Published in final edited form as:

J Abnorm Child Psychol. 2015 January ; 43(1): 163–176. doi:10.1007/s10802-014-9884-7.

Bidirectional Influences of Anxiety and Depression in Young Children

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Abstract

Anxiety and depression tend to co-occur in children. Studies indicate that higher levels of anxiety are associated with subsequent higher levels of depression, while depression may inhibit subsequent anxiety. It is important to increase our understanding of the temporal sequencing of these disorders and, particularly, to determine if suppression effects account for the inhibitory association. In addition, further information about these relationships in young children is needed. Participants were a diverse (20.4 % Hispanic, 16.7 % African American; 49.1 % boys) community sample of 796 children with data available at ages 4, 5, and 6–7 years. Anxiety and depression symptoms were assessed using the Child Symptom Inventory and symptom count measures from the Diagnostic Interview Schedule for Children-Parent Scale-Young Child version. The results indicated: (a) anxiety and depression were relatively stable over time; (b) anxiety at age 4 and 5 was a significant positive predictor of subsequent depression; (c) while an inhibitory effect of depression on subsequent anxiety was found, that inhibitory effect was due to negative suppression, and higher levels of depression were actually associated with subsequent anxiety; (e) consistent with a significant suppression effect, when depression was included as a predictor, the association between anxiety at ages 4 and 5 and anxiety one year later increases in magnitude. Both anxiety and depression are associated with higher levels of one another in the subsequent year. Implications for prevention are discussed.

Keywords

Anxiety; Depression; Bidirectional influences; Suppression effects; Preschoolers

In the last 20–30 years, considerable attention has been paid to the relationship between anxiety and depression in children. Consistent with findings among adults (1992), the authors of several reviews (Brady and Kendall 1992; King et al. 1991; Seligman and Ollendick 1998) have concluded that there is a high level of comorbidity between disorders of anxiety and depression when they are assessed as categorical constructs, and moderate to high correlations between anxiety and depression when measured as symptom dimensions.

This high rate of co-occurrence, has led to a debate about whether anxiety and depression are, in fact, distinct disorders. Results from the few studies using confirmatory factor analysis to examine the latent factor structure of anxiety and depression suggest that viewing them as separate, but correlated, constructs is preferable (Chorpita et al. 1998; Cole et al. 1997, 1998; Snyder et al. 2009; Strickland et al. 2011).

Despite the high rate of co-occurrence, it is important to increase our understanding of the temporal sequencing of these disorders. The clinical implications of any temporal sequence are clear; if one disorder typically precedes the other, then preventive efforts to reduce the development of the subsequent disorder could be beneficial, and understanding the mechanisms by which that developmental progression occurs would improve such efforts.

In reviewing studies on the temporal sequence of anxiety and depression, Cole et al. (1998) have noted that methodological issues limit the conclusions that can be derived from this research. Studies examining the age of onset of anxiety and depressive disorders (Hershberg et al. 1982) have found that anxious children tend to be younger than depressed children, but such studies are not necessarily informative about the sequence with which these symptoms or disorders develop (Cole et al. 1998). Results from studies using within-subject designs with clinical samples (Strauss et al. 1988a, b) suggest that children with comorbid anxiety and depressive disorders were more likely to have developed anxiety disorders first. In community studies of anxiety and depressive disorders, Pine et al. (1998) found that anxiety disorder diagnosed between ages 9 and 18 predicted adult depressive disorders, while depression at age 9–18 predicted adult anxiety, even while controlling for the presence of a comorbid disorder. Costello et al. (2003) found that depression diagnosed in 9–10 year olds was associated with subsequent anxiety, but anxiety was not associated with subsequent depression when controlling for an anxiety disorder at age 9–10. Bufferd et al. (2012) found that preschoolers with anxiety disorders at age 3 were at increased risk of a depressive disorder at age 6 after controlling for initial depressive disorder, but not vice versa. While all three studies controlled for the presence of the comorbid disorder (e.g., when examining anxiety as a predictor of subsequent depression, the presence of an initial anxiety disorder was controlled for), they did not control for the initial level of symptoms of the comorbidity. Thus, anxiety may have been a significant predictor of subsequent depression because the children with anxiety exhibited high levels of depressive symptoms but did not meet full diagnostic criteria for a disorder, and vice versa.

In a third set of studies (Lewinsohn et al. 1995; Reinherz et al. 1989; Reinke and Ostrander 2008; Sanford et al. 1995) with samples of older children and adolescents, anxiety and depression were examined dimensionally rather than categorically. The categorical approach considers traits or disorders to be dichotomous, (i.e., present or not). The dimensional approach assumes that symptoms of a disorder are distributed across a continuum and does not assume that disorders are discrete entities. The issues involved in using a categorical vs. a dimensional approach are thoroughly discussed elsewhere (Beauchaine 2003; Kraemer et al. 2004; Widiger and Samuel 2005). To summarize, proponents of dimensional approaches cite the lack of evidence that many disorders are distributed as discrete classes rather than dimensionally, and that the high rates of comorbidity among DSM-IV disorders suggest they are not discrete entities (Kraemer et al. 2004; Widiger and Samuel 2005). Kraemer et al. (2004) note that: (a) the approaches are fundamentally equivalent; (b) which approach to use depends upon the question being asked; and (c) dimensional approaches are generally to be preferred when engaged in hypothesis-testing because of the loss of power associated with the use of categorical variables.

While data from these dimensional studies indicate that anxiety at a younger age predicts depression at a later age, none of these studies controlled for the prior level of symptoms of depression (Cole et al. 1998). Similarly, Moffitt et al. (2009) found no difference in the rate at which older children and adolescents with major depressive disorder (MDD) developed anxiety concurrently or later when compared to the rate at which older children and adolescents with generalized anxiety disorder developed MDD concurrently or later. In all of these studies, the findings may merely have reflected the well-known correlation between depression and anxiety present in the younger age group.

To date, three studies (Cole et al. 1998; Keenan et al. 2009; Snyder et al. 2009) of children or adolescents have tried to move beyond the limitations of the prior reports. All three are longitudinal studies conducted with community samples, thereby reducing overrepresentation of comorbidity of anxiety and depression that can occur in clinical samples (Caron and Rutter 1991; Cole et al. 1998), and all three included dimensional measures of anxiety and depression to allow for the assessment of anxiety-depression relationships across the full range of such symptoms.

Cole et al. (1998) examined the stability of symptoms of depression and anxiety between grades 3 and 6. Results indicate that the stability of anxiety and of depression was high for models using either parent or child reports. In addition, the cross-lag path coefficients of anxiety with subsequent depression were significant for both parent and child reports, indicating that higher levels of anxiety were associated with subsequent higher levels of depression, even after controlling for prior level of depression. Unexpectedly, parent-reported child depression predicted *less* anxiety subsequently, which Cole et al. considered to be a possible inhibitory effect in which higher levels of anxiety actually reduce subsequent levels of depression.

In the second study, Snyder et al. (2009) used linear growth models to examine the bidirectional effects of anxiety and depression symptoms at 6-month time intervals between ages 5.3 and 9.3 years. Consistent with Cole et al.'s (1998) findings, higher levels of parent-

rated anxiety symptoms were associated with higher levels of subsequent depression symptoms in three of five time periods, and higher levels of depression were associated with lower subsequent anxiety levels in two time periods. A similar pattern was noted for teacher reports. Like Cole et al., they concluded that anxiety symptoms had a facilitative relationship with subsequent depression, while depressive symptoms had an inhibitory relationship with subsequent anxiety. Of note, when models included cross-lagged paths, the autocorrelations (i.e., from anxiety at time X to anxiety at time X+1, and depression at time X to depression at time X+1) were not significant. The absence of significant autocorrelations differs from the results of most other studies.

In the third study, Keenan et al. (2009) examined the relationship between anxiety and depression based on care-giver reports provided annually for girls ages 6–12 years. In one set of analyses, Keenan et al. examined the relationship between symptoms of depression and separation anxiety; in the second set of analyses, they examined the relationship between depression symptoms and combined generalized and social anxiety symptoms. The stability of depression, separation and generalized/social anxiety were all high, and half of the paths from separation anxiety to depression and generalized/social anxiety to depression were significant. In contrast, the relationship between depression and both subsequent separation anxiety and generalized/social anxiety was significant for only 1 of 12 paths. They concluded that: (a) anxiety and depression were moderately stable; (b) anxiety was associated with subsequent depression; and (c) information about depression did not improve prediction of subsequent anxiety.

The results of these studies are similar to one another in certain important respects. All three found significant homotypic continuity over time for anxiety and depression, suggesting some stability and predictive ability for anxiety with subsequent anxiety and depression with subsequent depression. All three are also consistent in finding that anxiety is associated with subsequent depression. Two (Cole et al. 1998; Snyder et al. 2009) of the three studies, however, found that depression may sometimes have an inhibitory relationship with subsequent anxiety, while the third found no strong relationship between depression and subsequent anxiety. An important feature of the third study (Keenan et al. 2009), however, is that the correlations between depression and anxiety were small (0.15–0.19). Correlations between anxiety and depression of such a low magnitude are unusual in this literature and raise concerns about the generalizability of the findings.

Similarly, there is a statistical issue with the Cole et al. and Snyder et al. studies that could account for the unexpected result that depression may lead to less subsequent anxiety, thus casting doubt on the conclusion that depression inhibits later development of anxiety. Specifically, it is possible that this is a spurious finding due to suppression effects that are obscuring the true relationship between depression and later anxiety. A suppressor variable increases the predictive validity of another variable (or set of variables) when included in a regression equation (Conger 1974). Suppression may be present when: (a) the absolute value of the path coefficient of a predictor is greater than its bivariate correlation with the criterion, or (b) the two have different signs (Kline 2011). Suppression can result in a “surprise” relationship (Kline 2011) between the predictor and the criterion (e.g., in the regression analysis, depression at time X is negatively correlated with anxiety at time X+1,

but the bivariate correlation between depression at time X and anxiety at X+1 is positive). Suppression is more likely to occur when there is a strong association between predictor variables (Gaylord-Harden et al. 2010).

Suppression can take several forms. In negative suppression, predictor 1 shows either a positive or negative association with the criterion variable in a regression equation that does not include predictor 2. When predictor 2 is added to the regression equation, however, the sign of the path coefficient for predictor 1 changes direction, becoming the opposite of the sign of the path coefficient in the equation that did not include predictor 2. In addition, when negative suppression occurs, the magnitude of the path coefficient for predictor 2 increases compared to the magnitude of the predictor 2 path coefficient found in a regression equation that did not include predictor 1. Of course, other patterns of change in predictive power can occur that are not suppressive effects. For example, redundancy can be observed when the predictive power of two correlated predictors both decline when both are entered into the regression analyses (Paulhus et al. 2012).

In the Cole et al. (1998) report, there are indications that suppression effects may be occurring. In that report, the predictors are anxiety and depression at time X, and the criterion is anxiety at time X+1. While the path coefficient for anxiety at Time 1 and anxiety at Time 2 were not reported, the correlations between the measured indicators of anxiety at Time 1 and Time 2 were significant and positive. Similarly, the correlation between depression at Time 1 and anxiety at Time 2 also was positive. When both were examined as simultaneous Time 1 predictors of Time 2 depression, the path coefficient from depression was negative, suggesting a negative suppression effect. It is also likely that suppression was affecting the Snyder et al. (2009) results because the autocorrelations that had been significant and positive were no longer significant when the cross-lagged relationships were included. Because the magnitude of the nonsignificant autocorrelations in the cross-lagged model, and the correlations between cross-lagged variables were not reported (e.g., anxiety at time X with depression at time X, depression at time X with anxiety at X+1, etc.), it is not clear whether suppression (the magnitude of a variable increases as the direction of the sign of the other changed direction) or collinearity (two correlated variables reducing the magnitude of the relationship between *each* predictor with the outcome) was operating.

Awareness of suppression can do more than allow the researcher to detect problems in a model; it can contribute to a better understanding of the phenomenon being studied (Gaylord-Harden et al. 2010; Paulhus et al. 2012), particularly when suppression effects can be replicated (Paulhus et al. 2012). Gaylord-Harden et al. suggest that attempts to control for suppression statistically are likely to be less productive than examining the patterns by which suppression is having an effect. To determine whether suppression effects are present and what their significance may be, Gaylord-Harden et al. recommend examining components of the model (i.e., the relationship of each latent predictor factor, or combinations of factors, with the criterion) separately and comparing these responses to those when multiple factors are examined.

The aim of the present study was to shed more light on the temporal sequencing of symptoms of depression and anxiety in young children, by using structural equation

modeling (SEM) to examine bidirectional effects of symptoms of anxiety and depression, and to determine if suppression effects contributed to the inhibitory association of depression with subsequent anxiety noted in prior work. Based on the prior literature, we hypothesized that (a) anxiety and depression would be reasonably stable from preschool to early grammar school years (ages 4 to 6 years); (b) in models including bidirectional influences, higher levels of anxiety would be associated with higher levels of depression subsequently; (c) while models including bidirectional influences might reflect an inhibitory effect of depression on subsequent anxiety, subsequent analyses would show that this finding was due to suppression effects, and that higher levels of depression were associated with higher levels of subsequent anxiety.

The present study retained the strengths of studies by Cole et al., Keenan et al., and Snyder et al. in the following ways: (a) using a community sample to avoid over-representation of comorbidity; (b) examining anxiety and depression dimensionally in order to assess the whole range of symptoms; (c) assessing symptoms at multiple time points; and (d) using SEM to examine combinations of cross-lags and autocorrelations while estimating measurement error. In addition, we also used multiple indicators of the latent constructs of anxiety and depression (unlike Cole et al. and Snyder et al. who used a single indicator to assess each latent factor). Suppression effects may be underestimated due to measurement error (Holmbeck et al. 2002), and in designs using a single measure, it is not possible to estimate measurement error.

Finally, another strength of the present study is that we examined the longitudinal relationships between depression and anxiety in younger children (ages 4, 5, 6), during the transition from preschool through kindergarten and into early grammar school. Examining the temporal relationships between these disorders as they first begin to develop in preschool children will help to clarify the developmental progression of these disorders. The presence of anxiety disorders in young children has long been recognized (Egger and Angold 2006; Lavigne et al. 2009), and there is increasing awareness of the occurrence of depressive symptoms and disorder in young children as well (Luby et al. 2002, 2003, 2006). Furthermore, there is considerable evidence for the stability of symptoms of childhood anxiety and depression (Harrington et al. 1996; Ollendick and King 1994) over time. Studies documenting this stability often include a small number of preschool children in samples primarily consisting of older children; however, far fewer studies have documented the stability of anxiety and depression symptoms or disorders that only included children who were preschoolers at study onset. There is also growing evidence for the long-term stability and clinical significance of preschool depression. Studies by Luby and colleagues indicate that depression occurs in preschool children (Luby et al. 2002, 2003), impairs the child's functioning (Luby et al. 2009a) and is relatively stable for at least 24 months (Luby et al. 2009b); thus, it is not a developmentally transient phenomenon. Examining the temporal relationships between anxiety and depression as they first begin to develop in preschool children will help to clarify the developmental progression of these disorders.

Method

Participants

Data were collected as part of a longitudinal study of the development of symptoms of psychopathology among 4-year-old preschool children as they transitioned into early grammar school, ages 6–7 years. A diverse sample of 796 4-year-old children was recruited from 13 Chicago Public School preschool programs and 23 primary care pediatric clinics located throughout Cook County, Illinois. At the time of the initial interview, eligible children: (a) were 4 years of age; (b) spoke English or Spanish; (c) had lived with the parent who participated in the study for at least 6 months; (d) obtained a standard score on the Peabody Picture Vocabulary Test 70 (Dunn and Dunn 1997); (f) did not meet criteria for an Autism Spectrum Disorder, were not enrolled in a special education class for children with developmental delays or intellectual disabilities and did not have a school IQ test score below 70.

Of the initial sample of 796 4-year-olds (mean age=4.44, $SD=0.32$), there were 391 (49.1 %) boys and 405 (50.9 %) girls. Parent-reported racial/ethnic group membership included: 433 (54.4 %) White, non-Hispanic; 133 (16.7 %) African American; 162 (20.4 %) Hispanic; 19 (2.4 %) Asian; and 35 (4.4 %) multi-racial or “Other.” Race/ethnicity was not reported by 14 (1.8 %) parents. All social classes (Hollingshead 1975) were included, with 303 (38.1 %) children in the highest class (Class I), 290 (36.4 %) in Class II, 79 (9.9 %) in Class III, 63 (7.9 %) in Class IV, and 61 (7.7 %) in Class V. Other details about the age-4 sample are available (Lavigne et al. 2009). At the Time 2 visit, the mean age was 5.11 years ($SD=0.35$); at Time 3, the mean age was 6.20 ($SD=0.46$).

There were 627 children and parents (78.8 %) who participated in all three waves of data collection and had valid data for the main measures used in the present study. The sample of parents and children who completed all three waves of data collection differed from those who did not with respect to: (a) race, with a greater proportion of minority participants dropping out, $\chi^2(5, N=(796))=77.7, p=0.001$; (b) SES, with a greater proportion of lower SES groups dropping out, $\chi^2(4, N=(796))=69.61, p=0.001$; and (c) age, with those who dropped out being on average 25 days older at study entry, $t(773)=2.41, p=0.02$.

Before conducting the analyses, missing data and item frequencies were examined separately within each age group. Within each of the three age groups, less than 5 % of the data were missing. Because data were not missing completely at random (MCAR; Little's test, $\chi^2(137, N=796)=246.53, p<0.001$), listwise deletion is less appropriate than imputing the missing data (Graham 2009). Missing data were imputed using SPSS V15.0 Expectation Maximization methodology which uses maximum likelihood procedures utilizing all study variables (child age, sex, race/ethnicity, SES, all anxiety and depression symptom measures for all 3 age groups) to estimate values. With the imputation, the final sample N was 796.

Measures

Demographics—Parents completed a demographic questionnaire to provide information concerning the child's age, race/ethnicity, and parental education and occupation

information which was coded for socioeconomic status using the Hollingshead Four-Factor Index of Social Status (Hollingshead 1975).

Child Psychopathology: Depression and Anxiety

While a multi-informant approach to studying developmental psychopathology is desirable, it is difficult to use this approach in studying preschoolers. Preschoolers are too young to reliably provide self-reports, and it is too difficult for observers to provide ratings across the wide range of situations that would be necessary to determine a true rating of a child's anxiety and depression. Teacher ratings can be useful, but parent and teacher ratings differ substantially most likely as a result of situation-specific behaviors in children (Drabick et al. 2007, 2011). As a result, researchers have argued that parent and teacher ratings should be examined independently. While there has been concern about the depression-distortion hypothesis, in which parent ratings of their child's behavior are thought to be influenced by parental depression, there is little evidence to support this (Richters 1992). In addition, no research presently indicates that parental affect affects ratings of anxiety and depression differentially. Given these limitations, the present report utilized parent ratings of child anxiety and depression, recognizing that patterns might look different if teacher ratings were used.

Depression—A structured parent interview and parent-reported questionnaires assessing symptoms of child depression were used as the three manifest indicators of the latent depression factor. These included: (a) the symptom count measure from the depression scale of the Diagnostic Interview Schedule for Children-Parent Scale-Young Child version (Fisher and Lucas 2006), test-retest reliability=0.57. The DISC-YC was administered by trained graduate students who were taught to use the computer-based interview by DISC-trained staff. They practiced conducting interviews until they were able to reproduce the skip patterns used by other trained interviewers, a procedure recommended by Shaffer, Fisher, Lucas, Dulcan, & Schwab-Stone (Shaffer et al. 2000). Parents completed the major depression scale of the Child Symptom Inventory (CSI) (Gadow and Sprafkin 1997, 2000), a parent-report problem behavior checklist with items derived from DSM-IV diagnostic criteria (alphas=0.70–0.80); and (c) the CSI dysthymia scale, alpha=0.74. The CSI has excellent convergent, divergent and discriminant validity (Sprafkin et al. 2002). Alphas for the depression measure in this sample were 0.74 at age 4, 0.72 at age 5, and 0.72 at age 6–7.

Anxiety—The three manifest indicators used to estimate the latent factor of symptoms of child anxiety included: (a) the symptom count measure from the generalized anxiety scale of the DISC-YC; and (b) the generalized anxiety and separation anxiety scales of the CSI. Alphas for the anxiety measure in this sample were 0.77 at age 4, 0.78 at age 5, and 0.80 at age 6–7.

Procedure—Research assistants approached parents at pediatric offices and preschools and informed them about the study. Questionnaires were mailed to interested parents, and completed either before or during a home visit. At the home visit, the PPVT-III was administered. Parents were then re-contacted approximately 1 and 2 years after the initial visit for a home visit resulting in collecting Time 1 data at age 4 (Time 1), Time 2 at age 5,

and Time 3 at age 6 or, for a few children, at age 7 years. Written consent to participate was obtained each year. This study was approved by the appropriate Institutional Review Boards.

Data Analysis

In all of the tested models, errors associated with the measured indicators were allowed to correlate for the same measure (e.g., the CSI anxiety scale at age 4, 5, and 6–7; the CSI depression scale at age 4, 5, and 6–7, etc.) across the three assessment periods. In addition, we followed the procedure of Cole et al. (1998) in allowing the residuals of anxiety and depression to covary within each time period. All paths were set to be free, allowing the magnitude of similar path coefficients to vary across time periods.

In assessing model fit, we felt that a moderately good fit was acceptable for two reasons. First, we did not anticipate that overall model fit would be very strong because other factors (e.g., family factors, child temperament, etc.) that might contribute to the development of anxiety and depression alone were not included in the models. Second, when examining components of the overall model, some important paths might be excluded (e.g., autocorrelations were not included in each model). As a result, overall model fit might not be ideal. Because our interest was in examining the strength of particular relationships between anxiety and depression rather than in generating a full model of factors associated with anxiety and depression, a moderately good model fit seemed acceptable.

In assessing overall goodness of fit, we followed Brown's method (Brown 2006) in reporting a χ^2 but not interpreting its value because it is inflated by large sample sizes. Multiple fit indices were used, including an index adjusting for model parsimony (root mean square error of approximation; RMSEA), and comparative fit indices (non-normed fit index, NNFI, and comparative fit index, CFI). Criteria for good-fitting models were: RMSEA approximately 0.06 or lower for a good fit and approximately 0.08 for a moderately good fit, NNFI 0.95, CFI > 0.95. Completely standardized path coefficient weights are included in the figures and text; values of such path coefficients can exceed one in magnitude (Joreskog 1999). Following Kline (1998), we describe path coefficients as small (< 0.10), medium (around 0.30), and large (> 0.50). LISREL 08.8 and SPSS 20.0 were used to conduct the analyses. To evaluate the statistical significance of differences in goodness-of-fit between nested CFA models in invariance testing, we used a modified version of the *SB* χ^2 (Satorra and Bentler 2001) that yields a more accurate scaled difference test for LISREL (Bryant and Satorra 2012).

Results

Preliminary Analyses: Means, Ranges and Correlations Between Measured Indicators and Latent Factors

Scores on each of the scales are presented in Table 1. Children in the sample showed a wide range of scores on each scale. While mean scores for the normative samples for the CSI and DISC are not available, the norms manual (Gadow and Sprafkin 1997) for the CSI reports the number of children in the age 3–6 sample exceeding a screening cutoff (two or more

symptoms occurring *often* or *very often*) for generalized anxiety and SAD. For both types of problems, the prevalence at that level in the normative sample was 4 %. In this sample, for GAD it was somewhat higher: 15 % at age 4, 11.8 % at age 5, and 10.4 % at age 6. For SAD, the prevalence of 2+ “often” or “very often” scores was very similar: in this sample, it is 06.3 % at age 4, 4.3 % at age 5, and 3.1 % at age 6.

For depression and dysthymia, the norms manual reports the percent of children with a symptom count ≥ 1 rather than the percent of children who screened high. In the normative sample, 2.8 % had at least 1 depression symptom. In this sample at least one depressive symptom was reported for 3.1 % of the children at age 4, 3.1 % at age 5, and 4.1 % at age 6. For dysthymia, 1.7 % of the normative sample reported at least one depression symptom; in this sample, 1.4 % at age 4, 1.0 % at age 5, and 2.1 % at age 6 reported at least one depression symptom.

The correlations among the measured indicators of anxiety and depression are presented in Table 2. The results indicate: (a) at each time period, the measured indicators of anxiety and depression are positively, and significantly, correlated with one another; (b) between each time period and the subsequent time period, the autocorrelations are all positive and significant, (i.e., each indicator of anxiety at Time 1 correlates with each indicator of anxiety at Time 2, etc.); (c) the measured indicators for each cross-lag (i.e., anxiety at Time 1 with depression at Time 2, depression at Time 1 with anxiety at Time 2, etc.) are all positively and significantly correlated.

Longitudinal Models

Stability of Latent Constructs: Anxiety and Depression Modeled as

Autocorrelated, but Unrelated Factors—Model 1 posits that anxiety and depression are both autocorrelated but unrelated to one another across time periods, i.e., that anxiety at time X is correlated with anxiety at time X+1, and depression at time X is correlated with depression at time X+1, but paths from a disorder at time X to the other disorder at time X +1 were not included in this model. Model 1 was examined to assess the stability of the latent constructs and to illustrate the magnitude of the autoregressive path coefficients when the cross-lagged paths (from anxiety to subsequent depression, and vice versa) were not included. A preliminary analysis to illustrate the direction and magnitude of the autoregressive effects without cross-lagged paths is necessary to determine if suppression effects are present in subsequent analyses. Because prior studies have shown that the cross-lagged path coefficients are significant, we did not anticipate that the fit for this model would be good; rather, it was included because the information about the autoregressions is important for the aims of the present study (i.e., to determine if suppression could account for some previous findings).

As expected, this model (Fig. 1) showed relatively poor fit overall, $SB\chi^2(116)=900.46$, $p<0.001$, $RMSEA=0.09$, $NNFI=0.95$, $CFI=0.97$), meeting criteria on 2 of the 3 indicators. Completely standardized path coefficients for latent anxiety factors were large for both Time 1 to Time 2, 0.76 ($p<0.001$) and for Time 2 to Time 3, 0.80 ($p<0.001$). Completely standardized path coefficients for depression were large for Time 1 to Time 2, 0.54 ($p<0.001$) and for Time 2 to Time 3, 0.52 ($p<0.001$).

Anxiety and Depression Modeled as Autocorrelated, With Bidirectional Cross-Lags—In Model 2 (Fig. 2), anxiety and depression were examined as having both autoregressive (i.e., anxiety at Time 1 was associated with anxiety at Time 2, etc.) and cross-lagged associations at the subsequent time period (i.e., anxiety at Time 1 was associated with depression at Time 2, and depression at Time 1 was associated with anxiety at Time 2, etc.).

Model 2 showed a moderately good fit, $SB\chi^2(112)=743.64$, $p<0.001$, $RMSEA=0.08$, $NNFI=0.96$, $CFI=0.97$. Goodness of fit was better for Model 2 than Model 1, $SB\chi^2(4)=115.45$, $p<0.001$. In this model, anxiety at time X was a statistically significant, large predictor of anxiety at time X+1, with a positive association between anxiety at time X and X+1. This is consistent with the results for anxiety in the autoregressive Model 1. In contrast, while depression at time X was a large, positive predictor of depression at time X+1 in model 1, depression at time X became a large, negative predictor of depression at time X+1 in Model 2.

In the cross lagged paths, anxiety at time X was a large, positive predictor of depression at time X+1. This is consistent with the positive correlations between anxiety at time X and depression at time X+1 (see Table 1). In contrast, depression at time X showed a strong negative association with anxiety at time X+1, while the correlations of time X depression and time X+1 anxiety were positive. The finding that depression at time X appears to have an inhibitory effect on time X+1 anxiety is consistent with the findings of Cole et al. and Snyder et al. The reversals of the signs in the bidirectional model, in which both anxiety and depression at time X predicted anxiety and depression at time X+1, raise the possibility of suppression effects rather than a true inhibitory effect in which depression decreases subsequent anxiety.

Cross-lag Model Without Autocorrelations—Following Gaylord-Harden et al.'s (2010) procedure for examining suppression effects, an additional model (Model 3; Fig. 3) that included the bidirectional cross-lagged paths without including autocorrelations was then examined. Comparisons of the relationships between anxiety and depression in the three models allowed for comparisons of predictors of anxiety and depression as predictors of subsequently anxiety or depression separately as well as simultaneously.

As expected, exclusion of the autocorrelations in Model 3 resulted in a somewhat poorer fit for the overall model, $SB\chi^2(116)=1048.56$, $p<0.001$; $RMSEA=0.10$, $NNFI=0.95$, $CFI=0.96$), but results for specific paths could be examined. Completely standardized path coefficients for anxiety at Time 1 to depression at Time 2 (0.56, $p<0.001$) and for anxiety at Time 2 to depression at Time 3 (0.52, $p<0.001$) were statistically significant and large in magnitude; path coefficients were significant and large for depression at Time 1 to anxiety at Time 2 (0.67, $p<0.001$) and from depression at Time 2 to anxiety at Time 3 (0.61, $p<0.001$). Rather than having an inhibitory association between depression and subsequent anxiety, this model suggests that there is a bidirectional influence in which higher anxiety levels are associated with higher depression levels subsequently, while higher depression levels also predict higher anxiety at each subsequent time.

Examining Suppression Effects—To examine suppression effects, differences in the associations between two predictors at time X with the outcome at time X+1 are examined when modeled separately and when both are included in the model. In negative suppression two predictors both have a positive relationship with an outcome when examined separately. When both are included as predictors simultaneously, however, the magnitude of one predictor increases, while the sign of the other becomes negative. This occurs for anxiety and depression at Time 1 as predictors of anxiety at Time 2.

We first examined suppression effects when Time 1 anxiety and depression were predictors of Time 2 anxiety. When Time 1 anxiety is a predictor for Time 2 anxiety without including Time 1 depression as a predictor, the standardized path coefficient of anxiety at Time 1 as a predictor of Time 2 is 0.80. When anxiety and depression at Time 1 were both included as predictors of Time 2 anxiety (as in Model 2), the path coefficient for Time 1 anxiety predicting Time 2 anxiety increased to 3.01. While the path coefficient for Time 1 depression as a predictor of Time 2 anxiety was positive when it was the sole predictor of Time 2 anxiety (0.67), that path coefficient became negative when both Time 1 anxiety and depression predicted Time 2 anxiety (−2.33, model 2). This pattern is consistent with negative suppression. The Sobel test, which can be used to examine the magnitude of the increase in the path coefficient for Time 1 anxiety predicting Time 2 anxiety, was significant (Sobel $Z=4.59$, $p=0.0001$).

Negative suppression was also present for the prediction of Time 3 anxiety from Time 2 anxiety and depression. The path coefficient for Time 2 anxiety predicting Time 3 anxiety without including Time 2 depression was 0.77; when depression at Time 1 is included, the path coefficient for Time 1 anxiety with Time 2 anxiety increased to 2.77. When Time 2 depression was the sole predictor of Time 3 anxiety, its value was positive (0.61), but became negative (−2.06) when Time 2 anxiety was also included. The increase in the path coefficient for Time 2 anxiety to Time 3 anxiety was significant (Sobel $Z=3.11$, $p=0.002$). The pattern of suppression described above appears to be present in the two prior reports.

In the present study, a second pattern of suppression was also evident when time X anxiety and depression predict subsequent depression at time X+1. When Time 1 depression was the sole predictor of Time 2 depression, its path coefficient was positive (0.54). When Time 1 anxiety was the sole predictor of Time 2 depression, its path coefficient was also positive (0.56). When both were entered as predictors of Time 2 depression, the path coefficient for Time 1 anxiety as a predictor of Time 2 depression increased significantly to 3.07 (Sobel $Z=4.09$, $p=0.0001$), and the path coefficient for Time 1 depression with Time 2 depression became negative (−2.45). When Time 2 depression was the sole predictor of Time 3 depression, its path coefficient was positive (0.52). When Time 2 anxiety was the sole predictor of Time 3 depression, its path coefficient was also positive (0.52). When both were entered as predictors of Time 3 depression, the path coefficient for Time 2 anxiety as a predictor of Time 3 depression increased significantly to 2.60 (Sobel $Z=2.98$, $p=0.003$), and the path coefficient for Time 2 depression with Time 3 depression became negative (−2.01).

Discussion

The aims of this study were to examine: (a) the longitudinal stability of symptoms of depression and symptoms of anxiety in a sample of younger children than has been studied previously; and (b) to determine if higher levels of symptoms of anxiety were associated with higher levels of subsequent symptoms of depression; and (c) if the inhibitory effects of depression on subsequent anxiety noted in prior studies may be attributable to suppression effects. There are three previous studies (Cole et al. 1998; Keenan et al. 2009; Snyder et al. 2009) that have examined the longitudinal relationship between depression and anxiety while controlling for initial levels of these types of symptoms in older children. Each of these studies indicated that there is an autoregressive effect in which anxiety predicts subsequent anxiety, and depression predicts subsequent depression. The stability of each type of symptom over time, sometimes referred to as homotypic continuity, was high in all three studies. Similar to the results with older children, the present results indicate that the homotypic stability of both anxiety and depression in younger children is also high. This finding extends our knowledge of the stability of both depression and anxiety over time to an important developmental period.

Prior longitudinal studies have differed somewhat in their conclusions about heterotypic continuity, i.e., the relationship between each construct with the other at a subsequent point in time. Both Cole et al. (1998) and Snyder et al. (2009) concluded that a high initial level of anxiety was an important predictor of subsequent depression. In contrast, Keenan et al. (2009) did not find anxiety to be an important predictor of subsequent depression.

The results of the present study are consistent with the findings of Cole et al. and Snyder et al. with regard to high initial anxiety as a predictor of subsequent depression. In the present study, when anxiety was modeled as a predictor of subsequent depression while including an estimate of initial depression, there was a strong, positive association between anxiety at Time 1 and 2, and depression at Time 2 and 3, respectively. The results of this study, and those of Cole et al. and Snyder et al., differ from those of Keenan et al. A key difference in the findings across these studies is that the measures of anxiety and depression used in the Cole et al., Snyder et al., and the present study were all moderately to highly correlated, while those in the Keenan et al. study showed low correlations. The pattern of findings suggests that, when the measures of anxiety and depression are moderately to highly correlated when measured contemporaneously at the first assessment, anxiety will emerge as a strong predictor of subsequent depression, possibly stronger than the initial level of depression. However, if the measures of anxiety and depression are not moderately or highly correlated, or perhaps if specific types of anxiety or depression are assessed, then anxiety may not be a strong predictor of subsequent depression.

In prior reports, both Cole et al. and Snyder et al. found evidence supporting an inhibitory association between depression and subsequent anxiety in children, suggesting that initial higher levels of depression may result in lower levels of subsequent anxiety. In those studies, the total score on the depression measure was associated with the total score on the subsequent anxiety measure. The results of the present study suggest that the negative association of total depression scores with subsequent anxiety is associated with negative

suppression. Although suppression effects are often treated as statistical artifacts whose effects should be eliminated, researchers are increasingly recognizing that careful analysis of suppression effects can enhance the understanding of underlying theory about the relationship between constructs (Gaylord-Harden et al. 2010; Paulhus et al. 2012). This is particularly true when the suppression effects are replicable across studies, as they are for the association of depression with subsequent anxiety in this and in Cole et al. and Snyder et al.

In examining suppression effects Paulhus et al. noted that the construct underlying the unshared variance (i.e., “pure” depression” or “pure” anxiety in the present study) may differ from the construct that includes the shared variance (i.e., the variance shared by anxiety and depression). With regard to anxiety and depression, there is considerable theoretical and empirical support for temperamental negative affect (NA) contributing to the development of both anxiety and depression (Muris and Ollendick 2005). Thus, NA may account for the shared variance between anxiety and depression in young children. “Pure” anxiety (or depression-free anxiety, i.e., the portion of variance for anxiety that is not shared with depression) may be particularly maladaptive, and is associated with subsequent anxiety and with subsequent depression, as the present data indicate. In fact, pure anxiety is more strongly associated with subsequent symptoms (both pure anxiety and total anxiety) than is total anxiety. While total depression (both pure depression and depression shared with anxiety) is associated with high levels of subsequent anxiety and depression, pure depression (or anxiety-free depression, i.e., the portion of variance for depression that is not shared with anxiety) is associated with both less subsequent anxiety and less subsequent depression.

Although the latter seems counterintuitive, it is possible that this association is accounted for by differences in the strategies used by parents in response to symptoms of anxiety or depression in their young children. Parents of children exhibiting higher levels of pure depression may use strategies akin to behavioral activation (e.g. arranging more enjoyable activities that, over time, may lead to lower levels of depression). That same strategy of behavioral activation may also lead to greater exposure to anxiety-arousing situations. Exposure is a major component in the treatment of anxiety. Thus, exposure of these children to more situations also may decrease their future anxiety symptoms. For this reason, pure depression may lead to less subsequent depression, whereas total depression (i.e., pure depression plus the portion of variance shared with anxiety) may generate parental strategies that are more consistent with parental reactions to childhood anxiety, such as limiting their child’s exposure to novel situations. The association between anxiety in children and parental overprotectiveness (McLeod et al. 2007) is well established; thus, high levels of child anxiety are likely to elicit overprotective parenting strategies that prevent exposure. Avoiding anxiety-evoking situations leads to increases in subsequent anxiety due to a reinforcement of the avoidance response as well as to less participation in potentially enjoyable activities, which could contribute to future depression. This process would account for the present finding that high levels of anxiety increase subsequent levels of both anxiety and depression. Further research will be needed to determine if, indeed, parenting strategies mediate the temporal relationship between initial and later symptoms of pure anxiety and depression. Additional research is also needed to determine if the characteristics of pure depression (i.e., anxiety-free depression) or pure anxiety (i.e., depression-free

anxiety) are associated with specific symptoms, cognitive processes, or other factors not associated with the shared variance of anxiety and depression.

Results of this study have implications for both the assessment and treatment of internalizing disorders in young children. First, given that these data suggest that pure depression in young children is related to later anxiety, and that anxiety is related to later depression, follow-up assessments of pre-schoolers who present with either type of symptom (e.g. anxiety) should include an assessment not only of the original type of symptom (e.g. anxiety), but also of the other type (e.g., depression). Second, since depression and anxiety are relatively stable even at younger ages, early intervention programs are crucial. The present data also suggest that early intervention strategies should target both anxiety and depression symptoms, regardless of the specific disorder with which the child presents. Third, clinicians who, based on previous research, assumed that depression inhibits anxiety and, consequently, were less concerned about young children with depression symptoms developing later identity should reconsider their treatment approach. Results of the current study suggest that, in fact, clinicians should be more vigilant about possible increases in anxiety among young children exhibiting depressive symptoms.

This study also makes clear that further research is needed to understand the mechanisms by which anxiety leads to subsequent depression, as Cole et al., and Snyder et al. previously suggested. Rather than examining how depression may inhibit subsequent symptoms of anxiety, the results of the present study, as well as the reexamination of prior findings that did not recognize the suppression effects point to the need to focus on (a) the mechanisms by which high levels of anxiety contribute to subsequent high levels of depression; and (b) how an understanding of suppressor effects can improve the prediction of subsequent anxiety and depression to more effectively target interventions.

The present study has certain limitations. First, because of the age of the children, self-reports of anxiety and depression could not be used. Also we could not use teacher-reports because of the difficulties involved in gathering such data from teachers at more than 300 different schools that the children attended. Teacher reports show only low levels of agreement with parent reports (Drabick et al. 2010), and the pattern of results could be substantially different for teacher reports. Second, the present study was conducted with a narrow age range of children as they transition into grammar school. The findings are generally consistent with those of other studies with older children. Nonetheless, a study of a single sample over an extended age range would be valuable. Third, while this study was conducted with a diverse sample, future studies with larger diverse samples would allow for multi-sample comparisons of the replicability of the models in each specific race/ethnic/age group. Fourth, anxiety measures used did not include social phobia or specific phobia, and future studies may need to consider the relationships of these anxiety disorders to depression.

Finally, while there are advantages to examining anxiety/depression relationships in community samples (as was done in the present study), there are also advantages to doing so in clinical samples. Studies conducted in both types of samples can be valuable and which sample is most appropriate will depend on the research question. Community samples: (a)

reduce the overrepresentation of comorbidities that may occur in clinical samples (Caron and Rutter 1991; Cole et al. 1998); (b) avoid biases present in referral patterns for mental health services. (Lavigne et al. 1998); (c) include children ranging across the whole spectrum of symptom severity, ranging from no symptoms through mild symptoms, those with subthreshold levels of disorder and that who would meet criteria for a disorder; (d) have certain statistical advantages because the variance of measures will not be as restricted as it might be when children only at the higher levels of the severity continuum are included in the sample. For these reasons, community samples are advantageous when studying basic psychological mechanisms (e.g., how anxiety affects the development of subsequent depression) and risk factors. Clinical samples can be advantageous to determine how those processes might be specific to individuals with disorders. Clinical samples might be more useful for questions such as (a) what is the likelihood that a patient with a diagnosis of anxiety will meet criteria for a depressive disorder subsequently?; (b) among patients meeting clinical criteria for comorbid disorders, which is more likely to remit first?; (c) are the factors associated with comorbidity in clinic samples different from those in community samples?

Despite these limitations, the present study shows that symptoms of depression and anxiety that emerge during the preschool period are relatively stable. Moreover, these data also show that symptoms of anxiety, do, indeed lead to later symptoms of depression, and that, contrary to previous results, pure depression symptoms also lead to later anxiety symptoms. Future prevention and intervention programs for pre-school children should target both types of symptoms.

Acknowledgments

This research was supported by National Institute of Mental Health grant MH 063665.

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Fig. 1.

Model 1: Autoregressions only. Structural model for autocorrelations without cross-lagged paths, showing the relationships between latent factors included in each model. Each latent factor included 3 measured indicators not indicated. * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

Fig. 2.

Model 2: Bidirectional influences with autoregressions. Structural model for autoregressions and cross-lagged paths, showing the relationships between latent factors included in each model. Each latent factor included 3 measured indicators not indicated. * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

Fig. 3.

Model 3: Bidirectional influences without autoregressions Structural model for cross-lagged paths without autoregressions, showing the relationships between latent factors included in each model. Each latent factor included 3 measured indicators not indicated. * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

Table 1

Symptom prevalence in the CSI normative sample and the present sample

Scale	Study Mean (Range) for total score	CSI norms: % exceeding cutoff	Study: % exceeding screening cutoff (anxiety); symptom counts (depression)
DISC generalized anxiety (highest possible total score=12)			
Age 4	1.42 (0–9)		
Age 5	1.27 (0–11)		
Age 6	1.45 (0–9)		
CSI generalized anxiety (highest possible total score=30)		4 % (ages 3–6 combined)	
Age 4	4.92 (0–28)		15 %
Age 5	5.18 (0–11)		11.80 %
Age 6	4.89 (0–20)		10.40 %
CSI separation anxiety (highest possible total score=30)		4 % (ages 3–6 combined)	
Age 4	2.28 (0–20)		
Age 5	2.22 (0–16)		6.30 %
Age 6	4.89 (0–20)		4.30 %
DISC major depression (highest possible total score=20)			3.10 %
Age 4	2.84 (0–16)		
Age 5	2.35 (0–15)		
Age 6	2.11 (0–14)		
CSI major depression (highest possible total score=33)		1 or more: 2.8 % (ages 3–6 combined)	
Age 4	1.16 (0–13)		3.10 %
Age 5	1.23 (0–11)		3.10 %
Age 6	1.20 (0–13)		4.10 %
CSI dysthymia (highest possible total score=21)		1 or more: 1.7 % (ages 3–6 combined)	
Age 4	0.78 (0–9)		1.40 %
Age 5	0.84 (0–8)		1.00 %
Age 6	0.89 (0–11)		2.10 %

^aMean total scores were not provided for the normative sample. Instead, screening cutoffs and number of symptoms were provided. Screening cutoff for both generalized anxiety and separation anxiety is 2 symptoms occurring “often” or “very often.” screening cutoffs not provided for MDD and dysthymia; rather, the percent of children with 1 or more symptoms was provided

Table 2

Correlations between measured indicators of anxiety and depression within and across ages

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17
Wave 1																	
1. CSI anxiety (wave 1)	1																
2. CSI separation anxiety (wave 1)	0.55**	1															
3. DISC GAD (wave 1)	0.44**	0.36**	1														
4. CSI depression (wave 1)	0.63**	0.50**	0.46**	1													
5. CSI dysthymia (wave 1)	0.63**	0.50**	0.43**	0.93**	1												
6. DISC MDD (wave 1)	0.56**	0.38**	0.59**	0.60**	0.57**	1											
Wave 2																	
7. CSI anxiety (wave 2)	0.59**	0.36**	0.32**	0.39**	0.36**	0.38**	1										
8. CSI separation anxiety (wave 2)	0.38**	0.55**	0.23**	0.29**	0.28**	0.25**	0.50**	1									
9. DISC GAD (wave 2)	0.37**	0.26**	0.50**	0.34**	0.31**	0.44**	0.47**	0.31**	1								
10. CSI depression (wave 2)	0.43**	0.32**	0.34**	0.49**	0.46**	0.43**	0.57**	0.37**	0.42**	1							
11. CSI dysthymia (wave 2)	0.45**	0.33**	0.33**	0.48**	0.47**	0.41**	0.59**	0.36**	0.44**	0.91**	1						
12. DISC MDD (wave 2)	0.44**	0.30**	0.44**	0.45**	0.40**	0.60**	0.45**	0.24**	0.56**	0.55**	0.56**	1					
Wave 3																	
13. CSI anxiety (wave 3)	0.52**	0.33**	0.22**	0.34**	0.33**	0.31**	0.58**	0.34**	0.30**	0.39**	0.42**	0.35**	1				
14. CSI separation anxiety (wave 3)	0.33**	0.47**	0.17**	0.26**	0.26**	0.17**	0.31**	0.57**	0.24**	0.31**	0.29**	0.22**	0.45**	1			
15. DISC GAD (wave 3)	0.41**	0.36**	0.44**	0.39**	0.35**	0.38**	0.42**	0.34**	0.54**	0.39**	0.42**	0.38**	0.47**	0.36**	1		
16. CSI depression (wave 3)	0.42**	0.31**	0.29**	0.46**	0.41**	0.36**	0.39**	0.32**	0.32**	0.48**	0.48**	0.38**	0.61**	0.41**	0.48**	1	
17. CSI dysthymia (wave 3)	0.42**	0.31**	0.28**	0.44**	0.42**	0.35**	0.36**	0.32**	0.32**	0.43**	0.46**	0.35**	0.61**	0.40**	0.47**	0.94**	1
18. DISC MDD (wave 3)	0.42**	0.27**	0.32**	0.43**	0.39**	0.54**	0.32**	0.19**	0.38**	0.36**	0.39**	0.51**	0.50**	0.29**	0.52**	0.52**	0.53**

Child symptom inventory; GAD generalized anxiety disorder; MDD major depressive disorder