Published in final edited form as:

J Child Fam Stud. 2017 February ; 26(2): 591–602. doi:10.1007/s10826-016-0575-6.

Early Pubertal Timing and Childhood Family Adversity Interact to Predict Newlywed Women's Anxiety Symptoms

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Abstract

The contextual amplification hypothesis posits that girls' early pubertal timing will predict anxiety and depression symptoms most strongly when early puberty occurs under adverse conditions. Research supporting this hypothesis has consistently linked early pubertal timing occurring in adverse contexts to symptoms during adolescence, but little is known about the link to adult symptoms. The present study examined the extent to which women's reports of early pubertal timing and childhood family adversity interact to predict symptoms of anxiety and depression during the first two years of marriage. Married women ($N = 226$) completed questionnaires within 7 months into their first marriage (Time 1) and approximately 19 months later (Time 2). Analyses indicated that at both Time 1 and 2, women's reports of earlier pubertal timing predicted anxiety symptoms only when women reported a history of greater childhood family adversity. Additional analyses indicated that the interaction of earlier pubertal timing and greater childhood family adversity predicted symptoms of traumatic intrusions and panic, but not social anxiety, at Time 1, and panic symptoms at Times 1 and 2. These findings expand our understanding of the relation of early pubertal timing to adult emotional health and the family conditions that moderate this relation.

Keywords

early pubertal timing; childhood family adversity; anxiety; depression; newlywed women

Introduction

Puberty is a dramatic transitional period in girls' development, triggering rapid biological and social change, and increasing risk for psychological health problems (Mendle, 2014b). There are significant inter-individual differences in the timing of pubertal development (e.g., Belsky, Steinberg, & Draper, 1991), and the maturation disparity hypothesis suggests that girls who enter puberty earlier than their peers are at higher risk for developing symptoms of anxiety and depression (Ge & Natsuaki, 2009). This vulnerability is hypothesized to occur because early developing girls may be more frequently placed in social situations that are inappropriate for their cognitive and emotional development (e.g., Mendle, Harden, &

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Conflict of Interest: The authors declare that they have no conflict of interest.

Brooks-Gunn, 2010). These girls are unprepared to cope with increased pressures that result from being perceived as older and more mature than they actually are because they have a significant “maturation gap” between their physical and psychosocial maturity (Rudolph, Troop-Gordon, Lambert, & Natsuaki, 2014; Sontag, Graber, Brooks-Gunn, & Warren, 2008). Research over the past two decades, however, has been inconsistent with regard to whether there is a simple main effect between early pubertal timing and symptoms of anxiety and/or depression in adolescence. Several reviews of this work have emphasized that additional research is needed to clarify the social circumstances that affect the strength of the relation between early pubertal timing and psychopathology (Galvao et al., 2013; Negri & Susman, 2011; Reardon, Leen-Feldner, & Hayward, 2009).

The contextual amplification hypothesis directly addresses this paucity of main effects by suggesting that girls’ early pubertal development is more strongly linked to anxiety and/or depression symptoms when early puberty occurs in the context of psychosocial adversity (Ge & Natsuaki, 2009). In this view, early developing girls are likely to be in need of a supportive and protective social environment to help them cope successfully with the added stress of the maturation gap (Mendle, Leve, Van Ryzin, & Natsuaki, 2013; Sontag, Graber, Brooks-Gunn, & Warren, 2008). Thus, although early pubertal timing alone is not a consistent predictor of concurrent or later anxiety and/or depression symptoms for girls, early puberty is a potent risk factor for anxiety and depression symptoms in adolescence when it occurs within stressful social contexts (e.g., Negri & Susman, 2011; Rudolph & Troop-Gordon, 2010; Winer, Parent, Forehand, & Bresland, 2015).

In conjunction with the contextual amplification hypothesis, other research on pubertal timing and contextual stress posits that the childhood family environment may be an important predictive factor of girls’ pubertal timing (e.g., Belsky, Steinberg, & Draper, 1991). Specifically, a growing body of work indicates high levels of childhood adversity are more often associated with earlier as opposed to later pubertal development (see Ellis, 2004 for a review). Some research demonstrates that girls who grow up in environments defined by neglect (low parental investment), abuse (including childhood sexual abuse), and general chaos are more likely to experience earlier pubertal timing whereas girls who grow up with cohesive family relationships and higher frequency of contact with biological parents are more likely to experience later pubertal timing (e.g., Bergevin, Bukowski, & Karavasilis, 2003; Boynton-Jarrett et al., 2013; Ellis, 2004; Zabin, Emerson, & Rowland, 2005). Thus early pubertal timing occurring in the context of family stress may be conceived as an evolved and potentially adaptive response to childhood family adversity, as earlier onset of sexual maturity increases the timeline in which a female can mate and bear children across the lifespan and thereby improve evolutionary fitness (e.g., Saxbe & Repetti, 2009). But evolutionary fitness in one domain (i.e., earlier sexual maturity) may entail tradeoffs in another (i.e., psychological health) and in contemporary social environments early pubertal timing in the context of childhood family stress may be a key environment that engenders negative psychological symptoms during adolescence (e.g., symptoms of depression and anxiety) (e.g., Ellis, Figueredo, Brumbach, & Schlomer, 2009).

In contrast to this understanding of adolescent developmental outcomes, we know very little about the interactive effects of pubertal timing and childhood context on later adult anxiety

and depressive symptoms. The few studies that examine direct effects of early puberty on adult symptoms are inconsistent, with one finding no relation to depressive symptoms at age 21 (Foster, Hagan, & Brooks-Gunn, 2008), one finding a relation to anxiety and depression diagnoses in a clinical sample at age 24 (Graber, Seeley, Brooks-Gunn, & Lewinsohn, 2004), and another finding associations between retrospectively reported early and late pubertal timing to a range of lifetime anxiety disorders (Weingarden & Renshaw, 2012). Only one study (Copeland et al., 2010) examined adversity moderators of young adult outcomes, finding that early pubertal timing in a context of adolescent conduct disorder predicted depressive diagnoses at ages 19–21.

Furthermore, while the direct relation between childhood family adversity and later anxiety or depressive symptoms in adulthood is quite robust (for a review see Taylor, Lerner, Sage, Lehman, & Seeman, 2004), the literature has rarely examined whether family adversity broadly predicts symptoms of dysphoria (non-specific symptoms of depression and anxiety) or may be more symptom specific to anxiety or/and depression. Moreover, the majority of research on the direct relation of early pubertal timing to affective symptoms has primarily focused only on depressive symptoms or on a non-specific amalgam of internalizing or general dysphoria symptoms (e.g., Negri & Susman, 2011). The tripartite model of anxiety and depression proposes that high negative affect defines both depression and anxiety, whereas low positive affect and loss of interest or pleasure are unique to depression, and somatic tension and physiological hyperarousal are unique features of anxiety (e.g., Clark & Watson, 1991). More contemporary explorations of this model have divided anxiety and depression symptoms into “fear” (predominantly symptoms found in anxiety and physiological hyperarousal) and “distress” (predominantly symptoms found in depression) (e.g., Watson, 2005). Exploring the interactive effects of childhood family adversity and pubertal timing on both anxiety and depression symptoms in a single adult sample may help determine if the relation of childhood family adversity and early pubertal timing to general dysphoria can be further extrapolated to independent anxiety or depression symptom clusters and thereby improve prevention and intervention efforts.

Importantly, when studying the etiology of depression and anxiety in women, the major life transition of marriage may be a unique and underexplored window of mental health vulnerability (e.g., Brock & Lawrence, 2011; Brock & Lawrence, 2014; Gottman, 1999; Lavner, Karney, & Bradbury, 2014). Women’s psychological health in the first years of marriage is an essential factor in predicting later marital satisfaction and divorce, as well as diverse physical and mental health outcomes (e.g., Birditt, Brown, Orbach, & McIlvane, 2010; Caughlin, Huston, & Houts, 2000; Huston & Caughlin, 2001; Kiecolt-Glaser, Bane, Glaser, & Malarkey, 2003; Whitton et al., 2007). Although child and adolescent experiences in family relationships are known to influence women’s emotional health in the context of marriage (e.g., Busby, Walker, & Holman, 2011; Roisman, 2007), we know very little about whether childhood family adversity exacerbates effects of early pubertal timing for women’s mental health during the important family transition of early marriage. Examining the predictive effects of interactions between early pubertal timing and childhood family adversity in the current study provides a novel test of the contextual amplification model of pubertal timing in an adult sample at a vulnerable life transition, and may provide a

foundation for further examination of these processes on long-term anxiety and depression within marriages.

The present study examined women's reports of their childhood family environments as a moderator of the relation of early pubertal timing to their anxiety and depressive symptoms within the first seven months of marriage, and whether these symptoms persisted approximately 19 months later. A primary purpose of this study was to expand our understanding of the moderating effects of family environments and pubertal timing on anxiety and depressive symptoms occurring many years after the end of puberty. We hypothesized that women who reported experiencing early pubertal timing would evidence greater symptoms of anxiety and depression during the early years of marriage but only when they also had experienced greater childhood family adversity. We examined a community sample of women because although adverse family environments may include extreme cases of neglect and abuse, varying levels of adverse family dysfunction are present across families. Studying the outcomes of natural variation of family risk in community samples (in contrast to only targeting samples of maltreated individuals) can facilitate our broader understanding of the full range of the impact of common family dynamics and dysfunction. Furthermore, we specifically set out to explore a community sample of newlywed women in the first stages of forming their own families — an important and underexplored window of anxiety and depression vulnerability for women (e.g., Huston & Caughlin, 2001). Finally, an additional aim of the study was to clarify whether early pubertal timing in the context of family adversity is best characterized as a risk for general, non-specific symptoms of dysphoria in adulthood or a risk for specific types of anxiety or depressive symptoms.

Method

Participants

Data were obtained from a larger longitudinal study, the [name of project removed for masked review] (for details, see [citation removed for masked review]). Adult women who were married for the first time (n at Time 1 = 226, n at Time 2 = 203) and living in New England ($M_{age} = 27.70$, $SD = 4.80$, 92% White) were recruited with their husbands from marriage license records to participate in a short-term longitudinal study investigating mental and physical health in early marriage. The first study visit (Time 1) occurred within the first seven months of marriage; the second visit (Time 2) was approximately 19 months later. At Time 1, no women in the sample had children (a condition of eligibility for entry into the study). As of Time 2, 49 women had given birth to a child.

Procedures

All study procedures were approved by the Institutional Review Board at [name of University removed for masked review]. All participants were consented before participating in any study procedures in accordance with the approved IRB. Participants completed a telephone screen and, if qualified, were scheduled for lab visits. For each of the two experimental sessions, couples separately answered a series of computerized questionnaires, which were the focus of the current analyses. Participants were seated in a comfortable, but

stimulus-neutral room, and monitored by a research assistant. Participants also engaged in other tasks, such as providing saliva samples and discussing a disagreement, that were not the focus of the current research.

Measures

Perceived Childhood Family Adversity—The 13-item Risky Families Questionnaire (Taylor et al., 2004), administered at Time 1, was used to retrospectively assess total perceived abuse, neglect, family conflict, and household disorganization from ages 5 through 15. The Risky Families Questionnaire, which was adapted from the Adverse Childhood Experiences (ACE) instrument, was designed to assess the relation of childhood experiences, including family stress, to mental and physical health outcomes in adulthood (Felitti et al., 1998). The Risky Families Questionnaire is a gold standard for parsimonious retrospective assessment of adverse childhood experiences when prospective data are unavailable (e.g., Carroll et al., 2013; Cho, Bower, Kiefe, Seeman, & Irwin, 2012; Maleck & Papp, 2015). The Risky Families Questionnaire and the broader ACE measure have been reliably correlated with adverse mental and physical health outcomes in adulthood across diverse samples (Carroll et al., 2013; Dube et al., 2001; Dube et al., 2009; Dube et al., 2005). The Risky Families Questionnaire has additionally been validated against clinical interviews of individuals' experiences during childhood conducted and coded by trained clinical interviewers (Taylor et al., 2004, 2006). In the present study, participants rated aspects of their childhood family environment on a series of 5-point Likert scales ranging from 1 (not at all) to 5 (very often), with items related to *neglect* (e.g., “How often would you say you were neglected while you were growing up, that is, left on your own to fend for yourself?”), *family conflict* (e.g., “How often would you say there was quarreling, arguing, or shouting between your parents?”), *abuse* (e.g., “How often did a parent or other adult in the household push, grab, shove, or slap you?”), and *household disorganization* (e.g., “Would you say the household you grew up in was chaotic and disorganized?”). Positively worded items were reverse coded (i.e., “How often did a parent or other adult in the household make you feel that you were loved, supported, and cared for?”). Total scores can range from 13 to 65. In our current sample, Cronbach's α was measured at .87.

Perceived Pubertal Timing—Perceptions of pubertal timing were assessed by retrospective self-report using the pubertal timing item from the Pubertal Development Scale (PDS) (Petersen, Crockett, Richards, & Boxer, 1988). The PDS is the most widely used self-report measure of pubertal development and the perceived pubertal timing item is a well-established and effective method for assessing retrospective perceptions of the timing of pubertal changes compared to peers (Coleman & Coleman, 2002; Dorn, 2006; Dubas, Graber, & Petersen, 1991; Negri & Susman, 2011). The specific item read: “Please try to remember when the following occurred: Compared to your same-sex peers (age-mate peers), when would you say you began to experience changes due to puberty, including changes in physical development? Participants rated their relative timing on a scale from 1 (“much earlier than most of my peers”) to 5 (“much later than most of my peers”). This measure is treated as a continuous variable in the current research. For descriptive purposes it was calculated that, 31% of women experienced timing that was a little or much earlier than most of their peers (ratings of 1 or 2), 44% of women experienced pubertal timing at about the

same time as their peers (rating of 3), and the remaining 25% of women experienced pubertal timing that was a little or much later than their peers (ratings of 4 or 5). This distribution is consistent with previous work using retrospective reports of women's perceived pubertal timing compared to peers (e.g., Smith & Powers, 2009; Weingarden & Renshaw, 2012). Of key importance, although other more "objective" methods exist for the assessment of pubertal development (e.g., Tanner staging via physical exam) (see Moore, Harden, & Mendle, 2014), *perceptions* of pubertal timing relative to one's peers appears to be an equally, if not stronger, predictor of later psychological maladjustment, and may uniquely tap into the complex social and cultural value placed on early pubertal development in adolescent social environments (e.g., Dimler & Natsuaki, 2015; Mendle, 2014a; Moore et al., 2014). As a result, the current study specifically used perceptions of pubertal timing as compared to peers as the pubertal variable of interest.

Beck Anxiety Inventory-II—The 21-item, Beck Anxiety Inventory-II (Beck, Epstein, Brown, & Steer, 1988) was used to evaluate current symptoms associated with DSM-IV-TR diagnoses for current anxiety disorders. Scores range from 0 to 63; scores of 8–15 are associated with *mild* anxiety, scores of 16–25 are associated with *moderate* anxiety, and scores of 26–63 are associated with *severe* clinical anxiety (Beck et al., 1988). In our sample, Cronbach's α was measured at .90 for Time 1 and .90 for Time 2.

Inventory of Depressive Symptomatology: Self-Report—To assess for current symptoms of depression, participants answered the 30-item Inventory of Depressive Symptomatology: Self-Report (Rush, Gullion, Basco, Jarrett, & Trivedi, 1996). This measure evaluates all symptoms required for a DSM-IV-TR diagnosis of a major depressive episode, and improves on other standard self-report measures of depression symptomatology because each item assesses a single symptom only and all items are equally weighted. The Inventory of Depressive Symptomatology correlates highly with the Hamilton Rating Scale for Depression ($r = .88$), the Beck Depression Inventory ($r = .93$), and the clinician-rated version of the IDS ($r = .91$) (e.g., Biggs et al., 2000; Rush et al., 1996; Rush, Carmody, & Reimnitz, 2000). Scores can range from 0 to 84; scores of 14–25 are associated with clinically significant *mild* symptoms of depression, scores of 26–38 are associated with clinically significant *moderate* symptoms of depression, and scores of 39 and above are associated with clinically significant symptoms in the *severe* range. Each individual item is rated on a 0–3 scale (higher numbers indicating greater symptom severity). In our sample, Cronbach's α was .87 for Time 1 and .81 for Time 2.

Inventory of Depression and Anxiety Symptoms—To further clarify specific symptom clusters of anxiety and depression, participants answered the 64-item Inventory of Depression and Anxiety Symptoms (Watson et al., 2007, 2008). The Inventory of Depression and Anxiety Symptoms was designed to contain multiple scales assessing specific symptoms of anxiety (e.g., panic, social anxiety, traumatic intrusions) and depression (e.g., insomnia, suicidality, appetite loss). The scales demonstrate strong discriminant validity, strong criterion validity with associated DSM-IV anxiety and mood disorders, and have been used in a broad range of both clinical and community samples (e.g., Watson et al., 2007, 2008). Participants indicated the extent to which they had

experienced symptoms “during the past two weeks, including today” on a 5-point Likert scale ranging from “*not at all*” to “*extremely*.” In the current study, the 10-item *dysphoria* subscale (e.g., “I felt inadequate”) and three subscales measuring anxiety specific symptom clusters were explored in follow-up analyses. Anxiety specific subscales were the 8-item measure of *panic* (e.g., “My heart was racing or pounding”); the 4-item measure of *traumatic intrusions* (e.g., “I had nightmares of something bad that happened”); and the 5-item measure of *social anxiety* (e.g., “I was worried about embarrassing myself socially.”) All subscales were determined to be reliable, and in our sample Cronbach’s α was measured for dysphoria (Time 1 = .88, Time 2 = .88) panic (Time 1 = .84, Time 2 = .77), traumatic intrusions (Time 1 = .78, Time 2 = .75), and social anxiety (Time 1 = .85, Time 2 = .84).

Parent’s highest level of education—Participants’ self-reported parental education was used as a proxy for childhood socioeconomic status and was controlled in all analyses because childhood socioeconomic status can play a significant role in adverse family environments and is important to include in models of childhood family adversity (e.g., Lehman, Taylor, Kiefe, & Seeman, 2005). A 9-point scale was used to measure parent’s highest level of education: 1 = Grade school, or no high school, 2 = Some high school, 3 = High school diploma, 4 = G.E.D., 5 = Associate’s degree 6 = Vocational degree, 7 = Bachelor’s degree, 8 = Master’s degree, 9 = Ph.D., M.D., J.D. Results were consistent when SES was not included as a covariate.

Data Analyses

We tested a series of moderation models in SPSS 22 using the computational tool PROCESS (Hayes, 2013). Childhood family adversity, pubertal timing and their interaction were simultaneously entered into the regression model given that our primary hypothesis was concerned with the interaction rather than conditional effects. When significant interactions emerged, figures were created that illustrated the form of the interaction by depicting the regression lines of the relation between pubertal timing and psychological symptoms at varying levels of childhood family adversity (mean level and 1-/+ SD from the mean). Pubertal timing and childhood adversity were measured at Time 1. Anxiety (Beck Anxiety Inventory-II), depression (Inventory of Depressive Symptoms: Self-Report), and dysphoria (Dysphoria Subscale of Inventory of Depression and Anxiety Symptoms) were measured at Times 1 and 2. Three models were run at each time point for a total of six models. Parent education (a proxy for SES) served as a covariate in all models. Based on our initial findings, follow-up moderation analyses were conducted to examine the interaction between pubertal timing and childhood family adversity on unique anxiety disorder symptom clusters (i.e., panic disorder, PTSD, and social anxiety disorder) using subscales of the Inventory of Depression and Anxiety Symptoms at Time 1 and 2. All independent variables in our analyses were mean centered and interaction terms were calculated within PROCESS. Six participants did not complete all items of the Risky Families Questionnaire and were dropped from analyses.

Results

Descriptive analyses and correlations between all variables are included in Table 1. Based on published clinical assessment standards of the Beck Anxiety Inventory-II (Beck et al., 1988) and the Inventory of Depressive Symptomology: Self-Report (Rush et al., 1996; Rush et al., 2000), at Time 1, 31% (70 of 226) of women presented with clinically significant symptoms of anxiety (mild = 48, moderate = 13, severe = 9) and 31% (70 of 226) of women reported clinically significant symptoms of depression (mild = 55, moderate = 11, severe = 4). At Time 2, 26% of women (53 of 202) reported clinically significant anxiety symptoms (mild = 35, moderate = 10, severe = 8) and 35% (71 of 203) of the women presented with clinically significant depression symptoms (mild = 57, moderate = 13, severe = 1). In all analyses there was a main effect of childhood family adversity on the dependent variable of interest (See Tables 2 and 3). Pubertal timing and parent education exerted no main effects on dependent variables of interest.

At Time 1 and persisting at Time 2, childhood family adversity significantly moderated the relation between pubertal timing and women's anxiety symptoms ($p < .05$ at both time points, see Table 2), but childhood adversity did not moderate the association between pubertal timing and depressive symptoms (Time 1, $F(1,213) = 2.51$, $p = .11$; Time 2, $F(1, 191) = .40$, $p = .53$) or generalized dysphoria symptoms (Time 1, $F(1,213) = .43$, $p = .51$; Time 2, $F(1,190) = .49$, $p = .48$). As displayed in Figure 1, higher levels of childhood family adversity coupled with early pubertal timing predicted greater symptoms of anxiety at Time 1 and at Time 2. Follow-up Johnson-Neyman simple slope analyses of both Time 1 and Time 2 analyses indicated that the relation of early pubertal timing to anxiety was significant only for women (Time 1, $n = 40$; Time 2, $n = 38$) with the highest levels (1 SD above the mean) of childhood family adversity (Time 1, $p = .005$, Time 2, $p = .01$). The interaction between total childhood family adversity and pubertal timing accounted for 1.8% of the variance in anxiety symptoms in women above and beyond childhood family adversity and parent education at Time 1 and 1.9% of the variance at Time 2.

To better understand which specific types of anxiety symptoms may underlie the relation of early pubertal timing and family adversity to anxiety, we ran follow-up moderation models with the three anxiety-specific symptom subscales (traumatic intrusions, panic, and social anxiety) of the Inventory of Depression and Anxiety Symptoms as outcome variables at Time 1 and Time 2. As shown in Table 3, earlier pubertal timing in the context of family adversity predicted higher levels of traumatic intrusions and panic symptoms at Time 1. Panic symptoms persisted at Time 2 as an outcome of the interaction of early pubertal timing and family adversity. Follow up Johnson-Neyman simple slopes analyses indicated that traumatic intrusions (Time 1, $p = .002$) and panic symptoms (Time 1, $p = .002$, Time 2, $p = .004$) were significant only for women with higher levels of childhood adversity (see Figures 2 and 3).

Discussion

Although studies of the direct relation of early pubertal timing to symptoms of anxiety and depression have produced mixed and inconsistent findings, early pubertal timing is a

consistent predictor of adolescent and early adult anxiety and/or depression symptoms when early pubertal timing occurs within adverse contexts (e.g., Blumenthal, Leen-Feldner, Trainor, Babson, & Bunaciu, 2009; Mendle et al., 2013; Rudolph & Troop-Gordon, 2010). The key role of adverse environments as a moderator of early pubertal timing effects is emphasized by the contextual amplification hypothesis (e.g., Ge, Brody, Conger, Simons, & Murry, 2002; Ge & Natsuaki, 2009; Negri & Susman, 2011; Winer et al., 2015). Our study tested a novel application of the contextual amplification hypothesis with an understudied sample of newlywed women for three primary reasons, all aimed at further informing the development of, and highlighting potential intervention targets for, women's psychological health problems.

First, we sought to clarify the role of *family* adversity (as opposed to peer problems or general life stress) as a moderator of the relation of early pubertal timing to anxiety and depression symptoms. Understanding which social contexts may activate early pubertal timing's relation to anxiety or depression may allow policy makers, researchers, and practitioners to work together to more thoughtfully intervene with at-risk girls and women. Secondly, we sought to clarify whether early pubertal timing and adverse family environments predict only anxiety symptoms, only depressive symptoms, or instead predict both syndromes and/or generalized dysphoria. This clarification is important as both psychological and pharmacological treatments for anxiety and depression may contain both distinct and overlapping components (e.g., Garber & Weersing, 2010). Third, because there is very little information on whether early pubertal timing and adverse family environments predict women's anxiety and depression symptoms into adulthood, we examined these relations in a sample of newlywed women, during a developmental window of stress, anxiety, and depression vulnerability (e.g., Brock & Lawrence, 2011; Kiecolt-Glaser et al., 2003; Lavner et al., 2014).

As was anticipated, childhood family adversity was positively associated with anxiety and depressive symptoms at Time 1 (during the first seven months of marriage) and at Time 2 (approximately 19 months later). Early pubertal development alone was not associated with women's symptoms of anxiety or depression, however, consistent with the contextual amplification hypothesis, when early pubertal timing occurred within a context of childhood family adversity, early puberty predicted anxiety symptoms at Time 1 and Time 2 (see Table 2), but did not predict symptoms of depression or general dysphoria at either time point. In follow-up analyses that examined symptom clusters of anxiety subtypes, earlier pubertal timing interacted with family adversity to predict traumatic intrusions at Time 1 and panic symptoms at Time 1 and Time 2 (see Table 3).

Our significant models for global anxiety, panic symptoms (e.g., symptoms associated with Panic Disorder), and traumatic intrusions (e.g., symptoms associated with Post-Traumatic Stress Disorder) can perhaps be best understood within broader research related to the impact of childhood family adversity and other developmental risks on later vigilance and maladaptive stress coping processes into adulthood (e.g., Del Giudice, Hinnant, Ellis, & El-Sheikh, 2012). Children who grow up in abusive, highly conflictual, or neglectful homes may develop highly reactive stress response systems to navigate their unsupportive home environments (e.g., Del Giudice et al., 2012; Repetti, Taylor, & Seeman, 2002). While this

strategy may be adaptive in the short-term for youth growing up in stressful contexts, as children age, their taxed and ultimately dysregulated stress system may promote symptoms of hyperarousal and related anxiety. As girls who experience early pubertal timing are at risk for experiencing more risky social environments (e.g., higher levels of substance use, earlier dating and sexual relationships), and as pubertal onset enhances individual predispositions to emotional processing mechanisms of both threats and rewards (e.g., Quevedo, Benning, Gunnar, & Dahl, 2009) the combination of early pubertal timing and a highly stressful or unsupportive family context may trigger or amplify the development of anxiety symptoms into adulthood. Our research further supports the idea that early pubertal timing may function as a tipping point in the relation between girl's youth adversity and later maladjustment (e.g., Mendle et al., 2013). Relatedly, our work documents that off-time late pubertal timing alone or in the context of family adversity may not be a risk factor for anxiety or depression, however, whether late timing functions as a protective factor, or is simply no different than on-time development, is still contested in the literature and warrants future exploration. Additionally, the fact that anxiety symptoms were present within the first seven months of marriage *and* that symptoms persisted more than a year later is important. While the acute transition to marriage may lead to a short-term increase in anxiety symptoms, symptom persistence more than a year later indicates the potentially lasting effects of the contextual amplification process on anxiety symptoms in adult women.

Finally, using the lens of the tripartite model (Clark & Watson, 1991; Watson, Clark, et al., 1995; Watson, Weber, et al., 1995), our results may indicate one etiological process (i.e., early pubertal timing in the context of family stress) contributing to the physiological hyperarousal of anxiety (i.e., traumatic intrusions and panic) rather than general dysphoria or depression in newlywed women. These outcomes for anxiety (particularly hyperarousal symptoms) are especially interesting as past research on early pubertal timing has more often focused on predicting depressive or dysphoric symptoms rather than anxiety symptoms (e.g., Negri & Susman, 2011; Rudolph, Troop-Gordon, Lambert, & Natsuaki, 2014).

Limitations of this work suggest several directions for future research. Although our measures of childhood family adversity and pubertal timing were designed to be used retrospectively and possess high reliability with related prospective assessment procedures (e.g., Dubas, Graber, & Petersen, 1991; Taylor et al., 2004), prospective, long-term longitudinal designs in future studies that span early childhood through the early years of marriage would eliminate the possibility that participants' current mood states or situations bias their retrospective memories. As this study was specifically focused on women's perspectives, variables in the model were taken from a single reporter. As this is a potential issue of shared method variance, the use of multiple reporters, including partner's perspectives on constructs of interest (which were unavailable in the current research), could strengthen the confidence of findings in future work. Furthermore, future studies could work to integrate measures of chronic developmental stress (e.g., childhood adversity) and acute current adult stress (e.g., marital conflict) to determine if current stressors may partially explain increases in psychological symptoms.

The current sample was limited to women married to men, the majority of whom were white and relatively well-educated. To evaluate the generalizability of our findings to newlywed

women more broadly, it would be important to examine these questions among more diverse samples (e.g., across different race, ethnicity, and income groups, and among women in same-sex marriages) as girl's pubertal timing may have differential effects based on race, ethnicity, and related contextual understandings of puberty (e.g., Deardorff et al., 2013; Hamlat et al., 2014; Natsuaki, 2013). Furthermore, whereas the current study measured a broad domain of childhood family adversity, future research could focus on specific types of adversity (e.g., childhood sexual abuse) which may be especially relevant to early pubertal onset and later psychological health (e.g., Foster et al., 2008; Mendle, et al., 2013; Natsuaki, Leve, & Mendle, 2011). In the current study, childhood adversity was not directly associated with early pubertal timing. This may have been because the vast majority of our community sample of women did not experience extreme childhood stress to the degree that might prompt early pubertal onset. Future research could compare clinical to community samples of women to determine if only individuals who experience the highest levels of childhood family adversity (e.g., court documented childhood maltreatment) experience earlier pubertal onset, if at all. Finally, future research could determine if the relations documented in this study are further modified by the quality of women's adult marital relationships. It is notable, however, that the contextual amplification hypothesis regarding the importance of child and adolescent experiences is supported even in the absence of accounting for current environmental conditions, which were not the focus of this study.

Overall, our findings document the associations between childhood family adversity, perceived pubertal timing compared to peers, and later anxiety symptomology in newlywed women. Our findings may be useful for life course developmental psychopathology researchers by providing further evidence for the contextual amplification model of pubertal timing, particularly possible effects of early pubertal timing on the mental health of women growing up in higher stress family contexts. For clinicians treating girls and women with anxiety symptoms, who additionally report adverse childhood experiences, a sensitivity to pubertal timing and related experiences during adolescent development may be warranted to further inform prevention and intervention efforts.

Acknowledgments

This research was supported by a grant to Paula R. Pietromonaco and Sally I. Powers from the National Cancer Institute of the National Institutes of Health under award number R01CA133908. The content is solely the responsibility of the authors and does not necessarily represent the official views of the National Institutes of Health.

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Figure 1.
Interaction between pubertal timing and childhood family adversity predicts anxiety symptoms at Time 1 (left) and persists at Time 2 (right).

Figure 2.
Interaction between pubertal timing and childhood family adversity predicts panic symptoms at Time 1 (left) and persists at Time 2 (right).

Figure 3.
Interaction between pubertal timing and childhood family adversity predicts traumatic intrusions at Time 1.

Table 1

Descriptive Statistics and Correlation Matrix of All Study Variables

Time 1 Variables	<i>n</i>	<i>SD</i>	<i>M</i>	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
1. Family Adversity	220	9.33	28.06	-														
2. Pubertal Timing	225	.96	2.92	.020	-													
3. Parent Education	223	2.09	6.14	-.089	.051	-												
4. Anxiety	226	7.51	7.01	.269**	-.114	-.043	-											
5. Depression	226	7.60	11.77	.295**	-.052	-.002	.595**	-										
6. Dysphoria	226	6.26	16.93	.257**	-.025	.019	.629**	.752**	-									
7. Social Anxiety	226	3.49	7.47	.244**	-.023	-.023	.489**	.522**	.632**	-								
8. Panic	226	3.05	9.50	.277**	-.096	-.045	.792**	.515**	.554**	.430**	-							
9. Traumatic Intrusions	226	2.43	5.45	.243**	-.115	-.082	.571**	.480**	.570**	.472**	.517**	-						
Time 2 Variables																		
10. Anxiety	202	7.01	6.50	.175*	-.112	-.069	.635**	.610**	.546**	.381**	.520**	.538**	-					
11. Depression	203	7.91	12.16	.290**	-.093	-.083	.561**	.763**	.622**	.446**	.462**	.482**	.708**	-				
12. Dysphoria	202	6.23	16.76	.139	-.038	-.016	.461**	.621**	.639**	.401**	.340**	.396**	.686**	.807**	-			
13. Social Anxiety	202	3.21	7.36	.183**	-.053	-.022	.355**	.544**	.532**	.617**	.311**	.362**	.599**	.619**	.667**	-		
14. Panic	202	2.39	9.30	.205**	-.125	-.018	.514**	.504**	.469**	.400**	.536**	.403**	.734**	.576**	.578**	.612**	-	
15. Traumatic Intrusions	202	1.92	5.08	.207**	-.012	-.098	.278**	.350**	.366**	.353**	.190**	.475**	.510**	.468**	.503**	.467**	.385**	-

Note. Family Adversity = Risky Families Questionnaire. Anxiety = Beck Anxiety Inventory-II. Depression = Inventory of Depressive Symptomatology: Self-Report. Parent Education = Participants' parents' highest level of education. Dysphoria = Inventory of Depression and Anxiety Symptoms Dysphoria Subscale. Social Anxiety = Inventory of Depression and Anxiety Symptoms Social Anxiety Subscale. Panic = Inventory of Depression and Anxiety Symptoms Panic Subscale. Traumatic Intrusions = Inventory of Depression and Anxiety Symptoms Traumatic Intrusions Subscale.

* $p < .05$.

** $p < .01$.

Moderation Model Predicting Women's Anxiety Symptoms from the Interaction of the Risky Families Questionnaire and Pubertal Timing at Time 1 and Time 2

Table 2

Variables	b	SE	R ²	F	Change in R ²	F Change in R ²
ANXIETY SYMPTOMS AT TIME 1						
Parent Education	-.07	.24				
Family Adversity	.23 ***	.05				
Pubertal Timing	-.87	.51				
Family Adversity X PT	-.13 *	.06	.11 ***	6.30 ***	.018 *	4.25 *
ANXIETY SYMPTOMS AT TIME 2						
Parent Education	-.22	.24				
Family Adversity	.13 *	.05				
Pubertal Timing	-.74	.52				
Family Adversity X PT	-.12 *	.06	.07 *	3.38 *	.019 *	3.92 *

Note. Parent Education = Participants' parents' highest level of education included as covariate. Family Adversity = Risky Families Questionnaire. PT = Pubertal Timing. Anxiety Symptoms = Beck Anxiety Inventory-II.

* $p < .05$.

** $p < .01$.

*** $p < .001$.

Table 3

Moderation Model Predicting Women's Anxiety Specific Symptoms (Panic and Traumatic Intrusions) from the Interaction of the Risky Families Questionnaire and Pubertal Timing at Time 1 and Time 2

Variables	b	SE	R ²	F	Change in R ²	F Change in R ²
PANIC SYMPTOMS AT TIME 1						
Parent Education	-.0397	.0954				
Family Adversity	.095***	.021				
Pubertal Timing	-.266	.207				
Family Adversity X PT	-.071**	.025	.120***	7.2632***	.0343**	8.2938**
TRAUMATIC INTRUSIONS AT TIME 1						
Parent Education	-.0834	.0763				
Family Adversity	.0658***	.0171				
Pubertal Timing	-.2200	.1658				
Family Adversity X PT	-.0576**	.0197	.1085***	6.4788***	.0359**	8.5786**
PANIC SYMPTOMS AT TIME 2						
Parent Education	-.0189	.0810				
Family Adversity	.0549**	.0187				
Pubertal Timing	-.2670	.1766				
Family Adversity X PT	-.0497*	.0213	.0838***	4.3444**	.0262**	5.4239**

Note. Parent Education = Participants' parents' highest level of education included as covariate. Family Adversity = Risky Families Questionnaire. PT = Pubertal Timing. Panic = Inventory of Depression and Anxiety Symptoms Panic Subscale. Traumatic Intrusions = Inventory of Depression and Anxiety Symptoms Traumatic Intrusions Subscale.

* $p < .05$.

** $p < .01$.

*** $p < .001$