments completed to date on three different occasions (Waves 1, 2, and 3) and one more assessment currently under way (Wave 4). Twins were recruited from local schools and advertisements in the Los Angeles community. The sample is representative of the ethnic and socio-economic diversity of the greater Los Angeles area (Baker et al. 2007). The ethnic composition of the entire sample is representative of Southern California: 37% Hispanic, 27% Caucasian, 14% Black, 4% Asian, and the remaining 17% of mixed/other (Muthen and Muthen 1998–2007).

The present study utilized data from Waves 2 and 3, when the twins were ages 11–13 (mean = 11.89, $SD = 0.69$) and 14–16 years old (mean = 14.69, $SD = 0.63$), respectively. The sample includes both same-sex male and female monozygotic (MZ) and dizygotic (DZ) pairs, as well as opposite-sex DZ pairs, a total of 184 pairs with available SPQ data in Wave 2 and 481 pairs in Wave 3. Among the twin pairs with available data, 93% (171 pairs) and 90% (430 pairs) had complete data from both co-twins, for Waves 2 and 3, respectively, although several pairs had only singletons in cases where the SPQ was not completed due to time constraints during the laboratory visit. Some twin pairs in the larger study sample of 724 pairs either did not participate in Waves 2 or 3 or participated through mail surveys in which the SPQ-C was not included. Thus, the total sample on which the present analyses were based include 355 individuals (48% male, 52% female) at Wave 2 and 911 (47% male, 53% female) at Wave 3. A total of 100 twin pairs participated in both Waves 2 and 3, in which data are available for both twins at both Waves. The distribution across sex and zygosity for the present analyses was: N (individuals) = 89 MZ male twins, 93 MZ female twins, 45 DZ male twins, 55 DZ female twins, and 73 DZ opposite sex twins for Wave 2 and 180 MZ male twins, 197 MZ female twins, 141 DZ male twins, 166 DZ female twins, and 227 DZ opposite sex twins for Wave 3.

Measures

During Wave 2 and Wave 3, the twins were asked to complete the child version of the Schizotypal Personality Questionnaire (SPQ-C; Raine and Baker 2003) specifically developed for the Southern California Twin Project. The SPQ-C is a downward extension of the adult Schizotypal Personality Questionnaire-Brief (SPQ-B) self-report questionnaire (Raine and Benishay 1995) which covers the nine symptoms of SPD outlined in the Diagnostic and Statistical Manual of Mental Disorders-4th ed (DSM-IV) (American Psychiatric Association 1994). The SPQ-B in turn is a short form of the full 74-item Schizotypal Personality Questionnaire (SPQ) (Raine 1991). Consisting of 22 statements requiring a dichotomous response (1 = YES, 0 = NO), the SPQ-C was based on questions from the brief scale. Questions that appeared most reliable and valid from the full SPQ were chosen during the development of the SPQ-B; subsequently, these same questions were chosen during the construction of the SPQ-C. Fourteen of the original items on the brief SPQ-B scale for adults remained identical in the SPQ-C child version, and eight were modified to be more appropriate for children. Internal consistencies for the SPQ-C subscales ranged from $\alpha = 0.81$ to 0.92 for Wave 2 and $\alpha = 0.80$ to 0.90 for Wave 3.

For the SPQ-B in adults, both exploratory and confirmatory factor analysis have suggested a three-factor solution: Cognitive–Perceptual deficits (likened to the positive symptoms of schizophrenia), Interpersonal-Affective deficits (akin to negative symptoms in schizophrenia), and Disorganization (Raine et al. 1994). The Cognitive–Perceptual factor captured constructs of referential ideation, odd belief or magical thinking, unusual perceptual experience, and paranoid ideation. The Interpersonal-Affective factor captured constructs of excessive social anxiety, lack of close friends, inappropriate or constricted affect, and paranoid ideation. Lastly, the Disorganization factor captured constructs of odd behavior and speech patterns (Raine 1991). The three factors and total score from the SPQ-B have found to have internal consistencies ranging from $\alpha = 0.72$ to 0.80 , correlations with the full 74-item SPQ ranging from $r = 0.89$ to 0.94 , scale scores correlated with independent clinical ratings of DSM-III-R schizotypal traits (average $r = 0.62$), and test–retest correlations across a two-month interval between $r = 0.86$ to 0.95 (Axelrod et al. 2001; Raine and Benishay 1995). In addition, this three-factor solution has been replicated in at least 14 independent samples (Bedwell and Donnelly 2005; Reynolds et al. 2000), across several populations (Gruzelier et al. 1995; Gruzelier and Kaiser 1996; Chen et al. 1997; Reynolds et al. 2000) and in samples of both schizophrenic inpatients and outpatients (Vollema and Hoijsink 2000).

Zygosity determination

Zygosity was determined through DNA microsatellite analysis [>7 concordant and zero discordant markers = monozygotic (MZ); one or more discordant markers = dizygotic (DZ)] for 87% of the same-sex twin pairs. For the remaining same-sex twin pairs, zygosity was established by questionnaire items about the twins' physical similarity and the frequency with which people confuse them (Lykken 1978). The questionnaire was used only when DNA samples were insufficient for one or both twins. When both questionnaire and DNA results were available, there was 90% agreement between the two (Baker et al. 2007).

Analyses

Factor analyses of the SPQ-C items—In order to examine the latent structure of schizotypal traits, both exploratory and confirmatory factor analyses were employed for the 22 SPQ-C items, separately within each wave of assessment. It was hypothesized that the factors would be inter-correlated and therefore exploratory principal component analyses (PCA) with promax oblique rotations were performed using SPSS 17.0 (Statistical Package for the Social Sciences 1998) on the 22 SPQ-C items for one randomly selected member of

each twin pair. The scree plot of eigenvalues was used to determine the number of factors to extract. The criterion cutoff was set at ± 0.35 for the item loadings. Using this criterion, each item loaded most highly on one distinct factor. Subsequent confirmatory factor analyses (CFA) were performed on the other twin of each pair using Mplus (Muthen and Muthen 2003). One and three-factor solutions were examined in CFA and compared to one another. Goodness of fit was determined by non-significant chi-square values (χ^2), RMSEA of < 0.05 , and CFI > 0.90 .

SPQ-C subscale formation—SPQ-C subscales were computed as means of items loading on the three factors identified in factor analyses. Since all three subscale means were moderately skewed, they were transformed using a ranking and normalization procedure (BLOM option in SPSS) (van den Oord et al. 2000). These standardized normal scores were used in all genetic analyses. Preliminary analyses revealed that there were no mean differences between the sexes for either the SPQ-C Wave 2 subscales ($F(3,351) = 0.791, p = 0.49$) or the SPQ-C Wave 3 subscales ($F(3, 907) = 0.36, p = 0.78$), and there were no mean differences in zygosity groups for SPQ-C Wave 2 subscales ($F(3, 348) = .21, p = 0.88$) or SPQ-C Wave 3 subscales ($F(3, 904) = .326, p = 0.81$). Thus, all subsequent analyses combined males and females into two groups: (1) all male and female MZ pairs ($N = 182$ individuals from 88 pairs) and all male, female, and opposite sex DZ twins ($N = 173$ individuals from 83 pairs) for Wave 2; and (2) all male and female MZ twins ($N = 377$ individuals from 181 pairs) and all male, female, and opposite sex DZ twins ($N = 534$ individuals from 267 pairs) for Wave 3.

Genetic analyses—Intraclass correlations and cross-twin cross-trait phenotypic correlations were first examined for indications of genetic and environmental variance and covariance among the SPQ-C scales (Neale and Cardon 1992). Saturated models, which estimate the variances, covariances and means of schizotypy subscale scores separately for twins in the zygosity groups were then fit in both univariate and multivariate analyses. These models were used as baseline models to which subsequent models of genetic and environmental influence were compared. Univariate models were fit separately to each SPQ-C subscale within each wave, to provide an initial estimation of the relative contribution of genetic (A), shared environmental (C) and non-shared (E) environmental factors to individual differences in schizotypal traits. All saturated models with means constrained across twins and zygosity fit the data for every SPQ scale ($p > .05$).

To investigate the extent to which genetic and environmental influences are stable or changing across adolescent development a series of multivariate genetic models were fit to the six subscales combined across Waves 2 and 3 simultaneously, (i.e., three subscales in each of two waves of assessment): (1) a Cholesky decomposition model, which estimated the genetic and environmental variance and covariance among the six subscales; (2) a one-factor common pathway model, in which a single latent factor exerted common influences on the six subscales, as well as the genetic and environmental effects unique to each subscale; and (3) a two correlated factor common pathway (CP) model, such that each of the two correlated common factors exerted common influences on the three subscales within each wave. The Cholesky model in which constraints of equality in variances and covariances across co-twins and zygosity groups are placed was used as the baseline to which all subsequent models were compared.

All models were fit with the structural equation program Mx (Neale et al. 2003), using a maximum likelihood estimation procedure for raw data. This raw maximum likelihood approach yields a goodness of fit index calculated as two times the log-likelihood of the data given the model ($-2LL$). The difference between $-2LL$ for two nested models follows a chi-square (χ^2) distribution, with degrees of freedom (df) equal to the difference in df for the two

models (Neale and Cardon 1992). A non-significant χ^2 is interpreted to mean that the more constrained model provides a statistically equivalent fit to the data compared to the less constrained, saturated model; on the other hand, a significant χ^2 is indicative of poor fit for the constrained model because it fits the data significantly worse than the saturated model. In addition to the likelihood-ratio χ^2 -test, the Akaike's Information Criterion (AIC) was computed for each fitted model (Akaike 1987). The AIC is a widely used index of fit, such that smaller AIC values are indicative of the most parsimonious and well-fitting model. The AIC was evaluated in all models according to this rationale in order to identify the best fitting models in both univariate and multivariate analyses.

Results

Principal component analyses of SPQ-C items

Table 1 presents exploratory factor loadings for the 22 SPQ-C items on each of the three factors, separately for each wave of assessment. The Principal Components Analysis (PCA) of the 22 SPQ-C items in a random sample of one twin from each pair yielded three large components that accounted for 69.20% of the total variance at Wave 2 and 69.08% at Wave 3. Simple structure for this three-factor solution was evident at both Waves 2 and 3, in that each item loaded most strongly on the intended factor (loadings exceeding 0.35 in all cases), and showed small or near-zero loadings (all under 0.35) for the other two unintended factors. A Cognitive-Perceptual factor (Factor 1) captured aspects of referential ideation, odd belief or magical thinking, unusual perceptual experience, and paranoid ideation. A Disorganization factor (Factor 2) captured odd behavior and speech patterns. Lastly, an Interpersonal-Affective factor (Factor 3) captured excessive social anxiety, lack of close friends, inappropriate or constricted affect, and paranoid ideation. The three factors in this adolescent sample mirrored those previously reported for both the SPQ original scales, as well as the SPQ-B (brief) scale, which were both created to assess the nine symptoms of schizotypal personality disorder outlined in the DSM-IV in community samples of adults (Raine 1991; Raine et al. 1994; Raine and Benishay 1995).

Though the SPQ-C is a newly developed measure, it is based largely on the SPQ and SPQ-B, both well-validated and widely used measures in adult samples. In the current sample, the factor structure of the SPQ-C remains consistent with its predecessors as well as classical schizophrenia symptom dimensions. Additionally, significant phenotypic ($r = -0.09$ to -0.44) and genetic correlations ($r_g = -0.14$ to -0.62) between schizotypal traits during Waves 2 and 3 and several event-related potential (ERP) paradigms have been found in our sample (Ericson et al. 2008). Known for their involvement in the pathogenesis of schizophrenia spectrum disorders, deficits in the P50, P300a, and P300b ERPs have been demonstrated in schizophrenia patients, their unaffected relatives, as well as individuals with elevated levels of schizotypal personality traits. Therefore, it is reasonable to assume that since the SPQ-C demonstrates significant relationships with known biological risk factors for schizophrenia spectrum disorders in our sample, that the measure is accurately assessing schizotypal traits.

In confirmatory factor analyses based on data from the other twin from each pair, a three-factor model fit the data according to the fit statistics for both Wave 2 and Wave 3 (see Table 2). A one-factor model was also tested, but did not fit the data well according to the fit criteria. A 3-factor solution is consistent with that previously found in adult samples (Mason et al. 2005; Mata et al. 2003, 2000; Raine 1991).

Given the patterns in both exploratory and confirmatory factor analyses, three SPQ-C subscales were then created by computing the means of items using unit weights: Cognitive-Perceptual, Interpersonal-Affective, and Disorganization. The three SPQ-C subscales and

total score had internal consistencies ranging from $\alpha = 0.81$ to 0.92 for Wave 2 and $\alpha = 0.80$ to 0.90 for Wave 3. Table 3 presents the means and standard deviations for the three subscales and SPQ-C total score prior to transforming the data. As stated earlier, there were no significant differences among means across the sexes or zygosity groups for any of the SPQ subscales or for the SPQ total score.

Correlations

All SPQ-C subscales within waves were significantly correlated with each other (r ranging from 0.40 to 0.65; $p < 0.05$). Correlations for each subscale between waves ranged from 0.26 to 0.48 ($p < 0.05$), giving a first indication of longitudinal stability. Intraclass twin correlations (see Table 4) were significant ($p < 0.05$) for each subscale for MZ pairs and consistently higher than DZ correlations, indicating genetic influences. The only DZ correlations that reached significance were for the Wave 2 Cognitive-Perceptual and Interpersonal-Affective and Wave 3 Cognitive-Perceptual subscales. The DZ cross-twin cross-trait correlations were lower than the MZ cross-twin cross trait correlations, suggesting that genetic factors contribute to the covariation among the three schizotypal symptom dimensions.

Univariate model fitting results

Model fitting results and ACE parameter estimates, along with 95% confidence intervals for each subscale within each wave are presented in the Appendix. For each variable, a saturated model that freely estimated the variances and covariances across twins and zygosity groups, was fit to the data. For the Cognitive-Perceptual scale in Wave 2, an ACE model was found to fit the data better in comparison to the saturated model ($\chi^2 = 0.89$, $df = 3$, $p = 0.89$). Next, C was dropped from the model. This AE model provided a slightly better fit when compared to the full ACE model ($\Delta\chi^2 = 1.72$, $df = 4$, $p = 0.79$) than did the CE model ($\Delta\chi^2 = 2.98$, $df = 4$, $p = 0.15$). Though both the CE and AE models fit the data, according to the AIC statistic, the AE was deemed the best fitting model. In addition, C was estimated near zero in the ACE model for the Cognitive-Perceptual subscale, and never exceeded 0.02 for any subscale. Lastly, in order to evaluate the power of this sample to reject alternative hypotheses, models that dropped the genetic and shared environmental estimates were fit to the data. The E only ($\Delta\chi^2 = 40.1$, $df = 5$, $p = < 0.01$) model fit poorly. Thus, the best fitting model for the Cognitive-Perceptual scale included genetic and non-shared environmental influences (i.e., AE model).

The aforementioned steps were completed for each subscale individually within each wave, and an AE model was found to be best-fitting and most parsimonious for each variable. Genetic influences accounted for about half of the variance in each of these subscales, while the non-shared environment accounted for the remaining half of the variance (see Appendix Table 6).

Multivariate model fitting results

Next, a series of multivariate models were fit to Waves 2 and 3 simultaneously, in order to estimate the genetic and environmental stability of schizotypal traits between the two assessments, spanning a critical time in adolescent development (age 11–16). A full Cholesky decomposition model was used as a baseline to which a one-factor Common Pathway model (Model 3, Table 5), a two-factor Common Pathway model in which factor loadings were constrained to be equal across the two waves (Model 4a, Table 5), and a two-factor Common Pathway model in which factor loadings were unconstrained across waves (Model 4b, Table 5) were compared. Among these models, the two-factor Common Pathway model, with subscale loadings free to vary between the two waves (Model 4b), provided the better fit of the data based on AIC criterion, and did not significantly differ from the

Cholesky model ($\chi^2 = 41.72$; $\Delta df = 32$; $p = 0.12$). From this unconstrained two-factor Common Pathway model all shared environmental pathways (for both common and specific factors) could be dropped as its fit was not significantly different from Model 4c ($\Delta\chi^2 = 2.98$; $\Delta df = 9$; $p = 0.96$). This was deemed the best-fitting and most parsimonious model.

Parameter estimates from the best-fitting model are provided in Fig. 1, along with 95% confidence intervals. Factor 1 (at age 11–13) was primarily explained by genetic influences (63%; i.e., $0.79^2 = 0.63$) and non-shared environmental influences (37%; i.e., $0.61^2 = 0.37$). For Factor 2 (at age 14–16) 78% (i.e., $0.60^2 + 0.65^2 = 0.78$) of the variance was explained by genetic influences, and 22% (i.e., $0.22 = 0.17^2 + 0.44^2$) was explained by non-shared environmental effects. Continuity of both genetic and unique environmental factors was found across waves. The stability ($r = 0.58$) in the latent factor over time was largely (81%) explained by stable genetic effects (i.e., $0.79 \times 0.60 / [(0.79 \times 0.60) + (0.61 \times 0.17)] = 0.81$). The remaining 29% (i.e., $0.61 \times 0.17 / [(0.79 \times 0.60) + (0.61 \times 0.17)] = 0.29$) was explained by stable unique environmental factors. Further, the latent schizotypy Wave 3 variance could be decomposed into that due to stable genetic influences (36%), stable environmental influences (3%), Wave 3-specific genetic influences (42%), and Wave 3-specific environmental influences (19%).

For the Cognitive-Perceptual subscale at Wave 3 and the Interpersonal-Affective and Disorganization subscales at Waves 2 and 3, scale-specific variance could be explained by both genetic and non-shared environmental effects. For the Cognitive-Perceptual subscale (Wave 2), the scale-specific genetic variance was non-significant as the 95% CI contained zero; for this subscale only scale-specific, non-shared environmental effects were significant. Although significant genetic and non-shared environmental covariation was found between waves of assessment at the latent level, suggesting that factors are somewhat stable through development, scale-specific genetic and non-shared environmental factors for the Interpersonal-Affective and Disorganization subscales at both time-points, as well as the Cognitive-Perceptual subscale at Wave 3, were found as well. Schizotypal traits appear to be significantly stable across development, although these longitudinal data demonstrate that new genetic and environmental processes are also at play during a later stage in development in adolescent boys and girls.

Discussion

This study sought to address gaps in the literature related to the measurement and development of schizotypal personality traits during adolescence, by implementing a newly created instrument, the SPQ-C, which was based on the established SPQ-B (the only self-report instrument based on all nine DSM-IV criteria for schizotypal personality disorder) (Raine and Benishay 1995; American Psychiatric Association 1994). Similar to that which is often found in adults, a phenotypic three-factor structure emerged in our sample of adolescent twins measured on two occasions between early to middle adolescence. This finding supports the notion that it is possible to assess schizotypal traits as early as age 11 years. Each of the three subscales—Cognitive-Perceptual, Disorganization, Interpersonal-Affective—was influenced by genetic and non-shared environmental factors, but not environmental factors shared by twins. The finding that shared environment does not contribute to schizotypy in adolescents is consistent with past twin studies of schizotypal personality traits in adults (Kendler et al. 1991; Kendler and Hewitt 1992; Linney et al. 2003; Miller 1994; MacDonald et al. 2001).

SPQ-C subscales based on these three factors were moderately inter-correlated, and the MZ cross-trait correlations were greater than the DZ cross-trait correlations, suggesting potential genetic covariation among them. Longitudinal genetic analyses demonstrated that

schizotypal traits are moderately stable across development, with significant genetic overlap among the three factors assessed on two independent occasions during different age ranges in adolescence. Additionally, new genetic and environmental factors contributed to the latent factor variance at the later assessment, which is suggestive of significant change in schizotypal traits across adolescent development. Nonetheless, for some subscales specific variance was also evident within each wave, highlighting the importance of distinguishing among different factors underlying schizotypal traits. While unique genetic and non-shared environmental variance in the Cognitive-Perceptual scale in Wave 3, and the Interpersonal and Disorganization sub-scales in both Waves 2 and 3 were significant, (i.e. what was not explained by the common schizotypy factor), no unique genetic variance was found for the Cognitive-Perceptual subscale at Wave 2. It should be noted that these unique variance components reflect effects that are specific with respect to both time (wave) and scale. Though the three factors were moderately correlated and shared a common genetic etiology, there was some genetic specificity to two of the three scales. This specificity remained consistent across the two assessments, further supporting the notion of a multidimensional phenotypic structure underlying the SPQ-C.

There was also new genetic and non-shared environmental variance for the latent schizotypy factor as well as the subscales at age 14–16 years. In particular, the longitudinal analyses speak to the importance of considering the three factors underlying schizotypy; results demonstrated both continuity as well as new variance in schizotypal traits between the two occasions, the former being primarily (but not entirely) due to stability in genetic influence and the latter being due to both genetic and non-shared environment. These findings add to the growing behavioral genetics literature indicating that adolescent schizotypy, a potential endophenotype for schizophrenia spectrum disorders, is primarily influenced by genetic and non-shared environmental factors.

To our knowledge, this is the first study to uncover one latent factor of schizotypal traits at two separate time-points. For example, in an adult, unselected sample, Linney et al. (2003) proposed that at least two latent factors are needed to account for the covariation among the symptom dimensions, namely a positive symptom dimension and a negative symptom dimension. This discrepancy gives rise to the notion that perhaps there is something quantitatively different about schizotypal traits during adolescence as compared to during adulthood. Since the SPQ-C has shown promise in its assessment of the widely agreed upon subcomponents to schizotypal personality or schizotypy, additional studies are needed to test the psychometric properties of the SPQ-C in greater depth, including the investigation of its clinical relevance and criterion validity through clinical interviews for SPD and other diagnostic measures of schizophrenia spectrum disorders. Future longitudinal studies encompassing both adolescence and adulthood will also provide the opportunity to investigate developmental changes in the structure of schizotypal traits over time.

Strengths and limitations

The results must be interpreted in light of the limitations of our sample. First, though our sample size was moderate in comparison to other twin studies on schizotypy, a larger sample size would undoubtedly increase power to detect the effect of common environment (C) and to detect potential sex differences. Past research has often reported sex differences in schizotypal traits assessed in college-aged and adult samples, namely that females tend to endorse more of the cognitive-perceptual items (positive symptom factor) and men tend to score higher on the interpersonal-affective (negative symptom factor) (Raine 1992). However, this was not the case in our adolescent sample, suggesting that sex differences potentially develop later in adolescence or even in early adulthood.

There are several strengths to the current study. This is the first study to investigate psychometrically-based schizotypal personality during adolescence, using a measure designed specifically for the assessment of schizotypal traits during childhood and adolescence. Another major strength of this study is that it employs a community sample that is representative of Los Angeles County. Past research on schizotypy has focused on undergraduates, adults, or unaffected relatives of schizophrenic patients. In contrast, this study examines the genetic etiology of schizotypal traits in an unselected sample of adolescent twins. This is particularly informative because schizophrenia research, in any respect, is characteristically rife with potential confounds, such as anti-psychotic medications, acute psychosis, or hospitalization. None of the subjects in the current sample were taking such medications, nor was there any history of severe mental illness or institutionalization, thus eliminating these confounds.

Conclusion and future directions

The present findings help clarify the phenotypic factorial structure of schizotypal traits and the etiological processes underlying this personality structure at a critical time in development. These results may contribute to the development of new strategies to accurately identify individuals at putative risk for schizophrenia spectrum disorders, prior to the manifestation of clinical symptoms. In addition, longitudinal studies have the potential to shed light on the nature of schizotypal traits during adolescence versus during early adulthood. Future studies investigating traits from adolescence to adulthood would provide important research into the continuity of such traits over time as well as potential relationships with other important and known risk factors for schizophrenia spectrum disorders.

A next step for behavioral-genetic research on schizotypal traits is to examine putative endophenotypes, both phenotypically and then at the biometric level. Conceptually, endophenotypes are tools that hold the unique potential to reveal genetic and neurobiological underpinnings of psychopathology. Schizophrenia spectrum endophenotypes—such as p50, MMN, and p300 components of the auditory evoked potential (AEP)—have been found to be highly heritable, reliable, and informative indices of neurobiological dysfunction in adults (for reviews see Turetsky et al. 2007; Hall et al. 2007). However, few studies have examined the relationship between ERP dysfunction and schizotypal traits and no studies have investigated their potential genetic association. Preliminary analyses revealed significant phenotypic and genetic correlations between ERP dysfunction and SPQ-C scores and the next step in this line of research is to examine further the genetic covariation amongst these variables during development (Ericson et al. 2008). These unanswered yet potentially informative questions seem like viable next steps in research on schizotypal personality and the potential role it plays in the schizophrenia spectrum liability puzzle.

Lastly, future studies of schizotypal traits both in unaffected children and adolescents and in those with prodromal symptoms (or identified as prodromes) are needed to investigate further the emerging clinical, behavioral, and physiological processes at play during development. Studies employing clinical interviews are also needed to validate the newly created measure, the SPQ-C, as it has now shown promise in its assessment of the widely agreed upon subcomponents to schizotypal personality or schizotypy.

Acknowledgments

This study was funded by NIMH (R01 Laura A. Baker). Catherine Tuvblad was supported by post-doctoral stipends from the Swedish Council for Working Life and Social Research (Project 2006-1501) and the Sweden-America Foundation. Adrian Raine was supported by NIMH (Independent Scientist Award K02 MH01114-08). We thank

the Southern California Twin Project staff for their assistance in collecting data, and the twins and their families for their participation. We also appreciate the help of Erin Skrok in developing child-appropriate wording for the SPQ-C items.

References

- Akaike H. Factor analysis and AIC. *Psychometrika*. 1987; 52:42–48.
- American Psychiatric Association. Diagnostic and statistical manual of mental disorders. 4. Author; Washington, DC: 1994.
- Axelrod SR, Grilo MC, Sanislow C, McGlashan TH. Schizotypal Personality Questionnaire—Brief: factor structure and convergent validity in inpatient adolescent. *J Personal Disord*. 2001; 15:168–179.
- Baker LA, Barton M, Lozano DI, Raine A, Fowler JH. The Southern California Twin Register at the University of Southern California: II. Twin Res Human Genetics. 2006; 9(6):933–940. [PubMed: 17254433]
- Baker LA, Jacobson KC, Raine A, Lozano DI, Bezdjian S. Genetic and environmental bases of childhood antisocial behavior: a multi-informant twin study. *J Abnorm Psychol*. 2007; 116(2):219–235. [PubMed: 17516756]
- Bedwell JS, Donnelly RS. Schizotypal personality disorder or prodromal symptoms of schizophrenia? *Schizophr Res*. 2005; 80:263–269. [PubMed: 16122908]
- Chang C-J, Chen WJ, Liu S-K, Cheng JJ, Ou Yang W-C, Chang H-J. Morbidity risk of psychiatric disorders among the first degree relatives of schizophrenia patients in Taiwan. *Schizophr Bull*. 2002; 28:379–392. [PubMed: 12645671]
- Chapman LJ, Chapman JP, Raulin ML. Body-image aberration in schizophrenia. *J Abnorm Psychol*. 1978; 87:399–407. [PubMed: 681612]
- Chapman LJ, Chapman JP, Kwapil TR, Eckblad M, Zinser MC. Putatively psychosis-prone subjects 10 years later. *J Abnorm Psychol*. 1994; 103:171–183. [PubMed: 8040487]
- Chen WJ, Hsiao CK, Lin CCH. Schizotypy in community samples: the three-factor structure and correlation with sustained attention. *J Abnorm Psychol*. 1997; 106:649–654. [PubMed: 9358696]
- Chen WJ, Liu SK, Chang CJ, Lien YJ, Chang YH, Hwu HG. Sustained attention deficit and schizotypal personality features in nonpsychotic relatives of schizophrenic patients. *American Journal of Psychiatry*. 1998; 155:1214–1220. [PubMed: 9734545]
- Claridge GS. 'The schizoprenias as nervous types' revisited. *Br J Psychiatry*. 1987; 151:735–743. [PubMed: 3332195]
- Claridge, G. Theoretical background and issues. In: Claridge, G., editor. *Schizotypy. Implications for illness and health*. Oxford University Press; New York: 1997.
- Claridge GS, Brooks P. Schizotypy and hemisphere function-I. Theoretical consideration and the measurement of schizotypy. *Personal Individ Differ*. 1984; 5:633–648.
- Claridge G, Hewitt J. A biometrical study of schizotypy in a normal population. *Personal Individ Differ*. 1987; 8:303–312.
- Claridge G, McCreery C, Mason O, Bentall R, Boyle G, Slade P, Popplewell D. The factor structure of 'schizotypal' traits: a large replication study. *Br J Clin Psychol*. 1996; 35:103–115. [PubMed: 8673026]
- Ericson M, Tuvblad C, Lozano DI, Raine A, Baker LA. Genetic and environmental overlap among schizophrenia spectrum endophenotypes and schizotypal traits during childhood and adolescence (conference presentation). *Behav Genet*. 2008; 38:625.
- Erlenmeyer-Kimling L. Early neurobehavioral deficits as phenotypic indicators of the schizophrenia genotype and predictors of later psychosis. *American Journal of Medical Genetics*. 2001; 105:23–34. [PubMed: 11424986]
- Gottesman II, Gould TD. The endophenotype concept in psychiatry: etymology and strategic intentions. *Arch Gen Psychiatry*. 2003; 160(4):636–645.
- Gruzeliier J, Burgess A, Stygal J, Irving G, Raine A. Patterns of cognitive asymmetry and syndromes of schizotypal personality. *Psychiatry Research*. 1995; 56:71–79. [PubMed: 7792344]

- Gruzelier, Kaiser J. Syndromes of schizotypy and timing of puberty. *Schizophr Res.* 1996; 21(3):183–194. [PubMed: 8885046]
- Hall MH, Rijdsdijk F, Kalidindi S, Schulze K, Kravariti E, Kane F, Sham P, Bramon E, Murray RM. Genetic overlap between bipolar illness and event-related potentials. *Psychological Medicine.* 2007; 37:667–678. [PubMed: 17224092]
- Kendler KS. Diagnostic approaches to schizotypal personality disorder: a historical perspective. *Schizophrenia Bull.* 1985; 11(4):538–555.
- Kendler KS, Gruenberg AM, Strauss JS. An independent analysis of the Copenhagen sample of the Danish adoption study of schizophrenia, II: the relationship between schizotypal personality disorder and schizophrenia. *Arch Gen Psychiatry.* 1981; 38(9):982–998. [PubMed: 7283669]
- Kendler KS, McGuire M, Gruenberg AM, O'Hare A, Spellman M, Walsh D. The Roscommon Family Study. I Methods, diagnosis of probands, and risk of schizophrenia in relatives. *Archives of General Psychiatry.* 1993; 50:527–540. [PubMed: 8317947]
- Kendler K, Hewitt J. The structure of self-report schizotypy in twins. *J Personal Disord.* 1992; 6:1–17.
- Kendler KS, Ochs A, Gorman A, Hewitt J, Ross D, Mirsky A. The structure of schizotypy: a multi-trait twin study. *Psychiatry Res.* 1991; 36:19–36. [PubMed: 2017520]
- Laurens KR, Hodgins S, Maughan B, Murray RM, Rutter ML, Taylor EA. Community screening for psychotic-like experiences and other putative antecedents of schizophrenia in children aged 9–12 years. *Schizophr Res.* 2007; 90:130–146. [PubMed: 17207968]
- Lenzenweger MF. Schizotaxia, schizotypy, and schizophrenia: Paul E. Meehl's blueprint for the experimental psychopathology and genetics of schizophrenia. *J Abnorm Psychol.* 2006; 115(2):195–200. [PubMed: 16737381]
- Lin CC, Su CH, Kuo PH, Hsiao CK, Soong WT, Chen WJ. Genetic and environmental influences on schizotypy among adolescents in Taiwan: a multivariate twin/sibling analysis. *Behav Genet.* 2007; 37(2):334–344. [PubMed: 16967335]
- Linney YM, Murray RM, Peters ER, MacDonald AM, Rijdsdijk F, Sham PC. A quantitative genetic analysis of schizotypal personality traits. *Psychol Med.* 2003; 33(5):803–816. [PubMed: 12877395]
- Lykken. The diagnosis of zygosity in twins. *Behav Genet.* 1978; 8:437–473. [PubMed: 570035]
- MacDonald A III, Pogue-Geile MF, Debski TT, Manuck S. Genetic and environmental influences on schizotypy: a community-based twin study. *Schizophr Bull.* 2001; 27:47–58. [PubMed: 11215549]
- Mason O, Claridge G, Jackson M. New scales for the assessment of schizotypy. *Personal Individ Differ.* 1995; 18:7–13.
- Mason O, Linney Y, Claridge G. Short scales for measuring schizotypy. *Schizophr Res.* 2005; 78:293–296. [PubMed: 16054803]
- Mata I, Gilvarry CM, Jones PB, Lewis SW, Murray RM, Sham PC. Schizotypal personality traits in nonpsychotic relatives are associated with positive symptoms in psychotic probands. *Schizophr Bull.* 2003; 29:273–283. [PubMed: 14552503]
- Mata I, Mataix-Cols D, Peralta V. Schizotypal Personality Questionnaire-Brief: factor structure and influence of sex and age in a nonclinical population. *Personal Individ Diff.* 2005; 38:1183–1192.
- Mata I, Sham PC, Gilvarry CM, Jones PB, Lewis SW, Murray RM. Childhood schizotypy and positive symptoms in schizophrenic patients predict schizotypy in relatives. *Schizophrenia Research.* 2000; 44:129–136. [PubMed: 10913744]
- Meehl P. Schizotaxia revisited. *Arch Gen Psychiatry.* 1989; 46:935–944. [PubMed: 2552952]
- Miller, MB. Unpublished Ph.D. dissertation. University of Wisconsin; Madison: 1993. A Study of Schizotypal Traits in Young Male Twins.
- Miller, MB. A study of schizotypal traits in young male twins. *Dissertation Abstracts International: Section B: The Sciences and Engineering.* Vol. 54. 1994. p. 5397-B
- Muthen, LK.; Muthen, BO. *Mplus User's Guide.* Muthen & Muthen; Los Angeles, CA: 1998–2007.
- Neale, MC.; Cardon, LR. *Methodology for genetic studies of twins and families.* Kluwer Academic Publications; Dordrecht, The Netherlands: 1992.
- Neale, MC.; Boker, SM.; Xie, G.; Maes, H. *Mx: Statistical modeling.* Department of Psychiatry, Medical College of Virginia; Richmond, VA: 2003.

- Peters ER, Day S, Garety PA. The Peters et al. Delusions Inventory (PDI): new norms for the 21-item version. *Schizophr Res.* 1996; 18:118.
- Poulton R, Caspi A, Moffitt TE, Cannon M, Murray R, Harrington H. Children's self-reported psychotic symptoms and adult schizophreniform disorder: a 15-year longitudinal study. *Arch Gen Psychiatry.* 2000; 57:1053–1058. [PubMed: 11074871]
- Raine A. The SPQ: a scale for the assessment of schizotypal personality based on DSM-III-R criteria. *Schizophr Bull.* 1991; 17(4):555–564. [PubMed: 1805349]
- Raine A. Sex differences in schizotypal personality in a non clinical population. *J Abnorm Psychol.* 1992; 101:361–364. [PubMed: 1583234]
- Raine, A.; Baker, LA. Unpublished questionnaire. Department of Psychology, University of Southern California; 2003. The SPQ-C: a brief child version of the Schizotypal Personality Questionnaire.
- Raine A, Benishay D. The SPQ-B: a brief screening instrument for schizotypal personality disorder. *J Personal Disord.* 1995; 9:346–355.
- Raine A, Reynolds C, Lencz T, Scerbo A, Triphon N, Kim D. Cognitive-perceptual, interpersonal, and disorganized features of schizotypal personality. *Schizophr Bull.* 1994; 20(1):191–201. [PubMed: 8197415]
- Reynolds CA, Raine A, Mellingen K, Venables PH, Mednick SA. Three-factor model of schizotypal personality: invariance across culture, gender, religious affiliation, family adversity, and psychopathology. *Schizophrenia Bull.* 2000:603–618.
- Rossi A, Daneluzzo E. Schizotypal dimensions in normals and schizophrenic patients: A comparison with other clinical samples. *Schizophrenia Research.* 2002; 54:67–75. [PubMed: 11853980]
- SPSS Inc. SPSS Base 8.0 for Windows User's Guide. SPSS Inc; Chicago: 1998.
- Turetsky BI, Calkins ME, Light GA, Olincy A, Radant AD, Swerdlow NR. Neurophysiological endophenotypes of schizophrenia: the viability of selected candidate measures. *Schizophr Bull.* 2007; 33(1):69–94. [PubMed: 17135482]
- Van den Oord EJ, Simonoff E, Eaves L, Pickles A, Silberg J, Maes H. An evaluation of different approaches for behavior genetic analyses with psychiatric symptom scores. *Behav Genet.* 2000; 30:1–18. [PubMed: 10934795]
- Vollema MG, Hoijtink H. The multidimensionality of self-report schizotypy in a psychiatric population: An analysis using multidimensional Rasch models. *Schizophrenia Bulletin.* 2000; 26(3):565–575. [PubMed: 10993398]
- Vollema MG, Sitskoorn MM, Appels MCM, Kahn RS. Does the Schizotypal Personality Questionnaire reflect the biological-genetic vulnerability to schizophrenia. *Schizophrenia Research.* 2002; 54:39–45. [PubMed: 11853977]
- Vollema MG, Van den Bosch RJ. The multidimensionality of schizotypy. *Schizophr Bull.* 1995; 21:19–32. [PubMed: 7770738]
- Walker EF, Bollini A. Pubertal neurodevelopmental and the emergence of psychotic symptoms. *Schizophr Res.* 2002; 54:17–23. [PubMed: 11853974]
- Walker EF, Neumann CC, Baum K, Davis DM. The developmental pathways to schizophrenia: potential moderating effects of stress. *Dev Psychopathol.* 1996; 8:647–665.
- Walker EF. Adolescent changes in stress sensitivity and the expression of vulnerability to psychopathology. *Development and Psychopathology.* 2001; 13:721–732. [PubMed: 11523856]

Appendix

See Table 6.

Fig. 1.

Two-Factor Common Pathway Model of SPQ-C Subscales (Model 4c) during Waves 2 and 3. *Note.* A = variance due to additive genetic influences; E = variance due to non-shared environmental influences; a = variance due to additive genetic influences unique to each subscale; e = variance due to non-shared environmental influences unique to each subscale; Paths indicate parameter estimates (and 95% confidence intervals)

Table 1

Loadings from exploratory factor analyses for three extracted factors using promax rotation: Wave 2 (age 11–13 years) and Wave 3 (age 14–16 years)

	Wave 2			Wave 3		
	Factor 1: Cognitive	Factor 2: Disorganization	Factor 3: Interpersonal	Factor 1: Cognitive	Factor 2: Disorganization	Factor 3: Interpersonal
I often get the feeling that other people are watching me when I am out playing or shopping	0.59	-0.01	-0.23	0.72	-0.03	-0.04
I often have to stop people from taking advantage of me	0.55	-0.04	0.14	0.38	-0.07	0.30
I sometimes think that people can read my mind	0.52	0.10	0.10	0.52	0.03	0.06
I sometimes feel there is a person or spirit around me, even when there is no one there	0.52	-0.11	-0.21	0.70	-0.05	-0.04
I sometimes feel distracted by far-off sounds that I'm not normally aware of	0.51	0.24	-0.11	0.60	0.09	-0.01
I sometimes think that common events or objects have a special message for me	0.43	0.24	-0.24	0.74	-0.06	-0.11
I often pick up on hidden threats or put-downs from what people say or do	0.43	0.07	0.20	0.48	0.17	0.14
I have had experiences like seeing flying saucers, or known something will happen before it does	0.42	0.30	0.14	0.45	0.15	-0.07
I am an odd, unusual person	0.23	0.79	-0.03	-0.08	0.87	-0.03
I am a strange person	-0.02	0.79	-0.23	-0.11	0.88	-0.05
I sometimes use words in unusual ways	0.25	0.68	-0.23	0.25	0.46	-0.04
I sometimes ramble on when I am talking	0.14	0.68	-0.18	0.18	0.38	-0.03
I sometimes act oddly	0.21	0.53	-0.18	0.06	0.66	-0.05
I find it hard getting people to understand what I am saying	0.18	0.44	0.13	0.28	0.36	0.19
I find it best not to let other people know too much about me	0.13	-0.01	0.65	0.15	-0.09	0.58
I feel very uneasy talking to people I do not know well	-0.03	-0.21	0.65	-0.02	-0.13	0.71
I tend to keep my feelings to myself	-0.04	-0.03	0.58	-0.01	-0.05	0.64

	Wave 2			Wave 3		
	Factor 1: Cognitive	Factor 2: Disorganization	Factor 3: Interpersonal	Factor 1: Cognitive	Factor 2: Disorganization	Factor 3: Interpersonal
I often keep in the background on social situations	-0.15	0.03	0.55	-0.11	0.01	0.66
I feel I have to be on my guard even with friends	0.30	0.13	0.50	0.15	0.27	0.43
I am a bit unfriendly and distant	-0.13	0.09	0.48	-0.02	0.12	0.44
I feel very uncomfortable in social situations involving people I don't know well	0.10	-0.26	0.45	0.00	-0.14	0.71
I find it hard to make close friends	-0.14	-0.23	0.43	-0.25	0.25	0.56

Table 2

Model fit indices for confirmatory factor analyses—Waves 2 and 3

	Wave 2		Wave 3	
	1 Factor (22 items)	3 Factor (22 items)	1 Factor (22 items)	3 Factors (22 items)
χ^2	220.672	172.080	795.783	551.486
Free parameters	44	47	44	47
p	<.001	<.001	<.001	<.001
RMSEA	.060	.048	.098	.050
CFI	0.912	0.946	0.756	0.885

Table 3

Means, standard deviations (SD) and number of individual participants (N) for SPQ-C total score and three sub-factors at Wave 2 (age 11–13 years) and Wave 3 (age 14–16 years), separately by Zygosity (MZ, DZ)

	Wave 2		Wave 3	
	MZ Mean (SD)	DZ Mean (SD)	MZ Mean (SD)	DZ Mean (SD)
SPQ-C Total	0.31 (0.24)	0.28 (0.22)	0.32 (0.22)	0.34 (0.24)
Cognitive-Perceptual	0.27 (0.26)	0.26 (0.25)	0.27 (0.26)	0.29 (0.27)
Disorganization	0.29 (0.31)	0.25 (0.29)	0.34 (0.32)	0.38 (0.34)
Interpersonal-Affective	0.35 (0.28)	0.32 (0.25)	0.35 (0.29)	0.34 (0.29)
	<i>N</i> = 182	<i>N</i> = 173	<i>N</i> = 377	<i>N</i> = 534

Table 4
Intraclass and cross-twin cross-trait correlations between SPQ-C subscales, by zygosity during Wave 2 and Wave 3

	MZ			DZ		
	Cognitive-Perceptual	Disorganization	Interpersonal-Affective	Cognitive-Perceptual	Disorganization	Interpersonal-Affective
Wave 2 (age 11–13)						
Cognitive-Perceptual	.58*			.33*		
Disorganization	.37*	.43*		.21	.22	
Interpersonal-Affective	.37*	.23*	.48*	.12	.22	.30*
Wave 3 (age 14–16)						
Cognitive-Perceptual	.49*			.23*		
Disorganization	.49*	.55*		.26	.25*	
Interpersonal-Affective	.31*	.32*	.46*	.26	.11	.03

Note. MZ monozygotic, DZ dizygotic; intraclass correlations on each diagonal in bold; cross-twin cross-trait correlations are below the diagonal;

* $p < .05$

Table 5

Multivariate genetic model fitting for Waves 2 and 3 longitudinally

	Overall fit			
	-2LL (df)	AIC	χ^2 (Δdf)	<i>p</i>
1. Saturated constrained	8789.83 (3632)	1525.83	–	
2. Cholesky	8966.45 (3729)	1508.45	176.62 (97)	<0.01
3. 1-Factor common pathway	9118.09 (3766)	1586.09	151.64 (37)	<0.01
4a. 2-Factor common pathway-constrained factor loadings	9018.49 (3764)	1490.49	52.04 (35)	0.03
4b. 2-Factor common pathway-unconstrained factor loadings	9008.17 (3761)	1486.17	41.72 (32)	0.12
4c. Reduced 2-factor common pathway-unconstrained factor loadings (i.e. drop common C and unique C)	9011.15 (3770)	1471.15	44.70 (41)	0.31

Note. -2LL = -2(log-likelihood), AIC Akaike's Information Criterion, χ^2 difference in log-likelihoods between sub-models, Δdf change in degrees of freedom, A variance due to additive genetic influences, C variance due to shared environmental influences, E variance due to non-shared environmental influences, Best-fitting model and fit values shown in bold

Table 6

Univariate model fit indices and parameter estimates (with 95% confidence intervals) for SPQ-C subscales

	-2LL (df)	AIC	χ^2 (df)	p	A	C	E
Wave 2							
Cognitive-Perceptual							
Saturated	896.71 (347)	–	–				
ACE	897.60 (350)	197.60	0.89 (3)	0.89			
AE	898.43 (351)	196.43	1.72 (4)	0.79	0.53 (0.38, 0.65)	–	0.47 (0.35, 0.61)
CE	899.69 (351)	197.69	2.98 (4)	0.15			
E	936.81 (352)	232.81	40.1 (5)	<0.01			
Disorganization							
Saturated	880.40 (347)	–	–				
ACE	881.28 (350)	181.28	0.87 (3)	0.83			
AE	881.36 (351)	179.36	0.96 (4)	0.91	0.42 (0.25, 0.55)	–	0.58 (0.45, 0.74)
CE	883.17 (351)	181.17	2.77 (4)	0.59			
E	903.84 (352)	199.84	23.44 (5)	<0.01			
Interpersonal-Affective							
Saturated	919.33 (347)	–	–				
ACE	923.95 (350)	223.95	4.62 (3)	0.20			
AE	925.20 (351)	223.20	5.87 (4)	0.21	0.46 (0.31, 0.58)	–	0.53 (0.42, 0.68)
CE	927.53 (351)	225.53	8.2 (4)	0.09			
E	955.31 (352)	251.31	35.98 (5)	<0.01			
Wave 3							
Cognitive-Perceptual							
Saturated	2375.68 (904)	–	–				
ACE	2377.61 (907)	563.61	1.93 (3)	0.59			
AE	2377.61 (908)	561.61	1.93 (4)	0.75	0.53 (0.42, 0.62)	–	0.47 (0.38, 0.58)
CE	2394.59 (908)	578.59	18.91 (4)	<0.01			
E	2447.77 (909)	629.77	72.09 (5)	<0.01			
Disorganization							
Saturated	2340.85 (904)	–	–				
ACE	2343.45 (907)	529.45	2.60 (3)	0.45			

	-2LL (df)	AIC	χ^2 (df)	p	A	C	E
AE	2343.45 (908)	527.45	2.60 (4)	0.62	0.57 (0.47, 0.65)	-	0.43 (0.35, 0.53)
CE	2363.94 (908)	547.94	23.09 (4)	<0.01			
E	2427.73 (909)	609.73	86.88 (5)	<0.01			
Interpersonal-Affective							
Saturated	2427.30 (904)	-	-				
ACE	2438.63 (907)	624.63	11.33 (3)	0.10			
AE	2435.63 (908)	622.63	8.33 (4)	0.08	0.38 (0.26, 0.49)	-	0.62 (0.51, 0.74)
CE	2452.56 (908)	636.56	25.26 (4)	<0.01			
E	2471.61 (909)	653.61	44.31 (5)	<0.01			

Note -2LL = -2(log-likelihood), AIC = Akaike's Information Criterion, BIC = Bayesian Information Criterion, df = degrees of freedom, A = additive genetic effects, C = shared environmental effects, E = nonshared environmental effects