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ORIGINAL ARTICLE

Perceived Family Environment and Symptoms of Emotional Disorders: The Role of Perceived Control, Attributional Style, and Attachment

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Abstract Findings from decades of research suggest that a perceived lack of parental care and overprotection are positively related to later symptoms of emotional disorders in children and adolescents. The present study used a cross-sectional design to evaluate models investigating reported family environment during childhood, current attachments, control-related cognitions, and current symptoms of emotional disorders in adolescence. It was hypothesized the effect of a perceived controlling and rejecting family environment during childhood would influence current depression and anxiety, and that these effects would be partially accounted for by the quality of current attachments, perceived control, and attributional style. A sample of 234 university students was assessed. Regression analyses of variables, including analyses of indirect effects, were conducted. As predicted, current attachment, perceived control, and attributional style helped to account for relationships between some family variables, and depression and anxiety. Findings are discussed with respect to the interplay of family variables and models of emotional disorders.

Keywords Parenting · Perceived control · Attachment · Attributional style · Depression and anxiety

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Introduction

For the past several decades, researchers have examined the effects of family variables such as parenting on later socioemotional outcomes in offspring. For example, Parker, Tupling, and Brown (1979) used factor analytic studies to conclude that parental behaviors and attitudes towards children have two dimensions: care and protection. The ''care'' variable describes parenting characterized by affection, emotional warmth, empathy, and closeness on one end of the spectrum, and emotional indifference, coldness, and neglect on the other. The ''overprotection'' variable describes parenting characterized by intrusion, control, and prevention of independent behavior on one end, and the allowance of independence and autonomy on the other end. The Parental Bonding Instrument (Parker et al., 1979) was developed to study these parenting dimensions and has prompted much research.

With some exceptions (e.g., Lewinsohn & Rosenbaum, 1987; MacKinnon, Henderson, Scott, & Duncan-Jones, 1989), numerous empirical studies with both clinical and community samples have confirmed that perceived low parental care and overprotection in childhood are associated with depression and anxiety disorders later in life (e.g. Gerlsma, Emmelkamp, & Arrindell, 1990; Kendler, Myers, & Prescott, 2000; Neale et al., 1994; Wolfradt, Hempel, & Miles, 2003). However, much less research has focused on *how* the early environment creates or influences vulnerabilities for these emotional disorders. Although several lines of research have suggested possible cognitive factors that may mediate the relationship between early environment and later maladjustment, there is not yet a consensus on which are most important.

Control, care, contingency, and attachment

One hypothesis is that early experiences with attachment figures may influence the development of a sense of contingency between actions and outcomes and hence of perceived control over the environment. Through contingent relationships with caregivers, it is thought that the child begins to learn that their actions and behaviors can evoke a predictable outcome (e.g., see Thompson, 1998). By contrast, a history of lack of control may put individuals at eventual risk for experiencing negative emotions through the development of a generalized tendency to perceive events as not within one's control (see Chorpita & Barlow, 1998; Mineka & Zinbarg, 1996, 2006; Schneewind, 1995). A second and related hypothesis from attachment theory suggests that parental inconsistency or neglect creates a risk for emotional disorders by affecting the individual's belief that he or she is worthy of care (see Bowlby, 1973, 1977) and by affecting the individual's ability to develop relationships that can support him or her in times of stress (see Bowlby, 1988). The study of personal control, attachment, and negative emotions has relied on the examination of constructs representing thoughts and beliefs about mastery, contingency, and the quality of interpersonal relationships.

In terms of control-related cognitions, two theoretical approaches have been widely researched including *locus of control* (e.g., Levenson, 1974, 1981) and *learned helplessness/hopelessness* (e.g., Abramson, Seligman, & Teasdale, 1978). Research has suggested that people's perceptions of control over specific negative life events, in conjunction with the kinds of causal attributions made for those events, affect the development of negative emotions. For example, findings from cross-sectional studies generally indicate that a more *external locus of control* correlates with greater

depressive and anxious symptomatology (e.g., Beautrais, Joyce, & Mulder, 1999; Chorpita & Barlow, 1998; Clarke, 2004; see Preston & Benassi, 1996, for a meta-analysis). Unfortunately, however, it is not known whether an external locus of control is merely a correlate or symptom of depression or anxiety, or whether it serves as a risk factor. In addition, a pessimistic attributional style is also observed in depressed (and sometimes anxious) individuals (e.g., Buchanan & Seligman, 1995; Mineka, Pury, & Luten, 1995; Sutton et al., 2007, Submitted). Moreover, some prospective studies also suggest that negative attributional style may serve as a risk factor for the development of major depression (e.g., Alloy et al., 2006), or depressed mood in the wake of a stressful life event (e.g., Metalsky & Joiner, 1992; Metalsky, Joiner, Hardin, & Abramson, 1993).

Research into the hypothesized role of attachment has also examined inter-relationships between the quality of caregiver attachment in childhood, the quality of current interpersonal relationships, and emotional disorders. An extensive literature has explored the relationship between perceived early attachments to parents and attachment styles as adults (see Parker, Barrett, & Hickie, 1992; Westen, 1998, for reviews). In general, results suggest that adults with insecure attachment styles described their parents as less caring or more inconsistent in their parenting than those who reported secure adult attachments. Other studies have, in turn, linked poor quality current attachment relationships and low social support to depression and anxiety (e.g., Bifulco, Moran, Ball, & Bernazzani, 2002; Carnelley, Pietromonaco, & Jaffe, 1994; Mikulincer & Orbach, 1995; Roberts, Gotlib & Kessel, 1996). Additionally, in a longitudinal study of women who had recently graduated from high school, Hammen et al. (1995) found that insecure current attachment both related to fewer positive parental memories of care and predicted the onset and severity of depressive symptoms at a 12-month follow-up interview in the face of an interpersonal stressor.

In sum, research supports the view of the importance of perceived control and secure attachments in adjustment. In addition, developmental factors influenced by family environment and parental relationships likely impact the formation of these cognitive and interpersonal vulnerabilities to emotional disorders.

Mediational models

The family environment has been hypothesized to affect control-related cognitions and the ability to form close relationships later in life. A few studies have attempted to examine whether control-related cognitions account for the relationships between parent and family variables, and depression and anxiety (e.g., Chorpita, Brown, & Barlow, 1998; Taris & Bok, 1997; Whisman & Kwon, 1992). For example, in a cross-sectional study of college students, Whisman and Kwon (1992) concluded that the relationship of early low parental care to more severe depressive symptoms in college was accounted for by a depressive attributional style and dysfunctional attitudes. In another cross-sectional study with clinically anxious and non-anxious children, Chorpita et al. (1998) found that perceived control over the environment accounted for the relationship between current family over-control and current childhood anxiety. Finally, in a longitudinal study with a non-clinical adult sample, Taris and Bok (1997) examined the relationship between perceived low parental care in childhood, locus of control, and depression, and found some evidence that lower feelings of personal controllability accounted for the relationship between early low parental care and depression.

Several other studies have explored the degree to which the quality of adult relationships accounts for the effects of early relationships on adult emotional disorders. In general the literature suggests that perceived lower parental care in childhood was related to higher levels of depression, and that this relationship was partially accounted for by how secure people felt in current relationships (e.g., Gittleman, Klein, Smider, & Essex, 1998; Strahan, 1995). This evidence is consistent with the notion that the quality of the parent-child relationship influences the child's ability to form a social network, which in turn has been shown to be related to the development of emotional disorders.

Unfortunately, there are a number of limitations of research to date on this topic. For example, most studies of mediational models including parent and family variables have generally explored either interpersonal attachment variables or control-related cognitive variables – but not both in the same study. Two recent studies have examined models predicting both symptoms of anxiety and depression that included both cognitive and attachment variables in samples of undergraduates (Safford, Alloy, Crossfield, Morocco, & Wang, 2004; Williams & Riskind, 2004). Both studies found that control-related cognitive variables and attachment style variables improved the models' ability to predict both anxiety and depressive symptoms, but neither study included parent and family variables in their analyses of indirect effects. Theoretically, responsive and caring parenting would give the child a sense that he or she can evoke responses from others and to some extent can influence their environment in an effectual way, but also would foster the expectation that relationships with others can be satisfying and nurturing. Similarly, parenting characterized by over-control might limit both the child's exposure to mastery experiences and peer relationships and foster the expectation that relationships with others are intrusive and controlling. In these ways, associations of parental care and overprotection with anxiety and depression might be accounted for by both control-related cognitions and the quality of interpersonal attachments.

Other limitations are that some studies have examined only maternal variables or a composite of maternal and paternal variables in the analysis, even though maternal and paternal variables might be expected to contribute to the prediction of distress in different ways (e.g., Phares & Compas, 1992; Phares, Field, Kamboukos, & Lopez, 2005). Similarly, perceptions of paternal and maternal care and overprotection might differ as a function of child gender, thus participant gender and parent gender might interact in important ways in models predicting symptoms of emotional disorders. In addition, later work by Parker et al. (1997) examined ''dangerous and threatening'' parenting (i.e., verbal abuse, physical violence) in addition to parental care and overprotection. They found preliminary evidence suggesting that this dimension relates to both anxiety and depression, although much less research has examined this high conflict dimension of parenting in conjunction with the care and overprotection dimensions. A more thorough examination of these mediational models would analyze paternal and maternal variables separately, would include participant gender, would include a measure of conflict in the family, and would include both control-related variables and interpersonal attachment variables. Finally, such a study should examine the relationships of these variables to both anxiety and depression instead of to one or the other as in most prior studies.

A hierarchical model of emotional disorders

The understanding of the relationships between anxiety and depression has become increasingly sophisticated. Although anxiety and depression seem reasonably distinct

from one another, at the diagnostic and self-report levels it has become evident that there is also a great deal of overlap between the two (e.g., Clark & Watson, 1991; Maser & Cloninger, 1990). Many studies of children, students, and adults have shown that nearly all self-report measures and clinician rating scales of anxiety and depression are highly correlated, with coefficients in the .45 to .75 range (e.g., Clark & Watson, 1991; Costa & McCrae, 1992). At the diagnostic level, there is also a great deal of comorbidity between the diagnosis of depression and various anxiety disorders and vice versa (e.g., Maser & Cloninger, 1990; Mineka, Watson, & Clark, 1998).

Over the past 15 years the most influential theory explaining the overlap between mood and anxiety symptoms and disorders has been the tripartite model of anxiety and depression (e.g., Clark & Watson, 1991). Building on earlier work of Watson and Tellegen (1985), Clark and Watson (1991) proposed that negative affect is the affective dimension which characterizes both anxiety and depression. Moreover, they hypothesized that a second basic affective dimension—positive affect—is what distinguishes depression from anxiety, with depression (but not anxiety) being characterized by low levels of positive affect. In addition, based on factor-analytic studies, they proposed that a specific factor—anxious arousal (somatic anxiety)—seemed to be unique to anxiety/panic. Later, however, this factor was found to be primarily related to panic-like anxiety but not general anxiety (Mineka et al., 1998). Since it was proposed in 1991, the tripartite model has been supported by both exploratory and confirmatory factor analytic studies (e.g., Geisser, Cano, & Foran, 2006; Keough & Reidy, 2000; Nitschke, Heller, Imig, McDonald, & Miller 2001).

The tripartite model has important implications for the assessment of emotional disorders and for research examining predictors of anxiety and depression. For example, in one study examining parent and family variables and the tripartite model of negative emotions, Safford et al. (2004) discovered that perceived maternal care was most strongly associated with the anhedonic depression scale ($\Delta R^2 = .09$) and perceived paternal care was most strongly associated with the general distress scale ($\Delta R^2 = .09$), whereas maternal overprotection was most strongly associated with the anxious arousal scale ($\Delta R^2 = .04$). Thus, research on family environment variables impacting the development of negative emotions might be advanced by focusing on both predictors of the general negative affect factor and predictors of the more specific dimensions of anhedonia and anxious arousal.

Overview of current research

The current study examined the interplay of the early environment measures with measures of current control-related cognitive variables and current attachment variables in predicting anxiety and depression viewed within the tripartite model framework. The variables included (1) both mother and father care and overprotection, (2) participant gender, (3) family environment variables including conflict and control, (4) adult attachment variables, (5) attributional style and control-related cognitive variables, and (6) symptoms of anxiety and depression. The relationships of these variables with each of the three factors in the tripartite model were assessed. Overall, lower care, higher overprotection, more family conflict, and more family control were expected to be associated with higher levels of emotional distress. Moreover, some variance in the relationships between maternal and paternal variables and different symptom scales was predicted, as seen by Safford et al. (2004), and the consistency of these models across participant gender was assessed. Finally, the current study was designed to replicate and

extend previous studies showing evidence for the roles of control-related cognitive styles and interpersonal attachment styles as factors that might account for the relationship between early parenting variables and negative emotions (e.g., Chorpita et al., 1998; Gittleman et al., 1998; Taris & Bok, 1997).

Method

Participants

Participants were 234 students (140 females and 94 males) randomly selected from the class lists of two introductory psychology courses at Northwestern University. The students participated as part of their course requirement. Of these initial 234 participants, 218 completed all of their questionnaires, but four participants omitted some items and were dropped from the sample. Thus, the final sample of 214 participants consisted of 125 females and 89 males.

Measures

The Parental Bonding Instrument (*PBI; Parker, Tupling, & Brown,* 1979) is a 25-item self-report measure composed of the two subscales reflecting recalled parental care and parental overprotection. Participants rate on a four-point scale how much each statement described each parent (Mother—MPBI; Father—FPBI) in the first 16 years of life with higher scores representing more care and more overprotection. Internal consistencies (Cronbach's alpha) are high (.90 to .95 for parental care and .86 to .87 for parental overprotection; Parker, 1989; Gittleman et al., 1998; Shams & Williams, 1995). For the current sample, reliability estimates are presented in Table 1. Three-month test-retest reliability is high (.86 for care and .85 for overprotection; Whisman & Kwon, 1992), and moderate consistency has been shown over extended periods up to 10 years (.63 to .72 for parental care and .56 to .68 for parental overprotection; Wilhelm & Parker, 1990). PBI scores have not been found to be influenced by mood states (Parker, 1989), and various research strategies have all supported the PBI as a measure of actual parenting (e.g., corroborative reports by siblings and parents; contrasting scores returned by MZ and DZ twins; correlation with interview-derived parental ratings; Wilhelm & Parker, 1990).

Family Environment Scale (*FES; Moos,* 1974) asks participants to indicate whether 90 statements about families are either true or false about their family. The measure administered to participants has ten 9-item subscales. Moos (1974) originally reported alpha coefficients for each scale ranging from .64 to .79. The Conflict subscale measuring levels of family violence and levels of conflict and the Control subscale measuring levels of family control, adherence to rules, and rigidity were the only subscales of interest for the current investigation, with higher scores representing more conflictual and controlling family environments.

The Adult Attachment Scale (AAS; Collins & Read, 1990*)* is an 18-item scale to measure adult attachment style dimensions based on Hazan and Shaver's (1987) categorical measure of attachment style. Its three subscales measured the extent to which an individual is comfortable with closeness, feels he or she can depend on others, and is anxious or fearful about being abandoned or unloved. Sperling, Foelsch, and Grace (1996) assessed the association between the AAS and other measures of adult

Table 1 Correlations of parent, family, attachment, and control-related cognitive variables

		1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.
1.	GD/NA	(.94)												
2.	AD	.57*	(.91)											
3.	AA	.66*	.25*	(.85)										
4.	MPBI-C	–.29*	–.39*	–.19*	(.88)									
5.	MPBI-O	.25*	.20*	.11	–.47*	(.87)								
6.	FPBI-C	–.33*	–.26*	–.20*	.41*	–.25*	(.91)							
7.	FPBI-O	.32*	.22*	.25*	–.40*	.55*	–.44*	(.85)						
8.	FES-Conflict	.25*	.22*	.24*	–.47*	.41*	–.31*	.43*	(.78)					
9.	FES-Control	.20*	.16*	.05	–.30*	.56*	–.18*	.53*	.37*	(.70)				
10.	ASQ	.43*	.23*	.29*	–.15*	.08	–.12	.17*	.26*	.23*	(.79)			
11.	AAS	–.50*	–.53*	–.37*	.44*	–.26*	.47*	–.41*	–.35*	–.22*	–.28*	(.81)		
12.	PUCN	.37*	.41*	.28*	–.27*	.19*	–.33*	.24*	.15*	.20*	.06	–.37*	(–)	
13.	POL	.42*	.42*	.28*	–.16*	.08	–.11	.09	.14*	.10	.24*	–.35*	.37*	(–)

Note: Reliabilities are presented in parentheses. GD/NA = General Distress/ Negative Affect, AD = Anhedonic Depression, AA = Anxious Arousal, MPBI-C = Maternal Care, MPBI-O = Maternal Overprotection, FPBI-C = Paternal Care, FPBI-O = Paternal Overprotection, FES-Conflict = Family Conflict, FES-Control = Family Control, ASQ = Attributional Style Questionnaire, AAS = Adult Attachment Scale, PUCN = Factor score for perceived lack of control, POL = Factor score for powerful others/luck

* $p < .05$

attachment and their findings support the construct validity of the measure. This study followed the suggestion of Sperling et al. (1996) and examined attachment on one secure-insecure dimension and utilized a composite of the three subscales (with Anxiety items reverse scored) to represent attachment. Three items explicitly referring to romantic relationships were omitted for this college-aged sample in which 32 participants indicated never being involved in such a relationship. Higher scores represent more secure current attachments.

The Expanded Attributional Style Questionnaire (*ASQ; Peterson & Seligman,* 1984) used in this study presents participants with 12 hypothetical negative events involving themselves and asks them to offer ''one major cause of the event.'' They then rate that event along three 7-point Likert scales according to its internality (7) vs. externality (1), stability (7) vs. instability (1), and globality (7) vs. specificity (1). Each of these dimensions has been shown to possess an acceptable level of internal consistency (.66, .85, and .88, respectively; Peterson & Villanova, 1988). A composite of the stability and globality scores for each of the twelve items was calculated to form the ASQ Generality subscale with higher scores representing a more negative attributional style. This subscale has been shown empirically and theoretically to reflect the construct most central to the reformulated helplessness and hopelessness models of depression (e.g., Abramson, Seligman, & Teasdale, 1978; Abramson, Metalsky, & Alloy, 1989; Whisman & Kwon, 1992).

The Mastery Scale (*Pearlin & Schooler,* 1978) is a 7-item scale that assesses the extent to which one regards one's life circumstances as being under one's control in contrast to being fatalistically determined. Participants rate on a 4-point Likert scale how much they agree with statements about personal control. Higher scores represent more mastery. Internal consistency has been reported in the acceptable range (e.g., .75 in Skaff, Pearlin, & Mullan, 1996).

The Internality, Powerful Others, and Chance Scales (*I, P, and C; Levenson,* 1974) is a 24-item scale that measures the extent to which people believe that they have control over their own lives. Participants rate on a 6-point Likert scale how much they agree with certain statements. Internality (I) measures the extent to which people believe they have control over their own lives (8 items), Powerful Others (P) concerns the belief that other persons control the events in one's life (8 items), and Chance (C) measures the degree to which a person believes that chance affects his or her experiences and outcomes (8 items). Reliabilities for the subscales have been shown to be reasonable (.64, .77, and .78 respectively; Levenson, 1981). Substantial correlations between the P and C scales have been reported (r = .59, Levenson, 1974; r = .40, Walkey, 1979). Correlations between the I scale and the P and C scales have been reported as r = .01 and r = –.19 respectively (Walkey, 1979). High scores for the sum of scores on the Powerful Others and Chance scales represent a more external locus of control. Test-retest reliabilities with a 1-week interval range between .60 and .79 (Levenson, 1981).

Mood and Anxiety Symptom Questionnaire (MASQ; Watson et al., 1995*)* consists of items garnered from the symptom criteria for the anxiety and mood disorders in *DSM-III-R (*American Psychiatric Association, 1987). Participants indicated to what extent they had experienced each symptom (1 = *not at all*, 5 = *extremely*) ''during the past week including today.''

The 65-item version of the MASQ we used had 3 scales: (1) the General Distress/Negative Affect Scale, (2) the Anxious Arousal Scale, and (3) the Anhedonic Depression Scale. Although the 27-item General Distress Scale is further subdivided into 3 subscales, we only used the overall General Distrss/NA (GD/NA) scale which contains item assessing general symptoms of both anxiety and depression. The Anxious Arousal Scale (AA) includes 17 items that describe symptoms of somatic hyperarousal (e.g., feeling dizzy or lightheaded, shortness of breath, dry mouth) thought to be specific to anxiety/panic (Watson et al., 1995). Finally, the Anhedonic Depression Scale (AD) includes 7 Loss of Interest items reflecting anhedonia, disinterest, and low energy (e.g., ''felt like nothing was enjoyable'') and 14 reverse-scored High Positive Affect items assessing positive emotional experiences (e.g., felt cheerful, optimistic, looked forward to things with enjoyment). Coefficient alpha for these three scales have been reported for students, adults, and patients and range from .78 to .93 (see Watson et al., 1995).

Procedure

Participants were asked to participate in a study about their thoughts and feelings. They were scheduled for small group sessions that lasted approximately 1 h and were asked to complete the packet of questionnaires. At the end of the session, participants were given information about resources available where they could talk to a therapist about any difficulties they were having in their lives.

Results

Factor analyses

Exploratory factor analysis was used to examine relationships between items in the Mastery Scale (MS) and the Internality, Powerful Others, and Chance Scales (IPC), and

the ASQ. To determine the number of factors to extract, Velicer's (1976) minimum average partial (MAP) test was conducted, as well as a parallel analysis (e.g., Hayton, Allen, & Scarpello, 2004). Both procedures were implemented using O'Connor's (2000) algorithms. Velicer's MAP analysis suggests the number of factors to extract by conducting a series of principal components analyses on the data to determine which number of components minimizes residual correlations after the components are partialed from variables in the data set. Parallel analysis compares eigenvalues in the data to a set of simulated data sets that have the same number of cases and variables, but whose variables are uncorrelated. Thus, a parallel analysis compares observed eigenvalues to those that arise from chance.

Velicer's MAP suggested three factors with a minimum average squared partial correlation of .0095. We conducted the parallel analysis by randomly shuffling the raw data, which results in uncorrelated variables but preserves distributions of variables. This method is recommended when variables are not continuous and cannot be assumed to be normally distributed (O'Connor, 2000). We used 1000 replications and examined the 95th percentile of each eigenvalue. The first 10 eigenvalues from the observed data were as follows: 6.82, 3.76, 2.42, 1.80, 1.64, 1.49, 1.46, 1.35, 1.27, and 1.24. The first 10 eigenvalues from the random data 2.05, 1.91, 1.82, 1.74, 1.67, 1.61, 1.56, 1.51, 1.46, 1.41. Thus, the parallel analysis suggested four factors because the first four factors in the raw data were larger than the corresponding eigenvalues from the simulated datasets. Because the MAP analysis and parallel analysis differed, we examined a scree plot as well as the factor pattern matrix for the three- and four-factor solutions. It was decided to extract three factors because a fourth factor did not account for much additional variance in the data (2.8%) and because the fourth eigenvalues for the observed and random data in the parallel analysis were close in value (1.80 vs. 1.74). In addition, although parallel analysis is highly accurate across various conditions, when it errs it tends to yield too many factors (Zwick & Velicer, 1986).

Principal axis factor extraction was used to obtain the three factors, and an oblique solution was obtained using direct oblimin rotation. Before rotation, the factors accounted for 14.2, 7.1, and 4.0% of the variance respectively. After rotation, factor loadings that were greater than |.30| were interpreted. The first factor was best described as a lack of perceived control factor, with all seven Mastery Scale items loading on it, as well as four internality items and two chance items from the I, P, and C scales (items #6, #14, #18, #19, #21, #23). The second factor was clearly an ASQ factor with all 12 items in the generality subscale loading on it, with no items from other scales having a loading of more than .3. Seven items from the powerful others scale and two chance items from the I, P, and C scales (items #3, #7, #8, #11, #13, #15, #16, #17, #22) loaded onto the third factor. One item from the Mastery Scale (#4) that loaded onto the first factor also loaded onto the third factor. The third factor was composed of items related to the belief that powerful people and/or luck determined the outcome of one's life. For subsequent analyses, factor scores for the first and third factors were saved to create variables for the perceived uncontrollability scale (PUCN) and the powerful others/luck scale (POL). Correlations between these factors, the ASQ, and other variables in the study are presented in Table 1. Table 2 presents the factor pattern matrix for the three-factor solution from the exploratory factor analysis.

Table 2 Factor pattern matrix

Item	Factor 1	Factor 2	Factor 3
Mastery Scale #1	**0.68**	–0.01	0.04
Mastery Scale #6	**–0.62**	0.06	0.06
I, P, and C #18	**–0.61**	0.04	0.14
I, P, and C #23	**–0.58**	–0.03	0.08
I, P, and C #19	**–0.52**	–0.05	–0.02
Mastery Scale #7	**–0.48**	–0.06	–0.17
Mastery Scale #3	**0.47**	–0.04	0.12
I, P, and C #21	**–0.40**	0.00	0.06
I, P, and C #14	**0.39**	0.05	0.21
Mastery Scale #2	**0.39**	0.12	0.20
Mastery Scale #4	**0.36**	0.16	**0.30**
Mastery Scale #5	**0.33**	0.17	0.24
I, P, and C #6	**0.32**	–0.09	0.27
I, P, and C #24	0.30	0.10	0.15
I, P, and C #2	0.29	–0.04	0.20
I, P, and C #10	0.28	–0.04	0.15
I, P, and C #5	–0.26	0.08	–0.07
I, P, and C #9	–0.23	–0.14	0.15
ASQ #4	0.03	**0.61**	0.06
ASQ #9	–0.16	**0.59**	0.04
ASQ #3	0.00	**0.59**	0.03
ASQ #6	–0.04	**0.54**	-0.05
ASQ #8	0.08	**0.50**	0.02
ASQ #12	–0.18	**0.47**	0.07
ASQ #7	–0.12	**0.47**	0.05
ASQ #10	0.17	**0.47**	0.01
ASQ #2	0.02	**0.45**	–0.04
ASQ #5	–0.13	**0.43**	0.16
ASQ #11	0.04	**0.38**	0.07
ASQ #1	0.10	**0.36**	–0.09
I, P, and C #11	0.10	–0.05	**0.68**
I, P, and C #22	–0.12	0.12	**0.63**
I, P, and C #15	–0.06	0.03	**0.63**
I, P, and C #3	0.05	–0.03	**0.61**
I, P, and C #17	0.07	0.12	**0.44**
I, P, and C #13	0.23	0.01	**0.43**
I, P, and C #16	0.10	0.08	**0.42**
I, P, and C #8	–0.01	0.13	**0.40**
I, P, and C #7	0.29	0.06	**0.37**
I, P, and C #20	0.05	–0.06	0.28
I, P, and C #12	0.22	–0.07	0.27
I, P, and C #4	–0.13	0.09	0.18
I, P, and C #1	–0.11	–0.17	0.02

Note. Factor loadings > = |.30| are listed in bold. ASQ = Attributional Style Questionnaire. I, P, and C = Internality, Powerful Others, and Chance Scales

Predictive models of symptom scales

The first goal of the present study was to examine which parent and family variables predicted higher scores on the symptom scales when considering the hierarchical structure of emotional disorders. Specifically, which early parent and family variables are predictive of the general distress factor that is common to depression and anxiety (GD/NA)? Similarly, what variables are predictive of the more specific components of

anxiety/panic (AA) and depression (AD)? Secondly, does participant gender interact with family variables?

To answer these questions, hierarchical regression analyses were conducted with all six parent and family variables, and participant gender, predicting each of the three symptom scales. Main effects were entered on step 1 of these regressions, and interactions with participant gender were entered on step 2. These regressions revealed that different configurations of parent and family variables explained unique variance in the prediction of scores on the three symptom scales above and beyond the influence of the other parent and family variables. As shown in Table 3, participants with low paternal care scored higher on GD/NA. Step 2 of this regression approached statistical significance ($p = .054$), so interaction effects were examined. Gender interacted with maternal overprotection and this effect was explored with follow-up simple regressions done separated within gender. The standardized regression parameters (βs) for maternal overprotection were –.20 ($p = .15$) for males and .26 ($p = .02$) for females. In other words, maternal overprotection was associated with more general distress in females, and less general distress in males. However, maternal overprotection was only a significant predictor of general distress in females.

For AD, low maternal care predicted higher levels of anhedonic depression. No interaction effects were found with gender. Finally, for scores on AA, paternal overprotection and high family conflict were associated with higher levels of AA over and above the other family variables. The set of interactions with gender accounted for an additional contribution (6%) of the variance in AA. To clarify these effects, simple regression analyses were conducted. Maternal overprotection was positively related to AA ($\beta = .16$, $p = .17$) in females, but this relationship was not statistically significant.

Table 3 Regression results

	GD/NA		AD		AA	
	ΔR^2	β	ΔR^2	β	ΔR^2	β
Step 1	.18*		.17*		.11*	
Gender		–.13†		–.11		–.05
MPBI-C		–.10		–.32*		–.05
MPBI-O		.07		–.01		–.03
FPBI-C		–.21*		–.12		–.08
FPBI-O		.11		.02		.21*
FES-Conflict		.07		.02		.17*
FES-Control		.01		.04		-.14
Step 2	.05†		.01		.06*	
Gender × MPBI-C		.36		.07		–.23
Gender × MPBI-O		–.50*		–.26		–.52*
Gender × FPBI-C		–.16		–.10		–.10
Gender × FPBI-O		.26		.16		.36*
Gender × FES-Conflict		–.15		.02		–.32*
Gender × FES-Control		.19		–.05		.22

Note. Beta weights shown for step 1 are from the model *before* entering interaction terms on step 2. GD/NA = General Distress/ Negative Affect, AD = Anhedonic Depression, AA = Anxious Arousal, MPBI-C = Maternal Care, MPBI-O = Maternal Overprotection, FPBI-C = Paternal Care, FPBI-O = Paternal Overprotection, FES-Conflict = Family Conflict, FES-Control = Family Control. Gender is dummy coded such that 0 = female, 1 = male. * $p < .05$; † $p < .10$

For males, maternal overprotection was inversely related to AA ($\beta = -.31$, $p = .03$) such that higher maternal overprotection was related to less anxious arousal. Paternal overprotection was positively related to AA in males ($\beta = .45$, $p = .01$), but in females the relationship was negligible ($\beta = .07$, $p = .56$). Family conflict was positively related to AA for females ($\beta = .29$, $p = .01$), but in males the relationship between family conflict and AA was negligible ($\beta = -.08$, $p = .54$).

Analyses of indirect relationships

After examining the early parent and family variables predicting the symptoms scales, we examined whether the relationship between self-reported early family variables and current symptomatology might be accounted for by current levels of attachment and control-related cognitions. A strict test of mediation would require a longitudinal design in which the predictors preceded the mediators, which in turn preceded the dependent variables. However, cross-sectional designs can assess whether patterns of correlations are consistent with mediational models by testing the significance of indirect effects. It should be noted that tests of indirect effects would be consistent with a mediational model but cannot confirm the existence of mediated effects in our cross-sectional design. Rather, indirect effects include possible mediated relationships, as well as other third-variable effects (MacKinnon, Krull, & Lockwood, 2000). The zero-order correlations between the family variables, symptom scales, and potential mediators are presented in Table 1.

To assess potential indirect relationships, Mplus software, version 4.1 was used (Muthén & Muthén, 2006). Mplus provides parameter estimates and confidence intervals for indirect effects. The confidence intervals were formed using the bias-corrected bootstrap method recommended by Mackinnon, Lockwood, and Williams (2004) on the basis of simulation research. For our analyses, all family variables, participant gender, AAS, ASQ, and factor scores for PUCN and POL were regressed on GD/NA, AD, and AA. Then, potential mediators (AAS, ASQ, PUCN, POL) were regressed on all family variables, and participant gender. This structure allows for precise assessment of direct and indirect effects (e.g., parameters estimates and confidence intervals for the compound relationships, such as FPBI-C → DAAS → GD/NA). Indirect effects were only interpreted for significant main effects from the initial regression analyses (see Table 3). Five significant indirect effects were found and are summarized in Table 4.

To summarize these results, the association between low paternal caring and GD/NA can be accounted for, in part, by lower adult attachment and a sense of perceived lack of control. Similarly, the relationship between low maternal care and higher anhedonic depression can be accounted for, in part, by adult attachment and perceived lack of control. None of the indirect effects were significant for the parental overprotection variables. Finally, family conflict was positively related to anxious arousal in females, and this effect was partially accounted for by pessimistic attributional style.[1]

[1] Overall, attributional style accounted for the relationship between family conflict and AA. However, given that previous regression analyses revealed that the relationship between family conflict and anxious arousal was only significant for females, we presented the results for this test of indirect effects for females only.

Table 4 Summary of significant mediated effects

Relationship	Standardized indirect effect	95% Confidence interval
FPBI-C → AAS → GD/NA	–.15	–.30, –.06
FPBI-C → PUCN → GD/NA	–.09	–.22, –.02
MPBI-C → AAS → AD	–.16	–.31, –.07
MPBI-C → PUCN → AD	–.06	–.15, –.003
FES-Conflict → ASQ → AA (within females only)	.20	.04, .50

Note. FPBI-C = Paternal Care, AAS = Adult Attachment Scale, GD/NA = General Distress/ Negative Affect, PUCN = Factor score for perceived lack of control, MPBI-C = Maternal Care, FES-Conflict = Family Environment Scale-Conflict, AD = Anhedonic Depression, AA = Anxious Arousal, ASQ = Attributional Style Questionnaire

Discussion

The present study was conducted to test the relation between parental and family variables, control-related cognitions, current interpersonal attachment and symptoms of anxiety and depression. We followed the suggestion of Phares and Compas (1992) to clarify the role of parental variables in the development of psychopathology by keeping maternal and paternal variables separate in the analysis. Moreover, by examining these parent and family variables within the framework of the tripartite model of anxiety and depression, the current study attempted to identify the relationship between variables corresponding to perceptions of certain early family patterns and different components of anxiety and depression (some relatively specific to anxiety vs. depression and one for general distress) and whether the prediction models were consistent across participant gender. We also analyzed indirect relationships in an effort to understand whether attachment, attributional style, and a sense of mastery might help to account for these relationships. The major predictions were that reports of family environments characterized by perceptions of lower care and higher overprotection would be related to greater symptomology and that this relationship would be partially accounted for by an individual's difficulties in establishing close, supportive relationships with others and by an individual's perception of lack of control over the environment.

The role of both low parental care and high overprotection in the model predicting more symptoms of emotional disorders is the finding generally observed in the literature (see Gerlsma et al., 1990). However, in the current investigation, low paternal care was the best predictor of GD/NA, which is the common factor in depression and anxiety. Low maternal care was the best predictor of Anhedonic Depression, the factor specific to depression. Similar findings for both low paternal care and low maternal care were obtained by Safford et al. (2004). Gender of participants did not significantly interact with either of these predictors in our study (and was not examined in the Safford et al. (2004) study).

The pattern of predictors was different for the anxious arousal symptoms relatively specific to anxiety/panic. Higher paternal overprotection and family conflict both predicted higher levels of somatic anxiety. However, participant gender interacted with family variables in predicting somatic anxiety. Maternal overprotection was positively related to AA in females (although this effect was not significant), but negatively related to AA in males. The effect of paternal overprotection on AA was positive and significant for males and virtually zero for females. Family conflict emerged as a

significant predictor of high AA, but tests of interactions and follow-up regressions indicated that this effect was present for females and negligible for males.

Results of this study are consistent with the hypothesized role of control-related cognitions as mediators of the relationships between early parent and family variables and symptoms of emotional disorders. The effect of paternal care on GD/NA was accounted for, in part, by both current attachment quality and perceived uncontrollability. These results are consistent with the notion that early lower care may contribute to feelings of uncontrollability, lower mastery, and helplessness, and difficulties feeling secure in interpersonal relationships. Indeed, these variables may mediate the impact of paternal care on the development of non-specific symptoms of emotional disorders in late adolescence, but longitudinal studies would be needed to fully test such mediational models.

In terms of the other symptom scales, low levels of maternal care were related to higher more symptoms of AD, and poorer quality current attachment and perceived uncontrollability helped to account for this relationship. None of the tests for indirect effects on the relationship between overprotection and the symptom scales were significant. Finally, perceived family conflict was related to AA, and pessimistic attributional style helped to account for this relationship in females but not in males. Although this was not an a priori prediction, it is perhaps not surprising that a family environment that is high in conflict (and maybe violence) would be associated with more physiological symptoms of arousal in females. Results do suggest that participant gender is important in fully understanding the relationship between family environment, cognitive variables, and those symptoms of anxiety specific to anxiety/panic.

There are several methodological limitations of this study shared by many other related studies in the literature. First, our sample was an unselected college student sample and the results should ideally be replicated with a clinical sample of both depressed and anxious adults to determine whether similar relationships would emerge in predicting clinically significant levels of anxiety and depression. Second, research that examines adults' reports of parental behaviors may be influenced by recall biases (Gerlsma et al., 1990). Evidence from a number of studies, however, does support the general validity of the PBI (e.g., high test-retest reliability over intervals of as long as 10 years, high correlations between reports of siblings and twins, correlations between PBI ratings and the results of detailed interviews with respondents and parents, and correlations between PBI reports by adult subjects and their mothers' own PBI scores; Parker, 1989). Nevertheless, the current study did not obtain reports to corroborate participants' reports of their family environments as would have been ideal to rule out recall bias in this sample.

An additional limitation is that our theoretical understanding of the constructs examined as potential mediators and how these variables relate to each other is somewhat limited. The study of personal control has relied on constructs such as *mastery*, *controllability*, and *attributional style*, but just how these constructs converge is less clear. From our attempts to examine the factor structure of the locus of control and attributional style dimensions together, it was difficult to integrate the scales, which might be an artifact of the different response methods used by these scales. But nonetheless this issue remains unresolved. Moreover, the study of adult attachment is largely an extrapolation from the infant and child literature, and some have argued that it is unclear to what extent measures attempting to assess *attachment* adequately represent the construct (see Sperling et al., 1996).

Another limitation is that the tripartite model of negative emotions does not include factors specific to any anxiety disorders other than panic disorder (e.g., Mineka et al., 1998; Zinbarg & Barlow, 1996). Thus, further research will be necessary to clarify the relations among early family environment variables, parent and participant gender, control-related cognitions, current attachment and these other anxiety symptom dimensions.

A final limitation of the present study is its correlational nature. Consequently, it cannot indicate the causal status of any of the observed relationships. For example, one cannot determine from the results whether childrearing practices lead to anxiety and depression, or whether early symptoms of anxiety and depression in offspring might elicit certain patterns of childrearing. Additionally, it is unclear from the regression analyses and the self-report nature of the dependent measures whether some unknown underlying additional variables might account for the shared variance among the parent and family variables, the cognitive and attachment variables, and the symptom scales (e.g., shared genetic variance or overall negative response bias).

Despite these limitations, interesting relationships between parent variables and symptoms scales did emerge. Results suggest that reports of paternal overprotection predict anxious arousal for males, while low maternal care predicts anhedonic depression for both males and females. These findings are consistent with studies that found that a larger amount of variance in depression scores is explained by perceived parental rejection (or low care) than by perceived parental overprotection (e.g., Parker & Hadzi-Pavlovic, 1984; Whisman & Kwon, 1992). Results are also consistent with findings that a family environment characterized by limited opportunity for personal control (high overprotection) is associated with anxiety (see Chorpita & Barlow, 1998; Rapee, 1997), although parent and participant gender was important in understanding this relationship.

In conclusion, the relationship of both maternal and paternal variables to symptoms of emotional disorders observed in the present study have been relatively understudied. The fact that reports of low maternal and paternal care were significant unique predictors of different symptom dimensions after controlling for the influence of the others suggests that the functional significance of mother and father care may be somewhat different. Just what father and mother care contribute to the picture is not well understood, but the current findings support Phares and Compas' (1992) conclusions that it may be important to keep these variables separate in future studies in this area. Further research might also consider the implications of our findings that a perceived ''family conflict'' variable was a predictor of anxious arousal above and beyond the other predictors. These findings are consistent with the work of Parker et al. (1997) suggesting that conflict and intrusiveness appear to be important in the theoretical model for risk for the development of anxiety/panic. Finally, results of this study suggest that studies attempting to examine the specificity of care and overprotection to anxiety and depressive symptoms should consider the tripartite model as a useful framework for studying predictors of specific and nonspecific components of anxiety/panic and depression.

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