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Genetic Contributions to Continuity, Change, and Co-occurrence of Antisocial and Depressive Symptoms in Adolescence

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In adolescence, antisocial and depressive symptoms are moderately stable and modestly correlated with each other. We examined the genetic and environmental origins of the stability and change of antisocial and depressive symptoms and their co-occurrence cross-sectionally and longitudinally in a national sample of 405 adolescents. Monozygotic (MZ) and dizygotic (DZ) twins and full, half, and unrelated siblings 10–18 years of age from nondivorced and stepfamilies were studied over a 3-year period. Composite measures of adolescent self-reports, parent reports, and observational measures of antisocial and depressive symptoms were analysed in multivariate behavioural genetic models. Results indicated that the majority of the stability in and co-occurrence between dimensions could be accounted for by genetic factors. Nonshared environmental risks and, for antisocial symptoms, shared environmental risks also contributed to the stability. Genetic influences on change were observed, but only for antisocial behaviour. In addition, the longitudinal association between antisocial behavioural and later depressive symptoms was also found to be genetically mediated, but this effect was nonsignificant after controlling for stability. Results are discussed in light of the potential contributions of developmental behavioural genetic research in understanding individual differences in the stability and change of maladjustment.

Keywords: Behaviour problems, emotional disorder, adolescence, twins, comorbidity, genetics.

Abbreviations: BPI: Behaviour Problem Index; CDI: Child Depression Inventory; DZ: dizygotic; En: nonshared environment; Es: shared environment; Ga: additive genetic factors; GFI: Goodness-of-Fit Index; MZ: monozygotic; NEAD: Nonshared Environment and Adolescent Development project; RMSEA: root mean square error of approximation.

Introduction

Research findings on antisocial behaviour and depressive symptoms in adolescents from diverse samples and measures concur on two major points. First, almost without exception, extant research underscores the stability of both clusters of symptoms throughout adolescence and, to a less extent, into adulthood (Farrington, 1991; Feehan, McGee, & Williams, 1993; Harrington, Fudge, Rutter, Pickles, & Hill, 1990; Kandel & Davies, 1986; Lewinsohn, Clarke, Seeley, & Rohde, 1994; Robins & Ratcliff, 1978; Stanger, Achenbach, & McConaughy, 1993). A high level of stability is reported for both dimensional and categorical (diagnostic) definitions of psychopathology. The stability of individual differences and the continuity of the severity of impairment into adulthood is striking in light of the normative biological, social, and psychological changes throughout

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adolescence and early adulthood that are believed to militate *against* continuity. Second, the rate of co-occurring antisocial and depressive symptoms, measured as a correlation or as comorbid diagnoses, far exceeds chance and, because overlap is also observed in community samples, is unlikely to be related to referral biases or other methodological artefacts (Angold & Costello, 1993; Caron & Rutter, 1991; Verhulst & van der Ende, 1993). Again, the high rates of covariation are found for both dimensional and categorical definitions of psychopathology. High rates of co-occurring depressive and antisocial symptoms pose nosological and treatment dilemmas (Cantwell & Rutter, 1994), and complicate existing research findings on individual differences in the aetiology of depressive symptoms *or* antisocial behaviour examined in isolation (Capaldi, 1992; Dadds, Sanders, Morrison, & Rebgetz, 1992; Downey & Coyne, 1990; Harrington, Fudge, Rutter, Pickles, & Hill, 1991; Kovacs, Paulauskas, Gatsonis, & Richards, 1988).

Stability and Specificity of Psychosocial Risks

Research aimed at explaining the stability of symptoms and the co-occurrence of symptom clusters underscores the role of stable, overlapping, and developmentally linked psychosocial risk factors. For instance, available data indicate that individual differences in both antisocial and depressive symptoms have been associated with family discord and harsh parenting (Dadds et al., 1992; Downey & Coyne, 1990; Riess et al., 1995), peer rejection and poor social support (Lewinsohn et al., 1994; Patterson & Bank, 1989; Quiggle, Garber, Panak, & Dodge, 1992), school failure (Patterson, Reid, & Dishion, 1992; Patterson & Stoolmiller, 1991), stressful life events (Compas, Howell, Phares, Williams, & Giunta 1989), and socioeconomic disadvantage (Caspi & Moffitt, 1995; Dubois, Felner, Meaves, & Krier, 1994). These same risk factors are also thought to underlie the continuity of adjustment problems, as available data suggest that individual differences in psychosocial risk factors are quite stable among normal-risk, and especially among high-risk, populations (Dishion, French, & Patterson, 1995).

Explanations for the high rates of symptom co-occurrence from a psychosocial risk perspective generally focus on the tendency for psychosocial risks to covary and the lack of connections between specific risk factors and specific symptom clusters (Wrate, Rothery, McCabe, Aspin, & Bryce, 1995). Indeed, given the available findings on psychosocial risk and maladjustment, high rates of co-occurring adjustment problems is the expectation.

Some provocative findings from longitudinal research suggest an even more complicated picture of the relation between psychosocial risks and maladjustment. One finding requiring additional empirical attention is the tendency for antisocial symptoms to be associated with an increase in *subsequent* depressive symptoms (Capaldi, 1991; Kovacs et al., 1988; Rhode, Lewinsohn, & Seeley, 1991). The attendant failure experiences of antisocial behaviour, namely peer rejection and school and relationship failures, are believed to represent a risk factor for later depression. In other words, the consequences of one dimension of maladjustment may act as risk factors for the later emergence of another dimension of maladjustment. Alternatively, antisocial and depression symptoms may not be aetiologically distinct, but rather may index a common underlying developmental path (e.g. Caron & Rutter, 1991).

Behavioural Genetic Models of Risk

Interest in incorporating genetic and psychosocial risk models in the development of psychopathology has burgeoned in the past decade and has been stimulated by findings that mentally ill parents confer both psychosocial and genetic risks on their offspring (Rutter, 1990; Weissman, Warner, Wickramaratne, & Prusoff, 1988) and that individuals appear to elicit experiences or select environments that are related to their genetic predispositions (Scarr & McCartney, 1983). Available data from twin, adoption, and family studies document that a range of psychopathological symptoms and syndromes in young people can be attributed, in part, to genetic causes (Rutter et al., 1990; Todd, Neuman, Geller, Fox, & Hickok, 1993).

Antisocial behaviour and depressive symptoms have received considerable attention in this regard. To date, available studies indicate a significant but moderate genetic influence on depression or depressive symptoms in children and adolescents (Harrington et al., 1993; Puig-Antich et al., 1989; Silberg et al., 1994; Weissman et al., 1988; Wierzbicki, 1987). Research findings on the genetic influences on antisocial behaviour in adolescence are less clear—and certainly not as robust as studies of adults (DiLalla & Gottesman, 1989; Mednick, Moffitt, Gabrielli, & Hutchings, 1986). Especially in adolescence, the effects of shared environment in some studies appears to be more important than genetic influences (Simonoff, McGuffin, & Gottesman, 1994). Nonetheless, the weight of evidence favours at least a minor role ascribed to genetic factors, albeit for only certain subtypes of antisocial behaviour, notably aggressiveness (Edelbrock, Rende, Plomin, & Thompson, 1995; Plomin, Nitz, & Rowe, 1990; Rowe, 1983; Silberg et al., 1994).

Behavioural genetic methods can be used to examine not only the genetic and environmental influences on individual dimensions of psychopathology cross-sectionally, but also the genetic and environmental influences on stability and change (Plomin, 1986). Behavioural genetic methods may be employed to examine whether genetic influences mediate the stability of depressive and antisocial symptoms. To date, little is known about the genetic influences on stability of antisocial and depressive symptoms in adolescence, and this absence is an important missing link in developmental models of psychopathology. Extant findings on personality in adolescence and adulthood offer some clues, however. Available research suggests that behavioural continuity is largely genetically mediated, with nonshared (individual-specific) environmental experiences contributing the remainder. The reverse is found for change, which appears to be influenced largely by nonshared environmental influences, with genetic influences playing a minor role (Dworkin, Burke, Maher, & Gottesman, 1977; Loehlin, 1992; McGue, Bacon, & Lykken, 1993).

Behavioural genetic models may also be used to examine whether genetic risks are specific to a dimension of maladjustment or instead influence a profile of psychopathology. For example, the high correlation between antisocial and depressive symptoms may reflect nonspecific psychosocial risks and nonspecific genetic risks. A previous paper from this project supported the hypothesis of a common genetic risk underlying both depressive and antisocial symptoms (O'Connor, McGuire, Reiss, Hetherington, & Plomin, in press), but one family history study (Puig-Antich et al., 1989) and an adoption study (Cadoret, O'Gorman, Heywood, & Troughton, 1985) do not support this hypothesis. Thus, one aim in the current study is to replicate the previous finding of genetic mediation of the overlap of antisocial and depressive symptoms.

In addition to examining stability, change, and co-occurrence of dimensions, behavioural genetic models may examine longitudinal mediation of risks and psychopathology, as in the case of antisocial behaviour acting as a risk factor for later depressive symptoms (Capaldi, 1992). Genetic mediation of the longitudinal association between antisocial behaviour and depression would be expected if, for example, antisocial and depressive symptoms index a common underlying genetic pathway.

In a previous paper, we reported on the genetic and environmental influences on antisocial and depressive symptoms and the genetic and environmental sources of covariance between these dimensions (O'Connor et al., in press). The results indicated that approximately half of the variance in depressive and antisocial symptoms could be accounted for by genetic factors. Nonshared environment also accounted for a quarter and a half of the variance for antisocial behaviour and depression, respectively. Shared environmental factors made a significant contribution only to antisocial behaviour. In addition, covariation between antisocial and depressive symptoms was found to be both genetically and environmentally influenced. The previous analyses also indicated no significant differences by gender in the gene-environment pattern of univariate and bivariate analyses. In this report we present the genetic and environmental influences on depressive and antisocial symptoms at wave 2, on the stability of each dimension across waves, on the covariation between symptom clusters at wave 2, and on the prediction of depressive symptoms from earlier antisocial behaviour.

Three central hypotheses guide the current analyses. First, based on our findings at wave 1, we predicted significant genetic influences in individual differences in depressive and antisocial symptoms at wave 2, in the stability of depressive and antisocial symptoms across the 3 years, in the cross-sectional co-occurrence of depressive and antisocial symptoms at wave 2, and in the link between early antisocial symptoms and later depressive symptoms. The emerging research literature on longitudinal bivariate genetic analyses of personality led us to hypothesise that longitudinal change would largely be mediated by nonshared environmental influences. Finally, based on our wave 1 finding of shared environmental risks for antisocial behaviour, we hypothesised that shared environmental influence would be more salient for antisocial behaviour than for depressive symptoms.

Methods

Sample

The data in this study are part of the Nonshared Environment and Adolescent Development (NEAD) project (Reiss et al., 1994). Families in this study were drawn from several sources. Nondivorced families with nontwin siblings and a subset of the other families were recruited through random digit dialing. However, because random digit dialing procedures would be prohibitively expensive to identify the other relatively rare sibling and family types sought in the study, the remainder of the families were identified from two large national market surveys of over 675,000 families that spanned a wide range of geographic and socioeconomic characteristics (Reiss et al., 1994).

The wave 1 sample included two target same-sex adolescent siblings no more than 4 years apart in age from a total of 720 nondivorced and remarried families from 47 states. To avoid the increased stress associated with the marital transition, all remarried families were together for a minimum of 5 years. The average ages of the target children were 14.8 years and 12.6 years for the older and younger adolescents, respectively; the range in age was 10–18 years. Nondivorced families consisted of 93 MZ twin pairs, 99 DZ twin pairs, and 95 full-sibling pairs. Remarried families included 182 full-sibling pairs, 109 half-sibling pairs, and 130 unrelated-sibling pairs. Thus, the six groups of siblings differ in genetic relatedness from 100% (MZ twins) to 50% (DZ and full siblings) to 25% (half siblings) and 0% (unrelated siblings). The sample is generally middle-class but contains a wide range of educational and income levels. Fathers averaged 14 years of education and mothers averaged 13.6 years. The average family income was $25,000–35,000. Ninety-three per cent of the fathers and 94% of the mothers were Caucasian. Although the percentage of Caucasians in the sample is above national norms, this is not surprising given the large number of long-remarried stepfamilies that the study design required. Average ages of mothers was 38.1 years, and that of fathers was 41 years.

The nature of the larger study was to examine familial nonshared environmental influences on psychosocial development. Consequently, only families in which both adolescents were at least half-time residents in the home at wave 2 were considered eligible for the longitudinal study. Because data collection for wave 2 was approximately 3 years after wave 1, many families were ineligible for the follow-up study. The full longitudinal sample consists of 405 families, including 63 MZ twins, 75 DZ twins, and 57 full siblings in nondivorced families; and 95 full siblings, 61 half siblings, and 44 unrelated siblings in stepfamilies. The zygosity of 10 of the 405 pairs of siblings could not be determined (see following). Of the 720 families at wave 1, 280 were considered "out of scope" for the follow-up study because one of the siblings no longer resided in the home at least half-time. Of the 440 families that were eligible, 405 or 92% agreed to participate. At wave 2 a subset of families that were eligible but refused to participate ($N = 8$) and the "out of scope" families ($N = 280$) were also asked to participate in a smaller study with a very reduced battery; information for this study was collected over the phone rather than in the family's home (families in the phone survey were not included in these analyses). Combining the larger study and the smaller phone study, at least some information was collected on over 95% of families. This retention rate is remarkably high given that the sample was identified in 47 states and was collected with the help of several research teams.

Attrition analyses to examine differences between the 35 families that refused to participate in the full study indicated that older age and less parental monitoring characterised the refusers from the 405 families who participated. There was also a trend for the older child in the refuser families to exhibit more antisocial behaviour and for mothers in the refuser families to display less parental warmth.

Procedure

For wave 1, families were visited in their homes by trained interviewers on two occasions about 2 weeks apart; each visit lasted approximately 2 hours. Mothers, fathers, and the two adolescent siblings completed an extensive battery of questionnaires during as well as between the home visits. In addition, during the visits parent-child problem-solving interactions were videotaped. Similar procedures were employed at wave 2 except that families were visited just once and a shortened battery was used.

Measures

Zygosity. Twin zygosity was determined using parent and interviewer reports on a questionnaire (Nichols & Bilbro, 1966). When compared to blood tests of single gene markers, this questionnaire correctly identifies greater than 90% of twins. Consistent with other studies, 6% of the twins could not be diagnosed definitively in this study; these twin pairs are excluded from all analyses.

Adolescent adjustment. Several authors make explicit the important differentiation between symptoms, syndromes, and disorders in studying psychopathology. As detailed elsewhere, this study employed a dimensional model of psychopathology based on symptom checklists rather than a categorical "affected-unaffected" dichotomy (Reiss et al., 1995). A dimensional model was adopted for several reasons. First, symptom checklists have a long history in clinical, developmental, and epidemiological research (Kandel & Davies, 1982). Second, available findings indicate that symptom checklists with children and adolescents tend to replicate research using diagnostic classifications with respect to concurrent and longitudinal validity (Kandel & Davies, 1986; Stiffman, Chueh, & Earls, 1992), and rates of co-occurring symptoms (Verhulst & van der Ende, 1993). Symptom checklists also discriminate clinic from nonclinic populations (Achenbach & Edelbrock, 1981). Third, available data suggest that the genetic-environment pattern of influence is similar for clinical diagnoses and dimensional modes of psychopathology (Livesley, Jang, Jackson, & Vernon, 1993; Nigg & Goldsmith, 1994). Finally, it would have been prohibitively expensive to train interviewers and collect diagnostic data on both adolescents and obtain similar reports from parents. In order to increase the reliability of the depression and antisocial constructs, a multimethod multirater design was used. The present strategy is analogous to research using diagnostic classifications in which adolescent self-report and parental reports are combined.

The *Child Depression Inventory* (CDI; Kovacs, 1981) is a widely-used 27-item inventory of depressive symptoms (e.g. depressed affect, poor appetite, poor sleep). The CDI has been previously reported to demonstrate good reliability, validity, and correspondence with clinical diagnoses (Kovacs, 1981). Adolescents completed the CDI; mothers and fathers completed the parent version of the CDI.

The *Behaviour Problem Index* (BPI; Zill, 1985) is a 32-item questionnaire that assesses the frequency of specific behaviours that discriminate clinic from nonclinic individuals; items on the BPI were adapted from the widely-used Child Behaviour Checklist (CBCL; Achenbach & Edelbrock, 1981). The BPI has been used in a national sample of over 15,000 children aged 4–17 years and demonstrates satisfactory reliability and validity (Zill, 1985). The two six-item Depression/Internalising (e.g. sudden changes in mood; felt sad or depressed) and Antisocial (e.g. got into trouble at school, bully/mean to others) factors were included in these analyses. Mother, father, and adolescent self-reports were included.

In addition to the questionnaire assessments, *Observational assessment* of depressed mood and antisocial behaviour was made by trained raters using a previously validated coding system (Hetherington & Clingempeel, 1992). Following the completion of a 10-minute dyadic videotaped interaction, coders rated each dimension on a five-point Likert scale. A total observational score of depressed mood and antisocial behaviour was averaged across the adolescent's behaviour in three dyadic interactions (i.e. with mother, father, and sibling).

A composite score for depressive and antisocial symptoms was created be aggregating measures across respondents. Mean differences and moderate correlations between parent and child reports of psychopathology are sometimes interpreted as evidence for analysing data from reporters separately. We proposed that by aggregating across sources of information a more reliable measure of adjustment would be obtained and the genetic and environmental estimates would be more reliable, i.e. not driven by methodological factors. There are, of course, several different strategies for analysing children's self-report and parent reports of children's symptoms; there is no best solution. When parent and child reports were analysed separately, the siblings' similarity (i.e. correlation) based on parent reports was higher than that based on children's self-reports in each sibling group. This is to be expected given that parent-reported sibling similarity is based on the *same* parent's rating whereas adolescent-reported sibling similarity is based on *different* siblings' ratings. However, for both parent and adolescent reports the *pattern* of correlation across sibling groups was essentially the same.

The depression composite included mother, father, and self-reports of the total score from the CDI, the depression factor from the BPI, and observers' reports of depressed mood based on the observational data. The internal consistency (Cronbach's alpha) for this composite was .73 for the older adolescent and .70 for the younger adolescent at wave 1 and .74 and .67 for the older and younger adolescent, respectively, at wave 2. To ensure comparability in for the longitudinal analyses, the composites include only the measures present at both waves. Time constraints at wave 2 required that a measure used at wave 1 (the Behaviour Events inventory) be dropped from the wave 2 battery. The antisocial composite consisted of mother, father, and self-report of the antisocial factor from the BPI and observers' reports of the adolescent's observed antisocial behaviour. The internal consistency of this composite is .65 for the older adolescent and .62 for the younger adolescent at wave 1 and .70 and .65 for the older and younger adolescent, respectively, at wave 2.

Data Analysis

A twin-sibling design was used in this study to decompose observed (phenotypic) variance into genetic and environmental components (Plomin, DeFries, & McClearn, 1990). Genetic influence is implied if the following pattern exists among the correlations and, in the case of bivariate analyses, among the cross-correlations: MZ twins > DZ twins and full siblings > half siblings > unrelated siblings. Shared environment is suggested if the correlations among all sibling pairs are similar and substantial. Nonshared environmental influence is suggested if the correlations among sibling pairs are small, and is most clearly observed as the dissimilarity of identical twins.

Rather than examining individual pairs of correlations, model-fitting techniques consider the entire pattern of correlations simultaneously. The modelling procedures for univariate and bivariate models are explained in detail elsewhere (Neale & Cardon, 1992), and will be only briefly discussed here. In univariate and bivariate behavioural genetic analyses sibling similarity is decomposed into similarity due to additive genetic factors (Ga) and similarity due to shared environmental experiences (Es); sibling dissimilarity is attributed to nonshared environment (En), or those experiences that make siblings dissimilar, and measurement error. Based on quantitative genetic theory, MZ twins are genetically identical and are therefore correlated 1.0 for additive genetic variance. Full siblings and DZ twins are correlated .5, half siblings are correlated .25, and genetically unrelated siblings are correlated 0. By definition, shared environment is correlated 1.0 for siblings who live in the same house and the nonshared environment correlation for all siblings pairs is 0 because it represents the influences that are unique to each sibling (see Plomin et al., 1990).

In the bivariate model the sibling cross-covariances, or sibling A's score on measure 1 with sibling B's score on measure 2, are analysed. The bivariate model includes genetic (Ga) as well as the shared (Es) and nonshared (En) environment estimates mediating the relationship between the two measured variables. The percentage of the phenotypic correlation that is due to each of the three sources can be computed by multiplying the common paths (i.e. the two paths connecting the measures) and dividing by the phenotypic correlation. The genetic (ga) and shared (es) and nonshared (en) environmental estimates unique to each measure are also examined in bivariate analyses. A significant specific genetic estimate indicates that there are genetic factors influencing individual differences in one measure that are independent of the genetic influences on the second measure. Several illustrations are provided below.

Several methodological conditions should be noted. First, in these analyses, we examine only additive genetic variance; nonadditive (dominant and epistatic) genetic variance was not modelled because we have little power to discriminate additive and nonadditive genetic influences. Second, consistent with other studies age, sex, age differences, and the age $\times$ sex interaction were partialled from the covariances prior to genetic modelling because these variables can artificially inflate sibling similarity—especially when both twins and nontwins are examined (McGue & Bouchard, 1984). Third, variance/covariance matrices (based on double-entered data) were analysed because they are sensitive to, and allow tests of, variance differences among groups (Cudeck, 1989; Neale & Cardon, 1992). Fourth, both univariate and bivariate behavioural genetic models make certain assumptions about the nature of the processes being estimated. Specifically, the models assume no gene-environment interaction or assortative mating. A more detailed discussion of the validity of these assumptions can be found in Loehlin (1992). In addition, the estimates obtained are unlikely to be biased by a twin-specific effect because the estimates are based on a range of sibling types. Finally, sample size precluded a test of whether the similar phenotypic correlations observed cross-sectionally and longitudinally for boys and girls were nonetheless influenced by a different pattern of genetic and environmental influences.

The significance of parameters in behavioural genetic models were examined by fixing the parameter to 0 and re-estimating the fit, the conventional manner used in behavioural genetic research (Neale & Cardon, 1992). A significant change in the chi-square with 1 *df* indicates a significant worsening of the fit and that the parameter should be retained in the model. For purposes of presentation, however, all estimates are provided and a parameter is *displayed* as 0 only if the model estimated the parameter to be 0. There is wide recognition that the chi-square test for the full model should be accompanied by alternative fit indices, and that each alternative fit index has advantages and limitations. Because of sample size and primary concern regarding residual variance, the root mean square error of approximation was chosen to accompany the chi-square test (Joreskog & Sorbom, 1993). Analyses were run using LISREL, 8th edition (Joreskog & Sorbom, 1993). Model-fitting results for the bivariate models are given in the figure captions.

Results

Univariate Results

Sibling correlations (Table 1) and model-fitting results (Table 2) for antisocial and depressive symptoms at wave 2 are similar to results from wave 1 (O'Connor et al., in press). For antisocial behaviour, the pattern of correlations suggests genetic influence, as the MZ correlation is greater than the DZ and full-sibling correlations which, in turn, are greater than the half-sibling and unrelated-sibling correlations. The pattern of correlations is more complex for depressive symptoms and does not follow a simple genetic cascade. MZ and full-sibling correlations are roughly equal, albeit greater than half-sibling and unrelated-sibling correlations; however, the correlation between unrelated siblings is .25. The implications of this pattern are addressed below. It should be noted that the genetic estimate is derived from the *pattern* of correlations.

Table 2 displays the univariate model-fitting results based on the covariances for antisocial and depressive symptoms at wave 2. For antisocial behaviour, genetic factors accounted for 67% of the variance, with 12% of the variance attributable to shared and 21% of the variance attributable to nonshared environmental influences. For depressive symptoms, 36% of the variance was attributed to genetic factors, with essentially all of the remaining variance attributable to nonshared environmental influences. The significant genetic parameter for depressive symptoms is a result of the full-sibling and half-sibling comparisons. The absence of MZ-DZ differences suggests caution in putting too much emphasis on the estimates obtained for depression.

The next set of analyses examined whether there were variance differences across sibling pairs and whether these differences influenced the parameter estimates. For these analyses, the variances in the MZ sibling group is fixed to 1 and the other sibling groups are allowed to vary, i.e. they are scaled relative to the MZ group. The significance of variance differences is evaluated as a change in chi-square with 5 *df*. Consistent with analyses reported from wave 1, significant variance differences across sibling groups were found for depressive symptoms ($\Delta\chi^2 = 16.83$ with 5 *df* is significant at $p < .05$) but not for antisocial symptoms ($\Delta\chi^2 = 6.44$ with 5 *df* is not significant at $p < .05$). For depressive symptoms larger variances were found for nontwin sibling pairs. The parameter estimates for the depressive symptoms model changed slightly, namely, a decrease in genetic factors and a corresponding increase in shared and nonshared influences (see Table 2). Although the consistent variance differences in depressive symptoms at both waves is noteworthy, there is little evidence that the substantive findings are altered.

Cross-sectional Genetic and Environmental Influences on Symptom Co-occurrence

Next, bivariate behavioural genetic models were used to examine the genetic and environmental influences on the overlap of antisocial and depressive symptoms at wave 2. The phenotypic correlation between dimensions at wave 2 for the entire sample was $r = .41$. The cross-sibling correlations at wave 2 (i.e. sibling A's antisocial behaviour and sibling B's depression) are given in the top half of Table 3. Figure 1 displays the results of bivariate analyses. Model-fitting statistics are provided in the figure caption. The results suggest that the phenotypic correlation of $r = .41$ between dimensions at wave 2 was almost entirely mediated by genetic factors (i.e. $[.61 \times .61]/.41$], with shared and nonshared environment adding minimally (i.e. $[20 \times .20]/.41$; $[.09 \times .09]/.41$, respectively). In addition, genetic variance unique to antisocial behaviour is suggested (ga = .56); in contrast, the genetic influences specific to depression fell just short of statistical significance (ga = .20). There were also significant nonshared environmental influences specific

Table 1
Intraclass Correlations for Antisocial Behaviour and Depression

	Sibling pair					
	MZ	DZ	FI	FS	HS	UN
Antisocial behaviour	.75	.55	.41	.34	.29	.17
Depression	.36	.36	.40	.09	.03	.25

FI = full siblings in nondivorced families; FS = full siblings in stepfamilies; HS = half siblings in stepfamilies; UN = unrelated siblings in stepfamilies.

Table 2
Genetic and Environmental Parameter Estimates for Antisocial and Depressive Symptoms

	Variance Estimates Based on Standardised Parameter Estimates					
	h^2	Es^2	En^2	χ^2 (*df*)	RMSEA	GFI
Antisocial behaviour						
Variances fixed	.67*	.12*	.21*	9.82 (9)	.013	.96
Variances free	.61*	.14*	.25*	2.38 (4)	.000	1.00
Depression						
Variances fixed	.36*	.07	.56*	23.58* (9)	.068	.95
Variances free	.22*	.14	.64*	6.75 (4)	.044	.99

The analyses were run using covariances with variances fixed and free across the sibling groups. For depression, the model that allowed variances to be equal across groups fit significantly better. RSMEA = root mean square error of approximation; GFI = goodness-of-fit index; *df* = degrees of freedom; h^2 = variance due to genetic factors; Es^2 = variance due to shared environmental factors; En^2 = variance due to nonshared environmental factors.

*$p < .05$.

Table 3
Cross-sectional and Longitudinal Cross-sibling Cross-correlations

	Sibling pair					
	MZ	DZ	FI	FS	HS	UN
Cross-sectional						
Antisocial behaviour and depression	.30	.20	.29	.18	.08	.16
Longitudinal						
Antisocial behaviour	.66	.41	.19	.33	.38	.07
Depression	.37	.29	.15	.08	.03	.15

The phenotypic correlation between antisocial behaviour and depressive symptoms at wave 2 was $r = .41$. The phenotypic stability correlations for antisocial behaviour was $r = .63$ and for depression it was $r = .59$. The cross-sibling, cross-dimension correlation examines one adolescent's score on depression with the sibling's score on antisocial behaviour; the cross-sibling, within-dimension longitudinal correlation examines one adolescent's score on (e.g.) depression at wave 1 with the sibling's score on depression at wave 2. The cross-sibling correlations are central to the bivariate analyses (see text).

See Table 1 for abbreviations.

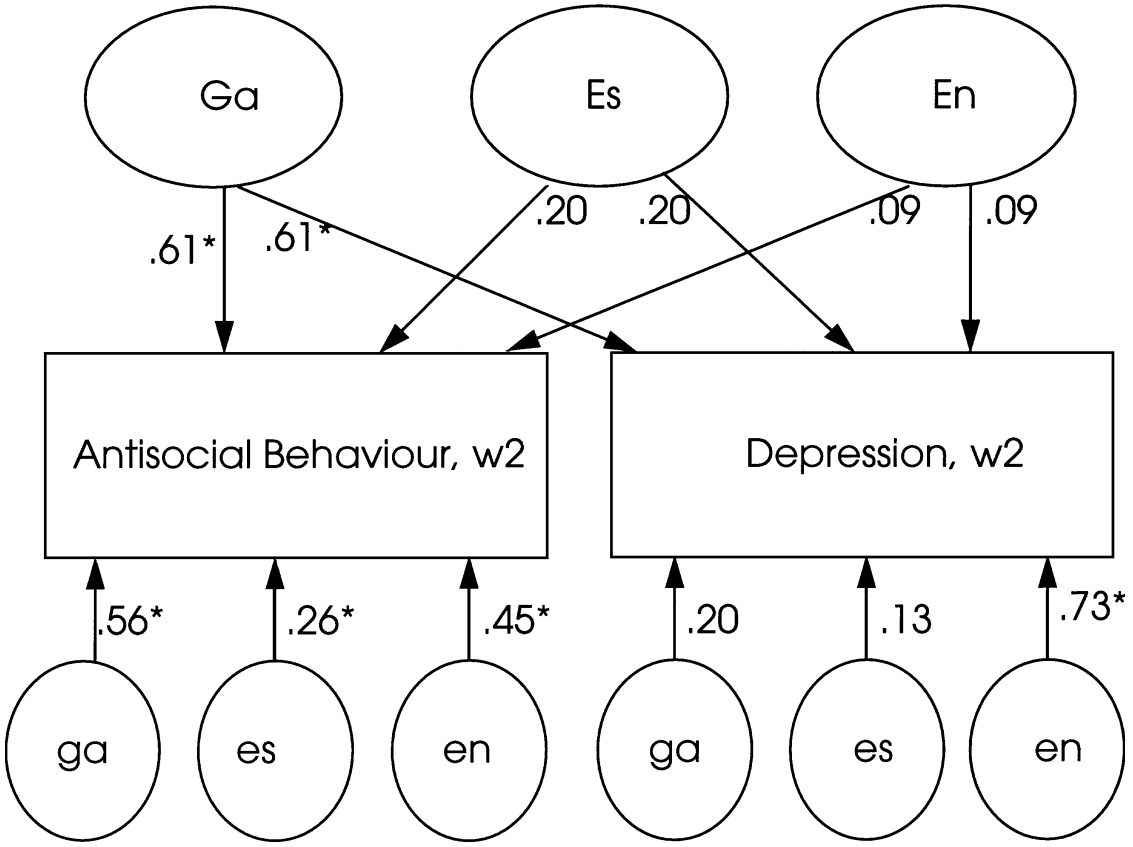

Figure 1. Additive genetic (Ga), shared environment (Es), and nonshared environment (plus error) (En) mediation of the co-occurrence of antisocial and depressive symptoms as well as the genetic (ga), shared environment (es), and nonshared environment (en) estimates specific to antisocial and depressive symptoms. Results for one sibling are presented. Significance of parameters was tested based on comparing the fit of the model with and without the path, with a change in chi-square evaluated with 1 *df* (Neale & Cardon, 1992). For the model, $\chi^2_{(27)} = 35$, $p > .05$; RMSEA = .03; GFI = .95. *$p < .05$. The model was re-analysed allowing variances for the antisocial behaviour and depression measures to vary across group; this alternative model did not fit significantly better, $\chi^2_{(17)} = 17$, $p > .05$; RMSEA = .01; GFI = .98. *$p < .05$. Paths given are based on the former, more parsimonious model. For each model, *df* is equal to the number of unique variances/covariances minus the number of estimated parameters. There are two unique variances and four unique covariances in each of the six groups because the data are double-entered. Thus, the model has 36 total degrees of freedom; there are 9 unique parameters estimated. For Figs. 1–3, the paths from the common genetic and environmental latent factors to the composites are set to be equal in order to estimate the unique genetic and environmental estimates for each composite.

to both dimensions of psychopathology (en = .45 for antisocial behaviour and .73 for depressive symptoms). Finally, only for antisocial behaviour was there evidence for significant shared environmental influence (Es = .28).

It is also possible to compute the per cent of variance explained by genetic and environmental influences on, for example, antisocial behaviour that is not shared with depression. For example, $.56^2/(.56^2 + .61^2)$, or approximately 46%, of the genetic variance on antisocial symptoms is independent of depression (Fig. 1).

Longitudinal Analyses: Within Dimensions

For the whole sample, the phenotypic stability for antisocial behaviour was $r = .63$ and for depression symptoms it was $r = .59$. Cross-sibling correlations across waves 1 and 2 (i.e. the correlation between one sibling's depression at wave 1 and the co-sibling's depression at wave 2) are displayed in the bottom half of Table 3. Genetic influence on stability is suggested for both antisocial and depressive symptoms as the cross-correlations roughly follow a genetic cascade. It is interesting that these longitudinal cross-sibling results for depression are clearer in pointing to genetic influence than are the sibling correlations at either wave. Model-fitting results are displayed in Figs. 2 and 3 for antisocial behaviour and depression, respectively; model-fitting statistics are provided in the figure captions.

For antisocial behaviour, 54% (i.e. $[.58 \times .57]/.63$) of stability of antisocial behaviour was genetically based (Fig. 2). Shared environment accounted for approximately one-third of the stability of antisocial behaviour (i.e. $[.44 \times .43]/.63$) and nonshared environmental influences accounted for 17% (i.e. $[.33 \times .33]/.63$). Similarly, genetics largely explained the stability of depressive symptoms (i.e. $[.66 \times .57]/.59$) (Fig. 3). However, unlike antisocial behaviour, the remaining portion of stability

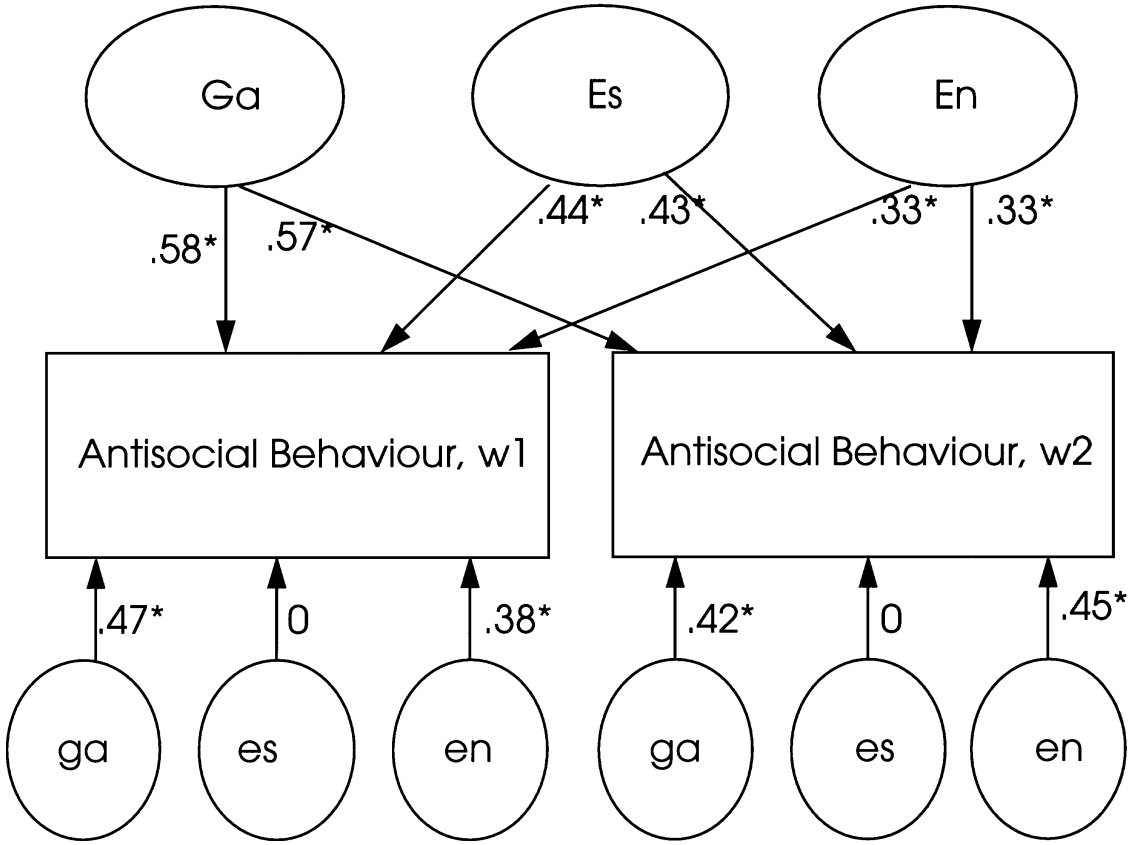

Figure 2. Additive genetic (Ga), shared environment (Es), and nonshared environment (plus error) (En) mediation of the stability of antisocial behaviour as well as the genetic (ga), shared environment (es), and nonshared environment (en) estimates specific to wave 1 (W1) and wave 2 (W2). Parameters estimated to be 0 are fixed to 0 in the figure. Significance of parameters was tested based on comparing the fit of the model with and without the path, with a change in chi-square evaluated with 1 *df* (Neale & Cardon, 1992). For the model, $\chi^2_{(29)} = 78$, $p < .05$; RMSEA = .07; GFI = .86. The model was re-analysed allowing variances for the antisocial behaviour measures to vary across group; this alternative model fit significantly better, $\chi^2_{(19)} = 44$, $p < .05$; RMSEA = .06; GFI = .92. *$p < .05$. Paths given are based on the latter, better-fitting model. In this model, the paths from the common latent factors differ slightly because the variances of antisocial behaviour at waves 1 and 2 differ slightly.

was explained entirely by nonshared environmental factors; shared environment did not contribute to stability.

Also shown in Figs. 2 and 3 are genetic and environmental influences on antisocial and depressive symptoms at wave 2 that are *independent* of genetic and environmental influences at wave 1. In other words, these residuals indicate genetic and environmental factors that contribute to discontinuity. Of particular interest is the finding that for depression there is no evidence of genetic contribution to change (i.e. in Fig. 3 *ga* at wave 2 = 0). However, there is evidence for significant genetic change in antisocial symptoms (i.e. in Fig. 2 *ga* at wave 2 = .42). Indeed, about one-third of the genetic variance on antisocial symptoms at time 2 involves genetic factors that are independent from genetic variance at wave 1 (i.e. $.42^2/[.42^2 + .57^2]$). There were no significant shared environment risks for antisocial behaviour or depression that were unique to wave 2. Finally, nonshared environmental risks specific to antisocial and depressive symptoms were found at both waves (*En* at time 2 = .45 and .65 in Figs. 2 and 3, respectively). Slightly less than one-quarter of the variance in antisocial symptoms at wave 2 could be explained by nonshared environmental influences, most of which are unique to wave 2 (i.e. $45^2/[.45^2 + .33^2]$).

Longitudinal Analyses: Between Dimensions

The hypothesis that the relationship between antisocial behaviour and later depression was genetically mediated was examined using the same bivariate methods outlined earlier. The cross-sibling cross-correlations between wave 1 antisocial symptoms and wave 2 depression are displayed in Table 4. Genetic influence is suggested by the intraclass correlations, as the MZ correlation is significantly greater than the full-sibling correlations, although as in the case of the univariate depression results (Table 1), the correlation for unrelated siblings does not suggest genetic influence. The model-fitting results indicate that over half of the phenotypic correlation of $r = .33$ is due to genetics, with 24% and 19% accounted for by shared and nonshared environmental factors, respectively (not tabled). Next, we examined the hypothesis that genetic and environmental influences on antisocial symptoms at wave 1 made an *independent* contribution to change in depression after accounting for stability in depressive symptoms. Behavioural genetic analyses were

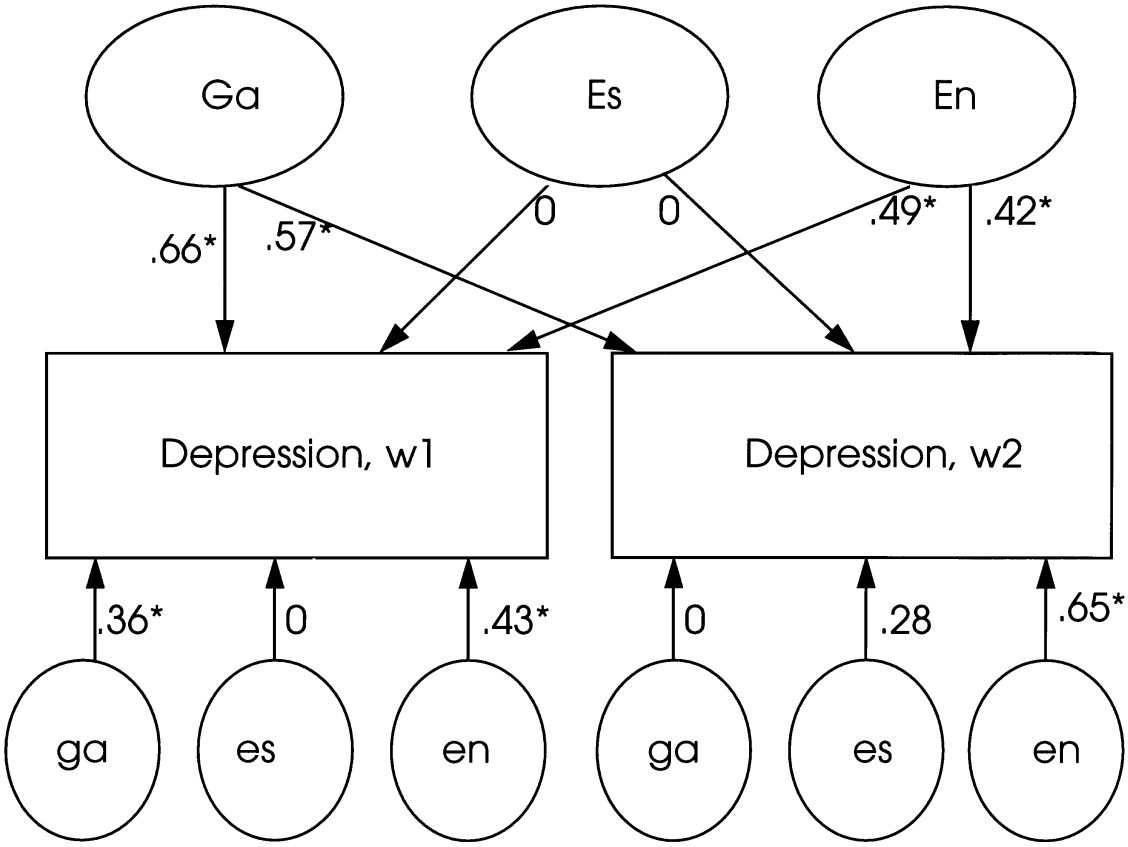

Figure 3. Additive genetic (Ga), shared environment (Es), and nonshared environment (plus error) (En) mediation of the stability of depressive symptoms as well as the genetic (ga), shared environment (es), and nonshared environment (en) estimates specific to wave 1 (W1) and wave 2 (W2). Parameters estimated to be 0 are fixed to 0 in the figure. Significance of parameters was tested based on comparing the fit of the model with and without the path, with a change in chi-square evaluated with 1 *df* (Neale & Cardon, 1992). For the model, $\chi^2_{(30)} = 78$, $p < .05$; RMSEA = .07; GFI = .85. The model was re-analysed allowing variances for the depression measures to vary across group; this alternative model fit significantly better, $\chi^2_{(19)} = 33$, $p < .05$; RMSEA = .04; GFI = .87. *$p < .05$. Paths given are based on the latter, better-fitting model. In this model, the paths from the common latent factors differ slightly because the variances of depression at waves 1 and 2 differ slightly.

Table 4
Longitudinal Cross-correlations: Wave 1 Antisocial Behaviour and Wave 2 Depressive Symptoms

	Sibling pair					
	MZ	DZ	FI	FS	HS	UN
Cross-correlation	.39	.19	.05	.06	.06	.36

The phenotypic correlation for Wave 1 antisocial behaviour and Wave 2 depression (uncorrected for stability) is $r = .33$.
See Table 1 for abbreviations.

pre-empted, however, as the phenotypic regression models indicated that antisocial symptoms at wave 1 made no unique prediction to wave 2 depressive symptoms after first controlling for wave 1 depressive symptoms. That is, because there is no phenotypic change, there can be no genetically mediated change.

Discussion

A longitudinal behavioural genetic design was used in the current study to illuminate two fundamental questions raised by research findings: what accounts for the marked stability in maladjustment and what accounts for the significant overlap between dimensions of maladjustment? The central findings were that genetic influences underlie the stability of antisocial and depressive symptoms over a 3-year period and that the co-occurrence of antisocial and depressive symptoms is mediated, in part, by genetic influences. Equally importantly, the findings support nonshared environmental risks in the stability of antisocial and depressive symptoms. Finally, intra-individual change was also related to genetic as well as shared and nonshared environmental risks, although the pattern of findings was different for antisocial and

depressive symptoms. These results are considered in the context of extant cross-sectional and longitudinal studies of psychopathology in adolescence.

Genetic and Environmental Influences within and between Dimensions

The cross-sectional genetic-environment pattern obtained in wave 2 for antisocial symptoms was very similar to that obtained in wave 1 (O'Connor et al., in press). The genetic estimate is large compared to some reports, but consistent with the finding that genetic influences appear largest on coercive, aggressive types of antisocial behaviours—similar to the definition of antisocial behaviour in this report (Plomin et al., 1990). In contrast, for depressive symptoms there was a decrease in heritability from wave 1 and a complementary increase in nonshared environment, from roughly one-third to approximately one-half (O'Connor et al., in press). The apparent change in the heritability of depression may be a result of age-based changes in the causes of individual differences in depressive symptoms (e.g. Thapar & McGuffin, 1994), but the inclusion of a wide age range at both waves renders such interpretations unlikely in this sample. Subsequent research on samples with more restricted age ranges are required to examine hypotheses regarding the timing of genetic influences on depressive symptoms in adolescence. Sample size precluded analyses to examine genetic influences in younger and older adolescents separately. One measure included in the wave 1 battery was dropped from the wave 2 battery. This does not explain the change, however, because the heritability estimates for the wave 1 composites with and without the measure were essentially identical. These findings heighten the need for further behavioural genetic studies of symptoms that focus on potential age-based changes and differences in the genetic-environmental profile derived from different research designs. More importantly, these data add to the increasing body of research documenting mild to moderate genetic influences on adolescent psychopathology.

These findings replicate the previous report in a second important way, namely, by documenting the significant genetic influences on the co-occurrence of antisocial and depressive symptoms. Whereas approximately half of the correlation between dimensions was attributable to genetic influence at wave 1, the follow-up analyses suggested an even greater genetic influence on syndrome co-occurrence and relatively little role played by shared or nonshared psychosocial risks. Although this difference may highlight a developmental change, as suggested above, the broad age range included in this study precludes developmental change hypotheses. Clearly, follow-up studies are required to specify *which* genetically influenced factors give rise to both antisocial and depressive symptoms. Specific hypothesised mechanisms include abnormalities in the same neurotransmitter system(s) that are common to both dimensions and similar biological substrates that underlie reactivity to stress (see Collins & Depue, 1992; Dahl et al., 1992; Post, 1992). Molecular genetics will undoubtedly provide some key answers to these questions in the near future (Plomin & Rutter, in press). Moreover, these findings parallel phenotypic research on the high rates of co-occurring behavioural problems in adolescents and underscore the need for future studies to examine *patterns* of adjustment problems in adolescence rather than isolated, individual dimensions of maladjustment.

Genetic Risks, Stability, and Change

Both dimensions of psychopathology examined in this report were, as expected, quite stable over the relatively short time span. The stability of antisocial behaviours ($r = .63$) and depressive symptoms ($r = .59$) is well within the range of coefficients reported in other studies of adolescents using dimensional and diagnostic models (Feehan et al., 1993; Stanger et al., 1993; Verhulst & van der Ende, 1993). The novel finding of this paper is that the stability of both antisocial and depressive symptoms was accounted for largely by genetic factors. This finding extends available cross-sectional genetic analyses of adolescent symptomatology and numerous longitudinal genetic analyses of a wide variety of measures throughout the life-span, essentially all of which implicate genetic influences on continuity in development (Pedersen, 1993). These data are also consistent with a recent study that found strong genetic influences on the stability of antisocial behaviour from childhood to adulthood using a retrospective design (Lyons et al., 1995).

Just as important as the finding of genetic continuity is the finding of genetic change for antisocial symptoms during this 3-year period. That is, significant genetic effects at wave 2 were obtained that were independent of genetic influences at wave 1. Indeed, about 40% of the genetic variance on antisocial behaviour at wave 2 is independent of genetic variance at wave 1. In other words, if genes are eventually found that account for genetic influences on antisocial symptoms in adolescence, these results suggest that *different* genes may contribute to variation in antisocial behaviour from early adolescence to early adulthood. This finding of genetic change warrants replication, especially because the essence of developmental behavioural genetics is to identify genetic change as well as continuity (Plomin, 1986), yet evidence for genetic change has been hard to document. This finding is consistent with developmental models of antisocial behaviour that propose changes in the underlying causes of antisocial behaviour (Moffitt, 1993).

The finding of significant genetic influences on antisocial and depressive symptoms—either cross-sectionally or longitudinally—is not to say that these behaviours are immutable or would be refractory to intervention. The reason is that heritability is a descriptive statistic given a particular population and is not readily translated into a likelihood of behavioural change given an alteration in environmental circumstances, such as intervention (Rutter, 1991). Furthermore, recent findings on the prevalence of gene-environment correlations (Plomin, 1994) suggest that finding genetic influence is only a rudimentary step towards understanding how genetic risks manifest as adjustment problems. For example, individuals at genetic risk may seek out and evoke environmental experiences consistent with, or that exacerbate, genetic risks (O'Connor & Rutter, 1996). The finding that depressive and antisocial symptoms are

genetically influenced cross-sectionally and longitudinally does not specify the mechanisms leading to psychopathology, but does suggest that genetically informative designs may be best equipped to address the more complex questions concerning risk processes.

Environmental Risks, Stability, and Change

As Plomin (1994) and others have argued, studies that include a genetic control are best suited to examine environmental sources of individual differences. These results are largely consistent with previous efforts in underscoring the salience of psychosocial risk factors in the development and stability of antisocial and depressive symptoms in adolescence (Cicchetti & Toth, 1995), but may be especially convincing because environmental risks were found that were *independent* of genetic risks.

Nearly one-third of the stability in antisocial behaviour was accounted for by shared environmental risks; in contrast *none* of the stability in depressive symptoms was explained by shared environment. These longitudinal findings reflect cross-sectional results which generally indicate that antisocial behaviour is one of the few behaviours moderately influenced by shared family risks (Plomin et al., 1990; Rowe, 1983; Silberg et al., 1994). Risk factors that are likely to operate on all children within a family to promote antisocial behaviour over time include socioeconomic disadvantage, marital discord, and sibling conflict (e.g. Dishion et al., 1994). In contrast to the results for antisocial behaviour, there is little evidence from the analytic models that there are parallel effects of shared environmental risks on depressive symptoms. It is important to note, however, that the correlation between unrelated siblings for depressive symptoms, when viewed separately from the other sibling groups, suggests that there may be some role for shared environmental effects.

Nonshared or unique environmental risk factors, which have been implicated in cross-sectional assessments of child and adolescent psychopathology (Plomin, 1994; Reiss et al., 1995), were found to underlie the stability of both antisocial and depressive symptoms. In other words, differences in environmental experiences between siblings persist over time and underlie the stable differences in siblings' levels of both depressive and antisocial symptoms. By way of illustration, consider the finding that approximately one-third of the stability in depressive symptoms was accounted for by nonshared environment. It may be that the adolescent who experiences more stressful events continues to experience more stressful events over time so that the differences in depressive symptoms between the adolescent and the sibling at wave 1 are maintained at wave 2. For antisocial behaviour, the nonshared effects were also significant but somewhat less influential. These findings do not suggest *which* nonshared risk factors may promote differences in siblings' adjustment, but several reports implicate parental differential treatment as a likely candidate (Reiss et al., 1995; Rodgers, Rowe, & Li, 1994).

Change in depressive and antisocial symptoms at wave 2 was also driven by nonshared experiences. That is, sibling differences in experiences between waves created adjustment differences at wave 2. This finding is consistent with developmental hypotheses and empirical results regarding the role of sibling-specific psychosocial influences in the increasing differentiation of siblings' personality and psychological adjustment (McCartney, Harris, & Bernieri, 1990; McGue et al., 1993).

A moderate correlation of nonshared environmental risks from wave 1 to wave 2 for both antisocial and depressive symptoms suggests that there are stable individual-specific risk factors that are consistent over time *and* that predispose to both depressive and antisocial symptoms. This is one of the first studies to demonstrate that there are *stable* nonshared risks in adolescent adjustment. Furthermore, the finding that the shared environmental risks are identical for antisocial behaviour over time but virtually unrelated for depressive symptoms was also striking, and suggests that the view of a continuity of environmental risks or "ecological constancy" (Caspi & Moffitt, 1995) may be greater for antisocial than depressive symptoms. The stability of nonshared risks contrasts with a recent study of adult female twins in which environmental influences were entirely transient or occasion-specific over a 17-month period; however, similarly to this study, genetic influences on the liability to depression were entirely stable (Kendler, Neale, Kessler, Heath, & Eaves, 1993). Numerous methodological differences exist between this and the Kendler et al. study, including age and measurement differences, the inclusion of nontwins, and the difference in follow-up periods. Clearly, further research on genetic and environmental stability to psychopathology across the life-span are needed.

A unique aspect of this study was to examine whether the prediction of depressive symptoms from earlier antisocial behaviour, a pathway previously noted by Capaldi (1992) and others, was mediated genetically. Two sets of findings are instructive. First, slightly more than half of the phenotypic correlation of $r = .33$ between antisocial behaviour at wave 1 and depressive symptoms at wave 2 could be accounted for by genetic influences, with shared and nonshared environmental influences each comprising roughly one-quarter of the remaining variance. However, depressive symptoms were quite stable within this relatively short time span and, after partialling out the stable component of depressive symptoms, there was no significant change to be explained, i.e. the partial correlation was essentially zero. Secondly, as an exploratory follow-up analysis, we also examined whether depression was related to change in antisocial behaviour approximately 2 years later, a pattern also noted by Kovacs et al. (1988) and others. The same pattern was obtained, i.e. after accounting for initial levels of antisocial symptoms there was no significant change to explain within the genetic design.

Limitations and New Directions

Several limitations of this study should be noted. First, the estimate of nonshared environment is confounded with measurement error. Second, the fit of the longitudinal models was significant at $p < .05$ and should be interpreted with caution; however, the fit of the models was satisfactory using other conventions (root mean square error of approximation). Third, because of the

nature of the complex twin-sibling design, genetic influences on depressive symptoms were found despite minimal MZ-DZ differences in correlations and covariances. In addition, significant variance differences among groups were found for depressive symptoms and when variances were included in the model there was a slight decrease in the estimate of genetic influence. For these reasons the results for the analyses of depressive symptoms should be interpreted with caution. In this vein, it is interesting to note that no clear consensus regarding genetic influences on depressive symptoms in children and adolescents has yet emerged from existing studies.

Other limitations of this report suggest directions for future research. First, in order to understand how genetic influences may interact with the considerable biological and social changes that occur throughout adolescence, research on narrow age groups is warranted. Longitudinal behavioural genetic analyses of narrow age groups could also examine whether the reported higher rates of stability of psychopathology in late adolescence to adulthood compared to pre- to mid-adolescence (Feehan et al., 1993) may be explained by genetic effects. Additionally, greater appreciation of the developmental changes in genotypic → phenotypic expression across the life-span and across developmental transitions in longitudinal studies will increase our understanding of genetic processes regulating the timing of developmental risks (Gottesman & Goldsmith, 1993). Further research is also needed to examine changes in the gene-environment pattern of influences that may result when alternative definitions of antisocial and depressive symptomatology are used (cf., Kendler, Neale, Kessler, Heath, & Eaves, 1992). There are to date no data documenting a clearly different pattern of findings when dimensional and categorical models of psychopathology are compared. Thus, behavioural genetic research may help bridge the gap between dimensional and categorical models of psychopathology.

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