

A longitudinal twin and sibling study of the hopelessness theory of depression in adolescence and young adulthood

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Background. Maladaptive cognitive biases such as negative attributional style and hopelessness have been implicated in the development and maintenance of depression. According to the hopelessness theory of depression, hopelessness mediates the association between attributional style and depression. The aetiological processes underpinning this influential theory remain unknown. The current study investigated genetic and environmental influences on hopelessness and its concurrent and longitudinal associations with attributional style and depression across adolescence and emerging adulthood. Furthermore, given high co-morbidity between depression and anxiety, the study investigated whether these maladaptive cognitions constitute transdiagnostic cognitive content common to both internalizing symptoms.

Method. A total of 2619 twins/siblings reported attributional style (mean age 15 and 17 years), hopelessness (mean age 17 years), and depression and anxiety symptoms (mean age 17 and 20 years).

Results. Partial correlations revealed that attributional style and hopelessness were uniquely associated with depression but not anxiety symptoms. Hopelessness partially mediated the relationship between attributional style and depression. Hopelessness was moderately heritable ($A = 0.37$, 95% confidence interval 0.28–0.47), with remaining variance accounted for by non-shared environmental influences. Independent pathway models indicated that a set of common genetic influences largely accounted for the association between attributional style, hopelessness and depression symptoms, both concurrently and across development.

Conclusions. The results provide novel evidence that associations between attributional style, hopelessness and depression symptoms are largely due to shared genetic liability, suggesting developmentally stable biological pathways underpinning the hopelessness theory of depression. Both attributional style and hopelessness constituted unique cognitive content in depression. The results inform molecular genetics research and cognitive treatment approaches.

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Introduction

Depression is very common, chronic, and increases markedly in adolescence (Costello *et al.* 2003; Ford *et al.* 2003; Hankin *et al.* 1998). Adolescent depression reliably predicts long-term mental health difficulties (Harrington *et al.* 1990; Dunn & Goodyer, 2006; Gregory *et al.* 2007) and carries burden of social and educational impairment (Puig-Antich *et al.* 1993; Katon *et al.* 2010; Riglin *et al.* 2014). Maladaptive

cognitions, such as biases in how individuals attend to, interpret and remember emotional information, have been implicated in the development and maintenance of depression (Jacobs *et al.* 2008), and are targeted by recommended first-line treatments such as cognitive behavioural therapy (CBT) (AACAP, 2007). Therefore, it is of high importance to understand the aetiology of depression-related cognitions operating across adolescence and emerging adulthood.

Hopelessness theory of depression

Negative attributional style and hopelessness are two maladaptive cognitions associated with adolescent depression. Negative attributional style refers to the attribution of negative events to internal (directed to the

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self), stable (likely to persist over time) and global (likely to affect many aspects of life) causes, and positive events to external, unstable and specific causes (Abramson *et al.* 1978). Hopelessness is an expectation that the future will be negative and that this outcome cannot be altered (Beck *et al.* 1974). A number of theories specified negative attributional style and hopelessness as causal cognitive vulnerabilities in depression, including the hopelessness theory of depression (Abramson *et al.* 1989). Specifically, the theory posits that in the context of negative life events, attributional style contributes to the formation of hopelessness, which in turn contributes to development of depression symptoms. In other words, hopelessness is thought to mediate the prospective relationship between attributional style and depression. Although to date many aspects of the theory have gained empirical support (Metalsky & Joiner, 1992; Joiner, 2001; Hankin, 2008), evidence for the mediation is mixed in both child and adult samples (Alloy & Clements, 1998; Abela, 2001; Hankin *et al.* 2001).

Cognitive specificity

Given high co-morbidity of depression with anxiety disorders (Angold *et al.* 1999; Costello *et al.* 2003; Kessler *et al.* 2005; Cummings *et al.* 2014), it is of theoretical and clinical interest to differentiate them based on maladaptive cognitions. The cognitive content hypothesis posits that although anxious and depressed individuals both have distorted cognitions, the content differs across these disorders (Beck & Perkins, 2001). Specifically, it is hypothesized that depressed individuals tend to think negatively about the self and focus on experiences of loss whereas anxious individuals focus on perceived threat or danger. In line with this model, cognitive concerns targeted in CBT tend to vary across anxiety and depressive disorders (Brewin, 1996).

The hopelessness theory of depression has been proposed specifically to explain some of the causal factors in depression. In support of this claim, evidence generally suggests that hopelessness is uniquely associated with depression and not anxiety (Alloy & Clements, 1998; Beck *et al.* 2001, 2006; Miranda & Mennin, 2007; Alloy *et al.* 2012; Hendriks *et al.* 2014). This indicates that negative inferences about the future might constitute unique content in depression that differentiates it from anxiety disorders. However, there is mixed evidence about the specificity of negative attributional style to depression (Ahrens & Haaga, 1993; Luten *et al.* 1997; Waschbusch *et al.* 2003; Hankin *et al.* 2004; Brozina & Abela, 2006; Reardon & Williams, 2007), suggesting that maladaptive interpretations of events might instead be a transdiagnostic cognitive risk factor

for internalizing problems. Notably, the specificity of attributional style and hopelessness has largely been studied in adults. As maladaptive cognitions are thought to emerge in development (Cole *et al.* 2008; Field & Lester, 2010), it is important to gain a better understanding of disorder-specific and transdiagnostic cognitions in depression and anxiety in young people.

Aetiology

There is growing evidence that individual differences in maladaptive cognitions stem from both genetic and environmental influences. Our team has previously shown that attributional style is moderately heritable in adolescence (Lau *et al.* 2006; Lau & Eley, 2008; Zavos *et al.* 2010). These studies have also found genetic and environmental overlap between attributional style, depression and anxiety symptoms, suggesting that maladaptive cognitive processes in part represent a genetic vulnerability to internalizing problems, in addition to being reflections of the individual's environment (Beck, 2008). This also indicates that both inherited predispositions and environmental circumstances might play a role in the hopelessness theory of depression, an idea originally hypothesized by Abramson *et al.* (1989). To date, a composite measure of hopelessness and guilt was found to be moderately heritable in adults, with substantial influence of the individual-specific environment (Jang *et al.* 2004), however no study has yet investigated the aetiology of hopelessness in young people, or its genetic and environmental associations with depression or attributional style. Examining aetiological influences on the joint associations between attributional style, hopelessness and depression could help to clarify some of the mechanisms that underpin these relationships in the hopelessness theory of depression. It may also help to disentangle common and specific influences on these traits.

Aims

The current study investigated the hopelessness theory of depression from an aetiological perspective. Three waves of data from a large epidemiological sample of adolescent twins and siblings were employed. First, the potential cognitive specificity of negative attributional style and hopelessness to depression *v.* anxiety symptoms was investigated, both concurrently at mean age 17 years, and prospectively across adolescence and young adulthood. Based on the existing, largely adult literature we hypothesized that hopelessness would be uniquely associated with depression while attributional style would be associated with both depression and anxiety. Second, we aimed to determine whether hopelessness mediates the

relationship between attributional style and depression, both concurrently and across time. In line with the hopelessness theory of depression, we expected to observe at least partial mediation. Third, we explored what proportion of variance in hopelessness was accounted for by genetic and environmental influences. We hypothesized that similarly to attributional style, hopelessness would be moderately heritable, in line with adult estimates of hopelessness and guilt. Fourth, we examined the shared aetiology between attributional style, hopelessness and depression. We expected that common genetic influences would explain most of the shared variance between these three traits, in line with the generalist genes hypothesis (Eley, 1997), which proposes that traits co-vary due to shared genetic influences, while non-shared environmental influences are generally symptom-specific and contribute to the differentiation between the traits. Based on the generalist genes hypothesis, we hypothesized that there would be no unique genetic association between hopelessness and depression independent of attributional style.

Method

Sample

We used data from waves 2–4 (hereon referred to as times 1–3, respectively) of a longitudinal twin and sibling study, the Genesis 1219 (G1219; McAdams *et al.* 2013). The study was given ethical approval by the Research Ethics Committee of the Institute of Psychiatry, King's College, London, South London and Maudsley NHS Trust and Goldsmiths, University of London. Informed consent was obtained from parents of adolescents aged <16 years and from participants aged ≥ 16 years. The sample characteristics are presented in Table 1. Concurrent analyses were conducted at time 2, the only time at which hopelessness was measured. Longitudinal analyses spanned times 1–3, using attributional style measure at time 1, hopelessness at time 2 and depression and anxiety symptoms at time 3.

Measures

Attributional style (times 1 and 2)

Participants completed the revised Children's Attributional Style Questionnaire (Thompson *et al.* 1998); a 24-item forced-choice questionnaire that described a positive or negative event (e.g. 'You get an A on a test') and asked about its possible cause (e.g. 'I am clever'). The measure assesses three dimensions of attributional style (internal-external, global-specific, stable-unstable), with an overall lower

composite score indicating more negative attributional style. The measure demonstrates moderate internal consistency reliabilities ranging $\alpha = 0.40$ – 0.60 , moderate 6-month test–retest reliability of 0.53 and small to moderate criterion-related validity assessed through association with depression symptoms ($r = -0.40$) (Thompson *et al.* 1998).

Hopelessness (time 2)

Participants completed the Hopelessness Scale (Beck *et al.* 1974), consisting of 20 true-false items assessing feelings of hopelessness (e.g. 'My future seems dark to me'). Theories of hopelessness suggest that it may be underpinned by three subfactors reflecting feelings about the future, loss of motivation and future expectations (Beck *et al.* 1974), although unidimensional solutions have also been reported (Dozois & Covin, 2004). In the current analyses items were summed to create a total score. The measure has sound psychometric properties in both clinical and healthy adults and adolescent samples, with high internal consistencies up to $\alpha = 0.90$ and high 3-week test–retest reliability of 0.85, demonstrating robust validity with related constructs, such as depression symptoms, and suicidal ideation and intent independently of depression (Beck *et al.* 1974; Young *et al.* 1992; Dozois & Covin, 2004).

Depression symptoms (times 2 and 3)

Participants completed the Short Mood and Feelings Questionnaire (Angold *et al.* 1995), a 13-item unidimensional self-report measure assessing how often depression symptoms occurred in the past 2 weeks. Responses were summed to give a total depression score. The measure demonstrates good reliability and validity (Angold *et al.* 1995).

Anxiety symptoms (times 2 and 3)

At time 2 adolescents completed the Spence Children's Anxiety Scale (Spence, 1998); a 38-item self-report questionnaire tapping anxiety disorder related symptoms, such as generalized anxiety, panic, separation anxiety and social anxiety symptoms. At time 3 participants completed the Revised Symptoms of Anxiety Scale (Gregory *et al.* 2011), an age-appropriate version of the Revised Child Anxiety and Depression Scale (Chorpita *et al.* 2000), consisting of 36 self-report items designed to assess DSM-IV anxiety disorder symptoms. Responses were summed to create total scores. The measures have good reliability and validity (Spence, 1998; Birmaher *et al.* 1999; Chorpita *et al.* 2000; Gregory *et al.* 2011). Internal consistencies for all measures in the current study and descriptive statistics are presented in Table 2.

Table 1. Sample characteristics

	Time 1	Time 2	Time 3
No. of pairs	1372	866	896
Female/male pairs (%)	768 (56)/604 (44)	520 (60)/346 (40)	547 (61)/349 (39)
Age: mean (years, months) (range)	15.0 (12.0–21.0)	17.0 (14.0–23.0)	20.0 (18.0–27.0)
Zygoty (MZ/DZS/DZO/sib/unknown)	350/313/334/330/45	234/207/232/182/11	230/214/232/201/19

MZ, Monozygotic; DZS, dizygotic same sex; DZO, dizygotic opposite sex.

The inclusion of siblings inevitably resulted in large age ranges; however 72% of the participants were twins with a tighter age range (e.g. at time 2, age s.d. = 1.11, range = 15–19 for twins, age s.d. = 1.97, range = 15–23 for siblings).

Attrition was predicted by socio-economic status (responses were more likely from individuals with parents reporting higher qualifications and home ownership), delinquency (individuals reporting lower levels of delinquent behaviour were more likely to stay in the study) and sex (females were more likely than males to remain in the study), but not by zygosity and internalizing symptoms.

Phenotypic analyses

Phenotypic analyses were conducted using Stata (StataCorp, 2007). Concurrent (time 2) and longitudinal (across times 1–3) associations between variables were first explored using full and partial correlations. Partial correlations allowed investigating unique associations between two variables over and above associations accounted for by other variables (e.g. unique association between attributional style and depression symptoms controlling for hopelessness and anxiety symptoms). Next, the Sobel-Goodman test was used to test whether hopelessness mediates the relationship between attributional style and depression, concurrently and across time (Preacher & Hayes, 2004). Bootstrapping was used to obtain confidence intervals. All phenotypic analyses were conducted on untransformed and unregressed variables for comparison with other published samples. Analyses were conducted on a random selection of one twin from each twin pair to ensure the independence of observations.

Twin analyses

The twin design compares the similarity between monozygotic (sharing 100% of their genes) and dizygotic (sharing on average 50% of their segregating genes) twin pairs. Differences in within-pair correlations allows estimations of the influences of additive genetics (A), shared environment (C, factors that contribute to phenotypic similarity between siblings) and non-shared environment (E, factors that contribute to phenotypic differences between siblings). Quantitative genetic methods are described comprehensively elsewhere (Rijsdijk & Sham, 2002; Plomin et al. 2013).

Twin models were fitted using OpenMx (Boker et al. 2011) within R (<http://www.R-project.org>) (TeamRDC, 2010), a structural equation modelling package for

genetically informative data. As is standard in model fitting analysis, all variables were regressed for age and sex (McGue & Bouchard, 1984), and depression at both waves and hopelessness were log transformed. Transformation did not have an impact on the relationship between the variables in a way that would alter interpretation.

Models were fitted using raw data maximum likelihood. The core fit statistic was minus twice the log likelihood (-2LL) of the observations. This is not an overall measure of fit, but provides a relative measure of fit, since differences in -2LL between models are distributed as χ^2 . To examine the overall fit of the genetic model we compared the -2LL to that of a saturated model (which fully describes data using the maximum number of free parameters, estimating variances, covariances and means for the raw data to get a baseline index of fit). The fit of sub-models was assessed by χ^2 difference tests, Akaike's Information Criterion (AIC) and Bayesian Information Criterion, with lower values suggesting a better fit. If the difference between the AIC of two models was <10, the more parsimonious model was selected (Wagenmakers & Farrell, 2004). Information about the precision of parameter estimates was obtained by likelihood-based confidence intervals.

Univariate twin analyses

Univariate genetic analyses were conducted for all variables. Males and females showed differences in variance on all variables and a scalar was fitted to account for this difference. Qualitative and quantitative sex differences were tested to see whether males and females differ in type and magnitude of genetic and environmental influences, but such differences were not found. The exception was attributional style at time 1, which showed quantitative sex differences, however differences in magnitude were small with

Table 2. Descriptive statistics and phenotypic correlations: full correlations below diagonal and partial correlations above diagonal

(a) Descriptive statistics	N (individuals)	Mean (s.d.)	Skew	Cronbach's α
Attributional style time 1	2562	4.30 (3.31)	-0.50	0.61
Attributional style time 2	1570	4.37 (3.50)	-0.58	0.66
Hopelessness time 2	1573	3.66 (3.25)	1.66	0.82
Depression time 2	1590	6.25 (5.33)	1.14	0.79
Depression time 3	1549	6.45 (5.73)	1.26	0.90
Anxiety time 2	1569	20.62 (12.80)	1.21	0.87
Anxiety time 3	1552	25.06 (14.88)	1.18	0.94

(b) Concurrent correlations	Attributional style time 2	Hopelessness time 2	Depression time 2	Anxiety time 2
Attributional style time 2	-	-0.31 (-0.37 to -0.25)	-0.23 (-0.29 to -0.16)	-0.04 (-0.11 to 0.03)
Hopelessness time 2	-0.47 (-0.52 to -0.42)	-	0.27 (0.21 to 0.33)	0.07 (0.00 to 0.14)
Depression time 2	-0.46 (-0.51 to -0.40)	0.50 (0.45 to 0.55)	-	0.51 (0.48 to 0.56)
Anxiety time 2	-0.32 (-0.38 to -0.26)	0.36 (0.30 to 0.42)	0.61 (0.57 to 0.65)	-

(c) Longitudinal correlations	Attributional style time 1	Hopelessness time 2	Depression time 3	Anxiety time 3
Attributional style time 1	-	-0.26 (-0.33 to -0.19)	-0.09 (-0.17 to -0.01)	-0.03 (-0.11 to 0.05)
Hopelessness time 2	-0.28 (-0.35 to -0.21)	-	0.17 (0.09 to 0.25)	0.08 (0.00 to 0.16)
Depression time 3	-0.23 (-0.30 to -0.15)	0.33 (0.26 to 0.40)	-	0.59 (0.54 to 0.64)
Anxiety time 3	-0.20 (-0.27 to -0.12)	0.27 (0.20 to 0.34)	0.62 (0.57 to 0.67)	-

s.d., Standard deviation.

95% Confidence intervals (CIs) are presented in parentheses. CIs not inclusive of zeros indicate significant correlations. Non-overlapping CIs mean significant difference between the values.

Partial correlations between two variables (e.g. attributional style and hopelessness) controlled for the associations with the other two variables (e.g. depression and anxiety symptoms).

Higher scores indicate more positive attributional style, greater helplessness and greater depression and anxiety symptoms.

overlapping 95% confidence intervals [$A_M=0.38$ (95% CI 0.25–0.49) *v.* $A_F=0.49$ (95% CI 0.39–0.58) and $E_M=0.49$ (95% CI 0.39–0.58) *v.* $E_F=0.51$ (95% CI 0.42–0.61)] and for simplicity homotypic models are presented. Finally, comparisons indicated that covariances, means and variances could be equated across dizygotic twins and singleton siblings for all variables, thus siblings were modelled alongside twins in the analyses.

Multivariate twin analyses

First, a one-factor independent pathway model was fitted to examine the genetic and environmental overlap on the three variables (Fig. 1a). The model allows one set of common (A_C , and E_C) and variable-specific (A_S , and E_S) genetic and environmental influences on each variable. The model tests whether there is a single set of common aetiological factors that influence attributional style, hopelessness and depression symptoms, accounting for their associations, in addition to variable-specific factors.

Next, the Cholesky decomposition (Fig. 1b) was used to examine whether there are any genetic and environmental influences shared between hopelessness and depression when accounting for the genetic overlap with attributional style. The Cholesky decomposition assumes three distinct sets of genetic and environmental influences on a variable at each time point. A_1 and E_1 are influences on the first variable (paths a_{1_1} and e_{1_1}) that can also influence the remaining two variables (paths $a_{1_{2-3}}$ and $e_{1_{2-3}}$). A_2 and E_2 influence the second variable (paths a_{2_2} and e_{2_2}) and can also influence the third variable over and above the influences accounted for by A_1 and E_1 (paths a_{2_3} and e_{2_3}). A_3 and E_3 are unique, residual influences specific to the third variable only (paths a_{3_3} and e_{3_3}).

Both multivariate models were fitted concurrently at time 2, and longitudinally at times 1–3. Although any ordering of variables explained the variance-covariance matrix between the variables equally well, the order of variables was based on the hopelessness theory of depression. Longitudinal models allowed investigating how aetiological influences operated across development, reflecting the developmental predictions of the theory. However, in addition to variable-specific influences, influences at later time points can also reflect age-specific genetic and environmental innovation that characterizes adolescence (Hannigan *et al.* in press; Kendler *et al.* 2008; Waszczuk *et al.* 2016). Concurrent models allowed investigating variable-specific influences without the confounding effect of genetic and environmental innovation across time.

Results

Phenotypic results

Attributional style and hopelessness were moderately associated with internalizing symptoms (Table 2), both concurrently ($r=-0.47$ and 0.50 with depression, $r=-0.32$ and 0.36 with anxiety, respectively) and longitudinally ($r=0.-0.23$ and 0.33 with depression, $r=-0.20$ and 0.27 with anxiety, respectively). However, partial correlations indicated that when controlling for depression symptoms, neither attributional style nor hopelessness remained significantly associated with anxiety symptoms. Conversely, both maladaptive cognitive styles were uniquely associated with depression symptoms after controlling for anxiety, both concurrently and across time ($r_p=-.23$ and -0.09 for attributional style and $r_p=.27$ and 0.17 for hopelessness, respectively). Given the lack of unique association between both maladaptive styles and anxiety symptoms, only depression was taken forward to further analyses.

Mediation analyses revealed that hopelessness partially mediated the relationship between attributional style and depression symptoms (Fig. 2). Specifically, hopelessness mediated about 37% of this relationship concurrently (total effect = -0.71 , indirect effect via hopelessness = -0.26) (Fig. 2a), and about 38% longitudinally (total effect = -0.37 , indirect effect via hopelessness = -0.14) (Fig. 2b).

Twin modelling results

Model fit statistics for comparisons to saturated models, and testing whether parameters can be dropped, are presented in Supplementary Table S1. Model fit statistics corroborate AE models and in the full models C estimates are very small. However, for completeness full ACE models are presented in the Supplementary material (Tables S2, S3). Dropping C from the models did not have impact on the interpretation of the results. The associations between attributional style and depression symptoms, including univariate ACE results for these variables, have been reported before (Lau *et al.* 2006; Lau & Eley, 2008; Zavos *et al.* 2010).

Univariate results revealed that hopelessness was moderately heritable ($A=0.37$, 95% CI 0.28–0.47), with remaining variance accounted for by non-shared environmental influences ($E=0.63$, 95% CI 0.53–0.73). Attributional style and depression symptoms were also moderately heritable and univariate results for these variables have been presented before (Lau *et al.* 2006; Lau & Eley, 2008; Zavos *et al.* 2010).

Independent pathway models examined common (i.e. influencing all three variables) and variable-specific genetic and shared environmental influences

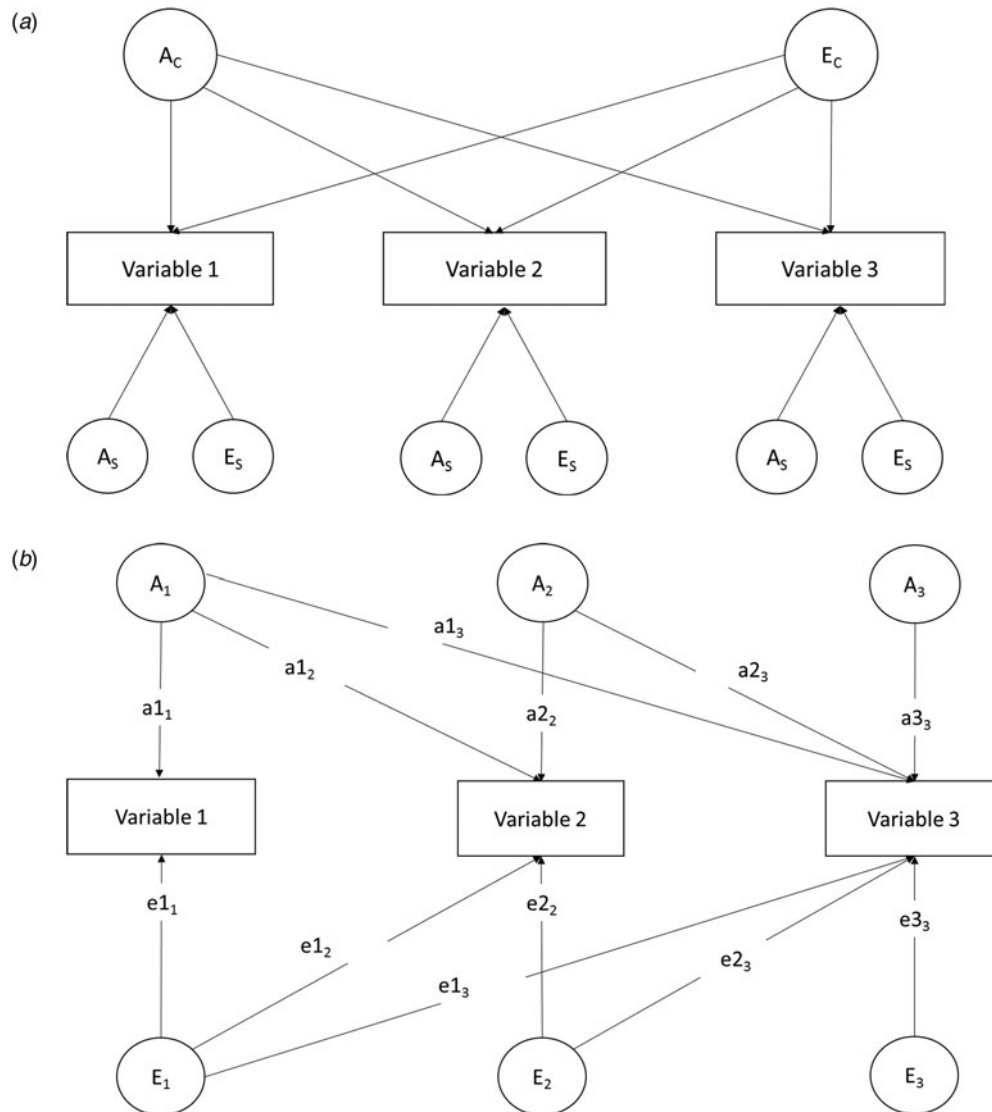


Fig. 1. (a) Independent pathways model, (b) Cholesky decomposition. AE models are presented for clarity. Model fit statistics corroborated AE models and in the full models C estimates were very small.

on attributional style, hopelessness and depression symptoms. Concurrently, common genetic influences accounted for majority of genetic influences on attributional style and depression ($A_c=0.31$ and 0.30 , accounting for 70% (95% CI 49–96) and 64% (95% CI 44–90) of total genetic variance in each variable, respectively), and about half genetic influences on hopelessness ($A_c=0.19$, accounting for 51% (95% CI 34–71) of total genetic variance in hopelessness) (Fig. 3a). Common non-shared environmental influences were significant and accounted for about a third of all non-shared environmental influences on variables [$E_c=0.21$, 0.26 and 0.11 on attributional style, hopelessness and depression, respectively, accounting for 37% (95% CI 21–57), 42% (95% CI 25–59) and 20% (95% CI 10–33) of total non-shared environmental variance].

All variable-specific influences were significant ($A_s=0.13$ – 0.18 , $E_s=0.35$ – 0.42).

Longitudinally, common genetic influences were the only source of common variance, and accounted for about half of genetic influences in attributional style time 1 [$A_c=0.22$, accounting for 51% (95% CI 37–67) of total genetic variance in attributional style time 1] and the majority of genetic influences on hopelessness time 2 and depression symptoms time 3 ($A_c=0.36$ and 0.30 , accounting for 83% (95% CI 64–100) and 77% (95% CI 56–100) of total genetic variance in each variable respectively). Furthermore, there were only significant variable-specific genetic influences on attributional style (Fig. 4a). Thus all genetic influence on both hopelessness and depression was common to all three variables. Common non-shared environmental

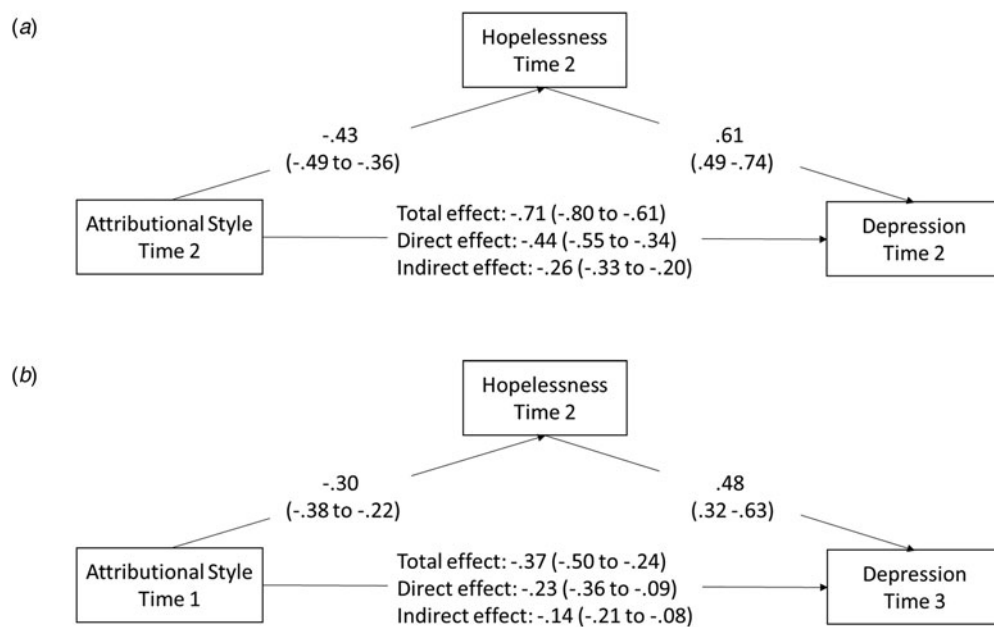


Fig. 2. Mediation analyses results: (a) concurrently at time 2, (b) longitudinally across times 1–3. 95% Confidence Intervals (CIs) are presented in brackets. CIs not inclusive of zeros indicate significant coefficients. Non-overlapping CIs mean significant difference between the values.

influences did not emerge (for longitudinal independent pathway model with E_c see Supplementary Table S2, and for model fit comparisons see Supplementary Table S1 note), instead, all non-shared environmental influences were large and variable-specific ($E_s = 0.57$ – 0.61).

Cholesky decompositions examined whether there are any genetic and environmental influences shared between hopelessness and depression when accounting for the genetic overlap with attributional style. Both concurrent and longitudinal Cholesky decompositions indicate that after accounting for genetic influences shared with attributional style, there were no significant additional genetic associations between hopelessness and depression (standardized path a_{23} was non-significant in Figs 3b and 4b). Although non-shared environmental influences were largely variable-specific, when accounting for non-shared environmental influences shared with attributional style, there was a small but significant non-shared environmental association between hopelessness and depression in concurrent analyses (standardized path $e_{23} = 0.02$).

Discussion

This study is the first to investigate the hopelessness theory of depression from an aetiological perspective, both within and across time-points during adolescence and into young adulthood. Phenotypic results

supported the theory, as hopelessness partially mediated the relationship between attributional style and depression symptoms, and both maladaptive cognitive styles were unique to depression and not to anxiety (the latter phenotype was therefore dropped from twin analyses). Twin modelling results revealed that a set of common genetic influences largely accounted for the association between attributional style, hopelessness and depression symptoms, indicating shared genetic liability to maladaptive cognitions and depression underpinning the hopelessness theory of depression.

Aetiology of hopelessness theory of depression

Current phenotypic results indicate that hopelessness partially mediates the association between attributional style and depression symptoms, both concurrently and across development, as predicted by the hopelessness theory of depression. This is in line with some (Alloy & Clements, 1998), but not all (Abela, 2001; Hankin *et al.* 2001) previous studies. Furthermore, although prospective longitudinal results indicated that about a third of the association between early attributional style and later depression was mediated via hopelessness, both attributional style and hopelessness were also uniquely associated with depression symptoms, indicating that they may be independent cognitive risk factors for developing depression in adolescence.

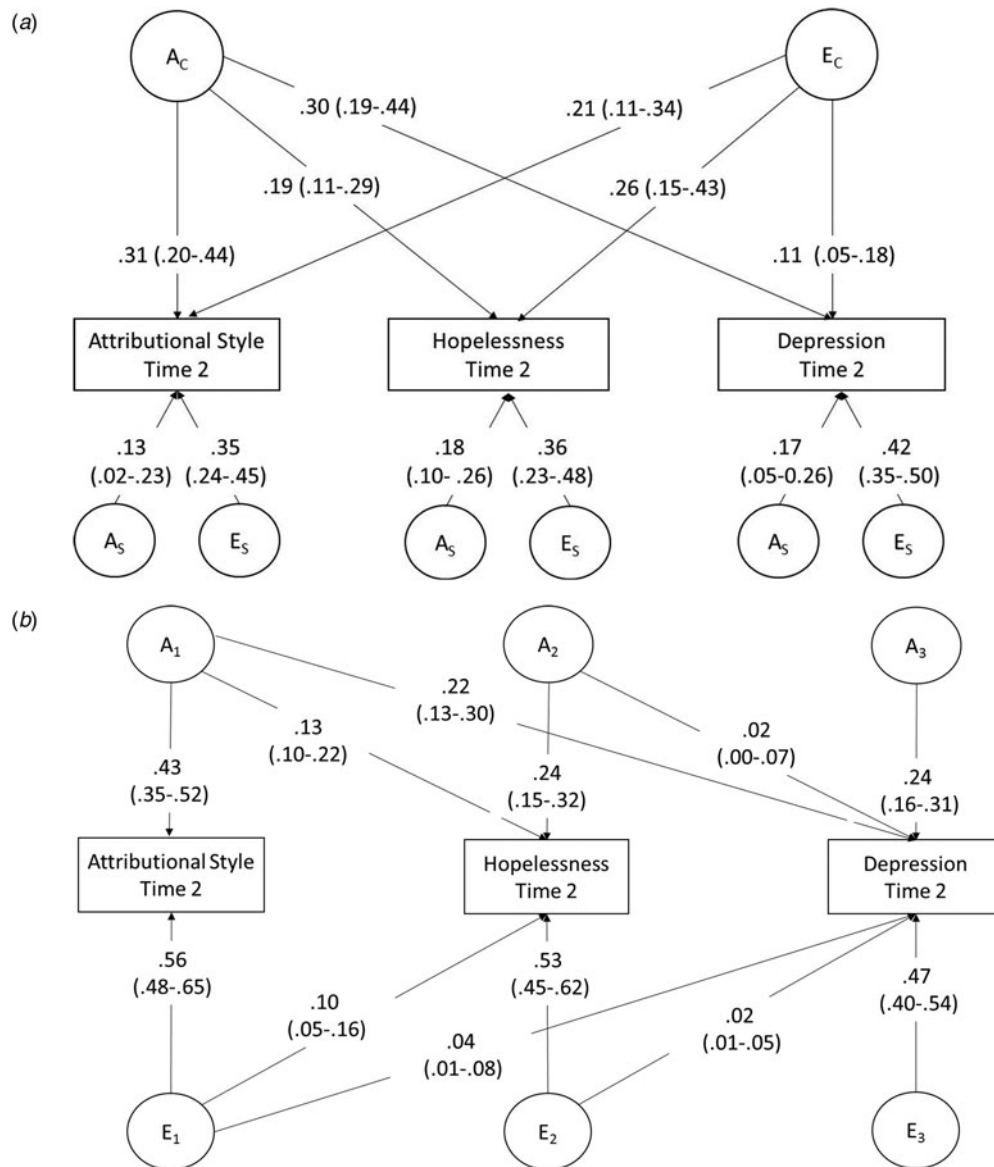


Fig. 3. Concurrent multivariate results: (a) Independent pathways model, (b) Cholesky decomposition. All paths are squared. Square root of these values should be taken to obtain variance path.

Univariate twin modelling results highlighted the role of both genetic and environmental influences in the aetiology of adolescent hopelessness. The current heritability estimate was moderate, in line with the previous estimate of heritability of a composite hopelessness and guilt trait in adults (Jang *et al.* 2004), but extends previous research by using a more comprehensive, validated measure of hopelessness and expands understanding of its aetiology to a novel developmental period.

Most of the genetic influences on each of the two maladaptive cognitions and depression symptoms were shared concurrently and across development. Furthermore, there were no shared genetic influences

between hopelessness and depression over and above genetic influences shared with attributional style. Thus, as expected, the association between maladaptive cognitions and depression was explained largely by underlying genetic liability, in line with the generalist genes hypothesis (Eley, 1997). The results are also in agreement with previous findings that associations between many different cognitive biases and internalizing problems are largely due to shared genetic influences (Lau & Eley, 2008; Zavos *et al.* 2010; Chen & Li, 2013; Moore *et al.* 2013; Waszczuk *et al.* 2013; Brown *et al.* 2014; Lau *et al.* 2014). The results are suggestive of developmentally stable biological pathways underpinning the hopelessness theory of depression.

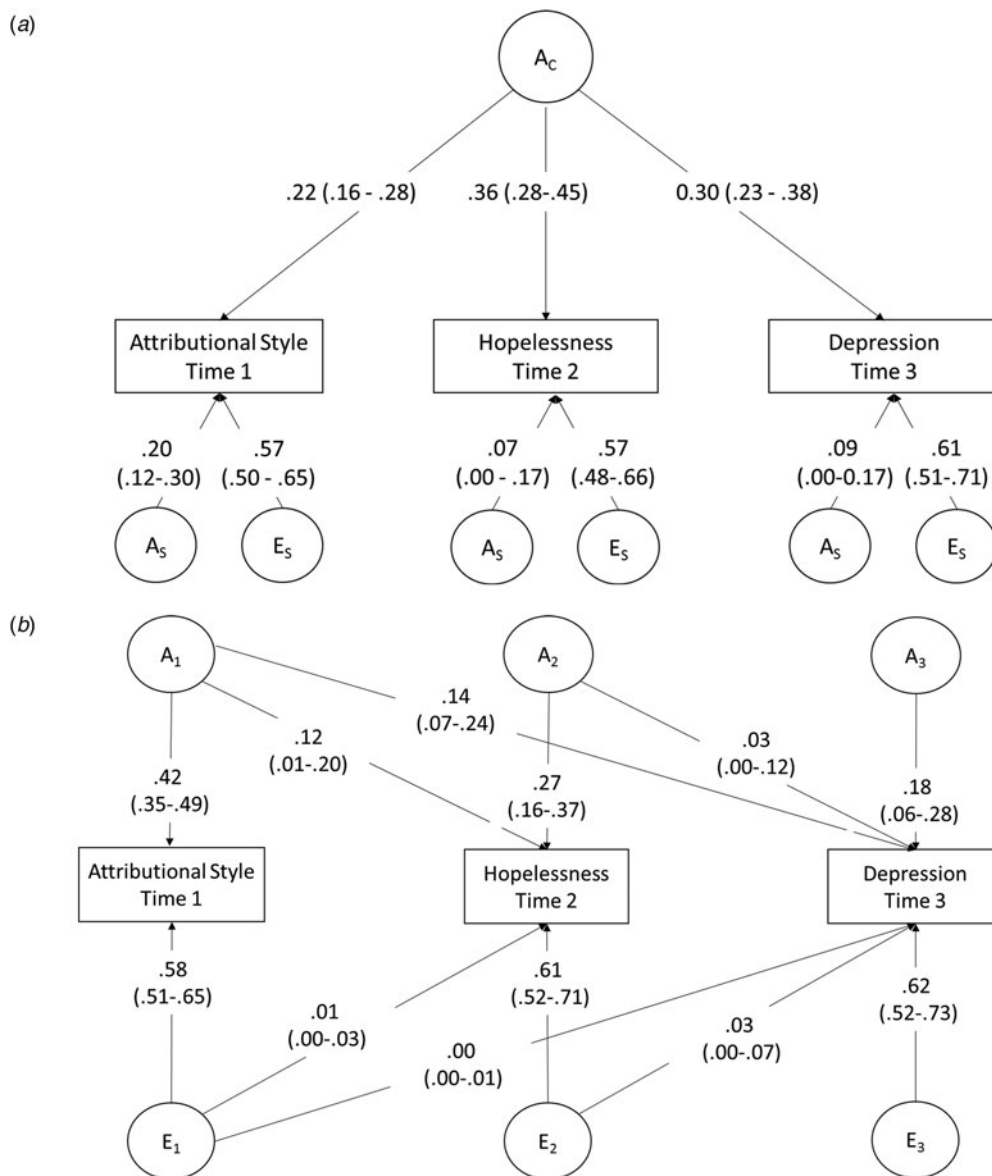


Fig. 4. Longitudinal multivariate results: (a) Independent pathways model, (b) Cholesky decomposition. All paths are squared. Square root of these values should be taken to obtain variance path.

Evidence for shared genetic effects has implications for molecular genetic studies, supporting the argument that including cases with a range of depression-related phenotypes would lead to increasing power to detect shared susceptibility loci (Hettema *et al.* 2015). It also suggests that the role of specific genes in the aetiology of internalizing symptoms and maladaptive cognition should be investigated as it might be possible to combine the genetic markers to create polygenic risk scores to predict an individual’s vulnerability to depression (Demirkan *et al.* 2011).

Conversely, non-shared environmental influences were largely trait-specific, especially in longitudinal analyses. This indicates that environmental influences contribute to differences between cognitive

vulnerabilities and depression, possibly explaining some of the phenotypic specificity observed. However, a significant common non-shared environmental factor suggests that it might be possible to identify environmental influences that contribute to the hopelessness theory of depression. In line with the theory, these could be negative life events that interact with attributional style in a diathesis-stress manner. Future studies should aim to identify these specific environmental influences to inform targeted clinical and resilience interventions in adolescence. Nonetheless, this common non-shared E environmental factor might also to some extent reflect time-specific measurement error, as it does not replicate in the longitudinal analyses.

Cognitive specificity

Evidence for phenotypic specificity in associations between attributional style, hopelessness and depression, and unique environmental influences acting on these symptoms, has implications for therapeutic interventions. Furthermore, identifying disorder-specific maladaptive cognitions, and also those shared between co-morbid disorders such as depression and anxiety, could continue to inform the tailoring of CBT programmes to a given diagnosis. Current findings indicate that both attributional style and hopelessness are independently and uniquely associated with depression symptoms, but not with anxiety symptoms in adolescence. This is in line with previous research finding that hopelessness is a maladaptive cognition specific to depression (Beck *et al.* 2001, 2006; Alloy *et al.* 2012; Hendriks *et al.* 2014). Conversely, the finding that attributional style is unique to depression supports some previous studies in adults and children (Rodriguez & Pehi, 1998; Hankin *et al.* 2004; Brozina & Abela, 2006), but does not support the view, based largely on adult literature, that attributional style is a transdiagnostic cognitive risk factor for both depression and anxiety (Luten *et al.* 1997; Alloy *et al.* 2012). Taken together, these results indicate that negative thoughts and interpretations about the present and future events constitute central and unique cognitive content in adolescent depression, in line with cognitive specificity hypothesis (Beck & Perkins, 2001). This supports the clinical evidence that modifying attributional style and hopelessness in CBT prevents and reduces adolescent depression, as well as other important depression-related symptoms such as suicidality (Brent *et al.* 1998; Voelz *et al.* 2003; Stanley *et al.* 2009). However, our findings also imply that targeting attributional style and hopelessness may not be as effective in reducing adolescent anxiety symptoms. Future research should aim to explore the unique and transdiagnostic content of depressive cognitions in more detail by combining multiple cognitive distortions within a single study.

Limitations

The genetically-informative, representative sample and multiple time points are strengths of the study. However, a number of limitations are noteworthy. First, it was beyond the scope of the current study to investigate all aspects of the hopelessness theory of depression, such as the generally supported diathesis-stress interaction between negative life events and attributional style (Abela, 2001; Hankin *et al.* 2001; Abela & Sarin, 2002). Furthermore, Abramson *et al.* (1989) posited that the theory is specific to 'hopelessness depression', however the distinction of this

depression subtype from major depression is debated (Alloy & Clements, 1998), and the theory has generally been studied with broad measures of depression. Future research should explore phenotypic and aetiological associations between different dimensions of depression, attributional style and hopelessness within the context of the theory, which was beyond the scope of the current study. It should also investigate whether there are bidirectional associations between attributional style, hopelessness and depression (Zavos *et al.* 2010). Second, our analyses used self-report internalizing symptoms. Results should be replicated in clinical samples and using lifetime diagnostic interviews. However, symptoms of internalizing disorders are important markers of psychopathology (Pickles *et al.* 2001; Fergusson *et al.* 2005; Balázs *et al.* 2013). Common psychiatric disorders are now considered to be the extremes of quantitative traits (Plomin *et al.* 2009; Insel *et al.* 2010) and there is evidence that differently defined internalizing problems have the same aetiology (Kendler *et al.* 1987, 1992a, b). Nonetheless, reliance on self-report data may be associated with shared method variance that could inflate the correlations. Third, there was attrition in the sample – although not for internalizing symptoms. Attrition bias might complicate estimation of trait prevalence; however it is unlikely to affect the estimation of between trait associations (Wolke *et al.* 2009). Last, there are limitations inherent to the twin design, discussed comprehensively elsewhere (Plomin *et al.* 2013). These have minimal and contrasting effects on parameter estimates which should be taken as indicative rather than absolute.

Conclusions

The current study is the first to study the aetiological underpinnings of the hopelessness theory of depression, demonstrating that associations between attributional style, hopelessness and depression symptoms are largely due to shared genetic liability, suggesting developmentally stable biological pathways underpinning this influential theory. Furthermore, both attributional style and hopelessness were not related to anxiety, thus constituted unique cognitive content in depression. The results inform molecular genetics research and treatment approaches, as identifying specific cognitions in depression can inform the design of more precise clinical interventions for this disorder across development.

Supplementary material

For supplementary material accompanying this paper visit <http://dx.doi.org/10.1017/S0033291716000489>.

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Declaration of Interest

None.

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