Predictors and Pathways From Infancy to Symptoms of Anxiety and Depression in Early Adolescence

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Data from a prospective 11-year longitudinal survey were used to identify early predictors and pathways to symptoms of anxiety and depression at 12–13 years of age, and to examine whether there were unique predictors of anxious versus depressive symptoms. Structural equation modeling was used to explore longitudinal relations between contextual (maternal distress, family adversities, and social support) and temperamental (shyness and emotionality) risk factors in their prediction of informant-consistent symptoms of anxiety and depression. The results show that early risk factors can explain 38% of the variance in boys' covarying symptoms of anxiety and depression in early adolescence, and 25% of variance in girls' covarying symptoms. Two main pathways were identified. One pathway was through temperament, as nearly all risk factors were partly mediated through child emotionality in midchildhood. Another pathway was through early contextual risk factors, with all direct and indirect contextual impact from before 5 years of age. Family adversity uniquely predicted depressive symptoms. These findings underscore the persisting impact of contextual predictors in families with children less than 5 years of age. The importance of early interventions to prevent adolescent internalizing problems is stressed.

Keywords: anxiety, depression, maternal distress, family adversities, social support

A major theme in studies of development is the question of lasting effects of early experiences on child psychology over and above changes in risk factors (O'Connor, 2006). Identification of early predictors with persisting effects on symptoms of anxiety and depression is of practical significance for early intervention and prevention efforts (Kovacs & Devlin, 1998). Results from longitudinal studies have indicated that comorbid cases of anxiety and depression are more severe and persistent than single ones, with more serious risk history (Costello, Mustillo, Erkanli, Keeler, & Angold, 2003; Merikangas et al., 2003; Moffitt, Harrington, et al., 2007). It is emphasized that antecedent risk factors of this group should be targeted as early as possible for preventive purposes (Moffitt, Caspi, et al., 2007), indicating that it might be more important to predict overlapping symptoms of anxiety and depression than symptoms of either alone. We need more knowledge about early predictors and the mechanisms through which predictors contribute to the development of psychopathology (Rothbart & Bates, 2006; Rutter, 2003).

Internalizing problems in children, such as anxiety and depression, are found to be hard to identify and treat (Costello & Angold, 2006). The boundaries between different child disorders, or between normality and disorder, are generally characterized by uncertainty (Fergusson, Horwood, & Boden, 2006; Rutter, 2003). A well-supported finding is that subthreshold levels of emotional symptoms in early adolescence put youths at heightened risk for later psychiatric disorders (Clark, Rodgers, Caldwell, Power, & Stansfeld, 2007; Fergusson et al., 2006; Jaffee et al., 2002). Hence, it is crucial to identify pathways from earlier ages to symptoms of anxiety and depression in early adolescence.

The specific developmental period during which children and their families are exposed to risk factors, and the complex interplay between these factors, may affect the development of internalizing problems (Cicchetti & Toth, 1998; Essex, Klein, Cho, & Kraemer, 2003). Juvenile onset depression is found to be associated with a higher frequency of early childhood risk factors than adult onset depression (Jaffee et al., 2002). By continuously measuring risk factors across time, we may identify sensitive periods in which the children might be negatively affected by exposure to specific risk factors.

In the current study, we measured child temperamental shyness and emotionality, family adversities, lack of social support, and maternal distress continuously through childhood. These temperamental and contextual risk factors are well-documented risk factors for internalizing problems and disorders in adolescence (Essau, 2004; Essex et al., 2003; Gaylord, Kitzmann, & Lockwood, 2003; Ge, Natsuaki, & Conger, 2006; Hammen & Brennan, 2003; Jaffee et al., 2002; Leech, Larkby, Day, & Day, 2006; Masi et al., 2003). Most studies that have investigated mediating relations between risk factors of internalizing problems have either (a) used cross-sectional data or longitudinal data with a relatively

short age-span, (b) had small samples, or (c) used statistical methods that do not account for measurement errors (Appleyard, Egeland, Van Dulmen, & Sroufe, 2005; Essex et al., 2006; Gaylord et al., 2003; Leech et al., 2006; Luby, Belden, & Spitznagel, 2006; MacPhee & Andrews, 2006; Masi et al., 2003). Several authors have therefore emphasized the need for examining the long-term longitudinal relations between risk factors, and especially factors representing both nature and nurture (Rutter, Moffitt, & Caspi, 2006; Zahn-Waxler, Crick, Shirtcliff, & Wood, 2006). It is stressed that longitudinal research measuring stressors and potential mediators at each of several time points are needed to test models of mediating mechanisms that lead to psychopathology (Grant et al., 2003).

On the basis of previous research and models of risk factors and psychopathology, several potential pathways to internalizing problems may be hypothesized (Cicchetti & Toth, 1998; O'Connor, 2006; Rothbart & Bates, 2006). We would expect that both family adversities and lack of social support increase the level of maternal distress, which again predict the child's mental health (Essex et al., 2006; Thompson, Flood, & Goodvin, 2006). Maternal distress can also affect family adversities (Essex et al., 2006; Rutter, Moffitt, & Caspi, 2006). Family adversities are found to be easier to handle if families have good social support (Rodgers & Rose, 2002; Thompson et al., 2006). However, lack of social support may contribute to more family adversity and thereby affect the child's symptom level (Leech et al., 2006). Increased levels of maternal distress can make the mothers less responsive and less emotionally available for their children and thereby increase their children's levels of negative emotionality or shyness, which again make them more vulnerable for developing internalizing problems (Cicchetti & Toth, 1998; Cummings & Davies, 1994). On the other side, we might also expect that child emotionality may contribute to increased levels of maternal distress, insofar as highly emotional children tend to acquire coercive tendencies more often than others (Rothbart & Bates, 1998).

Recent research in both the study of resilience and the study of risk pathways has had a strong focus on the biology of mental health. Several researchers have noted that there is a need to promote studies on contextual and relational factors and how they mediate the environment with regard to child development (Luthar & Brown, 2007; Zucker, 2006). To inform the studies of risk and resilient pathways, it is important to focus on what makes children vulnerable, whether there are sensitive periods for contextual impact on child mental health, and what are the mediating relations between different predictors.

However, few studies have tested the longitudinal relations between temperamental and contextual risk factors and their effect on internalizing symptoms. How to measure or test such complex interplay is a challenge when investigating mediating, reciprocal, and dynamic relations between several variables. A variable that serves as a stressor in one relation might turn into a mediator in another relation. An exploratory approach to the relations between the specific variables would—when put together in one model—examine all relations across development at the same time. Rutter, Kim-Cohen, and Maughan (2006) have pointed out that there is a need for identifying mediating processes, insofar as we know too little to develop an all-encompassing model of continuities in childhood and onwards. We are unaware of prior attempts to simultaneously explore the longitudinal relations between the tem-

peramental and environmental factors mentioned above and their direct and indirect pathways to adolescent internalizing problems.

Symptoms of anxiety and depression are often classified as internalizing problems in childhood (Campbell, 1995; Mathiesen & Sanson, 2000). Identification of differential predictors of internalizing symptoms may help us to clarify whether there are different paths of risk to common versus unique symptoms of anxiety and depression. Although some clinical studies have found that classification of children into groups of depression and anxiety was predicted by different factors (Moffitt, Caspi, et al., 2007; Phillips, Hammen, Brennan, Najman, & Bor, 2005), population-based studies give inconclusive evidence for selective pathways in childhood (Essex et al., 2006; Leech et al., 2006; Mesman & Koot, 2000). By using structural equation modeling to conduct a confirmatory factor analysis, we have the possibility to differentiate between the common and unique variance in parent- and child-reported symptoms of anxiety and depression.

The aim of this investigation was to identify early predictors and their pathways from 1.5 years of age and onwards to covarying symptoms of anxiety and depression (core internalizing problems) at 12–13 years of age. Second, we wanted to examine whether there are unique predictors of anxious versus depressive symptoms in early adolescence.

Method

Sample and Procedure

In this study, we used data from the Tracking Opportunities and Problems Study—a prospective population-based longitudinal study focusing on the mental health of children and their families. More than 95% of Norwegian families with children attend the public health services, which include 8–12 health screenings during the first 4 years of the child's life. All families from 19 geographic health care areas that visited a child health clinic in 1993 for the scheduled 18-month (Time 1 [t1]) vaccination visit were invited to complete a questionnaire. Of the 1,081 eligible families, 939 (87%) participated at t1. The parents who participated at t1 received a similar questionnaire when the children were 2.5 years of age (Time 2 [t2]: $n = 781$), 4.5 years of age (Time 3 [t3]: $n = 750$), 8–9 years of age (Time 4 [t4]: $n = 535$) and 12–13 years of age (Time 5 [t5]: $n = 613$). The adolescents ($n = 566$) filled in their own questionnaires at t5. The questionnaires were administered by the health care workers at t1–t3. At subsequent waves, questionnaires for parents (t4) and parents and children (t5) were sent by mail. The parents could choose whether the mother or the father should complete the questionnaire at t1–t4, and at t5 the mothers were encouraged to answer (besides the adolescents). Less than 25 fathers participated at each wave, thus, the paternal questionnaires have not been analyzed in the current article.

The 19 health care areas were chosen on the basis of being overall representative of the diversity of social environments in Norway: 28% of the families lived in large cities, 55% lived in densely populated areas, and 17% lived in rural areas. Maternal age ranged from 19 to 46 years at t1, with a mean of 30 years ($SD = 4.7$). The sample was predominantly ethnic Norwegian families from the middle class. This is largely representative for ethnicity in Norway in general, as only 2.3% of the population in 1993 came from non-Western societies (Statistics Norway, 2006).

Data from the child health clinics showed that nonrespondents at t1 did not differ from respondents with respect to maternal age, education, employment status, number of children, or marital status. The sample at t5 included 46% boys; of the mothers, 23.6% had 11 years schooling or less, 46.8% had between 12 and 15 years of schooling, and 27.1% had a college or university education of 4 years or more. Of the mothers, 53.5% worked full time outside the home, and 75.1% reported that they were doing well economically. Furthermore, 80.8% of the mothers lived with a spouse/partner. Taking all waves into account, there were few questions that respondents refrained from answering, and the items showed a satisfactory level of variance. Analyses of sample attrition from t1 to t5 showed that the remaining families were not significantly different from the dropout sample in terms of maternal distress, family adversities, social support, and child temperament. In addition, we ran Cox proportional hazard regression to account for the dropout at different waves. In the Cox regression, we get an estimate of the hazard ratio, which is the effect of an explanatory variable on the risk of, in this case, dropout. Maternal education, maternal age, child gender, urban/rural status, and each predictor in the main model at t1 (maternal distress, family adversities, social support, child emotionality, and child shyness) were entered in the survival analysis, first one by one and then all predictors together. The results from the Cox regression show that only years of education influenced dropout throughout the study (hazard ratio = 0.88; 95% confidence interval = 0.83–0.93), indicating that the mothers had a lower risk for dropout the more years of education they had.

We carried out statistical modeling using missing data estimation techniques using the multinomial logistic regression (MLR) procedure in Mplus (Muthén & Muthén, 2006), and we applied full information maximum likelihood (FIML), which takes advantage of participants with partial data. Assuming the data are missing at random (MAR) conditional on covariates included in the model, FIML estimation increases power and decreases potential attrition bias by adjusting for bias related to model variables. This procedure estimates the parameters in the model directly, and the uncertainty of missingness is included in the model chi-square. Even if MAR is not literally true, FIML is still the recommended approach (Schafer & Graham, 2002) because it outperforms common alternative approaches, such as complete case analysis or single imputation strategies.

Measures

Both parent- and child-reported *symptoms of depression* were measured with the Short Mood and Feeling Questionnaire (SMFQ; Angold et al., 1995). The SMFQ is a uni-dimensional child- and parent-reported scale designed for use in epidemiological studies of depression in children and adolescents, consisting of 13 items (e.g., "I feel miserable or unhappy"). Two items about restlessness and poor concentration were omitted because of space limitations in the questionnaire. The remaining 11 items addressed the affective and cognitive components of depression found to be the best predictors of depressive status (Angold et al., 1995). The child and the parent are asked to rate depressive symptoms on a 3-point scale (i.e., *not true*, *sometimes true*, and *true*). The SMFQ consists of items from the Mood and Feeling Questionnaire, which is translated, back-translated, and validated in another Norwegian sample

of adolescents 13–14 years of age (Sund, Larsson, & Wichstrøm, 2001). The SMFQ has reported high levels of sensitivity and specificity (Angold et al., 1995). Cronbach's alphas were .83 for the parent report, .86 for the child report, and .88 for a combination of the parent and child reports. We combined the parent- and child-reported items into indexes using mean scores. The two depression indices were used as indicators of a latent depression factor in a confirmatory factor analysis.

Symptoms of anxiety were measured with the Generalized Anxiety Disorder (GAD) scale—a subscale of the Coolidge Personality and Neuropsychological Inventory for Children (CPNI; Coolidge, Thede, Stewart, & Segal, 2002). The GAD scale consists of 12 parent-reported items derived from criteria from three different anxiety disorders (generalized anxiety disorder, separation anxiety disorder, and social phobia) in the *Diagnostic and Statistical Manual of Mental Disorders* (4th ed.; American Psychiatric Association, 1994). Because of lack of a suitable anxiety scale with both parent and child reports, we constructed a child-report questionnaire based on the CPNI/GAD scale parent report. The child-reported items were constructed by changing the wording of the parent-reported items (e.g., "My child worries too much" into "I worry too much"). The child and the parent were asked to rate anxious symptoms occurring within the last 2 months on a 4-point scale (i.e., *not true*, *seldom true*, *sometimes true*, and *always true*). The CPNI has been translated and back-translated in another Norwegian study (Kristensen & Torgersen, 2007). Cronbach's alpha was .78 for the maternal report, .86 for the child report, and .70 for a combination of the parent and child reports. The 12 anxiety items for respectively parent and child were combined into two mean indices and, by conducting a confirmatory factor analysis, used as indicators of a latent anxiety factor.

Core internalizing problems were operationalized as the underlying common factor in anxiety and depression. Thus, a latent factor accounting for the informant-consistent covariance between anxiety and depression was modeled to represent common internalizing problems. This approach implies separation of the unique and common aspects of anxiety and depression, and it enables the potential prediction of each of these three sources of variance.

Shyness and emotionality were assessed by the Emotionality, Activity, and Sociability Temperament Survey (EAS; Buss & Plomin, 1984), in which each temperament trait is measured by five items rated on a 5-point scale. Shyness refers to the tendency to be inhibited and awkward in new social situations, and emotionality refers to the tendency to become aroused easily and intensely. Each temperament trait is measured by five items rated on a 5-point scale ranging from 1 (*not typical*) to 5 (*very typical*). The items for each dimension are combined into a mean index. Because of ambiguity in the translation, we deleted one item from each of the dimensions. The reliability and validity of the EAS have earlier been well established with this data set (Mathiesen & Tambs, 1999). Cronbach's alphas for shyness were .75 (t1), .75 (t2), .77 (t3), and .77 (t4); and Cronbach's alphas for emotionality were .66 (t1), .68 (t2), .71 (t3), and .67 (t4).

The *family adversity* index was based on questions referring to experiences on enduring problems during the last 12 months in the following areas: housing, employment, economy, their own and partner's physical health, use of alcohol, relationship to partner, childcare arrangements, children's illnesses, and child rearing (Mathiesen & Sanson, 2000). The answers were dichotomized,

and a mean composite measure was formed by the 10 items. Cronbach's alphas for family adversities at t1–t4 were .63, .64, .69, and .62.

Maternal distress (symptoms of anxiety and depression) was measured by the 25-item version of the Hopkins Symptom Check List (Hesbacher, Rickels, Morris, Newman, & Rosenfeld, 1980). The reliability of the Hopkins Symptom Check List has earlier been well established in a Norwegian sample (Tambs & Moum, 1993). Two items—"thoughts of ending your life" and "loss of sexual interest or pleasure"—were excluded from the Norwegian questionnaire because some participants in the pilot-project perceived them as offensive. We used the overall mean of the 23 items, each rated on a 4-point scale. Cronbach's alphas for maternal distress at t1–t4 were .90, .89, .90, and .92.

The *social support* index was formed by taking the mean of nine items (each rated on a 5-point scale). The index measures the amount of social support that the mothers receive from close family, friends, and their partners by using three questions pertaining to each of the three groups to capture the following: (a) closeness and contact, (b) respect and responsibility, and (c) feeling of belonging (Dalgard, Bjørk, & Tambs, 1995; Mathiesen, Tambs, & Dalgard, 1999). Cronbach's alphas for social support at t1–t4 were .74, .78, .77, and .76.

Girls were coded as one, and boys were coded as zero. Thus, positive effects indicate that girls have higher levels in the predictor or outcome variable than boys, whereas negative effects indicate that boys have higher levels on the outcome. In the modeling analyses, gender was included as a predictor to control for gender differences and to identify possible mediators of such differences.

Statistical Analysis

We conducted structural equation modeling using Mplus Version 4.2 (Muthén & Muthén, 2006). A robust maximum likelihood estimation procedure was used to perform the analyses because of the lack of multivariate normality. We handled markedly negative skew in the distribution of the symptom scores by log transforming the data using SPSS Version 14. We standardized the measures, allowing for comparison between the anxiety and depression scales.

An exploratory approach to model fitting was adapted (Jöreskog & Sörbom, 1993; Kendler, Gardner, & Prescott, 2002, 2006). In the basic model, the outcome part comprised a higher order factor model, including four observed variables (child- and parent-reported indices of anxiety and depression), two first-order factors (anxiety and depression), and one second-order factor (core internalizing problems). The predictor and mediator part of the model included all five predictor variables observed at four time points. The basic model allowed cross-time, within-variable paths, representing the stability of the variables. Residual correlations within time points were allowed. Moreover, effects from predictor variables to outcome were allowed for the most proximal time point (t4) upon the general latent factor of internalizing problems (t5).

Next, in a stepwise fashion, we elaborated the model by including residual correlations within time points and regression paths between time points suggested by the modification indices. Relations that were not accounted for by the basic model were indicated according to the expected reduction in chi-square obtained by modeling them. Thus, we allowed significant cross-effects

among the predictors. Only effects that were time logical were included (e.g., from t2 to t3). Moreover, long-term direct effects (from predictors at t1–t3) upon the outcome were allowed. Finally, predictor effects upon the first-order latent outcome factors of anxiety and depression were allowed (i.e., effects on the unique aspects of anxiety and depression, by controlling for the effects mediated through the general factor of internalizing problems). The adapted strategy involved potentially omitting paths from the basic model that were shown to be insignificant as the model was developed. In general, the final model would contain all significant (and theoretically sound) paths and exclude nonsignificant (and time-illogical) paths (see, e.g., Kendler et al., 2002, 2006). The final model would include estimates of stability among predictors, cross-effects among predictors, direct effects from predictors upon outcomes, indirect effects through developmental pathways, and effects common and/or unique to anxiety and depression.

As a final step, we investigated gender-moderating effects. We did this by running a two-group path model, divided by gender. Correlations, intercepts, factor loadings, and regression loadings were initially constrained across gender. The regressions and correlation were freed in the inverse of the stepwise manner previously described—freeing parameters contributing with the highest reduction of chi-square.

Model fit was evaluated with Satorra–Bentler scaled statistic (S-$B\chi$2), the robust comparative fit index (CFI), and the root-mean-square error of approximation (RMSEA). The CFI provides a measure of the fit of a particular model relative to the null model, whereas the RMSEA provides a measure of the model fit relative to the population covariation matrix in which the complexity of the model is taken into account. To conclude that there is a good fit between the hypothesized model and the observed data, a CFI value greater than .95 and a RMSEA value of .06 or less are considered necessary (Hu & Bentler, 1998).

Results

Descriptives

Table 1 shows Cronbach's alphas, means, and standard deviations for the predictors and parent- and child-reported symptoms of anxiety and depression in raw scores. The symptom indices of anxiety and depression correlated .57, parent- and child-reported anxiety correlated .40, and depression reports correlated .44 ($p <$ .01). Correlations between cross-informant standardized symptoms of anxiety and depression and predictors are shown in Table 2. Except shyness at t1 and social support at t4, all predictors were correlated with anxious symptoms at all time points ($p < $ .05). Except shyness at t1 and t2, all predictors were significantly correlated with depressive symptoms ($p < $ .05).

Model Fitting

The base model with all characteristics internally regressed on each other over time and internalizing regressed on t4 characteristics had a S-$B\chi^2$ of 1,500.64 with 241 degrees of freedom (CFI = .748, RMSEA = .073). After the stepwise fitting to the data, the cross-gender model had a S-$B\chi^2$ of 189.91 with 190 degrees of freedom (CFI = 1.000, RMSEA = .000). The difference between the base model and the cross-gender model was substantial and

Table 1
Means and Standard Deviations for Symptoms of Anxiety and Depression and Their Predictors

Predictor/variable	t1		t2		t3		t4		t5	
	M	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*
Predictors										
Shyness	2.16	0.72	2.25	0.70	2.34	0.70	2.36	0.74		
Emotionality	2.42	0.72	2.44	0.71	2.52	0.69	2.20	0.67		
Family adversities	1.21	0.18	1.20	0.18	1.23	0.20	1.21	0.20		
Maternal distress	1.35	0.34	1.30	0.29	1.28	0.29	1.30	0.33		
Social support	4.26	0.60	4.21	0.60	4.12	0.63	4.11	0.62		
Outcome variables										
Anxiety									1.40[a]	0.32
Depression									1.23[a]	0.25
Anxiety parent report									1.26[a]	0.25
Anxiety child report									1.57	0.50
Depression parent report									1.19	0.25
Depression child report									1.27[a]	0.34

Note. t = Time.
[a] Scores were significantly higher for girls than for boys ($p < .01$).

significant ($\Delta\chi^2 = 1,238$, $\Delta df = 51$, $p < .01$). Thereafter, the model was divided by gender to investigate possible moderator effects. From the gender-divided model ($S-B\chi^2 = 496.46$, $df = 457$, CFI = .992, RMSEA = .013) to the final gender-divided model ($S-B\chi^2 = 447.59$, $df = 448$, CFI = 1.000, RMSEA = .000) there were seven cross-time paths and two within-time significant gender differences. The final model with gender specific differences fitted significantly better to the data than the gender-equal model ($\Delta\chi^2 = 49$, $\Delta df = 9$, $p < .01$).

Table 2
Correlations Between Symptoms of Anxiety and Depression and the Predictors

Predictor	Anxiety	Depression
Shyness t1	.05	.08
Emotionality t1	.14[b]	.12[b]
Family adversities t1	.13[b]	.14[b]
Maternal distress t1	.21[b]	.21[b]
Social support t1	−.13[b]	−.21[b]
Shyness t2	.10[a]	.08
Emotionality t2	.10[a]	.14[b]
Family adversities t2	.23[b]	.24[b]
Maternal distress t2	.24[b]	.21[b]
Social support t2	−.19[b]	−.23[b]
Shyness t3	.16[b]	.12[b]
Emotionality t3	.15[b]	.14[b]
Family adversities t3	.13[b]	.19[b]
Maternal distress t3	.18[b]	.19[b]
Social support t3	−.17[b]	−.25[b]
Shyness t4	.19[b]	.13[b]
Emotionality t4	.24[b]	.29[b]
Family adversities t4	.10[a]	.22[b]
Maternal distress t4	.12[a]	.21[b]
Social support t4	−.08	−.16[b]

Note. t = Time.
[a] Correlation is significant at the .05 level (two-tailed). [b] Correlation is significant at the .01 level (two-tailed).

Figure 1 shows the final model divided on gender with the latent factors of anxiety and depression, the second-order factor of core internalizing problems, the effects from the predictors to core internalizing problems at 12–13 years of age, and the unique effect on depression at t5. The estimated correlation between anxiety and depression was .76. Significant effects, factor loadings of the three latent factors, and the amount of explained variance in the indicators and latent factors are shown in Figure 1. Significant effects that were related to predictors with no direct or indirect relation with the outcome variables (including two of the cross-time gender differences), within-time relations between predictors, and residual correlations are not shown. The two significant within-time gender differences showed that maternal distress and emotionality at t4 were significantly related for boys only ($r = .19$, $p < .01$), and social support and shyness at t3 were significantly related for girls only ($r = −.20$, $p = .001$). All the gender-moderated, cross-time paths were significantly stronger for girls than for boys, and three of these paths were significant for girls only (see Figure 1).

Both temperamental factors had indirect effects from the three earlier measures of temperament with direct effects at t4 on adolescent internalizing problems (see Figure 1). Over and above this, shyness at t1 had an indirect effect through social support at t2, shyness at t2 and t3 had an indirect effect on internalizing problems through emotionality at t4, and emotionality at t1 had an indirect effect through shyness at t3. The contextual factors showed quite another pattern, with early direct and indirect effects. Maternal distress at t1 and life strain at t2 had a direct effect on later internalizing problems. Social support at t3 had a direct negative, hence protective, effect on internalizing problems. Several early contextual factors had indirect effects on internalizing problems through emotionality at different time points. There were also several indirect effects from early contextual factors on internalizing problems through social support at t3. The contextual factors at t4 did not have any impact on internalizing problems. Family adversity at t4 was the only predictor with a unique impact on depression. There were no statistically significant unique predictors of anxiety throughout childhood to adolescence.
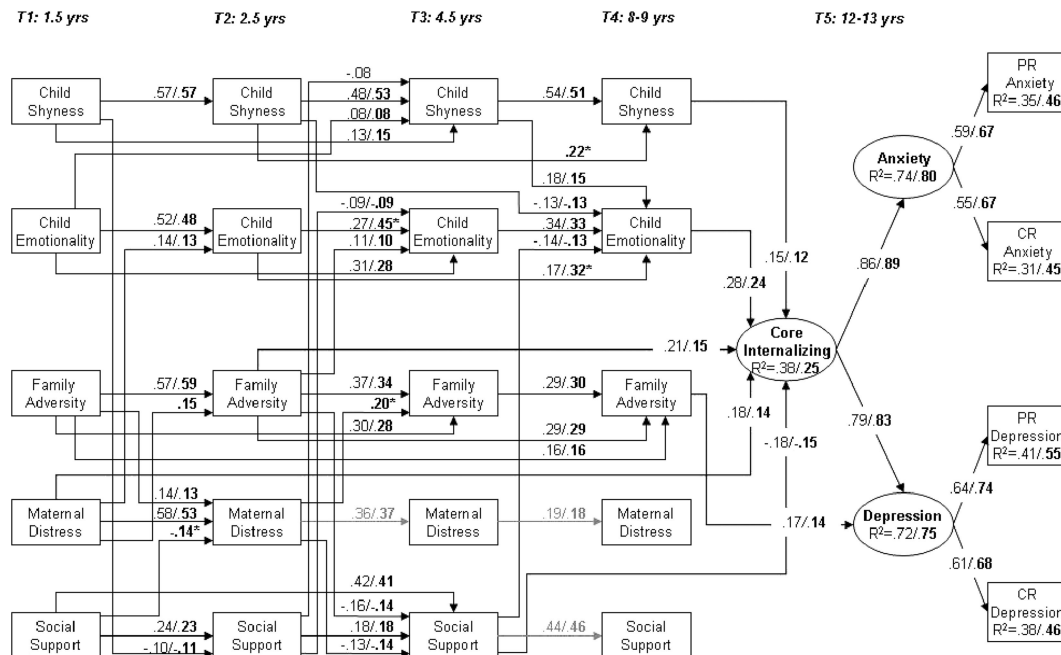
Figure 1. The full gender-divided model of Time 1–Time 4 predictors and latent factors of core internalizing, anxiety, and depression at Time 5 (PR = parent reported; CR = child reported). Only significant effects (*p* < .05), shown as standardized coefficients (betas), are noted on the paths. Coefficients for boys are shown in normal font, and coefficients for girls are shown in bold font. Significant gender-moderated paths are noted with an asterisk. T = Time.

Discussion

This study has examined the longitudinal relations between child temperamental shyness and emotionality, maternal distress, family adversities, and social support from early childhood and onwards, and their direct and indirect impact on symptoms of anxiety and depression in early adolescence. Unique predictors of symptoms of anxiety versus depression were also examined. We found direct impact of contextual predictors on adolescent problems already from the 2nd year of life. There were two main pathways to core internalizing problems (covarying symptoms of anxiety and depression). The first pathway was through temperament, as all risk factors were partly mediated by child emotionality in midchildhood. The second main pathway was through early contextual risk factors, with all direct and indirect contextual impact from before 5 years of age. A substantial part of variance in internalizing symptoms in boys (38%) and girls (25%) was explained by the predictors, and the girls had more and stronger relations between the predictors compared with boys. The results show a direct effect from family adversity in midchildhood to later symptoms of depression but no unique predictors of anxious symptoms.

The significance of early persisting effects on adolescent internalizing problems is consistent with previous research (Essex et al., 2006; Moffitt, Caspi, et al., 2007). However, this study differs from other studies regarding the continuous measures of risk factors across childhood, the longitudinal relations between risk factors, and predicting an outcome measure that represents the shared variance of parent- and child-reported symptoms of anxiety and depression. Five contributions are notable.

First, our results support and extend findings on the early impact of contextual factors on internalizing problems (Essex et al., 2006; Hammen & Brennan, 2003), indicating that children are more vulnerable to maternal distress, family adversities, and lack of social support in the period of early childhood (compared with midchildhood). Evidently, early childhood stands out as a vulnerable developmental period. Effect of maternal distress from early childhood is a well-documented finding (Cicchetti & Toth, 1998; Essex et al., 2006; Hammen & Brennan, 2003). More interesting is that the impact from 1.5 years of age is significant even when accounting for maternal symptoms and the other predictors later in childhood. Infancy is often associated with less sleep and more stress for the mother, and it has been argued that this period is an particularly vulnerable period for experiencing inadequate parent–child communication (Cicchetti & Toth, 1998). Studies on mother–child interaction and frontal brain asymmetry in children of depressed mothers indicate increased risk for developing child emotional problems (Dawson et al., 1999; Forbes et al., 2006; Henderson, Fox, & Rubin, 2001). Earlier onset maternal distress may also represent a more heritable condition (O'Connor, Heron, Golding, & Glover, 2003; Rutter, 2003), thus contributing to the explanation of why early maternal distress was more strongly related to internalizing at t5 than later measures of distress.

Another contextual factor with early impact is family adversities at 2.5 years of age. Developmentally, the child is acquiring increased mastery of the object world and increased autonomy while he or she still needs surveillance most of the time (Sroufe & Rutter, 1984). In Norway, this is a period in the child's life that is associated with transitions in the family setting—that is, mothers

have returned to work, and most children have started in some form of daycare. Thus, this may be a vulnerable period for exposure to adversities within the family. Caspi et al.'s (2003) findings on the serotonin transporter gene may also indicate that the early effects of adversity on internalizing symptoms are contingent on genetic susceptibility. As this Norwegian sample is relatively high functioning, these results support Essex et al.'s (2006) suggestion that at-risk children from higher socioeconomically conditions can be identified from infancy.

Second, both temperament traits showed relatively high stability across childhood, with predictive impact on adolescent internalizing problems from the most proximal time point. This finding is consistent with earlier findings on the relation between internalizing and temperamental shyness and negative emotionality (Gilliom & Shaw, 2004; Masi et al., 2003; Rothbart & Bates, 2006). Interestingly, emotionality seems to partly mediate the other predictors but is not mediated by any of the contextual factors, and the influence of child shyness on internalizing problems is only mediated by one of the contextual factors: social support. This finding supports Rothbart and Bates's (2006) suggestion of a process in which temperament might heighten the response from a contextual factor. Insofar as elevated levels of shyness and emotionality represent a biological vulnerability toward later internalizing problems, it is important to be aware of other risk or protective factors that can influence the level of these traits.

Third, our findings address the request from researchers in the field of developmental psychopathology about exploring different pathways to the same outcome (equifinality; Cicchetti & Toth, 1998; Kazdin, Kraemer, Kessler, Kupfer, & Offord, 1997). The results indicate that several childhood pathways contribute to internalizing problems in early adolescence, and that temperamental and contextual risk factors seem to behave differently. The contextual risk factors acted as both stressors and mediators for one another, hence, demonstrating how reciprocal and dynamic the longitudinal relationship between risk factors is in predicting later internalizing problems. Leve, Kim, and Pears (2005) found that maternal distress and child shyness at 5 years of age predicted internalizing problems from 5 to 17 years of age, and that maternal distress also predicted increasing internalizing trajectories for girls. They suggested that these two factors either had very powerful long-term effects or that they contributed to a series of environmental events that mediated early characteristics and later problems. In addition to supporting and extending their results by incorporating earlier years, our results indicate that maternal distress seems to trigger a cascade of other adverse contextual factors. This is consistent with earlier research finding that depressive symptoms are often stress generating and predict more chronic stress (Cole, Nolen-Hoeksema, Girgus, & Paul, 2006). Our findings on the mediating impact of social support is consistent with earlier findings that low social support mediates the relation between both maternal distress and family adversities on internalizing symptoms in preadolescence (Cummings & Davies, 1994). In a study of early predictors of anxiety and depression, Essex et al. (2003) showed that already from preschool years and onwards, girls had increased risk for developing symptoms of anxiety and depression as a result of exposure to maternal depression, family conflict, and divorce. In keeping with this study, we found more environmental effects among girls' internalizing paths.

Fourth, family adversity experienced when the children were 8–9 years of age predicted symptoms of depression without affecting anxious symptoms. This result supports Jaffee et al.'s (2002) finding that childhood risk factors (e.g., parental psychopathology and family instability) of juvenile-onset depression remained significant after controlling for comorbidity with anxiety disorders. Eaves, Silberg, and Erkanli (2003) found in their analysis of epigenetic pathways to adolescent depression that genes that are specific to depression, and not shared with risk of earlier anxiety, have a large additional effect on sensitivity to environmental stress. They also found that genetic vulnerability to anxiety led to an increased sensitivity to life events that then resulted in an increased risk for depressive symptoms. To the extent that maternal distress represents a genetic liability in children, then our study supports Eaves et al.'s findings from a developmental behavioral perspective.

Fifth, the childhood risk factors explained 38% of variance in internalizing problems in boys in early adolescence, and 25% of the variance in girls. Knowing that this is a population-based sample, with a limited number of risk factors compared with the "real" world and probably less variance to explain compared with at-risk samples, the predictors in this study explained a substantial part of the variation in the level of internalizing symptoms. Anxious and depressive symptoms were highly correlated, and most of the variance in both measures was explained by the core internalizing factor, suggesting that symptoms of anxiety and depression could be treated as one phenomenon in early adolescence. However, the finding that one risk factor predicts depressive symptoms uniquely indicates that we should not discard the usefulness of separating the symptoms. It is somewhat surprising that we did not find any unique predictors of anxiety because anxious symptoms are expected to show up earlier than depressive symptoms. It may be that earlier measures of anxious symptoms are needed for differential prediction. In addition, the anxiety measure in this study is mainly focused on anxiety-related symptoms (as in overanxious and generalized anxiety disorders) as opposed to fear-related symptoms (as in panic or phobia; Varner, Wickramaratne, & Weissman, 2008). Anxiety-related symptoms are found to be more overlapping with depressive symptoms (Kendler, Neale, Kessler, Heath, & Eaves, 1992); thus, our measure of anxiety may have contributed to the lack of unique predictors of anxiety symptoms.

The results presented here should be interpreted with respect to the following limitations. First, this study utilized a predominantly nonclinical sample, in which most families had ethnic majority background and were—at the outset—well functioning. In this population, it would be expected that the predictors generally show lower levels of risk compared with at-risk samples. Thus, the results may be underestimating the effects of the risk factors on internalizing problems. There is a possibility of nonignorable nonresponse (e.g., if depressed youths were unwilling to participate because of the depression), implying that the MAR assumption would not hold. However, we believe our analyses support the MAR assumption as far as possible. Further research is needed to see whether our findings transcend local conditions and ethnic variation. The predictor index of family adversities had only moderate alphas, ranging from .62 to .69. This index is formed by different stressful life events and would not be expected to demonstrate very high levels of internal consistency. The implication

of such an alpha on the analysis may, however, be that the relation between the variables is underestimated. Several of the predictor measures were relatively short and more general, which may limit the area of generalizations that can be made. The use of shorter and more general symptom rating scales may contribute to the difficulty of identifying unique predictors, as anxious and depressive symptoms in early adolescence are found to be hard to differentiate for parents and the youths themselves (Treiber & Mabe, 1987). Another limitation was the reliance in the study on maternally reported factors, and the biases this may have introduced. There is a risk of inflated associations between variables because of shared method variance. One of the main objections against maternal ratings is that both perspective and context may influence the reports. This is partly accounted for by using reports from different informants of the outcome variables. Finally, the overall design of the study did not allow exploration of genetics, but the measures of child temperament and maternal symptoms of anxiety and depression may act as indicators of genetic predispositions.

In closing, the findings in the current study answered two related questions with substantial practical implications. First, there were lasting effects of early experiences on adolescent internalizing problems over and above the subsequent risk factors. Second, specific detrimental pathways from childhood were also identified through the risk factors. The vulnerability of children with high levels of temperamental shyness and especially emotionality is underscored. An important contribution of this study is that a few theoretically important early childhood risk factors can explain a substantial amount of the variance in internalizing problems in early adolescence, and (maybe most important) maternal distress at 1.5 years of age predicts heightened levels of internalizing symptoms in young adolescents even when later levels of maternal distress were accounted for. If most of a child's development of psychopathology is determined in early childhood, then we should target our intervention effort mainly to families with children in their first years of life. If, however, the potential for impact from risk and protective factors are relatively similar at any point during the childhood years, early intervention might not be that crucial. The results presented here emphasize the importance of early interventions to prevent later development of symptoms of anxiety and depression in youths. For early detection of at-risk groups, pre- and postpartum screening of maternal distress should be part of a general follow-up of pregnant women and mothers of infants. Considering the potential negative impact of early maternal distress on family life and child outcomes, such an investment could have important public health implications.

References

American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.

Angold, A., Costello, E. J., Messer, S. C., Pickles, A., Winder, F., & Silver, D. (1995). Development of a short questionnaire for use in epidemiological studies of depression in children and adolescents. *International Journal of Methods in Psychiatric Research, 5,* 237–249.

Appleyard, K., Egeland, B., Van Dulmen, M. H. M., & Sroufe, L. A. (2005). When more is not better: The role of cumulative risk in child behavior outcomes. *Journal of Child Psychology and Psychiatry, 46,* 235–245.

Buss, A. H., & Plomin, R. (1984). *Temperament: Early developing personality traits.* Hillsdale, NJ: Erlbaum.

Campbell, S. B. (1995). Behavior problems in pre-school children: A review of recent research. *Journal of Child Psychology and Psychiatry, 36,* 113–149.

Caspi, A., Sugden, K., Moffitt, T. E., Taylor, A., Craig, I. W., Harrington, H., et al. (2003, July 18). Influence of life stress on depression: Moderation by a polymorphism in the 5-HTT gene. *Science, 301,* 386–389.

Cicchetti, D., & Toth, S. L. (1998). The development of depression in children and adolescents. *American Psychologist, 53,* 221–241.

Clark, C., Rodgers, B., Caldwell, T., Power, C., & Stansfeld, S. (2007). Childhood and adulthood psychological ill health as predictors of midlife affective and anxiety disorders—The 1958 British Birth Cohort. *Archives of General Psychiatry, 64,* 668–678.

Cole, D. A., Nolen-Hoeksema, S., Girgus, J., & Paul, G. (2006). Stress exposure and stress generation in child and adolescent depression: A latent trait-state-error approach to longitudinal analyses. *Journal of Abnormal Psychology, 115,* 40–51.

Coolidge, F. L., Thede, L. L., Stewart, S. E., & Segal, D. L. (2002). The Coolidge Personality and Neuropsychological Inventory for Children (CPNI): Preliminary psychometric characteristics. *Behavior Modification, 26,* 550–566.

Costello, E. J., & Angold, A. (2006). Developmental epidemiology. In D. Cicchetti & D. J. Cohen (Eds.), *Developmental psychopathology* (2nd ed., pp. 41–75). Hoboken, NJ: Wiley.

Costello, E. J., Mustillo, S., Erkanli, A., Keeler, G., & Angold, A. (2003). Prevalence and development of psychiatric disorders in childhood and adolescence. *Archives of General Psychiatry, 60,* 837–844.

Cummings, E. M., & Davies, P. T. (1994). Maternal depression and child development. *Journal of Child Psychology and Psychiatry and Allied Disciplines, 35,* 73–112.

Dalgard, O. S., Bjørk, S., & Tambs, K. (1995). Social support, negative life events and mental health—A longitudinal study. *British Journal of Psychiatry, 166,* 29–43.

Dawson, G., Frey, K., Panagiotides, H., Yamada, E., Hessl, D., & Osterling, J. (1999). Infants of depressed mothers exhibit atypical frontal electrical brain activity during interactions with mother and with a familiar, nondepressed adult. *Child Development, 70,* 1058–1066.

Eaves, L., Silberg, J., & Erkanli, A. (2003). Resolving multiple epigenetic pathways to adolescent depression. *Journal of Child Psychology and Psychiatry, 44*(7), 1006–1014.

Essau, C. A. (2004). The association between family factors and depressive disorders in adolescents. *Journal of Youth and Adolescence, 33,* 365–372.

Essex, M. J., Klein, M. H., Cho, E., & Kraemer, H. C. (2003). Exposure to maternal depression and marital conflict: Gender differences in children's later mental health symptoms. *Journal of the American Academy of Child and Adolescent Psychiatry, 42,* 728–737.

Essex, M. J., Kraemer, H. C., Armstrong, J. M., Boyce, T., Goldsmith, H. H., Klein, M. H., et al. (2006). Exploring risk factors for the emergence of children's mental health problems. *Archives of General Psychiatry, 63,* 1246–1256.

Fergusson, D. M., Horwood, L. J., & Boden, J. M. (2006). Structure of internalising symptoms in early adulthood. *British Journal of Psychiatry, 189,* 540–546.

Forbes, E. E., Shaw, D. S., Fox, N. A., Cohn, J. F., Silk, J. S., & Kovacs, M. (2006). Maternal depression, child frontal asymmetry, and child affective behavior as factors in child behavior problems. *Journal of Child Psychology and Psychiatry, 47,* 79–87.

Gaylord, N. K., Kitzmann, K. M., & Lockwood, R. L. (2003). Child characteristics as moderators of the association between family stress and children's internalizing, externalizing, and peer rejection. *Journal of Child and Family Studies, 12,* 201–213.

Ge, X., Natsuaki, M. N., & Conger, R. D. (2006). Trajectories of depressive symptoms and stressful life events among male and female adoles-

cents in divorced and nondivorced families. *Development and Psychopathology, 18,* 253–273.

Gilliom, M., & Shaw, D. S. (2004). Codevelopment of externalizing and internalizing problems in early childhood. *Development and Psychopathology, 16,* 313–333.

Grant, K. E., Compas, B. E., Stuhlmacher, A. F., Thurm, A. E., McMahon, S. D., & Halpert, J. A. (2003). Stressors and child and adolescent psychopathology: Moving from markers to mechanisms of risk. *Psychological Bulletin, 129,* 447–466.

Hammen, C., & Brennan, P. A. (2003). Severity, chronicity, and timing of maternal depression and risk for adolescent offspring diagnoses in a community sample. *Archives of General Psychiatry, 60,* 253–258.

Henderson, H. A., Fox, N. A., & Rubin, K. H. (2001). Temperamental contributions to social behavior: The moderating roles of frontal EEG asymmetry and gender. *Journal of the American Academy of Child and Adolescent Psychiatry, 40,* 68–74.

Hesbacher, P. T., Rickels, R., Morris, R. J., Newman, H., & Rosenfeld, M. D. (1980). Psychiatric illness in family practice. *Journal of Clinical Psychiatry, 41,* 6–10.

Hu, L. T., & Bentler, P. M. (1998). Fit indices in covariance structure modeling: Sensitivity to underparameterized model misspecification. *Psychological Methods, 3,* 424–453.

Jaffee, S. R., Moffitt, T. E., Caspi, A., Fombonne, E., Poulton, R., & Martin, J. (2002). Differences in early childhood risk factors for juvenile-onset and adult-onset depression. *Archives of General Psychiatry, 59,* 215–222.

Jöreskog, K. G., & Sörbom, D. (1993). *LISREL 8 user's reference guide.* Chicago: SSI.

Kazdin, A. E., Kraemer, H. C., Kessler, R. C., Kupfer, D. J., & Offord, D. R. (1997). Contributions of risk-factor research to developmental psychopathology. *Clinical Psychology Review, 17,* 375–406.

Kendler, K. S., Gardner, C. O., & Prescott, C. A. (2002). Toward a comprehensive developmental model for major depression in women. *American Journal of Psychiatry, 159,* 1133–1145.

Kendler, K. S., Gardner, C. O., & Prescott, C. A. (2006). Toward a comprehensive developmental model for major depression in men. *American Journal of Psychiatry, 163,* 115–124.

Kendler, K. S., Neale, M. C., Kessler, R. C., Heath, A. C., & Eaves, L. J. (1992). Major depression and generalized anxiety disorder—Same genes, (partly) different environments. *Archives of General Psychiatry, 49,* 716–722.

Kovacs, M., & Devlin, B. (1998). Internalizing disorders in childhood. *Journal of Child Psychology and Psychiatry and Allied Disciplines, 39,* 47–63.

Kristensen, H., & Torgersen, S. (2007). The association between avoidant personality traits and motor impairment in a population-based sample of 11–12-year-old children. *Journal of Personality Disorders, 21,* 87–97.

Leech, S. L., Larkby, C. A., Day, R., & Day, N. L. (2006). Predictors and correlates of high levels of depression and anxiety symptoms among children at age 10. *Journal of the American Academy of Child and Adolescent Psychiatry, 45,* 223–230.

Leve, L. D., Kim, H. K., & Pears, K. C. (2005). Childhood temperament and family environment as predictors of internalizing and externalizing trajectories from ages 5 to 17. *Journal of Abnormal Child Psychology, 33,* 505–520.

Luby, J. L., Belden, A. C., & Spitznagel, E. (2006). Risk factors for preschool depression: The mediating role of early stressful life events. *Journal of Child Psychology and Psychiatry, 47,* 1292–1298.

Luthar, S. S., & Brown, P. J. (2007). Maximizing resilience through diverse levels of inquiry: Prevailing paradigms, possibilities and priorities for the future. *Development and Psychopathology, 19,* 931–955.

MacPhee, A. R., & Andrews, J. J. W. (2006). Risk factors for depression in early adolescence. *Adolescence, 41,* 435–466.

Masi, G., Mucci, M., Favilla, L., Brovedani, P., Millepiedi, S., & Perugi, G. (2003). Temperament in adolescents with anxiety and depressive disorders and in their families. *Child Psychiatry & Human Development, 33,* 245–259.

Mathiesen, K. S., & Sanson, A. (2000). Dimensions of early childhood behavior problems: Stability and predictors of change from 18 to 30 months. *Journal of Abnormal Child Psychology, 28,* 15–31.

Mathiesen, K. S., & Tambs, K. (1999). The EAS Temperament Questionnaire—Factor structure, age trends, reliability, and stability in a Norwegian sample. *Journal of Child Psychology and Psychiatry and Allied Disciplines, 40,* 431–439.

Mathiesen, K. S., Tambs, K., & Dalgard, O. S. (1999). The influence of social class, strain and social support on symptoms of anxiety and depression in mothers of toddlers. *Social Psychiatry and Psychiatric Epidemiology, 34,* 61–72.

Merikangas, K. R., Zhang, H. P., Avenevoli, S., Acharyya, S., Neuenschwander, M., & Angst, J. (2003). Longitudinal trajectories of depression and anxiety in a prospective community study—The Zurich cohort study. *Archives of General Psychiatry, 60,* 993–1000.

Mesman, J., & Koot, H. M. (2000). Child-reported depression and anxiety in preadolescence: II. Preschool predictors. *Journal of the American Academy of Child and Adolescent Psychiatry, 39,* 1379–1386.

Moffitt, T. E., Caspi, A., Harrington, H., Milne, B. J., Melchior, M., Goldberg, D., et al. (2007). Generalized anxiety disorder and depression: Childhood risk factors in a birth cohort followed to age 32. *Psychological Medicine, 37,* 441–452.

Moffitt, T. E., Harrington, H., Caspi, A., Kim-Cohen, J., Goldberg, D., Gregory, A. M., et al. (2007). Depression and generalized anxiety disorder—Cumulative and sequential comorbidity in a birth cohort followed prospectively to age 32 years. *Archives of General Psychiatry, 64,* 651–660.

Muthén, L. K., & Muthén, B. (2006). *Mplus user's guide* (4th ed.) Los Angeles: Author.

O'Connor, T. G. (2006). The persisting effects of early experience on psychological development. In D. Cicchetti & D. J. Cohen (Eds.), *Developmental psychopathology* (2nd ed., pp. 202–234). Hoboken, NJ: Wiley.

O'Connor, T. G., Heron, J., Golding, J., & Glover, V. (2003). Maternal antenatal anxiety and behavioural/emotional problems in children: A test of a programming hypothesis. *Journal of Child Psychology and Psychiatry and Allied Disciplines, 44,* 1025–1036.

Phillips, N. K., Hammen, C. L., Brennan, P. A., Najman, J. M., & Bor, W. (2005). Early adversity and the prospective prediction of depressive and anxiety disorders in adolescents. *Journal of Abnormal Child Psychology, 33,* 13–24.

Rodgers, K. B., & Rose, H. A. (2002). Risk and resiliency factors among adolescents who experience marital transitions. *Journal of Marriage and the Family, 64,* 1024–1037.

Rothbart, M. K., & Bates, J. E. (1998). Temperament. In N. Eisenberg (Ed.), *Handbook of child psychology: Vol. 3. Social, emotional, and personality development* (pp. 105–176). New York: Wiley.

Rothbart, M. K., & Bates, J. E. (2006). Temperament. In N. Eisenberg (Ed.), *Handbook of child psychology: Vol. 3. Social, emotional, and personality development* (6th ed., pp. 99–166). Hoboken, NJ: Wiley.

Rutter, M. (2003). Commentary: Nature–nurture interplay in emotional disorders. *Journal of Child Psychology and Psychiatry and Allied Disciplines, 44,* 934–944.

Rutter, M., Kim-Cohen, J., & Maughan, B. (2006). Continuities and discontinuities in psychopathology between childhood and adult life. *Journal of Child Psychology and Psychiatry, 47,* 276–295.

Rutter, M., Moffitt, T. E., & Caspi, A. (2006). Gene–environment interplay and psychopathology: Multiple varieties but real effects. *Journal of Child Psychology and Psychiatry, 47,* 226–261.

Schafer, J. L., & Graham, J. W. (2002). Missing data: Our view of the state of the art. *Psychological Methods, 7,* 147–177.

Sroufe, L. A., & Rutter, M. (1984). The domain of developmental psycho-pathology. *Child Development, 55,* 17–29.

Statistics Norway. (2006). *Immigration and Immigrants 2006* (Annual report). Series: Statistical Analyses (SA 87). Available from http://www.ssb.no/english/subjects/02/sa_innvand_en/sa87/

Sund, A. M., Larsson, B., & Wichstrøm, L. (2001). Depressive symptoms among young Norwegian adolescents as measured by the Mood and Feeling Questionnaire (MFQ). *European Child and Adolescent Psychiatry, 10,* 222–229.

Tambs, K., & Moum, T. (1993). How well can a few questionnaire items indicate anxiety and depression? *Acta Psychiatrica Scandinavica, 87,* 364–367.

Thompson, R., Flood, M. F., & Goodvin, R. (2006). Social support and developmental psychopathology. In D. Cicchetti & D. J. Cohen (Eds.), *Developmental psychopathology* (2nd ed., pp. 1–37). Hoboken, NJ: Wiley.

Treiber, F. A., & Mabe, P. A. (1987). Child and parent perceptions of children's psychopathology in psychiatric outpatient children. *Journal of Abnormal Child Psychology, 15,* 115–124.

Varner, V., Wickramaratne, P., & Weissman, M. (2008). The role of fear and anxiety in the familial risk for major depression: A three-generation study. *Psychological Medicine, 38,* 1543–1556.

Zahn-Waxler, C., Crick, N. R., Shirtcliff, E. A., & Wood, K. E. (2006). The origins and development of psychopathology in females and males. In D. Cicchetti & D. J. Cohen (Eds.), *Developmental psychopathology* (2nd ed., pp. 76–138). Hoboken, NJ: Wiley.

Zucker, R. A. (2006). Alcohol use and the alcohol use disorders: A developmental–biopsychosocial systems formulations covering the life course. In D. Cicchetti & D. J. Cohen (Eds.), *Developmental psychopathology* (2nd ed., pp. 620–656). Hoboken, NJ: Wiley.

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