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The developmental interplay between the p-factor of psychopathology and the g-factor of intelligence from age 7 through 16 years

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Abstract

Intelligence and mental health are the core pillars of individual adaptation, growth, and opportunity. Here, we charted across childhood and adolescence the developmental interplay between the p-factor of psychopathology, which captures the experience of symptoms across the spectrum of psychiatric disorders, and the g-factor of general intelligence that describes the ability to think, reason, and learn.

Our preregistered analyses included 7,433 twin pairs from the Twins Early Development Study (TEDS), who were born 1994 to 1996 in England and Wales. At the ages 7, 9, 12, and 16 years, the twins completed two to four intelligence tests, and multi-informant measures (i.e., self-, parent- and teacher-rated) of psychopathology were collected.

Independent of their cross-sectional correlations, p- and g-factors were linked by consistent, bidirectional, and negative cross-lagged paths across childhood and adolescence (from −.07 to −.13 with 95% CIs from −.03 to −.15). The cross-lagged paths from intelligence to psychopathology were largely due to genetic influences, but the paths from psychopathology to intelligence were driven by environmental factors, and increasingly so with age.

Our findings suggest that intelligence and psychopathology are developmentally intertwined due to fluctuating etiological processes. Understanding the interplay of g- and p-factors is key for improving children's developmental outcomes.

Keywords: adolescence; childhood; cross-lagged twin model; intelligence; p-factor

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Introduction

Intelligence – the ability to think, reason, and learn – has been frequently implicated in developmental psychopathology (e.g., Astle & Fletcher-Watson, 2020; Pennington & Ozonoff, 1996). Yet, little is known about the developmental interplay between intelligence and psychopathology and its etiology over the course of childhood and adolescence. This is particularly true when we look for studies that treated both – intelligence and psychopathology – as continuous dimensions that describe quantitative differences between people, by contrast to discrete qualitative categories or disorders (Egger & Angold, 2006). Although intelligence has long been recognized to vary between people by degree rather than by type, the same logic has only been applied to developmental psychopathology in recent decades (Caspi et al., 2014; Lahey et al., 2012; Martel et al., 2017; Peters & Ansari, 2019).

Historically, developmental psychopathology differentiated typical and atypical populations, normal and abnormal development, and types of mental disorders (Cicchetti, 1989). A number of studies have challenged this approach: developmental psychopathologies are now thought to be both transdiagnostic and dimensional, rather than distinct categories of disorders (Achenbach & Edelbrock, 1978; Plomin et al., 2016; Rhee et al., 2014). The notion that psychopathologies are transdiagnostic stemmed initially from the observation that comorbidity – the co-occurrence of two or more disorders in the same individual – is the norm rather than the exception in developmental psychopathology (Egger & Angold, 2006; Rhee et al., 2014). Indeed, up to 50% of individuals diagnosed with a mental illness are going to develop two or more comorbidities within the next year (Kessler et al., 2005). Genetically sensitive studies have shown that this high comorbidity results, at least partly, from common genetic causes that explain the co-occurrence and correlation of mental disorders (Cross-Disorder Group of the Psychiatric Genomics Consortium, 2013; Lahey et al., 2011; Plomin et al., 2016; Zhao & Nyholt, 2017).

Dimensional models propose that mental disorders lie on the continuum between pathology and normality (Egger & Angold, 2006), with developmental psychopathology spanning the full range of variation from subclinical to clinical. Several higher-order dimensions have been differentiated, including externalizing (e.g., aggressive, delinquent, conduct behaviors), internalizing

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(e.g., anxious, depressed, withdrawn behaviors), attention problems, and thought disorder (e.g., Caspi et al., 2014; Harden et al., 2020; Martel et al., 2017). Across studies, correlations between these higher-order dimensions of developmental psychology hover around 0.5 (Wright et al., 2013), giving rise to a common variance factor that has been named "p-factor" and that can be observed at both phenotypic and genetic levels across childhood and adolescence (e.g., Allegrini et al., 2020; Caspi & Moffitt, 2018; Grotzinger et al., 2019; Lahey et al., 2011; Martel et al., 2017; Michelini et al., 2019; Schaefer et al., 2018; Selzam et al., 2018).

By contrast to developmental psychopathology, cognitive abilities, such as fluid reasoning, visual and auditory perception, and processing speed, have been known for well over a century to converge on one higher-order factor that captures their common variance (Carroll, 1993; Spearman, 1904). This factor, known as the general factor of intelligence or g-factor (Deary, 2012), encompasses other higher level factors of cognitive ability, including executive function (i.e., the effortful mental processes to control and direct attention) and working memory (i.e., working mentally with information that is no longer perceptually present; Diamond, 2013). For example, in 811 Texan children aged 7–15 years, a common latent factor from a battery of 16 executive function measures, including three tests for working memory, correlated .91 with a general factor of intelligence, extracted from four tests, including block design, matrix reasoning, vocabulary, and similarities (Engelhardt et al., 2016). The same study also found that individual differences in the latent factors of executive function and general intelligence have a common genetic origin (Engelhardt et al., 2016). These and other findings (e.g., Colom et al., 2004) suggest that the g-factor, executive function, and working memory describe the same cognitive dimension of individual differences.

A handful of studies previously explored contemporaneous linear associations between p- and g-factors in children and adolescents (cf. Caspi et al., 2020). For example, in 2,395 children from Brazil aged 6–12 years, executive function was significantly associated with p (beta = −.24; Martel et al., 2017), and in a sample of 415 children from the United States aged 8–12 years, p and a working memory factor correlated −.25 (Huang-Pollock et al., 2017). A third study found that a p-factor correlated −.21 with a g-factor in 1,300 US children, and this association was mainly due to shared environmental igenetic influences (45% and 40%, respectively; Grotzinger et al., 2019). Together, these existing studies suggest that p and g are negatively associated in childhood and adolescence, although they cannot elucidate causes and the nature of both domains' interplay over the course of development (see Papachristou & Flouri, 2020, for longitudinal associations between children's cognitive ability and externalizing and internalizing behavior problems).

A plausible theoretical explanation for the reported negative associations between p- and g-factors is that general intelligence protects against experiencing psychopathology symptoms (i.e., unidirectional negative effect), for example when children's intelligence helps them overcome anxieties that are typical in education settings (Schillinger et al., 2018). The reverse direction of influence is also possible: psychopathology may impair intelligence when it hinders children to maximize their learning opportunities. For example, attending school been shown to increase intelligence (Ritchie & Tucker-Drob, 2018) but children with poor mental health experience more often exclusions from educational settings (Ford et al., 2018) and thus, are likely to be disadvantaged in the development of intelligence.

Finally, g- and p-factors may have bidirectional influences on each other over the course of development, whereby higher g at an earlier age predicts experiencing fewer psychopathology symptoms at a later age, and higher p at an earlier age forecasts lower intelligence at a later one. Identifying the direction of associations between p- and g-factors is key for designing and implementing effective interventions that improve children's life chances, yet no previous study has tested bidirectional associations between the p- and g-factors over time. For example, if p predicts long-term changes in g but not vice versa, interventions that target p are likely to also alter g, whereas g-focused interventions would be unlikely to transfer benefits to p. Understanding if p causes changes in g or vice versa for genetic or environmental reasons is crucial for intervening effectively. If p and g influenced each other primarily due to genetics, interventions that focused on environmental changes would be unlikely to disrupt the p-g association. By contrast, if environmental factors linked p and g, interventions that altered children's surroundings could be expected to reduce influences of p on g and vice versa. Another relevant issue is if associations between p- and g-factors and their etiology vary across age or developmental periods; for example, it is possible that one factor predicts the other due to shared genetic influences in childhood but later in adolescence, the p-g-factor association might be driven by environmental factors.

The aims of current study were twofold. First, we sought to establish whether g- and p-factors were inter-related across childhood and adolescence because of unidirectional effects from one to the other (e.g., p → g or g → p) or because of bidirectional influences (i.e., p ↔ g). Second, we explored the extent to which the developmental interplay between g- and p-factors could be attributed to genetic, shared environmental, and nonshared environmental factors. To achieve these aims, we fitted a biometric genetic version of the autoregressive cross-lagged twin model (Malanchini et al., 2017) to longitudinal, multi-informant data from a large cohort study that was representative of the UK population. Our model tested, for example, if cross-lagged predictions from p at an earlier assessment age to g at a later assessment age remained significant after accounting for (a) shared genetic and environmental influences that are common to both constructs and (b) those influences – including nonshared environmental influences, for example if one twin experienced school exclusion but the other did not – that are shared with g at the earlier assessment age (Malanchini et al., 2017; McAdams et al., 2020). At the same time, the reverse direction of causality was also tested (i.e., g → p). The biometric genetic model differs from other autoregressive cross-lagged twin models, in that all variables and paths, including the cross-lagged ones, are decomposed into additive genetic (A), shared (common) environmental (C), and nonshared environmental (E) factors (i.e., ACE factors; McAdams et al., 2020). Our analyses quantified genetic and environmental influences on (a) the cross-lagged paths from earlier p to later g and vice versa, independent of the genetic and environmental influences on p and g at each assessment age, (b) the traits' stability over time, and (c) the cross-sectional correlations between traits within each assessment age.

Method

Sample

The Twins Early Development Study (TEDS) recruited families of twins born in England and Wales from 1994 through 1996 (Haworth et al., 2013). Twins who suffered from severe medical

problems currently or at birth (e.g., postnatal surgery); whose mothers reported severe medical problems during pregnancy; and whose first language was not English were excluded. The analysis sample included 14,866 children (from 7,433 families) for whom at least one measure of cognitive ability was available at age 7. Of those, 6,630 twins (3,315 families) also contributed data at age 9; 12,056 twins (6,028 families) at age 12; and 9,950 twins (4,975 families) at age 16. The sample size at age 9 was reduced because, due to funding restrictions, only a subsample of TEDS (approximately half) was invited to participate in the assessment wave. At each assessment age, parent-, teacher-, and self-ratings of psychopathology were available for >98% of the children who had completed least one measure of cognitive ability (cf. Table S6).

TEDS was at its inception representative of the UK population in comparison with census data and remains so, despite some attrition (Rimfeld et al., 2019). The socioeconomic status, derived from fathers' and mothers' occupation, education, and mothers' age at first birth, of the analysis sample (mean = 0.08, $SD = 0.99$) was minimally higher than that of the TEDS sample at inception (mean = 0, $SD = 1$; Table S1; von Stumm et al., 2020; see Supplementary Materials for details of the socioeconomic status measure). Parents provided written informed consent prior to data collection. TEDS project approval (05.Q0706/228) was granted by the ethics committee for the Institute of Psychiatry, Psychology and Neuroscience at King's College London.

Measures

Data were collected at ages 7, 9, 12, and 16 years. At each assessment age, children, parents, and teachers each provided ratings on 5–8 measures of psychopathology (i.e., multi-informant psychopathology assessment). The measures are listed below and described in detail in the Supplementary Materials. We followed Allegrini et al.'s (2020) modeling approach for extracting the p-factors (detailed below), creating psychopathology composite factor scores across raters and reporting the corresponding results in the main manuscript. We also report the results in full separately for each group of raters in the Supplementary Materials.

The twins also completed two to four intelligence tests at each assessment age, using phone-, booklet-, and web-based testing. The psychometric properties of the respective g-factors, including their high reliability and stability across ages and assessment modalities, have been previously reported (e.g., Rimfeld et al., 2019; von Stumm & Plomin, 2015).

Psychopathology measures

Age 7 measures. Parents and teachers completed the five subscales of the Strengths and Difficulties Questionnaire (SDQ; hyperactivity, conduct problems, peer problems, emotional problems, and prosocial behavior (reversed); Goodman, 1997), as well as the Antisocial Process Screening Device (APSD; Frick & Hare, 2001) and a measure of autism traits that was specifically created for TEDS.

Age 9 measures. The SDQ (Goodman, 1997) and the Childhood Autism Spectrum Test (Scott et al., 2002; Williams et al., 2005) were self-, parent- and teacher-reported. Parents and teachers also rated APSD (Frick & Hare, 2001) and aggression (a mean of proactive and reactive scales; Dodge & Coie, 1987).

Age 12 measures. The SDQ (Goodman, 1997), APSD (Frick & Hare, 2001), and Childhood Autism Spectrum Test (Scott et al., 2002; Williams et al., 2005) were self-, parent- and teacher-reported. Parents also completed the Moods and Feelings Questionnaire (Angold et al., 1995) and Conners' Parent Rating Scale that assesses ADHD symptoms (Conners et al., 2003).

Age 16 measures. The SDQ (Goodman, 1997) and the Moods and Feelings Questionnaire (Angold et al., 1995) were self-, parent- and teacher-reported, and The Autism Quotient was parent- and self-reported (Baron-Cohen et al., 2001). Parents also completed the Conners' Parent Rating Scale (Conners et al., 2003), the Inventory of Callous Unemotional Traits (Kimonis et al., 2008) and the Anxiety-Related Behaviors Questionnaire (Eley et al., 2003).

Intelligence measures

Age 7 measures. Children were tested on verbal and nonverbal abilities by telephone (Petrill et al., 2002), using a testing booklet mailed to the parents with two verbal tests (Similarities and Vocabulary from the Wechsler Intelligence Scale for Children (WISC-III-UK); Wechsler, 1992), two nonverbal tests (Picture Completion from the WISC-III-UK and Conceptual Grouping from the McCarthy Scales of Children's Abilities; McCarthy, 1972).

Age 9 measures. Participants were mailed a test booklet with two verbal tests, including vocabulary and general knowledge, adapted from the multiple-choice version of the WISC-III-UK (Wechsler, 1992) and two nonverbal tests, including Puzzles and Shapes, adapted from tests of the Cognitive Abilities Test 3 (Davis et al., 2008; Smith et al., 2001).

Age 12 measures. Testing was web-based and conducted at home via computers, using age-matched versions of the tests previously used at age 9 (i.e., two verbal and two nonverbal tests; Kaplan et al., 1998; Raven et al., 1996; Wechsler, 1992).

Age 16 measures. Twins completed web-based adaptations of Raven's Standard and Advanced Progressive and the Mill-Hill Vocabulary Scale using their home computers (Raven et al., 1998; Raven et al., 1996).

Statistical analysis

We conducted the analyses in R (R Core Team, 2014) with the following packages: TwinAnalysis (https://github.com/IvanVoronin/TwinAnalysis), OpenMx (Neale et al., 2016), and mlth.data.frame (https://github.com/IvanVoronin/mlth.data.frame). We used Full Information Maximum Likelihood, under the assumption that data were missing at random.

For each scale, z-standardized residuals were derived, regressed on sex and age differences within each assessment point (i.e., age differences in weeks at each assessment wave). To account for nonindependence of observations, one twin from each pair was randomly selected for the phenotypic analyses. At each age, we modeled a g-factor from the observed cognitive scores (i.e., four tests at ages 7, 9, and 12 years, and two tests at age 16), and p-factors from the psychopathology scales scores (i.e., parent-rated: seven scales at ages 7 and 16 years, eight scales at ages 9 and 12 years; child-rated: six scales at age 9, five scales at age 12, six scales at age 16; teacher-rated: seven scales at age 7, 8 scales at age 9, six scales at age 12). Our approach to modeling the p-factors followed prior analyses of these data by Allegrini et al. (2020), who acknowledged that some of the measures (e.g., peer problems, prosocial behavior (reversed), autism traits) were not previously

used in other factor models of general psychopathology. Akin to Allegrini et al. (2020), we adopted here a hypothesis-free approach to modeling single factors per assessment age that capture a general psychopathology trait across diverse domains. We included all available psychopathology measures in TEDS at each age, even though some were only administered once (e.g., aggression). Factor loadings for p- and g-factors at each age per rater and across raters (i.e., combined p-factor scores) are reported in the Supplementary Tables S2–S5. We retained factor scores from each factor analysis and used them in our cross-lagged twin models (details below). We report the cross-lagged twin model results with p-factor scores combined across raters in the main manuscript below and those with rater-specific p-factor scores in the Supplementary Materials and Tables.

A cross-lagged model specifies (a) paths that connect different measures across time-points (i.e., cross-lagged effects), and (b) paths that connect the same measure across time-points (i.e., stability). The stability and cross-lagged paths take the form of partial regression coefficients, which are independent of each other and adjusted for the pre-existing association between p- and g-factors. We used bootstrapped 95% confidence intervals to interpret the strengths of the cross-lagged and stability paths.

The biometric genetic version of the autoregressive cross-lagged twin model, which was fitted here, specifies time-specific genetic and environmental components of (co)variance in a standard bivariate genetic model (Neale & Cardon, 1992; Malanchini et al., 2017). The differentiation of additive genetic effects (A), shared environmental influences (C), and nonshared environmental factors (E) for each trait is based on the fact that identical (monozygotic; MZ) and non-identical (dizygotic; DZ) twins differ in their average correlation for the genetic component (1 versus 0.5) but have the same degree of correlation for shared (1) and nonshared environmental factors (0). In the bivariate twin analysis, MZ and DZ correlations are compared across traits: that is, one twin's p-factor score is correlated with the co-twin's g-factor score. If these cross-trait cross-twin correlations are greater for MZ than for DZ twins, this implies that genetic factors contribute to the phenotypic correlation between the two traits. The ACE cross-lagged model allows examining the etiologies of the cross-lagged associations between p- and g-factors over time. Other multivariate twin methods, such as Cholesky decompositions, specify the cross-lagged paths in separate models, which prohibits direct comparisons of the cross-lagged paths' effects, both phenotypically or etiologically. The ACE cross-lagged model overcomes this limitation by estimating all the paths within the same model, therefore allowing for comparison and interpretation of the developmental associations between constructs and of their etiology. The approach has been described in detail by Malanchini et al., (2017; see also McAdams et al., 2020).

Results

Descriptive statistics and correlations

Descriptive statistics for the latent p- and g-factors at each age for each rater and across raters (i.e., composite p-factor scores) and their intercorrelations are reported in Supplementary Table S6 and Figure S1. The g-factor correlations were positive and increased over time: The association of $r = .41$ between g-factor scores at ages 7 and 9 increased to $r = .59$ between ages 12 and 16. Correlations between p-factors across ages were positive and ranged from $r = .33$ between age 7 and at 16 to strong $r = .60$ between age 9 and at 12. These correlation patterns substantiate that p- and g-factor scores extracted from different measures at different assessment ages converge reliably on comparable underlying trait dimensions, in line with previous analyses of these data (Allegrini et al., 2020; von Stumm & Plomin, 2015). Correlations between p- and g-factor scores were small and negative, ranging from −.12 to −.18. Correlation patterns varied across raters (Figure S1). For example, the correlations ranged from $r = -.09$ between parent-rated p at age 7 and g at age 16 to $r = -.31$ between parent-rated p at 12 and g at 9, from $r = -.08$ between teacher-rated p at age 7 and g at age 12 to $r = -.17$ between teacher-rated p at age 12 and g at age 9, and from $r = -.03$ between g at age 7 and self-rated p at age 16 to $r = -.22$ between g at age 9 and self-rated p at age 12.

Developmental associations between p and g: Phenotypic cross-lagged models

We examined the stability and change in cross-rater p- and g-factors and in their cross-lagged paths. Table 1 reports the standardized path estimates for the associations between g-factor scores and the p-factor scores combined across raters at ages 7, 9, 12 and 16.

Both g and p were substantially stable from age 7 through to 16 years (i.e., stability paths in Table 1). This stability increased over development, particularly for g, where the autoregressive path increased from .44 between ages 7 and 9 to .61 between ages 12 and 16. A smaller increase in developmental stability was observed for p, from .54 to .61. For both p and g, the respective confidence intervals of the stability paths did not overlap, suggesting that the increases in stability were statistically significant.

From childhood onwards, g and p were negatively associated (i.e., cross-sectional correlation paths), with the contemporaneous relationships between g and p diminishing substantially after age 12. This suggests that the negative correlations between p and g at age 12 and p and g at age 16 are almost entirely accounted for by their relationships at previous points in development.

We found that over and above their stability and contemporaneous correlations, p and g reciprocally influenced each other (i.e., significant cross-lagged paths). These influences from g to subsequent p and vice versa occurred across all assessment ages and were fairly consistent (Table 1). The cross-lagged paths were of small effect size, confirming our hypothesis, but they were all associated with 95% confidence intervals that excluded 0.

Genetic and environmental influences on the p- and g-factor developmental interplay: ACE cross-lagged model

We applied the ACE cross-lagged model to examine to what extent genetic (A), family-wide (C) and individual specific (E) environmental factors contributed to the developmental associations between p and g. While the heritability of p was substantial and remained stable from age 7 ($h^2 = .74$) to 16 ($h^2 = .75$), the heritability of g, in line with previous findings (Haworth et al., 2010), increased substantially from age 7 ($h^2 = .38$) to 16 ($h^2 = .50$; Supplementary Tables S7 and S8).

Figure 1 illustrates the results focusing on the relative importance of A, C and E for each cross-sectional and cross-lagged path, expressed as percentages. For example, Figure 1 shows that 74% of the cross-sectional correlation between p and g at 7 ($r = -.19$, Table 1) were accounted for by genetic factors, while 18% were due to shared environmental factors, and 8% to nonshared environmental factors.

The ACE cross-lagged model showed that the high stability of p over development was almost entirely due to A (85%), while C and

Table 1. Cross-lagged model results for the phenotypic association between p- (across raters) and g-factors from ages 7 through to 16 years

	Path	Standardized estimate	Lower-bound 95% CIs	Upper-bound 95% CIs
Stability paths	P 7 → P 9	0.54	0.52	0.56
	G 7 → G 9	0.44	0.41	0.47
	P 9 → P 12	0.64	0.62	0.66
	G 9 → G 12	0.58	0.55	0.61
	P 12 → P 16	0.61	0.59	0.63
	G 12 → G 16	0.61	0.57	0.64
Cross-sectional correlation paths	P 7 ⇔ G 7	−0.19	−0.22	−0.16
	P 9 ⇔ G 9	−0.17	−0.19	−0.14
	P 12 ⇔ G 12	−0.03	−0.05	−0.01
	P 16 ⇔ G 16	−0.04	−0.07	0.00
Cross-lagged paths	P 7 → G 9	−0.11	−0.15	−0.08
	G 7 → P 9	−0.12	−0.15	−0.09
	P 9 → G 12	−0.07	−0.11	−0.04
	G 9 → P 12	−0.13	−0.15	−0.10
	P 12 → G 16	−0.07	−0.11	−0.03
	G 12 → P 16	−0.10	−0.13	−0.07
Variances and residual variances	P 7	1.00	–	–
	G 7	1.00	–	–
	P 9	0.67	0.65	0.69
	G 9	0.78	0.75	0.80
	P 12	0.52	0.50	0.54
	G 12	0.63	0.59	0.66
	P 16	0.59	0.56	0.61
	G 16	0.60	0.57	0.64

Note: CIs = confidence intervals, P = p-factor of psychopathology, G = g-factor of general intelligence, → regression paths, ⇔ correlation paths.

E (7% and 8%) accounted for a relatively small portion of the developmental stability of p. The developmental stability of g, which increased over development, was also primarily due to A (67%), followed by C (31%) but not E.

Genetic factors accounted for most of the initial contemporaneous correlation between g and p (A = 74%, C = 18%, E = 8%). Yet, shared environmental factors accounted for most of the correlation between p and g at age 9 ($r = -.17$, A = 10%, C = 74%, E = 16%) and at age 12 ($r = -.03$, A = 15%, C = 72%, E = 13%). Both genetic and environmental factors influenced the cross-sectional association between p and g observed at age 16 ($r = -.04$, A = 54%, C = 23%, E = 23%), although the non-significance of the phenotypic correlation makes an interpretation of the ACE contributions difficult.

The genetic contribution to the cross-lagged paths from p to subsequent g decreased over time (46%, 39%, 18%), as did the influence of shared environmental factors (44%, 41%, 15%), while the contribution of nonshared environmental factors increased (10%, 19%, 67%). The cross-lagged links from g to subsequent p followed a less consistent developmental pattern, with genetic factors accounting for most of the developmental cross-lagged paths from ages 7 to 9 and 12 to 16 (66% and 54%, respectively), while shared environmental factors accounted for most of the link between ages 9 and 12 (87%). The contribution of nonshared environmental factors to the cross-lagged paths from g to subsequent p was small overall (average E = 11%).

Developmental associations between p and g differ by rater

The results of the cross-lagged models with parent- and teacher-reported p-factors (Tables S9 and S10) were highly consistent with the pattern of associations observed for the combined p-factor across raters (reported above). Those with self-reported p differed in the way that 3 of 4 cross-lagged paths between p and g were associated with confidence intervals that included 0 (i.e., not significant; see Table S11 and Supplementary Materials for details). The contributions of genetic and environmental effects on the stability of p were highly consistent across raters, with genetic influences accounting for most of the autoregressive paths. However, the contributions of genetic and environmental effects to the cross-sectional and cross-lagged paths were rater-specific (see Tables S12–S14 and Supplementary Materials for details).

Discussion

Intelligence and mental health are the core pillars of individual adaptation, growth, and opportunity (Pettersson et al., 2020). Here, we charted their developmental interplay from childhood through adolescence, operationalizing both domains as p- and

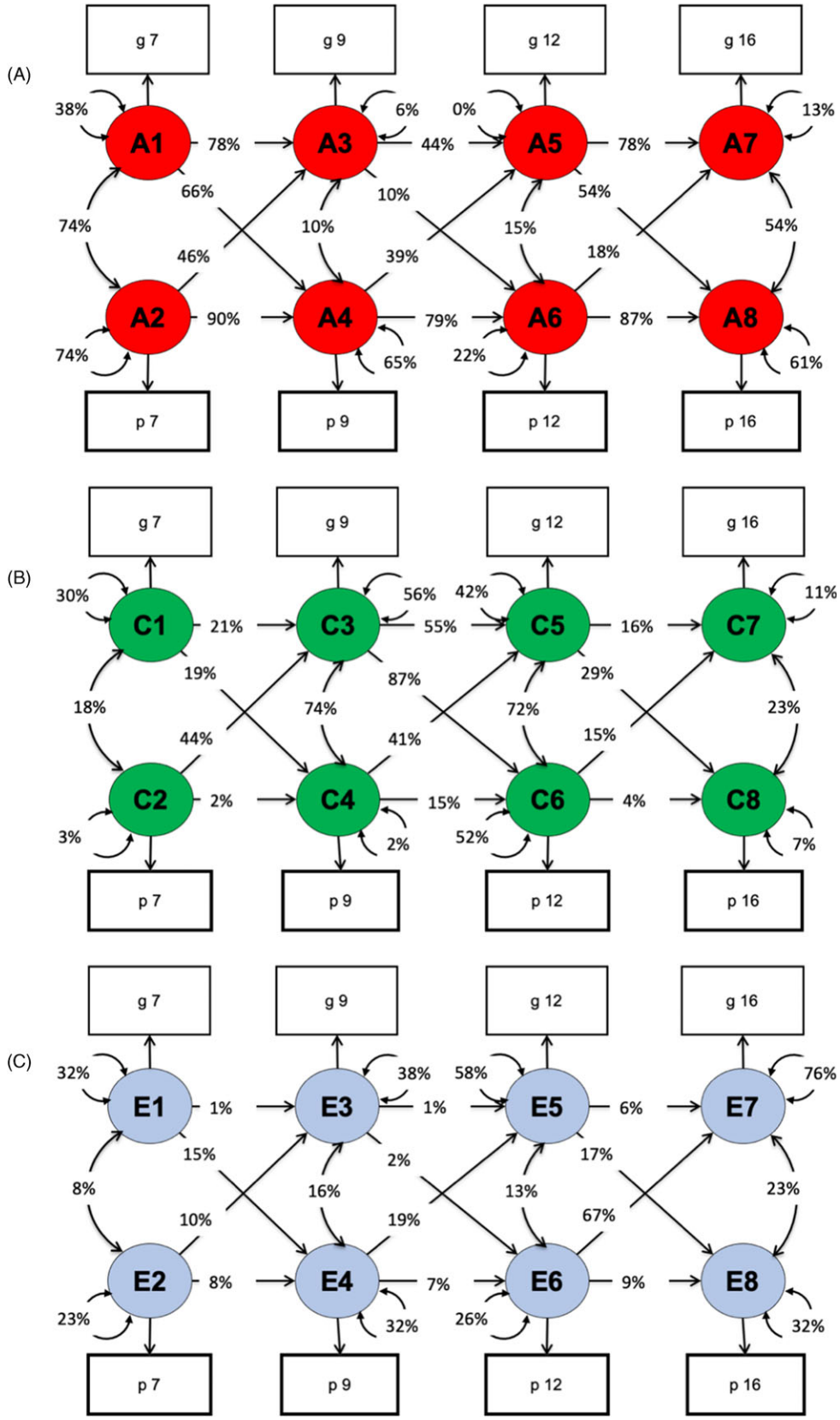

Figure 1. Genetic and environmental decomposition of the developmental associations between p, the general factor of psychopathology and g, the general factor of cognitive ability. Panel A presents the percentage of each association that is accounted for by genetic effects (A). Panel B shows the percentage of each path that is accounted for by environmental factors shared between siblings (C). Panel C shows the percentage of each association that is accounted for by environmental factors unique to each child (E). The ACE cross-lagged model paths are expressed as % of variance of the phenotypic associations. The double-headed arrows mirror the univariate ACE estimates for g- and p-factors; specifically, they indicate the ACE decompositions of p and g's phenotypic variance at the first assessment (i.e., age 7 years) and of p and g's respective residual variance at each subsequent assessment (i.e., ages 9, 12, and 16 years). Model fit: CFI = .99; TLI = .99; RMSEA = .01; diffLL (224) = 387.25, $p < .001$.

g-factors, respectively, that we derived behavior ratings and from cognitive ability test scores. Our results converged on four principal findings.

First, we showed that intelligence and psychopathology (i.e., combined p-factor scores) share consistent, bidirectional, negative associations from age 7 through to 16 years. Notably, their reciprocal associations were independent of the two domains' contemporaneous intercorrelations and respective stability. Higher g-factor scores at earlier ages predicted lower subsequent p-factor scores, suggesting that intelligence might protect against experiencing psychopathology. Conversely, we observed significant, slightly weaker cross-lagged paths from p- to g-factor scores, indicating that higher psychopathology was associated with reduced intelligence. Our findings indicate that the development of intelligence is consistently conditioned on that of psychopathology and vice versa across childhood and adolescence.

Second, the genetic and environmental origins of the cross-lagged paths from intelligence to psychopathology differed from those in the reverse direction (i.e., from psychopathology to intelligence). The estimates of genetic and environmental influences on the cross-lagged paths were in our model independent of the genetic and environmental influences on p- and g-factors at each assessment age. Paths from psychopathology to intelligence were with age increasingly due to nonshared environmental influences, while genetic and shared environmental influences were initially strong but diminished by age 16. Thus, over time, the experiences that are unique to a child within a family become related to the effects of p on g: a child's poor mental health may mean they cannot fully take advantage of their learning opportunities, for example because they miss periods of school or struggle to concentrate during lessons. For the cross-lagged paths from intelligence to psychopathology, genetic influences were dominant from ages 7 to 9 and 12 to 16 years, but shared environmental influences accounted for most of the association from ages 9 to 12. It is possible that the transition from primary to secondary school, which occurred for our sample between the ages of 9 and 12 years, brings about greater influence of the shared family environment on the effects of g on p, when parents select secondary schools according to what they perceive their children's educational needs to be (Riemann et al., 2020). Interestingly, the family-wide or shared environmental influences on the cross-lagged paths from p at age 7 and 12 years to g at 9 and 16 years, respectively, were positive, but the corresponding genetic and nonshared environmental influences were negative (Table S8), suggesting that shared environmental influences work in the opposite direction to genetic and nonshared environment effects. Thus, some of the shared environmental factors that contribute to higher levels of psychopathology may also partly drive subsequent higher levels of g. For example, closely monitoring their homework (i.e., family-wide environments) may increase anxiety in children but also enhance their education, which in turn has been shown to benefit intelligence (Ritchie & Tucker-Drob, 2018). While our interpretations remain speculative until further research corroborates the current results, our findings suggest that distinct etiologies underlie the consistent, bidirectional, negative associations between g and p across childhood and adolescence.

Third, we observed that the cross-lagged paths between intelligence and psychopathology and their genetic and environmental etiology differed across raters. Finding cross-rater differences aligns with a growing body of evidence for the distinct roles of objective and subjective experiences in developmental psychopathology (Baldwin et al., 2019; Danese & Widom, 2020). In particular, the pattern of our cross-lagged model results was different for the p-factor derived from self-ratings, which was less stable than p-factors from parent and teacher ratings and was only weakly associated with intelligence. We also observed cross-rater differences in p-factor loadings of the psychopathology measures, suggesting that the degree to which the extracted p-factors represent internalizing and externalizing behavior problems may vary as a function of rater. Elucidating the origin and meaning of the cross-rater differences in the p-factor and its associations with other constructs should be a priority for future research, because knowing why children, parents, and teachers differ in their perceptions of developmental psychopathology is key to improving the accuracy of diagnostic tools and the effectiveness of interventions and treatments.

Finally, we observed negative correlations between contemporaneous assessments of g- and p-factors, in line with previous reports (Grotzinger et al., 2019; Harden et al., 2020; Hatoum et al., 2018; Huang-Pollock et al., 2017; Martel et al., 2017). These correlations were highest at age 7, when they were largely due to genetic influences, and reduced after age 9, when shared and nonshared environmental influences became dominant. This shift suggests that intelligence and psychopathology might become more differentiated over development for environmental reasons. Thus, interventions at earlier ages are likely to exert effects on both g and p domains, while later interventions might have more domain-specific effects.

Limitations

Our study's first limitation pertains to the measurement instruments that served as observed indicators for the p- and g-factors and that differed across assessment waves. This raises the question of whether the developmental changes that we observed in the etiology of the interplay between p- and g-factors can be attributed to measurement differences across assessment waves. Because both general psychopathology and intelligence were assessed with valid, age-appropriate measures throughout the study's years, we argue that their respective latent factors map the same construct space and are comparable across time. Substantiating this claim, we observed considerable stability of the cross-rater p-factor, which even exceeded the stability of the g-factor (Table 1; cf. Bartels et al., 2002), reflecting that the extracted p-factors mapped the same construct space, even if their measures (i.e., observed indicators) were not administered at all assessment waves. Because we focused on single higher single order factor for p and g, our findings do not allow concluding about the etiology and developmental interplay of specific psychopathology symptoms or cognitive domains.

Second, our p-factors were based on multi-informant behavior ratings on screening measures for psychopathology; however, data from medical records or clinical diagnoses were not available. Third, we extracted single factor models from the psychopathology measures at each assessment age, in line with previous analyses of the current data (Allegrini et al., 2020), although bifactor or correlated factor models may have fitted the data better (Caspi et al., 2014; Lahey et al., 2011, 2012). Our aim here was to elucidate the developmental interplay between general psychopathology and general intelligence, rather than empirically testing the factor structure that underlies psychopathology. Future research is needed to confirm if a single p-factor can adequately represent dimensions of developmental psychopathology. Fourth, data on general intelligence and psychopathology were collected from our sample every 2–3 years starting at age 7 years. Yet, early-life and

more frequent assessments may have achieved a better understanding of the developmental interplay between intelligence and psychopathology. Finally, our sample has suffered attrition, which can cause sampling biases, although these were likely to be modest here. Moreover, our sample was UK-based; generalizing our findings across other contexts is not possible.

Conclusions

We showed in cross-lagged analyses of a large, longitudinal twin study that dimensional measures of intelligence and psychopathology share consistent, bidirectional, negative associations across childhood and adolescence. These reciprocal associations appear to stem from different genetic and environmental etiologies. The cross-lagged paths from intelligence to psychopathology were largely due to genetic influences, but the paths from psychopathology to intelligence were driven by environmental factors, and increasingly so with age. Our findings suggest that intelligence and psychopathology are transdiagnostic and dimensional traits that are developmentally intertwined due to fluctuating etiological processes.

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