Psychological Medicine, 2005, **35**, 1825–1835. © 2005 Cambridge University Press
doi:10.1017/S0033291705005829 Printed in the United Kingdom

Internalizing and externalizing problems in adolescence: general and dimension-specific effects of familial loadings and preadolescent temperament traits

J. ORMEL[1,2*], A. J. OLDEHINKEL[1,2,3], R. F. FERDINAND[3], C. A. HARTMAN[1,2], A. F. DE WINTER[1,2], R. VEENSTRA[4], W. VOLLEBERGH[5,6], R. B. MINDERAA[7], J. K. BUITELAAR[8] AND F. C. VERHULST[3]

[1] *Department of Psychiatry, University Medical Centre, Groningen*; [2] *Graduate School of Behavioural and Cognitive Neurosciences & Graduate School for Experimental Psychopathology, University of Groningen*; [3] *Department of Child and Adolescent Psychiatry, Erasmus Medical Centre, Rotterdam*; [4] *Department of Sociology, University of Groningen*; [5] *Trimbos Institute, Utrecht*; [6] *Department of Psychology, University of Leiden*; [7] *Department of Psychiatry and Graduate School of Behavioural and Cognitive Neurosciences, University of Groningen*; [8] *Department of Psychiatry, Radboud University Medical Center, Nijmegen, The Netherlands*

ABSTRACT

Background. We investigated the links between familial loading, preadolescent temperament, and internalizing and externalizing problems in adolescence, hereby distinguishing effects on maladjustment in general *versus* dimension-specific effects on either internalizing or externalizing problems.

Method. In a population-based sample of 2230 preadolescents (10–11 years) familial loading (parental lifetime psychopathology) and offspring temperament were assessed at baseline by parent report, and offspring psychopathology at 2·5-years follow-up by self-report, teacher report and parent report. We used purified measures of temperament and psychopathology and partialled out shared variance between internalizing and externalizing problems.

Results. Familial loading of internalizing psychopathology predicted offspring internalizing but not externalizing problems, whereas familial loading of externalizing psychopathology predicted offspring externalizing but not internalizing problems. Both familial loadings were associated with Frustration, low Effortful Control, and Fear. Frustration acted as a general risk factor predicting severity of maladjustment; low Effortful Control and Fear acted as dimension-specific risk factors that predicted a particular type of psychopathology; whereas Shyness, High-Intensity Pleasure, and Affiliation acted as direction markers that steered the conditional probability of internalizing *versus* externalizing problems, in the event of maladjustment. Temperament traits mediated one-third of the association between familial loading and psychopathology. Findings were robust across different composite measures of psychopathology, and applied to girls as well as boys.

Conclusions. With regard to familial loading and temperament, it is important to distinguish general risk factors (Frustration) from dimension-specific risk factors (familial loadings, Effortful Control, Fear), and direction markers that act as pathoplastic factors (Shyness, High-Intensity Pleasure, Affiliation) from both types of risk factors. About one-third of familial loading effects on psychopathology in early adolescence are mediated by temperament.

* Address for correspondence: Prof. dr. J. Ormel, Department of Psychiatry, University Medical Centre, Groningen, Hanzeplein 1, 9700 RB Groningen, The Netherlands.
(Email: j.ormel@med.umcg.nl)

INTRODUCTION

This report extends earlier cross-sectional work on the temperament–psychopathology relationship by adding a prospective component and familial loadings, as indexed by lifetime parental psychopathology. The earlier work found diverging temperament profiles between four groups of preadolescents, namely, those with (1) neither internalizing (anxiety, depression, somatization) nor externalizing (aggressive and rule-breaking behaviour) problems; (2) only internalizing problems; (3) only externalizing problems; and (4) both internalizing and externalizing problems (Oldehinkel *et al.* 2004). The temperament trait of Frustration was mainly related to maladaptation in general, whereas High-Intensity Pleasure (i.e. pleasure derived from intense or novel activities) and Shyness steered the conditional probability of externalizing *versus* internalizing problems. Fear and Effortful Control were associated with both the severity and direction of internalizing and externalizing problems, respectively. These associations are intuitively appealing and in line with other work (e.g. Fowles, 1993; Rothbart & Bates, 1998; Widiger *et al.* 1999; Rothbart *et al.* 2000; Shiner & Caspi, 2003) that showed that internalizing problems are typically associated with personality features from the domain of negative affectivity (or neuroticism) and a relatively overactive anxiety-related behavioural inhibition system whereas externalizing problems often go together with traits from the domain of novelty-seeking and a relatively overactive approach-related behavioural activation system.

The earlier work did not take into account familial influences and was cross-sectional. It is well known that both temperament and psychopathology are partly under genetic control and share their genetic determinants to some extent (Eaves *et al.* 1999; Plomin *et al.* 2001; Benjamin *et al.* 2002). In addition, non-genetic familial influences may also operate on both temperament and psychopathology (Rutter *et al.* 1997). Consequently, the association between temperament and psychopathology could be spurious rather than causal, due to the impact of familial influences on both. Another possibility is that temperament mediates familial influences on psychopathology. Aetiological models often assume that familial influences on psychopathology operate partly via temperament (Plomin, 1994; Rutter & Silberg, 2002) but empirical tests rarely address internalizing and externalizing problems simultaneously. Hence, it is not clear to what extent mediation of familial influences via temperament actually occurs and how specific the mediation is with regard to the major dimensions of psychopathology. The second problem with the earlier work was its cross-sectional nature, which hampered the interpretation of statistical associations. The current study related baseline measures of familial loading and preadolescent temperament to mental health as manifested in early adolescence.

Limited agreement between informants and informant-related method variance are important issues in the study of child and adolescent psychopathology (Kraemer *et al.* 2003). Informants provide partly unique information about children's behaviour by observing the children in different contexts from their own perspective (e.g. Achenbach *et al.* 1987; Renouf & Kovacs, 1994). Since simple combination rules work as well, if not better, than more complicated ones (Bird *et al.* 1992; Piacentini *et al.* 1992), we will compare two simple combinations: (1) the mean score of the standardized parent, child, and teacher ratings, and (2) the highest score of the three informants, that is, using for each dimension the informant with the highest rating. Each combination has strengths and weaknesses. The 'mean score' approach acknowledges that problems identified by multiple informants are more general and hence probably more severe than problems rated by only one informant. The 'highest score' approach recognizes that informants may be unaware of (part of) the child's behaviour and prevents this from resulting in too low a problem score if the mean score approach had been applied. Another problem arises if the same informant, often the parent, provides information on determinants and outcomes as well. Reliance on a single source of information is problematic if it leads to shared method variance because shared method variance will artificially inflate the associations. Therefore, we will examine whether results remain the same if the parent report on psychopathology is disregarded.

In sum, we will examine, in a large, population-based sample, the relationships between

familial loadings, preadolescent temperament, and psychopathology in early adolescence. Central questions regard the specificity of the associations in terms of general *versus* dimension-specific effects, confounding of the temperament–psychopathology association by familial loading, temperamental mediation of familial loading effects on psychopathology, and the robustness of findings across different composite multi-informant measures. Our last aim was to check the assumption that the pattern of relationships is invariant across gender, despite gender differences in temperament and psychopathology (e.g. Feingold, 1994; Verhulst *et al*. 1997).

METHODS

Sample

The Tracking Adolescents' Individual Lives Survey (*TRAILS*)

TRAILS is a new prospective cohort study of Dutch preadolescents. The present study involves data from the first (T1) and second (T2) assessment wave of TRAILS, which ran from March 2001 to July 2002 and September 2003 to December 2004, respectively. A detailed description of the sampling procedure and methods is provided in de Winter *et al*. (2005). Briefly, the TRAILS target sample involved all 10- to 11-year-old children living in the three largest cities and some rural areas in the North of The Netherlands. Of the eligible households, 76·0% ($n=2230$) were enrolled in the study (i.e. both child and parent agreed to participate). Responders and non-responders did not differ with respect to the prevalence of teacher-rated problem behaviour and the associations between sociodemographic variables and mental health indicators (de Winter *et al*. 2005).

Of the 2230 baseline (T1) participants, 96·4% ($n=2149$, 51·2% girls) participated in the first follow-up assessment (T2), which was held 2–3 years after T1 (mean number of months 29·44, S.D. = 5·37). Mean age at T2 was 13·55 (S.D. = 0·54).

Measures

Data collection

At T1 well-trained interviewers visited one of the parents or guardians (preferably the mother, 95·6%) in their homes to administer an interview covering a wide range of topics. Besides the interview, the parent was asked to fill out a written questionnaire. Children were measured at school, where they filled out questionnaires in groups, under the supervision of TRAILS assistants, but were also assessed individually. Teachers filled out a brief questionnaire for each TRAILS-child in their class. T2 involved only self-report questionnaires, to be filled out by the children (early adolescents now), their parents and their teachers. As in T1, the adolescents filled out their questionnaires at school, supervised by TRAILS assistants.

Lifetime parental psychopathology was assessed at T1 by means of the TRAILS Family History Interview (FHI), administered at the parent interview. Five spectra (or dimensions) of psychopathology were assessed: depression, anxiety, substance dependence, persistent antisocial behaviour, and psychosis. Each spectrum was introduced by a vignette (available on request) describing the main DSM-IV characteristics of the spectrum, followed by a series of questions assessing lifetime occurrence, professional treatment, and medication use. Biological parents were interviewed separately using a single informant, typically the mother. For each spectrum, we assigned each parent to one of the following categories: 0 = (probably) never had an episode, 1 = (probably) yes, or 2 = yes and treatment and/or medication. For antisocial behaviour, the last category was: 2 = (probably) yes and police involvement. Prevalence rates in mother and fathers respectively were, for depression: 27% and 15%; for anxiety: 16% and 6%; for substance dependence: 3% and 7%; and for antisocial behaviour: 3% and 7%. The FHI rates were by and large comparable to the CIDI–DSM-IV lifetime rates obtained by direct interviewing in NEMESIS (Bijl *et al*. 1998); the exception being fathers' rates for anxiety disorder and substance dependence that were 40% too low.

Subsequently, we calculated familial loadings for the domains of internalizing and externalizing disorders separately. Both are effectively a count of the number of lifetime disorders within each domain reported by the biological parents. As internalizing disorders we combined depression and anxiety; as externalizing disorders substance dependence and antisocial behaviour.

The empirical justification for the construction of the familial loadings is twofold (data available on request). First, disorders within each domain were more strongly correlated (on average 0·34) than disorders across domains (0·12), for mothers as well as fathers. Factor analysis of the disorder correlation matrix, for fathers and mothers separately, yielded two factors of internalizing and externalizing problems that were similar to the two-dimensional structure of common mental disorders (Krueger, 1999; Vollebergh *et al.* 2001; Kendler *et al.* 2003). Secondly, the pattern of associations between parental disorders and offspring psychopathology was similar for fathers and mothers, suggesting that the paternal and maternal indices could be combined without obscuring relevant details. In line with this also is that paternal disorders were weakly correlated with maternal disorders. For instance, paternal and maternal depression were associated (0·18) and so were paternal and maternal antisocial behaviour (0·26).

Child Temperament was assessed at T1 by the parent version of the short form of the Early Adolescent Temperament Questionnaire–Revised (EATQ-R) (Putnam *et al.* 2001; Ellis *et al.* 2004). The Dutch version of the EATQ-R (Hartman *et al.* 2000) identifies the dimensions of High-Intensity Pleasure (the pleasure derived from activities involving high intensity or novelty), Shyness (behavioural inhibition to novelty and challenge, especially social), Fear (worrying and unpleasant affect related to the anticipation of distress), Frustration (negative affect related to interruption of ongoing tasks or goal-blocking), Affiliation (the desire for warmth and closeness with others, independent of shyness or extraversion), and Effortful Control (the capacity to voluntarily regulate behaviour and attention). The six EATQ scales were examined on item-content overlap with the psychopathology measures and some problematic items were removed (see *Purified scales*).

Child psychopathology was assessed at T1 and T2 with the parent-rated Child Behaviour Checklist (CBCL) (Achenbach, 1991*a*), the Youth Self-Report (YSR) (Achenbach, 1991*b*) and the Teacher Checklist of Psychopathology (TCP). Their timeframe was the past 2–6 months. The TCP contains descriptions of problem behaviours corresponding to the syndromes of the CBCL and YSR (de Winter *et al.* 2005). We constructed broad-band scales of internalizing problems (consisting of items from Anxious/Depressed, Withdrawn/Depressed, and Somatic Complaints) and externalizing problems (with items from Aggressive behaviour and Rule-Breaking behaviour) (Oldehinkel *et al.* 2004). The TCP broad-band scores were calculated by summing the scores of the Anxious/Depressed, Withdrawn/Depressed and Somatic Complaints vignettes yielding an TCP internalizing problem score, and the scores on the Aggressive Behaviour and Rule-breaking Behaviour vignettes, yielding the TCP externalizing problems score.

Purified temperament and psychopathology scales

To examine item-content overlap between the T1 temperament and psychopathology scales, we performed series of exploratory (EFA) and confirmatory factor analyses on the T1 data (CFA) using SPSS 11 and MPLUS 3.11 software, according to Lemery *et al.* (2002). For both the CBCL and the YSR, separate analyses were performed for every combination of temperament traits (all EATQ scales) and psychopathology dimensions (internalizing, externalizing). The analyses were repeated until all remaining items loaded above 0·30 on the correct factor and below 0·30 on the wrong factor. All of this yielded the following scales (available on request): CBCL-Internalizing (from 24 to 19 items, Cronbach's $\alpha = 0{\cdot}82$); CBCL-Externalizing (from 23 to 18 items, $\alpha = 0{\cdot}86$); YSR-Internalizing (from 28 to 26 items, $\alpha = 0{\cdot}87$); YSR-Externalizing (from 23 to 19 items, $\alpha = 0{\cdot}84$); EATQ-Effortful Control (11 items, $\alpha = 0{\cdot}86$); EATQ-Frustration (five items, $\alpha = 0{\cdot}74$); EATQ-Fear (from five to three items, $\alpha = 0{\cdot}58$); EATQ-High-Intensity Pleasure (six items, Cronbach's $\alpha = 0{\cdot}77$); EATQ-Shyness (four items, $\alpha = 0{\cdot}84$); EATQ-Affiliation (from six to five items, $\alpha = 0{\cdot}66$).

Composite multi-informant indices for internalizing, externalizing, and total problems

For both internalizing and externalizing problems, we calculated composite scales. The multi-informant mean score measure represents the mean of the standardized parent, child, and teacher scores. The multi-informant highest

score measure reflects the standardized score of the informant with the highest score. We constructed total problem indices by adding the internalizing and externalizing scales. Finally, we constructed indices that did not include the parent-rated CBCL.

Statistical analysis

We used standardized values (*z*-scores) for all variables to achieve internally comparable regression coefficients. All analyses were performed by the statistical program STATA (StataCorp, 2003). To obtain a general impression of the associations amongst predictors and outcomes, product–moment correlations were calculated.

We estimated effects of familial loading of internalizing and externalizing pathology on temperament traits by means of six multiple linear regression analyses, one for each trait. Thus the familial loading effects were adjusted for each other.

Next, we estimated familial loading and temperament effects on T2 psychopathology by means of three sets of regression analyses. The first set addressed internalizing problems (after variance shared with externalizing problems was partialled out), the second set addressed externalizing problems (after variance shared with internalizing problems was partialled out), and the third set total problems (internalizing + externalizing). Each set consisted of three different multiple linear regression analyses. The first multiple regression analysis addressed the regression of psychopathology on familial loading. The second one addressed the regression of psychopathology on the six temperament traits. These two analyses yielded familial loading effects adjusted for the other familial loading variable and temperament effects adjusted for the other temperament variables, respectively. In the third multiple regression analysis both the familial loading and temperament variables were included as predictors and we estimated their independent effects on psychopathology, thus simultaneously adjusted for both familial loading and temperament. Comparing adjusted and unadjusted effects will inform on confounding and effect mediation. For instance, if temperament effects adjusted for familial loading are similar to unadjusted effects, we may conclude that familial loading does not confound or mediate the association between temperament and psychopathology. Likewise, if familial loading effects on psychopathology adjusted for temperament are similar to unadjusted effects, confounding or mediation by temperament is unlikely.

All regression analyses of T2 psychopathology were repeated with various measures to examine the robustness of the findings across several approaches. Measures were the T2 mean score and highest score psychopathology composites and their version without the parent ratings.

We examined gender effects by adding gender and interactions of gender and, respectively, familial loadings and temperament traits to the regression analyses.

RESULTS

Table 1 shows the associations between familial loading, temperament, and T2 psychopathology. Internalizing and externalizing symptoms were weakly correlated, as were internalizing and externalizing familial loading. Some temperament traits were moderately correlated as well, such as low Effortful Control with Frustration, others weakly, such as Frustration with Fear, and low Shyness with High-Intensity Pleasure and Affiliation.

Familial loading and temperament

Results of the regression analyses of temperament traits on familial loading are presented in Table 2. Adjusted for their overlap, both familial loadings independently influenced Frustration and Effortful Control. In contrast, Fear and High-Intensity Pleasure were influenced by either internalizing or externalizing familial loading, but not both.

Familial loading and psychopathology

After controlling for the overlap between internalizing and externalizing symptoms and adjusted for each other, familial loading had rather specific effects on psychopathology (data available on request). A high familial loading of internalizing disorders predicted internalizing but not externalizing symptoms whereas a high familial loading of externalizing disorders predicted externalizing but not internalizing

Table 1. *Pearson correlation matrix of T1 familial loadings, T1 temperament traits, and T2 composite psychopathology measures*

	T2 internalizing symptoms	T2 externalizing symptoms	T2 total symptoms	Familal loading: internal	Familial loading: external	Effortful Control	Frustration	Fear	Shyness	High-Intensity Pleasure	Affiliation
T2 internalizing symptoms	1·00										
T2 externalizing symptoms	0·27	1·00									
T2 total symptoms	0·82	0·77	1·00								
Familial loading: internalizing	0·19	0·12	0·19	1·00							
Familial loading: externalizing	0·08	0·18	0·15	0·25	1·00						
Effortful Control	−0·15	−0·31	−0·28	−0·12	−0·12	1·00					
Frustration	0·20	0·30	0·30	0·12	0·07	−0·40	1·00				
Fear	0·20	0·09	0·19	0·12	0·07	−0·22	0·27	1·00			
Shyness	0·17	−0·12	0·04	0·03	−0·03	−0·02	0·10	0·13	1·00		
High-Intensity Pleasure	−0·16	0·12	−0·04	0·01	0·06	0·04	−0·01	−0·18	−0·30	1·00	
Affiliation	0·00	−0·06	−0·03	0·04	0·02	0·12	−0·16	0·11	−0·29	0·15	1·00

Table 2. *Effects of T1 familial loadings on T1 temperament traits*[a]

	Regression coefficient (95% CI)[b]	
Dependent variable	Family loading of internalizing problems[b]	Family loading of externalizing problems[b]
Effortful Control	−0·09 (−0·14 to −0·05)***	−0·10 (−0·15 to −0·06)***
Frustration	0·11 (0·06 to 0·16)***	0·05 (0·01 to 0·09)*
Fear	0·11 (0·07 to 0·16)***	0·02 (−0·02 to 0·07)
Shyness	0·04 (−0·01 to 0·08)	−0·04 (−0·08 to 0·01)
High-Intensity Pleasure	−0·01 (−0·05 to 0·04)	0·06 (0·02 to 0·11)**
Affiliation	0·02 (−0·02 to 0·07)	0·01 (−0·03 to 0·06)

[a] All variables were standardized to mean zero and standard deviation 1.
[b] Familial loading effects are adjusted for one another.
*$p \leqslant 0{\cdot}05$, ** $p \leqslant 0{\cdot}01$, *** $p \leqslant 0{\cdot}001$.

symptoms. Total symptoms was predicted by both familial loadings.

Temperament and psychopathology

According to the unadjusted correlations in Table 1, high Frustration, low Effortful Control, and high Fear appeared to distinguish between maladaptive and adaptive behaviour, because they correlated with internalizing as well as externalizing and total symptoms. Shyness and High-Intensity Pleasure seemed predominantly dimension-specific markers. The latter two traits were not associated with total symptoms but correlated each with internalizing symptoms and also, but inversely, with externalizing symptoms.

After adjustment for the co-variance amongst the temperament traits and the overlap between internalizing and externalizing problems, the pattern of relationships slightly changed (data available on request). Frustration remained a predictor of internalizing, externalizing, and total symptoms (data available on request). Frustration remained a predictor of internalizing, externalizing, and total symptoms. But adjusted, Effortful Control and Fear had more specific effects. Effortful Control predicted externalizing but no further internalizing symptoms whereas Fear did the opposite. Both still predicted total symptoms. A second difference was that Affiliation emerged in the adjusted analyses as a trait that did not predict

Table 3. *Multiple linear regression analyses of, respectively, T2 internalizing problems, externalizing problems, and total amount of problems on T1 familial loadings and temperament traits*[a]

Predictors	Regression coefficients (95% CI)		
	Internalizing problems (adjusted for externalizing problems)	Externalizing problems (adjusted for internalizing problems)	Total amount of problems
Familial loading: internalizing	0·13 (0·09 to 0·17)***	0·01 (−0·03 to 0·05)	0·12 (0·07 to 0·16)***
Familial loading: externalizing	−0·01 (−0·05 to 0·04)	0·10 (0·06 to 0·14)***	0·07 (0·03 to 0·12)**
Effortful Control	−0·01 (−0·06 to 0·03)	−0·19 (−0·23 to −0·14)***	−0·16 (−0·20 to −0·11)***
Frustration	0·08 (0·04 to 0·13)**	0·16 (0·12 to 0·21)***	0·20 (0·16 to 0·25)***
Fear	0·07 (0·03 to 0·12)**	0·00 (−0·04 to 0·04)	0·07 (0·02 to 0·11)**
Shyness	0·16 (0·11 to 0·21)***	−0·16 (−0·20 to −0·11)***	0·01 (−0·04 to 0·05)
High-Intensity Pleasure	−0·14 (−0·19 to −0·10)***	0·12 (0·08 to 0·16)***	−0·02 (−0·07 to 0·02)
Affiliation	0·09 (0·05 to 0·14)***	−0·08 (−0·12 to −0·04)***	0·02 (−0·03 to 0·06)

[a] All variables are standardized to mean zero and standard deviation one. The outcome variables represent multi-informant mean score composite measures.

* $p \leqslant 0{\cdot}05$, ** $p \leqslant 0{\cdot}01$, *** $p \leqslant 0{\cdot}001$.

total symptoms but was related to both dimensions of psychopathology in different directions as were Shyness and High-Intensity Pleasure. High Affiliation predicted internalizing symptoms and low Affiliation externalizing symptoms.

Familial loading, temperament and psychopathology

Table 3 presents the results of the regression analyses that adjusted simultaneously for both familial loading and temperament, as well as the overlap between internalizing and externalizing problems. Frustration remained a general risk factor; Effortful Control, Fear, and both familial loadings still acted as dimension-specific risk factors, and Shyness, High-Intensity Pleasure, and Affiliation remained conditional direction markers that were unrelated to total symptoms but directed maladjustment towards either internalizing or externalizing problems.

Adjusting for familial loading did not reduce temperament effects on psychopathology, suggesting that familial loading did not confound or mediate the association between temperament and psychopathology. However adjusting for temperament reduced the effects of familial loadings on psychopathology, although they remained specific and statistically significant, as Table 3 shows. The effect of a high familial loading of internalizing pathology on offspring internalizing and total symptoms dropped from 0·17 and 0·18 to 0·13 and 0·12; the effect of a high familial loading of externalizing pathology on offspring externalizing and total symptoms dropped from 0·15 and 0·11 to 0·10 and 0·07. On average, temperament traits mediated one-third of the effect of familial loading on psychopathology.

Girls *versus* boys

Girls and boys differed at age 13–14 in psychopathology and temperament, in that girls had more internalizing problems [$t(2147) = 10{\cdot}21$, $p < 0{\cdot}001$] but fewer externalizing problems [$t(2147) = -5{\cdot}74$, $p < 0{\cdot}001$] and more problems in total [$t(2147) = 3{\cdot}46$, $p < 0{\cdot}001$]. Furthermore, we found gender differences in all temperament traits: girls had higher levels of Effortful Control [$t(1985) = 8{\cdot}20$, $p < 0{\cdot}001$], Fear [$t(1982) = 5{\cdot}11$, $p < 0{\cdot}001$], Shyness [$t(1984) = 4{\cdot}49$, $p < 0{\cdot}001$], and Affiliation [$t(1984) = 9{\cdot}55$, $p < 0{\cdot}001$]; and lower levels of Frustration [$t(1983) = -3{\cdot}35$, $p < 0{\cdot}001$] and High-Intensity Pleasure [$t(1980) = -5{\cdot}51$, $p < 0{\cdot}001$]. As might be expected, girls and boys did not differ with respect to familial loadings [internalizing: $t(2159) = 0{\cdot}51$, $p = 0{\cdot}61$; externalizing: $t(2165) = -0{\cdot}11$, $p = 0{\cdot}91$].

Interactions of gender with familial loadings and temperament traits were virtually all statistically non-significant. We found one gender by familial loading interaction ($p = 0{\cdot}011$), in that the association of familial loading of externalizing pathology with externalizing problems was stronger in boys than in girls. None of the gender by temperament interactions reached statistical significance.

Robustness

Additional analyses in which we used the highest score composite measures of psychopathology instead of the mean score composite measures yielded very similar regression coefficients (data available on request). We also repeated the multiple regression analyses with composite measures that excluded parent ratings. The pattern of associations did not change but the effects were generally slightly weaker (data available on request). This is not unexpected given the increase in measurement error due to the loss of a major indicator.

DISCUSSION

This study aimed to expand earlier work on the relationship between temperament and psychopathology in preadolescents in two ways: first by adding familial loading of internalizing and externalizing disorders and secondly by using a prospective design in which baseline familial loading and preadolescent temperament were related to psychopathology in early adolescence. Familial loadings acted as dimension-specific risk factors, in that familial loading of internalizing psychopathology predicted internalizing but not externalizing problems, whereas familial loading of externalizing disorders predicted externalizing but not internalizing problems (note that variance shared by externalizing and internalizing problems was partialled out). As a result they also predict independently the total amount of problems.

Adjusted for overlap between the preadolescent temperament traits, three types of relationship with later psychopathology could be distinguished: general risk factors, dimension-specific risk factors, and conditional direction markers. The latter do not increase risk but shape the problems, if any, in either the internalizing or externalizing direction. Adjustment for familial loading did not affect the effect of temperament traits on psychopathology, rendering the temperament–psychopathology association free of confounding by familial loadings. However, adjustment for temperament effects reduced the association of familial loading with psychopathology by one-third, suggesting that the effects of familial loadings on offspring mental health are partly mediated by offspring temperament.

Overall, the effects were weak but unequivocal and distinctive, and independent of gender despite significant gender differences in temperament and psychopathology. The findings regarding the familial loadings and most temperament traits provide support for the specificity hypothesis of partly different aetiologies of internalizing *versus* externalizing problems (e.g. Kendler *et al*. 2003; Krueger & Tackett, 2003). The findings regarding the temperamental trait of Frustration corroborate the notion that internalizing and externalizing problems also share risk factors (Neeleman *et al*. 2004). Stronger associations may be found with lifetime measures of offspring psychopathology and in late adolescence and adulthood.

The effects of familial loadings on offspring mental health have environmental and genetic origins, as the psychiatric disorders that underlie the familial loadings have complex aetiologies in which genetic and environmental factors interact (e.g. Rutter *et al*. 1997; Rutter & Silberg, 2002). Although the notion of $G \times E$ interplay makes it less useful to think in the terms of relative contributions, quantitative twin studies generally suggest that one-third to two-thirds of the variance in liability to internalizing and externalizing disorders has genetic origins (e.g. Kendler *et al*. 2003; McGuffin *et al*. 2002).

Limitations and strengths

The findings should be interpreted in the light of three limitations. First, the familial loadings for internalizing and externalizing disorders are crude approximations of essentially continuously distributed familial loadings, as they concern only parents and not other relatives and did not take into account age differences between families. Furthermore, we did not interview each biological parent in person but interviewed only one parent directly, usually the mother, and used this parent as informant for the other parent. The evidence on the drawbacks of family history interviews as compared with direct interviews of relatives is mixed (e.g. Caspi *et al*. 2001; Buecking *et al*. 2004), but generally points to underreporting of lifetime parental psychopathology. Except for fathers' anxiety and substance dependence and perhaps mother's antisocial behaviour, our prevalence rates were comparable to the lifetime rates of the large

NEMESIS study that used direct interviewing. Since we pooled paternal and maternal disorders in two broad familial loading indices, under- and overreporting of specific disorders may have averaged out somewhat. Yet, underreporting and thus underestimation of associations is possible. Secondly, we used the same parent as informant for lifetime parental psychopathology and offspring temperament. Since this parent was also one of the three informants on offspring psychopathology, the measures may share method variance. The reliance on a single source is problematic as it may inflate associations and cause bias. The first may have occurred but probably not the latter. When we removed the parent report from the multi-informant measures, the effects fell slightly but the pattern of relationships did not change. Thirdly, we could not control for potentially confounding factors that are correlated with parental psychiatric history and preadolescent temperament. For instance, chronic family discord might have influenced both parental and offspring mental health, and obstetric complications could have influenced both offspring temperament and mental health.

Major strengths include the large population-based sample; the prospective design; the use of composite measures of psychopathology based on multiple informants (parent, teacher, child); and the robustness of findings with different composite multi-informant measures with and without parent ratings of offspring psychopathology. Further assets include the use of purified measures of psychopathology and temperament from which items with similar content were removed, making it unlikely that the prospective association between temperament and psychopathology is inflated by item–content overlap.

Temperament trait-specific influences on psychopathology

Frustration acted as a general risk factor that predicted maladjustment whereas Effortful Control and Fear acted as dimension-specific risk factors. Shyness, High-Intensity Pleasure and Affiliation were direction markers, steering the conditional probability of internalizing *versus* externalizing problems. How well do these findings fit models of the temperament–psychopathology relationship (Clark *et al*. 1994; Costa & Widiger, 2002; Shiner & Caspi, 2003)? The spectrum model states that psychopathology represents the extremes of continuously distributed temperament traits or clusters, which implies substantial continuity of psychopathology over time. The vulnerability/resilience model assumes that, in the face of adversity, temperament sets in motion processes that cause the development of psychopathology (vulnerability) or protect against it (resilience). This model implies more within-person variation of psychopathology than the spectrum model (e.g. Ormel & Schaufeli, 1991). A third model is the pathoplastic model that asserts that temperament shapes the form of psychopathology but does not play a causative role. Our study suggests that different temperament traits fit different models; with Frustration acting in accordance with the vulnerability/resilience model, Effortful Control and Fear in accordance with the spectrum model, and Shyness, High-Intensity Pleasure and Affiliation in accordance with the pathoplastic model. Within each model the traits can exert their influence via person–environment correlation (temperament-related individual differences in exposure to environments), person–environment interaction (temperament-related individual differences in sensitivity to environments), or both (Plomin, 1994; Rutter *et al*. 1997).

The importance of specific temperament and personality facets

There is increasing evidence that temperament and personality are less distinct than often assumed (McCrae *et al*. 2000). Their close connection is clearly recognized in Rutter's (1987) definition of personality as 'the social and cognitive elaborations of temperament endowment: the thoughts, feelings, attitudes, and values that project early-emerging stylistic differences out into the world'. Our findings emphasize the importance of studying the role of personality in psychopathology at the level of the facets of the broad dimensions of personality. The big five are too crude for this task, since the very same dimension may subsume differentially operating facets. For instance, Neuroticism probably contains facets that largely act as general risk factors (e.g. vulnerability, angry hostility), while other facets of

Neuroticism have more dimension-specific effects (e.g. anxiety, depression for internalizing problems; impulsiveness for externalizing problems). Similar heterogeneity may characterize other broad dimensions of personality such as Conscientiousness and Extroversion.

CONCLUSION

We found meaningful general and dimension-specific relationships between familial loading, temperament and psychopathology. Since only one-third of the familial loading effects were mediated by temperament, additional mediators must play a role. The findings stress the significance of distinguishing (i) general risk factors, (ii) dimension-specific risk factors, and (iii) conditional direction markers that do not increase risk but steer the conditional probability of internalizing *versus* externalizing problems. In particular, the existence of general risk factors and conditional direction markers is important as they add two types of determinants to the types of spectrum-specific (internalizing, externalizing) and disorder-specific risk factors that have been demonstrated in studies of the structure of common mental and behavioural disorders and underlying genetic architecture (e.g. Kendler *et al*. 2003; Krueger & Tackett, 2003). Temperament traits may yield potentially valuable endophenotypes for psychiatric genetic research (e.g. Benjamin *et al*. 2002). In particular, the general risk factor Frustration and the dimension-specific risk factors Effortful Control and Fear are interesting candidates because they mediate familial loading effects and have differential relationships with psychopathology.

ACKNOWLEDGEMENTS

This research is part of the Tracking Adolescents' Individual Lives Survey (TRAILS). Participating centres of TRAILS include various Departments of the University of Groningen [J. Ormel (PI), S. M. Lindenberg, R. B. Minderaa, A. J. Oldehinkel, M. Reyneveld, A. F. de Winter]; the Erasmus Medical Center of Rotterdam [F. C. Verhulst (PI), R. F. Ferdinand]; the University of Nijmegen (J. Buitelaar); the Trimbos Institute and University of Leiden (W. A. M. Vollebergh).

TRAILS is financially supported by grants from the Netherlands Organization for Scientific Research (GB-MW 940-38-011, GB-MAG 480-01-006, ZonMw 100.001.001; NWO-175.010.2003.005), an unrestricted grant from the Department of Justice, and by the participating universities.

DECLARATION OF INTEREST

None.

REFERENCES

Achenbach, T. M. (1991*a*). Manual for the Child Behavior Checklist/4-18 and 1991 profile. University of Vermont: Burlington, VT.

Achenbach, T. M. (1991*b*). Manual for the Youth Self-Report and 1991 profile. University of Vermont: Burlington, VT.

Achenbach, T. M., McConaughy, S. H. & Howell, C. T. (1987). Child/adolescent behavioral and emotional problems: implications of cross-informant correlations for situational specificity. *Psychological Bulletin* **101**, 213–232.

Benjamin, J., Ebstein, R. P. & Belmaker, R. H. (2002). Personality genetics, 2002. *Israel Journal of Psychiatry and Related Sciences* **39**, 271–279.

Bijl, R. V., Ravelli, A. & van Zessen, G. (1998). Prevalence of psychiatric disorder in the general population: results of The Netherlands Mental Health Survey and Incidence Study (NEMESIS). *Social Psychiatry and Psychiatric Epidemiology* **33**, 587–595.

Bird, H. R., Gould, M. S. & Staghezza, B. (1992). Aggregating data from multiple informants in child psychiatry epidemiological research. *Journal of the American Academy of Child and Adolescent Psychiatry* **31**, 78–85.

Buecking, A., Rothen, S., Ferrero, F. & Preisig, M. (2004). Inter-informant agreement on anxiety disorders: family history versus direct interview information. Presented at the 12th symposium of the AEP, section Epidemiology and Social Psychiatry, 23–26 June, Mannheim, Germany.

Caspi, A., Taylor, A., Smart, M., Jackson, J., Tagami, S. & Moffitt, T. E. (2001). Can women provide reliable information about their children's fathers? Cross-informant agreement about men's lifetime antisocial behaviour. *Journal of Child Psychology and Psychiatry* **42**, 915–920.

Clark, L. A., Watson, D. & Mineka, S. (1994). Temperament, personality, and the mood and anxiety disorders. *Journal of Abnormal Psychology* **103**, 103–116.

Costa, P. T. Jr. & Widiger, T. A. (2002). *Personality Disorders and the Five-Factor Model of Personality*. APA: Washington, DC.

de Winter, A. F., Oldehinkel, A. J., Veenstra, R., Brunnekreef, J. A., Verhulst, F. C. & Ormel, J. (2005). Evaluation of non-response bias in mental health determinants and outcomes in a large sample of pre-adolescents. *European Journal of Epidemiology* **20**, 173–181.

Eaves, L., Heath, A., Martin, N., Maes, H., Neale, M., Kendler, K. S., Kirk, K. & Corey, L. (1999). Comparing the biological and cultural inheritance of personality and social attitudes in the Virginia 30000 study of twins and their relatives. *Twin Research* **2**, 62–80.

Ellis, L. K., Rothbart, M. K. & Posner, M. I. (2004). Individual differences in executive attention predict self-regulation and adolescent psychosocial behaviors. *Annals of the New York Academy of Sciences* **1021**, 337–340.

Feingold, A. (1994). Gender differences in personality: a meta-analysis. *Psychological Bulletin* **116**, 429–456.

Fowles, D. C. (1993). Biological variables in psychopathology: a psychobiological perspective. In *Comprehensive Handbook of Psychopathology* (ed. P. B. Sutker and H. E. Adams), pp. 57–82. Plenum Press: New York.

Hartman, C. A. (2000). Dutch translation of the Early Adolescent Temperament Questionnaire. Internal report, Department of Psychiatry, University of Groningen, The Netherlands.

Kendler, K. S., Prescott, C. A., Myers, J. & Neale, M. C. (2003). The structure of genetic and environmental risk factors for common psychiatric and substance use disorders in men and women. *Archives of General Psychiatry* **60**, 929–937.

Kraemer, H. C., Measelle, J. R., Ablow, J. C., Essex, M. J., Boyce, W. T. & Kupfer, D. J. (2003). A new approach to integrating data from multiple informants in psychiatric assessment and research: mixing and matching contexts and perspectives. *American Journal of Psychiatry* **160**, 1566–1577.

Krueger, R. F. (1999). The structure of common mental disorders. *Archives of General Psychiatry* **56**, 921–926.

Krueger, R. F. & Tackett, J. L. (2003). Personality and psychopathology: working toward the bigger picture. *Journal of Personality Disorders* **111**, 411–424.

Lemery, K. S., Essex, M. J. & Smider, N. A. (2002). Revealing the relation between temperament and behavior problem symptoms by eliminating measurement confounding: expert ratings and factor analyses. *Child Development* **73**, 867–882.

McCrae, R. R., Costa Jr., P. T., Ostendorf, F., Angleitner, A., Hrebickova, M., Avia, M. D., Sanz, J., Sanchez-Bernardos, M. L., Kusdil, M. E., Woodfield, R., Saunders, P. R. & Smith, P. B. (2000). Nature over nurture: temperament, personality, and life span development. *Journal of Personality and Social Psychology* **78**, 173–186.

McGuffin, P., Owen, M. J. & Gottesman, I. I. (eds) (2002). *Psychiatric Genetics and Genomics*. Oxford University Press: London.

Neeleman, J., Bijl, R. & Ormel, J. (2004). Neuroticism, a central link between somatic and psychiatric morbidity: path analysis of prospective data. *Psychological Medicine* **34**, 521–531.

Oldehinkel, A. J., Hartman, C. A., De Winter, A. F., Veenstra, R. & Ormel, J. (2004). Temperament profiles associated with internalizing and externalizing problems in preadolescence. *Development and Psychopathology* **16**, 421–440.

Ormel, J. & Schaufeli, W. B. (1991). Stability and change in psychological distress and their relationship with self-esteem and locus of control: a dynamic equilibrium model. *Journal of Personality and Social Psychology* **60**, 288–299.

Piacentini, J. C., Cohen, P. & Cohen, J. (1992). Combining discrepant diagnostic information from multiple sources – are complex algorithms better than simple ones? *Journal of Abnormal Child Psychology* **20**, 51–63.

Plomin, R. (1994). *Genetics and Experience. The Interplay Between Nature and Nurture*. Sage Publications: Thousand Oaks, CA.

Plomin, R., DeFries, J. C., McClearn, J. & McGuffin, P. (2001). *Behavior Genetics*. Freeman: New York.

Putnam, S. P., Ellis, L. K. & Rothbart, M. K. (2001). The structure of temperament from infancy through adolescence. In *Advances/Proceedings in Research on Temperament* (ed. A. Eliasz and A. Angleitner), pp. 165–182. Pabst Scientist Publisher: Miami, FL.

Renouf, A. G. & Kovacs, M. (1994). Concordance between mothers' reports and children's self-reports of depressive symptoms: a longitudinal study. *Journal of the American Academy of Child and Adolescent Psychiatry* **33**, 208–216.

Rothbart, M. K., Ahadi, S. A. & Evans, D. E. (2000). Temperament and personality: origins and outcomes. *Journal of Personality and Social Psychology* **78**, 122–135.

Rothbart, M. K. & Bates, J. E. (1998). Temperament. In *Handbook of Child Psychology: Vol. 3. Social, Emotional, and Personality Development* (ed. W. Damon), pp. 105–176. Wiley: New York.

Rutter, M. (1987). Temperament, personality and personality disorder. *British Journal of Psychiatry* **150**, 443–458.

Rutter, M., Dunn, J., Plomin, R., Simonoff, E., Pickles, A., Maughan, B., Ormel, J., Meyer, J. & Eaves, L. (1997). Integrating nature and nurture: implications of person-environment correlations and interactions for developmental psychopathology. *Development and Psychopathology* **9**, 335–364.

Rutter, M. & Silberg, J. (2002). Gene-environment interplay in relation to emotional and behavioral disturbance. *Annual Review of Psychology* **53**, 463–490.

Shiner, R. & Caspi, A. (2003). Personality differences in childhood and adolescence: measurement, development, and consequences. *Journal of Child Psychology and Psychiatry* **44**, 2–32.

StataCorp (2003). Stata Statistical Software: Release 8.0. Stata Corporation: College Station, TX.

Verhulst, F. C., van der Ende, J., Ferdinand, R. F. & Kasius, M. C. (1997). The prevalence of DSM-III-R diagnoses in a national sample of Dutch adolescents. *Archives of General Psychiatry* **54**, 329–336.

Vollebergh, W. A., Iedema, J., Bijl, R. V., de Graaf, R., Smit, F. & Ormel, J. (2001). The structure and stability of common mental disorders: the NEMESIS study. *Archives of General Psychiatry* **58**, 597–603.

Widiger, T. A., Verheul, R. & van den Brink, W. (1999). Personality and psychopathology. In *Handbook of Personality: Theory and Research* (ed. L. A. Pervin and O. P. John), pp. 347–366. Guilford: New York.