Parental criticism and adolescent internalising symptoms: Associations remain after accounting for shared genetic effects

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Ethical approval
Prior to the initiation of this study, the TOSS and TCHAD projects received ethical approval from the Institutional Review Boards at of the home institutions concerned: The Pennsylvania State University, USA, and Karolinska Institutet, Sweden.
Abstract

Objective
Parental criticism is associated with internalising symptoms in adolescent offspring. It is unclear whether these behaviours cause one another, and/or whether they are influenced by shared genes in related parent-offspring pairs. We use an Extended Children of Twins design to assess whether parent-reported criticism and offspring internalising symptoms remain associated after controlling for shared genes. To aid interpretation of our results and those of previous Children of Twins studies, we examine statistical power for the detection of genetic effects and explore the direction of psychosocial influences between generations.

Method
Data were drawn from two Swedish twin samples, comprising 876 adult twin pairs with adolescent offspring and 1030 adolescent twin pairs with parents. Parents reported on criticism towards their offspring, concurrently with parent and offspring reports of adolescent internalising symptoms. Extended Children of Twins structural equation models were used to examine intergenerational social and genetic mechanisms.

Results
Parental criticism was associated with adolescent internalising symptoms after controlling for genetic relatedness. No significant role was found for shared genes influencing phenotypes in both generations. Power analyses confirmed that any undetected genetic effects were small. Models could not distinguish the causal direction of possible psychosocial effects between generations.

Conclusion
The association between parent-reported criticism and adolescent internalising symptoms is not attributable to genetic confounding in this sample. As such, parental criticism may be involved in psychosocial family processes in the context of adolescent internalising. Future studies should seek to identify these processes and provide clarity on the direction of potential causal effects.
Introduction

Internalising symptoms are common among adolescents worldwide. They encapsulate problems relating to anxiety, depression, somatic symptoms and withdrawal, which typically co-occur and are rarely observed in isolation from one-another. Subthreshold internalising disorders are shown to cause substantial impairment during adolescence and constitute a major public health burden. If left untreated, subthreshold internalising problems can persist into adulthood, taking a chronic course and exerting significant impact on social, emotional and economic outcomes. Previous research suggests that the parent-child relationship is important for adolescent adjustment, during formative years of increasing offspring autonomy and separation from the parent-child dyad. Accordingly, parental criticism of their adolescent offspring has previously been associated with offspring internalising symptoms and disorder status.

Parental criticism reflects a distressed, unsupportive emotional climate or interaction pattern between the parent and child, typically assessed using coded speech samples (e.g.), or parent-report questionnaires (e.g.). Associations with adolescent internalising may be driven by social interactions within families, such that negative parenting behaviours influence, or are influenced by, mental health difficulties in offspring. In other words, parental criticism may contribute to the development of internalising symptoms in offspring and/or reflect the parent’s reaction to distressing child behaviours. It is also possible that genetic factors have a role to play, but this has scarcely been acknowledged in existing psychosocial research.

All human behaviours are influenced in part by genetic factors and all humans inherit their genes from their parents. As such, genetic factors shared between parents and offspring may at least partially account for correlations in their behaviour. Recent evidence suggests that genes linked to complex behaviours tend to have highly diffuse effects that are not specific to single behaviours. This means that the same genes could influence both internalising symptoms during adolescence and parenting behaviours in adulthood. If true, then it is important for researchers and clinicians to acknowledge the role of genetic factors in their work with parents and adolescents, to better conceptualise the influence of malleable psychosocial interactions on mental health outcomes.

The relative roles of genetic transmission and psychosocial influences in families can be studied using genetically sensitive research designs. The Children of Twins design is one such example. Here, adult identical and fraternal twin pairs and their children are compared for any given set of behaviours or traits. In the families of identical twins, a niece/nephew is just as genetically related to...
their uncle/aunt as to their own parent (see Figure 1), although they share their immediate rearing environment only with their parent. This allows for estimation of environmental mechanisms within the rearing environment that influence associations between generations. Comparisons between avuncular correlations (those between children and their parent’s twin) in MZ families and DZ families allow for the estimation of genetic influences on intergenerational associations. This is because genetic correlations are .50 in MZ families and .25 in DZ families. Put simply, the Children of Twins design indicates the extent to which psychosocial mechanisms versus genetic relatedness can explain parent-child associations.

**Figure 1 here**

Existing Children of Twins research on offspring internalising has provided evidence to support a social explanation for associations with parents’ emotional overinvolvement,18 harsh punishment19 and parent-offspring relationship quality.20 Just one study has explored parental criticism specifically, in relation to offspring somatic symptoms, with results again showing support for social mechanisms.21 Of these studies, only Narusyte et al.18 examined the direction of possible psychosocial effects between generations, finding a stronger influence of child-to-parent effects. Crucially, none of these studies included an assessment of statistical power to detect an influence of shared genes, meaning that their findings are difficult to critically interpret.

Statistical power in Children of Twins designs depends on several factors, each of which can limit accuracy to detect a role of genetic transmission in parent-child associations. Factors include the magnitude of the phenotypic parent-child correlation; proportion of the correlation attributable to genetic overlap; size of the avuncular genetic correlations (.50/.25 for families of identical/fraternal twins respectively, see dashed lines in Figure 1); total sample size; ratio of identical and fraternal twin families; heritability of the parent and offspring behaviours; and power to detect heritability within each generation.22,23 These factors vary across Children of Twins analyses that use different samples to study intergenerational associations involving different behaviours. When genetic influence is not detected in underpowered Children of Twins analyses, intergenerational covariance appears attributable only to psychosocial effects. Previously published Children of Twins studies may have been underpowered, resulting in the overestimation of non-genetic effects.

We used an Extended Children of Twins design to examine whether the association between parental criticism and adolescent offspring internalising symptoms remains after controlling for genetic
mechanisms. We performed simulations to determine statistical power in our sample to detect an effect of genetic transmission between generations, and explored the direction of causation for possible psychosocial effects between generations.

**Method**

Analyses involved combining data across two samples: (1) a Children of Twins sample where the twins were adult parents with adolescent offspring, and (2) a sample of adolescent twin pairs with one parent per pair. The adolescent twin sample increased our power to estimate heritability of adolescent internalising symptoms. It also increased the number of parent-child correlations going into the model, providing two estimates per twin pair.

**Children of Twins sample.** Data were drawn from the Twin and Offspring Study in Sweden (TOSS), comprising 387 identical (monozygotic, MZ) and 489 fraternal (dizygotic, DZ) twin families. Twin families consisted of same-sex adult twin pairs (mother-mother or father-father), with one genetically related adolescent child per twin. Sample eligibility required that each twin had been cohabiting with a partner (usually spouse) for a minimum of five years. Cousins (the offspring) were required to be the same sex and not differ in age by more than four years. Mean ages were 44.8 years for twins (SD=4.9; range 32-60) and 15.7 years for offspring (SD=2.4; range 11-22). 37% of twins (parents) and 52% of offspring were male.

**Adolescent twin sample.** Data were drawn from Wave 3 of the Swedish Twin Study of Child and Adolescent Development (TCHAD). Wave 3 was chosen as this contained the same offspring outcome variable and most closely matched adolescent ages compared to the TOSS. The sample comprised 416 MZ adolescent twin pairs and 614 DZ pairs (299 same-sex DZ pairs). One parent was included per pair of twin offspring. Adolescents in the TCHAD sample had a mean age of 16.7 years (SD=0.47; range 15-17). 49% of twins (adolescents) and 13% of parents were male.

**Measures:**

**Parental Criticism.** Parents reported on their critical perceptions of their adolescent offspring, using the validated 10-item critical remarks subscale of the Expressed Emotion measure. Self-reported questionnaires of parental criticism are useful in research to assess the parent’s own view of their feelings and experiences with the child. They cost less to collect and code compared to interview
assessments. Here, parents responded using a five-point Likert scale to indicate how often they agreed with each critical statement, e.g., “S/he makes me irritated”, “It is hard for us to get along” and “I find faults with him/her”.

**Adolescent Internalising symptoms.** Adolescent internalising symptoms were reported by parents and adolescents using the 30-item Internalising scale of the Child Behaviour Checklist and Youth Self-Report, respectively. These corresponding parent and child assessments use the same three-point Likert scale and were moderately correlated in both samples (see Table 1). The broad-band Internalising scale is the sum of the narrow-band syndrome scales for anxious/depressed, somatic and withdrawn behaviours, which share most of their genetic aetiology during adolescence. Evidence suggests that both parent and adolescent reports can accurately capture adolescent internalising phenotypes from different perspectives, with different biases associated with each method. We derived composite scores from a mean of parent and adolescent reports, to incorporate all available information. Using parent reports for both the parent and adolescent measure could inflate the phenotypic parent-offspring correlation, via shared method variance. To address this, we also conducted supplementary analyses using only self-reports for the adolescent phenotype.

**Table 1 here**

**Analysis:**

Residuals were taken to control for child age and sex prior to analyses. All variables were log transformed to correct for skew and standardized prior to model fitting. Structural equation models were fitted to the data using maximum likelihood estimation in the R (version 3.6.1) programme OpenMx (version 2.15.5). Combining data from both samples provides estimates for three different sets of influences, derived concurrently from a single model: (1) genetic and environmental influences on parental criticism, (2) covariance between the generations that is attributable to genetic relatedness versus non-genetic influences (which includes social effects), and (3) genetic and environmental influences on offspring internalising symptoms.
Genetic and environmental influences on parental criticism were estimated using twin pairs in the parent generation. Specifically, we quantified additive genetic (A), common environmental (C; which make family members similar to one another) and non-shared environmental (E; which make family members different to one another) effects. As illustrated in the upper half of Figure 2a, within-pair resemblance for parental criticism (i.e. criticism of their offspring) in MZ twin pairs results from both additive genetic and common environmental influences (A+C). In contrast, within DZ families, the parental twin correlation reflects their lower level of genetic resemblance (.5*A+C). This difference allowed us to estimate all genetic and environmental parameters on parental criticism, as in the regular twin design.

**Figure 2 here**

The key information for decomposing intergenerational covariance comes from the contrast in the avuncular (uncle/aunt-child) and parent-child correlations in families of MZ and DZ twins with children. Genetic sharing in the avuncular association in MZ families is equal to parent-child genetic sharing (Figure 1, rA=0.5). In contrast, genetic sharing in the avuncular association in DZ families is just 0.25. Comparing the magnitude of MZ and DZ avuncular associations provides the means to estimate genetic influence on the intergenerational association, which occurs as a result of genetic transmission from parents to offspring. Similarly, the contrast between the parent-child and the avuncular relationships provide the means to estimate the influence of the immediate family environment on the association between the generations (which includes social transmission, in either direction between generations). In the structural equation model, genetic transmission is estimated via the A1’ pathway, indexing genetic covariance between the parent and child behaviours. Any residual intergenerational association is estimated via the p pathway, capturing social transmission plus the effects of unmeasured confounders.

Comparing correlations between the offspring of MZ and DZ twins (i.e. cousins) allows for the estimation of genetic and non-shared environmental effects on offspring internalising symptoms. Since estimation of genetic effects relies upon comparisons between cousins in MZ families (lower half of Figure 2A, rA=.25) and cousins in DZ families (rA=.125), small genetic influences may not be identified between pairs with low genetic sharing, due to low power. Because cousins do not share a home environment, it is not possible to estimate the impact of the shared environment (typically defined as influences shared by siblings) on offspring phenotypes using these data. Therefore, the adolescent twin dataset was used to additionally inform on the aetiology of offspring.
internalising symptoms. Greater genetic resemblance between twin pairs means that the adolescent twin data has greater power to detect genetic effects than cousin data. It also allows for the estimation of shared environmental effects on the offspring phenotype.

Parental criticism in the adolescent twin dataset contributed to the estimation of the phenotypic parent-child correlation (Figure 2B-C). These data did not contribute directly to the estimation of parent aetiology or decomposition of the parent-child correlation. The same parent reported on their parenting of each twin; therefore, A and C correlations were fixed to unity for parents in the adolescent twin sample. Our approach is similar to that taken in previous studies using Extended Children of Twins models e.g.18, but distinct for two reasons. First, we specified data from DZ and MZ adolescent twins separately, to reflect the fact that MZ twins each inherit the same 50% of their parent’s DNA (Figure 2B), whereas DZ twins each inherit a random 50% (Figure 2C). Second, we included parameters to allow for differences in the parenting of adolescent twins by a single parent.22 These parameters (rEmz and rEdz) allow the within-parent correlations to differ between parents of MZ and DZ adolescent twins, and from the MZ correlation for adult twin parents (see differences in E1 specification across models in Figure 2, more details in supplementary materials).

The significance of pathways in our complete model (i.e., combining specifications across Figure 2) was tested by creating sub-models in which paths were consecutively fixed to zero. $\chi^2$ difference tests and Akaike’s Information Criterion (AIC) were used to assess whether sub-models yielded significantly worse fits to the data than the full model. All models were re-run using adolescent self-reports alone for child internalising symptoms.

**Power to detect the A1’ pathway.** As previously introduced, accurate estimation of A1’ (genetic influence on the parent-child correlation) relies upon adequate statistical power. Estimation of $p$ may be inflated if power to detect A1’ is inadequate, because $p$ is the residual parent-offspring covariance after accounting for genetic factors in A1’. With this in mind, we examined statistical power in our sample to detect an A1’ pathway of varied magnitudes. We simulated data to match our sample characteristics and systematically altered the strength of the A1’ pathway, reporting the corresponding change in our observed statistical power to detect it. We repeated these analyses for models using only adolescent self-report for child internalising problems, where the phenotypic parent-offspring correlation was lower.
Directionality of the $p$ pathway. The $p$ pathway captures the effects of social relationships and unmeasured confounding on the parent-child correlation. The $p$ pathway has traditionally been modelled as running from parent-to-child, although child-to-parent effects will also be captured. In our final analyses we tested a reciprocal causation version of the Extended Children of Twins model, to examine the direction of causation for intergenerational effects indexed by the $p$ pathway. We used the model introduced by Narusyte et al.,\textsuperscript{18} introducing some changes to correct model specification. Further details are provided in the supplementary materials.

Results

Table 1 presents descriptive statistics for parental criticism and adolescent internalising symptoms. Table 2 presents the parent-offspring correlation for MZ and DZ families. The phenotypic intergenerational correlation was .29 between self-reported parental criticism and the composite score for adolescent internalising symptoms, estimated using data from both samples.

**Table 2 here**

The different sets of within-pair correlations (twin pair, parent-child and avuncular) underpin the estimation of the parameters of the Children of Twins model and provide three types of information (Table 2). First, regarding parental criticism in the Children of Twins sample, the correlation between reports for MZ twin parents (.26) was greater than the correlation between DZ twin parents (.14), suggestive of parent genetic influence on parental criticism. Second, the parent-child correlations (.29, set to be equal across both family types) were more than twice the magnitude of the avuncular correlations for both MZ (.12) and DZ (.06) twin families. As such, the association was stronger between adults and adolescents who live together than those who do not, supporting a role for the social family environment in the association. The difference in the avuncular association for MZ versus DZ twin families suggests genetic factors influence the intergenerational association, although these correlations were smaller than the parent-child correlation. Third, the MZ cousin (.17) and twin (.60) correlations for internalising symptoms were approximately twice that of the DZ cousin (.09) and twin (.31) correlations, indicating genetic influences on adolescent internalising symptoms. The described pattern of results was consistent across models using the self-report approach for measuring child internalising symptoms (Table S1).

**Table 3 here**
**Model Fitting.** Full model results are depicted in Figure 3A. Factors indexing the common environment within each generation (C1 and C2) did not significantly account for measure variance in either generation, so were dropped from the model (Figure 3B). Exclusion of these parameters resulted in a more parsimonious model with no significant changes to model fit (Table 3). The heritability of parental criticism and adolescent internalising symptoms were estimated at 26% and 53% respectively. For the intergenerational association, the genetic transmission pathway (A1’) was not significantly different from zero (β=.05, 90% CI .00-.41). In contrast, the residual intergenerational pathway (p) was positive and significant (β=.24, 90% CI .18-.30). Subsequent nested models reinforced this observation (Table 3 shows model fit comparisons). Specifically, the association between parental criticism and adolescent internalising symptoms could be adequately explained by a model excluding passive genetic transmission, but not by a model including only passive genetic transmission. Results were consistent when using the self-report approach for measuring child internalising symptoms (Figure S1).

**Figure 3 here**

**Power to detect the A1’ pathway.** Table 4 presents results from power analyses tailored to our sample characteristics, exploring power to detect genetic transmission of varying magnitudes. There was 82% power to detect an A1’ estimate of .11 or greater, when parent and child heritability estimates are specified at .26 and .53 respectively. An A1’ estimate of .11 would account for 29% of the phenotypic parent-child correlation. Therefore, in these data, results suggest that at least 71% of the association between parental criticism and offspring internalising symptoms is not attributable to genetic transmission in this sample. The small A1’ estimate in Figure 3B could be a true finding for the role of genetic transmission (accounting for 13% of the phenotypic parent-child correlation), but statistical power is not sufficient to confirm this.

Statistical power was lower in models using only self-report data for adolescent symptoms, owing to the weaker phenotypic parent-child correlation. In these analyses, there was 82% power to detect an A1’ estimate of .13, accounting for 47% of the phenotypic parent-child correlation (Table S3). Therefore, in these data, results suggest that at least 53% of the association between parental criticism and adolescent-reported internalising symptoms is not attributable to genetic transmission.

**Table 4 here**
Directionality of the p pathway. To explore the direction of possible psychosocial effects between parental criticism and adolescent internalising symptoms, we applied a reciprocal causation version of the Extended Children of Twins model to our data. In this instance we were unable to distinguish parent-to-adolescent from adolescent-to-parent effects. Details on the model fitting process, results and discussion of these analyses are included in the supplementary materials.

Discussion

Our analyses reveal that parent reported criticism towards their adolescent child remains significantly correlated with adolescent internalising symptoms after controlling for genetic relatedness. Although both the parent and offspring behaviours were heritable, our results suggest that genetic overlap between the two could not explain their association. Specifically, we show that at least 71% of our reported parent-offspring association exists independently of shared genes and may arise from mechanisms within the immediate family. Statistical power was insufficient to prove that shared genes had a null effect in this sample. Overall, results confirm that adolescence may be an important period for family-based intervention, to interrupt possible psychosocial processes associated adolescent internalising symptoms and prevent their persisting into adulthood.

Results from previous Children of Twins studies examining parenting behaviours and offspring internalising outcomes support a role for social transmission in associations with parents’ harsh punishment, parent-offspring relationship quality, and parental criticism. However, these studies did not include power analyses for the detection of genetic transmission. We demonstrate that our key finding (i.e., that the association between parental criticism and adolescent internalising symptoms remains significant after controlling for genetic relatedness) is not attributable to a lack of statistical power. Given that previous studies have used either the same sample as us, or samples of a similar size, our power analyses indicate that their results are likely valid, although small effects genetic transmission may have gone undetected. We suggest that the presentation of power analyses in future publications using the Children of Twins design will aid interpretation of findings (see McAdams et al. for methodology). It is also important to explore how decisions regarding methodology (e.g., selection of measurement tools) may impact on statistical power and interpretation of results.
We maximised on data drawn from each sample using a composite measure of adolescent internalising symptoms, incorporating both parent and offspring reports. Parent and offspring reports were moderately correlated. Their partial agreement could reflect both shared perspectives and unique insights, alongside perceptual and reporting biases. For example, adolescents may report on symptoms that they have not disclosed to parents, while parents may identify symptoms that adolescents do not identify or wish to confront. However, using parent reports for parent and child traits introduced the risk that shared method variance could inflate parent-child correlations. Supplementary analyses using only adolescents’ self-reports of internalising symptoms showed a consistent pattern of findings to our primary analyses, although the phenotypic parent-child correlation was attenuated (.29 to .18). This attenuation reduced statistical power to identify genetic transmission (resulting in 80% power to detect a genetic effect accounting for 50% to of the phenotypic parent-child association, rather than 30%). Crucially, results from the more conservative supplementary analyses were in keeping with our primary results, suggesting that shared genetic influences do not explain the bulk (i.e., more than half) of the parent-offspring association.

Overall, our findings reinforce results from existing psychosocial study literature, where associations between parental criticism and child internalising have been studied without controlling for the role of shared genes. Our findings could have implications for interventions, as social and environmental mechanisms can be directly targeted by mental health practitioners. If parent-to-offspring effects are operating, then targeting parental criticism could lead to a reduction in adolescent internalising symptoms. On the other hand, if adolescents are eliciting criticism from their parents as a result of their own symptoms, then focussing on treating the adolescent’s symptoms could lead to changes in parental criticism. In the present study, it was not possible to identify the direction of effects. This could be because both parent-to-child and child-to-parent effects are operating in tandem. Or, unmeasured factors in the family environment could concurrently influence behaviours in both generations, for example sibling or spouse behaviours or socio-economic stressors.

Three complementary study designs could help to identify parent-to-child and child-to-parent effects. First, child or adolescent twin studies can provide information about the extent to which child/adolescent genes impact on the parenting that they receive, shedding light on evocative (child-to-parent) processes. A meta-analysis of child twin data identified significant effects of child genes (23-32%) on maternal negativity. This suggests that genetically influenced child behaviours evoke responses from parents via social mechanisms. Results from our adolescent twin sample provided
some evidence for an adolescent-to-parent process, as parent self-reports of criticism towards adolescent twins were almost twice as correlated for MZ (.73) versus DZ (.43) twin pairs. One bivariate child twin study showed that the same child genes influencing child anxiety also influence maternal control at age eight, suggesting that child anxiety may elicit maternal control at this age. Genetic influence from the parent genome on parenting behaviours is confirmed in Children of Twins studies, highlighting that parent-to-child effects are also possible (as suggested in the present study and others; e.g.20,21). To date, the only Children of Twins study to directly examine the direction of causation between negative parenting and adolescent internalising problems found evidence for child-based evocative effects, although we present supplementary information to detail omissions in their model specification. Together, twin findings support the possibility of transactional associations between adolescent internalizing symptoms and parental criticism.

Second, longitudinal studies using repeated assessments of parental criticism and adolescent internalising symptoms have generally identified transactional associations, with the possibility that child-to-parent effects are stronger. For example, one study with six time-frames during adolescence found stronger associations between offspring emotional symptoms and subsequent parent-reported criticism (using similar questionnaire items as in this study), compared to the reverse. Previously it has been shown that stability in the home environment, whilst modest, arises largely from parent-driven processes and family-wide effects, whereas child-driven effects are more associated with change over time. Furthermore, whilst the association between aspects of the home environment and offspring depression remains relatively stable from childhood into adolescence, the role of offspring genetic influences increases in adolescence. This indicates that adolescents influence their environments, and thus the parenting they receive, to a greater degree than is seen in younger children.

Third, studies that take an experimental or intervention-based approach can help to delineate parent-to-child and child-to-parent effects. For example, an experimental study showed that parental displays of controlling behaviours left children feeling less capable in a given task, with this effect moderated by child trait anxiety. Others found that a parenting skills training could reduce internalizing symptoms among young offspring, suggesting parent-to-child driven effects. Another experimental study found that negative parenting was reduced following the treatment of adolescent anxiety, highlighting the presence of child-to-parent effects. As such, it is important to recognise that the direction of causation in associations between parenting and adolescent outcomes cannot be assumed to run solely from parents to offspring.
Our study is strengthened by a sophisticated, genetically informative design and power analyses. There are also limitations to consider. We could not detect passive genetic effects accounting for less than 29% of the phenotypic parent-child correlation, or less than 47% when using only self-reported adolescent symptoms. Larger samples, including multiple children per twin, could harness power to test the null hypothesis for genetic transmission. Larger samples are also needed to examine age and sex effects, which may moderate parent-child associations. It would be preferable to examine longitudinal data and include two parents per child, to provide a developmental perspective and inform on family interactions. Results should be interpreted in the context of our measures, reflecting participants’ own perceptions and opinions, subject to social desirability bias. Parent and offspring reports of parental criticism were moderately correlated \( (r=.38) \) in the TOSS, although offspring reports were not used in this study because they were not collected in TCHAD. Speech samples are considered the gold-standard for assessing parent expressed emotion (e.g.,13), however these data are rarely available with the sample sizes required in genetically-sensitive research. Whilst speech samples or other researcher-coded assessments could reduce reporter bias, their results are specific to a window of observation, which may not generalise to parents’ everyday experiences and perceptions. We reduced risk of shared method variance by including adolescents’ self-reports of internalising symptoms. We note that the association with parent criticism was reduced when using only adolescent reports of internalising, although the overall pattern of results remained consistent.

The possibility of genetic confounding has been underacknowledged in existing psychosocial studies of parenting and offspring adjustment. Genetically informative research such as ours can contextualise results from those using more traditional designs and we emphasise the importance of including power analyses in this process. Here we show that the association between parental criticism and adolescent internalising symptoms remains significant after accounting for genetic relatedness. Existing studies of parental criticism and adolescent internalising symptoms can be interpreted with greater confidence. Literature from child twin, longitudinal and experimental studies all indicate the presence of transactional effects between generations. As such, parental criticism should be considered as part of the social environment relevant to the presentation of adolescent internalising symptoms, with adolescent symptoms potentially leading to an increase in parental criticism.
References

1. Merikangas KR, Avenevoli S. Epidemiology of mood and anxiety disorders in children and adolescents. Textbook in psychiatric epidemiology. 2002;2:657-704.

2. Sallis H, Szekely E, Neumann A, et al. General psychopathology, internalising and externalising in children and functional outcomes in late adolescence. Journal of child psychology and psychiatry, and allied disciplines. 2019;60(11):1183-1190.

3. Roberts RE, Fisher PW, Turner JB, Tang M. Estimating the burden of psychiatric disorders in adolescence: the impact of subthreshold disorders. Social Psychiatry and Psychiatric Epidemiology. 2015;50(3):397-406.

4. Betts KS, Baker P, Alati R, et al. The natural history of internalizing behaviours from adolescence to emerging adulthood: findings from the Australian Temperament Project. Psychol Med. 2016;46(13):2815-2827.

5. Goodman A, Joyce R, Smith JP. The long shadow cast by childhood physical and mental problems on adult life. Proceedings of the National Academy of Sciences of the United States of America. 2011;108(15):6032-6037.

6. Shankman SA, Lewinsohn PM, Klein DN, Small JW, Seeley JR, Altman SE. Subthreshold conditions as precursors for full syndrome disorders: a 15-year longitudinal study of multiple diagnostic classes. Journal of child psychology and psychiatry, and allied disciplines. 2009;50(12):1485-1494.

7. Laursen B, Collins WA. Parent-child relationships during adolescence. In: Handbook of adolescent psychology: Contextual influences on adolescent development, Vol. 2, 3rd ed. Hoboken, NJ, US: John Wiley & Sons Inc; 2009:3-42.

8. De Goede IHA, Branje SJT, Meeus WHJ. Developmental Changes in Adolescents’ Perceptions of Relationships with Their Parents. Journal of Youth and Adolescence. 2009;38(1):75-88.

9. Nelemans SA, Hale Iii WW, Branje SJT, Hawk ST, Meeus WHJ. Maternal criticism and adolescent depressive and generalized anxiety disorder symptoms: A 6-year longitudinal community study. Journal of Abnormal Child Psychology. 2014;42(5):755-766.

10. Silk JS, Ziegler ML, Whalen DJ, et al. Expressed Emotion in Mothers of Currently Depressed, Remitted, High-Risk, and Low-Risk Youth: Links to Child Depression Status and Longitudinal Course. Journal of Clinical Child & Adolescent Psychology. 2009;38(1):36-47.
11. Tompson MC, Pierre CB, Boger KD, McKowen JW, Chan PT, Freed RD. Maternal Depression, Maternal Expressed Emotion, and Youth Psychopathology. Journal of Abnormal Child Psychology. 2010;38(1):105-117.

12. Frye AA, Garber J. The Relations Among Maternal Depression, Maternal Criticism, and Adolescents’ Externalizing and Internalizing Symptoms. Journal of Abnormal Child Psychology. 2005;33(1):1-11.

13. Asarnow JR, Tompson M, Woo S, Cantwell DP. Is expressed emotion a specific risk factor for depression or a nonspecific correlate of psychopathology? J Abnorm Child Psychol. 2001;29(6):573-583.

14. McCarty CA, Lau AS, Valeri SM, Weisz JR. Parent-child interactions in relation to critical and emotionally overinvolved expressed emotion (EE): is EE a proxy for behavior? J Abnorm Child Psychol. 2004;32(1):83-93.

15. Hughes EK, Gullone E. Internalizing symptoms and disorders in families of adolescents: A review of family systems literature. Clinical Psychology Review. 2008;28(1):92-117.

16. Plomin R, DeFries JC, Knopik VS, Neiderhiser JM. Top 10 Replicated Findings From Behavioral Genetics. Perspectives on Psychological Science. 2016;11(1):3-23.

17. McAdams TA, Neiderhiser JM, Rijsdijk FV, Narusyte J, Lichtenstein P, Eley TC. Accounting for Genetic and Environmental Confounds in Associations Between Parent and Child Characteristics: A Systematic Review of Children-of-Twins Studies. Psychological Bulletin. 2014;140(4):1138-1173.

18. Narusyte J, Neiderhiser JM, D'Onofrio BM, et al. Testing different types of genotype-environment correlation: an extended children-of-twins model. Dev Psychol. 2008;44(6):1591-1603.

19. Lynch SK, Turkheimer E, D'Onofrio BM, et al. A genetically informed study of the association between harsh punishment and offspring behavioral problems. Journal of family psychology : JFP : journal of the Division of Family Psychology of the American Psychological Association (Division 43). 2006;20(2):190-198.

20. Hannigan LJ, Rijsdijk FV, Ganiban JM, et al. Shared genetic influences do not explain the association between parent-offspring relationship quality and offspring internalizing problems: results from a Children-of-Twins study. Psychol Med. 2018;48(4):592-603.

21. Horwitz BN, Marceau K, Narusyte J, et al. Parental criticism is an environmental influence on adolescent somatic symptoms. Journal of family psychology : JFP : journal of the Division of Family Psychology of the American Psychological Association (Division 43). 2015;29(2):283-289.
22. McAdams TA, Hannigan LJ, Eilertsen EM, Gjerde LC, Ystrom E, Rijsdijk FV. Revisiting the Children-of-Twins Design: Improving Existing Models for the Exploration of Intergenerational Associations. Behavior Genetics. 2018;48(5):397-412.

23. Verhulst B. A Power Calculator for the Classical Twin Design. Behav Genet. 2017;47(2):255-261.

24. Neiderhiser JM, Lichtenstein P. The Twin and Offspring Study in Sweden: Advancing our understanding of genotype-environment interplay by studying twins and their families. Acta Psychologica Sinica. 2008;40(10):1116-1123.

25. Lichtenstein P, Tuvblad C, Larsson H, Carlstrom E. The Swedish Twin study of CHild and Adolescent Development: the TCHAD-study. Twin research and human genetics : the official journal of the International Society for Twin Studies. 2007;10(1):67-73.

26. Hansson K, Jarbin H. New self-rating questionnaire in Swedish for measuring expressed emotion. Nordic Journal of Psychiatry. 1997;51(4):287-297.

27. Hale WW, 3rd, Keijsers L, Klimstra TA, et al. How does longitudinally measured maternal expressed emotion affect internalizing and externalizing symptoms of adolescents from the general community? Journal of child psychology and psychiatry, and allied disciplines. 2011;52(11):1174-1183.

28. Achenbach TM. Manual for the Child Behaviour Checklist and 1991 profile. In: University of Vermont DoP, ed: Burlington; 1991.

29. Achenbach TM. Manual for the Youth Self-Report and 1991 profile. In: University of Vermont DoP, ed: Burlington; 1991.

30. Waszczuk MA, Zavos HM, Gregory AM, Eley TC. The phenotypic and genetic structure of depression and anxiety disorder symptoms in childhood, adolescence, and young adulthood. JAMA psychiatry. 2014;71(8):905-916.

31. Thomas AM, Forehand R, Armistead L, Wierson M, Fauber R. Cross-informant consistency in externalizing and internalizing problems in early adolescence. Journal of Psychopathology and Behavioral Assessment. 1990;12(3):255-262.

32. De Los Reyes A, Kazdin A. Informant Discrepancies in the Assessment of Childhood Psychopathology: A Critical Review, Theoretical Framework, and Recommendations for Further Study. Psychological bulletin. 2005;131:483-509.

33. Neale MC, Hunter MD, Pritikin JN, et al. OpenMx 2.0: Extended Structural Equation and Statistical Modeling. Psychometrika. 2016;81(2):535-549.
34. Avinun R, Knafo A. Parenting as a Reaction Evoked by Children's Genotype: A Meta-Analysis of Children-as-Twins Studies. Personality and Social Psychology Review. 2014;18(1):87-102.

35. Eley TC, Napolitano M, Lau JY, Gregory AM. Does childhood anxiety evoke maternal control? A genetically informed study. Journal of Child Psychology and Psychiatry. 2010;51(7):772-779.

36. Hannigan LJ, McAdams TA, Plomin R, Eley TC. Etiological Influences on Perceptions of Parenting: A Longitudinal, Multi-Informant Twin Study. Journal of Youth and Adolescence. 2016;45(12):2387-2405.

37. Hannigan LJ, McAdams TA, Eley TC. Developmental change in the association between adolescent depressive symptoms and the home environment: results from a longitudinal, genetically informative investigation. Journal of child psychology and psychiatry, and allied disciplines. 2017;58(7):787-797.

38. Thirlwall K, Creswell C. The impact of maternal control on children's anxious cognitions, behaviours and affect: an experimental study. Behaviour research and therapy. 2010;48(10):1041-1046.

39. Cartwright-Hatton S, McNally D, White C, Verduyn C. Parenting skills training: an effective intervention for internalizing symptoms in younger children? Journal of child and adolescent psychiatric nursing : official publication of the Association of Child and Adolescent Psychiatric Nurses, Inc. 2005;18(2):45-52.

40. Silverman WK, Kurtines WM, Jaccard J, Pina AA. Directionality of change in youth anxiety treatment involving parents: an initial examination. Journal of consulting and clinical psychology. 2009;77(3):474-485.
Table 1. Raw data descriptive statistics

| Sample                                      | Reporter (N items) | Chronbach’s alpha | N   | Variance | Skew | Kurtosis | Mean (SD) | Pearson’s r correlation |
|---------------------------------------------|--------------------|-------------------|-----|----------|------|----------|------------|-------------------------|
| **Parental criticism**                      |                    |                   |     |          |      |          |            |                         |
| Children of Twins (TOSS)                    | Parent (10)        | .86               | 1,721 | 27.65   | .98  | 3.83     | 17.36 (5.26) |                         |
| Adolescent Twins (TCHAD)                    | Parent (10)        | .90               | 2,112 | 34.72   | 1.07 | 3.92     | 16.85 (5.89) |                         |
| **Offspring internalising symptoms**        |                    |                   |     |          |      |          |            |                         |
| Children of Twins (TOSS)                    | Parent (30)        | .xx               | 1,706 | 17.76   | 2.17 | 10.52    | 3.83 (4.21)  | \(r = .36\)             |
|                                            | Child (30)         | .86               | 1,669 | 43.78   | 1.35 | 5.30     | 8.70 (6.62)  |                         |
|                                            | Composite          | --                | 1,743 | 21.43   | 1.50 | 5.87     | 6.26 (4.63)  |                         |
| Adolescent Twins (TCHAD)                    | Parent (30)        | .xx               | 2,087 | 20.29   | 2.55 | 11.30    | 3.40 (4.50)  | \(r = .42\)             |
|                                            | Child (30)         | .88               | 2,313 | 50.92   | 1.35 | 5.02     | 8.34 (7.14)  |                         |
|                                            | Composite          | --                | 2,420 | 30.03   | 1.77 | 7.46     | 6.16 (5.48)  |                         |

\(^a\) Expressed Emotion measure. \(^b\) Child Behaviour Checklist. \(^c\) Youth Self-Report.
**Table 2.** Correlations in monozygotic (MZ) and dizygotic (DZ) families

|                        | MZ twin families | DZ twin families |
|------------------------|-----------------|-----------------|
| **Parental criticism** |                 |                 |
| Twin parent correlations on parental criticism for adolescent offspring | .26 | .14 |
| **Parental criticism and offspring internalising symptoms** |                 |                 |
| Parent-child correlation | .29 | .29 |
| Avuncular correlations | .12 | .06 |
| **Offspring internalising symptoms** |                 |                 |
| Cousin correlations on internalising symptoms | .17 | .09 |
| Twin correlations on internalising symptoms | .60 | .31 |

*a* Correlations taken from TOSS, the Children of Twins sample. *b* Correlations taken from TCHAD, the child twin sample.

**Table 3.** Model fit statistics

|                        | -2LL | df  | AIC  | \( \chi^2 \) | \( \Delta \) df | \( p \) | \( \chi^2 \) | \( \Delta \) df | \( p \) |
|------------------------|------|-----|------|--------------|-----------------|--------|--------------|-----------------|--------|
| Full model (ACE model) | 20361| 7545| 5271 | -            | -               | -      | -            | -               | -      |
| No C paths (AE model)  | 20361| 7547| 5267 | .08         | 2               | .96    | -            | -               | -      |
| AE model with no A1' path | 20365| 7548| 5269 | 3.9          | 3               | .28    | 3.8          | 1               | .05 |
| AE model with no p path | 20440| 7548| 5344 | 79           | 3               | <.001  | 79           | 1               | <.001 |
Table 4. Testing for power in the study sample to detect a genetic transmission pathway (A1’) of varying magnitudes between parental criticism and adolescent internalising symptoms, which show a phenotypic correlation (rPH) of .29

| Specified/estimated parameters | E1  | A1  | E2  | A2  | A1’ | p   | Power to detect A1’ (α = .05) |
|-------------------------------|-----|-----|-----|-----|-----|-----|-----------------------------|
| Study model                   | .74 | .26 | .39 | .48 | .05 | .24 | 13                          |
| Simulated models              | .74/.75 | .26/.26 | .39/.41 | .00/.01 | .53/.53 | .11/.11 | 62                         |
|                               | .74/.75 | .26/.25 | .39/.41 | .10/.11 | .43/.42 | .13/.13 | 54                         |
|                               | .74/.75 | .26/.25 | .39/.41 | .20/.22 | .33/.31 | .15/.15 | 48                         |
|                               | .74/.75 | .26/.25 | .39/.41 | .30/.32 | .23/.20 | .17/.17 | 39                         |
|                               | .74/.75 | .26/.25 | .39/.41 | .35/.36 | .18/.15 | .19/.19 | 33                         |
|                               | .74/.75 | .26/.25 | .39/.41 | .39/.40 | .14/.11 | .20/.21 | 29                         |
|                               | .74/.75 | .26/.25 | .39/.41 | .40/.41 | .13/.10 | .20/.21 | 28                         |

*We use simulated data to fit the study data structure, including 876 adult twin pairs with one child per twin and 1030 adolescent twin pairs with one parent per pair. Parameters were specified based on results from the study model, where heritability of parental criticism was .26 (A1) and heritability of adolescent internalising symptoms was .53 (A1’ + A2). Each row represents a new model. Specified values for A1’ are varied across models to manipulate the % of the phenotypic correlation (.29) attributable to A1’. Corresponding changes are made to the specified A2 and p estimates in each model to preserve heritability estimates and the phenotypic correlation. Values given for latent factors (E1, A1, E2 etc.) are variance components, whereas the value given for p is a standardised path coefficient (beta). Bolded row shows the model where 80% power to significantly detect A1’ was reached.
Figure 1. Genetic correlations between family members in the Children of Twins design for both monozygotic (MZ) and dizygotic (DZ) twin families.

- MZ=1.00/DZ=.50
- MZ=.50/DZ=.25
- MZ=.25/DZ=.125
Figure 2. Model specification for examining the association between parental criticism and adolescent internalising symptoms

A1=additive genetic effects on parent criticism; C1=shared-environmental effects on parent criticism; E1=nonshared environmental effects on parent criticism; A1'=genetic effects common to parent criticism and adolescent internalising symptoms; A2= additive genetic effects specific to adolescent internalising symptoms; C2=shared-environmental effects on adolescent internalising symptoms (not estimable using cousin data); E2=nonshared environmental effects on adolescent internalising symptoms; p=phenotypic effect of parental criticism on offspring adolescent internalising symptoms; rEdz/mz=within-parent correlation between E1 for parenting of child 1 and 2, allows parenting of each child to differ and varies according to offspring zygosity. The pathway between A1 and A1' is fixed to .50 because parents and children share approximately 50% of their genome. Variance paths have been omitted for simplicity, but for all latent factors, variance=1.

\[ r_{Edz/mz} = \text{within-parent correlation between E1 for parenting of child 1 and 2, allows parenting of each child to differ and varies according to offspring zygosity.} \]
\[ r_{A1A1'} = 0.50 \]
Figure 3. Model results, showing the association between parental criticism and adolescent offspring internalising symptoms

A. Full ACE model

B. Nested AE model (drop C1 and C2)

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A1=additive genetic effects on parent criticism; C1=shared environment effects on parent criticism; E1=nonshared environmental effects on parent criticism; A1'=genetic effects common to parent criticism and adolescent anxious-depression (pathway between A1 and A1' is fixed to .50 because parents and children share approximately 50% of their genome); A2= additive genetic effects specific to adolescent internalising symptoms; C2=shared environmental effects on adolescent internalising symptoms; E2=nonshared environmental effects on adolescent internalising symptoms. Residual intergenerational association in bold (standardised path coefficient).
Supplementary Materials

Sensitivity analyses, using only adolescent self-report to measure offspring symptoms

Table S1. Correlations in monozygotic (MZ) and dizygotic (DZ) families

| Parental criticism | MZ twin families | DZ twin families |
|--------------------|-----------------|-----------------|
| Parental criticism |                 |                 |
| Twin parent correlations on parental criticism for adolescent offspring | .25 | .14 |

Parental criticism and self-report offspring internalising symptoms

| Parental criticism and self-report offspring internalising symptoms | MZ twin families | DZ twin families |
|--------------------------------------------------------------------|-----------------|-----------------|
| Parent-child correlation | .18 | .18 |
| Avuncular correlations | .07 | .04 |

Self-report offspring internalising symptoms

| Self-report offspring internalising symptoms | MZ twin families | DZ twin families |
|---------------------------------------------|-----------------|-----------------|
| Cousin correlations on internalising symptoms | .13 | .07 |
| Twin correlations on internalising symptoms | .50 | .25 |

*Correlations taken from TOSS, the Children of Twins sample. Correlations taken from TCHAD, the child twin sample.

Table S2. Model fit statistics

|                  | -2LL  | df  | AIC   | Compare to full model | Compare to AE model |
|------------------|-------|-----|-------|-----------------------|---------------------|
|                  |       |     |       | x^2                   | x^2                 |
|                  |       |     |       | Δ df      | p       | Δ df      | p       |

| Model                | -2LL  | df  | AIC   | x^2 | Δ df | p   | x^2 | Δ df | p   |
|----------------------|-------|-----|-------|-----|------|-----|-----|------|-----|
| Full model (ACE model) | 20187 | 7368 | 5451  | -   | -    | -   | -   | -    | -   |
| No C paths (AE model) | 20187 | 7370 | 5447  | .11 | 2    | .95 | -   | -    | -   |
| AE model with no A' path | 20188 | 7371 | 5446  | .80 | 3    | .85 | .69 | 1    | .41 |
| AE model with no p path | 20212 | 7371 | 5470  | 24  | 3    | <.001 | 24  | 1    | <.001 |
Figure S1. Model results, showing the association between parental criticism and adolescent offspring internalising symptoms.

A1=additive genetic effects on parent criticism; E1=nonshared environmental effects on parent criticism; A1'=genetic effects common to parent criticism and adolescent anxious-depression (pathway between A1 and A1' is fixed to .50 because parents and children share approximately 50% of their genome); A2= additive genetic effects specific to adolescent internalising symptoms; E2=nonshared environmental effects on adolescent internalising symptoms. Residual intergenerational association in bold (standardised path coefficient).
Table S3. Testing for power in the study sample to detect a genetic transmission pathway (A1’) of varying magnitudes between parental criticism and adolescent internalising symptoms, which show a phenotypic correlation (rPH) of .18.

| Specified/estimated parameters | % rPH attributable to A1’ | Power to detect A1’ (α = .05) |
|-------------------------------|--------------------------|-----------------------------|
| **Study model**               |                          |                             |
| E1/A1                         | .74/.75                  | .26/.25                     | .50/.51 | .46/.45 | .01/.00 | .16/.17 | 7 | .07 |
| **Simulated models**          |                          |                             |
| E1/A1                         | .74/.74                  | .26/.26                     | .50/.51 | .00/.00 | .47/.48 | .01/.01 | 94 | 1.0 |
| E1/A1                         | .74/.74                  | .26/.26                     | .50/.51 | .10/.11 | .37/.37 | .03/.03 | 86 | 1.0 |
| E1/A1                         | .74/.74                  | .26/.26                     | .50/.51 | .20/.21 | .27/.27 | .05/.05 | 73 | 1.0 |
| E1/A1                         | .74/.74                  | .26/.26                     | .50/.51 | .30/.31 | .17/.15 | .08/.08 | 56 | .93 |
| E1/A1                         | .74/.74                  | .26/.26                     | .50/.51 | .32/.33 | .15/.14 | .09/.09 | 51 | .88 |
| **.74/.74**                   | **.26/.26**              | **.50/.51**                 | .34/.35 | .13/.12 | .10/.10 | **47** | **.82** |
| E1/A1                         | .74/.74                  | .26/.26                     | .50/.51 | .35/.36 | .12/.10 | .10/.10 | 45 | .78 |

*We use simulated data to fit the study data structure, including 876 adult twin pairs with one child per twin and 1030 adolescent twin pairs with one parent per pair. Parameters were specified based on results from the study model, where heritability of parental criticism was .26 (A1) and heritability of adolescent internalising symptoms was .47 (A1’ + A2). Each row represents a new model. Specified values for A1’ are varied across models to manipulate the % of the phenotypic correlation (.29) attributable to A1’. Corresponding changes are made to the specified A2 and p estimates in each model to preserve heritability estimates and the phenotypic correlation. Values given for latent factors (E1, A1, E2 etc.) are variance components, whereas the value given for p is a standardised path coefficient (beta). Bolded row shows the model where 80% power to significantly detect A1’ was reached.*
Testing directionality of the $p$ pathway

As a follow-up analysis to the unidirectional Children of Twins model that we have presented, we ran a reciprocal causation model that included causal paths running from child-to-parent as well as the parent-to-child path and genetic transmission path. The logic underlying reciprocal causation models has been discussed elsewhere, as has the application of these models to combined Children of Twins (CoT) and children-as-twin (CaT) data. The model that we used is given in Figure S1 and is based on the Narusyte model, with $r_{Emz}$ and $r_{Edz}$ parameters added. These are ‘within-person’ $E$ correlations, added to allow for differences in the parenting of adolescent twin 1 and the parenting of adolescent twin 2. This means that the within-person parenting correlations for the parents of MZ twins can differ from that of the parents of DZ twins. They can also both differ from the MZ twin correlation in the CoT sample. This is important because in previous applications of these models, model specification was such that within-person correlations and between-MZ-twin correlations were all specified as $A+C$, meaning that these 3 correlations were constrained to be the same. In our specification they can all vary, meaning that the within-person parenting correlations in the CaT sample are not brought into the estimation of parent aetiology. We have also specified the model to ensure that MZ twin children share an identical $A1'$ factor (in previous specifications they did not).

By applying a reciprocal causation model, we hoped to identify whether the association between parental criticism and offspring internalising could best be conceptualised as a parent-to-child effect, or a child-to-parent effect. Unfortunately, the model was unable to distinguish between the two (see Table S1). Whilst a genetic transmission only model (Model 5) was a significantly worse fit to the data than the full model (in agreement with our unidirectional model), it was possible to drop either the parent-to-child ($m$) pathway, or the child-to-parent ($n$) pathway. This was true when comparing back to the full model (Models 3 and 4 compared to Model 1), and when comparing back to the model with no genetic transmission (Models 6 and 7 compared to Model 2). There are several interrelated possibilities that may explain our inability to distinguish these paths from one another: (1) Our samples may be underpowered (as indicated by wide confidence intervals), (2) both parent-to-child and child-to-parent effects may be operating in tandem, (3) the aetiological structure of our phenotypes were not distinct enough. Heath and colleagues highlighted the need for phenotypes to have a distinct aetiological structure when using reciprocal causation models. Although child internalising is significantly more heritable than parental criticism in these data, it is possible that their aetiologies were not distinct enough. For example, it is possible that if $C2$ were significant, it would be easier to distinguish the $m$ and $n$ paths because $C2$ would impact upon the parent-child covariance structure.

1. Heath AC, Kessler RC, Neale MC, Hewitt JK, Eaves LJ, Kendler KS. Testing hypotheses about direction of causation using cross-sectional family data. Behav Genet. 1993;23(1):29-50.
2. Narusyte J, Neiderhiser JM, D’Onofrio BM, et al. Testing different types of genotype-environment correlation: an extended children-of-twins model. Dev Psychol. 2008;44(6):1591-1603.
Figure S2. Model specification for a two-sample reciprocal causation Children of Twins model, used to examine the association between parental criticism and adolescent internalising symptoms. A1 = Additive genetic effects on parental phenotype; C1 = shared-environmental effects on parental phenotype; E1 = nonshared environmental effects on parental phenotype; A1’ = genetic effects common to parental phenotype and offspring phenotype; A2 = genetic effects specific to offspring phenotype; C2 = shared-environmental effects on offspring phenotype (not estimable using cousin data); E2 = nonshared environmental effects on offspring phenotype; m = phenotypic effect of parent on offspring; n = phenotypic effect of child on parent; rEmz / rEdz = freely estimated correlations to allow the parenting of twin 1 and twin 2 to differ from one another, while also ensuring that these within-person correlations can differ across zygosity and can differ from MZ twin parent correlations. Measurement error (ε1 and ε2) contributes directly to the variance of both phenotypes.
Table S4. Standardised parameter estimates (95% confidence intervals) and model fit in nested two-sample reciprocal causation Children of Twins models \(^a\)

| Model | A1   | C1   | E1   | A2   | C2   | E2   | ε    | A1'  | m   | n   | -2LL (df) | AIC      | χ² (Adf) | p     |
|-------|------|------|------|------|------|------|------|------|-----|-----|------------|----------|----------|-------|
| 1     | .35  | -    | .29  | .54  | .02  | .04  | .34  | .06  | -.05| .41 | 20352.90 (7544) | 5264.90  |          | .91   |
|       | (.18, .46) | (.12, .79) | (.11, .65) | (.00, .20) | (.00, .72) | (.00, .45) | (.00, .32) | (.34, .62) | (.45, .56) | (.34, .62) | .00, .20 | (.00, .72) | (.00, .45) | (.00, .32) | (.34, .62) | (.45, .56) |
| 2     | .32  | -    | .36  | .58  | -    | .08  | .31  | .03  | .06 | .33 | 20352.91 (7545) | 5262.91  | 0.01     | .91   |
|       | (.18, .46) | (.13, .79) | (.41, .65) | (.00, .72) | (.00, .44) | (.00, .21) | (.00, .62) | (.46, .56) | (.34, .62) | (.46, .56) | .00, .21 | (.00, .44) | (.00, .21) | (.00, .62) | (.46, .56) | (.34, .62) | (.46, .56) |
| 3     | .27  | -    | .51  | .55  | -    | .23  | .22  | -   | .37 | .01 | 20353.05 (7546) | 5261.05  | 0.15     | .93   |
|       | (.18, .43) | (.16, .79) | (.40, .65) | (.00, .60) | (.00, .42) | (.00, .21) | (.00, .62) | (.38, .56) | (.38, .56) | (.38, .56) | .00, .21 | (.00, .42) | (.00, .21) | (.00, .62) | (.38, .56) | (.38, .56) |
| 4     | .34  | -    | .33  | .58  | -    | .06  | .33  | .04  | -   | .37 | 20352.93 (7546) | 5260.93  | 0.04     | .98   |
|       | (.25, .43) | (.18, .50) | (.43, .65) | (.00, .18) | (.19, .42) | (.00, .20) | (.25, .51) | (.25, .51) | (.25, .51) | (.25, .51) | .00, .20 | (.19, .42) | (.00, .20) | (.25, .51) | (.25, .51) | (.25, .51) |
| 5     | .27  | -    | .51  | .54  | -    | .24  | .22  | .00  | .40 | -   | 20353.02 (7546) | 5261.02  | 0.13     | .94   |
|       | (.18, .35) | (.39, .67) | (.39, .60) | (.16, .36) | (.08, .30) | (.00, .11) | (.00, .24, 53) | (.00, .24, 53) | (.00, .24, 53) | (.00, .24, 53) | .00, .11 | (.00, .24, 53) | (.00, .11) | (.00, .24, 53) | (.00, .24, 53) | (.00, .11) | (.00, .24, 53) | (.00, .11) |
| 6     | .34  | -    | .35  | .09  | -    | .05  | .32  | .55  | -   | -   | 20439.18 (7547) | 5345.18  | 86.3     | <.00  |
|       | (.27, .41) | (.22, .73) | (.00, .21) | (.00, .41) | (.00, .41) | (.42, .67) | (.42, .67) | (.42, .67) | (.42, .67) | (.42, .67) | .00, .41 | (.00, .41) | (.00, .41) | (.42, .67) | (.42, .67) | (.42, .67) | (.42, .67) |
| 7     | .36  | -    | .28  | .61  | -    | .03  | .36  | -   | -   | .46 | 20355.10 (7547) | 5261.10  | 2.05     | .15   |
|       | (.27, .44) | (.15, .41) | (.56, .66) | (.00, .10) | (.28, .44) | (.39, .54) | (.39, .54) | (.39, .54) | (.39, .54) | (.39, .54) | .00, .10 | (.28, .44) | (.39, .54) | (.39, .54) | (.39, .54) | (.39, .54) | (.39, .54) |
| 8     | .27  | -    | .52  | .55  | -    | .24  | .21  | -   | .38 | -   | 20353.05 (7547) | 5259.05  | 0.00     | .97   |
|       | (.18, .35) | (.41, .60) | (.49, .60) | (.16, .34) | (.12, .29) | (.33, .44) | (.33, .44) | (.33, .44) | (.33, .44) | (.33, .44) | .00, .34 | (.12, .29) | (.33, .44) | (.33, .44) | (.33, .44) | (.33, .44) | (.33, .44) |

\(^a\) A1=Additive genetic effects on parental phenotype; C1=shared-environmental effects on parental phenotype; E1=nonshared environmental effects on parental phenotype; A2=genetic effects specific to offspring phenotype; C2=shared-environmental effects on offspring phenotype (not estimable using cousin data); E2=nonshared environmental effects on offspring phenotype; ε=error term; A1'=genetic effects common to parental phenotype and offspring phenotype; m=phenotypic effect of parent on offspring; n=phenotypic effect of child on parent. Model 1 represents the full model, excluding C1 as advised in Narusyte et al. (2008). In subsequent nested models, empty cells (-) indicate that the parameter was dropped from the model. Model fit in Models 2 – 6 are compared to Model 1. Model 7 (A1’ and m dropped) and Model 8 (A1’ and n dropped) are compared to Model 3 (A1' dropped).