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Aetiological influences on stability and change in emotional and behavioural problems across development: a systematic review

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Abstract

Emotional and behavioural problems in childhood and adolescence can be chronic and are predictive of future psychiatric problems. Understanding what factors drive the development and maintenance of these problems is therefore crucial. Longitudinal behavioural genetic studies using twin, sibling or adoption data can be used to explore the developmental aetiology of stability and change in childhood and adolescent psychopathology. We present a systematic review of longitudinal, behavioural genetic analyses of emotional and behavioural problems between ages 0 to 18 years. We identified 58 studies, of which 19 examined emotional problems, 30 examined behavioural problems, and 9 examined both. In the majority of studies, stability in emotional and behavioural problems was primarily genetically influenced. Stable environmental factors were also widely found, although these typically played a smaller role. Both genetic and environmental factors were involved in change across development. We discuss the findings in the context of the wider developmental literature and make recommendations for future research.

Keywords

Childhood; adolescence; psychopathology; systematic review; behavioural problems; emotional problems; quantitative genetics; behavioural genetics; longitudinal

Introduction

Early-life emotional and behavioural problems constitute an important risk factor for future mental and physical health problems (e.g., Bardone et al., 1998; Harrington, Fudge, Rutter, Pickles, & Hill, 1990; Hofstra, van der Ende, & Verhulst, 2000, 2002; Kim-Cohen et al., 2003). However, these problems can begin to pose difficulties to individuals and their families from the moment they emerge in childhood or adolescence, with potential for associated disruption across a wide range of social, cognitive and educational domains (Eisenberg, Fabes, Guthrie, & Reiser, 2000; Hinshaw, 1992; Larsson & Frisk, 1999; McLeod & Kaiser, 2004). Studying the aetiology of the emergence and continuity of emotional and behavioural problems across development therefore has important implications for both current difficulty and future risk. First, identifying the importance of different aetiological

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factors at different stages of development can inform as to the optimal nature and timing of interventions. Second, understanding how childhood emotional and behavioural problems emerge and are maintained may help to clarify the pathways by which risk for future mental health problems is mediated.

Emotional and behavioural problems across childhood and adolescence

Emotional problems (often termed *internalising* problems) include anxiety, depression and associated symptoms and behaviours. Emotional problems are highly prevalent in childhood and adolescence (Costello, Mustillo, Erkanli, Keeler, & Angold, 2003; Merikangas, He, Brody, et al., 2010), with recent figures estimating lifetime prevalence at age 18 as high as 14.3% for mood disorders and 31.9% for anxiety disorders (Merikangas, He, Burstein, et al., 2010). In both cases, females (18% and 28% respectively) experience a greater number of emotional problems than males (10.5% and 26.1% respectively). Emotional problems begin early, with mean onset in childhood for anxiety disorders (Kessler, Chiu, Demler, Merikangas, & Walters, 2005) and early adolescence for depressive disorders (Kovacs & Devlin, 1998). Furthermore, temperamental factors, such as behavioural inhibition, that are associated with the development of emotional problems, are in evidence from very early in childhood (Caspi, Henry, McGee, Moffitt, & Silva, 1995; Degnan, Almas, & Fox, 2010; Rapee, Schniering, & Hudson, 2009). Individuals who experience emotional problems in childhood and adolescence are at a significantly increased risk of developing subsequent psychiatric problems in young adulthood and beyond (e.g., anxiety: Gregory et al., 2007; addiction: Lopez, Turner, & Saavedra, 2005; mood disorders: Roza, Hofstra, Van Der Ende, & Verhulst, 2003; suicidality: Weissman et al., 1999).

Behavioural (or *externalising*) problems include difficulties associated with attention, hyperactivity, conduct problems, aggression and antisocial behaviour. They are highly prevalent across both childhood and adolescence, with 20-23% of children estimated to experience a behavioural disorder by age 16 years (Costello et al., 2003; Merikangas, He, Burstein, et al., 2010). The high prevalence of behavioural problems is particularly driven by attention-deficit hyperactivity disorder (ADHD) and conduct problems, which have been shown to have 12-month prevalence rates of 8.6% and 2.1% respectively during middle childhood and adolescence (Merikangas, He, Brody, et al., 2010). In contrast to emotional problems, behavioural disorders are more common in males (23.5%) than females (15.5%; lifetime prevalence of any behavioural disorder at age 18; Merikangas, He, Burstein, et al., 2010). Behavioural disorders vary relatively widely across childhood and adolescence in terms of their age-of-onset (Kessler, Berglund, et al., 2005). However, temperamental dispositions that are predictive of later behavioural problems are again reliably observable very early in development (e.g. aggression: Tremblay et al., 2005).

Stability of psychopathological traits across development, sometimes referred to as *homotypic continuity*, is moderate for both emotional (Ollendick & King, 1994; Weems, 2008) and behavioural problems (Hofstra et al., 2000; Klein, Otto, Fuchs, Reibiger, & von Klitzing, 2015; Verhulst & van der Ende, 1995). While homotypic continuity is evident from early in development (e.g., Anselmi et al., 2008; Bufferd, Dougherty, Carlson, Rose, & Klein, 2012), it is typically thought to increase during adolescence for both emotional

(Costello et al., 2003; Ferdinand, Dieleman, Ormel, & Verhulst, 2007) and behavioural problems (Copeland, Shanahan, Costello, & Angold, 2009; Mesman & Koot, 2001; Sourander & Helstelä, 2005). However, the developmental presentation of emotional and behavioural problems is also characterised by change. Both childhood and adolescence are periods of extensive brain maturation, accompanied by profound changes in the social environment as well as in hormonal and physical development (Blakemore, 2008; Paus, Keshavan, & Giedd, 2008). Emotional and behavioural problems in childhood may therefore present in very different ways to these same problems in adolescence. Specifically, adolescence may be a period of phenotypic differentiation for emotional problems, with the generalised emotional problems that characterise earlier ages disaggregating, increasingly, into distinguishable mood and anxiety disorders (Cicchetti & Rogosch, 2002; Hallett, Ronald, Rijsdijk, & Eley, 2009; Waszczuk, Zavos, Gregory, & Eley, 2014). Depressive symptoms, in particular, are seen to increase after the onset of adolescence (Dekker et al., 2007; Ge, Conger, & Elder, 2001), driving an increase in the incidence of depression that is accompanied by the emergence of significant gender differences (Hankin et al., 1998; Thapar, Collishaw, Pine, & Thapar, 2012). Patterns are mixed across the traits underpinning behavioural problems in development, with mean levels of some externalising behaviours increasing in adolescence (e.g., status violations: Bongers, Koot, Ende, & Verhulst, 2004; antisocial behaviour: Moffitt, 1993) and others decreasing (e.g., aggression: Bongers et al., 2004; ADHD symptoms: Rutter, Kim-Cohen, & Maughan, 2006). In general, for both emotional and behavioural problems, there is evidence that the stability of relevant traits is disrupted during adolescence in particular (Hofstra et al., 2000).

Genetic influence on emotional and behavioural problems in development

The study of emotional and behavioural problems in childhood is complicated by the complex entanglement of genetic and environmental factors that underpin them (Rutter & Silberg, 2002; Rutter, 2004). Behavioural genetic designs can be used to unpick the aetiology of behavioural phenotypes and identify the relative contributions of genetic and environmental factors (Jaffee, Price, & Reyes, 2013; Rijsdijk & Sham, 2002; Turkheimer, 2000). In the context of this review, we use 'behavioural genetic designs' to refer to studies using genetically informative data from twin, sibling or adoption samples, where information about the differing degrees of genetic relatedness between participants within a sample - for example, identical and non-identical twin pairs - can be used to decompose the sources of variance in individuals' scores on quantitative measures of behaviour. Structural equation modelling of these data is used to produce estimates of the genetic and environmental influence on a given behaviour (Plomin, DeFries, Knopik, & Neiderhiser, 2013; Rijsdijk & Sham, 2002). These estimates, including the estimate of *heritability*, are population-level statistics that apply to the group from which the individuals were drawn. As such, they can only be used to make inferences about the aetiological origins of *differences* between individuals in a given population, rather than having an interpretable meaning for any one individual.

Genetically informative studies of measures of emotional and behavioural problems are numerous, and have typically shown that both genetic and environmental factors are important in their development (Burt, 2014; Lau & Eley, 2010; Rhee & Waldman, 2002;

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Rice, Harold, & Thapar, 2002; Rutter, Silberg, O'Connor, & Simonoff, 1999). Cross-sectional behavioural genetic estimates for measures of both emotional and behavioural problems tend to show age-related increases in heritability, suggesting that their aetiological architecture is not fixed across development (Bergen, Gardner, & Kendler, 2007). In some cases, increases in heritability may reflect relative decreases in the influence of (especially shared) environmental factors (e.g., Lamb et al., 2010; Scourfield et al., 2003). However, changes in the specific nature of the genetic and environmental influences that operate at different ages are also possible. For example, *genetic innovation* is observed when new genetic factors come online to influence emotional or behavioural problems developmentally. In this way, certain genes may only function from adolescence onwards. The influence of these new genetic factors may or may not result in an increase in heritability, depending on the extent to which earlier factors become less influential over time - a process termed *genetic attenuation* (Kendler, Gardner, Annas, et al., 2008). Different genetic influences may become influential as different behaviours and cognitive processes become involved in driving the symptoms of emotional and behavioural problems at different stages of development.

Studies that combine genetically informative data with developmental context are crucial in developing our understanding of the emergence and persistence of emotional and behavioural problems in development. Longitudinal behavioural genetic designs are, thus, ideally suited to the developmental study of psychopathology. With data collected from the same individuals at multiple time points, it is possible to estimate the extent to which various aetiological influences operate stably, contributing to continuity in the phenotype over time, or innovatively, driving phenotypic change (Boomsma, Busjahn, & Peltonen, 2002). In this way, developmental questions concerning the *extent* of continuity or discontinuity of various emotional and behavioural problems in childhood and adolescence can be expanded to address the aetiological *nature* of the developmental patterns that are observed. These longitudinal, behavioural genetic studies of emotional and behavioural problems in childhood and adolescence are the subject of this review.

The current review

The aim of the current review is to systematically collate, present and appraise the evidence regarding aetiological contributions to stability and change in emotional and behavioural problems in childhood and adolescence. In particular, this review will provide an overview of the degree of convergence of results on the sources of phenotypic stability and change across different samples, measures and modelling strategies. By reviewing studies of emotional and behavioural problems together, with a strict focus on the sources of variance in homotypic continuity and discontinuity, we aim to answer two interrelated questions. First: to what extent is phenotypic stability in childhood and adolescent psychopathology underpinned by genetic *versus* environmental factors? Second: to what extent are the genetic and environmental influences upon emotional and behavioural problems stable *versus* innovative across development?

Method

Search strategy

The literature search was performed using OvidSP on the following databases: *Ovid Medline* (1946 - April 2015); *Embase* (1947 - April 2015); *PsycINFO* (1806 - April 2015); *Journals@Ovid Full Text* (April 2015 update) and *PsycARTICLES Full Text.* We ran the searches for emotional and behavioural problems separately. Search terms for emotional problems were as follows: *depress*, mood, emotion*, affective disorder*, internali*, anxi*, worry,fear*, obses*, compul*, OCD, panic, phobi*, inhibit*, shy*, withdrawn, somatic*. Search terms for behavioural problems were: *behav*, attenti*, inattenti* externalising, externalizing, conduct disorder*, ADHD*, hyperactiv*, impuls*, disruptive*, problem*, aggress*, violen*, crimin*, deviant*, delinquen*, oppositional, ODD*. Both searches also included terms designed to produce a list of results that used genetic methods (*genes, genetic*, aetiolog*, etiolog*, twin*, adopt*, hertiab**) applied longitudinally (*longitudinal, stab*, change*, innovat*, continuity, development*, child* & adolescen*).* Age criteria were only applied during manual screening to avoid missing relevant studies that did not include 'childhood' or 'adolescence' in their titles. Additional search steps included scanning of the reference lists of recent studies (since 2010), and manual searches of journals containing the more than 5 of the studies identified via the database searches. This allowed us to reduce the possibility of non-indexed studies being missed.

Inclusion criteria

Studies were required to meet the following criteria for inclusion in this review:

1. The results must have been presented as a full paper published in a peer-reviewed journal[1]
2. The study design was required to include longitudinal, behavioural genetic analysis of data. Studies using other genetic designs (e.g., molecular genetic studies) were excluded. Studies using genetically informative data with designs that do not examine genetic influence (e.g., monozygotic twin differences) were also excluded.
3. Studies had to include any measure of at least one childhood emotional or behavioural problem. Phenotypes for inclusion were:
    - *Emotional problems:* depression or depressive symptoms; mood disorders; anxiety; worry; fear; obsessive-compulsive behaviours; panic symptoms; phobias; shyness; inhibition; general emotional problems, somatic complaints.
    - *Behavioural problems:* attention problems; hyperactivity; violent behaviour; conduct disorder; aggression; delinquency; peer deviance; criminality; general behavioural problems

[1]One exception was made to this criterion, in the case of a relevant study (Waszczuk, Zavos, Gregory, & Eley, 2016), the forthcoming publication of which we were aware due to the involvement of some of the authors of this review. We opted to include the results of this study in anticipation of publication at a similar time to this review.

4. Phenotypes must have been measured at least twice between the ages of 0 and 18 years, with data analysed longitudinally between these ages. Studies meeting these criteria but with further waves of measurement beyond the age of 18 years were included, with only results from waves within the 0-18 range presented in the review.

5. Studies were required to have been written in English.

Procedure

Study selection

The results of the database searches were checked for duplicates, which were removed. The titles of the remaining results were then used to identify those records that were clearly irrelevant to the current review (e.g., molecular genetic studies, review papers, commentaries). Following this process, the remaining records were exported from the database, along with their abstracts and other information. The abstracts, titles and format (e.g., full paper, conference abstract, etc.) of these records were then screened for further exclusions. All steps were carried out independently by two of the authors (LJH and NW) and, at this point, inclusion and exclusion lists were compared and discrepancies reviewed.

Final exclusions were made based on independent screening of full papers still included at this stage. Any further discrepancies and the inclusion or exclusion of studies using non-standard genetic models was discussed with a third author (TAM). The process of study selection is summarised in Figure 1.

Data extraction

Data were extracted from studies for presentation in the results section of this review. The fields for data extraction were the following: (1) study sample, including sample name if provided; (2) number of participants, given in terms of pairs of twins or siblings where possible; (3) phenotype(s) studied and measure(s) used, including mode(s) of measurement or (if questionnaire) reporter(s); (4) mean age(s) and age range(s) of participants at each eligible wave of data collection; (5) type of genetic model(s) used in analyses; (6) stability of phenotype(s) between all eligible waves and the proportion of this stability due to genes; (7) influence of stable genetic factors, given in terms of the proportion of phenotypic variance at the later age(s) explained by genetic factors from the earlier age(s); (8) influence of stable shared (if estimated) and non-shared environmental factors; (9) influence of new genetic factors, given in terms of the amount of phenotypic variance at later ages explained by factors emerging at these ages; and (10) influence of new shared (if estimated) and non-shared environmental factors.

Data extraction was carried out and cross-checked by LH and NW. We contacted the authors of studies for which the desired information was not included in either the paper or online supplementary materials.

Data synthesis—Among studies included in the review, the twin design was the most common (see 'Study sample' in Results section for details). An introduction to the use of

structural equation modelling in twin studies is presented in Box 1. The breadth of the measures of emotional and behavioural problems and methodological variation involved in the studies included in this review precluded a meta-analysis of findings. Specifically, the studies included in the review varied in phenotype, measure, reporter, age, interval of measurement, modelling strategy, and handling of sex differences. Consequently, we instead took steps to ensure that results could be presented in as comparable a manner as possible. However, studies differed in the way in which genetic and environmental influences on stability and change were presented. In part, this was due to the use of a range of different structural equation models across the included studies. The most common genetic model was the longitudinal Cholesky decomposition (Box 2, Figure B), in which components of variance from early waves are able to influence variance at later waves. Residual variance at later waves that is not explained by these earlier aetiological factors is then decomposed separately, with the resulting variance components free to explain variance at subsequent waves. Accordingly, we have presented (where possible) each study's findings regarding aetiological contributions to stability and change in the manner typically used in the description of a longitudinal Cholesky decomposition: in terms of the amount of variance at each wave (% of total) explained by stable, pre-existing factors *versus* emerging, new factors (see Box 2). Where modelling results could not be converted to this format, we have presented information on the aetiology of stability and change as described in Box 3 or as indicated on a case-by-case basis in the tables and text of the results section. The majority of studies included in this review used one, or a combination of the models presented in Box 2 & Box 3. In cases where the models used in a study were non-standard adaptations of the models presented in the boxes, we have used the authors' original terminology (e.g., 'Developmental model', 'Transmission model'). Space limitations preclude in-text descriptions of each individual model in this review and, as such, we refer readers to the original articles for full details.

We opted to exclude cross-lagged models from our review. This was because the stability-change interpretation sometimes applied to the results of cross-lagged model assumes that genetic influences are transmitted between waves in a manner that is directly proportionate to their relative importance at the first wave. In contrast, the Cholesky decomposition (and other included models) allows direct estimation of genetic influence on the covariation between traits (for further exploration of this issue, see Luo, Haworth, & Plomin, 2010).

Results

Study sample

In total, 58 separate eligible studies, published between 1993 and 2015, were identified and included in the review. Of these, 19 involved only emotional problems, 30 involved only behavioural problems and 9 involved at least one phenotype from each. 55 studies used twin data, while 5 used sibling or adoption data, either additionally or exclusively. Figure 2 illustrates the approximate ages spanned by studies included in the review.

Table 1 and Table 2 present the results from all studies included in the review. Studies are organised alphabetically by author name and presented along with basic information. The text summary below is organised by phenotype, with a view to highlighting any emerging

developmental themes in the results of studies of similar sub-types of emotional and behavioural problems. We have endeavoured to specify the age ranges of studies referred to in the text where possible. Where the terms 'childhood' and 'adolescence' are used descriptively without any age range specified in parentheses, we use these terms to refer to ages between 0-12 years and 13-18 years respectively.

Emotional Problems

Twenty-eight (28) studies of emotional problems included in this review are presented in Table 1. As noted in the methods section above, 9 studies included longitudinal analyses of both emotional and behavioural problems. These studies are included in both results tables, and are indicated by a superscript (g). The results of all studies involving analyses of emotional problems are described in the text below, grouped by phenotype as follows: (1) anxiety, fear and obsessive-compulsive behaviour; (2) depression and depressive symptoms; and (3) temperament and broad measures of emotional problems.[2]

Anxiety, fear and obsessive-compulsive behaviour—Eleven studies focused specifically on aspects of anxiety, fear, obsessive-compulsive and other related behaviours. The aetiology of stability and change in measures of anxiety was investigated in six of these studies, collectively spanning an age range of 4 to 18 years (Garcia et al., 2013; Lewis & Plomin, 2015; Trzaskowski, Zavos, Haworth, Plomin, & Eley, 2012; Waszczuk, Zavos, & Eley, 2013; Waszczuk et al., 2016; Zavos, Gregory, & Eley, 2012). Overall, the stability of anxiety and anxiety-related behaviours in childhood and adolescence tended to be accounted for by genetic factors, and change by environmental factors. In all six studies, wave-to-wave stability was moderate (*r*Ph range: .33-.54) and predominantly genetically- influenced (>50% *r*Ph due to genes; Garcia et al., 2013; Lewis & Plomin, 2015; Trzaskowski et al., 2012; Zavos et al., 2012; Waszczuk, Zavos, & Eley, 2013; Waszczuk et al., 2016). New genetic factors were also found to account for change across both childhood (Lewis & Plomin, 2015; Trzaskowski et al., 2012) and adolescence (Waszczuk et al., 2016). However, in two (Garcia et al., 2013; Zavos et al., 2012) of the three studies incorporating data from *late* adolescence (17-18 years; Garcia et al., 2013; Waszczuk et al., 2016; Zavos et al., 2012) no significant genetic innovation was found, suggesting that the involvement of new genetic factors for anxiety-related behaviours may wane later in development. Environmental influences were mainly found to contribute to change in anxiety. While these influences were non-shared (E) in most cases, in one study (Trzaskowski et al., 2012), longitudinal analyses produced evidence of shared (C) environmental influences playing a small but significant role in driving change in anxiety-related behaviours across middle childhood. Three studies, collectively spanning an age range of 4-17 years, analysed obsessive-compulsive symptoms (OCS; Bolhuis et al., 2014; Krebs, Waszczuk, Zavos, Bolton, & Eley, 2014; van Grootheest et al., 2007). Results from this group of studies were mixed. Stability in OCS was primarily underpinned by genetic factors in two studies, both of which included adolescents (Bolhuis et al., 2014; Krebs et al., 2014). However, in one study environmental factors were of equal importance for stability during middle childhood (van Grootheest et

[2]This category incorporates those studies that use a measure of temperament *or* a broader or more general emotional problems phenotype (usually 'internalising behaviour'). Therefore, it overlaps with the previous categories phenotypically, but not in terms of the studies included.

al., 2007). Non-shared environmental factors were found to drive change in all studies, while new genetic factors were again implicated in middle childhood but not late adolescence (17 years; Bolhuis et al., 2014). The aetiology of stability and change in fear was investigated in two studies, which used data collected in middle childhood and middle adolescence (Kendler, Gardner, Annas, et al., 2008; Trzaskowski et al., 2012). Genetic factors were found to be important for both developmental stability and change in fear both as an anxiety sub-type (Trzaskowski et al., 2012) and in terms of specific phobias (Kendler, Gardner, Annas, et al., 2008). In the case of the former, moderately high phenotypic stability (*r*Ph: .52) was found between the ages of 7 and 9, and this was predominantly explained (67%) by genetic factors. New genetic factors explained 34% of variance at age 9 (Trzaskowski et al., 2012). A similar pattern was found to extend into adolescence for situational and blood/injury fears but not animal fears, which displayed less overall (and genetic) stability (Kendler, Gardner, Annas, et al., 2008). Age- specific environmental influences were also found to drive change in both studies. These were primarily non-shared, although new shared environmental influences were also significant and explained, on average, more than 15% of new variance in middle childhood and early adolescence (Kendler, Gardner, Annas, et al., 2008; Trzaskowski et al., 2012).

Depression and depressive symptoms—Depression-related phenotypes were analysed in seven studies, with participants ranging from 5 to 18 years of age (Bolhuis et al., 2014; Lau & Eley, 2006; O'Connor, Neiderhiser, Reiss, Hetherington, & Plomin, 1998; Scourfield et al., 2003; Silberg et al., 1999; Tully, Iacono, & McGue, 2010; Waszczuk et al., 2016). These studies showed a broadly consistent pattern of results for the aetiology of stability in depressive symptoms across adolescence. This stability was moderate in six studies (*r*Ph range: .33-.59) and low in just one (.24; Tully et al., 2010). In five studies, genetic factors were predominant in driving stability in depression and depressive symptoms across a range of ages from middle childhood (Bolhuis et al., 2014; Lau & Eley, 2006; Silberg et al., 1999; Tully et al., 2010; Waszczuk et al., 2016). In contrast, substantial environmental contributions to stability were found in only two studies, both measuring depressive symptoms in early-to-middle adolescence. Shared environmental factors accounted for more than 70% of the continuity (*r*Ph: .39) in one (Scourfield et al. 2003) and non-shared environmental factors accounted for 46% of the continuity (*r*Ph: .59) in the other (O'Connor et al., 1998). Notably, both of these two-wave studies had wide within-wave age ranges, including children up to 8-9 years apart. Results from the overall group of studies of depression were also consistent in their estimation of the relative aetiological influences on change. New non-shared environmental factors were found to drive change in depressive symptoms, doing so predominantly in all studies, and exclusively in all but three. These three studies (Lau & Eley, 2006; Scourfield et al., 2003; Waszczuk et al., 2016) also found evidence of new genetic factors driving change. None of the studies found significant new shared environmental influences across development.

Temperament and broad measures of emotional problems—Temperament, in the form of shy, withdrawn or inhibited behaviour, was analysed in four studies, across a range of ages in childhood (1-12 years; Cherny, Fulker, Corley, Plomin, & DeFries, 1994; Hoekstra, Bartels, Hudziak, Van Beijsterveldt, & Boomsma, 2008; Plomin et al., 1993; van

den Oord & Rowe, 1997). Genetic factors were again found to be important for stability and, to a lesser but still significant extent, change. Environmental factors were found to be substantially involved in the stability of withdrawn behaviour in only one of the studies (van den Oord & Rowe, 1997). Large contributions to change from non-shared environmental factors were observed in three studies (Cherny et al., 1994; Plomin et al., 1993; van den Oord & Rowe, 1997). In the remaining study (Hoekstra et al., 2008), environmental factors were found to play only a minimal aetiological role overall. However, it should be noted that this study involved the decomposition of a rater-agreed phenotype, which somewhat limits the extent to which the results from this study are directly comparable with the other studies in this subset. Nine studies included broader measures of emotional problems, such as mixed anxiety and depression symptoms (Bartels, van den Oord, et al., 2004; Haberstick, Schmitz, Young, & Hewitt, 2005; Huizink, van den Berg, van der Ende, & Verhulst, 2007; Kendler, Gardner, & Lichtenstein, 2008; Nivard et al., 2015; Schmitz, Fulker, & Mrazek, 1995; van den Oord & Rowe, 1997; van der Valk, van den Oord, Verhulst, & Boomsma, 2003; van der Valk, Verhulst, Neale, & Boomsma, 1998). These studies collectively covered an age range of 3-17 years. In this group of studies, stability was again predominantly underpinned by genetic factors in the majority of cases. However, moderately influential stable shared environmental factors were also found in early childhood (Bartels, van den Oord, et al., 2004), middle childhood (van den Oord & Rowe, 1997) and early-/mid-adolescence (Huizink et al., 2007; van der Valk et al., 1998). Interestingly, in a study of teacher-reported internalising symptoms in children aged 7-12 years, no such environmental influences on stability were found. Instead this stability, which was found to be modest-to-moderate (*r*Ph range: .14-.38), was entirely genetic (Haberstick et al., 2005). Across this group of studies of broadly assessed emotional problems, phenotypic change resulted from both new genetic and new (primarily non-shared) environmental factors. New genetic factors tended to be less influential later in adolescence, although results of Nivard et al. (2015) are a notable exception. In three studies, collectively spanning ages 4-17 years, new genetic factors were not at all influential (Huizink et al., 2007; van den Oord & Rowe, 1997; van der Valk et al., 1998). Findings of moderate-to-large non-shared environmental contributions to change were more consistent across studies and ages.

Summary

Broad measures were used in the majority of studies of emotional problems, with specific studies of anxiety, depression and other symptoms of childhood emotional problems relatively fewer in comparison. Overall, in these studies of specific emotional problems such as anxiety, obsessive-compulsive symptoms and depressive symptoms spanning childhood and adolescence from age 4-18 years, stability was largely underpinned by genetic influences. Results were particularly consistent for studies of depressive symptoms in adolescence. A similar pattern was evident in studies of broader emotional problems, which covered almost the entire eligible age range (1-17 years), with some additional evidence that shared environmental influences may also play a role in producing stability in measures of these phenotypes. In contrast, non-shared environmental influences were typically found to drive change across all emotional phenotypes and all ages. In some specific phenotypes, such as fear, new shared environmental factors were also influential. There was some evidence that the transition from childhood to adolescence may be characterised by the

emergence of new genetic influences. Where present, genetic innovation tended to disappear by late adolescence, although this was not true in all cases.

Behavioural Problems

Thirty-nine (39) studies of behavioural problems are presented in Table 2. This table is structured identically to Table 1, and similarly includes all studies with a measure of behavioural as well as emotional problems (denoted by superscript [g]). The results of all studies of behavioural problems are summarised below in three groups: (1) ADHD, attention problems and impulsivity (2) aggression, anger, antisocial behaviour and conduct disorder; and (3) general behavioural problems.[3]

ADHD, attention problems and impulsivity—Sixteen studies reported analyses of a measure of either attention deficit hyperactivity disorder (ADHD) or related symptoms and behaviours. Nine of these studies included data from at least two ages between 2 and 8 years (Ebejer et al., 2010; Kan et al., 2013; Kuntsi, Rijsdijk, Ronald, Asherson, & Plomin, 2005; Lewis & Plomin, 2015; Polderman et al., 2011; Price et al., 2005; Rietveld, Hudziak, Bartels, van Beijsterveldt, & Boomsma, 2004; van den Oord & Rowe, 1997; Wang, Deater-Deckard, Petrill, & Thompson, 2012). Overall, stability in measures of ADHD symptoms was found to be largely genetic in these childhood studies. Additive genetic factors explained more than 65% of the wave-to-wave stability of ADHD symptoms in childhood in six of these studies. However, in three studies where non-additive (dominant) genetic effects (D) were also estimated (Ebejer et al., 2010; Kan et al., 2013; Rietveld et al., 2004), these also contributed to stability. Indeed, in one study (Ebejer et al., 2010), of inattention, non-additive genetic factors were by far the greatest source of phenotypic stability during this period, although they were found to be more influential in driving change elsewhere (age 3-7; Kan et al., 2013; Rietveld et al., 2004). Notably, new additive genetic influences on ADHD symptoms across this period were also found and, in some cases, these too were greater than the stable genetic factors that preceded them (e.g., Kuntsi et al., 2005; Polderman et al., 2011). Environmental influences on change in childhood ADHD-related symptoms were predominantly age-specific and non-shared. Where environmental influences on stability were found, the effects were limited, with stable shared and non-shared factors explaining between 3-17% later variance in childhood ADHD-related symptoms (e.g., Ebejer et al., 2010; Kan et al., 2013; Lewis & Plomin, 2015; Price et al., 2005; van den Oord & Rowe, 1997). The single exception to the overall trend in this group was a study by Wang et al. (2012), in which stability from shared environmental influences and change from non-shared environmental influences was found, with only small or non-significant genetic influences on attention regulation problems at ages 7 and 8 years. Eleven studies that reported analyses of ADHD-related symptoms used data from across middle childhood and adolescence (9-18 years; Bezdjian, Tuvblad, Wang, Raine, & Baker, 2014; Jaffee, Hanscombe, Haworth, Davis, & Plomin, 2012; Kan et al., 2013; Larsson, Lichtenstein, & Larsson, 2006; Larsson, Larsson, & Lichtenstein, 2004; Lewis & Plomin, 2015; Niv, Tuvblad, Raine, Wang, & Baker, 2012; Rietveld et al., 2004; Taylor et al., 2013;

[3]This category incorporates only those studies that use a broader or more general phenotype of behavioural problems (usually 'externalising behaviour'). Therefore, it overlaps with the previous categories phenotypically, but not in terms of the studies included.

van den Oord & Rowe, 1997). Wave-to-wave stability in attention problems across middle childhood was accounted for by a mixture of genetic and environmental stability (e.g., Kan et al., 2013; Rietveld et al., 2004). However, stability in broader ADHD phenotypes remained primarily genetically influenced both in this period and into adolescence. For example, across five studies (Jaffee et al., 2012; Larsson et al., 2006; Larsson et al., 2004; Lewis & Plomin, 2015; Taylor et al., 2013), stable genetic factors from earlier in development explained more than one third of variance, on average, in ADHD-related phenotypes at ages between 12 and 16 years. In each case, genetic factors accounted for more stability than any environmental factors. This pattern was replicated at age 12 in a study of a laboratory-based measure of impulsivity, but, notably, reversed at later ages where non- shared environmental factors accounted for most (55% and 75% respectively) of the wave-to-wave stability at ages 15 and 17 (Bezdjian et al., 2014). Non-shared environmental factors also became increasingly stable across adolescence in one study (Kan et al., 2013). Although phenotypic stability in ADHD and related behaviours generally remained moderate to high into adolescence, change was evident and was predominantly accounted for by a combination of genetic and non-shared environmental factors. In studies where estimates of shared environmental factors were significant, their contribution to change was generally small or non-significant (e.g., Larsson et al., 2004; Lewis & Plomin, 2015). The results regarding the relative magnitudes of new genetic and non-shared environmental influences emerging for ADHD symptoms across adolescence were mixed. In some studies (Bezdjian et al., 2014; van den Oord & Rowe, 1997) few or no new genetic influences were found, while in others (Chang, Lichtenstein, Asherson, & Larsson, 2013; Jaffee et al., 2012; Lewis & Plomin, 2015) new genetic influences were of greater magnitude than contemporary non- shared environmental influences.

Aggression, anger, antisocial behaviour and conduct problems—Twenty studies involved phenotypes relating to aggression, anger, antisocial behaviour (ASB), or conduct problems. Of these, seven involved longitudinal data on aggression or anger, incorporating data from children between the ages of 1 and 14 years (Eley, Lichtenstein, & Moffitt, 2003; Gagne & Hill Goldsmith, 2011; Haberstick, Schmitz, Young, & Hewitt, 2006; Lacourse et al., 2014; Tuvblad, Raine, Zheng, & Baker, 2009; van Beijsterveldt, Bartels, Hudziak, & Boomsma, 2003; Vierikko, Pulkkinen, Kaprio, & Rose, 2006). In early childhood, anger and aggression were found to be only moderately stable (*r*Ph range: .03-.48 up to age 7; Gagne & Hill Goldsmith, 2011; Lacourse et al., 2014; van Beijsterveldt et al., 2003). Stability was again predominantly genetic in this period, although stable shared environmental factors of roughly equivalent magnitude were found in one study (van Beijsterveldt et al., 2003). New genetic, shared and non-shared environmental factors influenced change in infant anger (Gagne & Hill Goldsmith, 2011) and aggression (Lacourse et al., 2014; van Beijsterveldt et al., 2003) in this period. In the remaining four studies of aggression (Eley et al., 2003; Haberstick et al., 2006; Tuvblad et al., 2009; Vierikko et al., 2006), from age 8 onwards, stability was generally higher, with genetic factors again accounting for the majority of this continuity. For example, in middle childhood, Haberstick et al. (2006) found that more than 75% of the stability in parent (*r*Ph range: .61-.78) and teacher (*r*Ph range: .33-.58) reports of aggressive behaviour between the ages of 8 and 12 years was explained by stable genetic factors. Two studies (Eley et al., 2003; Vierikko et al., 2006) included data from mid-

adolescence, but these studies had very different findings. In the first, stable genetic factors accounted for a substantial amount of the moderate-to-high stability of aggressive behaviour (84% of *r*Ph: .61) but little of the phenotypic change, which was attributable to both shared and non-shared environmental factors (Eley et al., 2003). In the other, aggression showed only low stability, influenced by stable environmental factors, with change resulting from new genetic factors (Vierikko et al., 2006). Methodological differences between these studies may offer a partial explanation for their discrepant findings. Specifically, the use of parent- and teacher-report respectively raises the possibility that these measures used in these studies may tap different, situation-specific behaviours. Nine studies, with a collective age range of 4-17 years, included analyses of aetiological stability and change in measures of generalised or nonaggressive antisocial behaviour (ASB; Eley et al., 2003; Harden, Quinn, & Tucker-Drob, 2012; Neiderhiser, Reiss, & Hetherington, 1996; Niv, Tuvblad, Raine, & Baker, 2013; O'Connor et al., 1998; Tuvblad, Eley, & Lichtenstein, 2005; van den Oord & Rowe, 1997; Van Hulle et al., 2009). One of these studies (Neiderhiser et al., 1996) presents analyses that are also included, in greater detail, in another study (O'Connor et al., 1998). Accordingly, only the latter is presented in the table and included in this overview. The results of the studies of antisocial behaviour suggest a pattern of moderate stability underpinned by both genetic *and* shared environmental influences (e.g. Niv et al., 2013; O'Connor et al., 1998; Tuvblad, Narusyte, Grann, Sarnecki, & Lichtenstein, 2011). To the extent that phenotypic change occurred across development, environmental factors - both shared and non-shared - were found to play a driving role. New genetic influences were generally of a smaller magnitude, but were present in mid-adolescence in particular. For example, in a study by Tuvblad, Eley, & Lichtenstein (2005), variance in boys' non-aggressive antisocial behavior at age 14 was primarily explained by new aetiological factors, with new genetic factors explaining 28% of the variance and new shared and non-shared environmental factors explaining 38% and 30% respectively. One notable exception was in Niv et al. (2013), where new genetic factors were more influential at age 14-15 than new non-shared environmental factors. Five studies used measures of conduct problems, spanning an age range of 4-17 years (Jacobson, Prescott, & Kendler, 2002; Jaffee et al., 2012; Lahey et al., 2009; Lewis & Plomin, 2015; Van Hulle et al., 2009). As in the group of studies of antisocial behaviour, significant duplication between two of these studies (Lahey et al., 2009; Van Hulle et al., 2009) mean that only the one with the greatest detail is presented in the table (Van Hulle et al., 2009). Overall, stability in conduct problems in childhood and adolescence was typically moderate-to-substantial, with wave-to-wave phenotypic correlations exceeding .45 in three studies (Jacobson et al., 2002; Jaffee et al., 2012; Lewis & Plomin, 2015). This stability was primarily genetically mediated, although stable shared and non-shared environmental factors were sporadically implicated across all studies. In terms of change, one study (Van Hulle et al., 2009) found that change in conduct problems in middle childhood and adolescence was entirely explained by a combination of shared and (primarily) non-shared environmental factors, with no new genetic influences coming online. However, the remaining studies all found additional genetic (as well as environmental) contributions to change, with new genetic factors explaining at least one fifth of variance at each wave, in all cases (Jacobson et al., 2002; Jaffee et al., 2012; Lewis & Plomin, 2015).

Behavioural problems (general)—Eight studies included general measures of behavioural problems, typically described as 'externalising behaviour', collectively spanning the ages of 2-17 years (Bartels, Boomsma, et al., 2004; Haberstick et al., 2005; Huizink et al., 2007; Schmitz et al., 1995; van der Valk et al., 2003, 1998; Wang et al., 2012; Wichers et al., 2013). In the studies that investigated the *childhood* aetiology of general behavioural problems, a pattern of mixed genetic and environmental stability was found. Evidence that stability in externalising behaviour between the ages of 3 and 10 years was the result of substantial stable shared environmental and genetic factors was found in three separate studies (Bartels, van den Oord, et al., 2004; Haberstick et al., 2005; van der Valk et al., 2003). Somewhat contrasting evidence was found in the one remaining study with data from this developmental period, in which environmental contributions to stability were non-shared, and relatively minor (Wang et al., 2012). Genetic contributions to stability were estimated to be substantial (explaining >30% variance) in all four studies, while the aetiology of phenotypic change was predominantly genetic and non-shared environmental in all cases. The results for general behavioural problems across *adolescence* were similarly consistent with respect to the origins of phenotypic stability. Genetic factors were again predominant, accounting for, on average, 79% of the phenotypic stability across three separate studies incorporating data from individuals aged 10-17 years (Huizink et al., 2007; van der Valk et al., 1998; Wichers et al., 2013). Genetic innovation was found to be important for driving phenotypic change in two of these three studies (van der Valk et al., 1998; Wichers et al., 2013), and also earlier, at age 12, in two further studies (Bartels, van den Oord, et al., 2004; Haberstick et al., 2005). Non-shared environmental contributions to change were found to be significant in all but one study (Schmitz et al., 1995), and were generally small-to-moderate in magnitude.

Summary

Studies of behavioural problems were typically designed to investigate either attention/hyperactivity related problems or aggressive/antisocial behaviour and conduct problems. Generalised measures of behavioural problems, such as externalising, were used in comparatively fewer studies. Overall, a pattern of predominant genetic influence on stability across development, for all behavioural problem phenotypes, was observed. The magnitude of this stability varied between phenotypes and across different ages. Additionally, environmental influences on stability were observed relatively consistently for some phenotypes (e.g., antisocial behaviour) and not for others (e.g. attention problems). Change was underpinned, variously, by genetic, shared and non-shared environmental factors, with limited consistency across different behavioural problem phenotypes. For the broadest measures of behavioural problems, including non-aggressive antisocial behaviour, new variance across childhood and adolescence was predominantly accounted for by genetic and non-shared environmental factors.

Discussion

Summary of findings and relation to the broader literature

We identified 58 longitudinal behavioural genetic studies of emotional and behavioural problems in this systematic review. Overall, the findings from the studies included were very

consistent. The majority found stability of psychopathology in childhood and adolescence to be primarily genetically mediated. This is in keeping with the aetiological and developmental picture established by previous work in the field, notably the significant genetic influence found on emotional and behavioural phenotypes from cross-sectional genetic studies at all ages (Lau & Eley, 2010; Middeldorp, Cath, Van Dyck, & Boomsma, 2005; Rhee & Waldman, 2002; Rice et al., 2002; Rutter et al., 1999); and the moderate stability of emotional and behavioural problems across childhood and adolescence (Costello et al., 2003; Hofstra et al., 2000; Ollendick et al., 1994).

Where stability due to environmental influences was found, it was generally of smaller magnitude (e.g. generalised internalising: Bartels, van den Oord, et al., 2004; Huizink et al., 2007; Kendler, Gardner, & Lichtenstein, 2008; antisocial behaviour: Eley et al., 2003; Harden et al., 2012; Jacobson et al., 2002; Niv et al., 2013). In the case of the shared environment, these results should be considered with respect to power demands, which are particularly high for estimating 'C' as has been discussed in detail elsewhere (Burt, 2009, 2014). Stability from both shared and non-shared environmental factors was seen, across results from studies of both emotional and behavioural problems.

This affirms that environmental factors can operate to produce phenotypic stability irrespective of whether they make individuals in the same family more or less similar. Previous research has identified some specific, stable sources of environmental influence on children's behaviour (e.g., SES, neighbourhood characteristics, parenting characteristics; Bradley, Corwyn, McAdoo, & Coll, 2001; Dumas et al., 2005; see Burt, 2014 for a review). Systematic rater effects could also play a role in producing phenotypic stability in some cases, although evidence from studies using multiple raters confirms that this does not account for all environmental stability.

Most studies found substantial phenotypic change, as well as stability, across development. In many cases, this was partially underpinned by genetic innovation. Genetically influenced phenotypic change can be the result of age-related biological changes, whether relating to brain development or hormonal variations (Blakemore, 2008; Kadosh, Linden, & Lau, 2013; Paus et al., 2008). In addition, the shifting importance of various social and environmental contexts for developing children and adolescents provides opportunities for new genetic influence to be mediated through *gene-environment interplay* - processes by which individuals' exposure and/or sensitivity to environmental factors is influenced genetically (Knafo & Jaffee, 2013; Rutter & Silberg, 2002; Scarr & McCartney, 1983). Aspects of this interplay have also been hypothesised (Scarr & McCartney, 1983), and shown (e.g., Brody et al., 2009; Elkins, McGue, & Iacono, 1997; Hannigan, McAdams, Plomin & Eley, 2016), to change across development. The idea that these processes may be especially important for phenotypic change in childhood and adolescence is supported by the relative lack of genetic innovation found in similar studies of adults (Gillespie et al., 2004; Kan et al., 2013; Nes, Røysamb, Reichborn-Kjennerud, Harris, & Tambs, 2007; Nivard et al., 2015; Takahashi et al., 2007; Van Den Berg, Willemsen, De Geus, & Boomsma, 2006). However, it is noteworthy that significant new genetic influences are found in young adulthood for some phenotypes (e.g. antisocial behaviour and alcohol abuse: Malone, Taylor, Marmorstein, McGue, & Iacono, 2004; obsessive-compulsive symptoms: van Grootheest, Cath, Hottenga,

Beekman, & Boomsma, 2009), suggesting some persistence of these processes beyond what is generally regarded as the end of the adolescent developmental period.

Phenotypic change was driven by non-shared environmental factors in the majority of studies. The non-shared environmental parameter in behavioural genetic studies includes time-specific measurement error, which could play a role in increasing the appearance of phenotypic discontinuity. However, non-shared environmental innovation was found to be substantial across studies using a range of measurement techniques in several different samples. Furthermore, the influence of 'true' non-shared environmental factors in driving phenotypic change is consistent with theoretical accounts of development, which emphasise the role of both stochastic and specific (e.g. illness, accidents, clinical interventions, different school experiences) influences in making children in the same family different from one another (Asbury, Dunn, Pike, & Plomin, 2003; Asbury, Dunn, & Plomin, 2006; Plomin, 2011; Turkheimer & Waldron, 2000).

Implications for researchers and clinicians

The predominance of genetic influence on stability in specific childhood and adolescent emotional and behavioural problems, as outlined in this review, has implications for genetic and developmental researchers alike. It is likely that the majority of genes affecting psychopathology in development have a broad effect across multiple traits and different life stages. As a result, extracting factors that reflect common variance across different traits and times may be beneficial in aiding the identification of these “general” genes (Eley, 1997). Shared genetic liability has long been viewed as at least a partial explanation for the widespread comorbidity in childhood psychopathology, including *between* emotional and behavioural problems (Caspi et al., 2013; Nadder, Rutter, Silberg, Maes, & Eaves, 2002; Silberg, Rutter, & Eaves, 2001). Substantial evidence for *pleiotropy* - whereby genes affect multiple different traits - has also emerged from molecular genetic studies (Hettema, Chen, Sun, & Brown, 2015; Trzaskowski et al., 2013). To date only a handful of studies have examined the extent to which this shared genetic influence operates developmentally to produce *heterotypic* continuity in emotional and behavioral problems (e.g. Roberson-Nay, Eaves, Hettema, Kendler, & Silberg, 2012; Silberg, Rutter, & Eaves, 2001; Waszczuk et al., 2016; Wertz et al., 2015). Future work exploring the overlap, and potential distinction, between genetic influences on homotypic and heterotypic continuity in these phenotypes is warranted. Identifying sources of common genetic liability to childhood and adolescent psychopathology *and* subsequent problems in adulthood also remains a priority. In the context of developmental psychopathology more broadly, researchers should aim to incorporate an awareness of the underlying genetic stability of many aspects of childhood and adolescent psychopathology into study design and the interpretation of results wherever possible.

While genetic innovation was found to be less prevalent than stability, it is of comparable developmental significance, providing an important source of change in symptomatology (Kendler, Gardner, & Lichtenstein, 2008; Kendler, Gardner, Annas, et al., 2008). Similarly, attenuation of genetic factors from early in development is also commonly seen alongside genetic innovation, in spite of the overall trend for heritability to increase with age (Bergen

et al., 2007). This combination of innovation and attenuation suggests that, despite early temperamental and behavioural factors predicting later life problems, their respective genetic aetiologies may still differ to a substantial degree. This has wide-ranging implications for research. For example, for molecular genetic approaches, which aim to identify specific regions of the genome associated with mental health problems, this may indicate that some genes are important only at one developmental stage. In order to identify such genes, approaches involving longitudinal data or the stratification of cross-sectional samples by age will be necessary (Traylor, Markus, & Lewis, 2014). Furthermore, the mechanisms through which genetic factors 'coming online' throughout development may contribute to the emergence of clinical psychopathology in late adolescence and young adulthood are not well understood, and could prove to be fruitful areas for further work.

The evidence presented in this review also has implications for clinicians. Most importantly, from a clinical perspective, the finding of genetic stability underpinning the development of emotional and behavioural problems should not be viewed deterministically. Genetic influence on individual differences in measures of psychopathology is indicative of the different levels of risk associated with inherited genetic factors. Understanding the extent to which these risk factors are the same across development is an important step in uncovering the mechanisms that underpin the emergence of psychiatric illnesses. However, genetic influence, stable or otherwise, does not preclude the possibility of effective treatment. This is most clearly exemplified by entirely genetic conditions, such as phenylketonuria (PKU), for which the routine and successful treatment is entirely environmental (limitations to diet). This example also demonstrates the benefit of being able to identify individuals who are at an increased genetic risk for a particular disorder. Although this is not yet a possibility for the kind of psychiatric problems associated with childhood and adolescent psychopathology, evidence of substantial genetic stability across development, as reviewed here, indicates that such an approach could facilitate early intervention in the future. Furthermore, recent work has begun to explore the possibility of using genetic information to predict the *kind* of treatment to which individuals may respond best (Eley et al., 2012; Lester & Eley, 2013). The potential value of this approach is again underlined by predominant genetic stability in developmental psychopathology. Environmental stability, where evident, should also be considered as important from a clinical perspective. To the extent that these stable environmental factors can be identified and are modifiable, interventions targeting them can be expected to have a lasting effect across development.

Understanding the aetiology of developmental change in psychopathology, in adolescence in particular, may provide clinically-relevant insights as to how risk for psychiatric disorders in young adulthood and beyond is mediated (Gregory et al., 2007; Moffitt & Caspi, 2001; Rutter et al., 2006). Genetic innovation for behavioural and emotional problems suggests that there are periods during development where individual differences in psychopathology are the result of genetically influenced behavioural or physiological changes. The evidence from studies in this review indicates that these potentially 'sensitive' periods occur throughout development, and certainly not limited to adolescence. This may indicate that the window for effective clinical intervention in childhood and adolescence may be equally wide. Understanding the nature of the underlying behavioural changes that result in genetic innovation in longitudinal genetic studies may also allow us to highlight *specific* behaviours

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as targets for interventions. The widespread evidence of environmental influences on change in measures of child and adolescent psychopathology could also have potential clinical significance. Firstly, this is because *specific* environmental factors may again be identifiable and modifiable. Secondly, because evidence of variability in these measures in childhood and adolescence reinforces the notion that, even in the context of genetic predisposition for psychiatric disease, external (i.e. environmental) factors can account for a substantial amount of variance in these phenotypes. The effects of a clinical intervention would likely appear in a similar way, if modelled in a genetically informative design. However, the largely time- specific nature of environmental influence on developmental psychopathology suggests that, for such interventions to be successful in the long-term, they may need to be actively maintained.

Strengths and limitations of the review

This review has several strengths, most notably that it is (to our knowledge) the first review of longitudinal behavioural genetic results for both emotional and behavioural problems. It thus provides a comprehensive overview of many years of research into the developmental aetiology of child and adolescent psychopathology. The coverage of the age span specified (0-18 years) is also unusually broad (see Figure 2). We believe that this makes the review a particularly useful resource for both genetic *and* developmental research and clinical practice.

The main limitation of this review concerns the different approaches taken to analysis in the studies, combined with the wide variety of measures used, which precluded meta-analysis. Our decision to incorporate a wide range of phenotypes from the field of developmental psychopathology made this limitation somewhat unavoidable, but we believe that it is largely counter-balanced by the benefits of presenting and reviewing these studies together. In particular, the broadly consistent nature of findings, in spite of considerable methodological heterogeneity (even within phenotypes), is evident and quite striking - even without the benefits of a meta-analysis. Other issues include methodological limitations of the individual studies, such as the heavy reliance in this field on questionnaire data, and related issues such as rater-bias and variations in reliability. Addressing such issues is beyond the scope of this review. More fundamental limitations and assumptions associated with twin and adoption studies and structural equation modelling remain as relevant as in the individual studies themselves. These have been explored and discussed in detail elsewhere (Derks, Dolan, & Boomsma, 2006; LoParo & Waldman, 2014; Plomin, Willerman, & Loehlin, 1976; Tomarken & Waller, 2005).

Conclusion and future directions

Overall, this review highlights several key ways in which genetically informative, developmentally-contextualised research into child and adolescent emotional and behavioural problems can inform the wider study of psychopathology. The morbidity and disruption associated with these problems at the time they present may be justification enough for studying their aetiologies, and the fact that they also represent significant risk factors for later life psychopathology only strengthens the case for this. However, ascertaining the mechanisms and pathways by which this risk is mediated will necessarily

require developmentally-contextualised approaches. The results of investigations into the aetiological architecture of stability and change in emotional and behavioural problems provide a strong empirical base for this process and, used in combination with findings from molecular, epidemiological, cognitive-experimental and clinical studies, can continue to play an important role in future.

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Box 1

Structural equation modelling in twin studies

The classical twin design involves comparing the degree of similarity between MZ (sharing 100% of their genes) and DZ (sharing on average 50% of their segregating genes) twin pairs. These relative differences in within-pair correlations allow estimations of the influences caused by additive genetic (A), shared environmental (C) and non-shared environmental (E) components of variance. The components can be estimated as follows:

$$A = 2(\mathrm{rMZ} - \mathrm{rDZ})$$
$$C = \mathrm{rMZ} - A$$
$$\mathrm{E} = 1 - \mathrm{A} + \mathrm{C}$$

The A component of variance captures the summed influence of all additive genetic effects on the phenotype and is evident when MZ twins are more phenotypically similar than DZ twins. C captures all non-genetic (i.e., environmental) similarity and is evident when DZ twin correlations are greater than half the magnitude of MZ twin correlations. E captures all non-genetic influences that make individuals in the same family different from one another, including measurement error. E is evident in the extent to which MZ twins, sharing all their genes and rearing environment, do not correlate perfectly for a given phenotype. Figure A shows an example of a univariate twin model, in which these components are estimated.

In cases where the extent of genetic similarity for a phenotype exceeds that which would be expected from the additive genetic overlap (e.g., for the twin model, if rDZ < 0.5 * rMZ), non-additive influences (D) are indicated and can be estimated instead of C. When estimated, D component of variance captures the effect of any interactions between alleles within (dominance) and across (epistasis) loci. For a more detailed introduction to structural equation modelling in behavioural genetics, see Plomin et al. (2013) and Rijsdijk & Sham (2002).

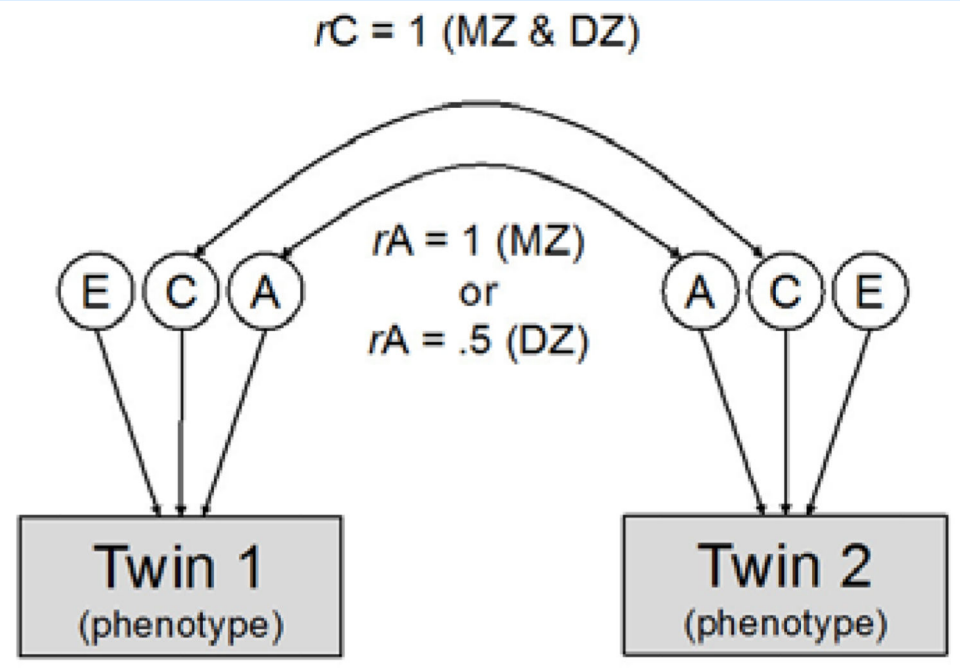

Figure A. Univariate twin model

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Box 2

Behavioural genetic analysis of longitudinal data: Cholesky decomposition, correlated factors solution and simplex model

In a longitudinal Cholesky decomposition (Figure B), the relative influence of stable, pre-existing aetiological factors is equivalent to the squared parameter estimates for cross-time paths into a variable (e.g., for T3, the paths running from A1/C1/E1 and A2/C2/E2 to T3). Similarly, the influence of new aetiological factors at T3 is equal to the squared estimates of the paths between variance components A3/C3/E3 and variable T3.

Where Cholesky decompositions were not presented in the studies, several sets of results could be presented comparably, thanks to the mathematical equivalence of other commonly used models (see Loehlin, 1996 for a full explanation). For example, Cholesky-equivalent estimates of aetiological contributions to stability and change can therefore be derived from the correlated factors solution (Figure C) and the simplex model (Figure D). The simplex model differs only in its separation of innovative effects (e.g., Ai), which can be transmitted along lateral paths, and time-specific effects (e.g., As), which are unique to one wave only.

Results from these models are presented entirely comparably in the majority of cases. For an outline of the differences in the conceptualisation of stability and change associated with common and independent pathway models, see Box 3.

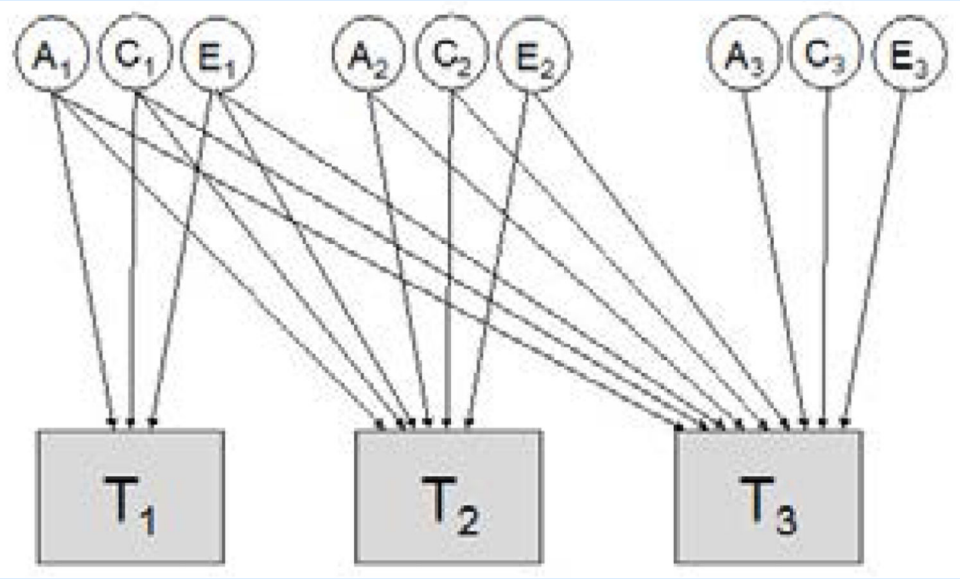

Figure B. Longitudinal Cholesky decomposition

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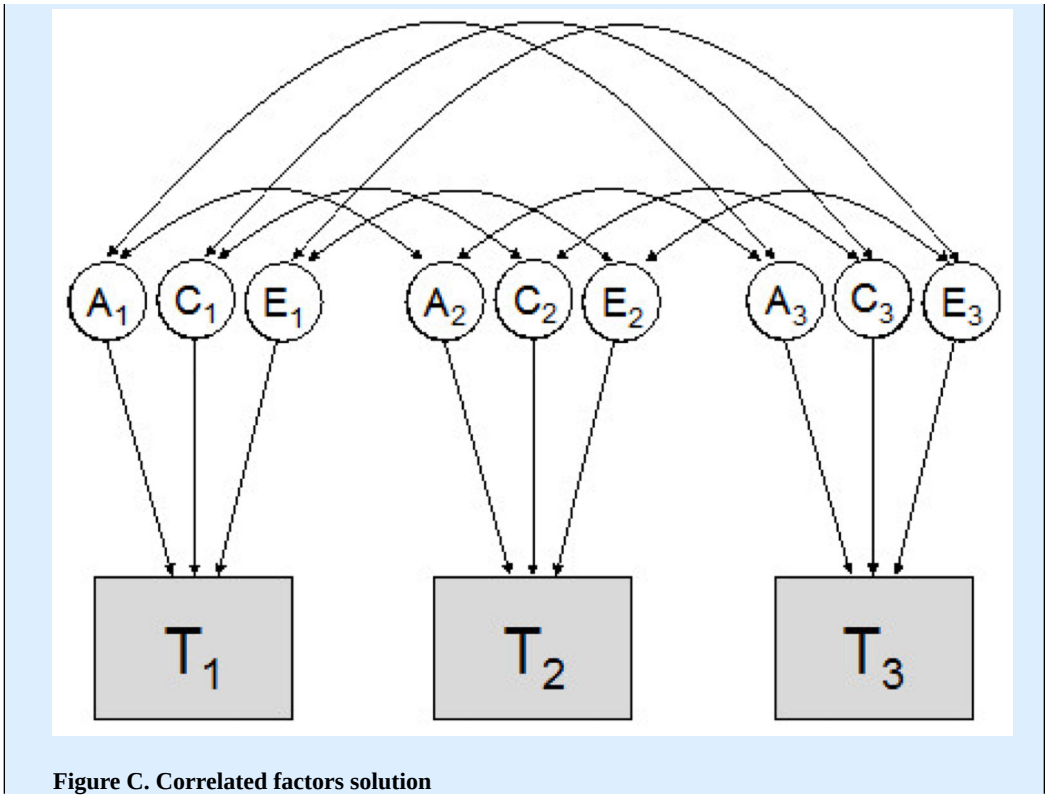

Figure C. Correlated factors solution

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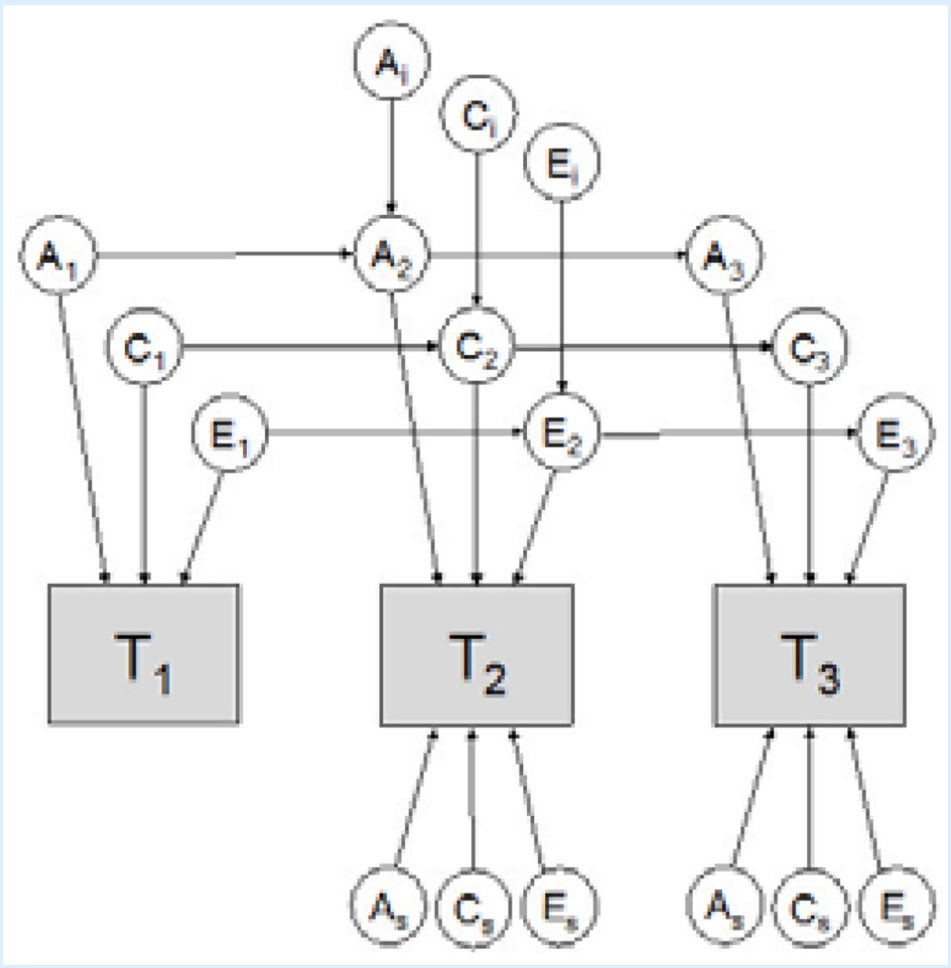

Figure D. Simplex model

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Box 3

Behavioural genetic analysis of longitudinal data: Common and independent pathway models

Figure E shows an independent pathway model, which was used in several studies included in this review. In this model, aetiological influences are sub-divided into those that are common to all waves (AC, CC, EC) and those specific to each wave (AS, CS, ES). In this way, the conceptualisation of aetiological stability is subtly different to the Cholesky decomposition and related models. In the independent pathway model, aetiological stability is estimated by squaring the path coefficient from a common aetiological factor (e.g., AC) to an observed variable (e.g. T2). New aetiological influences are time specific variance components at, in this example, T2 and T3.

The common pathway model (Figure F) is a more constrained model, for which the conceptualisation of stability and change is similar to the independent pathway model. Stable variance common to all waves is modelled as an underlying latent factor (L), with one set of variance components (AC, CC, EC). Stable aetiological influence at a specific wave is therefore represented by these variance components, weighted by the path loading between the latent factor and the observed variable.

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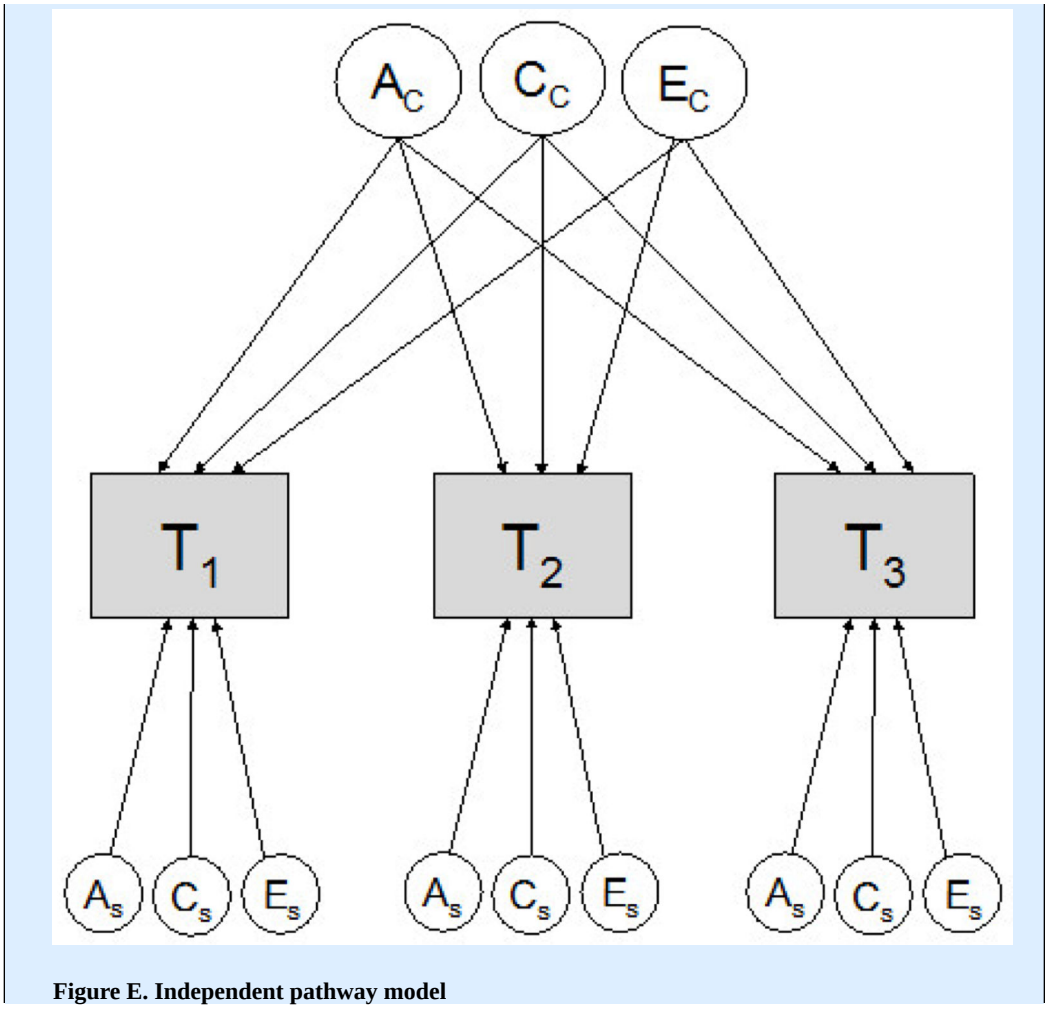

Figure E. Independent pathway model

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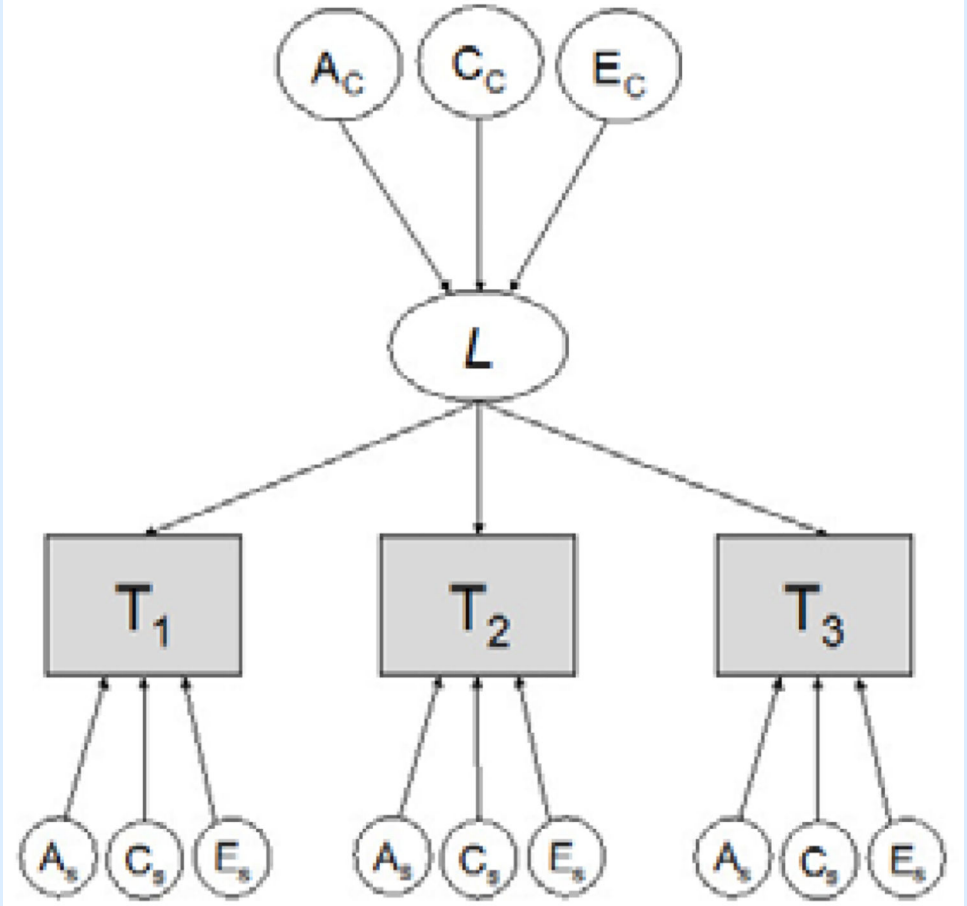

Figure F. Common pathway model

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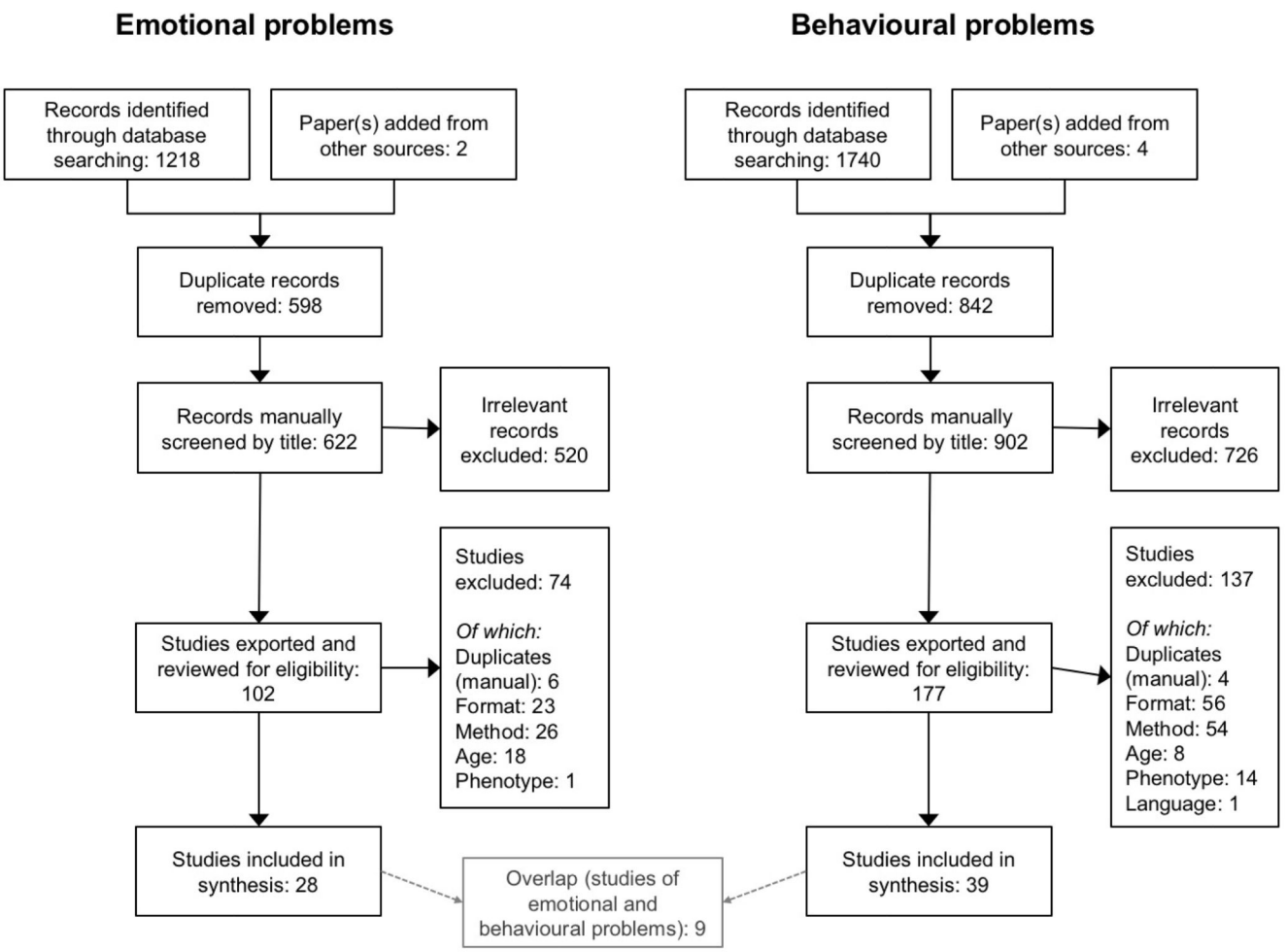

Figure 1. Selection process for inclusion of studies in the review synthesis

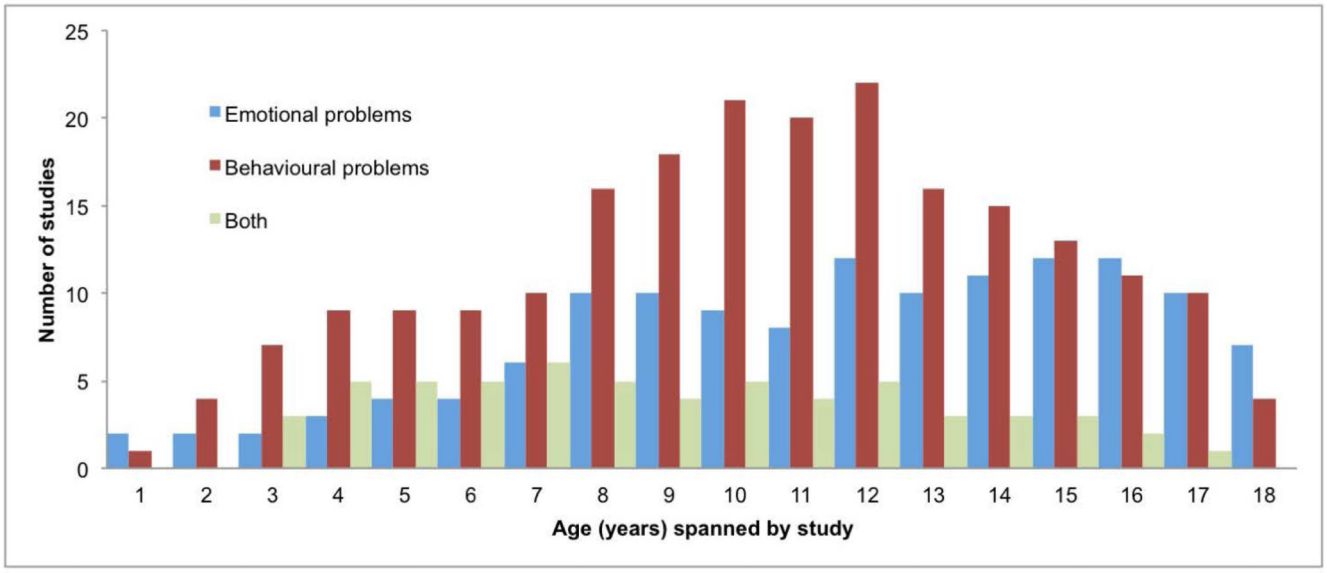

Figure 2. Frequency distribution showing the number of included studies at each age between 0-18 years

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Table 1

Included longitudinal quantitative genetic studies of emotional problems in childhood and adolescence

Emotional problems

Study	Sample (Name, N)	Phenotype(s) (measure; reporter)	Age at each wave[a]	Genetic model[b]	Phenotypic stability (*% due to genes where calculable*)	Influence of stable genetic factors[c]	Influence of stable environmental factors[d] C / E	Influence of new genetic factors[e]	Influence of new environmental factors[f] C / E
Bartels et al. (2004)[g]	Netherlands Twin Registry (NTR): 1481 - 5115 twin pairs	Internalising behaviour (CBCL; parent)	W1: 3 years	Developmental model	W1-W2: .38 *(52%)*	6%	19% / 1%	38%	9% / 27%
			W2: 7 years		W1-W3: .34 *(43%)* W2-W3: .62 *(51%)*	22%	25% / 3%	14%	9% / 28%
			W3: 10 years W4: 12 years		W1-W4: .31 *(32%)* W2-W4: .57 *(40%)* W3-W4: .68 *(38%)*	18%	29% / 8%	19%	8% / 18%
Bolhuis et al. (2014)	Genesis 1219 study (G1219): 1597 - 2651 *individual* twins	Depressive symptoms & obsessive compulsive symptoms (SMFQ / SCAS; self / self)	W1: *M* = 15 years (12-21)	Cholesky decomposition	Depressive symptoms (DS)				
			W2: *M* = 17 years (14-23)		W1-W2: .44 *(64%)*	18%	(2%)/3%	(0%) Controlling for: W1 OCS	(12%)/55% Controlling for: W1 OCS
					Obsessive compulsive symptoms (OCS)				
					W1-W2: .48 *(63%)*	18% Controlling for: W1 DS	(0%) / 4% Controlling for: W1 DS	(0%) Controlling for: W1&2 DS	(0%) / 52% Controlling for: W1&2 DS
Cherny, Fulker, Corley, Plomin, & DeFries (1994)	MacArthur Longitudinal Twin Study: 301 twin pairs	Shyness (home observation / laboratory task; observer)	W1: 1.2 years W2: 1.7 years	Common Pathway model (developmental)	Home observation:				
					W1-W2: .35 *(92%)*	23%	1% / 0%	0%	4% / 72%
					Laboratory task:				
					W1-W2: .29 *(55%)*	10%	7% / 0%	0%	25% / 58%
Garcia et al. (2013)	Minnesota Twin Family Study (MTFS): 756 twin pairs	Trait anxiety (STAI-C at W1 / STAI at W2 & W3; self)	W1: *M* = 14.18 years (*SD* = .51) W2: *M* = 18.16 years (*SD* = .65)	Cholesky decomposition	W1-W2: .42 (58%)	39%	(1%) / 4%	(0%)	(0%) / 55%

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Emotional problems

Study	Sample (Name, N)	Phenotype(s) (measure; reporter)	Age at each wave[a]	Genetic model[b]	Phenotypic stability (*% due to genes where calculable*)	Influence of stable genetic factors[c]	Influence of stable environmental factors[d] C / E	Influence of new genetic factors[e]	Influence of new environmental factors[f] C / E
			[W3: *M* = 21.46 years (*SD* = .77)]						
Haberstick, Schmitz, Young, & Hewitt (2005)[g]	Longitudinal Twin Study (LTS): 175-252 twin pairs	Internalising behaviour (TRF; teacher)	W1: 7 years	Developmental model	(Average across sex)				
			W2: 8 years		W1-W2: .30 *(100%)*	25%	0%	23%	52%
			W3: 9 years		W1-W3: .21 *(100%)* W2-W3: .33 *(100%)*	23%	0%	16%	61%
			W4: 10 years		W1-W4: .19 *(100%)* W2-W4: .28 *(100%)* W3-W4: .36 *(100%)*	30%	0%	6%	64%
			W5: 11 years						
			W6: 12 years		W1-W5: .26 *(100%)* W2-W5: .28 *(100%)* W3-W5: .28 *(100%)* W4-W5: .28 *(100%)*	25%	0%	12%	62%
					W1-W6: .14 *(100%)* W2-W6: .22 *(100%)* W3-W6: .28 *(100%)* W4-W6: .29 *(100%)* W5-W6: .38 *(100%)*	28%	0%	10%	62%
Hoekstra, Bartels, Hudziak, Van Beijsterveldt, & Boomsma (2008)	Netherlands Twin Register: 14889 twin pairs	Withdrawn Behaviour (CBCL; mother & father)	W1: 3 years	Cholesky decomposition (rater bias model)	Rater-agreed[h] withdrawn behaviour:				
			W2: 7 years		Males:				
			W3: 10 years		W1-W2: .30 *(87%)*	77%	0% / 1%	18%	0% / 2%
			W4: 12 years		W1-W3: .27 *(69%)* W2-W3: .56 *(50%)*	48% 25%	1% / 4% 0% / 0%	5%	0% / 1%
					W1-W4: .28 *(77%)* W2-W4: .55 *(68%)* W3-W4: .63 *(36%)*	59% 46% 13%	1% / 1% 0% / 6% 0% / 0%	7%	0% / 1%
					Females:				
					W1-W2: .29 *(72%)*	52%	1% / 1%	16%	0% / 1%
					W1-W3: .27 *(60%)* W2-W3: .56 *(39%)*	36% 15%	6% / 7% 0% / 0%	3%	0% / 1%

Europe PMC Funders Author Manuscripts

Emotional problems

Study	Sample (Name, N)	Phenotype(s) (measure; reporter)	Age at each wave[a]	Genetic model[b]	Phenotypic stability (*% due to genes where calculable*)	Influence of stable genetic factors[c]	Influence of stable environmental factors[d] C / E	Influence of new genetic factors[e]	Influence of new environmental factors[f] C / E
					W1-W4: .25 *(59%)* W2-W4: .48 *(63%)* W3-W4: .58 *(27%)*	35% 39% 7%	7% / 2% 0% / 6% 1% / 0%	5%	0% / 1%
Huizink, van den Berg, van der Ende, & Verhulst (2007)[g]	Dutch sample of adopted siblings: 106 biologically-related (BR) and 230 unrelated (BUR) pairs	Internalising behaviour (CBCL; adoptive parent)	W1: BR *M* = 12.5 years (SD = 1.2); BUR M = 12.4 years (SD = 1.2) W2: BR M = 15.8 years (SD = 1.2); BUR M = 15.6 years (SD = 1.2) [W3: 26 years]	Common pathway model (developmental)	W1-W2: .57 *(18%)*	23%	24% / 34%	(0%)	(0%) / 20%
Kendler, Gardner, Annas, et al. (2008)	Twin Study of Child and Adolescent Development (TCHAD): 1237 twin pairs, 16 single twins	Situational, animal, blood/injury fears (12-item rating scale; parent & self)	W1: 8-9 years (parent report only) W2: 13-14 years W3: 16-17 years [W4: 19-20 years]	Cholesky decomposition (rater bias model)	Rater-agreed[h] situational fears:				
					W1-W2: .44 *(81%)*	25%	0% / 6%	44%	5% / 20%
					W1-W3: .39 *(87%)* W2-W3: .78 (76%)	23% 28%	0% / 1% 0% / 13%	18%	4% / 12%
					Rater-agreed[h] animal fears:				
					W1-W2: .52 *(31%)*	8%	14% / 10%	36%	28% / 5%
					W1-W3: .53 *(15%)* W2-W3: .88 *(48%)*	2% 41%	22% / 12% 7% / 2%	2%	9% / 3%
					Rater-agreed[h] blood/injury fears:				
					W1-W2: .39 *(59%)*	12%	3% / 2%	55%	15% / 14%
					W1-W3: .30 (90%) W2-W3: .80 (79%)	17% 44%	0% / 0% 1% / 10%	13%	0% / 16%
Kendler, Gardner, & Lichtenstein (2008)	Twin Study of Child and Adolescent Development (TCHAD): 1237 twin pairs, 16 single twins	Mixed anxiety and depression (CBCL / YSR; parent / self)	W1: 8 to 9 years (parent report only) W2: 13 to 14 years W3: 16 to 17 years [W4: 19 to 20 years]	Cholesky decomposition (rater bias AE model)	Rater-agreed[h] mixed anxiety and depression:				
					W1-W2: .51 *(77%)*	21%	5%	69%	6%
					W1-W3: .48 *(85%)* W2-W3: .83 *(85%)*	23% 34%	(2%) 14%	28%	0%

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Emotional problems

Study	Sample (Name, N)	Phenotype(s) (measure; reporter)	Age at each wave[a]	Genetic model[b]	Phenotypic stability (*% due to genes where calculable*)	Influence of stable genetic factors[c]	Influence of stable environmental factors[d] C / E	Influence of new genetic factors[e]	Influence of new environmental factors[f] C / E
Krebs, Waszczuk, Zavos, Bolton, & Eley (2014)	Twins Early Development Study (TEDS): 3644 - 7834 twin pairs	Obsessive Compulsive behaviour (ARBQ; Parent)	W1: *M* = 4.04 years (*SD* = .13)	Cholesky decomposition	W1-W2: .45 *(75%)*	19%	(1%) / (3%)	42%	(1%) / 35%
			W2: *M* = 7.07 years (*SD* = .25)		W1-W3: .35 *(80%)* W2-W3: .44 *(70%)*	8% 13%	(0%) / (1%) (14%) / 4%	33%	(0%) / 27%
			W3: *M* = 9.02 years (*SD* = .29)		W1-W4: .23 *(78%)* W2-W4: .32 *(59%)* W3-W4: .37 *(72%)* W4: *M* = 16.32 years (*SD* = .68)	6% (2%) 8%	(25%) / (0%) (0%) / 1% (0%) / 2%	17%	(0%) / 38%
Lau & Eley (2006)	Genesis 1219 study (G1219): 1820 twin pairs	Depressive symptoms (sMFQ; self)	W1: *M* = 14 years (12-19 years)	Cholesky decomposition	W1-W2: .58 *(68%)*	26%	(5%) / 4%	10%	(7%) / 48%
			W2: *M* = 15 years (12-21)		W1-W3: .40 *(76%)* W2-W3: .45 *(62%)*	16% 27%	(1%) / 3% (1%) / (1%)	0%	(0%) / 51%
			W3: *M* = 17 years (13-23)						
Lewis & Plomin (2015)[g]	Twins Early Developmental Study (TEDS): >3000 twin pairs	Anxiety / peer problems (SDQ; parent)	W1: 4 years	Cholesky decomposition	Anxiety:				
			W2: 7 years		W1-W2: .38 *(69%)*	14%	6% / 1%	30%	10% / 37%
			W3: 9 years		W1-W3: .38 *(58%)* W2-W3: .51 *(51%)*	10% 6%	17% / 0% (0%) / 5%	29%	(1%) / 30%
			W4: 12 years		W1-W4: .31 *(73%)* W2-W4: .45 *(66%)* W3-W4: .51 *(60%)*	11% 10% 5%	7% / 0% (0%) / 2% (1%) / 4%	26%	(0%) / 35%
					Peer problems:				
					W1-W2: .29 *(89%)*	10%	(2%) / 1%	50%	(1%) / 37%
					W1-W3: .26 *(100%)* W2-W3: .45 *(95%)*	10% 21%	(2%) / (0%) (6%) / 3%	32%	(0%) / 25%
					W1-W4: .24 *(91%)* W2-W4: .41 *(82%)* W3-W4: .48 *(88%)*	7% 12% 10%	(3%) / 0% (0%) / 2% (0%) / 2%	42%	(0%) / 23%
O'Connor, Neiderhiser, Reiss,	Nonshared Environment & Adolescent Development (NEAD) Project: 405 individuals including twins	Depression (composite from depression sub-scale of BPI [3 raters], CDI [3 raters], and observational report; father/mother/self & observer)	W1: 10-18 years W2: 12-21 years	Independent pathway model	W1-W2: .59 *(64%)*	32%	(0%) / 18%	0%	(8%) / 42%

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Study	Sample (Name, N)	Phenotype(s) (measure; reporter)	Age at each wave[a]	Genetic model[b]	Phenotypic stability (*% due to genes where calculable*)	Influence of stable genetic factors[c]	Influence of stable environmental factors[d] C / E	Influence of new genetic factors[e]	Influence of new environmental factors[f] C / E
Hetherington, & Plomin (1998)[g]	and full, half and unrelated sibling pairs								
Plomin et al. (1993)	MacArthur Longitudinal Twin Study (MALTS): 300 twin pairs	Behavioural inhibition (IBR; observer)	W1: 1.2 years W2: 1.7 years	Cholesky decomposition	W1-W2: .34 *(100%)*	27%	(0%) / (4%)	18%	(0%) / 55%
Nivard et al. (2015)	Netherlands Twin Registry (NTR): 23,678 twin pairs	Mixed anxiety and depression ymptoms (ASEBA & CBCL subscale / YSR; mother / self)	W1: 3 years W2: 7 years W3: 10 years W4: 12 years (mother report) W4a: 12 years (self report) W5: 14 years W6: 16 years W7: 18 years [W9-30: 20 - 62 years]	Simplex model	W1-W2: .29 W2-W3: .58 W3-W4: .63 W4-W4a W4a-W5 W5-W6: 59 W6-W7: .70	12% 16% 11% 8% 31% 12% 10%	2% / 6% 2% / 6% 3% / 6% 11% / 4% 5% 5% 4%	49% 42% 50% 26% 20% 38% 43%	0% / 32% 0% / 34% 0% / 31% 0% / 48% 44% 45% 43%
Schmitz, Fulker, & Mrazek (1995)[g]	Colorado twin sample: 203 - 260 twin pairs	Internalising (CBCL; parents)	W1: 2.8 years W2: 7.6 years	Cholesky decomposition	W1-W2: .40 *(10%)*	(7%)	(26%) / (5%)	(30%)	(0%) / (32%)
Scourfield et al. (2003)	Population-based South Wales twin registry: 670 twin pairs	Depressive symptoms (MFQ; parent)	W1: 5-14 years W1: 8-17 years	Cholesky decomposition	W1-W2: .35 *(19%)*	(2%)	40% / 2%	23%	(0%) / 33%
Silberg et al. (1999)	Virginia Twin Study of Adolescent Behavioural Development (VTSABD): 273 female twin pairs	Depression (CAPA symptom scores; child)	W1: 8-14.5 years W2: 9.5-16 years	Reduced independent pathway model (incorporating life events)	Females only: W1-W2: .33 *(92%)*	30%	0%	0%	71%
Trzaskowski, Zavos, Haworth, Plomin, & Eley (2012)	Twins Early Development Study (TEDS): 3644 - 7834 twin pairs	Negative cognition / negative affect / fear / social anxiety (ARBQ; parent)	W1: 7 years	Cholesky decomposition (correlated factors)	Negative cognition:				

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Study	Sample (Name, N)	Phenotype(s) (measure; reporter)	Age at each wave[a]	Genetic model[b]	Phenotypic stability (*% due to genes where calculable*)	Influence of stable genetic factors[c]	Influence of stable environmental factors[d] C / E	Influence of new genetic factors[e]	Influence of new environmental factors[f] C / E
			W2: 9 years		W1-W2: .50 *(69%)*	23%	1% / 4%	34%	10% / 27%
					Negative affect:				
					W1-W2: .45 *(59%)*	14%	6% / 2%	32%	17% / 29%
					Fear:				
					W1-W2: .52 *(67%)*	21%	8% / 2%	37%	11% / 20%
					Social anxiety:				
					W1-W2: .54 *(71%)*	24%	6% / 4%	32%	14% / 21%
Tully, Iacono, & McGue (2010)	Minnesota Twin Family Study (MTFS): 756 twin pairs	Major Depressive Disorder (DSM-III-R DICA-R [W1], SCI for DSM-III-R [W2 & W3]; interview)	W1: *M* = 14.8 years (*SD* = .51) W2: *M* = 18.2 years (*SD* = .65) [W3: *M* = 21.5 years (*SD* = .77)]	Cholesky decomposition	W1-W2: .24 *(33%)*	(21%)	(4%) / (1%)	(5%)	(0%) / 68%
van den Oord & Rowe (1997)[g]	National Longitudinal Survey of Youth (NLSY): 436 full sibling pairs; 119 half sibling pairs; 122 cousin pairs	Anxious/depressed behaviour / dependent behaviour / peer conflict withdrawal (BPI; mother)	W1: 4-6 years W2: 6-8 years W3: 8-10 years	Genetic liability model (independent pathway model)	Anxious/depressed behaviour				
					W1-W2: .43 *(60%)*	28%	18% / 0%	0%	0% / 54%
					W1-W3: .42 (60%) W2-W3: .45 (60%)	26%	18% / 0%	0%	0% / 56%
					Dependent behaviour:				
					W1-W2: .42 *(59%)*	26%	19% / 0%	0%	0% / 56%
					W1-W3: .42 *(58%)* W2-W3: .44 *(59%)*	26%	18% / 0%	0%	0% / 56%
				Transmisison model	Peer conflict withdrawal				
					W1-W2: .42 *(69%)*	17%	14% / 0%	8%	0% / 60%
					W1-W3: .43 *(68%)* W2-W3: .43 *(68%)*	14%	15% / 0%	8%	0% / 64%

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Study	Sample (Name, N)	Phenotype(s) (measure; reporter)	Age at each wave[a]	Genetic model[b]	Phenotypic stability (*% due to genes where calculable*)	Influence of stable genetic factors[c]	Influence of stable environmental factors[d] C / E	Influence of new genetic factors[e]	Influence of new environmental factors[f] C / E
van der Valk, van den Oord, Verhulst, & Boomsma (2003)[g]	Netherlands Twin Registry (NTR): 1924 - 3873	Internalising behaviour (CBCL; parent)	W1: 3 years W2: 7 years	Indpendent pathway model	Males: W1-W2: .35 *(70%)*	24%	8% / 4%	22%	18% / 24%
					Females: W1-W2: .41*(58%)*	23%	8% / 4%	11%	27% / 27%
van der Valk, Verhulst, Neale, & Boomsma (1998)[g]	Dutch Adoption Sample: 222 biologically related adolescents adopted together (BR); 422 unrelated adolescents adopted together (BUR); 1484 adolescents adopted singly (AS)	Internalising (CBCL; Parent)	W1: 10-15 years W2: 14-17 years	Cholesky decomposition	W1-W2: .59 *(19%)*	(8%)	29% / 7%	(0%)	(4%) / 51%
van Grootheest et al. (2007)	Netherlands Twin Register: 8083 twin pairs	Obsessive Compulsive behaviour (CBCL subscale; parents)	W1: 7 years W2: 10 years W3: 12 years	Cholesky decomposition (rater bias model)	Males: Father ratings: W1-W2: .51 *(44%)* W1-W3: .44 *(43%)* W2-W3: .54 *(19%)* Mother ratings: W1-W2: .55 *(57%)* W1-W3: .43 *(54%)* W2-W3: .58 *(43%)* Females: Father ratings: W1-W2: .53 *(34%)* W1-W3: .44 *(35%)* W2-W3: .52 *(16%)* Mother ratings:	Information unavailable Information unavailable Information unavailable			

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Study	Sample (Name, N)	Phenotype(s) (measure; reporter)	Age at each wave[a]	Genetic model[b]	Phenotypic stability (*% due to genes where calculable*)	Influence of stable genetic factors[c]	Influence of stable environmental factors[d] C / E	Influence of new genetic factors[e]	Influence of new environmental factors[f] C / E
					W1-W2: .55 *(40%)*	Information unavailable			
					W1-W3: .47 *(27%)*				
					W2-W3: .58 *(36%)*				
Waszczuk, Zavos & Eley (2013)	Emotions, Cognitions, Heredity and Outcome (ECHO): 300 twin pairs	Anxiety symptoms (SCARED; self-report)	W1: 8 years	Cholesky decomposition.	Panic				
			W2: 10 years		W1-W2: .36 *(65%)*	28%	(2%)	(0%.	(0%) / 67%
								Controlling for: W1 Anxiety Sensitivity	
					Separation Anxiety				
					W1-W2: .35 *(89%)*	35%	(1%)	(0%)	(0%) / 61%
								Controlling for: W1 Anxiety Sensitivity	
					Generalized Anxiety				
					W1-W2: .37 *(66%)*	29%	(1%)	(0%)	(0%) / 67%
								Controlling for: W1 Anxiety Sensitivity	
Waszczuk et al. (2016)	Genesis 12-19: 896-1,372 twin and sibling pairs	Depression / anxiety disorder symptoms: panic; generalised anxiety; separation anxiety; social anxiety (SMFQ / SCAS; self)	W1: 12-21 years	Cholesky decomposition	Depression:				
			W2: 14-23 years		W1-W2: .47 *(83%)*	29%	(0%) / 2%	18%	(0%) / 51%
			[W3: 18-27 years]		Panic:				
					W1-W2: .43 *(60%)*	17%	(0%) / 3%	25%	(0%) / 54%
					Generalized anxiety:				
					W1-W2: .47 *(66%)*	21%	(0%) / 5%	18%	(0%) / 56%
					Separation anxiety:				
					W1-W2: .36 *(67%)*	14%	(0%) / (1%)	27%	(0%) / 59%

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Study	Sample (Name, N)	Phenotype(s) (measure; reporter)	Age at each wave[a]	Genetic model[b]	Phenotypic stability (*% due to genes where calculable*)	Influence of stable genetic factors[c]	Influence of stable environmental factors[d] C / E	Influence of new genetic factors[e]	Influence of new environmental factors[f] C / E
					Social anxiety:				
					W1-W2: .53 *(58%)*	23%	(0%) / 8%	15%	(0%) / 54%
Zavos, Gregory, & Eley (2012)	Genesis 1219 study (G1219): > 1300 twin pairs	Anxiety sensitivity (CASI at W1&2 / ASI W3; self)	W1: *M* = 14 years (12-19)	Cholesky decomposition (AE model)	W1-W2: .47 *(51%)*	13%	9%	19%	59%
			W2: *M* = 15 years (12-21)		W1-W3: .37 *(81%)* W2-W3: .48 *(67%)*	20% 14%	1% 3%	(1%)	61%
			W3: *M* = 17 years (14-23)						
			[W4: *M* = 20 years (19-27)]						

Notes –

[a]Mean (M) age given with range and standard deviation (SD) if available; where no range is presented this is because the information was unavailable and is not necessarily indicative of participants being the same age

[b]Models estimated additive genetic (A), common environmental (C) and unique environmental (E) components of variance unless otherwise stated;

[c / d]Results for influence of stable genetic and environmental factors given as % phenotypic variance in later wave(s) explained by factors from earlier wave(s) where possible; values are aligned with phenotypic stability coefficients in adjacent column – i.e. for values aligned with W2-W3 phenotypic stability, % variance explained is at W3 by stable factors from W2; where single values align with a group of stability coefficients, % variance explained is by stable factors from all prior waves; environmental factors are presented as C / E – where C was not estimated at all in the presented model, only one value is given; non-significant values are given in parentheses where known;

[e / f]Results for influence of new genetic and environmental factors given in same format as above; only one value is presented per parameter per wave, in alignment with the appropriate group of phenotypic correlations, i.e. values centrally aligned with W1-W3 and W2-W3 stability coefficients are new aetiological factors at W3;

[g]Study includes measures of both internalising and externalising and is therefore included in both tables

[h]All results (including correlations) pertain to latent factor rather than observed variables; rater-specific (in case of rater bias models) and scale-specific (in case of common pathway – phenotypic models) error not included

Measures: CBCL: Child Behaviour Checklist; TRF: Teacher Report Form; YSR: Youth Self Report form; SDQ: Strengths and Difficulties Questionnaire; BPI: Behaviour Problem Index; (S)MFQ: (Short) Mood and Feelings Questionnaire; SCAS: Spence Children's Anxiety Scale; STAI-C: Spielberger's State-Trait Anxiety Inventory for Children; ARBQ: Anxiety-Related Behaviours Questionnaire; CDI: Child Depression Inventory; IBR: Infant Behaviour Record; ASEBA: Achenbach System of Empirically Based Measurement; CAPA: Child and Adolescent Psychiatric Assessment; SCI: Structure Clinical Interview; DSM-III-R: Diagnostic and Statistical Manual III (Revised); DICA-R: Diagnostic Interview for Children and Adolescents (Revised); (C)ASI: (Child) Anxiety Sensitivity Index

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Table 2

Included longitudinal quantitative genetic studies of behavioural problems in childhood and adolescence

Behavioural problems									
Study	**Sample (Name, N)**	**Phenotype(s) (measure; reporter)**	**Age at each wave[a]**	**Genetic model[b]**	**Phenotypic stability (*% due to genes where calculable*)**	**Influence of stable genetic factors[c]**	**Influence of stable environmental factors[d] C / E**	**Influence of new genetic factors[e]**	**Influence of new environmental factors[f] C / E**
artels et al. (2004)[g]	Netherlands Twin Registry (NTR): 1481 - 5115 twin pairs	Externalising behaviour (CBCL; parent)	W1: 3 years	Developmental model	Males:				
					W2: 7 years				
					W1-W2: .54 *(64%)*	20%	15% / 1%	39%	11% / 14%
			W3: 10 years		W1-W3: .50 *(66%)* W2-W3: .73 *(74%)*	49%	13% / 1%	16%	7% / 13%
			W4: 12 years		W1-W4: .49 *(57%)* W2-W4: .69 *(69%)* W3-W4: .76 *(73%)*	48%	16% / 4%	16%	7% / 9%
					Females:				
					W1-W2: .57 *(54%)*	19%	20% / 1%	40%	7% / 13%
					W1-W3: .48 (45%) W2-W3: .70 *(60%)*	29%	25% / 3%	16%	11% / 16%
					W1-W4: .46 *(44%)* W2-W4: .66 *(58%)* W3-W4: .76 *(51%)*	38%	24% / 4%	13%	9% / 12%
Bezdjian, Tuvblad, Wang, Raine, & Baker (2014)	University of Southern California Twin Study of Rsik Factors for Anti-social Behaviour (RFAB): 179 - 560 twin pairs	Motor impulsivity (Go No-Go Task; laboratory)	W1: *M*= 9.60 years (9-10)	Independent Pathway model	W1-W2: .53 *(61%)*	40%	19%	0%	40%
			W2: *M*= 11.79 years (11-13)		W1-W3: .41 *(55%)* W2-W3: .63 (*45%)*	19%	34%	0%	46%
			W3: *M*= 14.87 years (14-15)		W1-W4: .31 *(56%)* 2-W4: .62 *(35%)* W3-W4: .60 *(25%)*	12%	52%	12%	25%
					W4: *M*= 17.28 years (16-18)				
Chang, Lichtenstein, Asherson, & Larsson (2013)	The Swedish Twin Study of Child and Adolescent Development (TCHAD): 1480 twin pairs	Attention problems (CBCL / YSR; parent / self)	W1: 8-9 years	Cholesky decomposition (rater bias model)	Rater-agreed[h] attention problems:				
			W2: 13-14 years		W1-W2: .64 *(88%)*	41%	(2%)	41%	(15%)
			W3: 16-17 years		W1-W3: .56 *(93%)* W2-W3: .85 *(80%)*	35% 22%	(1%) (15%)	26%	(1%)

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Behavioural problems

Study	Sample (Name, N)	Phenotype(s) (measure; reporter)	Age at each wave[a]	Genetic model[b]	Phenotypic stability (*% due to genes where calculable*)	Influence of stable genetic factors[c]	Influence of stable environmental factors[d] C / E	Influence of new genetic factors[e]	Influence of new environmental factors[f] C / E
Ebejer et al. (2010)	Australian Twin Registry / Colorado Birth Registry / Birth Registries of Norway & Sweden: 989 twin pairs	Inattention / hyperactivity-impulsivity (DBRS; parent)	W1: *M*= 6.3 years (SD = 0.3)	Inattention:	*(% = A+D)*	(A / D)		(A / D)	
			W2: *M*= 7.4 years (SD = 0.3)	Cholesky decomposition (ADE model)	W1-W2: .71 *(76%)*	(1%) / 46%	8%	(5%) / (17%)	22%
			W3: *M*= 8.4 years (SD = 0.3)		W1-W3: .61 *(71%)* W2-W3: .81 *(77%)*	(0%) / 32% (6%) / (19%)	10% 4%	(0%) / (0%)	28%
			(Age information from largest [Colorado; 489 pairs] sub-sample)	Hyperactivity-impulsivity:					
				Cholesky decomposition	W1-W2: .65 *(91%)*	44%	(0%) / 2%	24%	(8%) / 20%
					W1-W3: .61 *(88%)* W2-W3: .69 *(90%)*	37% 18%	(0%) / 3% (0%) / (1%)	18%	(0%) / 21%
Eley, Lichtenstein, & Moffitt (2003)	Young Twins Study (YTS - Sweden): 1186 twin pairs	Aggressive and non-aggressive antisocial behaviour (ASB; CBCL; parent)	W1: 8-9 years W2: 13-14 years	Cholesky decomposition (correlated factors; liability threshold model)	Aggressive ASB:				
					W1-W2: .61 *(84%)*	45%	1% / 0%	1%	30% / 24%
					Non-aggressive ASB:				
					W1-W2:. 49 *(44%)*	9%	21% / 0%	35%	21% / 14%
Gagne & Hill Goldsmith (2011)	Wisconsin twin sample: 735 - 1000 *individual* twins	Anger (Infant Behaviour Questionnaire / Locomotor Lab TAB; mother/laboratory)	W1: 1 year W2: 3 years	Cholesky decomposition	Parent report:				
					W1-W2: .34 *(85%)*	12%	1%	33%	55%
					Laboratory measure:				
					W1-W2: .03 *(0%)*	0%	0% / 0%	32%	23% / 45%
Haberstick, Schmitz, Young, & Hewitt, (2005)[g]	Longitudinal Twin Study (LTS): 175-252 twin pairs	Externalising behaviour (TRF; teacher)	W1: 7 years	Developmental model	(Average acr. sex)				
			W2: 8 years		W1-W2: .49	38%	5%	22%	36%
			W3: 9 years		W1-W3: .49 W2-W3: .59	40%	12%	13%	35%
			W4: 10 years		W1-W4: .43 W2-W4: .42	38%	11%	2%	49%

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Behavioural problems

Study	Sample (Name, N)	Phenotype(s) (measure; reporter)	Age at each wave[a]	Genetic model[b]	Phenotypic stability (*% due to genes where calculable*)	Influence of stable genetic factors[c]	Influence of stable environmental factors[d] C / E	Influence of new genetic factors[e]	Influence of new environmental factors[f] C / E
					W3-W4: .60				
			W5: 11 years		W2-W4: .42 W3-W4: .60	38%	11%	2%	49%
			W6: 12 years		W1-W5: .48 W2-W5: .44 W3-W5: .53 W4-W5: .46	43%	0%	8%	49%
					W1-W6: .35 W2-W6: .37 W3-W6: .38 W4-W6: .43 W5-W6: .50	37%	3%	12%	48%
Haberstick, Schmitz, Young, & Hewitt (2006)	Longitudinal Twin Study (LTS): 366 twin pairs	Aggressive behaviour (CBCL/TRF; parent / teacher)	W1: 7 years	Developmental model (AE only)	Parent report:				
			W2: 8 years [teacher-report]		W1-W3: .69 (*86%*)	66%	4%	10%	20%
			W3: 9 years		W1-W4: .64 (*92%*) W3-W4: .78 (*85%*)	66%	6%	10%	18%
			W4: 10 years W5: 11 years		W1-W5: .64 (*98%*) W3-W5: .74 (*95%*) W4-W5: .77 (*91%*)	73%	2%	11%	14%
			W6: 12 years		W1-W6: .61 (*99%*) W3-W6: .69 (*98%*) W4-W6: .70 (*96%*) W5-W6: .77 (*91%*)	69%	3%	10%	18%
					Teacher report:				
					W1-W2: .48 (*79%*)	40%	3%	21%	36%
					W1-W3: .45 (*92%*) W2-W3: .58 (*76%*)	45%	8%	12%	35%
					W1-W4: .39 (*98%*) W2-W4: .45 (*91%*) W3-W4: .57 (*78%*)	41%	5%	1%	53%
					W1-W5: .42 (*100%*) W2-W5: .44 (*100%*) W3-W5: .49 (*99%*) W4-W5: .46 (*98%*)	48%	2%	3%	47%
					W1-W6: .33 (*100%*) W2-W6: .35 (*100%*)	30%	2%	12%	56%

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Behavioural problems

Study	Sample (Name, N)	Phenotype(s) (measure; reporter)	Age at each wave[a]	Genetic model[b]	Phenotypic stability (*% due to genes where calculable*)	Influence of stable genetic factors[c]	Influence of stable environmental factors[d] C / E	Influence of new genetic factors[e]	Influence of new environmental factors[f] C / E
					W3-W6: .37 (*100%*) W4-W6: .35 (*100%*) W5-W6: .45 (*85%*)				
Harden, Quinn, & Tucker-Drob (2012)	National Longitudinal Study of Youth: Children and Young Adults (CNLSY): 2562 sibling pairs	Delinquency (CSAS-SRD/YA-SRD; self)	W1: 10-11 years W2: 12-13 years W3: 14-15 years W4: 16-17	Latent growth curve model	Range: .32 -.39	81% (intercept)	19% / 0% (intercept)	30% (slope)	0%/ 70% (slope)
Huizink, van den Berg, van der Ende, & Verhulst (2007)[g]	Dutch sample of adopted siblings:106 biologically-related (BR) and 230 unrelated (BUR) pairs	Externalising behaviour (CBCL; adoptive parent)	W1: BR *M*= 12.5 years (SD= 1.2); BUR M= 12.4 years (SD= 1.2) W2: BR M= 15.8 years (SD= 1.2); BUR M= 15.6 years (SD= 1.2) [W3:26 years]	Common pathway model (developmental)	W1-W2: .64 (92%)	92%	1% / 0%	0%	0% / 7%
Jacobson, Prescott, & Kendler (2002)	Virginia Twin Registry (VTR): 2580 twin pairs	Antisocial behaviour (11-item questionnaire [DSM-III-R conduct disorder symptoms]; self)	W1: <15 years W2: 15-17 years [W3: >18 years]	Cholesky decomposition	Males:				
					W1-W2: .56 (*18%*)	17%	6% / 16%	25%	0% / 36%
					Females:				
					W1-W2: .51 (*39%*)	14%	7% / 7%	36%	0% / 35%
Jaffee, Hanscombe, Haworth, Davis, & Plomin (2012)	Twins Early Developmental Study (TEDS): 6286 twin pairs	Disruptive behaviour (hyperactivity and conduct sub-scales of SDQ; parent)	W1: 9 years W2: 12 years	Cholesky decomposition (genetic cross-lag with household chaos)	Conduct:				
					W1-W2: .56 *(68%)*	29%	0% / 2%	21% (controlling for household chaos)	18% / 19% (controlling for household chaos)
					Hyperactivity:				
					W1-W2: .66 *(63%)*	34%	2% / 7%	36% (controlling for household chaos)	0% / 17% (controlling for household chaos)

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Study	Sample (Name, N)	Phenotype(s) (measure; reporter)	Age at each wave[a]	Genetic model[b]	Phenotypic stability (*% due to genes where calculable*)	Influence of stable genetic factors[c]	Influence of stable environmental factors[d] C / E	Influence of new genetic factors[e]	Influence of new environmental factors[f] C / E
Kan et al. (2013)	Netherlands Twin Registry (NTR): 2375 - 16103 twin pairs	Attention problems (Dutch OAS at W1 / CBCL W2-4 / YSR W5-7; parent / parent / self)	W1: 3 years W2: 7 years W3: 10 years W4: 12 years W5: 13-15 years (self-report) W6: 15-17 years (self-report) W7: 17-19 years (self-report) [Subsequent waves 19-60 years]	Simplex (ADE) model	W1-W2: .41 W1-W3: .29 W2-W3: .71 W1-W4: .18 W2-W4: .44 W3-W4: .62 *W1-W5: .05 *W2-W5: .11 *W3-W5: .15 *W4-W5: .25 *W1-W6: .03 *W2-W6: .06 *W3-W6: .09 *W4-W6: .14 W5-W6: .58 *W1-W7: .01 *W2-W7: .03 *W3-W7: .05 *W4-W7: .07 W5-W7: .29 W6-W7: . 51	*Proportion of genetic influences present at previous wave (A / D):* W2: 100% / 25% W3: 100% / 77% W4: 34% / 92% W5: 37% / 69% W6: 100% / 53% W7: 49% / 100% *denotes cross-rater correlations	*Proportion of environmental influences present at previous wave (E):* 4% 59% 50% 52% 53% 60%	*Proportion of genetic influences **not** present at previous wave (A / D):* 0% / 75% 0% / 23% 66% / 8% 63% / 31% 0% / 47% 51% / 100%	*Proportion of environmental influences not present at previous wave (E):* 96% 41% 50% 48% 47% 40%
Kuntsi, Rijsdijk, Ronald, Asherson, & Plomin (2005)	Twins Early Developmental Study (TEDS): 4719 twin pairs	ADHD symptoms (RRPSPC at W1 & (with additional items) W2 / SDQ hyperactivity-inattention sub-scale at W3 & W4 / CPRS at W5; parent)	W1: 2 years W2: 3 years W3: 4 years W4: 7 years W5: 8 years	Cholesky decomposition (AE only, with contrast effects)	W1-W2: .47 *(94%)* W1-W3: .39 *(92%)* W2-W3: .58 *(83%)* W1-W4: .46 *(59%)* W2-W4: .35 *(91%)* W3-W4: .45 *(87%)* W1-W5: .27 *(96%)* W2-W5: .40 *(93%)* W3-W5: .46 *(89%)* W4-W5: .49 *(86%)*	25% 17% 14% 10% 5% 7% 9% 9% 7% 6%	0% 0% 4% 0% 0% 1% 0% 0% 1% 2%	54% 45% 57% 55%	21% 19% 19% 11%
Lacourse et al. (2014)	Quebec Newborn Twin Study: 667 twin pairs	Physical aggression (3-item scale; mother)	W1: *M* = 1.6 years (1.5-2) W2: *M* = 2.7 years (2.5-3.1)	Cholesky decomposition	W1-W2: .32 *(82%)* W1-W3: .25 *(71%)* W2-W3: .39 *(58%)*	12% 5% 11%	(1%) / (0%) (3%) / (0%) (2%) / (0%)	51% 34%	(3%) / 31% 17% / 28%

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Study	Sample (Name, N)	Phenotype(s) (measure; reporter)	Age at each wave[a]	Genetic model[b]	Phenotypic stability (*% due to genes where calculable*)	Influence of stable genetic factors[c]	Influence of stable environmental factors[d] C / E	Influence of new genetic factors[e]	Influence of new environmental factors[f] C / E
			W3: *M* = 4.2 years (3.9-4.6)						
				Latent growth curve model	W1-W2: .32 *(100%)*	25% (intercept)	0% / 0%	9% / 33% (slope / resid.)	0% / 49%
					W1-W3: .25 *(100%)* W2-W3: .39 *(100%)*	26% (intercept)	0% / 0%	24% / 6% (slope / resid.)	20% / 24%
Larsson, Larsson, & Lichtenstein (2004)	Young Twins Study (YTS - Sweden): 1063 - 1106 twin pairs	ADHD symptoms (binary scaled 14-item checklist based on *DSM-III-R*; parent)	W1: 8-9 years W2: 13-14 years	Cholesky decomposition	Males:				
					W1-W2: .51 *(74%)*	36%	(2%) / 1%	38%	(0%) / 24%
					Females:				
					W1-W2: .51 *(79%)*	21%	(4%) / 3%	40%	(0%) / 32%
Larsson, Lichtenstein, & Larsson (2006)	Twin Study of Child and Adolescent Development (TCHAD): 1480 twin pairs	ADHD symptoms: inattention / hyperactivity-impulsivity (binary scaled 21-24 item checklist based on DSM-III-R; parent)	W1: 8-9 years W2: 13-14 years W3: 16-17 years	Independent pathway model	Inattention:				
					Males:				
					Range (across sex):				
					.34-.60 W2:	51%	Information unavailable	18%	Information unavailable
					W3:	45%		27%	
					Females:				
					W2:	56%	Information unavailable	6%	Information unavailable
					W3:	41%		21%	
					Hyperactivity-impulsivity:				
					Males:				
					W2:	54%	Information unavailable	18%	Information unavailable
					W3:	44%		22%	
					Females:				
					W2:	41%	Information unavailable	14%	Information unavailable
					W3:	33%		31%	

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Study	Sample (Name, N)	Phenotype(s) (measure; reporter)	Age at each wave[a]	Genetic model[b]	Phenotypic stability (*% due to genes where calculable*)	Influence of stable genetic factors[c]	Influence of stable environmental factors[d] C / E	Influence of new genetic factors[e]	Influence of new environmental factors[f] C / E
Lewis & Plomin (2015)[g]	Twins Early Developmental Study (TEDS): >3000 twin pairs	Conduct problems / hyperactivity (SDQ; parent)	W1: 4 years W2: 7 years W3: 9 years W4: 12 years W5: 16 years	Cholesky decomposition	Conduct problems:				
					W1-W2: .47 *(77%)*	22%	10% / 1%	40%	3% / 23%
					W1-W3: .43 *(72%)* W2-W3: .55 *(67%)*	16% 8%	17% / 0% (1%) / 3%	28%	8% / 18%
					W1-W4: .40 *(71%)* W2-W4: .52 *(73%)* W3-W4: .56 *(70%)*	14% 11% 8%	14% / 0% (3%) / 1% (4%) / 2%	23%	(0%) / 19%
					W1-W5: .28 *(96%)* W2-W5: .37 *(85%)* W3-W5: .39 *(74%)* W4-W5: .48 *(80%)*	12% 6% 2% 8%	3% / (0%) (0%) / 0% (1%) / 1% (0%) / 1%	41%	(0%) / 25%
				Cholesky decomposition (AE model)	Hyperactivity:				
					W1-W2: .54 *(52%)*	22%	11%	27%	40%
					W1-W3: .51 *(64%)* W2-W3: .65 *(65%)*	30% 11%	5% 8%	28%	19%
					W1-W4: .43 *(73%)* W2-W4: .57 *(71%)* W3-W4: .64 *(79%)*	27% 10% 5%	2% 5% 3%	31%	17%
					W1-W5: .31 *(89%)* W2-W5: .41 *(84%)* W3-W5: .45 *(89%)* W4-W5: .55 *(88%)*	21% 6% 2% 6%	0% 2% 1% 1%	38%	22%
Niv, Tuvblad, Raine, & Baker (2013)	University of Southern California Risk Factors for Antisocial Behavior Twin Study: 1204 individuals (750 participating families in the extended study)	Anti-social behaviour (common factor from: aggression & rule-breaking CBCL sub-scales; parent)	W1: 9-10 years W2: 14-15 years	Common pathway model (phenotypic)	Anti-social behaviour (latent factors)[h]:				
					W1-W2: .77 *(41%)*	23%	24% / 11%	42%	(0%) / (0%)
Niv, Tuvblad, Raine, Wang, & Baker (2012)	University of Southern California Risk Factors for Antisocial Behavior Twin Study: 1204 individuals (750 participating families in the extended study)	Impulsiveness (common factor from inattention, motor & non-planning BIS sub-scales; self)	W1: *M* = 11.89 years (*SD* = .69) W2: *M* = 14.69 (*SD* = .63)	Common pathway model (phenotypic; AE model)	Impulsiveness (latent factors)[h]:				
					W1-W2: .43 *(76%)*	18%	3%	32%	48%
O'Connor, Neiderhiser, Reiss,	Nonshared Environment and Adolescent Development (NEAD) Project: 405	Antisocial behaviour (composite from BPI [3 raters] and observational report; father / mother / self & observer)	W1: 10-18 years W2: 12-21 years	Independent pathway model	W1-W2: .63 *(52%)*	32%	18% / 11%	18%	(0%) / 20%

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Hetherington, & Plomin (1998)[g] - *see also Neiderhiser, Reiss, & Hetherington (1996)*	individuals including twins and full, half and unrelated sibling pairs								
Polderman et al. (2011)	Netherlands Twin Registry (NTR):445 twin pairs	Attention problems (CBCL; mother)	W1: 5 years W2: 7 years	Cholesky decomposition (correlated factors)	W1-W2: .39 *(91%)*	22%	0% / 1%	40%	3% / 34%
Price et al. (2005)	Twins Early Developmental Study (TEDS): 6840 twin pairs	ADHD symptoms (RRPSPC at W1 & [with additional items] W2 / SDQ hyperactivity-inattention sub-scales at W3; parent)	W1: 2 years W2: 3 years W3: 4 years	Common pathway model (developmental; AE model with contrast effects)	W1-W2: .55 *(94%)* W1-W3: .46 (96%) W2-W3: .60 (98%)	68% 50%	6% 5%	9% 27%	17% 19%
Rietveld, Hudziak, Bartels, van Beijsterveldt, & Boomsma (2004)	Netherlands Twin Registry (NTR): > 1096 twin pairs	Attention problems (Dutch OAS at W1 / CBCL at other waves; parent)	W1: 3 years W2: 7 years W3: 10 years W4: 12 years	Cholesky decomposition (correlated factors; ADE model)	Males: *(% = A+D)* W1-W2: .41 *(90%)* W1-W3: .38 *(92%)* W2-W3: .69 *(82%)* W1-W4: .35 (87%) W2-W4: .67 *(79%)* W3-W4: .75 *(76%)* Females: W1-W2: .41 *(90%)* W1-W3: .37 *(92%)* W2-W3: .68 *(81%)* W1-W4: .38 (89%) W2-W4: .65 (81%) W3-W4: .72 (79%)	(A / D) 11% / 7% 19% / 0% 13% / 27% 10% / 1% 5% / 28% 7% / 1% 21% / 1% 21% / 1% 18% / 25% 18% / 1% 12% / 17% 9% / 0%	1% 0% 5% 1% 6% 8% 1% 0% 6% 1% 6%	(A / D) 22% / 32% 9% / 3% 8% / 0% 36% / 15% 9% / 0% 5% 15% / 0%	27% 22% 15% 26% 21% 17%
Schmitz, Fulker, & Mrazek (1995)[g]	Colorado twin sample: 203 - 260 twin pairs	Externalising (CBCL; parents)	W1: 2.8 years W2: 7.6 years	Cholesky decomposition	W1-W2: .40 *(82%)*	(45%)	(3%) / (2%)	(12%)	(19%) / (19%)

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Study	Sample (Name, N)	Phenotype(s) (measure; reporter)	Age at each wave[a]	Genetic model[b]	Phenotypic stability (*% due to genes where calculable*)	Influence of stable genetic factors[c]	Influence of stable environmental factors[d] C / E	Influence of new genetic factors[e]	Influence of new environmental factors[f] C / E
Taylor et al. (2013)	Twins Early Developmental Study (TEDS): 5356 - 5983 twin pairs	ADHD traits (CPRS; parent)	W1: 8 years W2: 12 years	Cholesky decomposition (correlated factors)	Males:				
					W1-W2: .67 *(81%)*	42%	3% / 2%	32%	9% / 12%
					Females:				
					W1-W2: .65 *(78%)*	38%	4% / 3%	29%	13% / 13%
Tuvblad, Eley, & Lichtenstein (2005)	Twin Study of Child and Adolescent Development (TCHAD): 1226 twin pairs	Delinquency (sub-scale of CBCL / 34-item delinquency questionnaire; parent / self)	W1: *M* = 8.7 years (*SD* = 0.47) W2: *M* = 13.7 years (*SD* = 0.47)	Cholesky decomposition	Males:				
					W1-W2: .16 *(40%)*	1%	4% / (0%)	28%	38% / 30%
					Females:				
					W1-W2: .19 *(100%)*	10%	(0%) / (0%)	(21%)	36% / 32%
Tuvblad, Narusyte, Grann, Sarnecki, & Lichtenstein (2011)	Twin Study of Child and Adolescent Development (TCHAD): 2600 individual twins	Antisocial behaviour (externalising sub-scale of CBCL [W1] / 31-34 item delinquency questionnaire; parent / self)	W1: 8-9 years W2: 13-14 years W3: 16-17 years [W4: 19-20 years]	Common pathway model (developmental)	Males:				
					W1-W2: .11 *(53%)*	30%	12% / 3%	(3%)	22% / 30%
					W1-W3: .09 *(77%)* W2-W3: .55 *(65%)*	42%	16% / 4%	(0%)	(4%) / 34%
					Females:				
					W1-W2: .14 *(42%)*	32%	12% / 3%	17%	12% / 24%
					W1-W3: .12 *(69%)* W2-W3: .52 *(68%)*	46%	17% / 5%	(5%)	(4%) / 50%
Tuvblad, Raine, Zheng, & Baker (2009)	University of Southern California Twin Study of Risk Factors for Anti-social Behaviour (RFAB): 615 twin pairs	Reactive and proactive aggression (RPAQ; parent)	W1: *M* = 9.6 years (*SD* = 0.58) W2: *M* = 11.8 years (*SD* = 0.90)	Cholesky decomposition	Reactive agression:				
					W1-W2: .54 *(48%)*	23%	(1%) / 10%	(24%)	(9%) / 31%
					Proactive aggression:				
					W1-W2: .50 *(85%)*	53%	(0%) / 1%	(0%)	(3%) / 41%

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van Beijsterveldt, Bartels, Hudziak, & Boomsma (2003)	Netherlands Twin Registry (NTR): 1509 - 6488 twin pairs	Aggressive behaviour (CBCL; parent)	W1: 3 years	Simplex model	Males:				
			W2: 7 years		W1-W2: .48 *(65%)*	18%	10% / 0%	48%	8% / 16%
			W3: 10 years		W1-W3: .43 *(64%)* W2-W3: .73 *(78%)*	49%	9% / 3%	18%	7% / 14%
			W4: 12 years		W1-W4: .42 *(58%)* W2-W4: .67 *(76%)* W3-W4: .77 *(78%)*	53%	12% / 4%	14%	8% / 10%
					Females:				
					W1-W2: .48 *(60%)*	14%	14% / 0%	50%	6% / 16%
					W1-W3: .43 *(50%)* W2-W3: .73 *(67%)*	34%	19% / 3%	10%	7% / 18%
					W1-W4: .42 *(52%)* W2-W4: .67 *(68%)* W3-W4: .77 *(66%)*	46%	17% / 3%	12%	8% / 14%
van den Oord & Rowe (1997)[g]	National Longitudinal Survey of Youth (NLSY): 436 full sibling pairs; 119 half sibling pairs; 122 cousin pairs	Antisocial behaviour / headstrong behaviour / hyperactivity (BPI; mother	W1: 4-6 years	Genetic liability model (independent pathway model)	Antisocial behaviour:				
			W2: 6-8 years		W1-W2: .46 (*76%*)	28%	13% / 0%	0%	49%
			W3: 8-10 years		W1-W3: .44 (*73%*) W2-W3: .50 (*70%*)	32%	17%	0%	51%
					Headstrong behaviour:				
					W1-W2: .48 *(67%)*	32%	16% / 0%	0%	52%
					W1-W3: .48 (*67%*) W2-W3: .48 (*67%*)	32%	16% / 0%	0%	52%
					Hyperactivity				
					W1-W2: .48 (*81%*)	39%	9% / 0%	0%	52%
					W1-W3: .48 (*81%*) W2-W3: .48 (*81%*)	39%	9% / 0%	0%	52%
van der Valk, van den Oord,	Netherlands Twin Registry (NTR): 1924 - 3873	Externalising behaviour (CBCL; parent)	W1: 3 years	Indpendent pathway model	Males:				

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Verhulst, & Boomsma (2003)[g]			W2: 7 years		W1-W2: .55 (*53%*)	33%	23% / 5%	20%	7% / 12%
					Females:				
					W1-W2: .53 (*55%*)	36%	25% / 5%	14%	9% / 11%
van der Valk, Verhulst, Neale, & Boomsma (1998)[g]	Dutch Adoption Sample: 222 biologically related adolescents adopted together (BR); 422 unrelated adolescents adopted together (BUR); 1484 adolescents adopted singly (AS)	Externalising behaviour (CBCL; parent)	W1: 10-15 years W2: 14-17 years	Cholesky decomposition	W1-W2: .66 (*57%*)	26%	11% / 8%	22%	6% / 27%
Van Hulle et al. (2009) - *see also Lahey et al. (2009)*	Offspring of National Longitudinal Survey of Youth (NLSY79) sample	Antisocial behaviour: conduct problems / delinquency (BPI / SRD; mother / self)	W1: 4-9 years (mother-reported conduct problems only)	Cholesky decomposition	Conduct problems:				
					Males:				
			W2: 10-11 years		W1-W2: .59 *(85%)*	43%	5% / (ns)	2%	8% / 39%
			W3: 12-13 years		W1-W3: .52 *(90%)* W2-W3: .56 *(79%)*	38% 4%	14% / 6% 4% / (ns)	(ns)	6% / 29%
			W4: 14-17 years self-reported delinquency only)		Females:				
					W1-W2: .47 *(87%)*	25%	15% / (ns)	1%	16% / 41%
					W1-W3: .46 *(92%)* W2-W3: .44 *(66%)*	27% 8%	(ns) / 10% (ns) / (ns)	(ns)	13% / 35%
					Delinquency (controlling for conduct problems at W1):				
					W2-W3: .33 *(45%)*	29%	4% / (ns)	(ns)	61%
					W2-W4: .25 *(54%)* W3-W4: .38 *(68%)*	23%	(ns) / (ns)	(ns)	58%
						(ns) = non-significant estimate, value not reported			
Vierikko, Pulkkinen, Kaprio, & Rose (2006)	Finnish twin-family study (*FinnTwin12*): 1,041 twin pairs	Aggression (sub-scale of MPNI; teacher)	W1: 12 years W2: *M* = 14.3 years (14 - 15.6)	Cholesky decomposition (including sex-specific C)	Males:				
					W1-W2: .35 *(38%)*	7%	6% (sex-specific C)/ 0%	35%	0% / 52%
					Females:				

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					W1-W2: .26 *(0%)*	0%	31% / 1%	40%	0% / 28%
Wichers et al. (2013)	Twin Study of Child and Adolescent Development (TCHAD): 1480 twin pairs	Externalising behaviour (CBCL / YSR; parent / self)	W1: 8-9 years W2: 13-14 years W3: 16-17 years [W4: 19-20 years]	Rater bias model with Cholesky decomposition	Rater-agreed[h] externalising behaviour:				
					W1-W2: .73 (94%)	61%	(0%) / (1%)	26%	(4%) / 7%
					W1-W3: .71 (94%) W2-W3: .81 (86%)	58% (4%)	(0%) / (0%) (4%) / 6%	26%	(0%) / (0%)
Wang et al. (2012)	Western Reserve Reading Project: 204 twin pairs	Externalising behaviour + attention problems (CBCL; parent)	W1: *M* = 6.07 years (4.33-7.92) W2: *M* = 7.15 years (6.00-8.83) W3: *M* = 8.30 years (6.50-10.00)	Independent pathway model (combined externalising and attention problems)	Externalising:				
					W1-W2: .71(*82%*)	44%	(0%) / 12%	32%	(0%) / 12%
					W1-W3: .68 *(83%)* W2-W3: .76 *(86%)*	41% 27%	1% / 9% (0%) / (0%)	(16%)	(0%) / 7%
					Attention problems:				
					W1-W2: .47 *(7%)*	4%	44% / (0%)	(0%)	(0%) / 53%
					W1-W3: .35 *(0%)* W2-W3:.44 *(0%)*	(0%) (0%)	29% / 12% (0%) / 7%	(20%)	(0%) / (32%)

Notes –

[a]Mean (M) age given with range and standard deviation (SD) if available; where no range is presented this is because the information was unavailable and is not necessarily indicative of participants being the same age

[b]Models estimated additive genetic (A), common environmental (C) and unique environmental (E) components of variance unless otherwise stated;

[c / d]Results for influence of stable genetic and environmental factors given as % phenotypic variance in later wave(s) explained by factors from earlier wave(s) where possible; values are aligned with phenotypic stability coefficients in adjacent column – i.e. for values aligned with W2-W3 phenotypic stability, % variance explained is at W3 by stable factors from W2; where single values align with a group of stability coefficients, % variance explained is by stable factors from all prior waves; environmental factors are presented as C / E – where C was not estimated at all in the presented model, only one value is given; non-significant values are given in parentheses where known;

[e / f]Results for influence of new genetic and environmental factors given in same format as above; only one value is presented per parameter per wave, in alignment with the appropriate group of phenotypic correlations, i.e. values centrally aligned with W1-W3 and W2-W3 stability coefficients are new aetiological factors at W3;

[g]Study includes measures of both internalising and externalising and is therefore included in both tables

[h]All results (including correlations) pertain to latent factor rather than observed variables; rater-specific (in case of rater bias models) and scale-specific (in case of common pathway – phenotypic models) error not included

Measures: CBCL: Child Behaviour Checklist; TRF: Teacher Report Form; YSR: Youth Self Report form; SDQ: Strengths and Difficulties Questionnaire; OAS: Overactive Syndrome questionnaire; RRPSPC: Revised Rutter Parent Scale for Pre-school Children; CPRS-R: Conners Parent Rating Scale (Revised); BPI: Behaviour Problem Index; RPAQ: Reactive and Proactive Aggression Questionnaire; MPNI: Multidimensional Peer Nomination Inventory; DBRS: Disruptive Behaviour Rating Scale; CSAS: Child Self-Administered Supplement; (YA-)SRD: (Young Adult) Self-Report of Delinquency: BIS: Barratt Impulsiveness Scale