Published in final edited form as:
Child Dev. 2012 November ; 83(6): 1945–1959. doi:10.1111/j.1467-8624.2012.01821.x.

Growth in Externalizing and Internalizing Problems in Childhood: A Prospective Study of Psychopathology across Three Generations

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Abstract

Three generations of participants were assessed over approximately 27 years, and intergenerational prediction models of growth in the third generation's (G3) externalizing and internalizing problems across ages 3 to 9 years were examined. The sample included 103 fathers and mothers (G2), at least one parent (G1) for all of the G2 fathers (99 mothers, 72 fathers), and 185 G3 offspring (83 boys, 102 girls) of G2, with prospective data available on the G2 fathers beginning at age 9 years. Behavior of the G2 mother, along with father contact and mother age at birth were included in the models. Intergenerational associations in psychopathology were modest, and much of the transmission occurred via contextual risk within the family of procreation.

Keywords

childhood; externalizing; fathers; growth modeling; intergenerational; internalizing

Continuities in family socialization and contextual risks across generations, as well as genetic factors, are associated with the development of both externalizing (Conger, Belsky, & Capaldi, 2009; Simonoff, 2001) and internalizing (Enam, 2003; Kim, Capaldi, Pears, Kerr, & Owen, 2009; Kim, Capaldi, & Stoolmiller, 2003) problems in children. As a parent shares on average 50% of their genes with a biological child, accurate estimates of the magnitude of intergenerational associations in these problem behaviors are needed in order to interpret the role of contributing factors. The size of the associations places an upper bound on the contribution of all continuity factors combined, including genetic contributions. If the magnitude of associations is small, then genetic and environmental continuity factors may not be as strong as has often been assumed or may be subject to considerable moderation. Thus, intergenerational studies are critical to informing research and policy regarding cross-generation associations, including genetic heritability studies; yet until relatively recently, estimates of cross-generational associations were based largely on retrospective reports (Thornberry, 2009).

No prior intergenerational studies, of which we are aware, have predicted growth in children's problem outcomes; thus, key issues remain to be addressed. The purpose of the current study was to examine risk for child internalizing and externalizing problems prospectively across three generations. It was not the purpose of the study to focus on co-occurrence of these problems, but rather to shed light on heritability and other transmission issues for each of these dimensions of psychopathology that can be evident relatively early in childhood. To achieve this, hypotheses were tested across three generations from the Oregon Youth Study (OYS). Separate prediction models grouped by gender were run for growth in internalizing and externalizing symptoms in children (Generation 3; G3) across

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early development (from ages 3 to 9 years)—including prediction from the parallel areas of symptoms in paternal grandparents (Generation 1; G1), in fathers during their childhoods, and in both parents (Generation 2; G2) during early adulthood. The inclusion of both G2 parents' risk behaviors is a strength of the study, given that fathers' influences are often neglected in developmental research. Models also included father contact (a combination of living with the child and the number of days the child saw the father) with the G3 child during early childhood and the mother's age at birth of the child. The final period of measurement for G3 was at the same developmental stage—age 9 years—as the childhood assessment for their fathers.

The current study builds on an earlier study with the OYS sample (Kim et al., 2009) that examined intergenerational transmission of internalizing and externalizing behaviors across just two generations within the same model. The current study examines pathways across three generations for each area of psychopathology and examines prediction to growth in externalizing or internalizing across ages 3 to 9 years; whereas the outcome for G3 in Kim et al. was a single score (the average of psychopathology at ages 18–21 months and 3 years), rather than growth.

The underlying conceptual model for the current study is the Dynamic Developmental Systems (DDS) model (e.g., Capaldi, in press). The process of development, including the development of externalizing and internalizing behavior, may be conceptualized as due to transactions across and within physiological, psychological, and social systems, with constant feedback and interaction over time. The DDS model is an extension of the general lifespan and ecological models and further articulates both developmental and social influence processes. With regard to the current study, the interaction of the characteristics of the developing individual, which may be in part genetically inherited—such as temperament risk, including irritability and impulsivity (Caspi & Bem, 1990)—with his or her immediate social environment, occur within and are influenced by larger contextual factors affecting the family (e.g., psychopathology of both parents, age at parenthood, father presence). These larger contextual factors affect the individual in childhood particularly via family resources and interactions.

Intergenerational associations are expected partially because of evidence of genetic effects related to temperamental risks for externalizing via low behavioral inhibition related to noradrenaline (Rogeness et al., 1984) and serotonin (Kruesi et al., 1990) levels in the brain. Genes affecting dopamine function have been found to be associated with impulsiveness (Limosin et al., 2005). Candidate polymorphisms in the serotonergic system are implicated in internalizing disorders, including anxiety, depression, and stress (Collier et al., 1996).

In a meta-analyses involving participants under age 18 years, Burt (2009) found that 59% of the variance in externalizing symptoms and 51% of the variance in internalizing symptoms was explained by additive genetic influences (the effect of individual genes summed over loci), with an additional 15% of the variance in externalizing and 16% of the variance in internalizing being explained by shared environmental influences. These estimates would lead to the expectation of considerably higher cross-generational associations than have been found by intergenerational studies thus far. Prospective correlations between generations for the developmental periods of childhood and adolescence tend to be small to moderate for antisocial behavior (e.g., Capaldi, Pears, Patterson, & Owen, 2003; Conger, Neppl, Kim, & Scaramella, 2003; Kim et al., 2009; Thornberry, Freeman-Gallant, Lizotte, Krohn, & Smith, 2003; Thornberry, Freeman-Gallant, & Lovegrove, 2009), with maximum associations generally in the .30 range. Correlations tend to be small for internalizing symptoms (e.g., . 18, Kim et al.; .19, van Meurs, Reef, Verhulst, & van der Ende, 2009) and small to moderate for cross-generational paths from major depressive disorder to internalizing symptoms

(e.g., .22 to .26, Olino et al., 2008). There are a number of differences in designs across the studies that may account for this, particularly the fact that twin studies usually involve one rater (e.g., the mother) for each twin at one point in time, whereas intergenerational studies often involve different individuals as raters (often the mothers or parents from two generations) and many years between the ratings.

The conceptual model of the current study addresses the ways in which intergenerational risks are related to the process of *development* of problem behaviors, including possible differential growth across childhood. A pattern of early onset then decreasing overt externalizing behaviors in early childhood has been found in a number of studies (e.g., Gilliom & Shaw, 2004; Mathiesen, Sanson, Stoolmiller, & Karevold, 2009). Further, there is evidence that both boys and girls showed improvement in physical aggression (hitting, biting, and kicking) from ages 2 to 11 years, although girls appeared to improve more rapidly than boys from approximately ages 4 to 8 years (Tremblay, Masse, Pagani-Kurtz, & Vitaro, 1996). Thus, for both boys and girls, intergenerational risks may predict to initially higher levels of externalizing (intercept), failure to make normative improvements across childhood (slope), or both.

The few studies that have examined growth in internalizing symptoms in the early years of childhood indicate a contrasting pattern to that for externalizing, namely a gradual increase in internalizing symptoms across early childhood for both boys and girls (e.g., Gilliom & Shaw, 2004; Mathiesen et al., 2009). This may be related to emotional and cognitive developments in the child that enable the parent to recognize and identify the symptoms as internalizing. For both boys and girls, intergenerational risks may predict to initially higher levels of internalizing, more rapid growth in symptoms across childhood, or both. In sum, the overall developmental trajectories of externalizing and internalizing symptoms in childhood appear to differ, although it is well established that the behaviors are associated (Angold, Costello, & Erkanli, 1999; Capaldi, 1991; Gilliom & Shaw). Thus, the questions regarding prediction to growth in early childhood also differ.

The intergenerational conceptual prediction model for externalizing outcomes is shown in Figure 1. The model for G3 internalizing outcomes is identical in design to provide comparability of models, but includes prediction from depressive symptoms in G1 and G2. G2 psychopathology was included in the model in late childhood and again in early adulthood to examine the possibility that for developmental reasons intergenerational associations in psychopathology might be evident from G2 childhood to G3 but not from G2 early adult psychopathology to G3 childhood psychopathology. Inclusion of G2 early adult psychopathology, however, allowed for testing of more proximal associations of G2 with G3 behavior (e.g., paternal behavior experienced by the child in toddlerhood and to age 3 years) and for testing whether any effects of G2 childhood psychopathology were explained (mediated) by continuities into early adulthood. For both externalizing and internalizing, respectively, it was predicted that G1 psychopathology would be associated with the intercept of psychopathology for G3. However, this association was hypothesized to be mediated by G2 fathers' psychopathology (childhood and adulthood). That is, primary paths of transmission were predicted from one generation to the next via risk factors at a number of levels, rather than by risks that skipped a generation.

Of note, transmission of psychopathology both genetically and environmentally from G1 to G3 that is not mediated by that in G2 is possible (Olino et al., 2008), perhaps because of inheritance via recessive alleles, variation in gene expression (epigenetic variation), and environmental variation affecting gene expression. Thus, it was possible that some association might be present between G1 and G3 psychopathology that was not mediated by G2 psychopathology.

It also was expected that because of assortative mating (e.g., Krueger, Moffitt, Caspi, Bleske, & Silva, 1998; Mathews & Reus, 2001) and partner influences, psychopathology of G2 fathers and mothers would be associated. Psychopathology in G2 mothers and fathers was expected to be associated with both the intercept and slope of psychopathology in G3. Finally, two aspects of risk for the G2 family of procreation were examined, namely young age of the mother at birth of the G3 child and low father contact with the G3 child in early childhood. The first risk is known to be associated with more problematic outcomes for children, including increased risk for externalizing problems (Black et al., 2002). Thus, including mother's age at birth of the child helps clarify the predictive role of parental psychopathology versus risk due to having very young parents. Mother's age at birth of the child was also examined as a possible mediator of G2–G3 associations.

Regarding the absence of G2 fathers during early development, it was hypothesized that low father contact with the child in the early years would increase risk for G3 problems initially and over time, because such absences are related to a number of risk factors such as lower family income and maternal stress (Jackson, Brooks-Gunn, Huang, & Glassman, 2000). In keeping with this prediction, Jaffee, Moffitt, Caspi, and Taylor (2003) found that the amount of time children spent living with their fathers was inversely related to children's conduct problems at age 5 years. Consistent with the DDS model, we also considered that G2 fathers' environmental influences on G3 children's outcomes (e.g., via parenting) should depend on having contact, whereas fathers' transmission of genetic risk to their children should not; thus, the models should include exposure to father in the early years of childhood to help clarify influences. Additionally, Jaffee et al. reported that father absence does not always confer risk, because children of highly antisocial fathers had more conduct problems if they resided with their fathers. Thus, we explored whether G3 children whose fathers had higher contact with them in early childhood: (a) would show different intercept and slopes or (b) different (i.e., moderated) associations between their fathers' psychopathology in adulthood and their own.

The current study builds on prior work on prospective intergenerational associations in psychopathology in a number of ways. First, effects of father psychopathology from both childhood and adulthood were included in the model to test ways in which parents' childhood and adult histories of problem behavior impact their children's behaviors. Second, intergenerational associations were examined to some degree for both G2 parents. Third, the focus on growth in outcome behaviors across ages 3 to 9 years represents an advance over prior studies that have generally predicted to psychopathology at one point in time only.

Method

Participants

Prospective measures from three generations of participants in the OYS and the ongoing Three Generational Study (3GS) were used to test hypotheses. Original recruitment of G1 parents and their sons (G2) targeted all fourth-grade boys attending schools in higher juvenile crime-rate areas of a medium-sized metropolitan area in the Pacific Northwest. A 74% recruitment rate resulted in sample of 206 families who were 90% Euro American and predominantly lower and working class (Hollingshead, 1975). Five young men in G2 have died, and retention rates were 95% or higher at each year for the remaining men. All biological and step children (G3) of G2 were originally eligible to participate in 3GS, as well as the G2 mothers of G3; the sample was later limited to the first two biological offspring of each partner of the G2 men. Many men in the OYS had either not yet had a biological child at the time of the current study or had a child who was too young to have completed at least two time points of the 3GS (an inclusion criteria for the current study).

Shown in Figure 2 is the sample included in the current study. Multiple children from one father (which he may have had with different partners) could be included in the study.

The 103 G2 men included in the analyses had significantly higher levels of childhood antisocial behavior and their G1 parents showed higher mean levels of depressive symptoms and antisocial behavior than men not included in the analysis. The 185 G3 children in the current analysis sample did not significantly differ from others in the sample on any of the externalizing and internalizing outcomes. For the G2 mothers, those included in the analysis showed higher depressive symptoms than the mothers not in the analysis.

Procedures

During the OYS, G1 parents and G2 fathers were assessed using yearly multimethod, multiagent assessments starting when the G2 youth were ages 9–10 years (OYS Wave 1 [W1]) and continuing until they were ages 31–32 years. The current study uses data from the first 3 years of OYS, and assessments included interviews and questionnaires for the G2 youth and the G1 parents. Data were also collected from school records, teacher questionnaires, classroom peer nominations, official court records, interviewer and observer ratings, and coding of parent-child interactions in the home.

The 3GS assessments of the G2 parents and their G3 offspring started when the children were approximately age 21 months (3GS Time 1 [T1]) and included laboratory tasks as well as interviews and questionnaires. Each yearly assessment involved an appointment with the G2 mother and then one with the G2 father within the next 2 weeks.

Measures

Constructs for the current study were taken from the following developmental periods across the three generations.

1. G1 parents', and G2 fathers' behavior (in late childhood) was assessed in both the first and third years of the OYS (mean ages for the G1 fathers and mothers = 35.8 and 33.1 years, respectively; G2 mean ages = 10.1 and 12.0 years, respectively).
2. The G2 fathers' and mothers' behavior in adulthood was assessed at the first two time points of the 3GS, when the G3 children were approximately 21 and 39 months (3 years) of age (mean ages for the G2 fathers and mothers = 26.1 and 24.8 years, respectively).
3. G3 psychopathology was assessed using G2 parents' reports at four time points (ages 3.2, 5.2, 7.3, and 9.2 years; Time 2 [T2] through Time 4 [T4], respectively) T1 was not included because the Child Behavior Checklist measures were not used at T1.

Because of space considerations, descriptions of measures are abbreviated. Data reduction strategies for the OYS and 3GS have been described elsewhere (e.g., Capaldi, 1991; Capaldi et al., 2003) and are also described in more detail at http://www.oslc.org/appendices/cd_externalizing_3gs_capaldi.pdf. Constructs for G1 and G2 antisocial behavior and depressive symptoms have also been described elsewhere (e.g., Capaldi et al., 2003; Kim et al., 2009) and also are available in detail for the current study at the same web address. They are described briefly below.

G1 antisocial behavior during G2 fathers' late childhood (G2 ages 9 to 12 years)—Mother and father antisocial behavior constructs each comprised of self-reports, official arrest records, drivers license suspensions (both cumulative through OYS W1), and staff ratings.

G1 depressive symptoms during G2 fathers' late childhood (G2 ages 9 to 12 years)—G1 parents' depressive symptoms were assessed by three self-report indicators including the 20-item Center for Epidemiologic Studies Depression Scale (CES-D; Radloff, 1977). For G1 antisocial behavior and depressive symptoms, the mean of mother and father scores was used, and for depressive symptoms, the mean of the Wave1 [W1] and Wave 3 [W3] constructs was taken.

G2 fathers' late childhood antisocial behavior and depressive symptoms (ages 9 to 12 years)—Antisocial behavior was measured using the mean of the antisocial construct and the total number of arrests through OYS W3 from official court records. The final constructs were the means of the respective OYS W1 and W3 constructs.

G2 father contact in G3 early childhood (G3 ages 21 to 39 months)—Father contact with each child was assessed by: (a) interviews of the G2 fathers from the OYS regarding their contact with each child on a scale from 1 'never see' to 8 'live with full time' (e.g., the value 4 was 'Visit 2–3 times a week') and (b) the 3GS T1 and T2 parent interviews, wherein the G2 father and mother each reported on the living situation of the child and changes in status (i.e., parents in and out of the home) from the child's birth to the time of the current interview. Information regarding with whom the child had lived since birth was used to calculate estimates of the percentage of days since birth that the G3 child had lived with the G2 father. Measures were then standardized to a *Z*-distribution and combined. Mother and father reports from the 3GS were first combined within wave and then combined with the father report from the OYS wave closest to the 3GS time point, and then the T1 and T2 measures were combined to arrive at a continuous measure of father contact. Of the 185 children included in the study, 100 lived full time with their fathers through age 3 years. Note that 58 of the children who lived full time with their father had siblings (42 men had just one child in the current study).

G2 mother age at birth of G3—Birthdates of the 3GS offspring and their G2 parents were used to calculate the age of the G2 father and mother at the birth of the G3 child. As they were highly associated ($r = .56$, $p > .01$), only the mother's age was used (mean = 22.73, $SD = 3.88$, range = 15.2 to 37.0).

G2 fathers' and mothers' adult antisocial behavior (G3 ages 21 to 39 months)—Assessment of antisocial behavior for the G2 mothers and the G2 fathers differed slightly. For the mothers, the raw frequencies from 16 items on the National Youth Study Delinquency Scale (Elliot, Ageton, Huizinga, Knowles, & Canter, 1983) were recoded to a 9-point scale and then summed. For the G2 fathers, more information was available through their participation in the OYS. Thus, their antisocial behavior was measured as the mean of their observed OYS construct score and the number of arrests during the OYS assessment wave closest to each of the 3GS T1 and T2. The constructs were first standardized on the OYS sample and then standardized again based upon the 3GS analyses sample to help control for these differences in measurement. The final score for each parent represented the mean of the scales available at 3GS T1 and T2.

G2 fathers' and mothers' depressive symptoms (G3 ages 21 to 39 months)—The CES-D was used to assess the G2 fathers' and mothers' depressive symptoms. Each observed construct was formed as the mean of the indicators available from parent questionnaires that were completed at the 3GS T1 and T2 assessments.

G3 externalizing behavior (G3 ages 39 months to 10 years)—G3 externalizing behavior was measured using father and mother reports on 11 items from the Child Behavior

Checklist (CBCL; Achenbach, 1991) that were highly comparable on both the early childhood (CBC/2–3) and childhood (CBC/4–18) versions: "Can't concentrate," "Can't sit still," "Doesn't feel guilty after misbehaving," "Destroys own things," "Destroys others things," "Disobedient at home," "Gets in many fights," "Physically attacks people," "Screams a lot," "Stubborn, sullen, irritable," and "Temper tantrums." The items were summed and log transformed for both the mother and father. The final score was the mean of father and mother report at each time point.

G3 internalizing behavior (G3 ages 39 months to 10 years)—The internalizing scores for the G3 children were formed from 10 variables that were identical across the CBCL measures for early childhood and childhood versions (Achenbach, 1991): "Worrying," "Nervous, high-strung or tense," "Self-conscious, easily embarrassed," "Too fearful or anxious," "Unhappy, sad, depressed," "Aches or pains without known medical cause," "Headaches without medical cause," "Stomachaches without medical cause," "Vomiting without medical cause," and "Withdrawn." Items were summed and then the scales were log transformed for the father and mother reports individually at each time point. The mean of parents' reports formed the composite score at each time point. A table providing descriptive data for the raw and transformed externalizing and internalizing scores, along with a figure of the means by age, is available at http://www.oslc.org/appendices/cd_externalizing_3gs_capaldi.pdf.

Data Analytic Strategies

Latent growth modeling and observed predictor path modeling were conducted using Mplus 6.11 (Muthén & Muthén, 1998–2010). The complex samples option was used to adjust standard errors to account for the nesting of multiple G3 children (i.e., siblings) within G2 fathers. This option involves using a sandwich estimator, a widely used approach (rather than the inverse Fisher Information matrix) for the computation of standard errors when data are not independent (Asparouhov, & Muthen, 2005). This was used in conjunction with maximum likelihood with robust standard errors and χ^2 to estimate the models while accounting for nonindependence of observations. Externalizing and internalizing outcomes were analyzed separately in a series of growth models with increasing constraints to growth and associated parameters as follows: (a) an unconditional correlational model; (b) an unspecified single-factor outcome model; (c) an unspecified two-factor-growth model involving a linear spline model whereby T1 was set to 0, Time 4 (T4) was set to 6, the intervening time points were estimated, and the shape of the slope was not constrained (Meredith & Tisak 1990); (d) a model imposing equal error variances; (e) a model testing whether the intercept and slope were significantly associated; (f) a linear model in which loadings were set to 0, 2, 4, and 6, respectively, because of the 2-year gaps between observations; and (g) a quadratic growth model. The best fitting model of this series of nested models was used to specify the intercept and slope-factor outcomes for further modeling of intergenerational predictors (which were transformed and standardized). A model was then run for both growth outcomes that included grouping by gender, and these models were modified to include additional parameters or constraints to improve model fit and parsimony.

As the current sample size is relatively small in comparison to the complexity of the models tested, a stepwise approach was used to evaluate predictors of the G3 outcome growth factors and identify parsimonious models. First, a saturated model was run with all predictors present. All paths possible were estimated within temporal ordering constraints, and included paths from G1 and G2 behavior when G2 was aged 9–12 years to G2 mother and father behavior when the G3 was aged 21 to 35 months, age of mother at birth of G3, and father contact, as well as to G3 childhood intercept and slope. Paths were also present

from G2 mother and G2 father behavior, mother age at G3 birth, and G2 father contact with G3 to intercept and slope of G3 behavior from ages 3 to 9 years. All possible covariances within time point (G2 age 9–12 years and G3 age 21–39 months) were also estimated. Next, all regression paths that were nonsignificant ($p > .10$) for both genders were trimmed one at a time in an iterative fashion, starting with the regression path with the highest p-value. Finally, covariances that were nonsignificant for both genders were trimmed to arrive at the final prediction models.

There were no missing data for G1 or G2 late childhood predictors, G2 father age at G3 birth, G2 father contact, or for the G2 father's adult predictors. G2 mother's age at first birth was missing in one case, the G2 mother's delinquent behavior was missing for seven children, and G2 mother's CES-D was missing for five children. Regarding the outcome data, there were missing data because of the intergenerational (rather than cohort-based) design (i.e., not all G3 children had yet reached the age to participate in all of the assessment time points used in the current study). Of the 185 G3 children in the current analyses, 98.4% had data at age 3, 95.7% at age 5, 80% at age 7, and 53.5% at age 9 years. The maximum likelihood algorithm was used to derive parameter estimates in the presence of missing data, and the Yuan-Bentler T2 χ^2 was used because it provides tests that are robust to nonnormality and nonindependence of observations (Muthén & Muthén, 1998–2010). Chi-Square difference tests reported in nested model tests have been adjusted using the scaling correction factors from the output as described on the Mplus website (http://www.statmodel.com/chidiff.shtml).

Results

Correlations between G3 Externalizing and Internalizing Scores

The correlations between G3 externalizing and internalizing scores within each time point ranged from $r = .33$ to .53, $p < .05$, and did not differ discernibly by child gender.

Externalizing Behavior

Unconditional growth model—The best fitting and most parsimonious unconditional growth model of externalizing behavior was the nonspecified two-factor growth factor with equal outcome error variances and uncorrelated intercept and slope. Addition of gender in a two-group model did not significantly improve the fit, although allowing the error variances to vary between (but not within) gender did result in a significant improvement in fit to arrive at the final two-group linear-spline growth model. Testing gender differences (i.e., group equalities) in the growth parameters showed significant differences in slope mean (boys −.02, girls −.04), $\chi^2(1, N = 185) = 3.85$, $p < .05$, indicating that girls improved more over time in externalizing symptoms than did boys, but with no significant differences in slope variance (both .01), intercept mean (boys .70, girls .72), or intercept variance (boys .02, girls .03). Further information regarding the unconditional growth models tested and fit statistics is available from the first author.

Prediction model—The correlation matrices of predictors and the four time points of externalizing outcomes for boys and girls are shown in Table 1. Both boys (above the diagonal) and girls (below the diagonal) in G3 showed significant associations across the four assessment time points for externalizing behaviors, indicating some stability in rank order in this behavior over time. Associations of the predictors with externalizing in G3 children were generally weak and did not reach significance when patterns for boys and girls were examined separately.

Shown in Table 2 are the χ^2 difference tests regarding the significance in change in model deviance from the prior tested model for each of the growth-model steps described in the analytic plan. Shown in Figure 3 are the findings for the final prediction model to the intercept and slope of externalizing behavior for the G3 children, grouped by gender. Note also that slope factor loadings are shown. As antisocial behavior for G1 and the G2 men in childhood were measured at the same point (OYS W1), this association was tested as a covariance rather than a directional pathway. Sex of the G3 offspring was not expected to be related to the associations among the G1 and G2 predictors; thus, for greater parsimony, these were constrained to be equal. Findings indicated that antisocial behavior in G1 parents was significantly associated with their G2 son's antisocial behavior in childhood, but did not show any additional prediction to the G2 men's antisocial behavior in adulthood or to any other outcomes over and above variance explained by G2 antisocial behavior. G2 fathers' antisocial behavior in childhood was significantly predictive of their antisocial behavior in adulthood, a younger age of G2 mothers at the birth of G3, and lower G2 father contact with the G3 child at ages 2–3 years. In adulthood, G2 fathers' and mothers' antisocial behavior were associated regardless of G3 gender. G2 mothers' antisocial behavior was associated both with their younger age at the birth of G3 and less G2 father-contact time during the child's early years.

Predictions of the intercept of G3 externalizing behavior (at age 3 years) depended on the gender of the child. For girls only, the externalizing intercept was predicted in the hypothesized direction by the level of the G2 father's adult antisocial behavior, which was the only significant predictor of intercept for girls. Unexpectedly, for G3 boys, G2 father antisocial behavior was negatively associated with the intercept of externalizing behavior. In addition, G3 boys were likely to have higher levels of externalizing behavior at age 3 years if their mother was younger when they were born. Significant predictors to slope were found only for boys; boys were likely to show more growth or less decrease in externalizing behavior from ages 3–9 years if their mother was older when they were born. This may indicate that the protective effect on boys' externalizing behaviors at age 3 years (i.e., intercept) of having an older mother attenuated significantly over time (i.e., less decrease in externalizing); alternatively, because these boys overall had lower levels of these behaviors at age 3 years, they had less potential to show a decrease.

Overall, the two-group path model exhibited acceptable fit, χ^2 73.91 (88*df*), $p = 0.86$, with a 90% confidence interval for the RMSEA of .00–.03 and a TLI of 1.05. Regarding variance explained in the outcome, R^2 for the intercept was .08 (nonsignificant) for girls and .24 ($p < .01$) for boys. For the slope, the R^2 was .001 (nonsignificant) for girls and .12 ($p < .10$) for boys. Thus, the model did not explain significant variance in either the intercept or slope of externalizing for girls, and explained significant variance in the intercept but not slope of externalizing for boys.

Of note, although the correlations between the intercept and slope were constrained to be zero in the models, when freely estimated the correlations for externalizing were nonsignificant ($r = -.23$; $p = .26$) for girls and ($r = -.38$; $p = .12$) for boys.

For G3 girls, there was a significant total indirect effect of G2 father's childhood antisocial behavior on higher initial levels of externalizing ($\beta = .14$, $p = .02$), specifically via the G2 father's antisocial behavior in adulthood ($\beta = .11$, $p = .03$). For G3 boys, the total indirect effects of G2 fathers' childhood antisocial behavior were nonsignificant ($\beta = .01$, $p = .93$ for intercept and $\beta = -.10$, $p = .08$ for slope).

Internalizing Behavior

Unconditional growth model—The best fitting and most parsimonious unconditional growth model of internalizing behavior was grouped by gender, showed linear growth, had equal error variances across groups at ages 3, 5 and 7 years, and freely estimated variance in age 9 years internalizing behaviors. There were significant differences by gender on intercept variance (boys .004, girls .01) but not on intercept mean (boys .17, girls .20), slope mean (both .02), or slope variance (both .001).

Prediction model—The correlation matrices for boys and girls for internalizing outcomes are shown in Table 3. Overall, G3 boys (above the diagonal) and girls (below the diagonal) showed significant associations across time (from ages 3 to 9 years) for internalizing behaviors, indicating some stability in rank ordering. However, for boys, internalizing behaviors at age 3 years were associated with those at age 5 but not at ages 7 or 9 years. As in the externalizing behavior models, associations between the predictors and internalizing behaviors were generally low and nonsignificant regardless of G3 gender. Of note, however, maternal depressive symptoms in adulthood showed the highest associations with offspring depressive symptoms.

Shown in Table 2 in the right-hand column are the χ^2 difference tests for the significance in change in model deviance from the prior tested model for each of the model steps described in the analytic plan. The final prediction model to growth in internalizing behaviors is depicted in Figure 4. G1 to G2 associations were constrained to be equal across G3 gender (which did not significantly worsen model fit). Depressive symptoms assessed in the G1 parents when their son was aged 9–10 years were significantly associated with depressive symptoms in their son at that time. G2 parents' depressive symptoms were significantly associated. The G2 father's level of depressive symptoms in adulthood was significantly predicted both by G1 depressive symptoms and by his level of childhood depressive symptoms.

G2 mothers' depressive symptoms predicted G3 boys' and girls' internalizing behavior intercepts at age 3 years. Again, similar to externalizing models, G2 mothers' younger age at the birth of G3 predicted both higher levels of internalizing symptoms at age 3 years and lower growth in internalizing behaviors through age 9 years for boys. Neither the G2 father's depressive symptoms nor his contact with his child until age 3 years was predictive of intercept or slope of G3 internalizing behaviors.

Overall, the model showed adequate fit, $\chi^2 = 59.06$ (88 *df*), $p = 0.99$, with a 90% C.I. for RMSEA of .00–.00 and a TLI of 1.14. The model explained significant variance in the intercept for boys ($R^2 = .40$, $p < .05$) but not girls ($R^2 = .17$, $p < .10$) and did not explain significant variance in the slope of internalizing across childhood for either boys ($R^2 = .23$, $p < .10$) or girls ($R^2 = .02$, *ns*). Although the correlations between the intercept and slope were constrained to be zero in the models, when freely estimated the correlations for internalizing were nonsignificant for both girls ($r = .33$; $p = .32$) and boys ($r = -.32$; $p = .19$).

For G3 girls, the total indirect effect of G2 fathers' childhood depressive symptoms on the intercept of internalizing symptoms was significant overall ($\beta = 0.13$, $p < .05$); in part this occurred via G2 mothers' depressive symptoms ($\beta = .10$, $p < .05$). For G3 boys, there was a significant total indirect effect of G2 fathers' childhood depressive symptoms on the intercept of internalizing problems ($\beta = .24$, $p < .01$), which was comprised of a significant indirect path via G2 mothers' depressive symptoms ($\beta = .15$, $p < .05$). G2 fathers' childhood depressive symptoms were also indirectly associated with less growth in internalizing via G2 mother's age at birth of G3 ($\beta = -0.12$, $p < .05$).

Moderation of Intergenerational Associations by G2 Father Contact and Mother Age at Birth

Finally, we examined whether prediction from G2 mother's and father's psychopathology to G3 externalizing and internalizing behaviors was moderated by either the father contact in the child's early years or the mother age-at-birth variables – thus the interaction terms (e.g., between father contact and father antisocial behavior in predicting G3 externalizing behavior) were examined. Because of the size of the models, moderation (i.e., interaction effects) was tested in models including only the G2 predictors and G3 growth outcomes. There were no significant interactions predicting to intercept and slope of G3 externalizing behavior. For G3 internalizing behavior, the interaction of father depressive symptoms by father contact was positively associated ($\beta = .30$, $p < .05$) with the intercept for girls, indicating that higher levels of contact (through age 3 years) with a father with higher levels of depressive symptoms was linked with higher levels of internalizing for girls at age 3 years. There was a similar positive and significant (when standardized) path from the mother age at birth by father depressive symptoms interaction to the intercept of the growth model for girls ($\beta = .61$, $p < .01$). For boys and girls, there was a positive association from an interaction of mother age at birth by father depressive symptoms to the slope of internalizing ($\beta = .65$, $p < .001$ for boys; $\beta = .54$, $p < .05$ for girls), indicating accelerated growth in symptoms if a child was born to a young mother and a father with higher levels of depressive symptoms.

Discussion

Three generations of participants were assessed over approximately 27 years in this study of predictors of the development of children's externalizing and internalizing problems across ages 3 to 9 years. Consistent with prior work (e.g., Gilliom & Shaw, 2004), externalizing behaviors in G3 children decreased for both boys and girls across the 6-year period, with girls showing greater decreases. Findings for the prediction models indicated that associations between G1 psychopathology and the development of similar behaviors in G2 were generally consistent with prior work; for both antisocial behavior and depressive symptoms, they were in the moderate ($\beta = .30$ to .40) range. Interestingly, the G1 parents' depressive symptoms were a significant predictor of the G2 son's depressive symptoms in early adulthood in addition to his own depressive symptoms at age 9–10 years. This may indicate that the intergenerational transmission of such symptoms is stronger in adulthood because of reasons related to the developmental timing of gene expression, social learning, or early adult contextual risk. This point underscores the importance of examining intergenerational associations between problems assessed at comparable developmental periods.

Contrary to hypotheses, associations between paternal grandparents' and G2 fathers' antisocial behavior and depressive symptoms, and later similar psychopathology in G3, generally failed to reach statistical significance. Thus, this risk was not significant for boys' externalizing, was only on the maternal side for internalizing, and was not as strong as expected. This was surprising given that the parental and grandparental phenotypes represent the product of genetic risk, shared contextual risks (e.g., low income, neighborhoods), and learned social risks (e.g., poor-parenting skills). This suggests that a number of factors – likely including biological (e.g., the influence of multiple genes), contextual, and social factors – moderate cross-generational associations for externalizing and internalizing behaviors. This seems to indicate that, when using prospective data to predict childhood psychopathology, cross-generational associations, at least through fathers, may not be as large as anticipated for the externalizing and internalizing domains. Thus, genetic and environmental factors related to discontinuity across generations may be more numerous or stronger than those related to continuity.

Findings should be viewed in the context of prior studies with the current sample. Capaldi et al. (2003) found a significant association between the G2 father's antisocial or delinquent behavior in midadolescence and G3's early temperamental risk (at age 22 months) for externalizing in the form of higher levels of activity and anger, with boys and girls combined in the model. Consistent with the current study, Kim et al. (2009) examined associations between G2 father's delinquency in adolescence and G3 externalizing behavior in toddlerhood and found that father delinquency was positively associated with G3 externalizing behavior for girls but not for boys. Unlike the Kim et al. study, however, the association of father depressive symptoms with G3 internalizing behavior was nonsignificant for both boys and girls; whereas in the Kim et al. study, there was a significant association for girls.

The present and prior studies have tested somewhat different theoretical models at different developmental stages for G2 and G3. In addition, because of the nature of intergenerational studies in which the third generation of children is born over many years, the sample size has increased over the years. Finally, as growth was examined in the current study, measurement of G3 behavior was limited to measures available and developmentally appropriate from ages 3 to 9 years – so from preschool to later elementary school, namely the parent version of the CBCL. A more robust construct involving multiple measures and reporters may have shown a stronger association to G2 behavior (and the more comprehensive measurement of G1 and G2 antisocial behavior may account in part for stronger associations across these generations). Nevertheless, the findings of the current study show some similarity to prior intergenerational studies in that associations have been consistently modest. Further effort is required to understand and resolve the very different estimates of family associations in psychopathology that come from different approaches (e.g., prospective intergenerational studies versus cross-sectional twin studies).

All told, the predictors explained limited variance in the outcomes. The amount of variance explained in the intercept for externalizing behavior at age 3 years was 24% for boys but fell below significance for girls at 8%. Prediction to the intercept of internalizing behavior was somewhat stronger, reaching 40% of the variance for boys but only 17% for girls (the latter not significant). For both externalizing and internalizing, a younger age of the mother at birth accounted for a portion of this variance – being a risk factor for boys in both areas of psychopathology.

Findings for prediction of *growth* in externalizing and internalizing through ages 9–10 years for the G3 children did not indicate that higher relative levels of growth was predicted by higher levels of the parallel areas of psychopathology in either the biological parents or paternal grandparents. Thus, it does not appear that intergenerational risks assessed here emerge more strongly across childhood. Again, a further possibility is that measurement of the outcome may be an issue. However, the parent CBCL is a well-validated measure of child psychopathology (Achenbach, 1991), and the patterns of growth across time in the current study are consistent with prior findings regarding the development of externalizing and internalizing behavior. It is possible that reporters with a broader and possibly more objective view, particularly teachers, may provide stronger indices, particularly of externalizing symptoms. However, using such informants restricts the ability to consider growth models prior to the school years.

The only risk factor significantly associated with the slopes of externalizing and internalizing was the contextual control variable of mother's age at birth, and then only for boys. In the case of both externalizing and internalizing symptoms, mothers' *older* ages at birth of their sons were related to higher growth, possibly indicating some recovery of boys with younger mothers from the early negative effects at age 3 years, or conversely that the

protective effects of having an older mother in early childhood dissipated once the child was older. Note that as the mothers were on average around age 23 years at the birth of the children, they were a relatively young group of mothers overall; therefore, risk associated with mother's younger age at birth was likely linked to particularly young ages at birth. These findings should be considered also in the context of the fact that internalizing symptoms were relatively low for boys and showed lower growth than for girls across childhood. A further possibility is that older mothers may be more sensitive observers of internalized affective states in their school-aged sons than are younger mothers.

The G2 men's depressive symptoms and antisocial behavior during late childhood were associated with later risk context for his family of procreation. Each predicted a younger age at parenthood for their partners and lower levels of contact with their child in the first 3 years of the child's life. G2 men's childhood depressive symptoms were also predictive of his partners' higher levels of depressive symptoms in adulthood. For both antisocial behavior and depressive symptoms, the G2 parents showed significant associations in adulthood. In addition, for antisocial behavior, higher levels for each of the G2 parents in adulthood were associated with lower levels of contact with his child, and the G2 mother's antisocial behavior was also related to her younger age at the birth of the child. Thus, some intergenerational risk was transmitted via young age of the G2 mother at the birth of G3, as this was predictive of a higher intercept of both externalizing and internalizing behaviors (for boys only) in G3.

Regarding moderational effects, the gender of the child was a moderator, as transmission pathways were substantively different for boys and girls. Whereas mothers' depressive symptoms showed a similar association to internalizing symptoms at age 3 years for boys and girls, antisocial behaviors in the prior two generations did not confer significant risk for externalizing behaviors at that age for boys, whereas girls showed risk associated with paternal antisocial behavior. Young maternal age at birth was a risk factor for symptoms of both forms of psychopathology at age 3 years for boys, whereas it was not a significant risk factor for girls. In addition to gender, father contact and mother age at birth were also examined for moderational influences on associations of G2–G3 psychopathology. There were no significant interactions on associations of intercept and slope of externalizing for G3. The finding of Jaffee et al. (2003) that children of fathers with higher levels of antisocial behavior had more conduct problems if they had more contact with their fathers was thus not replicated. However, a similar theme was found regarding G3 girls' internalizing behaviors in that higher levels of contact with a more depressed father were associated with higher levels of internalizing behaviors at age 3 years. Girls also were at risk for higher levels of internalizing symptoms at age 3 years if they were born to a younger mother and their father had higher levels of depressive symptoms, and both boys and girls experiencing this combination of risk factors were at higher risk for growth in symptoms across childhood.

Findings from the current study present some challenges to genetic research and analyses. When measured within a relatively strong and ecologically valid study design (i.e., prospectively), the overall heritability of depressive symptoms and antisocial behavior or externalizing does not appear to be strong. Associations appeared to be stronger in adulthood from G1 to G2 associations. This may indicate that genetic influences are stronger during the adult phase of gene expression for these areas of psychopathology. However, much genetic and developmental theory predicts that genetic risk will be manifest in early childhood via temperamental characteristics reflecting externalizing or internalizing problems (Lahey & Waldman, 2003); therefore, associations would be expected at young ages. Further, it has been theorized that, by adulthood, experienced social learning and contextual influences are likely to have had considerable influence, and that genetic

contributions to psychopathology in adulthood might be weaker than those to childhood psychopathology. These intergenerational questions therefore require further investigation.

The current study had a number of design strengths, including prospective data and measurements of psychopathology across three generations and 27 years, and a dynamic developmental approach to examining associations to psychopathology in G3 particularly in that growth in psychopathology was examined from ages 3 to 9–10 years. The study included measurement of psychopathology at overlapping ages (9–10 years) for the G2 father and G3 child. Limitations of the data included the fact that information on the psychopathology of the parents of the G2 mother was not included. However, pathways from the paternal grandparents to G3 outcomes were not significant; thus, strong effects from maternal grandparents' psychopathology would not be expected. Second, the sample size was limited both by the number of children born to the G2 fathers and by their ages (i.e., some were still too young for data at the later ages of childhood). However, the relatively similar pathways among the G1 and G2 constructs for boys and girls in G3, given that no gender differences in these pathways would be expected, seemed to indicate that the model estimates were relatively stable. Finally, although there are members of minority groups in the sample, it is predominantly Euro American; thus, the extent to which the findings would generalize to other ethnic groups within the U.S. is not clear and requires testing in additional samples.

Findings of the current study suggest that it is critical for future research to focus on identifying moderating influences on intergenerational continuities in psychopathology, including interactions within genetic and physiological systems, gene by environment interaction effects, and interactions among contextual and psychosocial factors. There is some indication in this study that associations across generations in psychopathology may be stronger in adulthood. This may indicate that genetic influences are stronger during the adult phase of gene expression for these areas of psychopathology. It may also indicate that other factors, including intergenerational contextual risk, show more evidence in adult behavior and outcomes than in child behaviors. Finally, prediction from G1 psychopathology to G3 psychopathology was not significant for either externalizing or internalizing and was of a modest magnitude for prediction from G2 psychopathology for both outcomes. Overall, these findings suggest that intergenerational risk for these areas of psychopathology may be complex and subject to considerable moderation.

Acknowledgments

The project described was supported by awards from National Institutes of Health, U.S. Public Health Service (NIH) to Dr. Capaldi: Award Number R01 DA 015485 (Adjustment Problems and Substance Use in Three Generations) from the National Institute of Drug Abuse (NIDA); 1R01AA018669 (Understanding Alcohol Use over Time in Early Mid-Adulthood for At-Risk Men) from the National Institute on Alcohol Abuse and Alcoholism (NIAAA); and HD 46364 (Risk for Dysfunctional Relationships in Young Adults) from the National Institute of Child Health and Development (NICHD). The content is solely the responsibility of the authors and does not necessarily represent the official views of the NIH, NIDA, NIAAA, or NICHD. We wish to thank Jane Wilson and Sally Schwader for their contributions.

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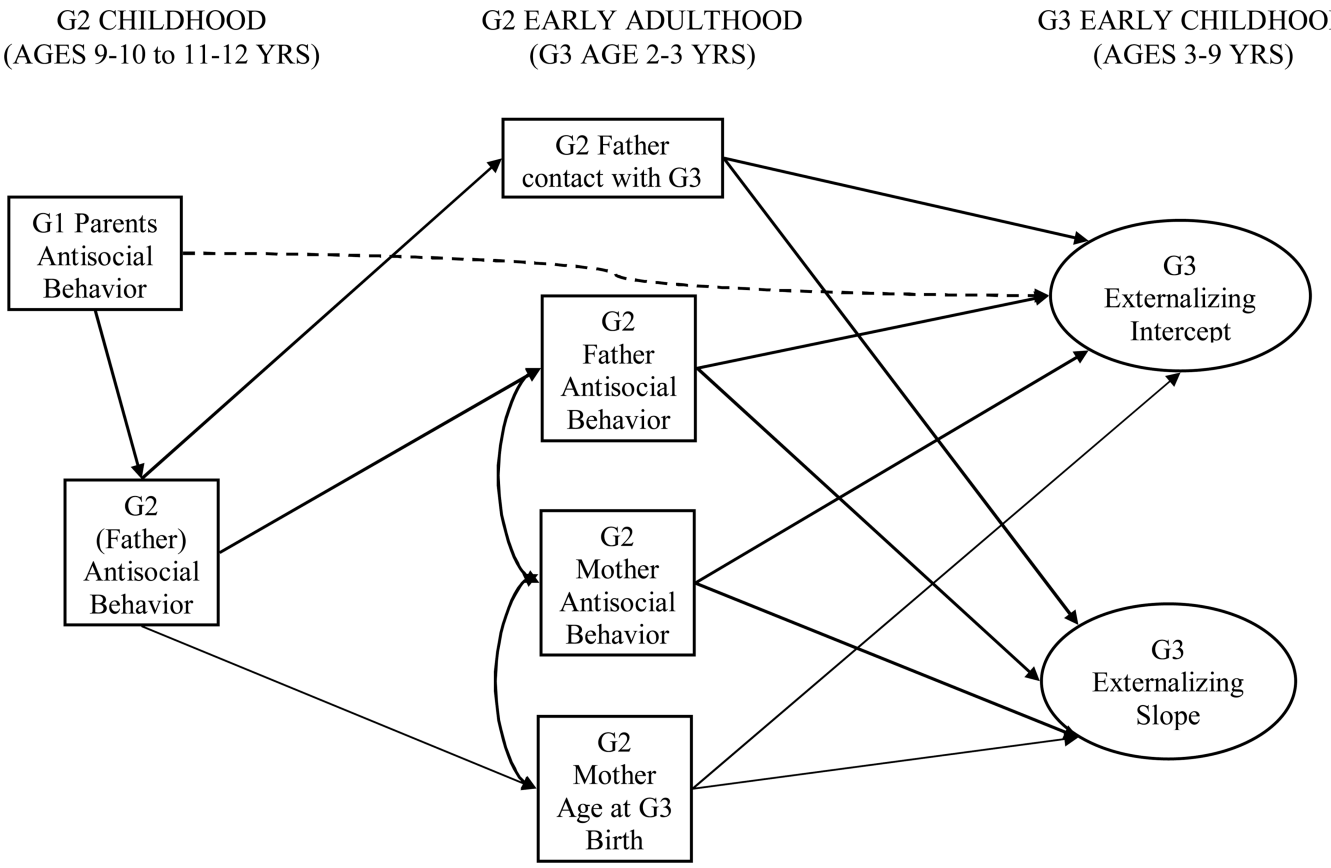

Figure 1.
Externalizing conceptual model.

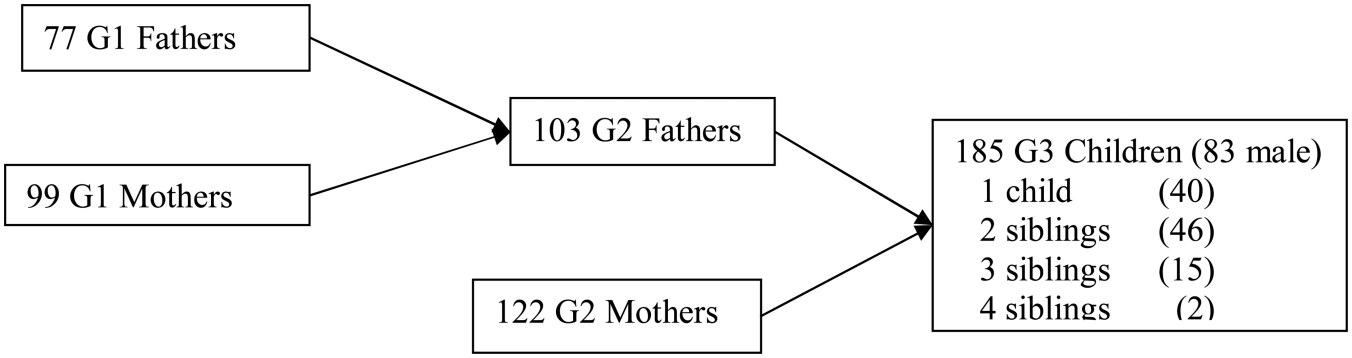

Figure 2.
Diagram of study participants.

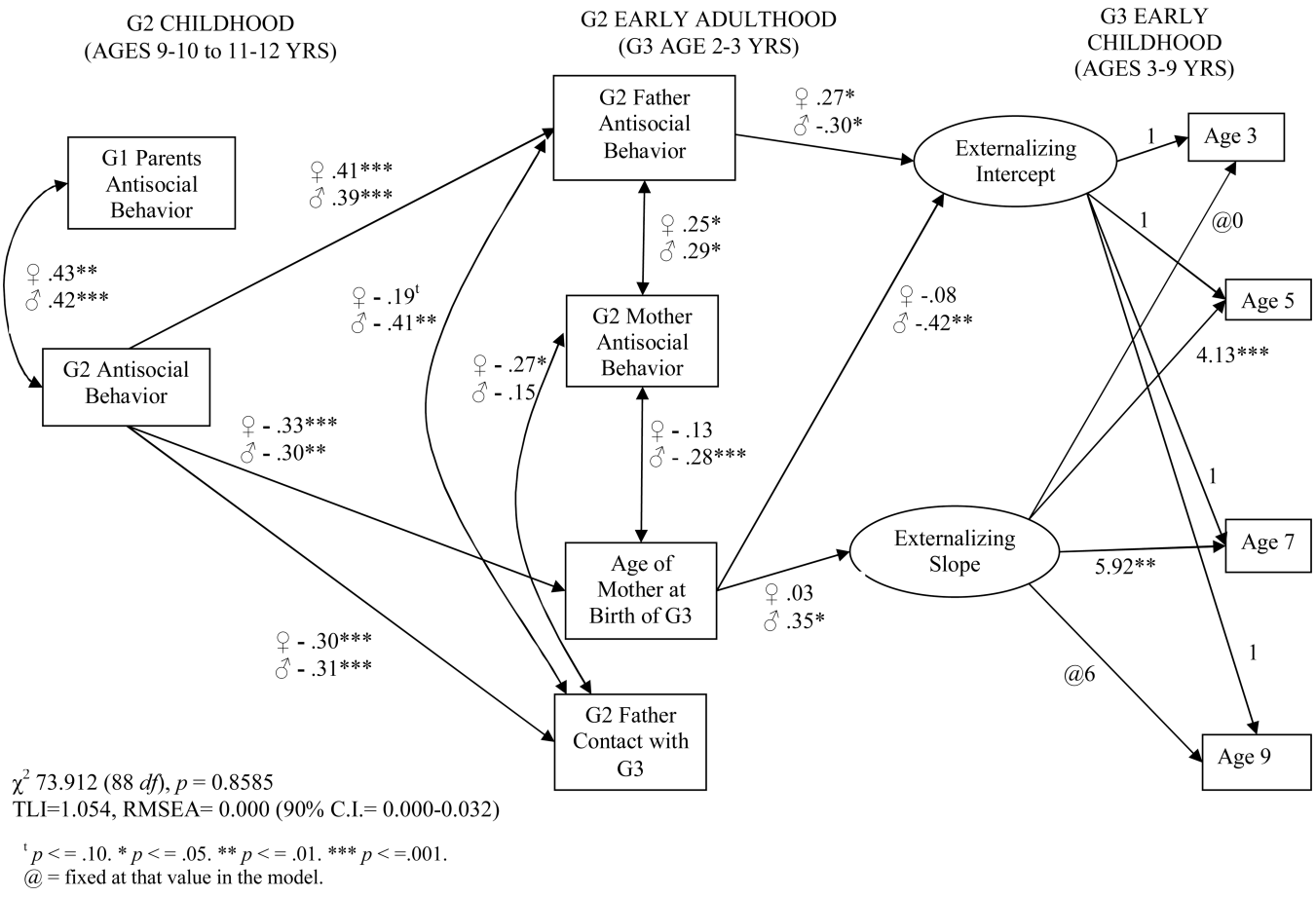

Figure 3.
Prediction model to the intercept and slope of externalizing behavior for the G3 children, grouped by gender.

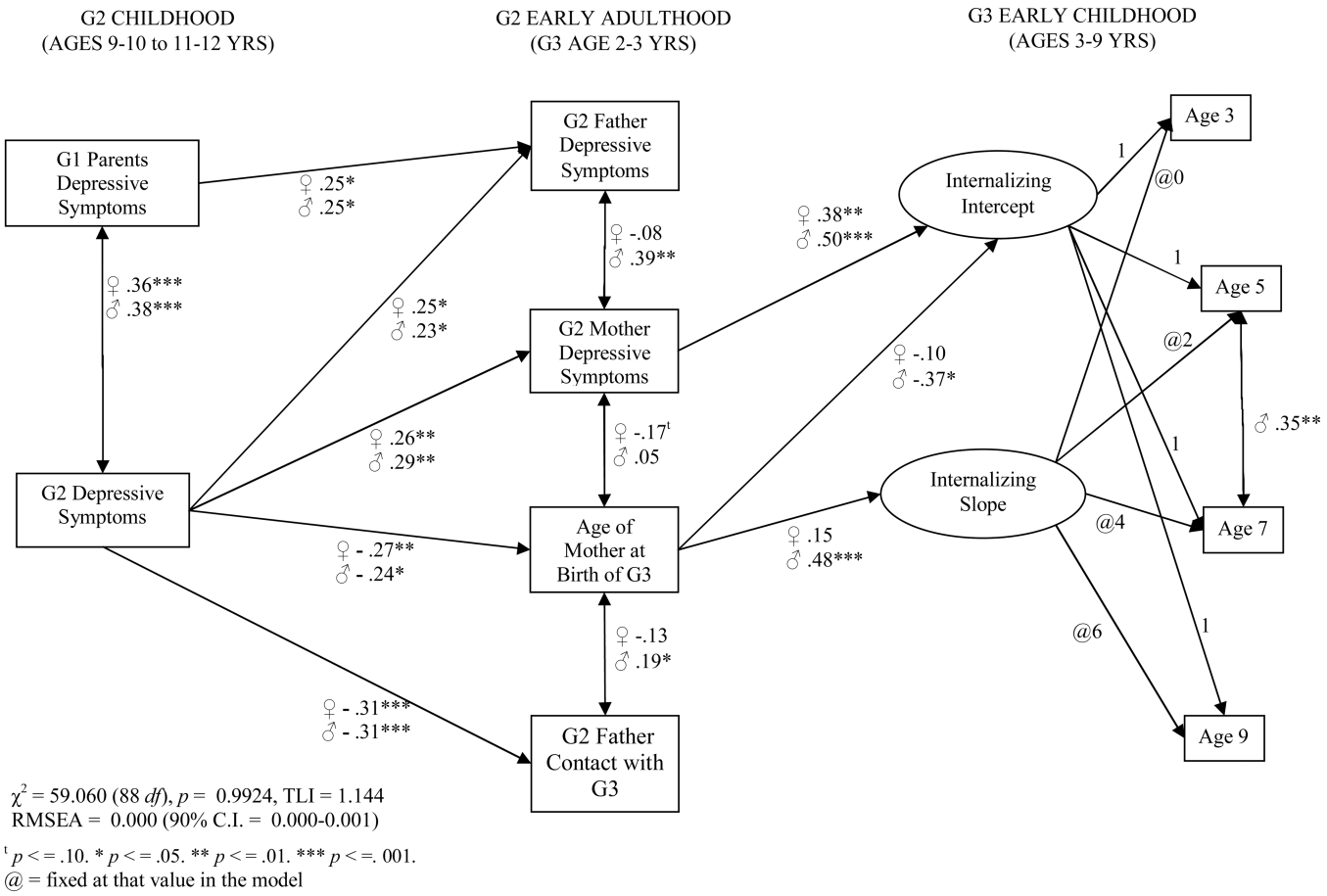

Figure 4.
Prediction model to the intercept and slope of internalizing behavior for the G3 children, grouped by gender.

Table 1

Correlations for Externalizing Model

	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.
1. G1 Antisocial behavior		.29**	−.24*	−.13	.14	−.26*	.07	.09	−.02	−.02	−.07
2. G2 Antisocial behavior (ages 9–12 years)	.51**		−.30**	−.40**	.47**	−.32**	.14	.14	−.14	.08	.23
3. G2 Age at birth of G3	−.15	−.27**		.28**	−.19t	.47**	−.26*	−.13	.08	.05	−.10
4. G2 Father cohabitation with G3	−.12	−.29**	.26**		−.51**	.27**	−.21t	.16	.16	.07	.07
5. G2 Father antisocial behavior in adulthood	.40**	.41**	.01	−.28**		−.26*	.32**	−.16	−.20t	−.02	.04
6. G2 Mother age at birth of G3	−.16	−.35**	.65**	.21*	−.05		−.33**	−.24*	.10	−.17	−.20
7. G2 Mother antisocial behavior in adulthood	.08	.12	−.16	−.29**	.27**	−.14		−.01	−.12	−.07	.12
8. G3 Externalizing (age 3 years)	.19t	.14	−.01	−.06	.28**	−.05	.18t		.28*	.29*	.32*
9. G3 Externalizing (age 5 years)	.17t	.09	−.06	−.07	.18t	−.16	.20t	.54**		.43**	.36*
10. G3 Externalizing (age 7 years)	.10	−.01	−.03	−.16	.16	−.10	.20t	.37**	.59**		.62**
11. G3 Externalizing (age 9 years)	.12	−.04	.07	−.07	.02	.10	.27t	.43**	.64**	.73**	

Note.

Boys are above the diagonal, girls below the diagonal.

** $p <= .01$.

* $p <= .05$.

t $p <= .10$.

Table 2

Growth Models Tested With Model Deviance Comparison Tests

	***N* = 185**	**Externalizing**	**Internalizing**
a	Unconditional correlational model	−21.68	−305.70
b	Single factor	−14.94 (χ^2 (2) = 4.39, p =.11)	−300.27 (χ^2 (2) = 6.74, p =.03)
c	Unspecified 2-factor growth	−17.19 (χ^2 (1) = 24.68, p <.01)	−300.08 (χ^2 (1) = 0.22, p =.64)
d	Equal error variance	−16.90 (χ^2 (3) = 0.30, p =.86)	−293.98 (χ^2 (3) = 5.20, p =.16)
e	Intercept and slope uncorrelated	−14.38 (χ^2 (1) = 2.11, p =.91)	−293.20 (χ^2 (1) = 0.63, p =.43)
f	Linear growth	1.76 (χ^2 (2) = 14.62, p <.01)	−286.74 (χ^2 (2) = 6.48, p =.04)
g	Quadratic growth	−13.10 (χ^2 (1) = 18.04, p <.01)	−287.93 (χ^2 (1) = 1.12, p =.29)
	Grouped by gender	−21.29 (χ^2 (5) = 7.96, p =.16)	−295.29 (χ^2 (5) = 8.91, p =.11)
	Final modified growth model	−33.46 (χ^2 (1) = 8.65, p <.01)	−308.59 (χ^2 (1) = 21.52, p <.01)
	Final model specifics	Unspecified growth (first and last fixed) with error variances different by gender	linear growth; free error variance age 9 and covariance of errors at ages 5 and 7 in boys
	[1]Satorra & Bentler, 2001		

Model Deviance calculation (−2*logliklihood; lower values are better) over scaled[1] χ^2 difference test on change in deviance over previous model

Ordered set of nested model comparisons:

1. All outcomes (four internalizing or externalizing variables) simply allowed to correlate.
2. All outcomes load on single internalizing or externalizing factor.
3. All outcomes load at 1 on an intercept factor, age 3 years outcome fixed at 0 and age 9 years outcome fixed at 6 on growth factor.
4. Fix residual variances of outcome variables as equal across time.
5. Fix covariation between intercept and outcome factors at 0.
6. All outcomes load at 1 on intercept, and outcomes from age 3, 5, 7, and 9 years, at 0, 2, 4, and 6 on slope factor.
7. As in Model 6, but with the addition of age 3, 5, 7, and 9 years, loaded 0, 4, 16, and 36 on quadratic growth factor.
8. Add grouping by gender (model coefficients estimated for males and females separately).
9. Final growth model (including specific constraints and modification detailed in Row 10) used in prediction models.
10. Specific model adjustments Models 9 used to reduce number of estimated parameters and increase growth model fit.

Table 3

Correlations for Internalizing Model

	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.
1. G1 Depressive symptoms		.34**	−.03	−.22*	.47**	−.15	.25*	.09	.05	.03	.07
2. G2 Depressive symptoms (ages 9–12 years)	.39**		−.19t	−.38**	.28*	−.28**	.24*	.11	−.03	−.01	.04
3. G2 age at birth of G3	−.08	−.20*		.28**	−.08	.47**	−.19t	−.11	−.08	−.09	.01
4. G2 Father cohabitation with G3	−.22*	−.25*	.26**		−.32**	.27*	−.22t	.04	.06	−.04	−.01
5. G2 Father depressive symptoms in adulthood	.27**	.36**	−.07	−.14		−.22t	.45**	.15	.05	.09	.04
6. G2 Mother age at birth of G3	−.13	−.26**	.65**	.21*	−.24*		−.08	−.17	−.05	.07	.34*
7. G2 Mother depressive symptoms in adulthood	.15	.30**	−.31**	−.20*	.06	−.24*		.31**	.20t	.27*	.21
8. G3 Internalizing (age 3 years)	.20*	.16	−.10	−.14	−.01	−.13	.22*		.27*	−.01	.06
9. G3 Internalizing (age 5 years)	.19t	.17t	−.17t	−.06	.15	−.07	.25*	.34**		.54**	.50**
10. G3 Internalizing (age 7 years)	.13	.04	−.10	−.19t	.11	−.03	.22t	.31**	.47**		.60**
11. G3 Internalizing (age 9 years)	.06	.14	.03	−.13	−.04	.10	.19	.43**	.46**	.66**	

Note:

Boys are above the diagonal, girls below the diagonal.

** $p <= .01$.

* $p <= .05$.

t $p <= .10$.