Interpersonal cognitive biases as genetic markers for paediatric depressive symptoms: Twin data from the Emotions, Cognitions, Heredity and Outcome (ECHO) study

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Abstract

Childhood depressive symptoms may arise from genetic and environmental risks, which act to bias the ways in which children process emotional information. Indeed previous studies show that several "cognitive biases" are heritable and share genetic and environmental risks with depressive symptoms. Intriguingly, past research suggests that many cognitive biases only reflect genetic risks for depressive symptoms from adolescence. The present study aimed to identify (i) when interpersonal cognitions mature as risk factors for depressive symptoms by examining whether these factors are stable and predict symptoms across time in childhood, (ii) the extent to which interpersonal cognitions reflect inherited/environmental risks on children's depressive symptoms. Results showed that compared to age 8, interpersonal cognitive biases were becoming more stable across time (from age 8 to 10 years: r's = 0.32 to 0.43) but only the absence of positive self/other perceptions, and negative peer and mother expectations at age 8 predicted depressive symptoms at age 10 (after controlling for depressive symptoms at age 8). The absence of positive self/other perceptions shared genetic influences with depressive symptoms within and across time. Across middle to late childhood, interpersonal cognitions begin to operate as vulnerability-trait factors for depressive symptoms, gradually reflecting distal genetic risks on symptoms.

Key words: twins, genetics, depression, children, cognitive bias

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Depressive symptoms are known to be prevalent in prepubescent children (e.g. Costello, Mustillo, Erkanli, Keeler, & Angold, 2003) and can affect both social and academic development (Edelsohn, Ialongo, Werthamer-Larsson, Crockett, & Kellam, 1992) and long-term mental health (Dunn & Goodyer, 2006)). In order to develop preventative interventions to attenuate these negative outcomes, it is important to understand the mechanisms by which symptoms of depression first develop. Models aimed at explaining why some children are more prone to developing depressive symptoms than others have underscored the role of both nurture and nature. Some of the most consistent findings regarding children's depressive symptoms relate to the role of stress, with stressful life events implicated in the onset of symptoms, but chronic difficulties and adversities as significant contributors too (Eley & Stevenson, 2000). Among the set of chronic difficulties, interpersonal problems such as poor parent-child relationships and negative peer relationships have been highlighted (K. D. Rudolph, Hammen, & Burge, 1997). Alongside environmental influences, data from family and twin studies also suggest the role of some genetic contributions to depressive symptoms in children (although studies vary over how large this contribution is) (Lau, Rijsdijk, Gregory, McGuffin, & Eley, 2007; Rice, Harold, & Thapar, 2002). There is also some suggestion that genetic and environmental factors correlate and interact (Rice, Harold, Shelton, & Thapar, 2006; Wilkinson, Trzaskowski, Haworth, & Eley, 2013). However what remains unknown is *how* genetic and environmental risks on symptoms are expressed.

One possibility that we have explored in previous studies is that genetic and environmental risks for children's depressive symptoms are expressed as biases in the way in which children process information about emotional events and activities (Eley et al., 2008; Gregory et al., 2007; Lau, Belli, Gregory, Napolitano, & Eley, 2012; Lau, Rijsdijk, et al., 2007). Similar to depressed adults (Kovacs & Beck, 1978), children with mood symptoms have been reported to show biases in automatic forms of information-processing such as in

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the interpretation and attribution of the causes of ambiguous, positive and negative events (Dineen & Hadwin, 2004; Gladstone & Kaslow, 1995). Analogous biases have also been shown in cognitions, which can reflect the cognitive products of maladaptive information-processing such as in their perceptions and expectations of daily events and activities, and of other people (Gregory et al., 2007; Rudolph et al., 1997). It is plausible that this collection of "cognitive biases" is shaped by more distal sources of genetic and environmental influence. There is also some work showing that negative patterns of information-processing and negative cognitions can be acquired through exposure to (Murray, Woolgar, Cooper, & Hipwell, 2001), and learning from negative social environments (Field, 2006; Haddad, Lissek, Pine, & Lau, 2011). In contrast, there is currently little work investigating the extent to which these maladaptive information processing styles and cognitions reflect markers of inherited risks on depressive symptoms in children. Addressing whether cognitive factors can reflect genetic vulnerability is important both for understanding genetic risk mechanisms and associated pathways, but also for dissecting depressive phenotypes in children into more genetically-homogenous subgroups that can inform the search for genes in molecular genetic studies.

Only a handful of studies have explored the genetics of maladaptive information-processing style and its association with depressive symptoms in children. One study showed that negative interpretational style (the tendency to draw negative interpretations of ambiguous words or scenarios) was moderately heritable (30%) and that genetic influences on this processing style overlapped with genetic influences on depressive symptoms (genetic correlation = 0.65) (Eley et al., 2008). However, in another study of the same sample, this time measuring attributional style (the tendency to attribute positive and negative events to internal or stable, global or specific and stable or unstable causes), genetic influences were minimal (Lau et al., 2012). Instead across two time-points (at age 8, 10 years), a pattern of

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shared and non-shared environmental influences contributed to negative attributions. Shared environmental influences are family-wide environmental influences that contribute towards the phenotypic similarity of family members growing up in the same environment, while non-shared environmental influences are individual-specific environmental influences that contribute towards differences among family members. Interestingly, data analysis also showed that the same shared environmental influences that contributed to negative attributions also tended to contribute to depressive symptoms (via a shared latent psychometric factor) – suggesting that in this period of middle to late childhood, attributional style may reflect family-wide environmental risks, rather than inherited risks on depressive symptoms. One study examined the genetics of negative interpersonal cognitions and links with depressive symptoms in 8-year-old children (Gregory et al., 2007). Negative interpersonal cognitions may reflect the products of biased information processing. This study found that shared environmental rather than genetic influences shaped both negative and positive perceptions about the self and others, and negative expectations of both peers and parents. Moreover, like the findings on attributional style, these dysfunctional interpersonal cognitions held shared and non-shared environmental influences in common with depressive symptoms – again suggesting that these negative cognitions are likely to reflect environmental experiences of children rather than inherited risks for symptoms at age 8.

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The paucity of studies investigating whether maladaptive information-processing styles and cognitions reflect genetic or environmental risks on children's depressive symptoms makes it difficult to draw firm conclusions across studies – particularly given mixed findings across these different processing styles and cognitions, with some studies reporting modest heritability of cognitive biases, and others suggesting that biases reflect environmental experiences. Adding additional complexity is that in adolescence, some

information-processing styles – attributional style in particular – have been found to be genetically influenced and to share genetic overlap with depressive symptoms (Lau & Eley, 2008; Lau, Rijsdijk, & Eley, 2006; Zavos, Rijsdijk, Gregory, & Eley, 2010). These age differences in the findings may be because attributional style does not fully mature (i.e. is not stable, does not show trait-like qualities or act as a diathesis-stress factor) until adolescence (Cole & Turner, 1993; Turner & Cole, 1994). As such, any mediation of genetic risk on depressive symptoms may be contingent on the developmental maturity of the specific process under assessment. In comparison, as interpretational style has been found to operate as a trait-vulnerability factor for emotional symptoms in early childhood (Pass, Arteche, Cooper, Creswell, & Murray, 2012; although see Haller et al., in press for the developmental timecourse of interpretational style), it may be more likely to reflect inherited risk for depressive symptoms from a young age, consistent with the findings from genetic designs. It is as yet unclear when negative interpersonal cognitions mature as stable, vulnerability factors for depressive symptoms, and if this has implications for whether these reflect inherited versus environmental risks. The present study addresses these questions. While the previous study (Gregory et al., 2007) analysed data from Wave 1 of the Emotions, Cognitions, Heredity and Outcomes (ECHO) twin study (when children were aged 8 years), here, we combined those data with data collected when children were aged 10 years. Using the two waves of data together, we first examine the extent to which these interpersonal cognitions are 'mature'. We explore this using two indices of developmental maturity: whether these factors showed stability across two time-points and whether these operate as trait-vulnerability factors by predicting depressive symptoms within and across (from age 8 to age 10 years). Next, we explore the extent to which these negative interpersonal cognitions reflect inherited and/or environmental risks for children's depressive symptoms across age 8 and 10.

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Methods

Sample

Data come from monozygotic (MZ) and dizygotic (DZ) twins taking part in the Emotions, Cognitions, Heredity and Outcome (ECHO) study (Eley, Gregory, Clark, & Ehlers, 2007). ECHO twins were selected from the Twins Early Development Study (Trouton, Spinath, & Plomin, 2002), a larger ongoing longitudinal sample of twins born in England and Wales during 1994-1996. The ECHO Wave 1 sample comprised 300 8-year-old twin pairs (mean age = 8.47 years, sd=0.18): 247 pairs were selected from TEDS on the basis of high parent-reported anxiety at age 7 (those scoring in the top 15% for anxiety); 53 control twin pairs not scoring in the top 15% of anxiety scores were also chosen to ensure coverage of the full range of scores on test measures. Eleven families were considered unusable at Wave 1 because of autistic spectrum disorders, severe receptive language impairments, or persistent attention problems in at least one of the twins, so only 289 families were contacted at Wave 2 approximately 2 years later (Lau, Gregory, Goldwin, Pine, & Eley, 2007). Of these, 250 families (87%) agreed to participate. Mean age of twins at Wave 2 was 10.09 years (sd = 0.26). At Wave 1, the sample was predominantly (80%) white, and all parents of children in the sample were in employment and had remained in education until the age of 18.

Data collection for ECHO took place at the Institute of Psychiatry in London, with a small number of families visited in their homes. Written informed consent was obtained from parents of all twins. Current analyses utilised Wave 1 and 2 data. Full data for all questionnaires across Waves were available for 245 twin pairs. Of these, there were 34 male MZ pairs; 48 female MZ pairs; 25 male DZ pairs; 42 female DZ pairs; and 96 opposite-sex DZ pairs. Because the ECHO sample is subject to selection biases (oversampling symptomatic children) and response biases (individuals with mothers reporting higher levels

of emotional symptoms and who experienced greater negative life events were less likely to participate at Wave 2), a weighting factor was constructed for use in subsequent analyses. This multiplied the ratio of the probability of selection of high symptom families to that of non-symptom control families, and the inverse of the predicted probability of families remaining at Wave 2 using significant predictors. By incorporating weights into analysis, parameter estimates are adjusted to reflect less weight being assigned to individuals from categories overrepresented (and greater weight to those under-represented) by the sampling process.

Measures

Depressive symptoms: These were measured using the Children's Depression Inventory (Kovacs, 1985), a 27-item self-report questionnaire adapted from the Beck Depression Inventory for use in children and adolescents. Individual items consist of three statements about the frequency with which a depressive symptom has occurred over the past two weeks, e.g. ``I am sad once in a while'' (0), ``I am often sad'' (1), and ``I am sad all the time'' (2). Total scores range between 0 and 54, with higher scores indicating higher levels of depression. One item concerning thoughts about suicide was removed from the questionnaire for ethical reasons (it was deemed inappropriate for 8-year-old children). The internal consistency (α) of the remaining items was 0.82 at 8 years and 0.80 at 10 years. The measure has demonstrated good discriminant and convergent validity in 6-16 year-olds (Hodges, 1990).

Peer perceptions: This was measured using the Perception of Peers and Self Questionnaire (Rudolph, Hammen, & Burge, 1995) which assesses children's perceptions of themselves and others in social contexts. It comprises 30 items, 15 of which concern beliefs about others (e.g. “Other kids are pretty helpful when you need them”, “Other kids can sometimes be pretty mean”) and 15 of which concern beliefs about the self (e.g. “I am a lot of

fun to be with", "It's a waste of other kids' time to be friends with me"). Children rate how much they agree with each item on 4-point Likert-like scales ranging between 1 (Not At All) and 4 (Very Much). Some items are positive ("Other kids are pretty easy to get along with"), others are negative ("Other kids are really out to get you"). Positive items are reverse-coded such that a higher total score indicates more negative perceptions. Rudolph and colleagues (1995) found test-retest reliabilities of $r = .69$, $p < .0001$ for both other and self sub-scales over a 1-month period, and $r = .55$, $p < .005$ and $r = .60$, $p < .002$ for these same sub-scales over a 5-month period.

Social expectations: The Children's Expectations of Social Behaviour Questionnaire (Rudolph, et al., 1995) provided an index of children's expectations about the prospective behaviour of their mothers and peers. Children are read 30 descriptions of hypothetical interpersonal situations (15 featuring their mother and 15 featuring peers), and instructed to choose the most likely outcome from 3 alternatives for each. The 3 alternative responses include a positive or accepting behaviour; an indifferent behaviour; and a negative, hostile or rejecting behaviour. For example: "You see some kids playing a game during break one day so you go over and ask if you can play with them. What do you think they might say?". The three responses are: "They might tell me to join in and make room for me" (positive), "They might just act like I wasn't even there and keep playing" (indifferent), and "They might say mean things about me and tell me to go away" (negative). Responses are rated 0 (positive), 1 (indifferent), or 2 (negative). Summing across items, higher scores indicate more negative expectations of social partners' behaviours. The CESBQ shows high test-retest reliability in 7-12 year-olds over a 5-month period: $r = .82$ (mother items), $r = .68$ (peer items).

Analyses

Phenotypic analyses: Confirmatory factor analyses were first used to replicate whether the same four interpersonal cognitive factors identified at Wave 1 (i.e. at age 8) also characterised children at Wave 2 (i.e. at age 10). These were conducted first for one half of the sample (where only one twin from each twin-pair was selected randomly) and then conducted in the second half, such that the second half reflected an internal replication of the first set of results. Correlational analyses were performed next between Wave 1 and 2 variables to identify: (i) significant concurrent phenotypic associations between interpersonal cognitive factors and depressive symptoms at Wave 2, (ii) the stability of each interpersonal cognitive factor from Wave 1 to Wave 2, (iii) significant phenotypic associations between Wave 1 depressive symptoms and Wave 2 interpersonal cognitive factors, and (iv) significant phenotypic associations between Wave 2 depressive symptoms and Wave 1 interpersonal cognitive factors. Again, these were conducted for each half of the sample separately.

Regression analyses were then conducted to investigate the extent to which Wave 2 depressive symptoms were predicted by each Wave 1 interpersonal cognitive factors over and beyond effects of Wave 1 depressive symptoms. These analyses were conducted in the whole sample to ensure maximal power, but while controlling for non-independence of cases by clustering observations within families.

Genetic analyses: First we estimated genetic and environmental effects on Wave 2 interpersonal cognitive factors. Of note, genetic and environmental influences on Wave 1 and 2 depressive symptoms and Wave 1 interpersonal cognitive bias measures have already been reported elsewhere (Gregory et al., 2007). Estimates of genetic and environmental influences are made by comparing within-pair similarity (twin correlations) among monozygotic (MZ) twins, who share 100% of their genetic makeup, and dizygotic (DZ) twins, who share on average 50% of segregating genes. Higher MZ compared to DZ resemblance is attributed to increased genetic similarity among MZ twins and used to estimate genetic (a^2) influences that

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are additive. Within-pair similarity not due to genetic factors is assigned as shared environmental variance (c^2) contributing toward resemblance among individuals in the same family. Finally, non-shared environmental influences (e^2) create differences among individuals from the same family and are estimated from within-pair differences between MZ twins, that is, the one minus MZ twin correlations although this term also includes measurement error.

Next, we used multivariate model-fitting analyses to confirm and refine these estimates on each measure, and also to examine the extent to which genetic, shared and non-shared environmental influences between interpersonal cognitive factors and depressive symptoms overlapped within and across time. Only three of the four Wave 1 interpersonal cognitive factors significantly predicted depressive symptoms at Wave 2 in phenotypic analyses. Thus, three independent pathway models (**Figure 1**) were run to investigate the extent to which a common genetic and/or environmental factor explained phenotypic overlap between the interpersonal cognitive factor and depressive symptoms within and across time – and the extent to which each measure was explained by specific genetic and/or environmental factors. A single model including all interpersonal cognitive factors as well as depression was not run owing to power constraints due to the sample size. Parameters in multivariate models are estimated from cross-twin cross-measure covariance matrices. As can be seen from Figure 1, two sets of genetic and environmental influences are assumed: 'Common' genetic and environmental factors (A_c, C_c, E_c) contribute to all measured variables while 'Specific' genetic and environmental influences account for unique variance on each variable at each time-point (A_{sD1}, A_{sD2}, A_{sI1}, A_{sI2}, C_{sD1}, C_{sD2}, C_{sI1}, C_{sI2}, E_{sD1}, E_{sD2}, E_{sI1} and E_{sI2},,). Total genetic, shared and non-shared environmental effects on each individual measure can be obtained by summing all specific genetic and environmental paths to that measure with common genetic and environmental influences.

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In order to maximise the sample, raw data were modelled. To assess model-fit, that is, how well observed values compare to expected values, a statistic, twice the negative loglikelihood (-2LL) of the data is produced. While -2LL does not represent an overall measure of model-fit, relative measures of fit, such as Chi-square (χ^2) can be obtained by subtracting the difference in the log-likelihood statistic of a tested model with that of a saturated model containing the same number of measured variables. A saturated model estimates the maximum number of parameters to describe variances, covariances and means of all measured variables from raw data. The lower the χ^2 relative to the degrees of freedom (i.e. a non-significant χ^2) generally indicates a better fit of the model to the data. Another consideration of model-fitting, parsimony, is to identify the best-fitting model with the fewest parameters. The Akaike's Information Criterion (AIC), which is calculated as $\chi^2 - 2df$ is an index of both goodness-of-fit and parsimony. When comparing AIC values across models, the more negative values indicate better fit relative to the number of parameters estimated. Models were fit to age-regressed and where appropriate log-transformed scores to minimize mean age effects and to correct for positive skew. Mean or variance differences between males and females identified by descriptive analyses were incorporated in genetic models.

Results

Interpersonal cognitive factors at Wave 2 (i.e. at age 10)

Confirmatory factor analyses conducted in each half of the Wave 2 twin sample showed that the factor structure at Wave 2 (i.e. at age 10) closely replicated that at Wave 1 (at age 8), with the same 4 factors emerging across waves and with similar items loading onto each (see supplementary table 1). The four factors were: Absence of Positive Peer/Self Perceptions, Negative Peer/Self Perceptions, Negative Expectations of Mother and Negative Expectations of Peers. Factor scores were generated by summing the scores of the items loading onto each factor. Each factor comprised 15 summed items and showed good

reliability with Cronbach's alphas = 0.85, 0.76, 0.78 and 0.67 respectively for Absence of Positive Peer/Self Perceptions, Negative Peer/Self Perceptions, Negative Expectations of Mother and Negative Expectations of Peers. Means and standard deviations of the 4 factors, together with depressive symptom measures at Wave 2 are reported in **Table 1**. Model-fitting analyses used to investigate mean sex differences on each measure revealed that at Wave 2, depressive symptoms and interpersonal perceptions and expectations were comparable across boys and girls. To assess whether depressive symptoms and these factor scores changed much at the mean level, Wave 1 variables are also included in **Table 1**. As can be seen mean levels were comparable across Waves.

Four sets of correlations (for each half of the sample) are presented in **Table 1**. The first set shows that all Wave 2 interpersonal cognitive bias measures correlated significantly with Wave 2 depressive symptoms suggesting cross-sectional associations between cognitions and symptoms. The second set shows significant cross-time correlations for all interpersonal cognitive bias measures from Wave 1 to Wave 2. The third set of correlations show that Wave 1 depressive symptoms (mostly) significantly correlated with all Wave 2 interpersonal cognitive bias measures. The last set of correlations show that Wave 1 interpersonal cognitive bias measures significantly correlated with Wave 2 depressive symptoms.

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Regression analysis to identify significant early predictors of Wave 2 depressive symptoms

Three of the four interpersonal cognitive factors at Wave 1 (absence of positive peer/self perceptions, negative expectations of peers and negative expectations of mothers) significantly predicted unique variance in depressive symptoms at Wave 2, over and beyond Wave 1 depressive symptoms in regression analyses (**Table 2**). Negative peer/self perceptions predicted depressive symptoms at Wave 2 at trend-level significance.

MZ and DZ twin correlations

MZ and DZ correlations for each Wave 1 and 2 measure are presented in **Table 3**. In Wave 1, depressive symptoms reflected a combination of modest genetic influences, shared environmental influences and largely non-shared environmental influences. As can be seen, the pattern of twin correlations for Wave 2 depressive symptoms support mainly shared environmental and non-shared environmental influences. For the *absence of positive peer/self perceptions*, both Wave 1 and Wave 2 twin correlations suggest modest heritability but again shared environmental and non-shared environmental contributions. While Wave 2 *negative peer/self perceptions* and *negative peer expectations* support modest heritability, Wave 1 twin data on these variables suggest mainly shared and non-shared environmental influences. For *negative mother expectations*, MZ and DZ twin correlations suggested mainly shared and non-shared environmental effects.

Genetic analysis to identify genetic influences on interpersonal cognitive factors and the genetic overlap with depressive symptoms across time

Parameter estimates from three independent pathway models are presented in **Table 4**. All models fit well, as indexed by the Root Mean Square Error of Approximation (RMSEA; Steiger, 1990; Steiger & Lind, 1980) and the Akaike Information Criterion (AIC; Akaike, 1974; 1987). All RMSEA values were =<.03, and all AIC values were < -29 – see **Table 4** for full fit statistics. Estimates of heritability, shared and non-shared environmental effects can be calculated by summing the appropriate common and specific factors together. Across all three models, adding up the estimates for common and specific genetic, shared and non-shared environmental effects for *Wave 1 depressive symptoms* estimates for total genetic influence varies from 0 to 0.33 and total shared environmental effects from 0.07 to 0.26.

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These estimates are similar to the ones reported in our previous paper. Again summing the common and specific genetic estimates for *Wave 2 depressive symptoms* yields similar total estimates that vary between 0.04 and 0.24 (falling within the 95% confidence interval estimates made in any one model), while total shared environmental effects are estimated between 0.16 and 0.29. At first glance these estimates appear to be discrepant to the MZ and DZ twin correlations, which suggest no genetic influence (**Table 3**). However, multivariate models also draw on cross-twin cross-measure MZ and DZ correlations (supplementary Table 2), these supply additional information that contribute to the heritability estimates of each individual measure. For example, it can be seen that correlations between Wave 2 depressive symptoms for twin 1, and Wave 1 depressive symptoms for twin 2, the MZ and DZ correlations are: 0.33 and 0.25. As the MZ cross-twin cross-measure correlation is larger than the corresponding DZ correlation, this suggests that some genetic influence is likely to contribute towards the relationship between these two measures (i.e. depressive symptoms at Wave 1 and Wave 2). Similarly, the MZ and DZ cross-twin cross-measure correlations for Wave 2 depressive symptoms and absence of positive self/other perceptions are 0.23 and 0.13. Again, the larger MZ correlation suggests the role of genes in explaining the phenotypic correlation between these measures. These additional pieces of information thus indirectly implicate genetic influences to each measure alone – explaining the discrepancy in the genetic estimates derived from multivariate models and the univariate MZ and DZ twin correlations.

The *absence of positive peer/self perceptions* measures support heritability at Wave 1 and Wave 2 (with total genetic estimates of 0.25 and 0.13) and non-shared environmental influences, again at both waves (with total estimates of 0.73 and 0.87). A similar pattern arose for *negative peer/self perceptions* measures, with total genetic estimates of 0.26 and 0.32 at Wave 1 and Wave 2 respectively and total non-shared environmental influences of

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0.74 and 0.64. Total shared environmental influences were minimal for both these interpersonal cognitive bias measures. *Negative mother expectations* showed no genetic effects at Wave 1 or Wave 2 (total estimates of 0 and 0), but rather supported modest shared (total estimates of 0.08 and 0.18) and large non-shared environmental (total estimates of 0.92 and 0.82) effects. Finally, for *negative peer expectations* genetic influences appear to play a modest role at Wave 2 (total estimate of 0.32), but not at Wave 1 (total estimate of 0.02), where shared environmental influences are relatively more important (total estimate of 0.30). Total non-shared environmental effects are estimated at 0.68 and 0.62 for this variable at Wave 1 and 2 respectively.

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In terms of common and specific genetic and environmental influences, significant common genetic effects were found between absence of *positive peer/self perceptions* and depressive symptoms within and across time, with some support also for common non-shared environmental influences. Specific non-shared environmental influences contributed to each variable too. Similarly, *negative peer/self perceptions* also shared common genetic influences with depressive symptoms within and across time, which largely accounted for common variance between these variables. Specific non-shared environmental influences were also important. *Negative peer expectations* and *negative mother expectations* showed a more distinct profile of effects, with no support for common genetic influences across measures. Instead, common variance across measures seemed to be explained by common shared and non-shared environmental influences. Again, specific non-shared environmental influences were generally apparent on each measure.

Discussion

In the present study, we first sought to examine whether interpersonal cognitive bias factors previously linked to internalising symptoms in children were maturing as stable, trait-like vulnerability factors to predict depressive symptoms within and across time. Cross-time

correlations between waves showed that all four interpersonal cognitive factors were moderately stable across time. Mean levels of these four factors were also comparable across waves. Although all four factors at Wave 1 correlated significantly with Wave 2 depressive symptoms, only three (absence of positive peer/self perceptions, negative peer expectations and negative mother expectations) significantly predicted depressive symptoms across time, over and beyond their associations with Wave 1 depressive symptoms. Second, we investigated the genetic and environmental contributions to these interpersonal cognitive factors and specifically whether they shared common genetic and environmental variance with depressive symptoms. Positive peer/self perceptions and negative peer/self perceptions were heritable and moreover, shared genetic risks with depressive symptoms. In contrast, negative peer and mother expectations generally reflected overlapping shared and non-shared environmental influences on depressive symptoms.

Drawing on the sparse data in this area, we had speculated that maladaptive information-processing styles and cognitions may only come to mediate inherited risks once they are developmentally-mature. Such a trend seems to characterise attributional style, which has been shown to only operate as a diathesis-stress factor for depressive symptoms in adolescence and not in childhood (Cole & Turner, 1993; Turner & Cole, 1994) – and to only reflect genetic risks from adolescence onwards (Lau & Eley, 2008; Lau, et al., 2006) and not in childhood (Lau, et al., 2012). Similarly, interpretational style, which may act as an early familial precursor for social anxiety symptoms, present in at-risk infants (Pass, et al., 2012), also shows (moderate) heritability in childhood (Eley et al., 2007). In comparison, negative cognitions such as perceptions and expectations of daily events and activities and of other people show minimal genetic influences in childhood, and according to our previous study, reflect recent environmental experiences instead (Gregory et al., 2007). It may then be that these negative cognitions have not yet begun to develop trait-like qualities in middle

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childhood. By including an additional wave of data two years later, we were able to show across ages 8 and 10 years that preadolescent children's positive and negative perceptions of the self and other people, and negative expectations of peers and their mother were showing moderate (rank-order) stability. With exception to negative self/other perceptions, these variables were also showing a temporal relationship with depressive symptoms across this period of middle to late childhood. These data are consistent with other studies explaining why preadolescent children with internalising symptoms might attract and respond differently to negative stressors such as peer rejection (Caldwell, Rudolph, Troop-Gordon, & Kim, 2004; K. D. Rudolph, et al., 1997).

If these interpersonal cognitive variables are beginning to show trait-like characteristics as risk factors for children's depressive symptoms, do they now also begin to reflect inherited risks? Our data provide tentative support for this hypothesis but only for the absence of positive self/other perceptions. This interpersonal cognitive bias factor alone was beginning to show some trait-like qualities; moderate stability, moderate temporal precedence and genetic effects that were shared with depressive symptoms within and across time. In contrast, negative perceptions of self and others showed genetic effects but their capacity to predict depressive symptoms across time was mediated through their concurrent effects on depressive symptoms. Negative expectations of peers and mothers while showing the capacity to predict depressive symptoms, did not show heritability or shared genetic effects with depressive symptoms. Together these data may suggest that these interpersonal cognitive biases are beginning to stabilise and may therefore only reflect genetic risks at later stages of development beyond childhood. Nonetheless, it is interesting to note that the absence of positive perceptions may emerge first as an inherited precursor of children's depressive symptoms.

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Why might maturation of particular cognitive traits elicit genetic contributions? Maturation is a gradual process involving protracted neural development but also experience-dependent pruning of these functions (Nelson, Jarcho & Lau, in press). As these developmental neural changes emerge, it is possible that the opportunity for inherited biological differences to manifest on cognition increases. Indeed, several well-known indices of cognitive ability and functioning reflect increasing heritability with age (Haworth et al., 2010). While these hypotheses on how genetic effects on cognitive processing wax and wane across development to differentially shape depressive symptoms are intriguing, more systematic investigations of cognitive vulnerability factors, their stability and heritability will have to be conducted.

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These data and conclusions are subject to a number of limitations. First, all measures here are self-report. Although there are strengths of using self-reported data particularly on internalising symptoms and cognitions concerning validity, these findings could be further strengthened with reports from peers or teachers. The exclusive reliance on self-reports may also have artificially increased associations between variables within and across time. Second, the sample size was relatively small and associated power was low, leading to wide confidence intervals that overlapped with zero among many of the parameter estimates of genetic models. Third, the cross-time correlations reported here may not be strong enough to conclude that cognitive biases are stable. The degree of the stability coefficients was moderate, indicating some degree of rank-order stability but also change. Furthermore, as only two waves of data were used in this study, it is difficult to conclude that these cognitive biases have stabilized at this stage of development. Finally, the usual caveats associated with twin analyses, such as violations of the equal environments assumption, assortative mating, and differences between twin and non-twin individuals, may collectively act to alter estimated parameters. Notwithstanding these limitations, our data provide some interesting

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findings on the role of negative cognitions – or rather the absence of positive self/other perceptions – as reflecting early inherited markers of risk for children's depressive symptoms.

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Table 1: Means (standard deviations) for Wave 2 measures, and correlations between variables at Wave 2 an
appearing in parentheses reflect internal replication in the other half of the sample; Correlations presented in
significance at p<0.05)

	Whole sample mean (SD)	Female mean (SD)
Wave 1 Depressive symptoms	10.27 (6.94)	9.87 (6.94)
Wave 1 Absence of positive peer/self perceptions	1.82 (0.52)	1.81 (0.52)
Wave 1 Negative peer/self perceptions	2.22 (0.51)	2.20 (0.50)
Wave 1 Negative expectations of mother	0.29 (0.25)	0.25 (0.22)
Wave 1 Negative expectations of peers	0.23 (0.30)	0.22 (0.32)
Wave 2 Depressive symptoms	8.22 (5.82)	7.89 (5.79)
Wave 2 Absence of positive peer/self perceptions	1.79 (0.47)	1.80 (0.47)
Wave 2 Negative peer/self perceptions	1.97 (0.47)	1.97 (0.48)
Wave 2 Negative expectations of mother	0.26 (0.22)	0.26 (0.22)
Wave 2 Negative expectations of peers	0.17 (0.23)	0.17 (0.24)
	Correlations with wave 2 depressive symptoms	Cross-time correlation from Wave 1 to Wave 2
Wave 2 Absence of peer/self perceptions	0.30 (0.39)	0.43 (0.35)
Wave 2 Negative peer/self perceptions	0.46 (0.54)	0.32 (0.26)

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Wave 2 Negative expectations of mother	0.34 (0.31)	0.35 (0.29)
Wave 2 Negative expectations of peers	0.42 (0.43)	0.33 (0.38)
Wave 1 Absence of peer/self perceptions	0.18 (0.26)	
Wave 1 Negative peer/self perceptions	0.18 (0.20)	
Wave 1 Negative expectations of mother	0.26 (0.36)	
Wave 1 Negative expectations of peers	0.27 (0.43)	

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Table 2: Regression analysis investigating the effects of Wave 1 interpersonal cognitive bias measures on de controlling for depressive symptoms at Wave 1

Model	Predictor	β	*t* statis
1	*Constant*	0.88	6.26, *p* <
	Wave 1 Depression	0.40	8.22, *p*<
	Wave 1 absence of positive perceptions	0.13	2.26, *p* =
2	*Constant*	0.91	6.08, *p* <
	Wave 1 Depression	0.40	8.14, *p* <
	Wave 1 negative perceptions	0.09	1.45, *p* =
3	*Constant*	1.81	10.41, *p* <
	Wave 1 Depression	0.25	4.27, *p* <
	Wave 1 negative expectations of peers	0.19	4.59, *p* <
4	*Constant*	1.39	9.54, *p* <
	Wave 1 Depression	0.38	7.57, *p* <
	Wave 1 negative expectations of mothers	0.17	4.00, *p* <

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Table 3: MZ and DZ twin correlations of Wave 1 and 2 variables

	MZ twin correlation	DZ twin correlation
Wave 1 depressive symptoms[a]	0.30	0.22
Wave 1 Absence of peer/self perceptions[a]	0.34	0.11
Wave 1 Negative peer/self perceptions[a]	0.07	0.08
Wave 1 Negative expectations of mother[a]	0.14	0.20
Wave 1 Negative expectations of peers[a]	0.21	0.19
W2 depressive symptoms	0.33	0.35
Wave 2 Absence of peer/self perceptions	0.12	0.07
Wave 2 Negative peer/self perceptions	0.46	0.13
Wave 2 Negative expectations of mother	0.18	0.21
Wave 2 Negative expectations of peers	0.42	0.04

[a] These data have been reported in our previous study (Gregory et al., 2007)

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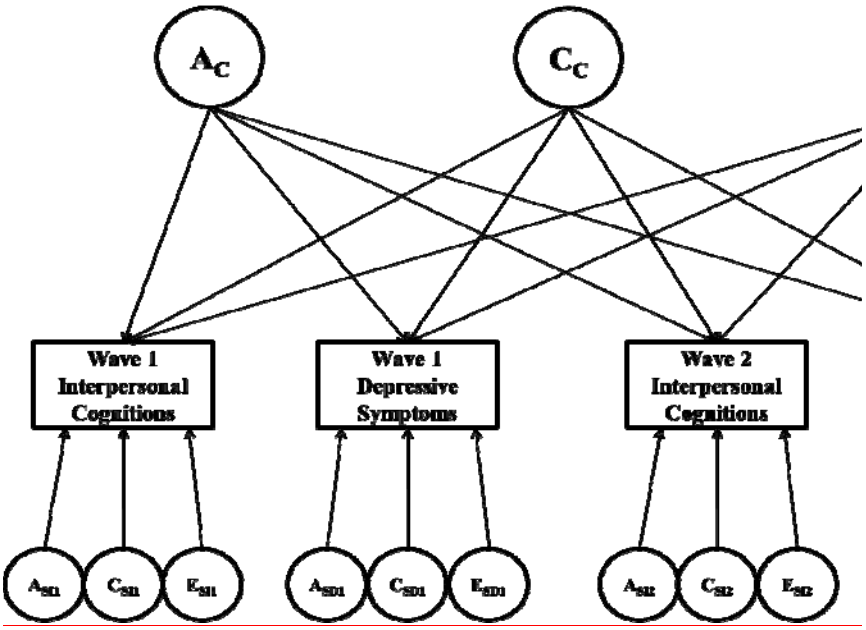
Table 4: Squared parameter estimates from Independent Pathways models of interpersonal cognitive factors over time. 95% confidence intervals are given in parentheses and significant estimates at this level are given

	Common Genetic	Common Shared Environment	Common Non-shared Environment	Specific Genetic
Model 1: Absence of Positive Perceptions of Peers and Self: -2*LL* = 4579.04 (1774); χ^2 = 226.49 (156); AIC = -85.				
Wave 1 Absence of Positive Self/Other Perceptions	**.25 (.05-.41)**	.02 (.00-.20)	**.05 (.01-.17)**	.00 (.00-.17
Wave 1 Depressive Symptoms	**.33 (.13-.49)**	.06 (.00-.23)	.00 (.00-.02)	.00 (.00-.10
Wave 2 Absence of Positive Self/Other Perceptions	**.13 (.03-.26)**	.00 (.00-.09)	**.87 (.32-.97)**	.00 (.00-.10
Wave 2 Depressive Symptoms	**.24 (.06-.45)**	.16 (.00-.35)	**.03 (.01-.10)**	.00 (.00-.10
Model 2: Negative Perceptions of Peers and Self: -2*LL* = 4559.45 (1774); χ^2 = 242.14 (156); AIC = -69.86; RMSEA				
Wave 1 Negative Self/Other Perceptions	**.26 (.14-.39)**	.00 (.00-.09)	.00 (.00-.10)	.00 (.00-.11
Wave 1 Depressive Symptoms	**.25 (.07-.43)**	**.09 (.01-.24)**	.01 (.00-.07)	.00 (.00-.15
Wave 2 Negative Self/Other Perceptions	**.21 (.03-.37)**	.03 (.00-.16)	**.09 (.04-.83)**	.11 (.00-.25
Wave 2 Depressive Symptoms	**.13 (.01-.36)**	**.24 (.04-.39)**	**.64 (.02-.76)**	.00 (.00-.08
Model 3: Negative Expectations of Mother Behaviour: -2*LL* = 4284.82 (1655); χ^2 = 282.66 (156); AIC = -80.86; RM				
Wave 1 Negative Mother Expectations	.00 (.00-.09)	.08 (.00-.21)	**.61 (.19-.99)**	.00 (.00-.19
Wave 1 Depressive Symptoms	.26 (.00-.47)	.07 (.00-.28)	**.13 (.03-.36)**	.00 (.00-.14

Wave 2 Negative Mother Expectations	.00 (.00-.27)	**.18 (.04-.31)**	**.04 (.01-.21)**	.00 (.00-.21
Wave 2 Depressive Symptoms	.18 (.00-.46)	.19 (.00-.40)	**.04 (.01-.17)**	.00 (.00-.13
Model 4: Negative Expectations of Peer Behaviour: -2*LL* = 3715.47 (1462); χ^2 = 282.66 (156); AIC = -29.34; RMSI				
Wave 1 Negative Peer Expectations	.02 (.00-.27)	**.14 (.01-.33)**	**.25 (.04-.82)**	.00 (.00-.34
Wave 1 Depressive Symptoms	.00 (.00-.47)	.26 (.00-.37)	**.22 (.01-.77)**	.00 (.00-.11
Wave 2 Negative Peer Expectations	.32 (.00-.61)	.06 (.00-.30)	.09 (.00-.47)	.00 (.00-.40
Wave 2 Depressive Symptoms	.04 (.00-.50)	**.24 (.04-.42)**	**.14 (.02-.33)**	.00 (.00-.16

Figure 1: Independent factor models investigating the extent to which each interpersonal cognitive factor sha
environmental influences (Ac, Cc, Ec) with depressive symptoms within and across time and the extent to wh
environmental influences are important (A_{sD1}, A_{sD2}, A_{sI1}, A_{sI2}, C_{sD1}, C_{sD2}, C_{sI1}, C_{sI2}, E_{sD1}, E_{sD2}, E_{sI1} and E_{sI2})



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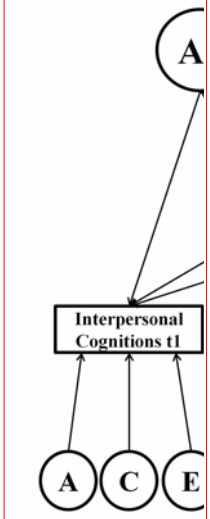

Supplementary Table 1: Factor loadings represent how questionnaire items load onto four interperson

at age 10)

Item	Positive Perceptions		Negati Percep		
Absence of Positive Perceptions of Peers and Self					
There are a lot of things about me that other kids really like	**.55**	**.46**	.13		
I am a lot of fun to be with	**.50**	**.56**	.19		
Once I am friends with someone, I know how to keep them as a friend	**.41**	**.59**	.06		
I have always been the kind of kid who makes friends really easily	**.56**	**.58**	.12		
Kids like to be around me because I can be a really good friend	**.64**	**.60**	.15		
I am good at making other kids feel better when they are upset	**.58**	**.56**	-.07		
I am good at making other kids laugh	**.48**	**.55**	-.06		
I can usually get other kids to play the games that I suggest	**.47**	**.51**	.15		
Other kids are pretty helpful when you need them	**.55**	**.51**	-.04		
Other kids are pretty easy to get along with	**.64**	**.56**	.23		

Other kids will try to cheer you up when you're upset	**.64**	**.56**	.04		
Other kids usually like you, even if you have some faults	**.54**	**.58**	.22		
Friends will take your side when other kids make fun of you	**.51**	**.52**	.22		
Once you're friends with someone they usually stay friends with you	**.60**	**.55**	.23		
Friends usually stick up for you when you are in trouble	**.56**	**.51**	.21		
Negative Perceptions of Peers and Self					
When other kids do not want to be around me, it's probably because there is something wrong with me	.10	.06	**.48**		
Sometimes I feel like I am too different from other kids	.19	.01	**.50**		
It's a waste of other kids' time to be friends with me	.25	.04	**.44**		
If another kid has something I want, I am NOT very good at getting a turn with it	.25	.22	**.37**		
I am NOT very good at getting other kids to let me join in their games	**.32**	.19	**.47**		
If another kid makes me angry or sad, I am NOT good at standing up for myself	.14	.14	.24		
If I get into a fight with another kid, I am NOT really good at ending it	.22	.23	.15		
Other kids can sometimes be pretty mean	.06	-.17	**.55**		
Other kids will try to put you down or tease you if they have a chance	.07	-.06	**.69**		

You never really know how other kids are going to act	-.12	.00	**.33**		
Other kids can not be trusted	.24	-.09	.28		
Other kids are really out to get you	.11	-.02	**.50**		
Once you get into a fight with a friend, it probably means that they will not be friends with you anymore	.16	.04	.26		
Friends often leave you out when there are other kids around to play with	.22	.02	**.49**		
Friends may gossip about you when you're not around	.09	.16	**.53**		
Negative Expectations of Mother Behaviour					
You tell your mother that you have won a prize at school	-.02	-.13	.07		
You give your mother a lopsided vase that you have made for her	-.02	-.12	-.09		
Your mother is teaching you a game, but you have difficulty understanding	-.27	.11	.00		
You return home to your mother after falling off your bike	-.16	-.13	-.12		
You tell your mother that some of the kids at school were making fun of you	.04	-.02	-.17		
You make your mother breakfast, but the toast is overdone	.04	-.04	-.23		
You ask your mother if she will take you to see a new movie	-.10	-.07	-.14		
You tell your mother that you can't finish a puzzle that you've started	.17	.12	-.24		

You have the lead role in a play but mother gets a call from a friend	.07	-.11	-.24		
You are scared in the night and wake your mother	.01	.00	.01		
You ask your mother for help with some difficult homework	.14	-.05	.04		
You're feeling yucky in the morning, and you see your mother	-.12	-.11	-.01		
You ask your mother to make something for a bake sale at school	-.15	.00	-.12		
You show your mother a test on which you didn't do very well	-.08	.06	-.13		
Your mother is going out, and you feel really ill	-.15	-.14	.16		
Negative Expectations of Peer Behaviour					
An older child picks on you in front of the kids in your class	-.11	-.21	**-.48**		
You are running for captain of your class and ask a friend to help you	-.16	.19	-.10		
You make a suggestion for a school project	-.29	-.16	**-.32**		
You go to school, and it's your birthday	-.18	-.13	-.14		
You're upset by something that happened at home and try to talk to a friend	-.08	.01	-.01		
You go to your friend's party and give them a present	.09	.06	-.03		
You ask another child to come to your house	**-.30**	.02	-.19		
You are playing a game in the playground and drop the ball	-.12	-.19	**-.33**		

You ask some other children if you can play with them	.03	-.10	**-.31**		
You get in trouble for passing a note that you didn't pass	**-.32**	-.12	-.25		
Your friend is supposed to be staying at your house but gets invited to a party	-.05	-.09	-.08		
You fall over in the playground and start crying	-.02	-.16	-.20		
Friends of a new friend start to tease you	-.19	-.03	-.08		
You are playing a game with some friends, but you keep getting the rules wrong	-.23	-.10	-.22		
You ask a friend to help you finish your science project	-.30	.13	-.01		

Supplementary Table 2: Cross-twin Cross-measure correlations for MZ and DZ twins

	MZ cross-twin cross-trait correlation
Wave 1 – Wave 2 depressive symptoms	.33
Wave 1 depressive symptoms – Wave 1 absence of peer perceptions	.15
Wave 1 depressive symptoms – Wave 2 absence of peer perceptions	.14
Wave 2 depressive symptoms – Wave 1 absence of peer perceptions	.23
Wave 1 depressive symptoms – Wave 1 negative peer/self perceptions	.07
Wave 1 depressive symptoms – Wave 2 negative peer/self perceptions	.26
Wave 2 depressive symptoms – Wave 1 negative peer/self perceptions	.06
Wave 1 depressive symptoms – Wave 1 negative expectations of mother	.18
Wave 1 depressive symptoms – Wave 2 negative expectations of mother	.20
Wave 2 depressive symptoms – Wave 1 negative	.21

expectations of mother	
Wave 1 depressive symptoms – Wave 1 negative expectations of peers	.21
Wave 1 depressive symptoms – Wave 2 negative expectations of peers	.16
Wave 2 depressive symptoms – Wave 1 negative expectations of peers	.10

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Wave 1 Depressive symptoms	10.27 (6.94)	9.87 (6.94)
Wave 1 Absence of positive peer/self perceptions	1.82 (0.52)	1.81 (0.52)
Wave 1 Negative peer/self perceptions	2.22 (0.51)	2.20 (0.50)
Wave 1 Negative expectations of mother	0.29 (0.25)	0.25 (0.22)
Wave 1 Negative expectations of peers	0.23 (0.30)	0.22 (0.32)

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Wave 1 Depressive symptoms	10.27 (6.94)	9.87 (6.94)
Wave 1 Absence of positive peer/self perceptions	1.82 (0.52)	1.81 (0.52)
Wave 1 Negative peer/self perceptions	2.22 (0.51)	2.20 (0.50)
Wave 1 Negative expectations of mother	0.29 (0.25)	0.25 (0.22)
Wave 1 Negative expectations of peers	0.23 (0.30)	0.22 (0.32)

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	MZ twin correlation	DZ twin correlation
Wave 1 depressive symptoms[a]	0.30	0.22
Wave 1 Absence of peer/self perceptions[a]	0.34	0.11
Wave 1 Negative peer/self perceptions[a]	0.07	0.08
Wave 1 Negative expectations of mother[a]	0.14	0.20
Wave 1 Negative expectations of peers[a]	0.21	0.19
W2 depressive symptoms	0.33	0.35
Wave 2 Absence of peer/self perceptions	0.12	0.07
Wave 2 Negative peer/self perceptions	0.46	0.13
Wave 2 Negative expectations of mother	0.18	0.21
Wave 2 Negative expectations of peers	0.42	0.04

[a] These data have been reported in our previous study (Gregory et al., 2007)