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The Role of Puberty in the Development of Depressive Symptoms into Young Adulthood

A dissertation submitted in partial satisfaction of the requirements for the degree

Doctor of Philosophy in Psychology

by

Sarah Madeline Thompson

2017

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ABSTRACT OF THE DISSERTATION

The Role of Puberty in the Development of Depressive Symptoms into Young Adulthood

by

Sarah Madeline Thompson

Doctor of Philosophy in Psychology

University of California, Los Angeles, 2017

Professor Constance L. Hammen, Chair

In addition to the biological and hormonal changes that characterize puberty, the pubertal transition is accompanied by significant psychological and social challenges. Research has established that pubertal experiences can lead to psychopathology among youth, with most studies examining the role of pubertal timing relative to peers. However, less is known about the mechanisms through which off-time development contributes to psychopathology, the impact of characteristics of puberty other than timing, and the influence of pubertal processes on mental health beyond adolescence. The present dissertation sought to explore these issues via three related studies. Given the marked increase in rates of depression during adolescence as well as the emergence of a gender disparity in depression prevalence that persists into adulthood, the dissertation focused on the association between pubertal experiences and depressive symptoms.

Study 1 utilized a large, community sample to test four psychosocial mechanisms that have been proposed to account for the link between off-time development and depressive

symptoms among youth. The results supported the stage termination, personal accentuation, and contextual amplification hypotheses among females, but did not support any hypotheses among males. Study 2 further explored contextual amplification by assessing which types of environmental stress exacerbate the effects of off-time development. Chronic interpersonal stressors interacted with pubertal timing among females while dependent episodic stressful events were associated with the highest level of depressive symptoms among early-maturing males. Study 3 examined whether pubertal synchrony, the degree to which various markers of puberty mature simultaneously, was associated with depression among females. Evidence suggested that asynchronous development led to the highest levels of depressive symptoms in young adulthood, particularly among late-maturing girls.

Together, the results of the dissertation indicate that females are particularly vulnerable to the influence of pubertal experiences on mental health and suggest several mechanisms through which puberty influences depression among females. Males appear to be less likely to react to variations in pubertal timing by becoming depressed. The findings suggest several pathways for future research including the integration of proposed biological and psychosocial mechanisms and the continued exploration of pubertal processes as a contributor to the gender gap in depression prevalence.

The dissertation of Sarah Madeline Thompson is approved.

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Selected Presentations

- Thompson, S. M., Hammen, C., & Brennan, P. A. (2016, October). *Contextual Amplification of Environmental Stress in the Association between Pubertal Timing and Depressive Symptoms*. Poster presented at the annual meeting of the Association of Behavioral and Cognitive Therapies, New York, NY.
- Thompson, S. M., Hammen, C., & Brennan, P. A. (2015, November). *Asynchronous Pubertal Development Predicts Depressive Symptoms in Young Adulthood*. Poster presented at the annual meeting of the Association of Behavioral and Cognitive Therapies, Chicago, IL.
- Thompson, S. M., Hammen, C., & Brennan, P. A. (2014, November). *Longitudinal Effects of Early Life Stress and Pubertal Timing in Depressive Symptoms: Late Pubertal Timing as a Risk Factor*. Poster presented at the annual meeting of the Association of Behavioral and Cognitive Therapies, Philadelphia, PA.
- Thompson, S. M., Hammen, C., & Brennan, P. A. (2013, November). *The Role of the Oxytocin Receptor Gene in the Association between Maternal Depression in Early Childhood and Offspring Depressive Symptoms*. Poster presented at the annual meeting of the Association of Behavioral and Cognitive Therapies, Nashville, TN.

Chapter 1: General Introduction

The transition to adulthood is marked by a set of interconnected biological, psychological, and social changes that delineate the boundary between youth and maturity. Of these changes, the occurrence of puberty is perhaps the most universally recognized sign of the transformation from child to adult. The pubertal transition is comprised of a variety of related experiences including biological and hormonal shifts, alterations in physical appearance, and social and psychological adjustments related to the increasing prominence of peer relationships and changes in sense of self. Although puberty is a universal developmental experience, individual variation in the pubertal experience as well as the myriad stresses associated with pubertal development can dramatically affect both short- and long-term outcomes across a variety of life domains.

The impact of various characteristics of the pubertal transition on mental health has been a growing area of research in the last several years. Characteristics of the pubertal experience have been shown to contribute to the development of psychopathology in adolescence (Mendle & Ferrero, 2012; Mendle, Turkheimer, & Emery, 2007; Graber, Lewinsohn, Seeley, & Brooks-Gunn, 1997) as well as adulthood (Graber, Seeley, Brooks-Gunn, & Lewinsohn, 2004; Gaysina, Richards, Kuh, & Hardy, 2015). However, much additional research is needed in order to fully elucidate the impact of puberty on psychological functioning, particularly with regard to the development of psychopathology. Relatively little is known about the mechanisms through which characteristics of the pubertal transition affect risk for psychopathology, including the biological, psychological, and social factors that may interact with elements of the pubertal experience to promote poor psychological functioning (Ge & Natsuaki, 2009; Rudolph, 2014). Additionally, the majority of studies have focused on a single characteristic of puberty without

regard for other pubertal features that may influence subsequent well-being (Mendle, 2014). Finally, most studies in this area have focused on the short-term effects of puberty, examining psychological outcomes solely in adolescence, while relatively few have explored the persistence of effects into young adulthood (Rudolph, 2014). Research that explores the mechanisms of observed associations between puberty and psychopathology, examines a wider array of pubertal characteristics, and focuses on long-term risk for psychopathology into adulthood is greatly needed.

Previous research exploring the impact of the pubertal transition on psychopathology has suggested that variations in puberty are associated with a variety of mental health conditions, including both internalizing and externalizing disorders (Mendle & Ferrero, 2012; Mendle *et al.*, 2007; Dimler & Natsuaki, 2015; Negriff & Susman, 2011; Rudolph, 2014). Among internalizing disorders, the influence of puberty on the development of depression has been the most consistently studied (Graber, 2013; Angold, Costello, & Worthman, 1998; Ge, Conger, & Elder, 2001a). However, there is also support for associations between the experience of puberty and eating disorders (Zehr, Culbert, Sisk, & Klump, 2007; McCabe & Ricciardelli, 2004) as well as a more limited group of findings linking characteristics of puberty with anxiety symptoms (Winer, Parent, Forehand, & Lafko Breslend, 2016; Weingarden & Renshaw, 2012; Blumenthal, Leen-Feldner, Trainor, Babson, & Bunaciu, 2009). Additional research has examined the association between characteristics of the pubertal transition and outcomes such as substance use (Castellanos-Ryan, Parent, Vitaro, Tremblay, & Seguin, 2013; Biehl, Natsuaki, & Ge, 2007; Wiesner & Ittel, 2002; Graber *et al.*, 1997) and delinquent behavior (Mrug *et al.*, 2014; Negriff, Susman, & Trickett, 2011; Haynie, 2003).

Pubertal Timing and Depressive Symptoms

The development of depressive symptoms has been of particular interest with regard to puberty due to the dramatic increase in rates of depression in adolescence (Hankin *et al.*, 1998) as well as the emergence of a gender gap in depression prevalence during these years that persists for much of adulthood (Kessler, 2003; Kuehner, 2003; Kessler, McGonagle, Swartz, Blazer, & Nelson, 1993). Given the large number of both men and women who are diagnosed with at least one lifetime episode of depression (Kessler *et al.*, 1993), examining factors that contribute to the development of depressive symptoms is especially important, particularly when one such factor may be the universal experience of puberty (Angold & Worthman, 1993; Angold *et al.*, 1998; Cyranowski, Frank, Young, & Shear, 2000; Conley, Rudolph, & Bryant, 2012; Conley & Rudolph, 2009).

Among studies examining the association between puberty and depressive symptoms, most have focused on a single characteristic of the pubertal transition: the timing of puberty relative to peers. Among females, the preponderance of evidence supports an association between early puberty and depression. Early-maturing girls exhibit higher levels of general psychological distress, including depressive symptoms, than on-time or late-maturing peers (Ge, Conger, & Elder, 1996; Siegel, Yancey, Aneshensel, & Schuler, 1999; Stice, Presnell, & Bearman, 2001; Kaltiala-Heino, Kosunen, & Rimpela, 2003; Ge *et al.*, 2003; Benoit, Lacourse, & Claes, 2013). Girls who mature earlier than peers also exhibit the highest levels of internalizing symptoms (Kaltiala-Heino, Marttunen, Rantanen, & Rimpela, 2003; Hayward *et al.*, 1997) and demonstrate the highest lifetime prevalence of major depression in adolescence (Graber *et al.*, 1997). Evidence from two longitudinal studies suggests that early-maturing girls are more likely to be depressed in young adulthood (Copeland *et al.*, 2010) and have a higher lifetime prevalence rate of depression (Graber *et al.*, 2004), although some evidence has

indicated that depression in young adulthood may be explained by adolescent conduct disorder rather than pubertal timing (Copeland *et al.*, 2010). A small body of research has also suggested that both early and late maturation may lead to increased risk of depressive symptoms in girls (Graber *et al.*, 1997; Natsuaki, Biehl, & Ge, 2009; Rudolph, 2014), highlighting the importance of examining potential curvilinear effects of pubertal timing.

Among males, the association between pubertal timing and risk for depression is less clear (Mendle & Ferrero, 2012). Some studies have suggested that early timing is associated with higher risk. For example, boys who mature earlier than peers have been shown to exhibit higher levels of depressive (Negri, Fung, & Trickett, 2008) and internalizing symptoms (Natsuaki, Klimes-Dougan, *et al.*, 2009; Ge, Conger, & Elder, 2001b). A study of African-American youth found that early-maturing boys exhibited significantly higher levels of depressive symptoms than on-time and late-maturing boys (Ge, Brody, Conger, & Simons, 2006). However, most of the studies linking early maturation to depressive and internalizing symptoms in boys examined mixed-gender samples and failed to conduct analyses separately by gender, suggesting that the obtained effects may have been driven by the strong association between early timing and depression in females.

Other research has supported the role of late timing in increasing risk for depression among boys. One study found that boys who perceived themselves as maturing later than peers demonstrated higher levels of depressive symptoms than on-time or early-maturing boys (Siegel *et al.*, 1999). Graber *et al.* (2004) reported that late-maturing males endorsed elevated depressive symptoms in young adulthood, and a longitudinal birth cohort study found that late timing in males was associated with higher levels of depressive and anxiety symptoms beginning in adolescence and continuing into adulthood (Gaysina *et al.*, 2015). Additional research has

indicated that off-time maturation more generally (e.g., either early or late timing) is linked with depressive symptoms in males (Graber *et al.*, 1997; Kaltiala-Heino, Kosnunen, *et al.*, 2003; Conley & Rudolph, 2009; Natsuaki, Biehl, *et al.*, 2009; Benoit *et al.*, 2013), although Conley and Rudolph (2009) found that late timing was associated with the highest levels of depressive symptoms overall (e.g., early-maturing boys showed higher levels of symptoms than on-time boys, but less than late-maturing youth). Additionally, two studies did not observe a link between timing and depressive symptoms in boys (Crockett, Carlo, Wolff, & Hope, 2013; Kaltiala-Heino, Marttunen, *et al.*, 2003).

Studies of puberty among females are generally of high quality and present a consistent case for an association between early timing and the development of depressive symptoms, with additional research needed to elucidate whether both early and late timing are associated with an elevated risk of depression in girls. Among males, published research has tended to support an association between timing and depressive symptoms, although the direction of the effect has yet to be established conclusively, with the majority of well-designed studies suggesting that late or both early and late timing are associated with increased depression. However, the vast majority of pubertal research examines simple associations between timing and symptoms with no clarification of the mechanisms of this effect. Data elucidating *why* off-time development is associated with depressive symptoms are sorely lacking.

Hypothesized Mechanisms of the Association between Puberty and Depressive Symptoms

Several hypotheses have offered explanations for the association between puberty and depressive symptoms, but empirical tests of these proposed mechanisms are limited. Consistent with the types of changes experienced during puberty, hypothesized mechanisms generally fall into one of two categories: (1) biological, or (2) psychosocial. To date, most theoretical work has

focused on pubertal timing and has attempted to explain why variations in timing might increase risk for psychopathology.

Although a plethora of biological and hormonal changes occur during the pubertal transition, the search for biological mechanisms has proven complex because proposed mechanisms must account for observed associations between timing and psychopathology. Thus, mechanisms cannot refer solely to normative biological processes that all adolescents experience during development. One proposed biological pathway through which puberty might lead to increased risk of depressive symptoms relates to the consequences of varying maturation rates among brain structures. The *temporal mismatch* hypothesis suggests that a discrepancy in the maturation of neural systems involved in affective processing and those associated with executive functioning can result in increased emotional reactivity in adolescence (Steinberg, 2005; Casey, Jones, & Somerville, 2011). Brain structures responsible for executive functioning and cognitive control (e.g., prefrontal cortex) are not fully developed until early adulthood and appear to mature independently of pubertal processes (Luna, Garver, Urban, Lazar, & Sweeney, 2004; Steinberg, 2005). Thus, adolescents lack the capacity to fully regulate their emotional responses.

Recent research also suggests that sex hormones associated with pubertal development may affect the connections between the prefrontal cortex and the neural structures associated with emotional processing (e.g., amygdala, ventral striatum), such that emotional reactivity among adolescents undergoing puberty is heightened (Ladouceur, 2012). Therefore, early-maturing youth may be at a particular disadvantage as they will experience increased reactivity to stressors long before they develop the cognitive resources to modulate their emotional responses. Increased reactivity has been hypothesized to account for the rise in the

prevalence of depression and other psychological conditions beginning in adolescence (Dahl, 2004; Ladouceur, 2012) and may therefore represent one mechanism through which variations in pubertal timing affect the development of depressive symptoms. Studies comparing biological measures of emotional reactivity (e.g., pupil dilation, eye blink startle, postauricular reflex) among depressed versus non-depressed youth or among early- versus on-time or late-maturing adolescents have offered some evidence in favor of this perspective (Silk *et al.*, 2009; Quevedo, Benning, Gunnar, & Dahl, 2009), but research explicitly linking pubertal timing to depressive symptoms via variations in biological measures of emotional reactivity is rare. However, one recent study found that pubertal status interacted with cortisol reactivity following a laboratory stress task to predict the onset of depression among adolescent girls (Colich, Kircanski, Foland-Ross, & Gotlib, 2015).

A second biological theory highlights the importance of hormonal shifts that occur as part of pubertal development. While hormonal changes are a normative part of puberty, research suggests that early maturation may be associated with unique hormonal processes. Early-maturing girls exhibit higher levels of follicle-stimulating hormone and estradiol than later-maturing peers (Vihko & Apter, 1984), and this difference persists into early adulthood (Apter, Reinila, & Vihko, 1989). Some research has suggested that the association between puberty and depressive symptoms in girls is fully mediated by changes in levels of estradiol and testosterone (Angold, Costello, Erkanli, & Worthman, 1999). In fact, circulating levels of androgens and estrogens are related to negative emotionality and mood in women across the lifespan (Steiner, Dunn, & Born, 2003). One study found that negative affect among adolescent girls was tied to hormonal levels, such that levels of negative affect were highest during periods when hormone levels were rising (Brooks-Gunn & Warren, 1989). This study also determined that heightened

hormone levels appeared to increase girls' sensitivity to social discord, such that they experienced increased negative affect following difficult interpersonal interactions. Sisk and Zehr (2005) have argued that the rise in gonadal hormones during puberty results in sex-differentiated reorganization of neural structures that can predispose females to develop psychopathology based on variations in pubertal timing, while others have suggested that stressful experiences during puberty may disrupt normative behavioral responses to gonadal hormones, leading to heightened stress reactivity and negative emotionality (Blaustein, Ismail, & Holder, 2016; Holder & Blaustein, 2014). Hormonal shifts represent a promising pathway through which variations in pubertal timing may exert effects on the development of depressive symptoms; however, the majority of studies to date have not obtained clear support for hormonal changes as a mediator of the association between puberty and symptoms of depression.

The final biological hypothesis that has been explored in the literature emphasizes the role of genetics in linking off-time development to psychopathology. Research has suggested that covariation between genes that contribute to the timing of puberty and those associated with risk for mental health conditions may be at least partially responsible for the link between timing and psychopathology. Recent studies have supported genetic covariation between off-time development and substance use (Corley, Beltz, Wadsworth, & Berenbaum, 2015) as well as early menarche and depressive symptoms among girls (Mendle, Moore, Briley, & Harden, 2016), although this result was observed only among females of high socioeconomic status. Other research has explored whether pubertal development might interact with genetic risk factors to promote psychopathology, although at least one study found that pubertal status did not moderate the interaction between chronic peer stress and the serotonin transporter gene in the prediction of depressive symptoms among youth (Hankin *et al.*, 2015). While genetic associations between

puberty and psychopathology offer a promising biological explanation for the link between timing and mental health, existing research is limited and further exploration is greatly needed.

Psychosocial explanations for the connection between puberty and depressive symptoms have also been proposed. The two most prominent emphasize the social and personal significance of off-time development relative to one's peers. The *stage termination* hypothesis highlights the specific risks associated with early pubertal timing. This theory suggests that puberty delineates the boundary between childhood and adolescence, and early-maturing youth are forced to terminate childhood prior to completing the developmental tasks associated with this stage of life (Petersen & Taylor, 1980). Therefore, upon entering adolescence and initiating puberty, early maturers have not yet attained the necessary psychological, interpersonal, and cognitive resources to cope effectively with the stresses of adolescence (Stattin & Magnusson, 1990; Caspi & Moffitt, 1991).

The stage termination hypothesis has primarily been used to explain the experience of girls. For example, early-maturing girls have been shown to engage in dating and sexual activity at a younger age than their later-maturing peers (Kim & Smith, 1998; Flannery, Rowe, & Gulley, 1993), behavior that has been associated with greater prevalence of depression and disordered eating (Kaltiala-Heino, Kosunen, *et al.*, 2003; Kaltiala-Heino, Rimpela, Rissanen, & Rantanen, 2001; Smolak, Levine, & Gralen, 1993). Pubertal development is also thought to distinguish early-maturing girls from their peers, as those who mature early are experiencing physical changes (e.g., menarche, breast and hip development, body hair growth) that set them apart from others of their age. In fact, some research has suggested that early-maturing girls are likely to have lower self-esteem and poorer body image than other adolescents (Williams & Currie, 2000; Benjet & Hernandez-Guzman, 2002). While on-time or late-maturing girls are likely to have

peers who have experienced puberty themselves, early-maturing females may have few friends with whom they can discuss the experience and seek support. Changes in body shape that accompany puberty are also at odds with Western ideals of thinness for women (Rudolph, 2014).

Unfortunately, while explanations consistent with stage termination appear reasonable, empirical tests of these proposed mechanisms are almost nonexistent, and many of the studies cited here are either correlational (Kaltiala-Heino, Kosunen, *et al.*, 2003; Kaltiala-Heino *et al.*, 2001), examine moderators rather than mediators of the link between pubertal timing and depressive symptoms (Smolak *et al.*, 1993), or explore one piece of this association without testing for mediation (e.g., test associations between early timing and body image without examining subsequent links to psychopathology; Williams & Currie, 2000; Benjet & Hernandez-Guzman, 2002). To date, only two studies have provided direct tests of mediation, concluding that early-maturing girls exhibit higher levels of negative self-focus and anxious arousal, reduced engagement coping, and more interpersonal stress than on-time or late-maturing females, which in turn leads to increased depressive symptoms (Rudolph, Troop-Gordon, Lambert, & Natsuaki, 2014; Conley *et al.*, 2012). Thus, while the stage termination hypothesis offers a promising explanation of the observed association between early timing and psychopathology among girls, empirical evidence in support of the mechanisms suggested by stage termination is lacking.

Among boys, the potential costs of early maturation have rarely been discussed. To date, only one study has tested stage termination in males, finding that negative self-focus, anxious arousal, social difficulties, and interpersonal stress mediated the association between early timing and depressive symptoms in boys (Rudolph *et al.*, 2014).

The *maturation disparity* hypothesis, though similar to stage termination, emphasizes the risks of both early and late timing (Petersen & Taylor, 1980). This perspective highlights the

personal significance as well as the interpersonal nature of pubertal development, in that youth undergoing puberty experience dramatic physical changes that are apparent to others and are generally able to compare their own development to the pubertal status of their peers (Simmons & Blyth, 1987). Thus, off-time maturation may set youth apart from their on-time peers and increase their sense of isolation or abnormality, adding an extra burden to the normative psychological stress that all adolescents experience as they undergo puberty (Caspi & Moffitt, 1991).

Though widely hypothesized to be a major contributor to the link between pubertal development and psychopathology, maturation disparity has not been extensively tested (Ge & Natsuaki, 2009). However, some have argued that studies focusing solely on linear effects of puberty tend to conclude that early timing produces the most detrimental outcomes, thereby ignoring the potential negative impact of late maturation that may be evident when examining curvilinear associations (Rudolph, 2014). Research that supports the maturation disparity perspective includes several findings that indicate both early and late timing are associated with higher levels of depressive symptoms than on-time maturation among boys (Kaltiala-Heino, Kosnunen, *et al.*, 2003; Conley & Rudolph, 2009; Natsuaki, Biehl, *et al.*, 2009; Benoit *et al.*, 2013) as well as more limited evidence in girls (Graber *et al.*, 1997; Natsuaki, Biehl, *et al.*, 2009). However, psychological and social pathways of this association have never been tested.

Both the stage termination and maturation disparity hypotheses also highlight potential gender differences in the experience of off-time development. While early-maturing boys are undergoing changes that are consistent with Western cultural ideals of masculinity (e.g., increase in height, increased musculature, deeper voice), early-maturing girls are moving away from ideals of attractiveness (e.g., thinness) and may be exposed to increasing levels of unwanted

sexual attention (Rudolph, 2014). Additionally, because girls generally start puberty one to two years earlier than boys, the earliest maturing girls begin the pubertal transition at a time when no other peers are undergoing the same experience, suggesting that early-maturing girls may be at even greater risk of social isolation and perceptions of abnormality than other off-time developers (Graber, 1997; Negriﬀ & Susman, 2011; Rudolph, 2014). Similarly, late-maturing boys experience puberty at a point when all of their peers (both boys and girls) have already matured, indicating that they may experience a heightened sense of isolation and deviance from others (Negriﬀ & Susman, 2011). Thus, theory suggests that the effects of pubertal timing on psychological well-being may vary greatly by gender. However, as reviewed above, while empirical evidence supports the existence of gender differences in the association between pubertal timing and depressive symptoms, clear distinctions between the psychological and social costs of early versus late development among females as compared to males have not been confirmed empirically (Mendle & Ferrero, 2012; Mendle *et al.*, 2007).

Two additional psychosocial hypotheses highlight the importance of the context in which pubertal development occurs. The *personal accentuation* hypothesis emphasizes the significance of personal attributes of the adolescent in contributing to psychopathology (Caspi & Moffitt, 1991; Ge & Natsuaki, 2009; Rudolph, 2014; Rudolph & Troop-Gordon, 2010). This theory is based on the idea that stressful life circumstances – such as the dramatic changes that youth experience during puberty – are likely to enhance the effects of pre-existing characteristics of the adolescent. Thus, youth with more negative personality traits and fewer psychological resources are more likely to demonstrate negative sequelae of puberty than adolescents possessing more positive characteristics.

Several studies have supported this hypothesis, with the majority examining personal accentuation among females. Caspi and Moffitt (1991) found that girls who demonstrated externalizing behavior in childhood exhibited higher levels of delinquent behavior and aggression in adolescence when they matured earlier than peers. Early-maturing girls without childhood behavior problems as well as on-time maturers with childhood externalizing behavior showed lower levels of problematic behavior at ages 13 and 15. Prior psychological distress (in early adolescence) has also been associated with increased levels of psychological distress later on, including heightened levels of depressive symptoms, among early-maturing girls (Ge *et al.*, 1996). Early pubertal timing has been linked to higher levels of depressive symptoms among adolescents with a prior history of depression, negative self-focus (among girls only), and depressive personality traits (Rudolph & Troop-Gordon, 2010). Additionally, self-regulation skills have been shown to moderate the association between pubertal timing and internalizing symptoms, such that the link between early timing and internalizing was specific to girls with poor self-regulation abilities (Crockett *et al.*, 2013).

The studies supporting personal accentuation to date have utilized longitudinal designs and large sample sizes as well as appropriate measures of relevant constructs. Thus, the evidence in support of personal accentuation is promising. However, explorations of the role of personal accentuation in boys have been less prevalent in the existing literature and only one study has examined the moderating role of personality characteristics in either females or males, despite the specific emphasis on features of personality in the personal accentuation hypothesis.

The *contextual amplification* hypothesis posits that contextual environmental stressors interact with characteristics of the pubertal experience to increase risk for psychopathology, including depression (Caspi, Lynam, Moffitt, & Silva, 1993; Ge & Natsuaki, 2009; Natsuaki,

Samuels, & Leve, 2014). Caspi *et al.* (1993) were the first to provide evidence in support of this idea; they found that the impact of early pubertal timing on delinquent behavior was moderated by features of the educational environment among adolescent girls in New Zealand. Early-maturing girls who attended mixed-sex schools exhibited higher levels of delinquent behavior than early-maturing girls attending same-sex schools. Ge *et al.* (1996) obtained similar results in a study predicting psychological distress among American girls, demonstrating that girls with mixed-sex friendships exhibited higher levels of distress than those with primarily same-sex friendships.

Most studies of contextual amplification have focused on outcomes associated with externalizing behavior, including aggression, substance use, and delinquent behavior (e.g., Sontag, Graber, & Clemans, 2011; Lynne-Landsman, Graber, & Andrews, 2010; Ge, Brody, Conger, Simons, & Murry, 2002; Mrug *et al.*, 2014; Obeidallah, Brennan, Brooks-Gunn, & Earls, 2004). However, research has increasingly explored contextual amplification processes in the prediction of depressive or internalizing symptoms. For example, early pubertal timing has been shown to interact with family stress and maternal depression history to predict adolescent depressive symptoms, such that early-maturing girls and boys exposed to high levels of recent family stress or who have a currently depressed mother show the highest levels of depressive symptoms (Rudolph & Troop-Gordon, 2010). Similarly, early-maturing girls experiencing high levels of stressful life events exhibit the most depressive symptoms in adolescence (Chen, Yu, Wu, & Zhang, 2015; Ge *et al.*, 2001a) while early-maturing boys have been shown to demonstrate the greatest level of internalizing symptoms in the presence of concurrent stressful life events (Ge *et al.*, 2001b).

Conley and Rudolph (2009) found that peer stress moderates the association between pubertal timing and depressive symptoms, such that early-maturing girls and late-maturing boys exhibit the highest levels of concurrent and longitudinal depressive symptoms in the context of high peer stress. A second study also found that peer problems are associated with higher levels of depressive symptoms among off-time youth, although this study concluded that early maturation was associated with the highest level of risk among both females and males (Winer *et al.*, 2016). Dating appears to moderate the association between pubertal timing and depressed mood (Natsuaki, Biehl, *et al.*, 2009) as well, while Benoit *et al.* (2013) determined that early timing led to increased depressive symptoms among boys and girls who perceived more rejection from their parents. Gender-specific effects were also found for dating and friendship behavior, such that early-maturing girls exhibited the highest levels of depressive symptoms when they initiated dating early and early-maturing boys showed the strongest link with depressive symptoms when they spent time with law-breaking peers.

Of note, childhood stressful experiences have been shown to predict earlier onset of puberty in girls (Ellis & Essex, 2007; Chisholm, Quinlivan, Petersen, & Coall, 2005; Ellis & Garber, 2000; Ellis, McFadyen-Ketchum, Dodge, Pettit, & Bates, 1999) suggesting that environmental context may not simply moderate the association between early timing and risk of psychopathology, but may actually serve as a common cause of both early timing and increased risk of psychopathology in adolescence (Viner, 2015).

Compared with the paucity of compelling evidence in support of the other proposed mechanisms, contextual amplification has perhaps the strongest existing support. Many studies have utilized longitudinal designs, and most examine contextual stressors that are highly theoretically relevant to the association between puberty and psychopathology (e.g., maternal

depression history and life stress in the prediction of depressive symptoms; affiliation with deviant peers in the prediction of delinquent behavior; Hammen, 2005; Hammen & Brennan, 2003; Keenan, Loeber, Zhang, Stouthamer-Loeber, & van Kammen, 1995). Unfortunately, few of these effects have been replicated, and the majority of studies have focused solely on chronic interpersonal stress, particularly among peers, with less attention paid to other environmental stressors. Among studies examining internalizing or depressive symptoms, the contextual amplification effects of non-interpersonal stressors have never been examined, despite evidence that non-interpersonal stress is also linked with the development of depression (Hammen *et al.*, 2009). Additionally, few studies have examined the impact of discrete stressful life events, although the distinction between episodic and chronic stress is highly relevant to the development of depression (Hammen, Kim, Eberhart, & Brennan, 2009; McGonagle & Kessler, 1990).

Other Characteristics of Puberty

In addition to pubertal timing, several other characteristics of the pubertal experience vary across individuals, might reasonably be expected to influence psychological well-being, and may therefore be associated with the development of depressive symptoms. Mendle (2014) and Rudolph (2014) have identified two particular features of puberty that necessitate further study.

Pubertal synchrony, the degree to which different markers of puberty develop simultaneously, is one characteristic of interest. In general, physical development does not occur with perfect simultaneity, with breasts developing first in the majority of girls and genitalia developing prior to the initiation of pubic hair growth in most boys (Susman & Dorn, 2013; Wheeler, 1991; Lee, 1980; Marshall & Tanner, 1970; Marshall & Tanner, 1969). However, although some degree of asynchrony is not uncommon, variations in pubertal synchrony have

long been theorized to influence psychological well-being. Eichorn (1975) was the first to hypothesize that the relative timing of various aspects of physical development could impact mental health, arguing that asynchronous maturation could lead to heightened anxiety. Brooks-Gunn and Warren (1985) also suggested that synchronous versus asynchronous development might be associated with psychopathology, although they did not predict the direction of the effect. Mendle (2014) offered explanations in both directions – adolescents might feel overwhelmed by the occurrence of multiple changes simultaneously, resulting in an association between synchronous development and psychopathology, or youth undergoing synchronous development may feel more psychologically secure in their experience of puberty, leading to an association between asynchronous development and psychopathology.

Hypotheses predicting an association between pubertal synchrony and psychopathology tend to align with the psychosocial mechanisms reviewed above, in that variations in synchrony are expected to result in alterations in self-image, stress, and perceptions of abnormality compared to peers that are likely to influence subsequent psychological functioning. Brooks-Gunn (1984) also highlights the particular stress that may be conferred by asynchronous development among girls, in that more advanced breast development is easily observable by others. Thus, studies examining the association between synchrony and psychopathology serve as a natural extension of the literature on the psychological effects of pubertal timing and may help to elucidate which psychosocial mechanisms link the experience of puberty to mental health outcomes.

Unfortunately, studies of synchrony are limited, and to the author's knowledge, pubertal synchrony has never been studied with regard to the development of psychopathology. Previous studies of synchrony have focused primarily on associations between synchronous versus

asynchronous development and other physical characteristics. Biro *et al.* (2003) found that girls who exhibited more advanced breast development than pubic hair development have higher body mass and a larger percentage of body fat than girls who demonstrate the opposite pattern of development. Girls with more advanced breast development also exhibit lower breast density than girls with more advanced pubic hair development or synchronous development (Novotny, Daida, Morimoto, Shepherd, & Maskarinec, 2011). Boys with medical conditions that delay or accelerate skeletal maturation (e.g., congenital adrenal hyperplasia) demonstrate matched delays or accelerations in the production of testosterone (Flor-Cisneros *et al.*, 2004), while boys who exhibit normal rates of skeletal maturation do not show such alterations in testosterone levels (Flor-Cisneros, Roemmich, Rogol, & Baron, 2006). Research has also suggested that girls in the early stages of puberty are most likely to exhibit asynchronous breast and pubic hair development while those in later stages demonstrate greater synchrony (Susman *et al.*, 2010). Thus, despite hypothesized associations between synchrony and psychopathology as well as the relevance of psychosocial theories of puberty and mental health to the study of synchronous development, almost nothing is known about the psychological impact of pubertal synchrony.

Another characteristic of puberty that is theoretically relevant to the development of psychopathology yet has received relatively little study is pubertal tempo, the speed of pubertal maturation. Evidence suggests that tempo varies significantly between markers of puberty (e.g., breast growth, genital development) and between individuals (Lee, 1980; Marshall & Tanner, 1970; Marshall & Tanner, 1969). For example, Marshall and Tanner (1970) note that in their study of 228 normal boys, full development of genitalia occurred over an average of three years. However, some boys achieved full development in less than two years while others required almost five. Among girls, the progression from the earliest stages of puberty to full maturation

across all markers of development can range between two and six years (Marshall & Tanner, 1969). Therefore, although trends in developmental speed are apparent, variability in tempo is not uncommon.

While some youth mature quickly, others experience a lengthy developmental transition, leading to varying effects on psychological well-being. The *maturation compression* hypothesis suggests that rapid progression through puberty may exert similar effects as the experience of off-time development, with adolescents unable to make necessary psychological and social adaptations to successfully navigate puberty (Mendle, Harden, Brook-Gunn, and Graber, 2010). Several studies have supported this perspective, although obtained results differ significantly by sex. Ge *et al.* (2003) found that African-American males who experienced a rapid pubertal tempo exhibited the highest increase in depressive symptoms. They found no evidence of an effect in girls. Similar results were obtained by Mendle *et al.* (2010), who found that pubertal tempo was unrelated to the development of depressive symptoms in girls, while both timing and tempo appeared to influence symptoms in boys, such that early timing and rapid tempo were associated with increased depression. Of note, rapid tempo appeared to exert effects on symptoms over and above the impact of timing, suggesting a unique role for pubertal tempo in the risk of developing depression. A later study replicated the effect of early timing and rapid tempo in the prediction of depressive symptoms among boys and found that this effect was fully mediated by changes in peer relationships (Mendle, Harden, Brooks-Gunn, & Graber, 2012).

However, other research has suggested that the association between pubertal tempo and depressive symptoms is not so straightforward. One study found that faster tempo was related to higher levels of internalizing symptoms among adolescent girls, but was not associated with internalizing in boys (Marceau, Ram, Houts, Grimm, & Susman, 2011). A recent study

conducted among females determined that rapid tempo was associated with increases in depressive symptoms between ages 10 and 13, while slower tempo was linked to higher levels of depressive symptoms at age 10 (Keenan, Culbert, Grimm, Hipwell, & Stepp, 2014), indicating that age may moderate the effect of tempo on depression. In contrast to the majority of findings, one study found that rapid pubertal tempo among boys was associated with *lower* levels of depressive symptomatology and did not find any association in girls (Laitinen-Krispijn, van der Ende, & Verhulst, 1999). However, this study was based on parental report of youth depressive symptoms, which may be somewhat less valid than studies utilizing self-report or both parent- and self-reports of depression. In summary, of the small number of studies that have examined pubertal tempo in relation to depressive symptoms, the majority have supported the maturation compression hypothesis among boys only, although others have suggested that the association between tempo and depressive symptoms may be more complex.

Like synchrony, hypotheses regarding the psychological effects of variations in tempo highlight similar processes to those predicted by psychosocial theories of timing and psychopathology. Rapid pubertal tempo is expected to reduce psychological and social preparedness for the challenges accompanying the pubertal transition (Mendle *et al.*, 2010), much like those predicted by the stage termination and maturation disparity hypotheses. Rapid tempo may also exacerbate the effects of negative personal characteristics or stressful environmental conditions on mental health. Thus, although the psychological implications of variations in synchrony and tempo have been less consistently studied than the role of pubertal timing, there is reason to expect that both synchrony and tempo exert similar effects on youth attempting to navigate the challenges of puberty.

Outcomes in Young Adulthood

Previous research on the role of puberty in the development of depressive symptoms has focused almost exclusively on effects in adolescence. However, young adulthood or emerging adulthood, characterized as the developmental period between ages 18 and 25 (Arnett, 2000), offers a particularly important window in which to assess the psychological impact of puberty. Depression that begins in adolescence frequently persists into young adulthood (Rao, Hammen, & Daley, 1999; Lewinsohn, Rohde, Klein, & Seeley, 1999), and youth with adolescent-onset depression may continue to generate stressful experiences once their symptoms have remitted, leading to increased risk of recurrent episodes (Daley *et al.*, 1997; Hammen, 1991). Additionally, the transition from adolescence to young adulthood is a critical developmental period that is associated with a number of significant psychosocial stressors (e.g., increased independence from family, initiation of higher education or full-time employment, pursuit of serious romantic relationships) that may precipitate the development of depressive symptoms. In fact, psychopathology tends to increase in the young adult years (Schulenberg & Zarrett, 2006).

Many of the changes experienced during young adulthood are also highly relevant to the psychological and social variables that are hypothesized to play a role in the association between puberty and psychopathology. For example, self-image and coping have been proposed as mediators of the link between pubertal experiences and the development of psychopathology in both the stage termination and maturation disparity hypotheses. These same characteristics are likely to be important factors in the ability of adolescents to successfully navigate the transition to young adulthood and have previously been shown to be associated with the development of depression (Orth, Robins, & Roberts, 2008; Lam & McBride-Chang, 2007). Consistent with the contextual amplification hypothesis, young adulthood may also offer an opportunity for youth to separate themselves from deleterious environmental contexts, such as stressful family situations

or the direct influence of parents with psychopathology, which could result in result in reduced risk of experiencing depressive symptoms (Hammen, Hazel, Brennan, & Najman, 2012; Luecken & Gress, 2010).

To date, only three studies have examined the psychological effects of puberty beyond adolescence. Two studies examining outcomes in young adulthood found that early-maturing females exhibited higher lifetime prevalence rates and higher levels of depression in young adulthood than on-time or late-maturing peers (Graber *et al.*, 2004; Copeland *et al.*, 2010). This effect was not observed among males. However, Copeland *et al.* (2010) determined that the higher prevalence of depression among young adult females in their study could be accounted for by adolescent conduct disorder. A third study found that late-maturing males experienced adolescent-onset affective symptoms that continued into midlife, while females with especially late initiation of menstruation demonstrated a lower risk of adult-onset affective symptoms (Gaysina *et al.*, 2015). However, this study did not include any assessments of psychopathology in young adulthood. Thus, despite the importance of understanding the association between pubertal experiences and psychopathology beyond adolescence, findings to date have been inconsistent and very little is known about the continuing influence of puberty into the young adult years.

Dissertation Overview

The present dissertation is designed to answer several questions regarding the association between puberty and the development of depressive symptoms. Of essential interest is the fact that puberty, a universal experience, nonetheless results in distinct psychological outcomes among different individuals. Thus, while puberty can be considered a normative stressful experience, variations in the particular nature of each individual's pubertal transition can

dramatically affect the level of stress associated with puberty as well as subsequent psychological well-being. The impact of puberty on the development of depression is of particular interest due to strong associations between stressful experiences and the occurrence of depressive symptoms independent of features of puberty (Hammen, 2005), as well as the marked increase in depression prevalence that occurs contemporaneously with pubertal development and persists into adulthood (Hankin *et al.*, 1998; Kessler *et al.*, 1993).

While the association between puberty and depression has been considered previously (for a review, see Graber, 2013), the vast majority of research to date has examined direct effects of pubertal timing on depressive symptoms in adolescence. Surprisingly little is known about the mechanisms through which puberty affects depressive symptoms, the role of characteristics other than timing, and the development of depressive symptoms beyond adolescence. The present dissertation therefore aims to address the following questions via three related studies.

1. What are the mechanisms through which variations in pubertal timing affect the development of depressive symptoms?

This question is addressed in Studies 1 and 2 with a focus on four hypothesized psychosocial mechanisms that have been prominently featured in the literature. Study 1 tests the stage termination, maturation disparity, personal accentuation, and contextual amplification hypotheses in a sample of 1070 community youth assessed annually from elementary school through one year post-high school as part of a study on predictors of substance use. Latent growth modeling is used to examine trajectories of depressive symptoms across adolescence and into young adulthood, and the study evaluates the relevance of each proposed mechanism by testing mediation and moderation effects of several variables that have been implicated in the link between timing and depressive

symptoms. Study 2 aims to extend the questions explored in Study 1 to a more nuanced examination of one of the four hypotheses: contextual amplification. Of particular interest is the specific influence of various types of contextual stress on the association between timing and depressive symptoms, including chronic interpersonal stressors, chronic non-interpersonal stressors, and episodic stressful life events. Study 2 utilizes a sample of 815 youth drawn from a longitudinal birth cohort study in which mother-child pairs were followed from pregnancy through youth age 20.

2. Do characteristics of puberty other than timing affect the occurrence of depressive symptoms?

Study 3 directly addresses this question by examining the role of pubertal synchrony in the development of depressive symptoms in adolescence and young adulthood. Drawing from the same sample of 815 youth used in Study 2, the third study explores the role of synchronous versus asynchronous morphological development in females by examining synchrony between breast and pubic hair development as a predictor of future depressive symptoms.

3. How does the pubertal experience affect depressive symptoms into young adulthood?

This question is addressed in each of the proposed studies. The sample utilized for Study 1, in which youth participated in annual assessments of depressive symptoms throughout high school as well as one year post-high school offers the ability to explore trajectories of depressive symptoms across the transition into young adulthood. In Studies 2 and 3, assessments were conducted at ages 15 and 20, allowing for an evaluation of discrete effects in adolescence and young adulthood.

Finally, the role of gender also serves as a key organizing principle of the dissertation, as pubertal processes appear to operate differently in females and males (Graber, 2013; Mendle & Ferrero, 2012; Mendle *et al.*, 2007) and may help to explain the development of the marked gender gap in depression prevalence that emerges in adolescence and persists into adulthood (Cyranowski *et al.*, 2000; Angold & Worthman, 1993). Studies 1 and 2 examine effects separately for females and males, while Study 3 includes females only due to limitations in the measurement of pubertal synchrony among males in the parent study.

**Chapter 2: Psychosocial Mechanisms of the Link between Pubertal Timing
and Depressive Symptoms (Study 1)**

Abstract

The timing of puberty relative to peers has been shown to influence the development of psychopathology among youth, such that early-maturing females and off-time males appear to be at the greatest risk of developing conditions such as depression. Several psychosocial hypotheses have been identified to account for the association between off-time development and depressive symptoms; however, empirical evidence in support of these hypotheses is limited. Moreover, few studies have examined trajectories of symptoms beyond adolescence or explored gender-specific mechanisms of the association between pubertal development and psychopathology. The present study sought to address these gaps in the literature by examining four prominent psychosocial explanations for the association between off-time pubertal development and depressive symptoms in a longitudinal community sample of 1070 youth followed from elementary school through one year post-high school. Latent growth modeling and conditional process analyses revealed support for aspects of the stage termination, personal accentuation, and contextual amplification hypotheses among females only, while the maturation disparity hypothesis was not supported among adolescents of either gender. Results provide some of the first empirical evidence of specific pathways through which early pubertal timing leads to depressive symptoms among girls and highlight critical gender differences in the mechanisms through which pubertal development contributes to psychopathology in adolescence and into young adulthood.

Introduction

Study 1 seeks to clarify the mechanisms through which variations in pubertal timing impact the development of depressive symptoms. As described above, although four psychosocial hypotheses have offered explanations for the association between puberty and psychopathology, empirical evidence in support of these hypotheses is lacking.

The stage termination and maturation disparity hypotheses refer to mediation models in which off-time development leads to poor coping, low self-image, and social skills deficits that contribute to increased levels of depression. Thus, empirical tests of these hypotheses require longitudinal studies in which potential psychosocial mediators of the link between timing and depressive symptoms can be explored. Unfortunately, very little research has examined the influence of off-time maturation on the specific mediators implicated by these hypotheses (Rudolph *et al.*, 2014; Conley *et al.*, 2012), and to the author's knowledge, no studies to date have directly tested the maturation disparity hypothesis.

The personal accentuation and contextual amplification hypotheses refer to moderation models in which the negative psychological effects of off-time development are enhanced by the presence of negative personal characteristics or stressful environmental contexts. Although both hypotheses have received support in the literature, many of the studies that provide empirical tests of personal accentuation have focused solely on girls. Additionally, although the personal accentuation hypothesis highlights the role of negative personality traits, only one study to date has examined features of personality as a possible moderator of the link between timing and depressive symptoms. This study assessed depressive personality traits and negative self-focus (Rudolph & Troop-Gordon, 2010), but was unable to examine more general personality characteristics such as those included in the Big Five personality taxonomy (Goldberg, 1990).

Furthermore, several of the studies testing facets of the personal accentuation and contextual amplification hypotheses have relatively small samples ($N < 200$), and most of the obtained effects have never been replicated. Thus, while existing studies have supported personal accentuation and contextual amplification, they have not yet provided clarification on several important issues, including the role of these processes in males, the influence of general personality traits on the association between timing and depressive symptoms, and the ability of these effects to be replicated and generalized to larger, more representative samples. Study 1 aims to provide direct empirical tests of the four psychosocial hypotheses in order to clarify the specific mechanisms through which pubertal timing affects the development of depression.

Additionally, most research on the association between timing and depressive symptoms and all of the existing studies examining psychosocial mechanisms of this association have explored outcomes solely in adolescence. As discussed previously, young adulthood is a particularly important developmental period in which to explore the influence of pubertal processes on psychopathology. Extending this research past adolescence would allow for the examination of trajectories through which variations in timing might influence depressive symptoms in the long-term and would help to identify the specific psychosocial processes by which puberty exerts distal psychological effects. Previous research has largely failed to explore symptom trajectories across development, with the notable exception of a single recent study that did not examine outcomes in young adulthood (Gaysina *et al.*, 2015). Study 1 examines the trajectory of depressive symptoms from grade 9 and through one year post-high school in order to clarify the long-term impact of timing on depression and further elucidate the mechanisms through which pubertal experiences contribute to psychopathology during a critical developmental period.

Finally, due to observed gender differences in the association between timing and depressive symptoms, it is possible that different processes underlie the association between puberty and depressive symptoms among females and males. Therefore, Study 1 explores the relevance of the four psychosocial mechanisms separately by gender.

Aims and Hypotheses

Aim 1. To assess the validity of the stage termination, maturation disparity, personal accentuation, and contextual amplification hypotheses in the association between pubertal timing and depressive symptoms.

1. Due to the established link between early timing and depressive symptoms among females (Graber, 2013; Mendle *et al.*, 2007) as well as the evidence suggesting that females' more maladaptive coping strategies and greater interpersonal orientation are partially responsible for gender differences in depressive responses to stress (Nolen-Hoeksema, 2001), it is hypothesized that, consistent with the stage termination hypothesis, psychological and social variables such as coping, perceived stress, self-image, and social competence will partially mediate the association between early maturation and depressive symptoms among females. In light of the mixed findings among males regarding the influence of timing on depressive symptoms (Mendle & Ferrero, 2012), it is expected that males will not show evidence of stage termination.
2. Given the consistent finding that early-maturing females demonstrate higher levels of depressive symptoms when compared to peers (Graber, 2013), it is not expected that females will demonstrate evidence consistent with maturation disparity, which would predict that both early- and late-maturing girls would exhibit increased depression. However, it is predicted that evidence in support of maturation disparity will be obtained

in males, such that males who mature off-time relative to peers (either early or late) will exhibit negative psychological and social characteristics that partially mediate the association between timing and depressive symptoms.

3. In light of associations between personality traits and other personal attributes in the development of depressive symptoms independent of puberty (Kendler, Gatz, Gardner, & Pedersen, 2006; Bagby, Quilty, & Ryder, 2008; Coyne & Whiffen, 1995), it is expected that both females and males will exhibit increased depressive symptoms in response to variations in timing (early development in girls, off-time maturation in either direction among boys) when they also possess personal vulnerabilities such as negative personality traits.

4. Due to the evidence supporting the contextual amplification hypothesis among both females and males (Benoit *et al.*, 2013; Rudolph & Troop-Gordon, 2010; Conley & Rudolph, 2009) as well as the relevance of environmental stressors to the occurrence of depressive symptoms independent of puberty (Hammen, 2005), it is hypothesized that both females and males will exhibit evidence of contextual amplification, such that environmental stressors moderate the association between timing and depressive symptoms. It is predicted that early-maturing girls experiencing contextual stressors will demonstrate the highest levels of depressive symptoms, while boys who mature off-time and experience high levels of environmental stress will exhibit more depressive symptoms than on-time peers.

Aim 2. To examine the influence of variations in timing on trajectories of depressive symptoms across adolescence and into young adulthood.

1. Given the paucity of research examining trajectories of depressive symptoms in response to variations in pubertal timing, as well as the mixed findings obtained by the three existing studies examining the effects of timing on symptoms beyond adolescence (Graber *et al.*, 2004; Copeland *et al.*, 2010; Gaysina *et al.*, 2015), no a priori hypotheses about trajectories of depressive symptoms are made.

Method

Participants

Study 1 utilized data from the Oregon Youth Substance Use Project (OYSUP), a longitudinal study examining predictors of youth substance use that was conducted between 1998 and 2012 by the Oregon Research Institute. Using stratified random sampling by school, grade, and gender, a total of 2127 youth attending one of fifteen elementary schools in western Oregon were identified as potential study participants. Parents of these children were sent a letter inviting them to enroll in the study and subsequently received a phone call to assess interest. Parents of 1075 students provided consent for their child's involvement, and a total of 1070 youth and their parents participated in the first assessment. Youth were recruited as part of grade cohorts (grades 1 through 5 at the start of the study) using a cohort-sequential design and were assessed regularly from elementary school through one year post-high school. A final assessment was conducted when youth were between ages 20 and 22, although data from the final assessment were not available for the current analyses.

Approximately half of the initial sample of 1070 was female ($N = 538$, 50.3%), and the majority of youth identified as Caucasian ($N = 918$, 85.8%). Of the remaining participants, 7.1% were Latino, 2.4% were Native American or Alaskan Native, 2.2% were Asian, 1.1% were African-American, and 1.7% identified as other or of mixed race/ethnicity.

A total of 64 students (6.0%) failed to participate in any assessments after grade 5, while a total of 112 did not participate in any assessments after grade 8 (10.5%). Youth who participated in at least one assessment in middle and high school were no different than non-participants at either timepoint on gender and race/ethnicity (Hampson, Tildesley, Andrews, Barckley, & Peterson, 2013; Andrews, Hampson, & Peterson, 2011). Of note, some participants missed one or more assessments, but continued to participate in assessments at subsequent timepoints.

Compared to the full sample of 1070, youth who completed an assessment during grade 9 ($N = 769$) showed no differences in gender ($\chi^2(1,1069) = 1.00, p = .34$), childhood depression history ($t(981) = -0.60, p = .55$), or pubertal timing ($t(939) = -1.42, p = .16$).

In comparison to the full sample, youth who participated in an assessment during grade 12 ($N = 876$) were more likely to be female ($\chi^2(1,1069) = 5.33, p = .02$). Youth who completed an assessment during grade 12 were no different from the full sample in terms of childhood depression history ($t(981) = 0.42, p = .68$) or pubertal timing ($t(939) = -0.18, p = .85$).

There was a nonsignificant trend such that youth who participated in an assessment one year post-high school ($N = 650$) were more likely to be female than those who did not participate ($\chi^2(1,1069) = 3.61, p = .06$). Individuals who completed an assessment one year post-high school were no different from the full sample with regard to childhood depression history ($t(981) = 0.18, p = .86$) or pubertal timing ($t(939) = -0.33, p = .74$).

Further information about the recruitment and composition of the sample is presented by Andrews, Tildesley, Hops, Duncan, & Severson (2003).

Procedures

Youth and their parents completed annual assessments beginning in elementary school and continuing through one year post-high school, other than a one-year break between T4 and T5 due to a lapse in funding. At each assessment point, youth and one or both parents completed a questionnaire comprised of multiple measures of substance use, psychosocial functioning, academic achievement, and health behaviors.

Many of the measures utilized in the OYSUP were shortened by the research team in order to reduce participant burden, including measures of self-image, social competence, personality traits, parental depressive symptoms, parent-child relationship conflict, and neighborhood stress. In order to develop these shortened versions of standard measures, pilot studies were conducted each time new questionnaires were developed in which an independent sample of youth and their parents completed the full measures on two occasions, two weeks apart. Factor analysis was conducted to determine which items exhibited the highest level of internal consistency and would be incorporated into the questionnaire. Internal consistency and test-retest reliability of the shortened measures were acceptable according to calculations by the research team.

If possible, all youth questionnaires were completed at school. Alternatively, students and their parents were invited to the Institute to complete the questionnaire. Those who were unable to travel to the Institute were assessed over the phone (grades 4-8) or completed a mailed questionnaire (high school and post-high school). Beginning in high school, youth were paid for their participation (\$20 during high school, \$30 post-high school). Parents completed mailed questionnaires at each assessment point and were paid \$25-\$30 for their participation each year.

Additionally, teachers completed annual mailed questionnaires for each of their participating students, and teachers and principals completed annual assessments of school

climate at T1 through T8. The present study utilized data collected from youth and their parents and did not incorporate teacher reports or assessments of school climate.

Families whose children no longer attended one of the fifteen participating schools continued to be contacted and asked to participate in each follow-up assessment. Families who lived within driving distance of the Institute were asked to come to the Institute and complete annual questionnaires. Families who lived outside of this range completed mailed questionnaires.

Additional information regarding study procedures is summarized in Andrews *et al.* (2003).

Measures

Pubertal status. Pubertal status was assessed using parent ratings on the Pubertal Development Scale (PDS; Petersen, Crockett, Richards, & Boxer, 1988). The PDS is a 5-item questionnaire that assesses growth spurt, body hair, and skin changes in addition to voice changes and facial hair development in boys, and breast development and menarcheal status in girls. Menarcheal status is assessed using a dichotomous response (yes/no) while all other items are measured using a 4-point scale ranging from 1 (development not yet started) to 4 (development seems complete). Menarcheal status was recoded for the present analyses such that a score of 1 indicated the absence of menarche and a score of 4 signified that menstruation had begun. The PDS demonstrates excellent reliability and validity and is moderately correlated with clinician ratings of the Tanner stages of physical development (Shirtcliff, Dahl, & Pollak, 2009; Brooks-Gunn *et al.*, 1987).

Parents completed the PDS on daughters' pubertal development during grades 4 and 5 and son's pubertal development during grades 6 and 7, due to sex differences in the typical age of puberty onset. In cases where ratings from both parents were available at a single timepoint,

individual parent ratings were averaged. The correlations among parent ratings of pubertal status ranged from .70 to .87 among parents of daughters and between .72 and .87 among parents of sons. Among participants for whom pubertal status was assessed more than once (i.e., a girl whose parents provided ratings during both grades 4 and 5), pubertal status at the more advanced grade was utilized in the present analyses (i.e., grade 5 for females and grade 7 for males).

Descriptive statistics for pubertal status and all other study variables are presented in Table 1. Females exhibited a range of PDS scores from 1 (no development) to 3.4 across grades 4 and 5 with a mean score of 1.65 (SD = 0.42) in grade 4 and a mean score of 1.98 (SD = 0.52) in grade 5. Males exhibited a range of 1 to 3.5 across grades 6 and 7, with a mean PDS score of 1.69 (SD = 0.48) in grade 6 and 2.07 (SD = 0.58) in grade 7. These scores confirm that participants were generally in the early stages of development at the assessment of pubertal status, and no participants had yet completed puberty.

Copies of all Study 1 measures, including the PDS, are located in the Appendix.

Pubertal timing. Pubertal timing was determined by regressing PDS scores on age at assessment point, separately by sex (Dorn, Dahl, Woodward, & Biro, 2006). The resulting residual scores reflect the timing of puberty relative to same-sex peers, such that higher residuals (e.g., positive residuals) signify earlier maturation.

Depressive symptoms. Depressive symptoms were measured using the Center for Epidemiological Studies Depression Scale (CESD; Radloff, 1977), a 20-item self-report questionnaire assessing symptoms within the past week. Participants completed the CESD beginning in 9th grade (T5) and continuing through one year post-high school. The CESD demonstrates high reliability and excellent concurrent validity with clinical ratings of depression in samples of adults as well as adolescents (Radloff, 1977; Garrison, Addy, Jackson, McKeown,

& Waller, 1991). The CESD exhibited high internal consistency in the present sample (Cronbach's α between .79 and .83 across assessment points).

Scores on the CESD among youth participating in the present study ranged between 0 and 60, with females exhibiting mean scores between 14.81 and 16.40 across adolescence and into young adulthood (Table 1). Males' mean level of depressive symptoms ranged between 10.88 and 13.20 in the same period. The percentage of females with levels of depressive symptoms that met or exceeded the clinical cutoff for mild depression (CESD score ≥ 16) ranged between 37.4% and 44.1% during grade 9 through one year post-high school. The percentage of males who met or exceeded the clinical cutoff for mild depression ranged from 22.4% in grade 9 to 34.3% one year post-high school. Complete data regarding the number and percentage of youth who met criteria for clinical levels of depression at each assessment point is presented in Table 2.

Mediators of the association between pubertal timing and depressive symptoms. For the present analyses, all proposed mediators were examined at the first assessment point at which data were available following the measurement of pubertal status but prior to the measurement of depressive symptoms in grade 9 through one year post-high school. Thus, mediators were measured among females by calculating the mean of scores in grades 6 and 7. Among males, mediators were evaluated in grade 8.

Coping. Youth coping was assessed using a 6-item self-report questionnaire developed by Wills (1990). Participants were asked to rate how frequently they tend to engage in various coping strategies (e.g., find someone to share my problem with; try to think of the good things in my life) when confronted with a difficult situation. This measure includes items assessing problem-focused, appraisal-focused, and emotion-focused coping techniques.

The coping measure was administered at each assessment beginning in grade 6 (T2) and continuing for the remainder of the parent study. Scores in the present sample ranged from 6-30, and higher scores reflect more effective coping. Females exhibited a mean score of 21.57 (SD = 4.78) across grades 6 and 7 ($r = .35$ across grades), and males demonstrated a mean coping score of 18.20 (SD = 5.80) in grade 8.

Perceived stress. Perceived stress was assessed by asking participants to read a list of 20 major life events derived from the Cornell Medical Index (Dornbusch, Mont-Reynaud, Ritter, Chen, & Steinberg, 1991) and select which events had occurred within the last 12 months. Stressful life events captured by this measure include episodic stressors related to family (e.g., my parents were divorced or separated), peers (e.g., I broke up with my boyfriend or girlfriend; one of my close friends died or became seriously ill), and self (e.g., I was suspended from school; I became seriously ill or was hospitalized). Youth completed the checklist annually beginning in 6th grade (T2). For the present analyses, perceived stress was measured among females by calculating the mean number of stressful life events in grades 6 and 7. Females reported a mean of 3.18 events (SD = 2.36) across grades 6 and 7 ($r = .39$). Males reported a mean of 2.96 stressful life events (SD = 2.53) in grade 8.

Self-image. Self-image was measured using an abbreviated, 6-item version of the Offer Self-Image Questionnaire (Offer, Ostrov, & Howard, 1984), a self-report measure that captures adolescent adjustment in 10 life domains. While the original measure contains 129 items, the brief version utilized for the present study was limited to questions regarding body image as well as more general self-concept (e.g., I am proud of my body; very often I think I am not at all the person I would like to be). Youth completed this measure at every assessment point beginning in 6th grade (T2). Scores ranged from 10-30, with higher scores indicating more positive self-

image. Females in the present sample exhibited a mean score of 23.72 (SD = 4.49) in grades 6 and 7 ($r = .53$), while males had a mean score of 24.40 (SD = 4.35) in grade 8.

Social competence. Social competence was evaluated using a modified version of the Self-Perception Profile for Adolescents (SPPA; Harter, 1988), a self-report measure examining adolescents' opinions about their social and academic competence. Participants were asked to select which of two opposing statements best describes them (e.g., some teenagers have a lot of friends versus other teenagers don't have very many friends). Youth then rated whether their selected statement is "sort of true" or "really true" of them. The original measure demonstrates good internal consistency and construct validity (Wichström, 1995).

A 10-item modified version was completed by youth in grades 6 through 12 (T4-T11), and the social competence subscale was calculated to assess self-perceptions of interpersonal abilities. Scores ranged from 1-4 with higher scores indicating greater levels of competence. Girls in the present sample had a mean social competence score of 3.18 (SD = 0.64) in grades 6 and 7 ($r = .59$), while boys had a mean score of 3.17 (SD = 0.65) in 8th grade.

Moderators of the association between pubertal timing and depressive symptoms. In order to best capture the personal and environmental context of pubertal development in the present study, proposed moderators, including personal characteristics and contextual factors, were measured as close to puberty as possible given the availability of data, but prior to the first assessment point at which outcomes were examined (i.e., between grades 4 and 8 among females, between grades 6 and 8 among males). A single composite score for each personal characteristic and contextual stressor was then created by averaging the data at each available assessment point for each participant (e.g., the mean level of extraversion across grades 7 and 8). This analytic strategy allowed for full representation of the persistent, contextual nature of the

relevant personal characteristics and environmental stressors and was designed to provide the most theoretically-consistent test of the personal accentuation and contextual amplification hypotheses. Table 3 presents correlation coefficients among the values of each personality trait and contextual factor at each assessment point, separately by gender.

Personality traits. Personality traits were measured using an adapted version of the “Mini-Markers” personality assessment (Saucier, 1994), which was designed to provide a brief, reliable measure of Big Five personality traits. The Mini-Markers asks youth to rate the degree to which each of 40 characteristics describe them (e.g., organized, talkative, creative) and demonstrates acceptable reliability (Saucier, 1994). As part of the OYSUP, a 10-item version was administered at every assessment between grade 7 and the post-high school assessment (T5-T9). For the present analyses, five personality traits were assessed, including extraversion, agreeableness, conscientiousness, emotional stability (characterized as the opposite of neuroticism), and openness to experience.

Personality traits were measured by taking the mean of scores in grades 7 and 8. Table 1 summarizes the mean, standard deviation, and range of each trait among females and males in the present sample.

Parental depressive symptoms. Parental depressive symptoms were measured using an abbreviated version of the CESD in which parents were asked to rate their depressive symptoms in the past week. The abbreviated version contains 10 of the original 20 items, and parents completed this measure at every assessment point beginning at T1 (youth grades 1-5).

For the present analyses, maternal and paternal scores on the CESD were assessed in grades 4 through 8 among females and in grades 6 through 8 among males. Correlations among maternal depression scores over time ranged between .44 and .73 among females and between

.56 and .63 among males (Table 3). Maternal depressive symptom scores ranged from 0 to 27 across all participants, and females exhibited an average maternal depression score of 6.08 (SD = 4.31) across grades 4 through 8, while males exhibited an average score of 5.79 (SD = 4.60) in grades 6 through 8 (Table 1). Correlations among paternal depression scores over time ranged between .40 and .64 among females and between .54 and .64 among males, with a mean score of 4.67 (SD = 3.59) among females and 4.92 (SD = 3.97) among males. Paternal depression scores ranged from 0-23 in the present sample.

Stressful life events. The checklist of stressful life events that was used to assess perceived stress as a mediator of the association between pubertal timing and depressive symptoms was also used to examine the moderating role of contextual life stress. Moderation analyses employed the mean level of stressful life events that occurred in grades 6 through 8 among both females and males. The total number of stressful life events ranged from 0 to 11. Among females, the mean number of events was 3.27 (SD = 2.12), while males reported an average of 3.03 events (SD = 2.13).

Parent-child relationship conflict. Relationship conflict between the participant and his or her parents was measured using an abbreviated version of the Conflict Behavior Questionnaire (CBQ; Prinz, Foster, Kent, & O' Leary, 1979). The original CBQ contains 73 items and demonstrates strong discriminant validity between distressed and non-distressed parent-youth dyads as well as high internal consistency (Cronbach's alpha ranging from .88-.95; Prinz *et al.*, 1979). In the present sample, youth rated the degree to which seven statements applied to the parent-child relationship (e.g., we have big arguments about little things; we never have fun together). Youth completed an annual CBQ for each parent beginning at T1, and maternal and paternal conflict were measured in the present analyses by calculating the mean CBQ score for

each parent across grades 6 through 8. Correlations among levels of maternal conflict over time ranged between .49 and .55 among females and between .37 and .55 among males (Table 3). Scores on the abbreviated CBQ ranged from 0 to 7, and the mean level of maternal conflict was 1.99 (SD = 1.48) among females and 1.86 (SD = 1.37) among males (Table 1). Correlations of paternal conflict scores over time ranged between .45 and .54 among females and between .49 and .52 among males. The mean level of paternal conflict was 2.02 (SD = 1.59) among females and 1.73 (SD = 1.50) among males.

Neighborhood stress. Neighborhood stress was captured using a modified version of the Community Violence Scale (Richters & Martinez, 1993). The modified version is a 7-item measure asking youth to rate how often they have witnessed or experienced various situations (e.g., seen someone get arrested; been beaten up, robbed, or attacked) within the past year. Youth completed this measure at each assessment beginning in 6th grade (T5). In the present analyses, neighborhood stress was determined by calculating the mean level of neighborhood stress across grades 6 through 8 for both females and males. Correlations across grades ranged between .40 and .57 among females and between .32 and .50 among males (Table 3). Neighborhood stress scores ranged between 7 and 21, and females reported a mean level of neighborhood stress of 9.52 (SD = 2.55), while males reported a mean of 9.47 (SD = 2.56).

Statistical Analyses

Latent growth modeling (Meredith & Tisak, 1990; Muthén, 1991) was used to evaluate trajectories of change in depressive symptoms across adolescence and into young adulthood. Latent growth models (LGMs) are designed to estimate change over time and offer a number of advantages when examining longitudinal datasets containing repeated assessments (Duncan & Duncan, 2004). An initial LGM was constructed to examine the main effect of pubertal timing

on trajectories of depressive symptoms from grade 9 through one year post-high school. As described below (see Results), several measurement models were constructed in order to select the most parsimonious model that provided the best fit to the data. As illustrated in Figure 1, the selected model contains two latent factors representing the intercept of depressive symptoms (i.e., depressive symptoms in 9th grade) and the linear slope of depressive symptoms between grade 9 and one year post-high school. Each of these latent factors is predicted by an observed linear pubertal timing variable as well as a quadratic timing variable formed by squaring the value of linear pubertal timing. The inclusion of quadratic timing allows for the exploration of curvilinear effects in which early and late timing are associated with similar outcomes, in light of research suggesting that the association between timing and psychopathology may be quadratic (e.g., Rudolph, 2014; Natsuaki, Biehl, *et al.*, 2009).

Following the exploration of main effects, two types of analyses were conducted to test the four psychosocial hypotheses. Mediation analyses were used to explore the stage termination and maturation disparity hypotheses, in which psychological and social variables are hypothesized to serve as mediators of the association between timing and depressive symptoms. Moderation analyses tested the personal accentuation and contextual amplification hypotheses, in which personal and environmental characteristics are hypothesized to interact with timing to predict depressive symptoms. LGMs were constructed to examine moderation effects. Due to limitations in testing mediation using LGMs, conditional process analyses were used to explore mediation.

LGMs were estimated using Mplus Version 6.12 (Muthén & Muthén, 1998-2010) using robust maximum likelihood procedures to account for the non-normal distribution of depressive symptoms. Full information maximum likelihood (FIML) methods were employed to account for

missing data. Model goodness of fit was assessed using several statistics. The chi-square statistic was used to evaluate absolute fit of the models, although large sample sizes can result in a significant chi-square statistic regardless of model fit. The comparative fit index (CFI), root mean square of approximation (RMSEA), and standardized root mean square residual (SRMR) were also examined (Bagozzi, & Yi, 2012; Bentler & Dudgeon, 1996). A CFI greater than or equal to .95, a RMSEA less than or equal to .05, and an SRMR less than or equal to .05 indicate good model fit (Hu & Bentler, 1998).

Conditional process analyses were conducted using the PROCESS macro for SPSS (Hayes, 2013), which uses bootstrapping techniques ($N = 5000$ resamples) to calculate bias-corrected 95% confidence intervals for estimates of direct and indirect effects. A significant indirect effect, as evidenced by a confidence interval that does not include zero, provides evidence of mediation.

All analyses were conducted separately among males and females in order to examine the distinct effects of pubertal timing by gender. Additionally, all analyses included childhood depressive symptoms as a covariate, based on scores on an adapted version of the Reynolds Child Depression Scale (RCDS; Reynolds, 1989). RCDS scores from the earliest possible assessment point (grade 4 for the majority of participants, grade 5 for the most advanced grade cohort) were used. Confirmatory analyses were conducted to ensure that the assessment points at which childhood depressive symptoms were evaluated reflect a time prior to the onset of puberty in the majority of the sample. Among girls whose level of childhood depressive symptoms was assessed in grade 4, a total of 314 (84.7%) had a PDS score of 2.0 or below, indicating minimal pubertal development. All girls assessed in 4th grade had a PDS score of 3.0 or below. Among females whose level of depressive symptoms was assessed in 5th grade, a total of 49 (59.0%) had

a PDS score of 2.0 or below, while 80 (96.4%) had a PDS score of 3.0 or below. Thus, although some girls had begun to show signs of puberty by the time depressive symptoms were measured, the majority of participants had not begun or were in the earliest stages of puberty, suggesting that covarying depressive symptoms in grades 4 and 5 is an appropriate means of controlling for the effect of childhood depression on the association between pubertal timing and subsequent depressive symptoms.

Mediation analyses examining the stage termination and maturation disparity

hypotheses. Four hypothesized mediators (coping, perceived stress, self-image, social competence) were examined with regard to the stage termination (early timing leads to increased depressive symptoms) and maturation disparity (both early and late timing are associated with increased depressive symptoms) hypotheses. Separate models were constructed for each proposed mediator.

All mediation models examined outcomes at two discrete points (grades 9 and 12) rather than as trajectories of symptoms over time, due to restrictions associated with conditional process analyses. Grade 12 was selected as the final assessment point in mediation analyses in order to explore the oldest age at which data on most participants were available ($N = 876$ participants had data on depressive symptoms in grade 12, whereas $N = 650$ participants had data on depressive symptoms one year post-high school).

Two sets of mediation models were constructed. The first predicted depressive symptoms in grades 9 and 12 from a linear pubertal timing variable, in order to test the stage termination hypothesis. To support stage termination, models would need to reveal that early timing was associated with declines in coping, self-image, or social competence or increases in perceived stress during middle school that at least partially explained increases in depressive symptoms in

9th or 12th grade. The second set of mediation analyses predicted depressive symptoms from a quadratic pubertal timing variable, in which linear timing was included as a covariate. These models were designed to test the maturation disparity hypothesis. In order to support maturation disparity, these models would need to indicate that both early and late timing were associated with declines in coping, self-image, or social competence or increases in perceived stress that at least partially explained subsequent increases in depressive symptoms.

Moderation analyses examining the personal accentuation and contextual amplification hypotheses. LGMs were constructed to examine the role of personal characteristics (Big Five personality traits) and contextual stressors (parental depressive symptoms, stressful life events, parent-child relationship conflict, and neighborhood stress) as moderating variables of the association between pubertal timing and depressive symptoms. Separate analyses were conducted for each hypothesized moderator.

As illustrated in Figure 2, the initial moderation model incorporated both linear and quadratic pubertal timing as well as interaction terms created by multiplying each of the pubertal timing variables by the moderator of interest. Examining the significance of the quadratic interaction term revealed the presence or absence of an interaction between quadratic timing and the moderator of interest in the prediction of the latent intercept and latent slope of depressive symptoms. In cases where the quadratic interaction term was nonsignificant, a second LGM was constructed excluding quadratic pubertal timing in order to test for the presence of a linear interaction (Figure 3).

In order to provide evidence in support of the personal accentuation or contextual amplification hypotheses, the moderation models would need to show that proposed moderators

interacted with either quadratic or linear pubertal timing to predict the intercept or linear slope of depressive symptoms.

Results

Descriptive Statistics

Table 1 presents descriptive statistics for all study variables by gender. Bivariate correlations among study variables are presented in Table 4.

Main Effects of Pubertal Timing on Depressive Symptoms

To examine the main effects of linear and quadratic pubertal timing on depressive symptoms across adolescence and into young adulthood, we constructed LGMs separately by gender. Among females, we examined three models in order to determine which would provide the best fit to the data. The first model included three latent factors representing an intercept, linear slope, and quadratic slope of depressive symptoms over time. The second model included only the intercept and linear slope factors, while the third model contained a single latent factor representing the intercept of depressive symptoms. The three-factor model provided good fit to the data ($\chi^2(14) = 30.22, p = .01$; RMSEA = 0.05 (90% CI: .02, .07); CFI = 0.97; SRMR = 0.03) while the two-factor model provided adequate fit to the data ($\chi^2(20) = 54.90, p < .001$; RMSEA = 0.06 (90% CI: .04, .08); CFI = 0.94; SRMR = 0.04). The one-factor model did not provide acceptable fit to the data ($\chi^2(25) = 85.32, p < .001$; RMSEA = 0.07 (90% CI: .05, .08); CFI = 0.90; SRMR = 0.05). We chose to utilize the most parsimonious model that also provided acceptable fit to the data, and therefore selected the two-factor model.

As illustrated in Figure 4, the effect of linear pubertal timing on the intercept of depressive symptoms among females was significant ($\beta = 0.17, SE = 0.06, p = .003$), while the effect of quadratic pubertal timing did not attain significance ($\beta = -0.09, SE = 0.06, p = .13$). This

indicates that pubertal timing was significantly associated with depressive symptoms in 9th grade, such that early-maturing girls exhibited the highest levels of depressive symptoms. There were no significant effects of linear or quadratic pubertal timing on the linear slope of depressive symptoms over time among females.

Among males, we again examined three separate LGMs in order to determine which would provide the best fit. The three-factor model included factors representing an intercept, linear slope, and quadratic slope of depressive symptoms over time and showed excellent fit to the data ($\chi^2(14) = 11.82, p = .62$; RMSEA = 0.00 (90% CI: .00, .04); CFI = 1.00; SRMR = 0.02). The two-factor model, which included latent factors representing the intercept and linear slope, also demonstrated excellent fit ($\chi^2(20) = 14.65, p = .80$; RMSEA = 0.00 (90% CI: .00, .03); CFI = 1.00; SRMR = 0.03). Finally, the one-factor model, containing only a latent intercept factor, showed reduced fit compared to the previous models ($\chi^2(25) = 51.71, p = .001$; RMSEA = 0.05 (90% CI: .03, .06); CFI = 0.93; SRMR = 0.05). Thus, the two-factor model was selected both for its relative parsimony and excellent model fit, as well as to remain consistent with the model utilized among female participants.

As illustrated in Figure 5, there were no significant effects of linear or quadratic pubertal timing on either the latent intercept or latent linear slope of depressive symptoms among males.

Of note, across all LGMs examining outcomes in females and males, the effect of childhood depressive symptoms on the latent intercept of depressive symptoms was highly statistically significant (all $ps < .003$), reaffirming the strong relationship between depressive symptoms in early childhood and adolescent depression.

Mediation of the Association between Pubertal Timing and Depressive Symptoms

In order to test the stage termination hypothesis, we examined mediation models in which linear pubertal timing predicted depressive symptoms in grades 9 and 12 via four proposed mediators (coping, perceived stress, self-image, and social competence). As summarized in Table 5, the direct effect of linear pubertal timing on depressive symptoms among females in 12th grade was significant in all analyses, such that earlier timing was associated with higher levels of depressive symptoms in 12th grade, even in the presence of a significant indirect effect. There were no other significant direct effects among females or males.

Examining possible mediators of the link between linear timing and depressive symptoms revealed two significant findings, both relating to social competence among females. Early maturation was associated with lower levels of social competence in grades 6 and 7, which in turn was linked to higher levels of depressive symptoms in both 9th (95% CI: .02, .71) and 12th (95% CI: .03, .60) grades. There was no evidence of social competence as a mediator among males. Furthermore, coping, perceived stress, and self-image did not emerge as significant mediators of the link between linear timing and depressive symptoms in 9th or 12th grade among youth of either gender (Table 5).

To test the maturation disparity hypothesis, we explored whether coping, perceived stress, self-image, or social competence would at least partially mediate the link between quadratic pubertal timing and depressive symptoms in grades 9 and 12. As summarized in Table 6, there was no evidence of significant direct effects of quadratic timing on depressive symptoms in 9th or 12th grade among females or males. However, one significant indirect effect emerged. Among males, both early and late maturation were associated with higher levels of coping in 8th grade, which in turn was associated with lower levels of depressive symptoms in 9th grade (95% CI: -.59, -.05). This is the opposite of what would be predicted by the maturation disparity

hypothesis and is also contrary to previous literature supporting associations between off-time maturation among males and negative outcomes, including increases in depressive symptoms (Benoit *et al.*, 2013; Natsuaki, Biehl, *et al.*, 2009; Conley & Rudolph, 2009; Graber *et al.*, 1997; Kaltiala-Heino, Kosnunen, *et al.*, 2003).

There was no evidence in support of coping as a mediator of the association between quadratic timing and 12th grade depressive symptoms among males or support for coping as a mediator among females. Perceived stress, self-image, and social competence did not emerge as mediators of the link between quadratic timing and depressive symptoms in 9th or 12th grade among youth of either gender (Table 6).

Moderation of the Association between Pubertal Timing and Depressive Symptoms by Personal Characteristics and Contextual Factors

Fit indices for all moderation analyses among females are located in Table 7, while fit indices for moderation analyses among males are presented in Table 8.

Personal accentuation.

Extraversion. Among females, there were no significant effects of quadratic pubertal timing, extraversion, or their interaction on the intercept or linear slope of depressive symptoms. When examining a model that excluded the quadratic timing variable, the effect of extraversion on the intercept of depressive symptoms was significant ($\beta = -0.17$, $SE = 0.08$, $p = .04$), such that higher levels of extraversion were associated with lower levels of girls' depressive symptoms in 9th grade. However, there was no evidence of significant interactions between linear timing and extraversion in the prediction of the intercept or linear slope of depressive symptoms.

Among males, there was no evidence of an interaction between quadratic timing and extraversion in the prediction of the latent intercept or linear slope of depressive symptoms.

However there was a significant main effect of extraversion on the intercept of depressive symptoms among males ($\beta = -0.23$, $SE = 0.10$, $p = .02$), such that more extraverted males showed lower levels of depressive symptoms in 9th grade. A similar pattern of results was observed in the model in which the quadratic timing variable was omitted, such that the only significant finding was the main effect of extraversion on the latent intercept of depressive symptoms ($\beta = -0.30$, $SE = 0.08$, $p < .001$).

Agreeableness. There were no significant interactions between quadratic or linear timing and agreeableness among females. However, the main effect of agreeableness on the intercept of depressive symptoms was significant in the model including quadratic timing ($\beta = -0.24$, $SE = 0.08$, $p = .003$) as well as the model excluding the quadratic variable ($\beta = -0.19$, $SE = 0.07$, $p = .01$), such that girls high in agreeableness exhibited the lowest depressive symptoms in 9th grade.

Among males, there were no significant main or interaction effects in the prediction of the latent intercept or linear slope of depressive symptoms in either model.

Conscientiousness. Among females, there were no significant main or interaction effects in the prediction of the latent intercept of depressive symptoms in the model including quadratic timing. However, the interaction of quadratic timing and conscientiousness significantly predicted the linear slope of depressive symptoms across adolescence and into young adulthood ($\beta = 1.09$, $SE = 0.46$, $p = .02$). As illustrated in Figure 6, highly conscientious females who matured earlier or later than peers exhibited a positive linear slope of depressive symptoms, while highly conscientious females who matured on-time exhibited a negative slope. Early-maturing females who were low in conscientiousness exhibited no change in depressive symptoms across high school, while late-maturing females who were low in conscientiousness exhibited a negative slope. To aid interpretation of the quadratic interaction effect, Figure 7

illustrates depressive symptoms among females over time based on pubertal timing and level of conscientiousness.

There was a significant main effect of conscientiousness on the latent intercept of depressive symptoms among females in the model omitting quadratic timing ($\beta = -0.15$, $SE = 0.07$, $p = .02$), such that girls with low levels of conscientious reported higher levels of depressive symptoms in 9th grade. There were no additional main or interaction effects in the model in which quadratic timing was omitted.

There were no significant effects among males in the model that included quadratic timing. In the model without the quadratic variable, there was a main effect of conscientiousness on the latent intercept of depressive symptoms among males ($\beta = -0.18$, $SE = 0.06$, $p = .004$), such that more conscientious boys showed lower levels of depressive symptoms in 9th grade.

Emotional stability. Among females, the interaction between quadratic timing and emotional stability was significant in the prediction of the intercept of depressive symptoms ($\beta = -0.52$, $SE = 0.24$, $p = .03$). As illustrated in Figure 8, there was a significant main effect of emotional stability on the intercept of depressive symptoms ($\beta = -0.19$, $SE = 0.08$, $p = .03$), such that females who were low on emotional stability demonstrated the highest levels of depressive symptoms in 9th grade. Among those with high levels of emotional stability, girls who matured on-time exhibited the highest depressive symptoms while those who matured earlier or later than peers demonstrated the lowest levels of symptoms. There was no evidence of significant main or interaction effects in the prediction of the latent linear slope of depressive symptoms among females in the model including quadratic timing or the model in which it was omitted.

Among males, there were significant main effects of linear pubertal timing ($\beta = 0.54$, $SE = 0.23$, $p = .02$) and emotional stability ($\beta = -0.25$, $SE = 0.09$, $p = .01$) on the latent intercept of

depressive symptoms in the model including quadratic timing. Early-maturing boys as well as those exhibiting lower levels of emotional stability had higher levels of depressive symptoms in 9th grade. There was no evidence of a quadratic interaction. In the model excluding quadratic timing, the main effects of linear timing ($\beta = 0.50$, $SE = 0.22$, $p = .02$) and emotional stability ($\beta = -0.23$, $SE = 0.07$, $p < .001$) in the prediction of the latent intercept remained significant. The interaction between linear timing and emotional stability did not significantly predict the latent intercept of depressive symptoms, and there were no effects of timing, emotional stability, or their interaction on the latent linear slope of depressive symptoms among males in either model.

Openness to experience. There were no significant effects of quadratic or linear timing, openness to experience, or their interactions on the intercept of depressive symptoms among females in either model. However, there was a significant interaction between quadratic timing and openness in the prediction of the linear slope of depressive symptoms ($\beta = 1.73$, $SE = 0.64$, $p = .01$). As presented in Figure 9, off-time females who were high on openness exhibited a positive linear slope of depressive symptoms, while those who were highly open to experience and matured on-time exhibited a negative slope. Among those low on openness to experience, the opposite pattern was observed, in which females who matured off-time exhibited a small decline in depressive symptoms across high school while those who matured on-time exhibited a small increase in symptoms over time. Additionally, the main effects of quadratic pubertal timing ($\beta = -1.58$, $SE = 0.66$, $p = .02$) and openness to experience ($\beta = -0.24$, $SE = 0.11$, $p = .03$) on the latent linear slope were statistically significant. Figure 10 depicts the effects of pubertal timing and openness to experience on depressive symptoms among females over time in order to assist in the interpretation of the quadratic interaction effect.

Among males, no significant effects were observed in the model that incorporated the quadratic timing variable. In the model omitting quadratic timing, there was a significant main effect of openness to experience on the latent intercept of depressive symptoms ($\beta = -0.17$, $SE = 0.07$, $p = .02$), such that higher levels of openness were associated with lower levels of depressive symptoms in 9th grade among boys. There was no evidence of a significant interaction between linear timing and openness to experience in the prediction of the latent intercept of depressive symptoms nor were there any significant predictors of the latent linear slope among males.

In summary, there was evidence that emotional stability moderated the association between pubertal timing and depressive symptoms in grade 9 among females, such that girls who were low on emotional stability had the highest level of symptoms overall, while among those with high emotional stability, girls who matured off-time exhibited the lowest depressive symptoms. There was also evidence of personal accentuation in the prediction of the linear slope of depressive symptoms between grade 9 and one year post-high school among females, such that early-maturing girls who were low on conscientiousness and openness to experience exhibited the highest levels of symptoms over time. Trait levels of extraversion and agreeableness did not show evidence of personal accentuation among females in the prediction of 9th grade depressive symptoms or the trajectory of symptoms over time. Males did not show any evidence of personal accentuation by Big Five personality traits.

Contextual amplification.

Maternal depressive symptoms. Among females, there was no evidence of interactions between quadratic or linear pubertal timing and maternal depressive symptoms in the prediction of the latent intercept or latent linear slope of youth depressive symptoms. However, there was a

significant main effect of maternal depressive symptoms on the intercept of youth depression in the model incorporating the quadratic timing variable ($\beta = 0.23$, $SE = 0.07$, $p = .002$) as well as the model without quadratic timing ($\beta = 0.26$, $SE = 0.06$, $p < .001$). In both cases, higher levels of maternal depression were associated with higher levels of girls' depressive symptoms in 9th grade.

Among males, there was a significant main effect of maternal depressive symptoms on the latent linear slope of depressive symptoms in the model including the quadratic timing variable ($\beta = 0.24$, $SE = 0.12$, $p = .04$) as well as the model in which quadratic timing was omitted ($\beta = 0.19$, $SE = 0.09$, $p = .02$), such that boys with more depressed mothers exhibited a steeper positive slope of depressive symptoms across high school. There was also a significant main effect of linear pubertal timing on the latent intercept of youth depressive symptoms in the model omitting quadratic timing ($\beta = 0.18$, $SE = 0.09$, $p = .05$), such that boys with high levels of maternal depressive symptoms exhibited the highest levels of depression in 9th grade. No other significant effects emerged among males.

Paternal depressive symptoms. There was no evidence of quadratic or linear interactions between pubertal timing and paternal depressive symptoms in the prediction of the latent intercept or latent linear slope of youth depressive symptoms among females. However, there was a main effect of paternal depression on the latent intercept of youth depressive symptoms in the model including quadratic timing ($\beta = 0.31$, $SE = 0.08$, $p < .001$) as well as the model in which it was omitted ($\beta = 0.33$, $SE = 0.06$, $p < .001$), such that girls whose fathers exhibited high levels of depression reported higher depressive symptoms themselves in 9th grade.

There were no significant effects of quadratic or linear pubertal timing, paternal depressive symptoms, or their interaction on the latent intercept or latent linear slope of males' depressive symptoms in either model.

Stressful life events. Among females, the interaction of quadratic timing and stressful life events did not attain statistical significance in the prediction of either the latent intercept or the latent linear slope. However, the main effects of linear pubertal timing ($\beta = 0.24$, $SE = 0.08$, $p = .004$), quadratic timing ($\beta = -0.21$, $SE = 0.08$, $p = .01$), and stressful events ($\beta = 0.24$, $SE = 0.07$, $p < .001$) were all significant in the prediction of the latent intercept of depressive symptoms. Early-maturing girls as well as those reporting the highest levels of stressful life events exhibited the highest levels of depression in 9th grade. The main effect of quadratic timing was also significant in the prediction of the linear slope of depressive symptoms among females ($\beta = 0.30$, $SE = 0.12$, $p = .01$), such that early- and late-maturing girls exhibited steeper positive slopes of depressive symptoms over time than girls who developed on-time.

In the model omitting quadratic timing, the main effects of linear pubertal timing ($\beta = 0.19$, $SE = 0.08$, $p = .02$) and stressful life events ($\beta = 0.33$, $SE = 0.05$, $p < .001$) were again significant in the prediction of the intercept of depressive symptoms among females, such that early timing and more stress were associated with higher levels of depression in grade 9. The main effect of stressful events was also significant in the prediction of the latent linear slope ($\beta = -0.26$, $SE = 0.07$, $p < .001$). Girls who reported more stressful events exhibited a larger negative slope of depressive symptoms across adolescence and into young adulthood.

Among males, there was a significant main effect of stressful life events on the latent intercept of depressive symptoms in the model including quadratic timing ($\beta = 0.27$, $SE = 0.09$, $p = .003$), such that boys who experienced higher levels of stressful life events during puberty

exhibited the highest levels of depressive symptoms in 9th grade. There was also a significant main effect of quadratic timing on the latent linear slope of depressive symptoms ($\beta = -0.29$, $SE = 0.12$, $p = .01$), such that males who matured on-time relative to peers exhibited a steeper positive slope of depressive symptoms over time than those who matured either early or late.

In the model in which the quadratic timing variable was omitted, there were significant main effects of linear timing ($\beta = 0.23$, $SE = 0.10$, $p = .02$) and stressful life events ($\beta = 0.18$, $SE = 0.06$, $p = .003$) on the latent intercept of depressive symptoms among males, such that earlier puberty and higher levels of stress were associated with more depressive symptoms in 9th grade. No additional significant effects were observed among males.

Maternal relationship conflict. There was no evidence of quadratic or linear interactions between pubertal timing and conflict with mothers in the prediction of the latent intercept or latent slope of depressive symptoms among females. However, there was a main effect of maternal conflict on the intercept of girls' depressive symptoms in the model including quadratic timing ($\beta = 0.21$, $SE = 0.07$, $p = .003$) as well as the model that omitted the quadratic timing variable ($\beta = 0.26$, $SE = 0.06$, $p < .001$), such that girls who reported high levels of conflict with their mothers in grades 6 through 8 reported the highest levels of depressive symptoms in 9th grade. Additionally, there was a significant main effect of linear timing on the latent intercept of depressive symptoms in the model omitting the quadratic timing variable ($\beta = 0.18$, $SE = 0.09$, $p = .04$), such that early maturation was association with higher levels of depressive symptoms in 9th grade.

There was a significant main effect of maternal conflict on the latent intercept of depressive symptoms among males in the model incorporating quadratic timing ($\beta = 0.28$, $SE = 0.08$, $p = .001$) as well as the model omitting quadratic timing ($\beta = 0.27$, $SE = 0.06$, $p < .001$). In

both cases, higher levels of maternal conflict during puberty were associated with higher levels of depressive symptoms among males in 9th grade. There were no other significant effects among males in either model.

Paternal relationship conflict. Among females, the interaction between quadratic pubertal timing and conflict with fathers in the prediction of the latent linear slope of youth depressive symptoms was significant ($\beta = -0.38$, $SE = 0.17$, $p = .03$). As illustrated in Figure 11, off-time females with low levels of paternal conflict exhibited the largest positive linear slope of depressive symptoms, while on-time females with low levels of conflict demonstrated a negative slope of depressive symptoms. On-time females reporting high paternal conflict exhibited a relatively flat slope of depressive symptoms over time, while off-time females with high levels of paternal conflict had a negative slope of symptoms. To aid in the interpretation of these findings, Figure 12 illustrates depressive symptoms among females over time according to pubertal timing and levels of paternal conflict. In addition to the significant interaction, the main effect of quadratic timing was also a significant predictor of the latent linear slope of depressive symptoms among females ($\beta = 0.40$, $SE = 0.12$, $p = .001$).

There was no evidence of an interaction between quadratic timing and paternal conflict in the prediction of the latent intercept of depressive symptoms among females. However, the main effects of quadratic timing ($\beta = -0.22$, $SE = 0.11$, $p = .03$) and paternal conflict ($\beta = 0.17$, $SE = 0.07$, $p = .01$) were significant predictors of the intercept. Girls with high levels of paternal conflict exhibited the highest levels of depressive symptoms in 9th grade, while late-maturing girls demonstrated lower levels of symptoms than either on-time or early-maturing girls. In the model omitting the quadratic timing variable, there was no evidence of a significant linear

interaction in the prediction of the intercept of depressive symptoms, although the main effect of paternal conflict on the intercept was once again significant ($\beta = 0.25$, $SE = 0.09$, $p < .001$).

Among males, there was a significant main effect of conflict with fathers on the intercept of youth depressive symptoms when the quadratic timing variable was included in the model ($\beta = 0.34$, $SE = 0.09$, $p < .001$) as well as when it was omitted ($\beta = 0.30$, $SE = 0.06$, $p < .001$). Boys who reported high levels of paternal conflict during puberty exhibited the highest levels of depressive symptoms in 9th grade. There were no other significant effects among males.

Neighborhood stress. There was a significant interaction between quadratic timing and neighborhood stress in the prediction of the latent linear slope of depressive symptoms among females ($\beta = -1.38$, $SE = 0.48$, $p = .004$). As illustrated in Figure 13, off-time females with low levels of neighborhood stress exhibited the steepest positive slopes of depressive symptoms over time. Late-maturing females with high levels of neighborhood stress exhibited the steepest negative slope of depression. Figure 14 presents depressive symptoms among females over time based on pubertal timing and levels of neighborhood stress. The main effect of quadratic timing was also a significant predictor of the latent linear slope ($\beta = 1.40$, $SE = 0.42$, $p = .001$) in this model.

There was no evidence of an interaction between quadratic timing and neighborhood stress on the latent intercept among females, although there was a significant main effect of neighborhood stress ($\beta = 0.35$, $SE = 0.08$, $p < .001$), such that girls living in more stressful neighborhoods had the highest levels of depressive symptoms in 9th grade. Similarly, there was no evidence of an interaction between linear timing and neighborhood stress in the prediction of the intercept of depressive symptoms in the model omitting quadratic timing, although the main effect of neighborhood stress on the intercept was once again significant ($\beta = 0.40$, $SE = 0.06$, p

< .001). As in the model incorporating the quadratic timing variable, higher levels of neighborhood stress predicted higher levels of 9th grade depressive symptoms among females.

Among males, there was a significant main effect of neighborhood stress on the intercept of depressive symptoms when the quadratic timing variable was included in the model ($\beta = 0.39$, $SE = 0.10$, $p < .001$) as well as when it was omitted ($\beta = 0.32$, $SE = 0.08$, $p < .001$). Boys who reported high levels of neighborhood stress during puberty exhibited the highest levels of depressive symptoms in 9th grade. There were no other significant effects among males.

In summary, there was some evidence of contextual amplification among females in the present study. Girls who reported high levels of paternal conflict and neighborhood stress exhibited the highest levels of depressive symptoms over time, while early-maturing girls with low levels of paternal conflict and neighborhood stress had lower initial levels of symptoms that increased between 9th grade and one year post-high school. Parental depressive symptoms, stressful life events, and maternal relationship conflict did not emerge as contextual amplifiers of timing's effect on depressive symptoms among females, although these characteristics frequently exerted effects on the intercept or slope of symptoms in girls, independent of timing. There was no evidence of contextual amplification among males, although every proposed moderator other than paternal depression was associated with males' depressive symptoms in 9th grade and several were also linked to the trajectory of depressive symptoms in males over time.

Discussion

The present study sought to provide a comprehensive test of four psychosocial hypotheses that have been theorized to account for observed associations between off-time pubertal development and depressive symptoms among youth. The current results are some of the first empirical findings (Rudolph *et al.*, 2014; Conley *et al.*, 2012) to support stage

termination among girls, indicating that poor interpersonal competence is one mechanism through which early maturation is linked to adolescent depression among females. Study 1 found no evidence of stage termination among males and did not support maturation disparity among youth of either gender. Findings regarding personal accentuation and contextual amplification were primarily related to the trajectory of depressive symptoms among females and generally suggested that personality traits and environmental stressors exert a stronger independent effect on adolescent depression than interactions between these experiences and pubertal timing. Taken together, the results indicate that early-maturing girls are at the highest relative risk for depression and provide some evidence for mechanisms of this effect. However, the present findings also suggest that off-time development may not exert a pronounced independent effect on the occurrence of depressive symptoms among youth who are otherwise at low risk for depression.

Pubertal Timing and Depressive Symptoms

The present results corroborate prior findings supporting the deleterious impact of early timing on psychological well-being among adolescent females (Graber, 2013; Mendle *et al.*, 2007), revealing that early-maturing girls demonstrate higher levels of depressive symptoms in 9th grade than female peers. However, there was no evidence that the linear trajectory of depressive symptoms across adolescence and into young adulthood was affected by pubertal timing among females. Early-maturing girls exhibited stable, high levels of symptoms between grade 9 and one year after high school while on-time and late-maturing girls experienced lower levels of symptoms that also remained stable over time. Although a priori hypotheses about the trajectory of symptoms were not made in the present study, research has suggested that pubertal timing can influence symptoms in young adulthood when examining static outcomes at a single

timepoint (Graber *et al.*, 2004), including evidence from the current study that early timing is associated with heightened levels of depression among females in 12th grade. Thus, it appears that early maturation among females is associated with high levels of depressive symptoms in adolescence that remain consistently elevated into young adulthood. The present study is one of the first to explore the influence of pubertal timing on the trajectory of symptoms over time (Gaysina *et al.*, 2015), and future research should continue to assess the long-term impact of pubertal processes on psychopathology among females. Given that the present study was limited to a linear trajectory of symptoms, studies examining non-linear trajectories (e.g., quadratic, cubic, piecewise) are particularly needed.

Somewhat surprisingly, no associations between timing and depressive symptoms were observed among males in the present sample. Previous studies regarding the association between timing and depression in males have reached varying conclusions about the impact of early, late, and both early and late development on psychopathology (Mendle & Ferrero, 2012), including two studies that failed to find an association between timing and depression (Crockett *et al.*, 2013; Kaltiala-Heino, Marttunen, *et al.*, 2003). The present study examined both linear and quadratic associations and conducted analyses separately by gender to increase the likelihood of capturing the true relationship between timing and depressive symptoms among males. Thus, it may be that compared to females, males are generally less likely to react to variations in timing by becoming depressed, perhaps due to gender differences in the salience or psychological significance of puberty (Rudolph, 2014). The consequences of timing among males may also be more apparent when examining outcomes other than depression, such as externalizing conditions (Dimler & Natsuaki, 2015; Mendle & Ferrero, 2012).

Stage Termination and Maturation Disparity

The present study is one of the first to support stage termination among females. Early-maturing girls reported reduced social competence in middle school, which in turn was associated with higher levels of depressive symptoms in grades 9 and 12. This suggests that girls who mature early may not yet have developed the interpersonal skills that would allow them to respond effectively to the challenges of adolescence by the time they initiate puberty, resulting in increased depression (Stattin & Magnusson, 1990; Caspi & Moffitt, 1991). Moreover, this skills deficit appears to have long-term effects on psychological well-being, with low social competence leading to heightened depressive symptoms through 12th grade, far beyond the end of pubertal development. Thus, early puberty and associated changes in interpersonal competence may represent one pathway through which women are more likely to develop depression across the lifespan, contributing to the gender disparity in depression prevalence. These findings suggest that in addition to treatments designed to ameliorate symptoms of depression, preventative efforts such as social skills training targeting early-maturing girls may be of particular use in reducing the overall prevalence of depression and diminishing the gender gap (Stice, Shaw, Bohon, Marti, & Rohde, 2009).

Of note, social competence was assessed using a self-report measure in the present study, leading to important questions regarding the degree to which interpersonal deficits are primarily a perception of the adolescent or reflect actual limitations in interpersonal skills among early-maturing females. Future research examining stage termination should seek to differentiate between subjective and objective assessments of social competence in order to provide more information about the specific mechanism through which early maturation is linked to higher depressive symptoms in girls.

Contrary to hypotheses, coping, perceived stress, and self-image did not serve as mechanisms of the association between early timing and depression in girls. Perhaps these mechanisms are only relevant to certain subgroups of adolescent females, such as those already at high risk of depression due to other factors (e.g., history of maternal depression, high levels of environmental stress). Given the small number of studies examining stage termination to date (Rudolph *et al.*, 2014; Conley *et al.*, 2012), future research should continue to test this hypothesis and seek to replicate the obtained results in order to ascertain what processes link early maturation with the occurrence of depressive symptoms among girls. In particular, moderated mediation analyses would help to identify which mechanisms are relevant to specific groups.

Consistent with study hypotheses, there was no evidence of stage termination among males. However, coping served as a mediator of the association between off-time development and boys' depressive symptoms in the present sample. Contrary to what would be predicted by the maturation disparity hypothesis, the obtained result suggests that early- and late-maturing boys exhibit more effective coping techniques, which in turn leads to lower levels of depressive symptoms in 9th grade. Given that this is the only significant finding to emerge from many analyses examining the impact of quadratic timing on depressive symptoms as well as the limitations of the coping measure used in the present study, it should be interpreted with caution. Future research should utilize comprehensive measures of coping that have demonstrated appropriate psychometric properties in an effort to replicate the current finding before meaningful conclusions can be drawn. In general, future studies should also seek to test maturation disparity more thoroughly, given that this is the first empirical test of the hypothesis to date. Consistent with study hypotheses, there was no evidence of maturation disparity among females in the present sample.

Personal Accentuation

Exploring the moderating role of personality traits in the association between pubertal timing and depressive symptoms revealed several interesting findings among females. Consistent with study hypotheses, emotional stability interacted with timing to predict depressive symptoms in 9th grade, although the result was not entirely consistent with the personal accentuation hypothesis. Girls with low levels of emotional stability demonstrated the most depressive symptoms overall, while among those with high emotional stability, individuals who matured on-time exhibited the highest level of depression. Thus, rather than emphasizing the interaction of timing and emotional stability, the results highlight the main effect of emotional stability on adolescent depressive symptoms.

Contrary to hypotheses, there was no evidence of personal accentuation with regard to the remaining four personality traits (extraversion, agreeableness, conscientiousness, openness to experience) in the prediction of females' depressive symptoms in 9th grade, although there was evidence of significant main effects of these traits. In combination with the finding related to emotional stability, this suggests that the direct influence of personality traits on adolescent depressive symptoms may be more relevant to girls at low risk for depression than the combined effects of pubertal timing and personality. Personal accentuation is inherently a hypothesis about risk for depression, and girls in a community sample may not surpass a necessary threshold of risk beyond which the effects of timing and negative personality traits on adolescent symptoms are particularly influential. Previous studies supporting personal accentuation have sometimes utilized samples selected to be at higher risk of depression than the general population (e.g., Rudolph & Troop-Gordon, 2010), which may result in a greater likelihood of observing personal accentuation effects when examining depressive symptoms at a particular timepoint. Thus, future

studies should continue to test personal accentuation in both low- and high-risk samples in order to identify the specific conditions under which negative personality traits exacerbate the effects of off-time development.

Given the paucity of existing research on the association between pubertal timing and trajectories of depressive symptoms over time, no a priori hypotheses were made regarding personal accentuation processes in the prediction of depressive symptoms between grade 9 and one year post-high school. However, both conscientiousness and openness to experience emerged as significant moderators of the association between timing and the trajectory of symptoms among females. As illustrated in Figures 7 and 10, early-maturing girls with low levels of conscientiousness and openness to experience exhibited the highest levels of symptoms across high school and showed a slight increase in symptoms over time. Late-maturing females with low levels of conscientiousness and openness as well as girls who were highly conscientious and open to experience regardless of timing had lower levels of depressive symptoms and generally exhibited flat or negative slopes.

These findings provide some of the first evidence supporting Big Five personality traits as contributors to personal accentuation processes in the prediction of symptom trajectories, indicating that early-maturing girls with low conscientiousness and openness to experience exhibit a relatively stable trajectory of high depressive symptoms into young adulthood. Consistent with stage termination, perhaps these girls experience a mismatch between physical and emotional maturity, leaving them unprepared to effectively address situations that may arise based on their level of physical development. For example, early-maturing girls with low openness to experience could be asked to engage in social interactions with which they are not comfortable (e.g., dating) and for which they have not developed adequate interpersonal skills,

leading to ongoing distress that results in higher levels of depressive symptoms over time. As discussed above, future research should seek to combine the exploration of moderators and mediators of the timing-depression link in order to identify the mechanisms that lead certain groups to exhibit higher levels of depressive symptoms than others. Of note, given that evidence of personal accentuation in the prediction of symptom trajectories was observed in a low-risk community sample in the present study, it is possible that personal accentuation processes among girls at high risk for depression might further disadvantage early-maturing females when examining trajectories of symptoms over time.

The present findings did not support personal accentuation with regard to extraversion or agreeableness when examining symptom trajectories among females, and contrary to hypotheses, males did not show any evidence of personal accentuation in the prediction of 9th grade depressive symptoms or the trajectory of symptoms over time. However, consistent with prior literature supporting a direct association between personality traits and depression (Bagby *et al.*, 2008; Kendler *et al.*, 2006), many of the Big Five personality traits significantly predicted depressive symptoms in grade 9 among both females and males regardless of pubertal timing. As discussed above, it is possible that adolescents at low risk for depression, such as those assessed in the present study, may not surpass a threshold of risk beyond which associations between timing, personality, and depressive symptoms would be consistently apparent. Thus, the independent effects of personality traits emerged as stronger predictors of symptoms than interactions between personality traits and timing in the majority of analyses conducted in the present sample. However, given that the current study is one of the few to test personal accentuation in males, future research should continue to explore personal accentuation among

both low- and high-risk boys to determine what, if any, personal characteristics influence the association between timing and psychopathology among males.

Contextual Amplification

Contrary to previous research supporting contextual amplification and against study hypotheses, there was no evidence that environmental stressors amplified the effects of pubertal timing in the prediction of 9th grade depressive symptoms among either females or males. As with the analyses exploring Big Five personality traits, many of the environmental stressors exerted strong independent effects on depressive symptoms, but did not interact with timing. Again, the use of a community sample may have limited the likelihood of obtaining evidence in support of contextual amplification given the strong direct associations between contextual stressors and depression. Previous research in support of contextual amplification processes on adolescent depressive symptoms has been conducted in both high-risk (Rudolph & Troop-Gordon, 2010; Conley & Rudolph, 2009) and community samples (Benoit *et al.*, 2013; Ge *et al.*, 2001a; Ge *et al.*, 2001b), indicating that contextual amplification of the negative effects of off-time maturation may still be relevant to the experiences of adolescents at low risk for depression.

Examining contextual amplification processes with regard to the trajectory of symptoms over time revealed that both paternal conflict and neighborhood stress serve to moderate the association between timing and depressive symptom trajectories among females. As illustrated in Figures 12 and 14, high levels of paternal conflict and neighborhood stress were associated with high levels of depressive symptoms that declined slightly between 9th grade and one year post-high school. Among girls with low levels of conflict with their fathers or neighborhood stress, early-maturing girls demonstrated a steep positive trajectory over time, such that their level of depressive symptoms increased consistently into young adulthood. Commensurate with the

findings on personal accentuation as well as analyses examining contextual amplification in the prediction of depressive symptoms in grade 9, these findings highlight the strong influence of environmental stressors on depressive symptoms among females at low risk for depression, regardless of pubertal timing. Additionally, the results suggest that early-maturing girls are at heightened risk of depression even when experiencing low levels of stress. While this conclusion might be overlooked in analyses that examine depressive symptoms at a single point in adolescence, the exploration of trajectories of symptoms revealed that even when girls experience low levels of stress and exhibit relatively low levels of symptoms in grade 9, early maturers are likely to exhibit increasing levels of depression over time. These findings indicate that contextual amplification processes may be more nuanced than was previously understood. Future studies examining trajectories of symptoms across adolescence and into later stages of adulthood will help to elucidate whether early-maturing girls with low contextual stress eventually experience levels of depressive symptoms that are similar to those of individuals in high-stress environments or if symptoms eventually stabilize. Studies that examine piecewise trajectories of depressive symptoms, in which the slope of symptoms is allowed to vary over time, are particularly needed.

Notably, childhood depressive symptoms showed a strong association with adolescent depressive symptoms in all analyses. This suggests that early onset of depression (i.e., prior to adolescence) may be such a powerful predictor of subsequent symptoms that other factors such as pubertal timing may not greatly enhance risk, particularly within a community sample. Given established associations between early childhood experiences, including prior depressive symptoms (Rudolph & Troop-Gordon, 2010; Ellis & Essex, 2007; Chisholm *et al.*, 2005; Ellis & Garber, 2000) and pubertal timing, future studies should aim to incorporate predictors of timing

in analyses examining psychological outcomes of puberty. Ideally, the field will continue to move toward testing comprehensive models of puberty that incorporate relevant predictors and outcomes of pubertal processes across multiple levels of analysis.

Strengths and Limitations

The present study has a number of strengths, including a large, community sample of youth assessed repeatedly from elementary school through one year post-high school. The inclusion of male participants is also a significant strength given the relative paucity of research on male puberty and psychopathology in the existing literature. Annual assessments allowed for the elucidation of exact temporal associations between variables of interest and ensured that relevant study variables could be measured at meaningful stages of development (e.g., examining pubertal timing among females and males at ages that reflect gender differences in the normative age of pubertal onset). Furthermore, repeated assessments of the same constructs provided data that were truly representative of the context in which puberty occurred and allowed for the exploration of trajectories of depressive symptoms over time. The ability to control for childhood depressive symptoms (measured prior to the onset of puberty in almost all cases) also serves as an important strength. Finally, the present study is one of the first to conduct empirical tests of the stage termination and maturation disparity hypotheses as well as one of the first to explore the impact of pubertal timing on trajectories of psychopathology, rather than examining a single timepoint in adolescence.

The study also has important limitations. The use of a sample recruited from youth living in Oregon necessarily limits the representativeness of the findings. Participants were primarily Caucasian, and most parents had more than a high school education (Andrews *et al.*, 2003). Given observed variations in pubertal development by characteristics such as race and ethnicity

(Kaplowitz, Slora, Wasserman, Pedlow, & Herman-Giddens, 2001; Anderson, Dallal, & Must, 2003), conclusions drawn from the present findings may not apply to all youth. The use of a low-risk, community sample may have also limited the generalizability of the findings and may not represent the experiences of youth at high risk for depression. While studies elucidating the influence of pubertal timing within community samples are needed, future studies should continue to examine both high-risk and community samples that incorporate youth from various racial and socioeconomic backgrounds in order to fully elucidate the mechanisms through which timing affects the development of depressive symptoms.

In addition, some of the measures employed to assess proposed mediating and moderating variables were shortened by the investigators of the parent study. Thus, we are unable to ascertain the precise psychometric properties of these measures. Furthermore, despite the large number of tests conducted, corrections for multiple tests were unable to be applied due to the nature of the present analyses, which did not always provide a specific significance value (e.g., conditional process analyses). Thus, the likelihood of Type I error (i.e., falsely concluding that an effect is present) may be heightened. Although all analyses controlled for prior depressive symptoms, the present study did not include measures of body mass index or adiposity, despite associations between these characteristics and pubertal timing (Biro *et al.*, 2003; Anderson *et al.*, 2003; Kaplowitz *et al.*, 2001). Future studies of puberty should assess and control for adiposity whenever possible. Finally, though the examination of trajectories of depressive symptoms over time represents a major strength, the present analyses were limited to a linear trajectory of symptoms. Future research should explore the use of higher order or piecewise trajectories that might better reflect changes in symptoms over time.

Conclusions

The present study provides an extensive exploration of the psychosocial mechanisms through which pubertal timing has been hypothesized to influence depressive symptoms among females and males across adolescence and into the early stage of young adulthood. Aspects of stage termination, personal accentuation, and contextual amplification were identified as mechanisms of this association among females, while the maturation disparity hypothesis did not explain associations between off-time development and depressive symptoms among adolescents of either gender. Of note, the use of a community sample at low risk for depression may have affected obtained outcomes, such that personality traits and contextual stressors often appeared to exert a stronger independent effect on depressive symptoms than interactions between these characteristics and pubertal timing. Together these findings confirm that early-maturing girls are vulnerable to the development of depressive symptoms and suggest several pathways through which early timing leads to negative outcomes, even when girls are otherwise at low risk for psychopathology.

The present results offer a fertile base for future research by highlighting the importance of examining trajectories of depressive symptoms over time and exploring outcomes beyond adolescence. Research should continue to assess both *how* puberty contributes to depression as well as *who* appears to be particularly vulnerable to the effects of off-time development. Analyses that explore mediation and moderation simultaneously may provide the most nuanced explanation of the processes that contribute to the link between timing and psychopathology.

**Chapter 3: Contextual Amplification Effects of Chronic and Episodic Stress on the
Association between Pubertal Timing and Depressive Symptoms (Study 2)**

Abstract

Depression is a widespread and pernicious condition that is known to dramatically increase in prevalence during adolescence. Variations in the timing of puberty have been identified as one contributor to depressive symptoms, with adolescents who mature off-time relative to peers at the greatest risk. Research suggests that the co-occurrence of environmental stressors during puberty can exacerbate the negative psychological effects of off-time development, a process known as contextual amplification. The present study sought to extend the contextual amplification literature by examining the effect of various types of environmental stressors (e.g., chronic interpersonal, chronic non-interpersonal, episodic) on the association between pubertal timing and depressive symptoms, considering non-linear associations between timing and depression, and assessing outcomes into young adulthood. Among a longitudinal sample of 725 youth, females exhibited the highest levels of depressive symptoms at age 15 when they matured either earlier or later than peers in the context of high levels of chronic interpersonal stress, while early-maturing boys showed the highest levels of depressive symptoms at age 15 in the context of high levels of dependent episodic stress. Following correction for multiple tests, neither females nor males showed contextual amplification effects of non-interpersonal chronic stress, and contextual amplification processes did not affect depressive symptoms in young adulthood. Results contribute to a nuanced understanding of contextual amplification, in which the nature of environmental stress as well as characteristics of the individual affect the relationship between puberty and depressive symptoms. Findings are discussed with regard to the importance of the psychosocial context of pubertal development.

Introduction

Study 2 expands upon the questions explored in Study 1 by providing a fine-grained analysis of contextual amplification processes in the association between pubertal timing and depressive symptoms. Contextual amplification was initially proposed as a hypothesis regarding the influence of social context on the association between timing and psychopathology (Caspi *et al.*, 1993) with the expectation that a distressing or problematic social environment would amplify the negative effects of off-time development. Previous research has generally supported this prediction, with the majority of studies focusing on the negative psychological impact of stressful peer contexts (Caspi *et al.*, 1993; Smolak *et al.*, 1993; Ge *et al.*, 1996; Blumenthal, Leen-Feldner, Trainor, Babson, & Bunaciu, 2009; Conley & Rudolph, 2009; Natsuaki, Biehl, *et al.*, 2009; Sontag *et al.*, 2011; Teunissen *et al.*, 2011; Benoit *et al.*, 2013; Mrug *et al.*, 2014). Additional research has indicated that stressful family (Rudolph & Troop-Gordon, 2010; Benoit *et al.*, 2013; Lynne-Landsman *et al.*, 2010; Ge *et al.*, 2002), and neighborhood (Obeidallah *et al.*, 2004; Ge *et al.*, 2002) environments are associated with increased psychopathology among youth who mature off-time.

Several studies have examined internalizing or depressive symptoms as an outcome of contextual amplification processes (Benoit *et al.*, 2013; Conley & Rudolph, 2009; Natsuaki, Biehl, *et al.*, 2009; Ge *et al.*, 1996; Rudolph & Troop-Gordon, 2010; Ge *et al.*, 2001a; Ge *et al.*, 2001b), with the majority assessing the role of chronic interpersonal stress (i.e., peer or family stress). While the focus on interpersonal stress is reasonable in light of clear associations between interpersonal stressors and depression (Hammen, Shih, & Brennan, 2004; Hammen, 2003), chronic stress in non-interpersonal domains has also been associated with the development of depression (Hammen *et al.*, 2009; Bonde, 2008; Mausner-Dorsch & Eaton,

2000), and many of the non-interpersonal stressors experienced by adolescents and young adults are particularly salient at these stages of life (e.g., academic difficulties, entering the workforce). In fact, previous research has linked off-time maturation with increased difficulties in non-interpersonal domains, such as academic achievement (Mendle & Ferrero, 2012; Mendle *et al.*, 2007), suggesting that the dual experience of non-interpersonal contextual stressors and off-time maturation may heighten risk for psychopathology. However, to the author's knowledge, no previous research has tested contextual amplification effects of non-interpersonal stress.

Additionally, few studies have examined the role of episodic stressful life events on the link between off-time development and depressive symptoms (Ge *et al.*, 2001a; Ge *et al.*, 2001b; Conley & Rudolph, 2009; Chen *et al.*, 2015). Episodic stressful events are strongly linked to the experience of depression, including initial onset of the disorder as well as its recurrence (Hammen *et al.*, 2009; Daley, Hammen, & Rao, 2000; Kessler, 1997). The few previous studies that have assessed episodic events have supported contextual amplification effects of episodic stressors, although one study utilized a composite chronic and episodic stress variable (Conley & Rudolph, 2009) that did not allow for an exploration of the separate impact of each type of stress. Additionally, although this limited body of literature has reliably suggested that the co-occurrence of early maturation and episodic stressful events are predictive of the worst outcomes in girls, the findings among males are not as clear, with Ge *et al.* (2001b) endorsing the detrimental effects of early timing and Conley and Rudolph (2009) emphasizing the risks associated with late timing in males.

Of note, episodic stressors are comprised of two discrete categories of events: those that are independent of the direct influence or action of the individual to whom they occur (e.g., death of a family member) and those that are in some way dependent on the actions of the individual

(e.g., romantic break-up). Thus, youth experiencing high levels of dependent events are actively contributing to stressful environmental contexts, a process that is particularly likely to occur among individuals with a history of depression (Hammen, 1991; Hammen, 2006). Previous research has established that females are especially likely to experience depressive symptoms in response to dependent stress (Kendler, Karkowski, & Prescott, 1999) and this relationship is apparent as early as adolescence (Rudolph & Hammen, 1999), suggesting that distinctions between independent and dependent events are important to explore in the examination of contextual amplification processes.

Contextual amplification has previously been tested primarily with regard to the accuracy of the hypothesis (e.g., whether or not contextual stressors moderate the impact of off-time development on psychopathology), with very little attention to the specific types of stress that may amplify the effects of timing on particular groups of adolescents. The present study seeks to elucidate the specific contextual amplification effects of chronic interpersonal, chronic non-interpersonal, independent episodic, and dependent episodic stress in the association between pubertal timing and depressive symptoms among youth followed into young adulthood. The distinct impact of each type of contextual stressor on the experience of females versus males is of particular interest in the present study, with the goal of increasing understanding of possible gender-specific pathways through which timing affects depressive symptoms.

Aims and Hypotheses

Aim 1. To examine the unique contribution of chronic stressful conditions and episodic stressful life events in the association between pubertal timing and depressive symptoms.

1. Given the relevance of chronic social stress to the development of depression among females (Hammen *et al.*, 2004; Hammen, 2003) as well as evidence in support of

contextual amplification effects of chronic interpersonal stress in both females and males (Benoit *et al.*, 2013; Rudolph & Troop-Gordon, 2010), it is predicted that chronic interpersonal stressors will result in heightened risk of depressive symptoms among early-maturing females and off-time males in adolescence as well as young adulthood. It is expected that this effect will be particularly strong among females.

2. It is hypothesized that non-interpersonal chronic stress will exhibit contextual amplification effects among both males and females. However, given the lack of previous research on contextual amplification due to non-interpersonal stress, no predictions are made regarding the expected direction of these effects (i.e., whether early, late, or both early and late timing will interact with non-interpersonal chronic stress to predict depressive symptoms among females and males).

3. In light of past research supporting the role of episodic stressful life events as moderators of the link between off-time development and depressive symptoms, it is expected that episodic stress will demonstrate evidence of contextual amplification among early-maturing females in adolescence and young adulthood. However, given discrepancies in the findings related to males (i.e., findings demonstrating contextual amplification effects of episodic stressors among both early- and late-maturing boys), predictions regarding the direction of the hypothesized effect in males are not made.

Aim 2. To examine the role of independent and dependent episodic stressors as contextual amplifiers of the link between pubertal timing and depressive symptoms.

1. It is predicted that both independent and dependent stress will result in contextual amplification among females and males, such that early-maturing females exhibit the highest levels of symptoms in the context of high levels of independent and dependent

stress. However, no hypotheses about the direction of the effect in males are made.

Additionally, it is predicted that dependent episodic stressors will exhibit the strongest effect among females.

Aim 3. To examine gender differences in the contextual amplification of off-time pubertal development on depressive symptoms in adolescence and young adulthood.

1. It is predicted that chronic interpersonal and dependent episodic stress will exhibit the strongest contextual amplification effects among females, such that early-maturing females with high levels of chronic interpersonal and dependent episodic stress will exhibit the highest levels of depressive symptoms. No hypotheses are made regarding the role of gender in the contextual amplification of pubertal timing by non-interpersonal chronic and independent episodic stress.

Method

Participants

Youth in the present sample were drawn from participants in the Mater Misericordiae Mothers' Hospital-University of Queensland Study of Pregnancy (MUSP; Keeping *et al.*, 1989), a longitudinal study of over 7000 youth born in Brisbane, Queensland, Australia between 1981 and 1984. As part of MUSP, mothers were assessed for depressive symptoms at four points between pregnancy and youth age 5 using the Delusions-Symptoms-States Inventory (DSSI; Bedford & Foulds, 1978). A total of 815 mother-child pairs were subsequently selected from the original MUSP sample to participate in a study examining the long-term effects of maternal depression on youth. In line with the goals of the study, this group was oversampled for maternal depression relative to the general population as determined by scores on the DSSI and later confirmed through diagnostic interviews at youth age 15 using the Structured Clinical Interview

for DSM-IV (SCID-I; First, Spitzer, Gibbon, & Williams, 1995). Mean maternal DSSI scores prior to youth age 5 were significantly associated with maternal depression diagnoses at age 15 ($b = 0.03$, $SE = 0.01$, $p < .001$).

Youth and their mothers completed follow-up assessments at youth ages 13, 15, and 20. Of the 815 youth who completed self-report and interview measures at ages 13 and 15, a total of 725 (89.0%) had at least partial data on pubertal status at age 13 and were included in the analyses examining outcomes at age 15 in the present study. The sample is approximately equally split between female ($N = 358$, 49.4%) and male ($N = 367$, 50.6%) participants. Individuals included in the analyses examining outcomes at age 15 are no different than the full sample of 815 youth in terms of gender ($\chi^2(1,814) = 0.01$, $p = .91$) or race/ethnicity ($\chi^2(1,792) = 3.03$, $p = .10$). There was a nonsignificant trend such that participants who were excluded from the analyses examining outcomes at age 15 were more likely to have a history of maternal depression by age 5 than those included in the analyses ($\chi^2(1,810) = 3.60$, $p = .06$).

Of the 725 participants included in the age 15 analyses, 328 females (91.6%) and 304 males (82.8%) were retained at the age 20 follow-up and were included in analyses examining outcomes at age 20. Participants lost to follow-up at age 20 were more likely to be male ($\chi^2(1,724) = 12.51$, $p = .001$). Participants lost to follow-up were no different from those retained at age 20 in race/ethnicity ($\chi^2(1,704) = 0.32$, $p = .62$), maternal depression history by age 5 ($\chi^2(1,720) = 0.02$, $p = .88$), or pubertal timing ($t(723) = 1.10$, $p = .27$).

The majority of participants identified as Caucasian ($N = 666$, 91.9%). The remainder identified as Asian ($N = 23$, 3.2%), Maori/Pacific Islander ($N = 7$, 1.0%), Aboriginal ($N = 6$, 0.8%), and other ($N = 3$, 0.4%). Data on race/ethnicity were not available for 20 participants.

Procedures

At youth age 13, the original MUSP participants completed a battery of interviews and self-report questionnaires, including an assessment of current physical development. The subsample of 815 mother-child pairs completed additional self-report and interview measures at youth age 15. Youth and their mothers completed a second follow-up assessment at youth age 20. Separate interviewers assessed mothers and youth, and all interviewers were blind to maternal depression status. All participants gave consent/assent at each assessment, and all procedures were approved by the Institutional Review Boards of the University of Queensland, Emory University, and the University of California, Los Angeles.

Measures

Pubertal status. Pubertal status was assessed at age 13. Youth were asked to rate their current morphological development by selecting which of five drawings most closely resembled their own bodies. These drawings illustrate stages of physical development as identified by Tanner (1969) and adapted by Morris and Udry (1980). Females rated current breast and pubic hair development while males rated current genital and pubic hair development. Self-ratings of the Tanner stages have been shown to be positively correlated with both physician and parent ratings of morphological development and demonstrate acceptable kappa coefficients (Brooks-Gunn, Warren, Rosso, & Garguilo, 1987; Duke, Litt, & Gross, 1980; Morris & Udry, 1980). Among females in the present sample, the mean self-rated Tanner breast stage was 3.59 (SD = 0.71) with a range of 1 to 5. The mean pubic hair stage for females was 3.81 (SD = 0.67) with scores ranging from 2 to 5. Among males, the mean self-rated Tanner genital stage was 3.34 (SD = 0.86) with a range of 1 to 5, while the mean pubic hair stage was 3.44 (SD = 0.94) with a range of 1 to 5. Table 9 illustrates the distribution of participants' Tanner stages separately by gender.

Pubertal timing. Pubertal timing was calculated by regressing self-rated pubertal status on chronological age separately by gender (Dorn *et al.*, 2006). Separate linear regressions were conducted for each metric of pubertal status (e.g., breast and pubic hair development in girls; genital and pubic hair development in boys). The resulting residuals reflect timing relative to same-sex peers, such that higher scores (e.g., positive residuals) indicate earlier development compared to peers.

Among girls, residuals for breast and pubic hair development were moderately positively correlated ($r = 0.54, p < .001$), while residuals for genital and pubic hair development among boys were highly positively correlated ($r = 0.77, p < .001$). Thus, the two residual scores were averaged for each participant in order to create a single metric of pubertal timing (e.g., Conley & Rudolph, 2009).

Chronic stress. Chronic stressful conditions were assessed using the UCLA Life Stress Interview (LSI; Hammen & Brennan, 2001) conducted at age 15. This semi-structured interview was used to gather detailed information about chronic conditions experienced within the past six months in several functional domains relevant to an adolescent (e.g., social life, family, academics). Interviewers then rated each domain according to the level of stress that would be experienced by a typical person faced with these circumstances. Domains were rated on a 5-point scale using behaviorally-specific anchors such that 1 represented superior circumstances (e.g., no impact or minimal impact of chronic stress on functioning) and 5 represented severely stressful conditions (e.g., significant impact of chronic stress on functioning). The LSI demonstrates high interrater-reliability as well as excellent predictive validity (Hammen & Brennan, 2001; Hammen *et al.*, 1995).

Youth were assessed for levels of chronic stress experienced in the six months prior to the age 15 assessment in order to capture contextual stressors that at least partially overlapped with the pubertal transition. Chronic stress in interpersonal (peer relationships, family relationships, close friendships, romantic relationships) and non-interpersonal domains (academics) were assessed. Descriptive information for all chronic stress domains in Study 2 is provided in Table 10.

Episodic stress. Episodic stress was also assessed using the LSI. Interviewers collected detailed information about the nature, circumstances, timing, and severity of episodic stressors that occurred in the year prior to the age 15 assessment. The interviewer then prepared a written narrative of each event and presented it to an independent rating team whose members were blind to the participant's actual reaction to the event. The team rated each event on objective severity (i.e., how much of an impact the event would have on a typical person under similar conditions) as well as independence (i.e., the degree to which the occurrence of the event was dependent on the actions and influence of the participant). Severity ratings ranged from 1 (no impact) to 5 (extremely severe impact), while independence ratings ranged from 1 (entirely independent of the individual) to 5 (totally dependent on the individual). As described above, the LSI exhibits excellent psychometric properties. The episodic stressors of interest included (a) all stressors coded as independent or fateful and (b) all stressors coded as dependent on the individual. Descriptive statistics regarding the episodic stress variables are provided in Table 10.

Depressive symptoms. Depressive symptoms at youth ages 15 and 20 were assessed using the Beck Depression Inventory-II (BDI-II; Beck *et al.*, 1996), a self-report measure that evaluates the severity of depressive symptoms within the past two weeks. The BDI-II demonstrates high internal consistency and convergent validity in clinical and community

samples of adults (Beck *et al.*, 1996), and its psychometric properties have also been replicated in community samples of adolescents (Osman, Barrios, Gutierrez, Williams, & Bailey, 2008). At age 15, youth in the present sample exhibited a mean BDI score of 6.02 (SD = 6.74) with scores ranging from 0 to 50. At age 20, the mean BDI score was 6.92 (SD = 8.29) with a range of 0 to 52. BDI-II scores in the present sample demonstrated high internal consistency at age 15 (Cronbach's $\alpha = .86$) and acceptable internal consistency at age 20 (Cronbach's $\alpha = .69$).

Statistical Analyses

Hierarchical regression analyses were used to examine three-way interactions between gender, pubertal timing, and contextual stress in the prediction of depressive symptoms in adolescence and young adulthood. Separate analyses were conducted for each contextual stress variable (e.g., chronic peer stress, chronic academic stress, independent episodic stress, dependent episodic stress, etc.), and several sets of analyses were performed. Given that the present sample was oversampled for maternal depression, all analyses controlled for maternal depression history by youth age 5. All analyses also controlled for prior youth depressive symptoms. Analyses examining outcomes at age 15 included participants' scores on the Anxious/Depressed subscale of the Youth Self-Report (YSR; Achenbach, 1991) at age 13 while analyses examining age 20 outcomes included age 15 depressive symptoms based on the BDI-II.

The first set of analyses was designed to explore whether gender moderated the association between pubertal timing and contextual stress in the prediction of depressive symptoms at ages 15 and 20. The first step of each regression included each of the control variables. In order to examine potential curvilinear effects of pubertal timing on depressive symptoms, the second step of each regression included gender, timing, and the contextual stressor of interest, as well as a quadratic timing variable formed by squaring the linear timing

variable. The third step of each regression included all two-way linear and quadratic interaction terms. The final step of the regression included the linear and quadratic three-way interactions between gender, timing, and the contextual stressor of interest.

Following the exploration of gender as a moderator, a second set of hierarchical regression analyses was conducted to examine the interaction of pubertal timing and contextual stress on depressive symptoms at ages 15 and 20 separately by gender. After splitting the dataset by gender, all analyses were conducted as described above in order to examine two-way quadratic and linear interactions between timing and each contextual stressor.

Of note, all analyses first examined potential curvilinear effects. In cases where there was no evidence of a quadratic effect (i.e., the quadratic interaction term was non-significant), subsequent analyses dropped all quadratic variables in order to examine linear interactions between pubertal timing and the contextual stressor of interest.

Due to the large number of tests conducted ($N = 80$), the false discovery rate control procedures outlined by Benjamini and Hochberg (1995) were utilized in order to correct for the potential false discovery of significant results associated with multiple tests. This method is particularly useful when testing scientifically-driven hypotheses and is not associated with many of the limitations accompanying methods designed to control the rate of false positive findings, such as the Bonferroni correction (Glickman, Rao & Schultz, 2014). For the present analyses, the false discovery rate was set to .05, such that at most 5% of the significant findings could be mistakenly concluded to be true positives.

Results

Descriptive statistics for all study variables are located in Table 10, organized by gender.

Chronic Interpersonal Stress

There was a significant three-way interaction between gender, quadratic pubertal timing, and chronic peer stress in the prediction of depressive symptoms at age 15 ($R^2\Delta = .02$, $F\Delta(1,695) = 10.25$, $p < .001$, Figure 15). Table 11 provides a summary of all three-way interaction results. Examining the interaction between quadratic pubertal timing and chronic peer stress separately by gender revealed that among females, this interaction was significant ($R^2\Delta = .04$, $F\Delta(2,342) = 7.93$, $p = .002$), such that girls with a high level of chronic peer stress who also matured off-time relative to peers exhibited the highest levels of depressive symptoms at age 15 (Table 12). The two-way interaction among males was not statistically significant (Table 13).

There was also a significant three-way interaction between gender, pubertal timing, and chronic family stress in the prediction of depressive symptoms at age 15, although this interaction was linear ($R^2\Delta = .02$, $F\Delta(1,699) = 14.04$, $p < .001$, Figure 16). Examining the interaction separately by gender revealed a significant linear interaction among females ($R^2\Delta = .02$, $F\Delta(1,344) = 11.59$, $p = .001$), but not males. Late-maturing females with high levels of chronic family stress exhibited the highest levels of depressive symptoms at age 15, and girls with high levels of family stress had more symptoms of depression overall than those with low family stress.

Gender did not moderate the association between pubertal timing and the other types of chronic interpersonal stress, including stress in a close friendship or romantic relationship, in analyses examining outcomes at age 15 (Table 11). Examining two-way interactions between pubertal timing and chronic interpersonal stressors initially revealed that there was a significant interaction between quadratic timing and chronic romantic stress among females; however, this effect failed to survive the correction for multiple tests. There was no evidence of additional significant two-way interactions between timing and chronic interpersonal stressors in the

prediction of adolescent depressive symptoms among females (Table 12). Among males, pubertal timing did not interact with any category of chronic interpersonal stress to predict depressive symptoms at age 15 (Table 13).

There were no significant findings among analyses examining three-way interactions between gender, pubertal timing, and chronic interpersonal stressors on depressive symptoms at age 20, nor were there any significant two-way interactions between timing and chronic interpersonal stress on outcomes at age 20 among females or males.

Chronic Non-Interpersonal Stress

The interaction between gender, pubertal timing, and chronic academic stress was not significant in the prediction of depressive symptoms at age 15 (Table 11). Additionally, there was no evidence of significant two-way interactions between timing and chronic academic stress on age 15 depressive symptoms among females or males (Tables 12 and 13). Although there was evidence of a three-way linear interaction among gender, pubertal timing, and academic stress on depressive symptoms at age 20, this interaction did not meet the significance threshold established by the false discovery control rate correction for multiple tests. Furthermore, while initial analyses suggested that the two-way linear interaction between pubertal timing and chronic academic stress was significant among females (Table 12), this finding also failed to meet the significance threshold after controlling for multiple tests. Thus, there was no statistically significant evidence of three- or two-way interactions between gender, pubertal timing, and chronic academic stress in the prediction of depressive symptoms at age 20.

Episodic Stressful Events

Examining the associations between gender, pubertal timing, and episodic stressful events revealed several significant findings. Although there was evidence in support of a three-

way linear interaction between gender, pubertal timing, and dependent episodic stress in the prediction of depressive symptoms at age 15 (Table 11), the interaction did not remain significant after controlling for multiple tests. However, there was a significant linear interaction between pubertal timing and dependent episodic stress among males ($R^2\Delta = .02$, $F\Delta(1,353) = 8.86$, $p = .003$). As illustrated in Figure 17, early-maturing boys with high levels of dependent stress demonstrated the highest level of depressive symptoms at age 15. There was no evidence of an interaction between pubertal timing and dependent episodic stress among females (Table 12). Furthermore, the three-way interaction between gender, timing, and independent episodic stress was non-significant, and independent episodic stress did not interact with pubertal timing among males or females to predict depressive symptoms at age 15.

Exploring outcomes at age 20 revealed that gender did not moderate the association between pubertal timing and dependent stress in the prediction of depressive symptoms in young adulthood. While there was evidence of a quadratic interaction between pubertal timing and dependent stress in the prediction of depressive symptoms at age 20 among males (Table 13), the effect was no longer significant following correction for multiple tests. There was no evidence of a three-way interaction between gender, pubertal timing, and independent stress, and pubertal timing did not interact with independent episodic stress to predict depressive symptoms at age 20 among males or females.

Discussion

The present study aimed to extend the existing literature on contextual amplification by exploring various types of stressors as well as identifying gender-specific effects of environmental stressors on the association between off-time pubertal development and depressive symptoms. The results suggest that both chronic interpersonal and dependent episodic

stressors serve as amplifiers of the detrimental psychological effects of off-time development. Furthermore, the findings support the role of gender as a moderator of the contextual amplification effects of chronic peer stress and highlight the unique contextual amplification processes that occur among females versus males, emphasizing the importance of chronic interpersonal stress among off-time females and dependent episodic stressful events among early-maturing males. The present study contributes to an increasingly nuanced understanding of contextual amplification that emphasizes the specific effects of various types of stress on psychopathology depending on concomitant developmental events and characteristics of the individual, including gender and stage of development.

Chronic Interpersonal Stress

Consistent with study hypotheses and prior literature, the present study supports the contextual amplification effects of interpersonal stressors among females who develop off-time and suggests that girls are more vulnerable to the combined effects of off-time development and chronic interpersonal stress than males. However, the results indicate that contextual amplification via chronic interpersonal stressors is not confined solely to early-maturing girls. Girls with high levels of chronic peer stress exhibited the highest levels of adolescent depressive symptoms when they matured earlier *or* later than peers, while girls experiencing high levels of family stress demonstrated the most depressive symptoms at age 15 when they underwent puberty later than others. These results contribute to the growing body of evidence that suggests off-time maturation in either direction can exert deleterious effects on females (Graber *et al.*, 1997; Natsuaki, Biehl, *et al.*, 2009; Rudolph, 2014) and supports co-occurring interpersonal stress as one contributor to the link between pubertal timing and depressive symptoms in girls.

The depressive effects of chronic interpersonal stress among females who matured off-time were no longer evident by young adulthood, as off-time girls who experienced high levels of chronic interpersonal stress in early adolescence did not exhibit significantly higher levels of depressive symptoms than peers at age 20. These findings indicate that girls at high risk for depression who mature early or late may be particularly vulnerable to the effects of interpersonal stress during adolescence, but increases in depressive symptoms associated with contextual amplification processes may be temporary. However, given that adolescent depression tends to continue into or recur in young adulthood (Rao *et al.*, 1999; Lewinsohn *et al.*, 1999) and the evidence that girls at risk for depression by virtue of off-time puberty have a heightened risk of developing proximal depressive symptoms in the context of significant interpersonal stressors, it is likely that this vulnerability portends continued risk for depression in the face of future stress. Of note, female gender and family conflict have been associated with a higher likelihood of depression recurrence in young adulthood (Lewinsohn, Rohde, Seeley, Klein, & Gotlib, 2000), suggesting that contextual amplification associated with interpersonal stress is particularly perilous among females and can establish an enduring risk for depression that could negatively affect well-being across the lifespan.

Contrary to hypotheses, males did not exhibit contextual amplification effects of chronic interpersonal stressors in adolescence or young adulthood, indicating that boys who mature off-time may be resilient to the effects of interpersonal stress despite vulnerability associated with variations in pubertal timing. Males are generally less likely than females to experience depressive symptoms following stress exposure (Hammen, 2005; Nolen-Hoeksema, 2001), and given gender differences in the meaning ascribed to puberty as well as the salience and importance of interpersonal harmony, males may show less reactivity to the stresses associated

with off-time development and co-occurring interpersonal stressors than females (Cyranowski *et al.*, 2000; Rudolph, 2002). Alternatively, males and females may have different ways of coping with challenging circumstances (Shih, Eberhart, Hammen, & Brennan, 2006; Matud, 2004). For example, girls are more likely than boys to seek social support as a means of coping with challenging experiences such as the pubertal transition (Eschenbeck, Kohlmann, & Lohaus, 2007; Hampel & Petermann, 2006; Piko, 2001). Thus, females who undergo puberty off-time and are simultaneously experiencing significant levels of interpersonal stress may not have access to an important coping mechanism, resulting in higher levels of depressive symptoms. Alternatively, given the greater cultural significance ascribed to puberty among females, girls may be more likely than boys to experience interpersonal stress that is directly related to off-time pubertal development (e.g., teasing or comments from peers or family members about pubertal development or lack thereof; pressure from a romantic partner to engage in sexual activity due to perceived physical maturity; Rudolph, 2014; McMaster, Connolly, Pepler, & Craig, 2002; Brooks-Gunn, 1984). Future research in the field of puberty should continue to explore gender differences in the development of psychopathology as well as the mechanisms through which interpersonal stress may impact the psychological outcomes of off-time pubertal development differentially by gender.

Chronic Non-Interpersonal Stress

Following corrections for multiple tests, chronic non-interpersonal stress did not significantly interact with pubertal timing to predict depressive symptoms regardless of gender. While this is contrary to research supporting the role of non-interpersonal stress in the development of depression (Hammen *et al.*, 2009; Bonde, 2008; Mausner-Dorsch & Eaton, 2000), it suggests that non-interpersonal stress in adolescence does not significantly exacerbate

the psychological effects of off-time pubertal development. This finding confirms the initial theory underlying the contextual amplification hypothesis; namely, it suggests that the *context* of pubertal development is critically important. Non-interpersonal stress may simply not be as salient as the interpersonal context in which puberty is embedded, given the social significance of physical maturation and associated changes in social roles. Furthermore, non-interpersonal chronic stress was operationalized in the present study by examining stress related to academics. Future studies should test the contextual amplification effects of non-interpersonal stress beyond academics, such as financial stress within the family. Future research should also seek to clarify whether non-interpersonal stress that co-occurs with off-time pubertal development could promote psychopathology through interpersonal pathways (e.g., poor academic performance might result in a change in class schedule, isolating adolescents from peers; financial strain might lead to interpersonal tension within the family; Morrison Gutman, McLoyd, & Tokoyawa, 2005; Barrera *et al.*, 2002).

Episodic Stressful Events

Contrary to hypotheses, females did not show contextual amplification effects of either independent or dependent episodic stressful life events on depressive symptoms and did not exhibit higher reactivity to dependent stressful events than males. This indicates that acute events, while potentially highly stressful, may not alter the environmental context of puberty among females to a degree that would contribute to psychopathology, whereas chronic interpersonal situations may have added psychological significance, due to their increased duration or broader impact. To date, only two studies outside of the present dissertation have examined the interaction between episodic stressful events and pubertal timing in the prediction of depressive symptoms among girls (Chen *et al.*, 2015; Ge *et al.*, 2001a), concluding that early-

maturing girls with high levels of episodic stress were more likely to be depressed. Given the conflicting findings obtained by the present study, future research examining contextual amplification effects of episodic stressful events among females is greatly needed. Furthermore, this research should continue to explore the relevance of both independent and dependent stressful events. Ge and colleagues (2001a) examined only those stressors categorized as “uncontrollable”, a designation that is roughly equivalent to independent events. Given the differential relevance of independent and dependent events to the maintenance of depression over time (Hammen, 1991), research examining contextual amplification processes should certainly seek to explore the unique effects of these stressors on psychological outcomes of puberty.

As predicted, dependent episodic stress emerged as a contextual amplifier of pubertal timing in the prediction of depressive symptoms among males at age 15. Early-maturing boys who experienced high levels of dependent stress demonstrated the highest levels of depressive symptoms in adolescence. Given that dependent stressors are directly related to the actions of the individual, boys may interpret dependent events as particularly stressful because they contributed to the occurrence of these stressors. Consistent with the stage termination hypothesis (Petersen & Taylor, 1980), boys who have matured physically at a young age may lack the necessary skills to interact effectively with their environment, leading them to feel distressed by the negative outcomes of their ineffectual behavior. Future research should seek to elucidate what types of dependent events are associated with early maturation among males in order to clarify how pubertal timing interacts with episodic stressors to promote psychopathology (e.g., risky behaviors, events related to poorly-developed interpersonal skills, etc.) as well as the mechanisms through which contextual amplification via dependent stress leads to heightened

depressive symptoms. Of note, the interaction between timing and dependent episodic stress among males in the prediction of age 20 depressive symptoms was initially significant, but did not survive correction for multiple tests. Thus, future research should also continue to explore whether contextual amplification via dependent stressors may vary based on stage of development.

More generally, future studies should explore the mechanisms through which the interaction of off-time pubertal development and contextual stress lead to heightened depressive symptoms, including whether these mechanisms differ based on the nature of the contextual stressor (e.g., chronic versus episodic, interpersonal versus non-interpersonal) or demographic characteristics such as gender or developmental stage (e.g., adolescence versus young adulthood). Consistent with the maturation disparity hypothesis (Petersen & Taylor, 1980), perhaps adolescents who develop off-time and experience high levels of concurrent environmental stress feel especially abnormal or isolated from peers, leading to the development of psychopathology. Psychological outcomes such as negative self-related cognitions or emotions such as guilt and shame may be particularly likely when the adolescent has actively contributed to the stressor, consistent with the findings among early-maturing males experiencing high levels of dependent episodic stress. Alternatively, perhaps co-occurring stress limits the ability of adolescents to seek valued forms of social support (Malecki & Demaray, 2003; Eschenbeck *et al.*, 2007; Hampel & Petermann, 2006) or engage in other productive coping mechanisms that would blunt the psychological impact of stress associated with off-time development. Additionally, given the association between stressful environments in early childhood and variations in pubertal timing (Ellis & Essex, 2007; Chisholm *et al.*, 2005; Ellis & Garber, 2000), individuals who develop off-time may be particularly vulnerable to the influence

of contextual stressors as they may not have learned adequate skills to cope effectively with stressful experiences and may have experienced significant environmental stress for much of their lives. Although there is a significant body of literature suggesting that early childhood stress predicts variations in pubertal timing, these studies are often conducted separately from those examining interactions between timing and later stressful experiences. Research that bridges the gap between these two areas of the literature could assist considerably in clarifying the association between pubertal timing and life stress. As the field progresses toward greater understanding of the myriad biological and psychosocial processes that comprise the pubertal transition, future studies should move toward a more nuanced understanding of the various mechanisms that contribute to the development of psychopathology among individuals who mature off-time.

Strengths and Limitations

The present study has a number of strengths, including the extension of the contextual amplification literature to examine the role of non-interpersonal stressors and episodic stressful events as well as the exploration of curvilinear effects of pubertal timing. The use of a large, mixed-gender sample followed longitudinally helps to elucidate the nuances of contextual amplification processes, including gender differences in contextual amplification, and contributes to a growing body of literature examining pubertal processes in males. Measurement of contextual stressors was highly detailed, did not rely upon self-report, and utilized an objective rating of stressor severity. Furthermore, the assessment of pubertal status was obtained contemporaneously and therefore not subject to retrospective reporting bias.

Advantages of the study should be considered alongside several limitations. The use of a single measure of puberty assessed at a relatively late timepoint is a notable drawback. Although

common in the contextual amplification literature (e.g., Benoit *et al.*, 2013; Teunissen *et al.*, 2011; Mrug *et al.*, 2014; Obeidallah *et al.*, 2004), a single measure of puberty necessarily fails to capture the dynamic processes inherent in a complex developmental process, and relatively late assessment may be unable to account for the unique experiences of early-maturing individuals. The field of puberty research should continue to move toward the adoption of measurement techniques that reflect the fluid nature of pubertal development, including the use of repeated assessments of development from late childhood through adolescence. Additionally, the present study utilized self-ratings of the Tanner stages to measure pubertal development. Although self-ratings have shown high correlations with physician and parent ratings (Brooks-Gunn *et al.*, 1987; Morris & Udry, 1980), some have raised doubts about their reliability and accuracy (Dorn *et al.*, 2006; Dorn, Susman, & Ponirakis, 2003). Furthermore, adiposity and body mass index have been associated with pubertal timing (Biro *et al.*, 2003, Kaplowitz *et al.*, 2001), but were not measured in the present sample. Thus, analyses were not able to control for the effects of these variables. Finally, the use of a sample that is primarily Caucasian and oversampled for maternal depression is not ideal, and future research on pubertal processes should seek to employ more inclusive and representative samples.

Conclusions

The present study highlights the complexity of contextual amplification processes and serves to extend the existing literature in several ways, including the exploration of stressors other than chronic interpersonal stress, the examination of curvilinear effects of pubertal timing, the focus on outcomes into young adulthood, and the exploration of gender differences in contextual amplification. Moving forward, future research should continue to refine our understanding of contextual effects of stress on pubertal processes by examining precise

categories of stressors, exploring the personal or demographic characteristics of individuals who are especially reactive to the amplifying effects of various types of stress, and seeking to elucidate the mechanisms through which contextual amplification contributes to the development of psychopathology. Increased understanding of the importance of the psychosocial context in which puberty occurs can only hasten the development of effective prevention and treatment efforts targeting youth at risk for depression and help to ameliorate the devastating effects of depression during adolescence and adulthood.

**Chapter 4: Pubertal Synchrony in the Prediction of Depressive Symptoms
among Females (Study 3)**

Abstract

Puberty is accompanied by numerous psychological and interpersonal challenges, including a dramatic rise in the prevalence of depression among girls. Pubertal timing has been identified as a potent predictor of depressive symptoms among females, but less is known about the impact of pubertal characteristics other than timing on psychopathology. The present study sought to address this gap in the literature by examining the effect of pubertal synchrony, the degree to which morphological indicators of puberty develop concurrently, on depressive symptoms in adolescence and young adulthood in a longitudinal sample. Among 355 female participants, asynchronous development at age 13 was associated with increased depressive symptoms at age 20, but not age 15. Additional analyses indicated that pubertal timing moderated the association between synchrony and depressive symptoms at age 20, such that girls who exhibited asynchronous development had the highest levels of depressive symptoms when they matured later than peers. Results provide initial empirical support for the role of pubertal synchrony in the development of depression among females and are discussed with regard to the biopsychosocial processes that may connect features of puberty with the long-term development of psychopathology.

Introduction

Study 3 aims to extend the research on puberty and psychopathology beyond explorations of timing by examining the association between pubertal synchrony and the development of depressive symptoms. As described previously, pubertal synchrony, the degree to which different indicators of puberty develop concurrently, has long been hypothesized to affect psychological well-being (Eichorn, 1975; Brooks-Gunn & Warren, 1985; Mendle, 2014), but has never been studied with regard to the development of psychopathology. Instead, as reviewed above, research to date has examined synchrony solely in relation to physical characteristics. The lack of studies exploring psychological implications of synchronous versus asynchronous pubertal development is particularly concerning given strong associations between pubertal timing and psychopathology, including depressive symptoms (Graber, 2013), as well as clear parallels between psychosocial explanations for timing's effects and the psychological and social processes that may be expected to result from variations in synchrony.

Furthermore, features of puberty including synchrony, timing, and tempo are necessarily “intertwined” (Mendle, 2014). The dynamic nature of pubertal development means that one adolescent can experience early maturation that progresses at a relatively slow tempo, while another initiates puberty on-time but exhibits a rapid tempo, such that their development eventually appears uniform. Similarly, individuals might display varying stages of morphological maturation at one age, but exhibit synchronous development at other points in adolescence, depending on the timing and tempo of maturation across each morphological feature. Thus, it has been hypothesized (Mendle, 2014) that pubertal characteristics may interact with one another to affect risk for psychopathology. For example, early timing may be perceived by girls as particularly deviant when development is also highly synchronous, such that multiple physical

markers are maturing early. Alternatively, asynchronous development may be interpreted as especially deviant when it also occurs off-time relative to peers. The interactive nature of pubertal characteristics has been supported by research suggesting that the duration of puberty is affected by the timing of pubertal onset (Marti-Henneberg & Vizmanos, 1997). However, very little research has examined interactive effects of pubertal characteristics in the prediction of psychopathology, although as described above one study found evidence for an interaction between timing and pubertal tempo in the prediction of externalizing behavior among boys (Marceau *et al.*, 2011).

The present study seeks to address marked gaps in the literature by examining the effect of pubertal synchrony on the development of depressive symptoms among females in a longitudinal sample. Additional analyses will explore potential interactive effects of synchrony and timing. The study is restricted to females due to methodological considerations associated with the sample. Consistent with the majority of previous research on synchrony in females, the parent study from which the present data are drawn assessed physical maturation according to Tanner stage ratings of breast and pubic hair development. However, among boys, genital development (e.g., penis size) and pubic hair development were examined. Unfortunately, these metrics are highly positively correlated ($r = 0.77$ in the present sample) and do not address the most important component of male morphological maturation: testicular development. Therefore, the present study examines pubertal synchrony solely among females.

Given that adolescent depression tends to persist or even worsen into young adulthood (Lewinsohn *et al.*, 1999; Rao *et al.*, 1999; Schulenberg & Zarrett, 2006) as well as previous findings suggesting that pubertal timing is associated with psychopathology beyond adolescence (Copeland *et al.*, 2010; Graber *et al.*, 2004), the analyses examine depressive symptoms at two

distinct points in adolescence and young adulthood. Depression was selected as the outcome of interest due to the strong association between other characteristics of the pubertal transition (e.g., timing) and increases in depressive symptoms among adolescent females (Cyranowski *et al.*, 2000; Graber, 2013; Mendle *et al.*, 2007). Additionally, in light of the interpersonal challenges that accompany puberty (Graber, 2013; Smetana, Campione-Barr, & Metzger, 2006) as well as the established link between interpersonal difficulties and depression among females (Hammen *et al.*, 2004; Hammen, 2003), depressive symptoms are theorized to be particularly germane to the study of pubertal synchrony among girls.

Aims and Hypotheses

Aim 1. To explore the influence of synchronous versus asynchronous development on depressive symptoms in adolescence and young adulthood among females.

1. Due to the lack of previous research on the association between pubertal synchrony and depression, study hypotheses were generated in accordance with theory. Mendle (2014) has offered theoretical explanations for an association between synchrony and psychopathology in both directions: (a) synchronous development may lead girls to feel more psychologically secure because they are experiencing all aspects of puberty simultaneously, suggesting an association between *pubertal asynchrony* and psychopathology, or (b) undergoing multiple changes simultaneously may lead synchronously developing girls to feel overwhelmed, suggesting an association between *synchronous pubertal development* and psychopathology. Thus, it is hypothesized that individuals will show varying levels of depressive symptoms in response to synchronous versus asynchronous development, but no predictions are made regarding the direction of the hypothesized effect.

Aim 2. To examine the interaction between pubertal synchrony and pubertal timing in the prediction of depressive symptoms.

1. Consistent with previous research demonstrating higher risk for depressive symptoms among girls exhibiting early timing (Graber, 2013; Mendle *et al.*, 2007), it is predicted that timing will moderate the hypothesized association between pubertal synchrony and depression, such that the effect of synchrony on depressive symptoms will be most deleterious among early-maturing girls.

Method

Participants

The present analyses include female participants from the MUSP subsample of 815 mother-child pairs described above. Of the 815 youth who participated in the assessments at ages 13 and 15, a total of 403 were female. Of these, 355 girls (88.1% of the female sample) had complete data on pubertal status and were included in analyses examining age 15 outcomes. Participants included in the age 15 analyses showed no differences in race/ethnicity ($\chi^2(1,394) = 0.19, p = .72$), maternal education ($\chi^2(1,399) = 0.01, p = .99$), maternal depression history by age 5 ($\chi^2(1,399) = 0.48, p = .50$), or maternal depression history by age 15 ($\chi^2(1,401) = 0.31, p = .64$) compared to the full sample of 403 girls. Of note, three female participants included in Study 2 did not have data on pubic hair development and were excluded from the present study because calculations of synchrony require complete data on both breast and pubic hair development.

A total of 326 female participants were retained at the follow-up assessment and included in analyses examining outcomes at age 20. The 29 participants lost to follow-up by the age 20 analyses were no different from those retained in the study at age 20 in race/ethnicity ($\chi^2(1,347) = 0.09, p = .99$), maternal education ($\chi^2(1,352) = 0.79, p = .60$), maternal depression history by

age 5 ($\chi^2(1,352) = 1.49, p = .25$), maternal depression history by age 15 ($\chi^2(1,354) = 0.84, p = .44$), pubertal synchrony ($\chi^2(2,354) = 3.14, p = .21$), or pubertal timing ($\chi^2(2,354) = 1.28, p = .53$).

The sample was primarily lower middle class, and the majority of participants' mothers had completed some high school ($N = 341, 96.1\%$).

Most female youth identified as Caucasian ($N = 331, 93.2\%$), while a minority of participants identified as Asian ($N = 12, 3.4\%$), Maori/Pacific Islander ($N = 2, 0.6\%$), Aboriginal ($N = 2, 0.6\%$), and other ($N = 1, 0.3\%$). Data on race/ethnicity were not available for seven participants. Race (Caucasian versus minority) was not associated with synchrony ($\chi^2(2,346) = 2.24, p = .33$) or timing ($\chi^2(2,346) = 1.88, p = .39$). Given previous research indicating that Asian females are more likely than Caucasian females to exhibit more advanced breast development (Novotny *et al.*, 2011), all analyses were repeated excluding non-Caucasian participants. There were no changes in the direction, significance, or effect sizes of the obtained results when excluding non-Caucasian participants.

Measures

Pubertal status. As described in Study 2, current pubertal status was assessed at youth age 13 by asking youth to rate their level of morphological development according to the Tanner scales. In the present sample, the mean self-rated Tanner breast stage at age 13 was 3.59 (SD = 0.71) with a range of 1 to 5. The mean self-rated Tanner pubic hair stage was 3.81 (SD = 0.67) with scores ranging from 2 to 5. Table 9 summarizes the distribution of pubertal status at age 13.

Pubertal synchrony. Consistent with prior research (Novotny *et al.*, 2011), pubertal synchrony was ascertained by calculating the difference between participants' breast and pubic hair Tanner stage ratings at the age 13 assessment. Participants were assigned to one of three

categories of synchrony. Those with a difference score of zero were classified as exhibiting synchronous development ($N = 227$, 63.9%). Girls whose pubic hair Tanner stage rating was greater than their breast Tanner stage rating were classified as exhibiting signs of *pubarche* ($N = 97$, 27.3%), while those whose breast development was more advanced than their pubic hair development were classified as exhibiting evidence of *thelarche* ($N = 31$, 8.7%). This method of classifying synchrony is commensurate with prior research (Novotny *et al.*, 2011).

Pubertal timing. As described in Study 2, timing was calculated by regressing pubertal status on chronological age and averaging the residual scores for breast and pubic hair development to create a single timing score for each participant.

Participants were then assigned to one of three categories according to their averaged residual score. A categorical timing variable was used in order to simplify the comparison of early-maturing, on-time, and late-maturing participants, without necessitating the use of a continuous quadratic variable. Commensurate with prior research (Dorn *et al.*, 2006; Stroud & Davila, 2008), participants were assigned to categories using a cutoff of ± 0.67 standard deviations from the mean in order to assign approximately 20% of the sample to each of the outlying groups. Girls above 0.67 standard deviations from the mean were classified as early-maturing ($N = 71$, 20.0%), while girls below -0.67 standard deviations from the mean were classified as late-maturing ($N = 84$, 23.7%). All other participants were classified as maturing on-time relative to peers ($N = 200$, 56.3%).

Depressive symptoms. Depressive symptoms were measured using the BDI-II as described in Study 2. At age 15, the mean BDI-II score in the present sample was 6.97 ($SD = 7.05$) with a range of 0 to 44. A total of 55 participants (15.5%) met criteria for at least mild depression at age 15, with BDI-II scores of 14 or above. At age 20, the mean BDI-II score was

8.05 (SD = 9.18) with scores ranging from 0 to 52. A total of 55 participants (16.9%) met criteria for at least mild depression at age 20. BDI-II scores in the present sample demonstrated high internal consistency at age 15 (Cronbach's $\alpha = .87$) and acceptable internal consistency at age 20 (Cronbach's $\alpha = .70$).

Statistical Analyses

Univariate analyses of covariance (ANCOVA) were employed to examine hypothesized differences between categories of pubertal synchrony in the prevalence of youth depressive symptoms at ages 15 and 20 as well as the hypothesized interaction between synchrony and timing. Synchrony was a categorical variable comprised of three categories: synchronous development, pubarche, and thelarche. Timing was also comprised of three categories: early-maturing, on-time, and late-maturing.

Given that the present sample was selected from the larger MUSP study by oversampling for maternal depression, all analyses included maternal depressive symptoms at youth age 5 as well as prior youth depressive symptoms as covariates as described in Study 2. All analyses also incorporated maternal education status as a covariate, due to the association between social disadvantage and pubertal timing (Ellis & Essex, 2007; Obeidallah, Brennan, Brooks-Gunn, Kindlon, & Earls, 2000). All analyses were adjusted for the observed mean of all covariates.

Results

Descriptive Statistics

Descriptive statistics for all study variables are presented in Table 14, stratified by pubertal synchrony and pubertal timing.

Pubertal Synchrony

Synchrony was not significantly associated with depressive symptoms at youth age 15 ($F(2,342) = 0.67, p = .51, \text{partial } \eta^2 = 0.00$). However, synchrony significantly predicted depressive symptoms at youth age 20 ($F(2,290) = 3.90, p = .02, \text{partial } \eta^2 = .03$). Girls with synchronous development at age 13 exhibited significantly fewer depressive symptoms at age 20 than girls demonstrating signs of either thelarche ($p = .03$) or pubarche ($p = .03$). The association between synchrony and age 20 depressive symptoms is illustrated in Figure 18.

Interactive Effects of Pubertal Synchrony and Pubertal Timing

The next set of analyses examined the interaction between synchrony and timing in the prediction of youth depressive symptoms. At age 15, the main effect of synchrony on depressive symptoms was not significant ($F(2,336) = 1.94, p = .15, \text{partial } \eta^2 = .01$). However, there was a main effect of timing on age 15 depressive symptoms ($F(2,336) = 4.29, p = .02, \text{partial } \eta^2 = .03$), such that late-maturing girls exhibited significantly higher levels of symptoms than on-time girls ($p = .01$). There were no significant differences between girls who matured on-time and those who developed earlier than peers. The interaction of synchrony and timing did not significantly predict depressive symptoms at age 15 ($F(4,336) = 1.31, p = .27, \text{partial } \eta^2 = .02$).

At age 20, there were significant main effects of synchrony ($F(2,284) = 8.02, p < .001, \text{partial } \eta^2 = .05$) and timing ($F(2,284) = 4.52, p = .01, \text{partial } \eta^2 = .03$) on depressive symptoms. Consistent with the findings reported above, girls with synchronous development exhibited significantly lower depressive symptoms at age 20 than girls showing signs of thelarche ($p = .01$) or pubarche ($p = .001$). Additionally, girls who developed on-time had significantly lower age 20 depressive symptoms than late-maturing girls ($p = .004$).

Examining the interaction between synchrony and timing revealed that timing moderated the association between synchrony and age 20 depressive symptoms ($F(4,284) = 2.66, p = .03$,

partial $\eta^2 = .04$), such that girls who exhibited asynchronous development had the highest levels of depressive symptoms when they matured later than peers. Late-maturing girls showing signs of either pubarche ($p = .01$) or thelarche ($p = .01$) exhibited significantly higher levels of depressive symptoms than those with synchronous development (Figure 19).

Of note, all analyses were repeated using a dichotomous synchrony variable in which girls exhibiting signs of thelarche and pubarche were collapsed into a single category in order to examine the general impact of asynchronous development on depression. The direction, significance, and effect sizes of all results at ages 15 and 20 were essentially unchanged.

Discussion

Study 3 sought to expand the literature on puberty and psychopathology by examining the role of pubertal synchrony in the development of depressive symptoms among females. Results suggest that asynchronous morphological development is associated with increased levels of depressive symptoms in young adulthood, but this association is not yet apparent in adolescence. Additional analyses found that pubertal timing moderated the association between synchrony and depressive symptoms in young adulthood, such that asynchronous development resulted in the highest levels of depressive symptoms among late-maturing girls. These findings represent the first empirical evidence of an association between asynchronous morphological development and psychopathology and offer preliminary support for an interactive, dynamic relationship between synchrony and timing in the prediction of depression among females.

Contrary to expectations, the effect of asynchronous development on depressive symptoms was not apparent until young adulthood. This suggests that the psychological effects of asynchronous development may emerge over time, so that by age 20 there is statistically significant evidence of heightened depression. This finding certainly does not exclude the

likelihood that asynchronous development has deleterious psychological effects on adolescents, but simply suggests that these effects become evident statistically by age 20. While young adulthood is known as a challenging transitional period (Arnett, 2014), many concerns associated with this stage are similar to those of adolescence, including body image, self-esteem, and peer and romantic relationships. Thus, it may be that the impact of asynchronous development on these concerns has reached a threshold of psychological significance by age 20, particularly as emerging adults separate from their families of origin (Markiewicz, Lawford, Boyle, & Haggart, 2006). Future studies examining the effect of asynchronous pubertal development on psychopathology in adolescence and young adulthood are greatly needed, both to replicate the current results and to provide additional insight into the emergence of clinically significant depressive symptoms by age 20.

In the present sample, asynchronous development resulted in the highest levels of depressive symptoms among late-maturing girls, contrary to the majority of research supporting an association between early timing and depression (Graber, 2013; Mendle *et al.*, 2007). This suggests that off-time development may be pernicious in either direction, a conclusion supported by previous research that has identified curvilinear associations between timing and depression (Conley & Rudolph, 2009; Graber *et al.*, 1997; Natsuaki *et al.*, 2009). Particularly when combined with asynchronous morphological development, late maturation may provoke concerns about abnormality and “deviance” from peers that could ultimately lead to heightened risk for depression.

While the present findings corroborate the extensive literature documenting the effect of timing on depressive symptoms and provide initial evidence that synchrony also influences mental health, additional research is needed to elucidate the psychological, interpersonal, and

biological mechanisms through which puberty affects psychopathology (Rudolph, 2014). In line with cognitive theories of depression (Beck, 1987), girls who mature asynchronously may develop the belief that puberty is an inconsistent, unpredictable process, while synchronous maturation may reinforce the belief that one is progressing appropriately toward developmental milestones. Consistent with the maturation disparity hypothesis, the detrimental effects of asynchronous development may be attributable to perceptions of deviance from peers or the belief that one is developing abnormally. Additionally, given the strong relevance of interpersonal stress to depression in females (Hammen, 2003), the interpersonal significance of asynchronous development should not be overlooked. Signs of thelarche may be associated with particular interpersonal challenges (Brooks-Gunn, 1984), such that girls may receive romantic attention before they have attained the psychological and interpersonal resources to cope with this experience (e.g., Petersen & Taylor, 1980; Stattin & Magnusson, 1990). Furthermore, consistent with the contextual amplification and personal accentuation hypotheses (Caspi, Lynam, Moffitt, & Silva, 1993; Caspi & Moffitt, 1991; Ge & Natsuaki, 2009), difficult environmental contexts and pre-existing personal vulnerabilities may aggravate the stresses of asynchronous maturation, leading to increased depressive symptoms. For example, girls with problematic peer relationships or low levels of emotional stability may cope with the stresses of asynchronous development in a particularly ineffective manner, resulting in higher levels of depression.

As discussed above, biological processes, including hormonal shifts, may also influence the association between synchrony and depressive symptoms (Angold *et al.*, 1999; Rudolph, 2014). Studies utilizing a biopsychosocial perspective are critically needed in order to understand the numerous contributing factors that link pubertal characteristics, including synchrony, with

depression. Additionally, by elucidating the mechanisms linking synchrony and timing to psychopathology, parents, educators, and medical professionals will be outfitted with the knowledge to develop preventative interventions designed to limit the deleterious psychological impact of asynchronous and off-time development on girls' emotional and social functioning.

Strengths and Limitations

Strengths of the present study include the use of a moderately large sample assessed at two points in adolescence and young adulthood as well as the ability to statistically control for prior depressive symptoms when predicting later symptoms. Retrospective reporting bias was avoided by measuring constructs concurrently, including pubertal status. Additionally, to the author's knowledge, this is the first study to explore associations between synchrony and psychopathology, addressing a notable gap in the literature on the psychological effects of puberty among young women and extending the limited prior research on features of puberty other than timing (Mendle *et al.*, 2010; Mendle *et al.*, 2012).

As discussed with regard to Study 2, the primary limitation of the present study is the use of a single, relatively late assessment of pubertal status. While the use of a single measurement of pubertal development is consistent with much of the research on puberty and psychopathology (e.g., Rudolph & Troop-Gordon, 2010; Stroud & Davila, 2008; Teunissen *et al.*, 2011), puberty is a highly dynamic process and the degree of synchrony among different markers of development may shift over time due to variations in timing and tempo (Susman *et al.*, 2010). The present study provides a snapshot of the psychological effects of synchrony at one point in development, but may fail to capture the broader effects of synchronous versus asynchronous maturation across the pubertal transition. Assessing pubertal status at age 13 is not ideal given the normative age of onset and duration of puberty among girls. Furthermore, later assessments

prevent comprehensive understanding of pubertal timing by failing to capture the interplay between timing and tempo (i.e., girls identified as late-maturing based on pubertal status at age 13 may have matured later than peers or may simply have progressed through puberty more slowly than others). In the future, the field of puberty research should move toward employing repeated measurements of pubertal status from late childhood through adolescence in order to capture the dynamic nature of pubertal development.

In addition, as discussed with regard to Study 2, self-ratings of the Tanner stages may not be the ideal measure of pubertal status. Furthermore, the use of a sample that was primarily Caucasian, limited to females, and oversampled for maternal depression is not optimal, and future studies should aim to explore associations between synchrony and psychopathology in more inclusive and representative samples. Finally, given the association between adiposity and pubertal development (Biro *et al.*, 2003; Kaplowitz *et al.*, 2001), the inability to control for body mass index or adipose tissue in the present analyses is not ideal, and future studies of puberty should aim to measure and control for adiposity.

Conclusions

The present study offers the first empirical evidence of the contribution of pubertal synchrony to the development of psychopathology, demonstrating that asynchronous pubertal development is associated with increased depressive symptoms in young adulthood, but not adolescence. This effect was particularly pronounced among girls who matured later than peers. Much additional research is needed, both to corroborate the present findings as well as to elucidate the complex interplay of psychological, interpersonal, and biological processes that contribute to the higher prevalence of depression among females beginning in adolescence. Precise, repeated measurements of pubertal development encompassing both morphological and

hormonal indicators will further advance the field and allow for greater understanding of the dynamic, interactive processes that comprise puberty. The present study represents a valuable step toward the expansion of research beyond the current focus on timing (Mendle, 2014). Additional studies exploring the role of synchrony in the development of psychopathology as well as the interactive effects of various characteristics of puberty will continue to further our understanding of the psychological correlates and consequences of the pubertal transition.

Chapter 5: General Discussion

The present dissertation sought to clarify several important issues regarding the relationship between puberty and the development of depression.

Mechanisms of the Association between Puberty and Depressive Symptoms

Studies 1 and 2 tested four major psychosocial hypotheses that attempt to explain the association between pubertal timing and depression. The *stage termination* hypothesis suggests that early-maturing youth are unable to complete the developmental tasks of childhood, leaving them unable to effectively manage the transition to adolescence (Petersen & Taylor, 1980; Stattin & Magnusson, 1990; Caspi & Moffitt, 1991). Study 1 found evidence in support of stage termination among females, such that early-maturing girls exhibited reduced social competence that was linked to greater depressive symptomatology in grades 9 and 12. This is consistent with two existing studies of stage termination, which also supported the stage termination hypothesis among girls by concluding that early timing was associated with higher levels of negative self-focus and anxious arousal, reduced use of engagement coping, and more interpersonal stress, all of which predicted subsequent depressive symptoms (Rudolph *et al.*, 2014; Conley *et al.*, 2012). These findings suggest that early development among girls appears to contribute to specific skill deficits, including lower social competence and reduced engagement coping, that are tied to subsequent increases in depression. Intervention should thus focus on building interpersonal and stress management skills among girls who have matured early or are at risk of early maturation due to genetic predisposition or childhood experiences (Perry, *et al.*, 2014; Ellis & Essex, 2007; Biro *et al.*, 2003). Of note, other characteristics hypothesized to account for the relationship between early timing and depressive symptoms, including coping, perceived stress, and self-image did not show evidence of stage termination among females in the present dissertation.

While one previous study found evidence for stage termination among males (Rudolph *et al.*, 2014), the results of the current dissertation suggest that stage termination is only relevant to females. This is consistent with the majority of existing literature that emphasizes the deleterious effects of early timing among girls only (Graber, 2013).

The *maturational disparity* hypothesis proposes that off-time development in either direction can lead to psychopathology due to increased feelings of isolation and abnormality among youth who mature earlier or later than peers (Petersen & Taylor, 1980; Caspi & Moffitt, 1991). Study 1 found no evidence of maturational disparity among females or males, suggesting that among adolescents at low-risk for depression, perceived differences from peers do not account for the link between timing and depressive symptoms. However, maturational disparity may be one explanation for the results of Study 3, in which girls who exhibited asynchronous morphological development had the highest levels of depressive symptoms in young adulthood, particularly when they matured later than peers. The intuitive explanation offered by the maturational disparity hypothesis is in line with existing research on the importance of peer acceptance among adolescents, particularly in relation to psychological well-being and risk for psychopathology (Rose & Rudolph, 2006; Demir & Urberg, 2004; Ladd & Troop-Gordon, 2003). Thus, although Study 1 did not support maturational disparity, future studies should continue to explore the relevance of this hypothesis to links between off-time development and psychopathology, particularly given that the present findings represent the first empirical test of maturational disparity with regard to pubertal timing. Additionally, future research should aim to identify the mechanisms by which variations in pubertal characteristics other than timing (e.g., synchrony, tempo) lead to depressive symptoms, including tests of maturational disparity.

The *personal accentuation* hypothesis highlights the role of personal characteristics of the adolescent in the association between timing and psychopathology, such that individuals who mature off-time relative to peers are expected to exhibit higher levels of psychopathology when they also possess negative personality traits or limited psychological resources (Caspi & Moffitt, 1991; Ge & Natsuaki, 2009). Study 1 offered limited evidence in support of personal accentuation among females. Trait levels of emotional stability interacted with pubertal timing to predict depressive symptoms during adolescence, although the observed association was not in the direction that would be predicted by personal accentuation. However, Study 1 also found that conscientiousness and openness to experience interact with timing to predict the trajectory of depressive symptoms among females into young adulthood, offering the first evidence of personal accentuation in the prediction of trajectories of symptoms over time. Extraversion and agreeableness did not interact with timing to predict symptoms among females, and there was no evidence of personal accentuation among males. As discussed above, these results must be interpreted alongside the strong independent effects that personality traits exerted on depressive symptoms and their trajectory in Study 1, regardless of gender. Thus, while the current findings provide some support for personal accentuation among females, they also suggest that the combined influence of timing and personality features may be less important among adolescents at low risk for depression.

Finally, Studies 1 and 2 explored the *contextual amplification* hypothesis, which proposes that co-occurring environmental stressors can intensify the negative psychological effects of off-time development. The results of Study 1 indicate that family and neighborhood stress can influence the trajectory of depressive symptoms into young adulthood among females, such that girls experiencing high levels of paternal conflict and neighborhood stress exhibit the most

symptoms over time, while early-maturing girls in low stress environments experience increasing levels of depression across adolescence. Parental depressive symptoms, stressful life events, and conflict with mothers did not interact with timing among females. Additionally, there was no evidence of contextual amplification among males in Study 1.

Consistent with the Study 1 results regarding paternal conflict among females, Study 2 confirmed that females with high levels of family stress are likely to exhibit the highest levels of depressive symptoms based on variations in pubertal timing; however, this study suggested that late-maturing girls with high chronic family stress show the most adolescent depression. Thus, in combination with the results of Study 1, the findings indicate that girls who mature off-time in either direction may be at heightened risk for depression when experiencing high levels of family stress. In addition, the results of Study 2 confirm that chronic interpersonal stress related to peers serves as a contextual amplifier of off-time development among females, with both early- and late-maturing girls showing high levels of adolescent depressive symptoms when they also experienced high levels of chronic peer stress.

Among males, Study 2 found that early-maturing boys with high levels of dependent stressful life events exhibit the highest levels of depressive symptoms in adolescence. In contrast, Study 1 did not support stressful life events as contextual amplifiers among males or females, although there was evidence that stressful events exerted a strong effect on adolescent depressive symptoms independent of pubertal timing. These divergent results may reflect the different study samples (low-risk adolescents recruited from the community versus high-risk youth oversampled for maternal depression) as well as the different measures of stressful events (self-report checklist versus semi-structured interview). Given the biases associated with life event checklists (Hammen, 2005), Study 1 may not have effectively captured participants' experience of episodic

stressful events. Additionally, Study 1 did not differentiate between types of episodic stress whereas Study 2 observed contextual amplification effects via dependent stressors only, suggesting that distinctions between independent and dependent events may be relevant to contextual amplification processes. Few existing studies have tested the contextual amplification effects of episodic stressors (Conley & Rudolph, 2009; Ge *et al.*, 2001a; Ge *et al.*, 2001b; Chen *et al.*, 2015), and the nuances of contextual amplification via episodic stressful events have yet to be fully elucidated. The results of the present dissertation suggest that these processes are complex and should continue to be explored in both community and high-risk samples using valid and reliable measures of stress exposure.

Together, the results of Studies 1 and 2 support contextual amplification among both females and males, with a particular emphasis on the deleterious effects of chronic interpersonal conditions among females. Females also showed contextual amplification effects related to neighborhood stress, as indexed by community violence. Consistent with prior findings regarding the association between interpersonal context and depression among women (Hammen, 2003), adolescent females appear to be especially sensitive to the social environment, particularly when early puberty has conferred vulnerability to environmental stressors. Among males, stressful events dependent in some way on the adolescent's own actions were identified as a contextual amplifier of early timing. Thus, in line with prior research (Winer *et al.*, 2016; Rudolph & Troop-Gordon, 2010; Conley & Rudolph, 2009; Ge *et al.*, 2001b), the present findings indicate that both chronic and episodic stressors serve to amplify the effects of off-time pubertal development and provide further evidence for the particular impact of specific categories of stress based on gender. Given the breadth of contextual amplification findings to date, including the results of the present dissertation, future research should continue to explore

what types of stress most strongly affect specific groups of adolescents in order to better understand how concurrent environmental stressors can alter the psychological impact of puberty in the short-term as well as into adulthood.

In summary, the results of the dissertation indicate that variations in pubertal timing are associated with depressive symptoms among females via several mechanisms, including stage termination, personal accentuation, and contextual amplification of chronic interpersonal and neighborhood stress. Among males, contextual amplification of dependent episodic stress predicted increased depressive symptoms in adolescence. However, no other mechanisms of the association between pubertal timing and depression were observed among males.

Characteristics of Puberty Other Than Timing

Study 3 was designed to explore the association between pubertal synchrony, the degree to which various morphological features of puberty develop simultaneously, and depression among females. The findings revealed that asynchronous pubertal development is associated with higher levels of depressive symptoms at age 20, but not age 15. Study 3 also demonstrated that pubertal timing moderated this association, such that girls who exhibited asynchronous development had the highest levels of depressive symptoms in young adulthood when they matured later than peers.

In addition to existing literature on pubertal tempo (Keenan *et al.*, 2014; Mendle *et al.*, 2012; Marceau *et al.*, 2011; Mendle *et al.*, 2010; Ge *et al.*, 2003), the current findings confirm that features of puberty other than timing influence the occurrence of depressive symptoms. Future research should continue to explore the psychological significance of characteristics beyond timing in order to develop a more sophisticated understanding of the myriad effects of puberty on youth well-being (Mendle, 2014; Rudolph, 2014). In addition, future studies should

aim to build on the results of Study 3 by examining interactions among various pubertal features. Given the inherent complexities of a years-long developmental process that involves multiple bodily systems, the synchrony, tempo, and timing of puberty are necessarily related. As more sophisticated measurement and analytical techniques are developed, future research should continue to examine how interactions among these characteristics affect the occurrence of psychopathology.

Outcomes in Young Adulthood

All three studies examined outcomes into young adulthood in order to elucidate the association between characteristics of puberty and depressive symptoms beyond the adolescent period. The results of Studies 1 and 3 support longitudinal effects of timing and synchrony on depressive symptoms into young adulthood among females, controlling for prior youth depression. Study 1 revealed that timing interacts with conscientiousness, openness to experience, paternal conflict, and neighborhood stress to predict trajectories of depressive symptoms between grade 9 and one year post-high school, while Study 3 found that asynchronous development predicted higher levels of depressive symptoms at age 20. These results corroborate the limited prior research that supports longitudinal effects of timing on depressive symptoms (Gaysina *et al.*, 2015; Copeland *et al.*, 2010; Graber *et al.*, 2004) and provide the first evidence that synchrony is associated with psychopathology beyond adolescence.

After applying a correction to account for the use of multiple tests, Study 2 did not support contextual amplification processes in the prediction of depressive symptoms at age 20. Given that Study 1 found evidence for contextual amplification among females when exploring trajectories of symptoms into young adulthood, it is possible that the divergent findings reflect

variation in the study samples as well as methodological differences. Study 1 utilized a community sample and examined linear trajectories over time, whereas Study 2 employed a sample at high risk for the development of depression and examined symptoms at a single timepoint. The mechanisms that account for the association between pubertal timing and depressive symptoms may vary between populations that differ in depression risk, but this possibility has rarely been explored in prior research. In line with the efforts made in the present dissertation to elucidate gender differences in the mechanisms linking puberty and depression, future studies should continue to assess group differences in the association between puberty and psychopathology (e.g., among samples at low vs. high risk for depression), particularly when examining mechanistic models. Additionally, although latent growth models examining symptom trajectories demonstrate increased power compared to other methods of assessing symptoms over time (Curran, Obeidat, & Losardo, 2010), only one previous study has investigated the association between pubertal development and trajectories of symptoms (Gaysina *et al.*, 2015). Thus, within the field of puberty research, little is currently known regarding the unique contribution of studies that assess symptom trajectories as compared to those that explore static symptoms at a particular timepoint. Future research should continue to clarify the effects of contextual amplification processes on psychopathology beyond adolescence by examining longitudinal trajectories of symptoms in addition to outcomes at specific, meaningful timepoints that reflect outcomes at critical stages of development.

The Role of Gender

Taken together, the dissertation findings highlight the importance of gender differences in the experience and psychological significance of puberty as well as the mechanisms through which puberty contributes to the development of depression. Almost all significant results were

obtained among females, with males showing little evidence of associations between pubertal characteristics and depressive symptoms. As discussed above, the psychological implications of pubertal development in males have been less studied than among females, and research examining associations between puberty and depressive symptoms in males has produced mixed results. The present findings suggest that males may simply be less likely than females to react to variations in pubertal timing by becoming depressed, particularly when examining community samples at low baseline risk for symptoms of internalizing disorders (e.g., Study 1). However, high-risk males oversampled for maternal depression showed significant contextual amplification effects of dependent stressful events on adolescent depressive symptoms (Study 2), suggesting that that high-risk boys may show stronger associations between timing and depression. Additionally, research on pubertal tempo has generally supported an association between tempo and internalizing symptoms among males (Mendle *et al.*, 2012; Mendle *et al.*, 2010; Ge *et al.*, 2003), indicating that tempo may be more salient or significant to males than timing. Furthermore, previous research has suggested that while the connection between timing and internalizing conditions among males is not conclusive, variations in timing are strongly linked to externalizing behaviors among boys (Dimler & Natsuaki, 2015; Mendle & Ferrero, 2012). Thus, evidence suggests that features of puberty can affect the development of psychopathology among males in certain circumstances, but may not show the pervasive effects that tend to be observed among females. Given the general scarcity of pubertal research among males, future studies should continue to explore the specific conditions under which pubertal characteristics such as timing, tempo, and synchrony affect internalizing and externalizing symptomatology in males.

Females showed a strong association between features of puberty and depressive symptoms in the present dissertation, with evidence in support of both early and late timing as contributors to depressive symptoms in adolescence and young adulthood. These findings corroborate prior research demonstrating the importance of puberty to mental health among females (Graber, 2013; Mendle *et al.*, 2007) and offer new information about the mechanisms through which timing influences depression as well as the role of synchrony in the development of psychopathology among girls. In light of clear gender differences in the present findings, it is likely that differential psychological reactions to puberty are important contributors to the notable gender gap in depression prevalence that emerges during adolescence (Kessler, 2003; Kuehner, 2003; Wade *et al.*, 2002; Kessler *et al.*, 1993), setting women on a trajectory of depression risk and recurrence that persists across the lifespan. Future research should continue to explore the gender-specific effects of puberty on depression as well as examine unique mechanisms of observed gender differences in order to further elucidate the contribution of pubertal processes to the disparity in depression prevalence among females and males.

Refining Mechanistic Explanations

The present dissertation identified several psychosocial mechanisms through which variations in pubertal timing are associated with depressive symptoms, including interpersonal skills deficits, negative personality traits, and contextual stressors. Biological mechanisms, including hormonal, neurological, and genetic explanations for the link between timing and depression, have increasingly been explored in the literature (Mendle *et al.*, 2016; Colich *et al.*, 2015; Blaustein *et al.*, 2016; Ladouceur, 2012; Sisk & Zehr, 2005). However, despite the growing body of research focused on explicating the association between puberty and depression, existing studies have yet to bridge the gap between psychosocial and biological

explanations. Future research should continue to build upon our understanding of the complex, interrelated processes through which variations in the experience of puberty affect risk for psychopathology by examining both psychosocial and biological variables, as well as their interaction. Mendle *et al.* (2016) provide one of the first studies to successfully integrate the biology and psychosocial context of puberty, determining that genetic predispositions toward early menarche were linked to higher levels of depressive symptoms, but only among girls with high socioeconomic status. Thus, studies that examine the intersection of hypothesized mechanisms (e.g., the effects of asynchronous brain maturation among off-time adolescents with pre-existing negative personality traits such as low conscientiousness; the interaction of genetic risk factors for depression and chronic interpersonal stress among early-maturing girls; the contribution of hormonal shifts to depressed mood among off-time boys with high levels of dependent episodic stress) may uncover nuances in the relationship between puberty and psychopathology that would be obscured when assessing a more limited set of possible explanations.

Additionally, most research to date has focused on examining predictors of variations in pubertal timing *or* the consequences of this variation (such as the present dissertation). An extensive body of literature suggests that biological and psychosocial factors such as obesity, exposure to chemicals that alter hormonal functioning, and environmental stressors in childhood can predispose children to mature off-time (Euling, Selevan, Hirsch Pescovitz, & Skakkebaek, 2008; Ellis & Essex, 2007; Biro, Khoury, & Morrison, 2006; Chisholm *et al.*, 2005; Ellis & Garber, 2000). However, attempts to link early risk factors for off-time development with observed outcomes of early or late maturation are rare. Furthermore, psychosocial predictors of off-time development (e.g., early life stress, obesity) have been shown to have a direct impact on

conditions often assumed within the puberty literature to be outcomes of variations in pubertal timing, such as depression (Klein *et al.*, 2009; Reeves, Postolache, & Snitker, 2008; Mustillo *et al.*, 2003). Given research highlighting the continuity of stress from early childhood through adolescence as a predictor of depression (Hazel, Hammen, Brennan, & Najman, 2008) as well as evidence linking early adversity and various risk factors known to be associated with pubertal timing (e.g., interpersonal stress, poor self-image, heightened stress reactivity; Natsuaki, Klimes-Dougan, *et al.*, 2009; Turner & Butler, 2003; Johnson *et al.*, 2002; Hammen, Henry, & Daley, 2000), it is possible that many of the consequences ascribed to variations in pubertal timing partially reflect processes that led to off-time development (Rudolph, 2014). Future research should seek to clearly establish the unique contribution of pubertal timing by examining models that incorporate both predictors and consequences of maturing off-time.

Furthermore, as evidenced in the present dissertation, several types of mechanisms account for the relationship between pubertal timing and depression. However, studies examining the combined effects of established mediating and moderating variables are rare, and little is known about which mechanisms are relevant for particular groups of youth (e.g., females versus males, individuals at low versus high risk for depression, etc.). As an example, given the evidence provided by Study 1 in support of stage termination among females, perhaps early-maturing girls with low emotional stability, conscientiousness, or openness to experience exhibit greater difficulty interacting successfully with peers, leading to subsequent increases in depression. Alternatively, perhaps other variables that have been hypothesized to explain the association between early timing and psychopathology might serve as mechanisms of personal accentuation or contextual amplification processes. For example, girls experiencing high levels of chronic peer, family, or neighborhood stress might have difficulty engaging in effective

copied behaviors, leading to higher levels of depression. Among males, the mechanism through which contextual amplification of dependent episodic stress leads to increases in adolescent depressive symptoms is even less clear, as Study 1 did not support stage termination or maturation disparity in boys. Future studies should aim to explore comprehensive models in which both mediating and moderating variables are assessed simultaneously in order to further refine our understanding of the processes through which variations in pubertal timing lead to depressive symptoms among particular groups.

Finally, as the field expands to examine metrics of pubertal development other than timing, future research should aim to identify the mechanisms through which characteristics such as tempo and synchrony contribute to psychopathology, including depression. Little is currently known about whether these mechanisms will prove to be similar or distinct from those that account for the link between timing and depression.

Strengths and Limitations

Overall, the present dissertation has a number of strengths that enhance its contribution to the literature. The findings are novel, expanding the study of puberty and depression by investigating hypothesized mechanisms of this association, exploring pubertal characteristics that have never previously been studied (i.e., synchrony), and extending the examination of outcomes into young adulthood. The inclusion of both female and male participants followed longitudinally with contemporaneous assessments of pubertal development allows for an in-depth and highly valid exploration of puberty's long-term effects. Additionally, studies included in the dissertation used a community sample of adolescents (Study 1) as well as a high-risk sample in which maternal depression was oversampled (Studies 2 and 3). Although direct comparisons between these samples were not conducted, the results offer some insight into the

ways in which the association between pubertal features and depressive symptoms might vary across low- and high-risk populations (i.e., high-risk samples may be more vulnerable to the effects of off-time maturation, particularly when off-time development occurs in the context of significant environmental stress or negative personality traits).

However, the measurement of pubertal development employed in the three studies is an important limitation of the present dissertation. Pubertal status was ascertained via self- and parent-ratings of physical maturation, and each of the studies assessed status at a single timepoint. Although these are common practices among studies of puberty and psychopathology (e.g., Benoit *et al.*, 2013; Conley & Rudolph, 2009; Rudolph, 2008), they necessarily limit the reliability of the findings due to limitations associated with non-physician ratings (Dorn *et al.*, 2006; Dorn *et al.*, 2003) as well as the challenges of capturing the intricacies of a dynamic developmental process at a single point in time. Future research should seek to employ repeated measures of development using multiple metrics (i.e., hormonal, biological, morphological) in order to capture the full experience of pubertal development and its association with psychopathology. In addition, youth participating in each of the studies were primarily Caucasian. Normative pubertal development is known to vary according to demographic characteristics such as race and ethnicity, and associations between characteristics of puberty and psychopathology have also been shown to vary by race (Hamlat *et al.*, 2015; Keenan *et al.*, 2014; Anderson *et al.*, 2003; Kaplowitz *et al.*, 2001). Thus, the composition of the samples used in the present dissertation limits the generalizability of the results and furthers the existing imbalance in understanding pubertal processes among Caucasian as compared to non-Caucasian individuals. Future research should seek to maximize inclusivity and representativeness in order to better understand the effects of pubertal features on psychopathology more generally.

The present dissertation examined outcomes related to solely to depression and did not explore biological explanations of the link between pubertal characteristics and psychopathology. Furthermore, beyond controlling for childhood depressive symptoms, maternal depression history, and maternal education, the studies included in the dissertation did not account for the substantial body of literature highlighting the association between early childhood experiences and variations in pubertal development. Thus, the present results reflect a limited model of the association between puberty and psychopathology. Future research should continue to expand models of puberty and psychopathology to explore outcomes other than depression, examine both psychosocial and biological mechanisms, and assess predictors as well as outcomes of variations in pubertal characteristics in order to provide the most comprehensive understanding of the link between puberty and psychological well-being across the lifespan.

Conclusions

The relationship between puberty, an intricate biological process imbued with cultural and personal significance, and depression, a disorder resulting from the interplay of biological, interpersonal, and cognitive factors, is necessarily complex. The present dissertation sought to clarify this relationship by exploring potential mechanisms of the association between pubertal timing and depression, examining features of pubertal development that have generally been overlooked in research to date, and assessing long-term outcomes. The findings contribute to an increasingly refined understanding of pubertal development and its relation to depression while raising a number of critical issues for future research, including the role of puberty as a contributor to gender differences in depression prevalence, the necessity of identifying the unique mechanisms through which puberty contributes to psychopathology among particular groups of youth, and the importance of assessing inclusive models of development that reflect

the varied biological and psychosocial experiences that comprise the pubertal transition. Future studies should continue to investigate the association between puberty and depression with the aim of identifying targets for prevention and treatment that will improve mental health outcomes during adolescence and across the lifespan.

Tables

Table 1. Descriptive statistics for Study 1 variables by gender.

Variable	Females (<i>N</i> = 538)		Males (<i>N</i> = 532)	
	Mean (SD)	Range	Mean (SD)	Range
Age at study-relevant grades				
Grade 4	9.96 (0.37)	9.1-11.0	-	-
Grade 5	10.96 (0.36)	10.1-11.9	-	-
Grade 6	-	-	12.05 (0.40)	10.4-13.4
Grade 7	-	-	13.12 (0.37)	12.2-14.5
Grade 9	15.21 (0.36)	14.5-16.3	15.25 (0.37)	14.4-16.5
Grade 12	18.21 (0.36)	17.2-19.5	18.24 (0.39)	17.2-20.5
Post-high school	19.30 (0.38)	18.4-20.8	19.37 (0.44)	18.4-21.1
Pubertal status, mean parent rating (PDS)				
Grade 4	1.65 (0.42)	1-3		
Grade 5	1.98 (0.52)	1-3.4		
Grade 6			1.69 (0.48)	1-3.2
Grade 7			2.07 (0.58)	1-3.5
Depressive symptoms (CESD)				
Grade 9	15.09 (11.87)	0-55	10.88 (8.31)	0-52
Grade 10	15.84 (12.44)	0-55	11.52 (9.18)	0-54
Grade 11	16.40 (11.56)	0-55	11.75 (9.56)	0-54
Grade 12	15.06 (11.68)	0-60	11.75 (9.84)	0-57
Post-high school	14.81 (11.43)	0-56	13.20 (10.77)	0-51
Childhood depressive symptoms ^a	18.91 (6.36)	13-48	17.68 (5.88)	13-52
Mediators ^b				
Coping	21.57 (4.78)	6-30	18.20 (5.80)	6-30
Perceived stress	3.18 (2.36)	0-13	2.96 (2.53)	0-12

Self-image	23.72 (4.49)	10-30	24.40 (4.35)	10-30
Social competence	3.18 (0.64)	1-4	3.17 (0.65)	1-4
Personality traits ^c				
Extraversion	8.62 (1.64)	2-10	8.37 (1.62)	2-10
Agreeableness	8.92 (1.21)	2-10	8.67 (1.34)	3-10
Conscientiousness	7.45 (1.80)	2-10	6.99 (1.83)	2-10
Emotional stability	4.96 (1.82)	2-10	5.75 (1.91)	2-10
Openness to experience	8.62 (1.44)	2-10	8.24 (1.53)	2-10
Contextual factors ^d				
Maternal depressive symptoms	6.08 (4.31)	0-22	5.79 (4.60)	0-27
Paternal depressive symptoms	4.67 (3.59)	0-18	4.92 (3.97)	0-23
Stressful life events	3.27 (2.12)	0-10	3.03 (2.13)	0-11
Maternal relationship conflict	1.99 (1.48)	0-7	1.86 (1.37)	0-6.5
Paternal relationship conflict	2.02 (1.59)	0-7	1.73 (1.50)	0-7
Neighborhood stress	9.52 (2.55)	7-20	9.47 (2.56)	7-21

Note. ^a Measured during grade 4 for all participants other than those in the oldest cohort, for whom childhood depressive symptoms were measured during grade 5. ^b Assessed among females during grades 6 and 7 and assessed among males during grade 8. ^c Assessed during grades 7 and 8. ^d Contextual factors were measured during grades 6-8 for all participants, other than maternal depressive symptoms, which were assessed during grades 4-8.

Table 2. Number and percentage of youth in Study 1 who met or exceeded the clinical cutoff for mild depression based on CESD score (≥ 16).

Grade	Females	Males
	<i>N</i> (%)	<i>N</i> (%)
9	144 (37.4%)	82 (22.4%)
10	192 (41.1%)	115 (25.9%)
11	205 (44.1%)	119 (27.0%)
12	172 (38.1%)	122 (29.2%)
One year post-high school	135 (39.8%)	105 (34.3%)

Note. Total *N* = 538 females and 532 males. Data on depressive symptoms were not available for all participants at all assessment points.

Table 3. Correlation coefficients among levels of each proposed moderator by grade level (Study 1).

Proposed Moderators	Females			Males		
	Grades 7 & 8			Grades 7 & 8		
Personality traits						
Extraversion	.63			.55		
Agreeableness	.43			.41		
Conscientiousness	.43			.46		
Emotional stability	.41			.41		
Openness to experience	.49			.53		
	Grades			Grades		
	6 & 7	6 & 8	7 & 8	6 & 7	6 & 8	7 & 8
Contextual factors						
Maternal depressive symptoms ^a	.60	.59	.63	.56	.61	.63
Paternal depressive symptoms ^a	.59	.40	.57	.64	.54	.54
Stressful life events	.39	.39	.35	.42	.25	.38
Maternal relationship conflict	.55	.49	.55	.44	.37	.55
Paternal relationship conflict	.51	.45	.54	.49	.51	.52
Neighborhood stress	.40	.47	.57	.50	.32	.48

Note. ^a Maternal and paternal depressive symptoms were assessed between grades 4 through 8 among females. Additional correlation coefficients across grade levels not presented above range between .44 and .73 for maternal depressive symptoms and between .40 and .64 for paternal depressive symptoms.

Table 4. Bivariate correlations among Study 1 variables.

Variable	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
1. Linear pubertal timing	-														
2. Quadratic pubertal timing	.18*	-													
3. Grade 9 CESD	.10*	.01	-												
4. Grade 10 CESD	.12*	.07*	.57*	-											
5. Grade 11 CESD	.11*	.00	.41*	.54*	-										
6. Grade 12 CESD	.13*	-.01	.42*	.45*	.53*	-									
7. One year post-high school CESD	.09*	.00	.36*	.38*	.44*	.53*	-								
8. Coping	-.01	.02	-.11*	-.05	-.07*	-.08	-.05	-							
9. Perceived stress	.10*	-.04	.17*	.16*	.11*	.09*	.12*	-.01	-						
10. Self-image	-.02	.05	-.35*	-.30*	-.26*	-.31*	-.22*	.30*	-.16*	-					
11. Social competence	-.04	-.03	-.27*	-.24*	-.18*	-.19*	-.16*	.19*	-.03	.30*	-				
12. Extraversion	-.08	.02	-.17*	-.17*	-.17*	-.22*	-.17*	.24*	.03	.29*	.48*	-			
13. Agreeableness	-.08	.01	-.12*	-.13*	-.09*	-.15*	-.14*	.28*	-.09*	.17*	.25*	.55*	-		
14. Conscientiousness	-.07	.02	-.11*	-.15*	-.14*	-.17*	-.10	.23*	-.03	.20*	.19*	.39*	.52*	-	
15. Emotional stability	-.04	-.02	-.24*	-.25*	-.22*	-.14*	-.07	-.05	-.14*	.14*	.00	-.20*	-.19*	-.22*	-
16. Openness to experience	-.11*	.03	-.11*	-.15*	-.13*	-.17*	-.16*	.27*	-.02	.20	.26	.44*	.51*	.49*	-.19*
17. Maternal depressive symptoms	.10*	.04	.16*	.16*	.12*	.19*	.18*	-.07	.08*	-.15*	-.12*	-.12*	-.08*	-.04	-.05
18. Paternal depressive symptoms	.04	-.03	.17*	.14*	.11*	.14*	.04	-.06	.10*	-.10*	-.07	-.07	.03	-.04	-.10
19. Stressful life events	.10*	-.03	.26*	.23*	.14*	.14*	.14*	-.03	.84*	-.21*	-.05	.04	-.07	-.04	-.20*
20. Maternal conflict	.09*	.02	.25*	.26*	.17*	.22*	.23*	-.17*	.20*	-.28*	-.15*	-.19*	-.24*	-.14*	-.15*
21. Paternal conflict	.06	-.01	.27*	.27*	.20*	.25*	.24*	-.11*	.12*	-.32*	-.21*	-.23*	-.12*	-.12*	-.17*
22. Neighborhood stress	.09*	.05	.30*	.34*	.28*	.25*	.26*	-.10*	.31*	-.18*	-.03	-.08	-.10*	-.11*	-.18*
23. Childhood depressive symptoms	.07*	-.01	.29*	.26*	.22*	.22*	.21*	-.07	.13*	-.30*	-.18*	-.16*	-.13*	-.16*	-.09*

Variable	16	17	18	19	20	21	22	23
1. Linear pubertal timing								
2. Quadratic pubertal timing								
3. Grade 9 CESD								
4. Grade 10 CESD								
5. Grade 11 CESD								
6. Grade 12 CESD								
7. One year post-high school CESD								
8. Coping								
9. Perceived stress								
10. Self-image								
11. Social competence								
12. Extraversion								
13. Agreeableness								
14. Conscientiousness								
15. Emotional stability								
16. Openness to experience	-							
17. Maternal depressive symptoms	-.05	-						
18. Paternal depressive symptoms	.03	.29*	-					
19. Stressful life events	-.02	.15*	.12*	-				
20. Maternal conflict	-.16*	.15*	.07	.26*	-			
21. Paternal conflict	-.18*	.13*	.10*	.20*	.51*	-		
22. Neighborhood stress	-.07	.12*	.15*	.43*	.28*	.22*	-	
23. Childhood depressive symptoms	-.16*	.12*	.02	.21*	.24*	.27*	.16*	-

Note. * $p < .05$

Table 5. Direct and indirect effects of linear pubertal timing on depressive symptoms in grades 9 and 12 via four proposed mediators, adjusted for childhood depressive symptoms (Study 1).

Mediator	Gender	Grade	N	Indirect Effect ^a			Direct Effect ^a		
				Effect	SE	95% CI	Effect	SE	95% CI
Coping	Females	9	319	.03	.10	(-.17, .24)	1.20	.65	(-.08, 2.48)
		12	374	.09	.10	(-.07, .33)	1.67*	.56	(.57, 2.77)
	Males	9	226	.02	.09	(-.16, .22)	.37	.54	(-.70, 1.45)
		12	265	-.004	.04	(-.15, .06)	.72	.61	(-.47, 1.91)
Perceived stress	Females	9	338	.19	.15	(-.10, .53)	.88	.61	(-.33, 2.09)
		12	396	.01	.04	(-.04, .14)	1.82*	.55	(.74, 2.89)
	Males	9	233	.11	.10	(-.05, .38)	.28	.56	(-.84, 1.39)
		12	277	.09	.10	(-.03, .36)	.55	.60	(-.64, 1.74)
Self-image	Females	9	338	.19	.19	(-.16, .59)	.89	.60	(-.30, 2.07)
		12	396	.11	.14	(-.15, .41)	1.72*	.53	(.68, 2.75)
	Males	9	233	-.07	.16	(-.42, .21)	.46	.54	(-.60, 1.52)
		12	276	-.23	.18	(-.65, .08)	.87	.58	(-.27, 2.01)
Social competence	Females	9	314	.29*	.17	(.02, .71)	.77	.64	(-.49, 2.03)
		12	372	.21*	.14	(.03, .60)	1.52*	.56	(.42, 2.62)
	Males	9	203	-.16	.15	(-.57, .05)	.32	.58	(-.82, 1.46)
		12	249	-.11	.11	(-.40, .03)	.65	.61	(-.55, 1.86)

Note. * $p < .05$, as evidenced by 95% confident interval that does not include zero.

Table 6. Direct and indirect effects of quadratic pubertal timing on depressive symptoms in grades 9 and 12 via four proposed mediators, adjusted for childhood depressive symptoms and linear pubertal timing (Study 1).

Mediator	Gender	Grade	N	Indirect Effect ^a			Direct Effect ^a		
				Effect	SE	95% CI	Effect	SE	95% CI
Coping	Females	9	319	.10	.08	(-.01, .34)	-.24	.47	(-1.17, .69)
		12	374	.07	.07	(-.04, .25)	-.15	.41	(-.95, .65)
	Males	9	226	-.23*	.13	(-.59, -.05)	1.00	.52	(-.01, 2.02)
		12	265	-.02	.05	(-.23, .04)	.55	.56	(-.56, 1.65)
Perceived stress	Females	9	338	-.16	.11	(-.40, .03)	-.05	.44	(-.91, .81)
		12	396	-.05	.05	(-.19, .01)	.05	.39	(-.72, .83)
	Males	9	233	.02	.04	(-.04, .16)	.60	.52	(-.43, 1.63)
		12	277	-.01	.05	(-.14, .07)	.46	.56	(-.64, 1.56)
Self-image	Females	9	338	-.11	.13	(-.39, .14)	-.11	.43	(-.95, .74)
		12	396	-.16	.10	(-.39, .03)	.16	.38	(-.59, .90)
	Males	9	233	-.02	.15	(-.32, .30)	.63	.51	(-.36, 1.63)
		12	276	.10	.16	(-.19, .44)	.36	.54	(-.70, 1.42)
Social competence	Females	9	314	.00	.11	(-.21, .22)	-.23	.45	(-1.12, .66)
		12	372	.08	.09	(-.06, .31)	-.11	.40	(-.90, .67)
	Males	9	203	-.01	.14	(-.28, .29)	.61	.55	(-.47, 1.68)
		12	249	.00	.10	(-.20, .21)	.53	.56	(-.58, 1.64)

Note. * $p < .05$, as evidenced by 95% confident interval that does not include zero.

Table 7. Fit indices of latent growth models estimating quadratic and linear interactions between pubertal timing and various hypothesized moderators in the prediction of depressive symptoms among females (Study 1).

Moderator	Highest Order Pubertal Timing Variable in the Model	Fit Indices				
		χ^2 (df)	<i>p</i>	RMSEA	CFI	SRMR
Extraversion	Quadratic	65.04(29)	< .001	.05	.94	.03
	Linear	53.05(23)	< .001	.05	.95	.04
Agreeableness	Quadratic	65.33(29)	< .001	.05	.94	.03
	Linear	54.00(23)	< .001	.05	.95	.04
Conscientiousness	Quadratic	67.33(29)	< .001	.05	.94	.03
Emotional stability	Quadratic	58.83(29)	.001	.04	.95	.03
	Linear	47.03(23)	.002	.05	.96	.03
Openness to experience	Quadratic	60.64(29)	.001	.05	.95	.03
Maternal depressive symptoms	Quadratic	64.46(29)	< .001	.05	.94	.03
	Linear	54.85(23)	< .001	.05	.95	.03
Paternal depressive symptoms	Quadratic	58.45(29)	.001	.04	.95	.03
	Linear	49.13(23)	.001	.05	.95	.03
Stressful life events	Quadratic	64.70(29)	< .001	.05	.95	.03
	Linear	53.72(23)	< .001	.05	.95	.03
Maternal relationship conflict	Quadratic	59.16(29)	.001	.05	.95	.03
	Linear	49.12(23)	.001	.05	.95	.03
Paternal relationship conflict	Quadratic	66.06(29)	< .001	.05	.94	.03
	Linear	54.55(23)	< .001	.05	.95	.03
Neighborhood stress	Quadratic	64.01(29)	< .001	.05	.95	.03
	Linear	50.49(23)	.001	.05	.95	.03

Table 8. Fit indices of latent growth models estimating quadratic and linear interactions between pubertal timing and various hypothesized moderators in the prediction of depressive symptoms among males (Study 1).

Moderator	Highest Order Pubertal Timing Variable in the Model	Fit Indices				
		χ^2 (df)	<i>p</i>	RMSEA	CFI	SRMR
Extraversion	Quadratic	26.08(29)	.62	.00	1.00	.02
	Linear	20.34(23)	.62	.00	1.00	.02
Agreeableness	Quadratic	19.79(29)	.90	.00	1.00	.02
	Linear	16.64(23)	.83	.00	1.00	.02
Conscientiousness	Quadratic	23.36(29)	.76	.00	1.00	.02
	Linear	18.21(23)	.75	.00	1.00	.03
Emotional stability	Quadratic	25.21(29)	.67	.00	1.00	.02
	Linear	20.52(23)	.61	.00	1.00	.03
Openness to experience	Quadratic	25.31(29)	.66	.00	1.00	.02
	Linear	18.66(23)	.72	.00	1.00	.02
Maternal depressive symptoms	Quadratic	21.45(29)	.84	.00	1.00	.02
	Linear	19.25(23)	.69	.00	1.00	.03
Paternal depressive symptoms	Quadratic	21.63(29)	.84	.00	1.00	.02
	Linear	17.60(23)	.78	.00	1.00	.02
Stressful life events	Quadratic	29.69(29)	.43	.01	1.00	.02
	Linear	24.09(23)	.40	.01	1.00	.03
Maternal relationship conflict	Quadratic	31.95(29)	.32	.01	.99	.03
	Linear	22.85(23)	.47	.00	1.00	.03
Paternal relationship conflict	Quadratic	21.88(29)	.83	.00	1.00	.02
	Linear	19.03(23)	.70	.00	1.00	.03
Neighborhood stress	Quadratic	25.22(29)	.67	.00	1.00	.02
	Linear	19.38(23)	.68	.00	1.00	.03

Table 9. Participants' pubertal status at age 13 assessment based on self-rated Tanner stages (Studies 2 and 3).

Tanner stage	Females		Males	
	Breast (<i>N</i> = 358)	Pubic hair (<i>N</i> = 355)	Genital (<i>N</i> = 367)	Pubic hair (<i>N</i> = 364)
Stage 1	1 (0.3%)	0 (0%)	8 (2.2%)	7 (1.9%)
Stage 2	18 (5.0%)	14 (3.9%)	45 (12.3%)	51 (14.0%)
Stage 3	131 (36.6%)	78 (22.0%)	155 (42.2%)	123 (33.8%)
Stage 4	183 (51.1%)	224 (63.1%)	134 (36.5%)	140 (38.5%)
Stage 5	25 (7.0%)	39 (11.0%)	25 (6.8%)	43 (11.8%)

Table 10. Descriptive statistics for study variables by gender (Study 2).

Variable	Total (<i>N</i> = 725)		Females (<i>N</i> = 358)		Males (<i>N</i> = 367)	
	Mean (SD)	Range	Mean (SD)	Range	Mean (SD)	Range
Age 13 Tanner stage						
Breast	3.59 (0.71)	1-5	3.59 (0.71)	1-5	-	-
Genital	3.34 (0.86)	1-5	-	-	3.34 (0.86)	1-5
Pubic hair	3.62 (0.84)	1-5	3.81 (0.67)	2-5	3.44 (0.94)	1-5
Depressive symptoms (BDI-II)						
Age 15	6.02 (6.74)	0-50	7.02 (7.06)	0-44	5.05 (6.26)	0-50
Age 20	6.92 (8.29)	0-52	8.01 (9.16)	0-52	5.66 (6.97)	0-47
Age 15 chronic stress						
Peer	2.28 (0.47)	1-4.5	2.27 (0.46)	1-4	2.29 (0.48)	1-4.5
Family	2.34 (0.59)	1-5	2.37 (0.60)	1-5	2.31 (0.57)	1-5
Close friendships	2.22 (0.51)	1-5	2.17 (0.47)	1-5	2.28 (0.54)	1.5-5
Romantic relationships	2.14 (0.37)	1-4	2.19 (0.42)	1-4	2.09 (0.31)	1-3
Academics	2.67 (0.93)	1-5	2.50 (0.84)	1-5	2.84 (0.98)	1-5
Age 15 episodic stress						
Independent stress	3.04 (2.78)	0-20	3.53 (3.09)	0-20	2.58 (2.35)	0-12.5
Dependent stress	2.95 (2.72)	0-15.5	3.27 (2.86)	0-15.5	2.63 (2.53)	0-12.5
	<i>N</i> (%)		<i>N</i> (%)		<i>N</i> (%)	
Caucasian race ^a	666 (91.9%)		334 (93.3%)		332 (90.5%)	

Note. ^aValues do not match the total number of participants due to missing race/ethnicity data (*N* = 20).

Table 11. Three-way interaction effects of gender, pubertal timing, and contextual stress variables on age 15 and age 20 depressive symptoms, adjusted for maternal depression history and prior youth depressive symptoms (Study 2).

	Type of Interaction	$R^2\Delta$	$F\Delta$	p Value of Model	p Value of Interaction
Age 15 depressive symptoms					
Chronic peer stress	Quadratic	.02	10.25	< .001	< .001*
Chronic family stress	Quadratic	.02	8.72	< .001	.89
	Linear	.02	14.04	< .001	< .001*
Chronic close friendship stress	Quadratic	.001	.49	.61	.39
	Linear	.00	.00	.97	.97
Chronic romantic relationship stress	Quadratic	.00	1.43	.24	.16
	Linear	.002	1.31	.25	.25
Chronic academic stress	Quadratic	.003	1.16	.32	.26
	Linear	.001	1.14	.29	.29
Independent episodic stress	Quadratic	.001	.43	.65	.36
	Linear	.00	.12	.73	.73
Dependent episodic stress	Quadratic	.01	4.67	.01	.25
	Linear	.01	5.35	.02	.02
Age 20 depressive symptoms					
Chronic peer stress	Quadratic	.001	.32	.73	.44
	Linear	.00	.10	.75	.75
Chronic family stress	Quadratic	.01	2.42	.09	.04
	Linear	.00	.28	.59	.59

Chronic close friendship stress	Quadratic	.003	1.04	.35	.15
	Linear	.00	.08	.78	.78
Chronic romantic relationship stress	Quadratic	.001	.44	.64	.48
	Linear	.001	.43	.51	.51
Chronic academic stress	Quadratic	.01	1.98	.14	.95
	Linear	.01	4.03	.05	.05
Independent episodic stress	Quadratic	.001	.48	.62	.85
	Linear	.001	.92	.34	.34
Dependent episodic stress	Quadratic	.01	1.85	.16	.08
	Linear	.00	.14	.71	.71

Note. *Statistically significant following false discovery rate control procedures to correct for multiple tests ($d = .05$, $N = 80$).

Table 12. Two-way interaction effects of pubertal timing and contextual stress variables in the prediction of age 15 and age 20 depressive symptoms among females, adjusted for maternal depression history and prior youth depressive symptoms (Study 2).

	Type of Interaction	$R^2\Delta$	$F\Delta$	p Value of Model	p Value of Interaction
Age 15 depressive symptoms					
Chronic peer stress	Quadratic	.04	7.93	< .001	.002*
Chronic family stress	Quadratic	.03	8.06	< .001	.47
	Linear	.02	11.59	.001	.001*
Chronic close friendship stress	Quadratic	.01	2.29	.10	.06
	Linear	.002	.71	.40	.40
Chronic romantic relationship stress	Quadratic	.02	5.30	.005	.02
Chronic academic stress	Quadratic	.001	.30	.74	.92
	Linear	.002	.80	.37	.37
Independent episodic stress	Quadratic	.001	.14	.87	.68
	Linear	.001	.59	.45	.45
Dependent episodic stress	Quadratic	.002	.33	.72	.98
	Linear	.00	.08	.78	.78
Age 20 depressive symptoms					
Chronic peer stress	Quadratic	.00	.09	.92	.68
	Linear	.00	.01	.93	.93
Chronic family stress	Quadratic	.01	2.45	.09	.06
	Linear	.001	.41	.52	.52
Chronic close friendship stress	Quadratic	.003	.47	.63	.34
	Linear	.00	.06	.81	.81

Chronic romantic relationship stress	Quadratic	.01	2.40	.09	.03
	Linear	.00	.00	.96	.96
Chronic academic stress	Quadratic	.02	2.93	.06	.58
	Linear	.02	6.31	.01	.01
Independent episodic stress	Quadratic	.01	1.95	.15	.32
	Linear	.01	1.77	.19	.19
Dependent episodic stress	Quadratic	.00	.05	.95	.97
	Linear	.00	.08	.78	.78

Note. *Statistically significant following false discovery rate control procedures to correct for multiple tests ($d = .05$, $N = 80$).

Table 13. Two-way interaction effects of pubertal timing and contextual stress variables in the prediction of age 15 and age 20 depressive symptoms among males, adjusted for maternal depression history and prior youth depressive symptoms (Study 2).

	Type of Interaction	$R^2\Delta$	$F\Delta$	p Value of Model	p Value of Interaction
Age 15 depressive symptoms					
Chronic peer stress	Quadratic	.01	2.24	.11	.05
	Linear	.001	.35	.56	.56
Chronic family stress	Quadratic	.01	1.95	.14	.44
	Linear	.01	3.06	.08	.08
Chronic close friendship stress	Quadratic	.003	.68	.51	.49
	Linear	.002	.79	.38	.38
Chronic romantic relationship stress	Quadratic	.001	.17	.85	.89
	Linear	.00	.14	.71	.71
Chronic academic stress	Quadratic	.01	1.27	.28	.14
	Linear	.001	.33	.57	.57
Independent episodic stress	Quadratic	.004	.88	.42	.20
	Linear	.00	.02	.90	.90
Dependent episodic stress	Quadratic	.03	5.61	.004	.15
	Linear	.02	8.86	.003	.003*
Age 20 depressive symptoms					
Chronic peer stress	Quadratic	.01	1.42	.24	.10
	Linear	.001	.19	.66	.66
Chronic family stress	Quadratic	.20	.84	.44	.20
	Linear	.00	.04	.84	.84

Chronic close friendship stress	Quadratic	.01	.68	.51	.25
	Linear	.00	.02	.90	.90
Chronic romantic relationship stress	Quadratic	.01	.68	.51	.51
	Linear	.003	.94	.33	.33
Chronic academic stress	Quadratic	.001	.13	.88	.66
	Linear	.00	.08	.78	.78
Independent episodic stress	Quadratic	.003	.44	.65	.36
	Linear	.00	.05	.83	.83
Dependent episodic stress	Quadratic	.02	3.13	.05	.03

Note. *Statistically significant following false discovery rate control procedures to correct for multiple tests ($d = .05$, $N = 80$).

Table 14. Descriptive statistics of study variables by pubertal synchrony (Study 3).

Variable	Synchronous pubertal development				Pubarche				Thelarche			
	Early-maturing	On-time	Late-maturing	Total	Early-maturing	On-time	Late-maturing	Total	Early-maturing	On-time	Late-maturing	Total
<i>N</i>	38	124	65	227	22	63	12	97	11	13	7	31
Caucasian race (%) ^a	36 (94.7%)	118 (95.9%)	60 (93.8%)	214 (94.3%)	18 (90%)	60 (98.4%)	12 (100%)	90 (92.8%)	10 (100%)	12 (92.3%)	5 (71.4%)	27 (87.1%)
Mean (SD) Tanner breast stage at age 13	4.34 (0.48)	4.00 (0)	2.91 (0.29)	3.74 (0.60)	3.91 (0.29)	2.98 (0.13)	1.92 (0.29)	3.06 (0.61)	5.00 (0)	4.00 (0)	3.00 (0)	4.13 (0.76)
Mean (SD) Tanner pubic hair stage at age 13	4.34 (0.48)	4.00 (0)	2.91 (0.29)	3.74 (0.60)	5.00 (0)	4.06 (0.25)	3.33 (0.65)	4.19 (0.58)	4.00 (0)	3.00 (0)	2.00 (0)	3.13 (0.76)
Mean (SD) BDI-II score at age 15	8.26 (5.93)	5.70 (5.97)	7.17 (6.84)	6.59 (6.27)	10.18 (8.62)	5.92 (6.10)	10.92 (13.06)	7.51 (8.03)	7.09 (5.80)	7.08 (6.22)	12.43 (15.25)	8.29 (8.86)
Mean (SD) BDI-II score at age 20	7.03 (7.32)	6.79 (8.40)	6.88 (7.33)	6.86 (7.88)	14.00 (14.24)	7.23 (7.50)	15.30 (13.34)	9.64 (10.49)	11.86 (11.11)	8.15 (8.44)	19.00 (17.13)	11.65 (11.91)

Note. ^aValues do not match the total number of participants due to missing race/ethnicity data (*N* = 7).

Figures

Figure 1. General form of main effects latent growth model (Study 1), in which pubertal timing predicts trajectories of youth depressive symptoms. Childhood depressive symptoms are included as a covariate.

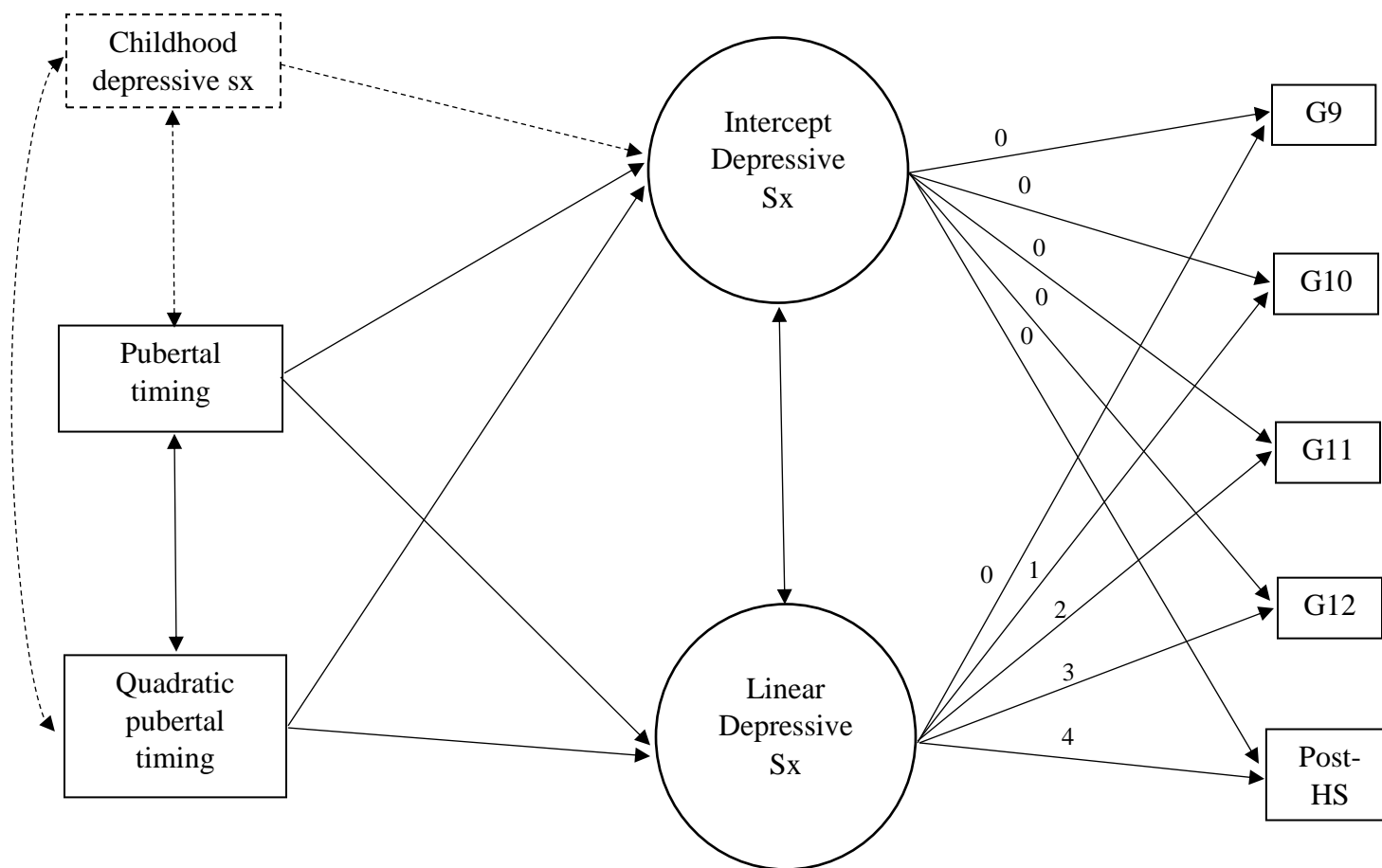


Figure 2. General form of the latent growth model used to test quadratic moderation (Study 1). Childhood depressive symptoms are included as a covariate.

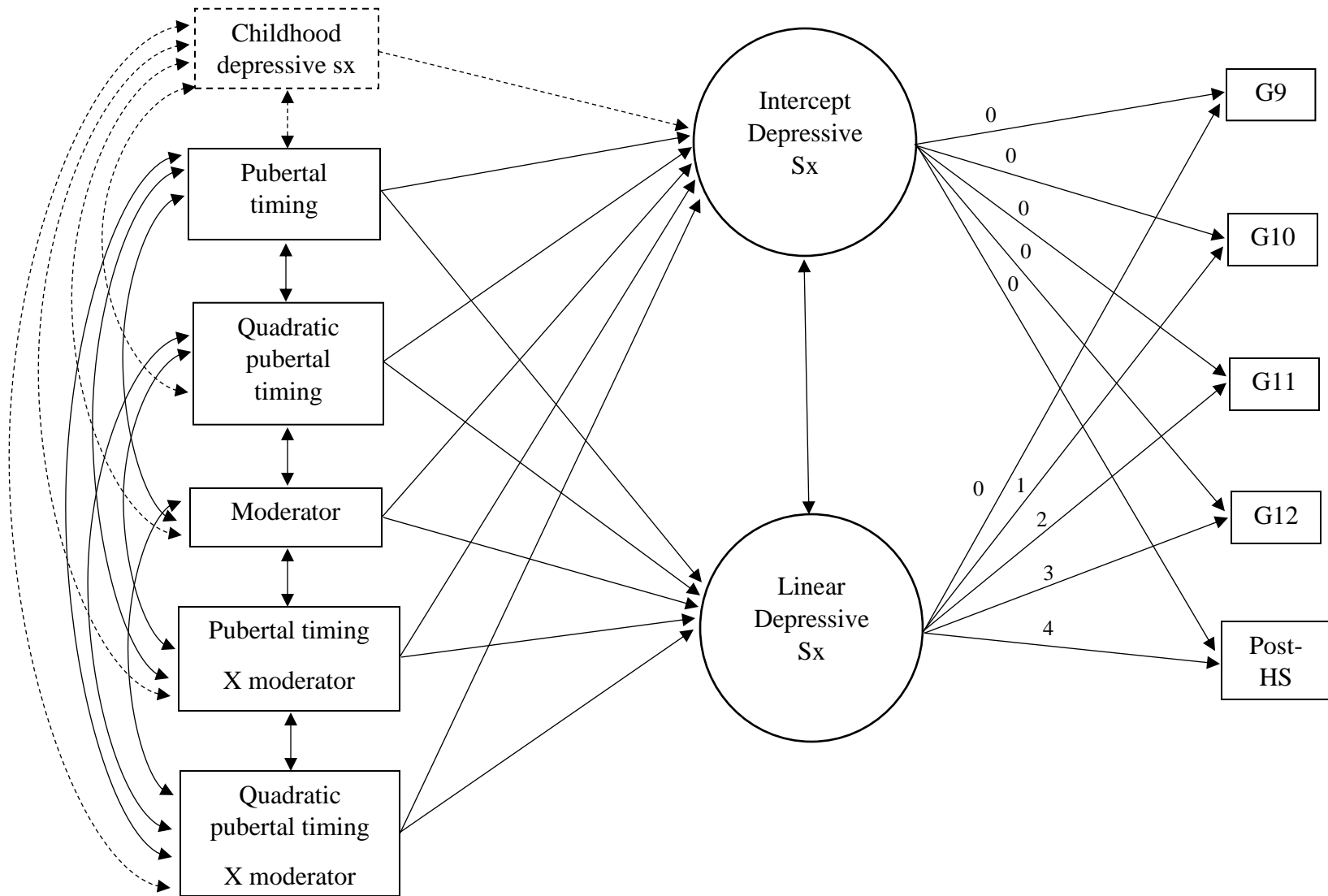


Figure 3. General form of the latent growth model used to test linear moderation, in which the quadratic pubertal timing variable and its interaction term have been omitted (Study 1). Childhood depressive symptoms are included as a covariate.

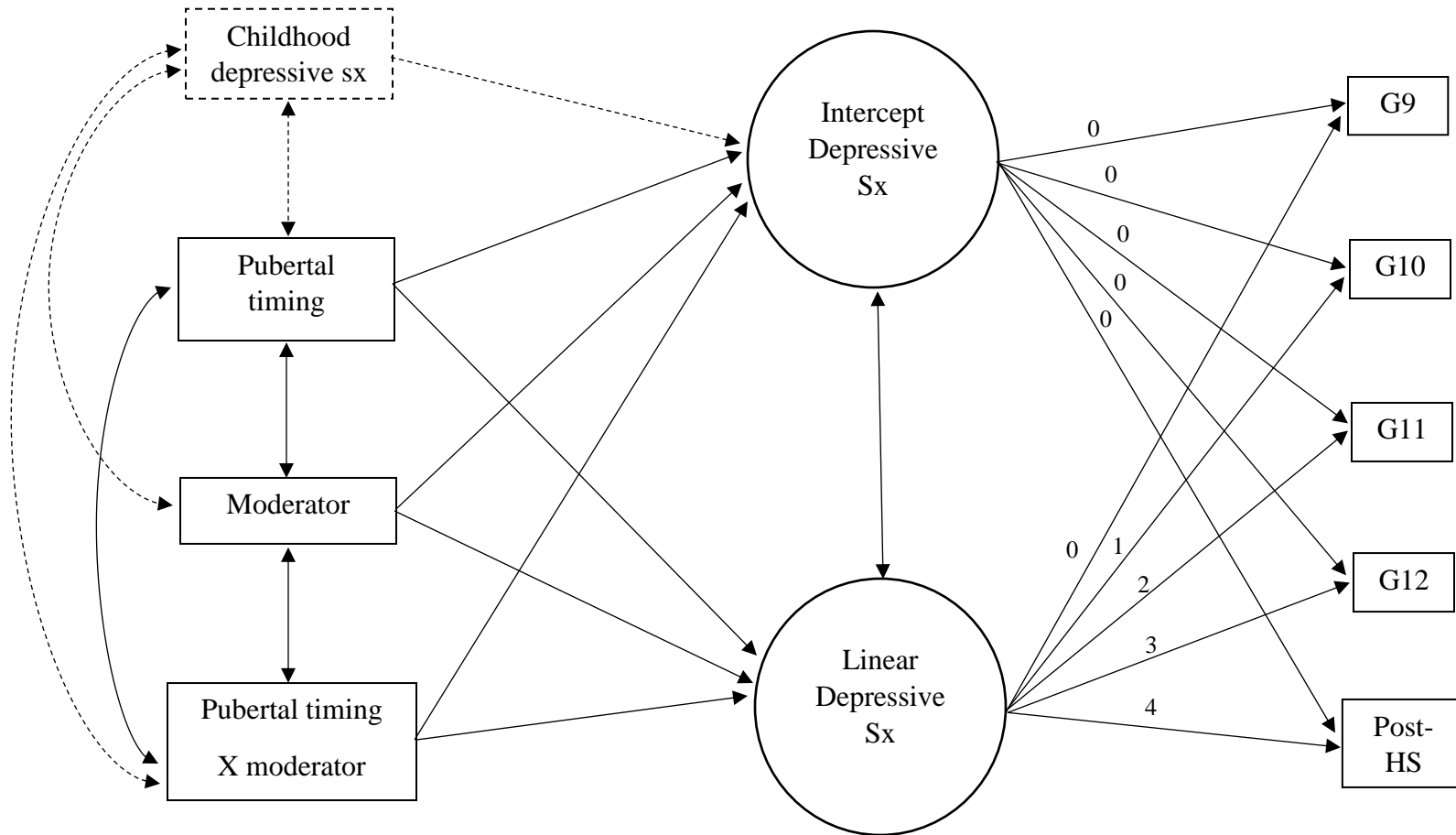


Figure 4. Main effects model in females (Study 1). Standardized path coefficients are presented. * $p < .05$, ** $p < .01$, *** $p < .001$

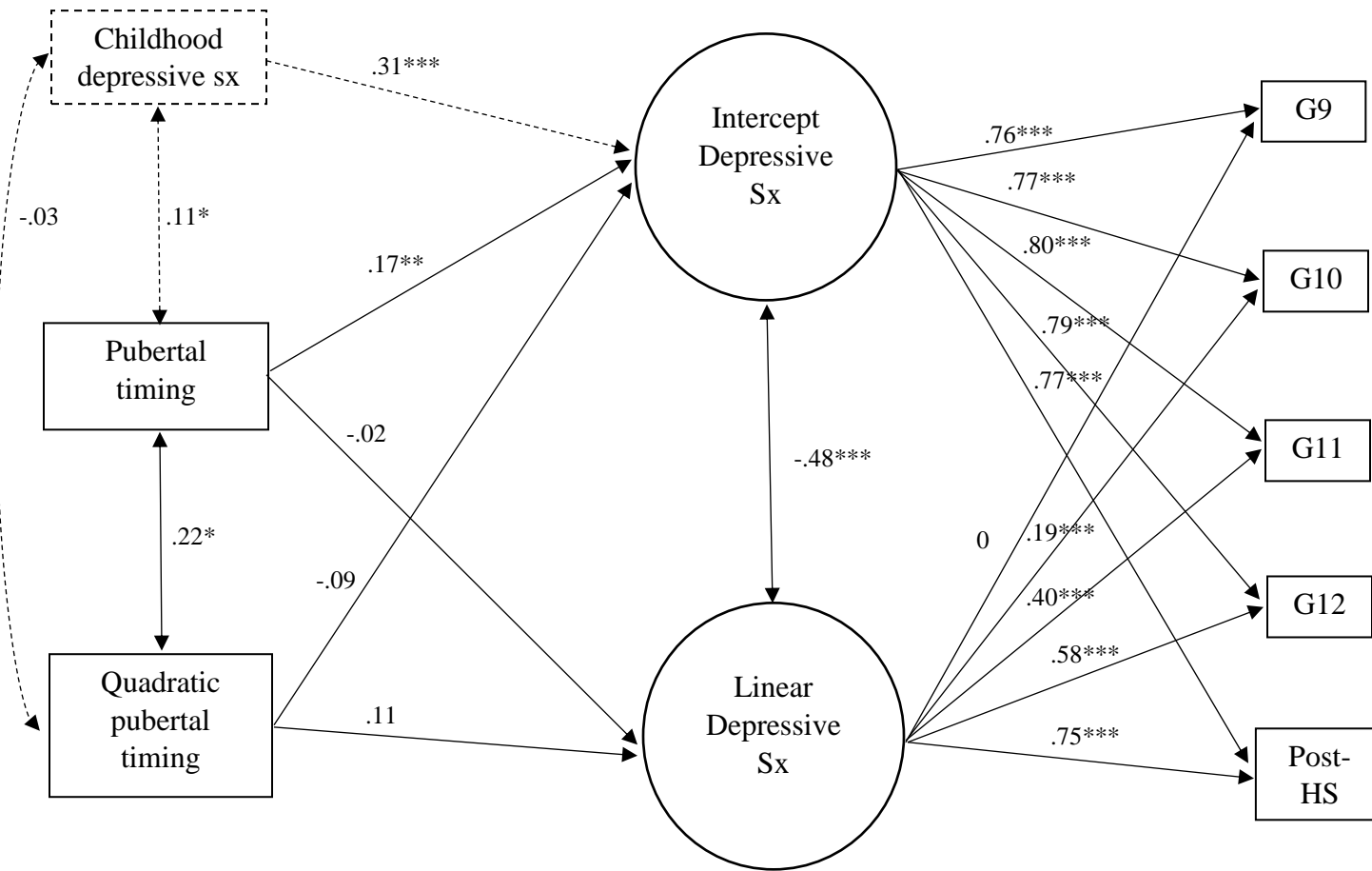


Figure 5. Main effects model in males (Study 1). Standardized path coefficients are presented. * $p < .05$, ** $p < .01$, *** $p < .001$

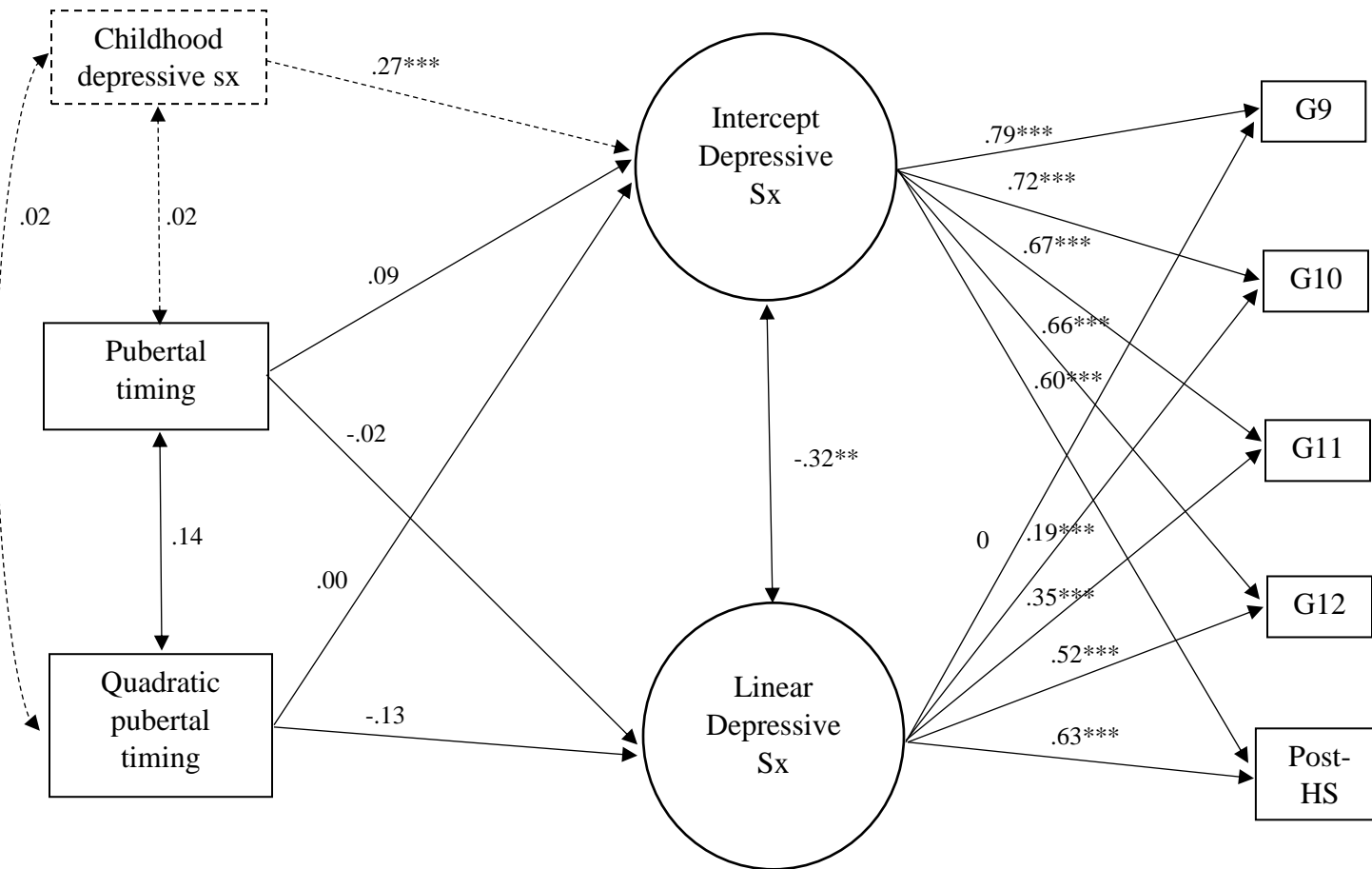


Figure 6. Significant interaction between quadratic pubertal timing and conscientiousness in the prediction of the latent linear slope of depressive symptoms among females from grade 9 through one year post-high school (Study 1). Early and late pubertal timing represent +/- 1.5 SD from the mean. High and low levels of conscientiousness represent +/- 1 SD from the mean.

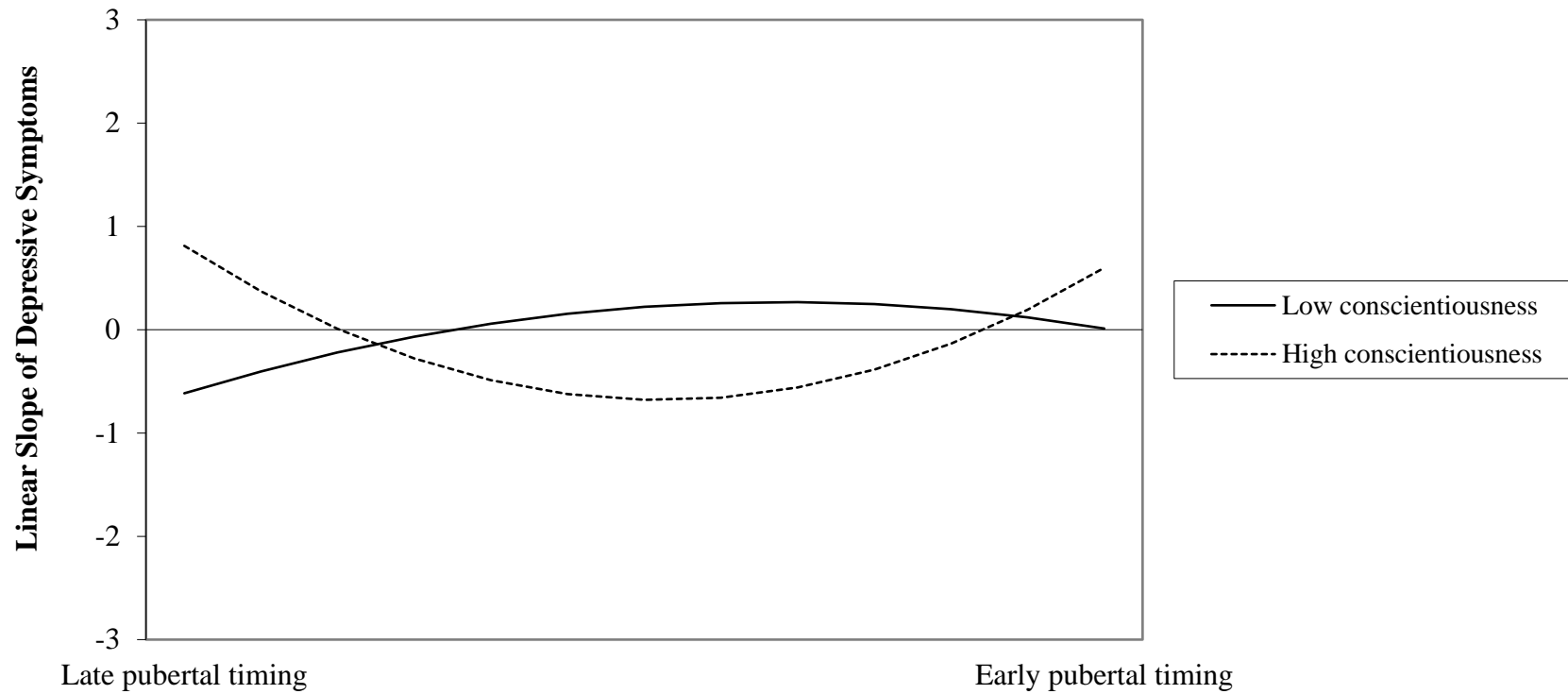


Figure 7. Depressive symptoms among females from grade 9 through one year post-high school by pubertal timing and level of conscientiousness, adjusted for childhood depressive symptoms (Study 1). Early and late maturation represent +/- 1 SD from the mean of pubertal timing. High and low levels of conscientiousness represent +/- 1 SD from the mean.

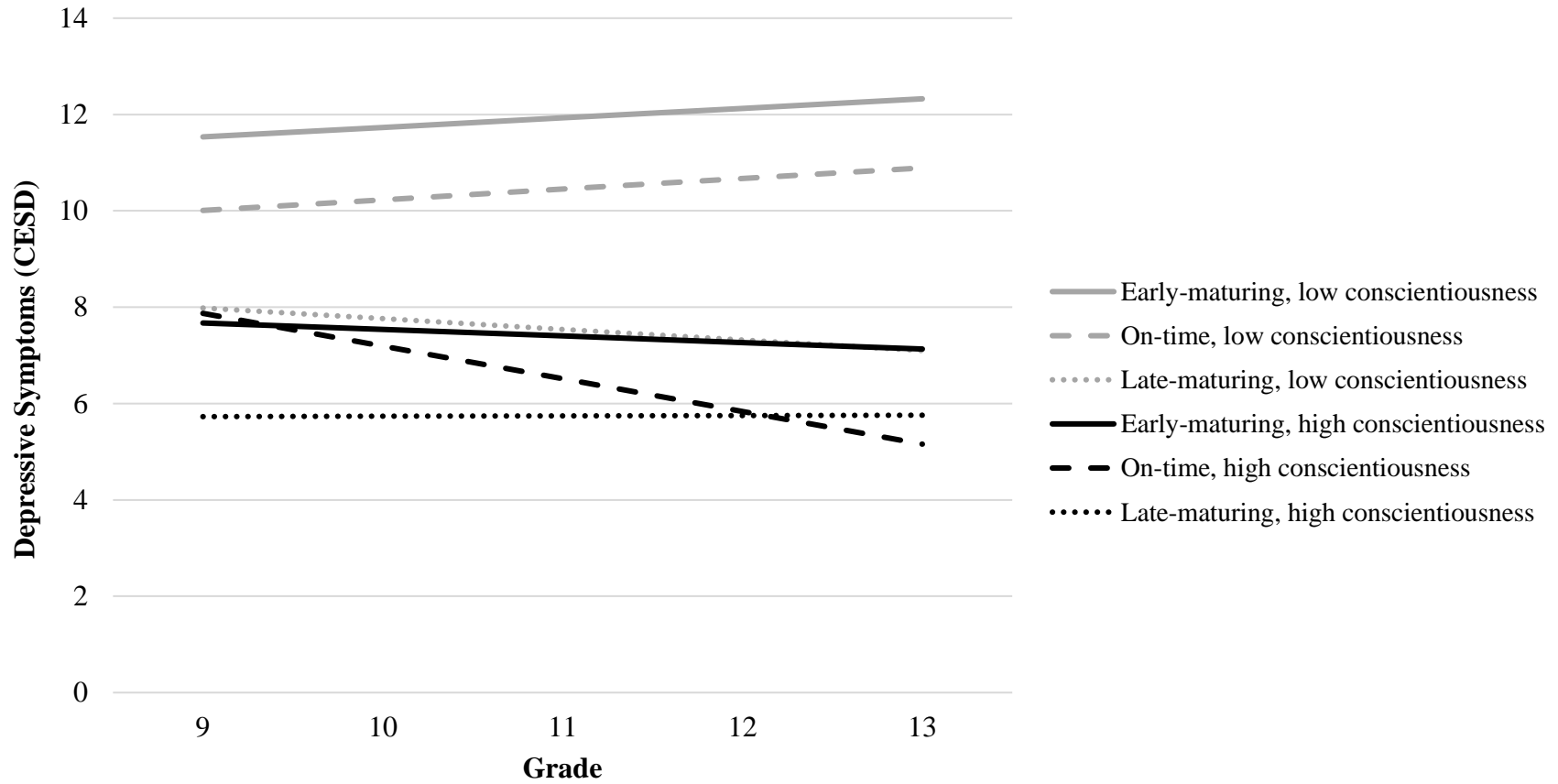


Figure 8. Significant interaction between quadratic pubertal timing and emotional stability in the prediction of the latent intercept of depressive symptoms among females (depressive symptoms in grade 9), adjusted for childhood depressive symptoms (Study 1). Early and late pubertal timing represent ± 1.5 SD from the mean. High and low emotional stability represent ± 1 SD from the mean.

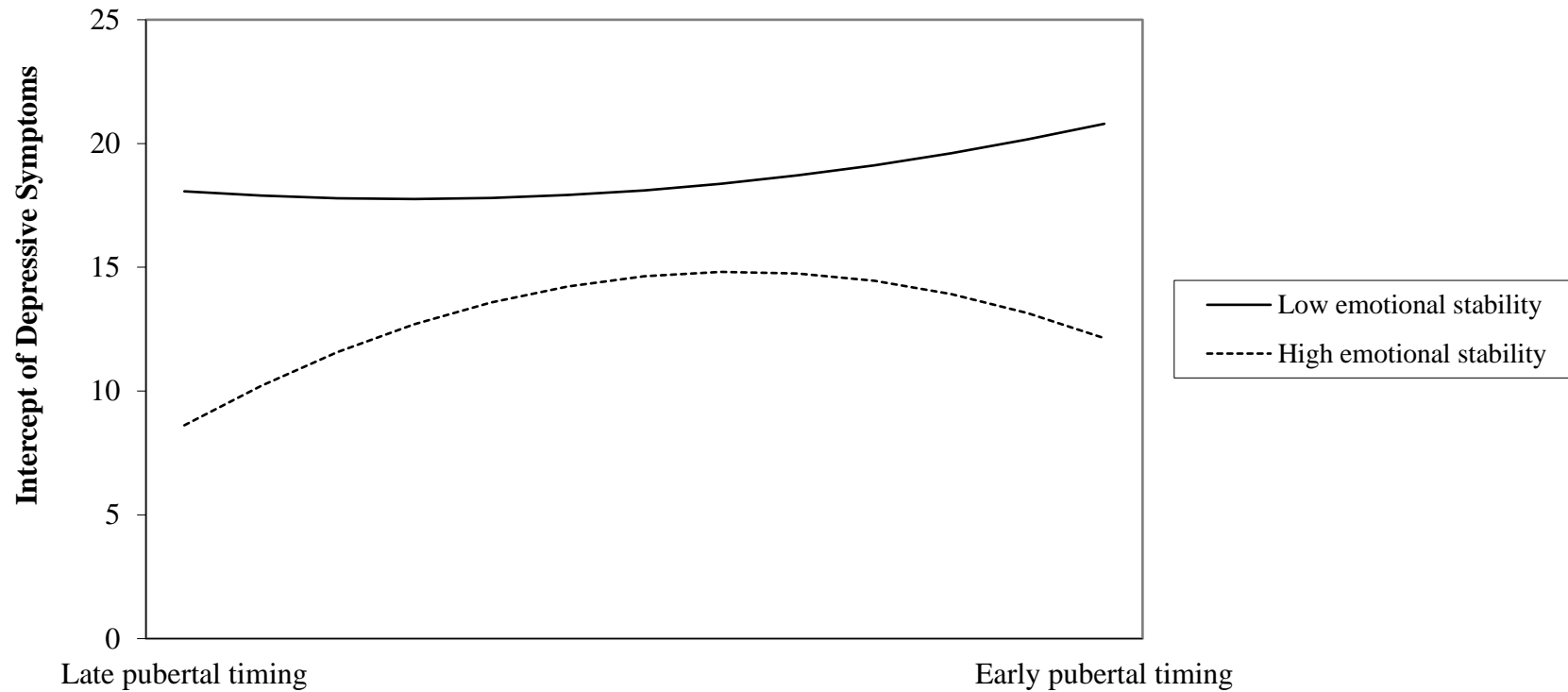


Figure 9. Significant interaction between quadratic pubertal timing and openness to experience in the prediction of the latent linear slope of depressive symptoms among females from grade 9 through one year post-high school (Study 1). Early and late pubertal timing represent +/- 1.5 SD from the mean. High and low levels of openness to experience represent +/- 1 SD from the mean.

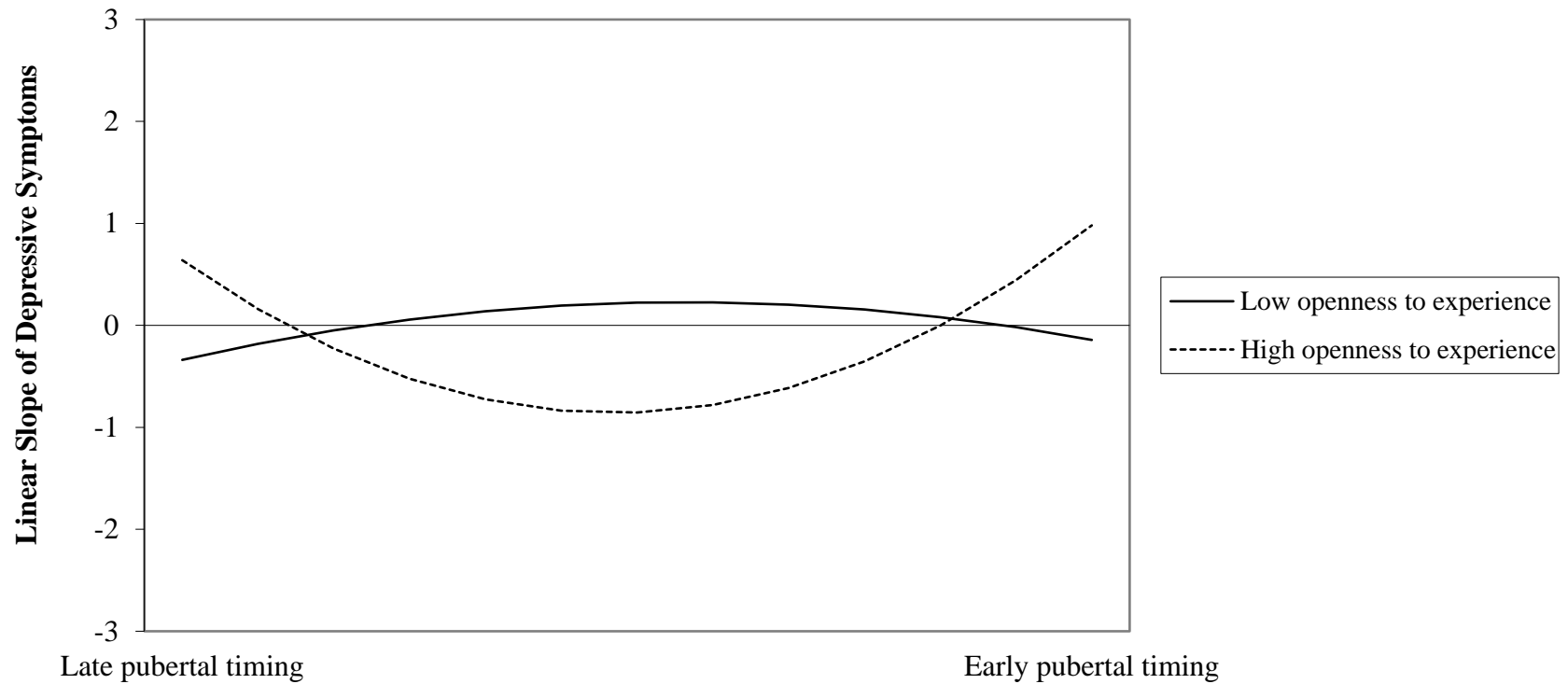


Figure 10. Depressive symptoms among females from grade 9 through one year post-high school by pubertal timing and level of openness to experience, adjusted for childhood depressive symptoms (Study 1). Early and late maturation represent +/- 1 SD from the mean of pubertal timing. High and low levels of openness represent +/- 1 SD from the mean.

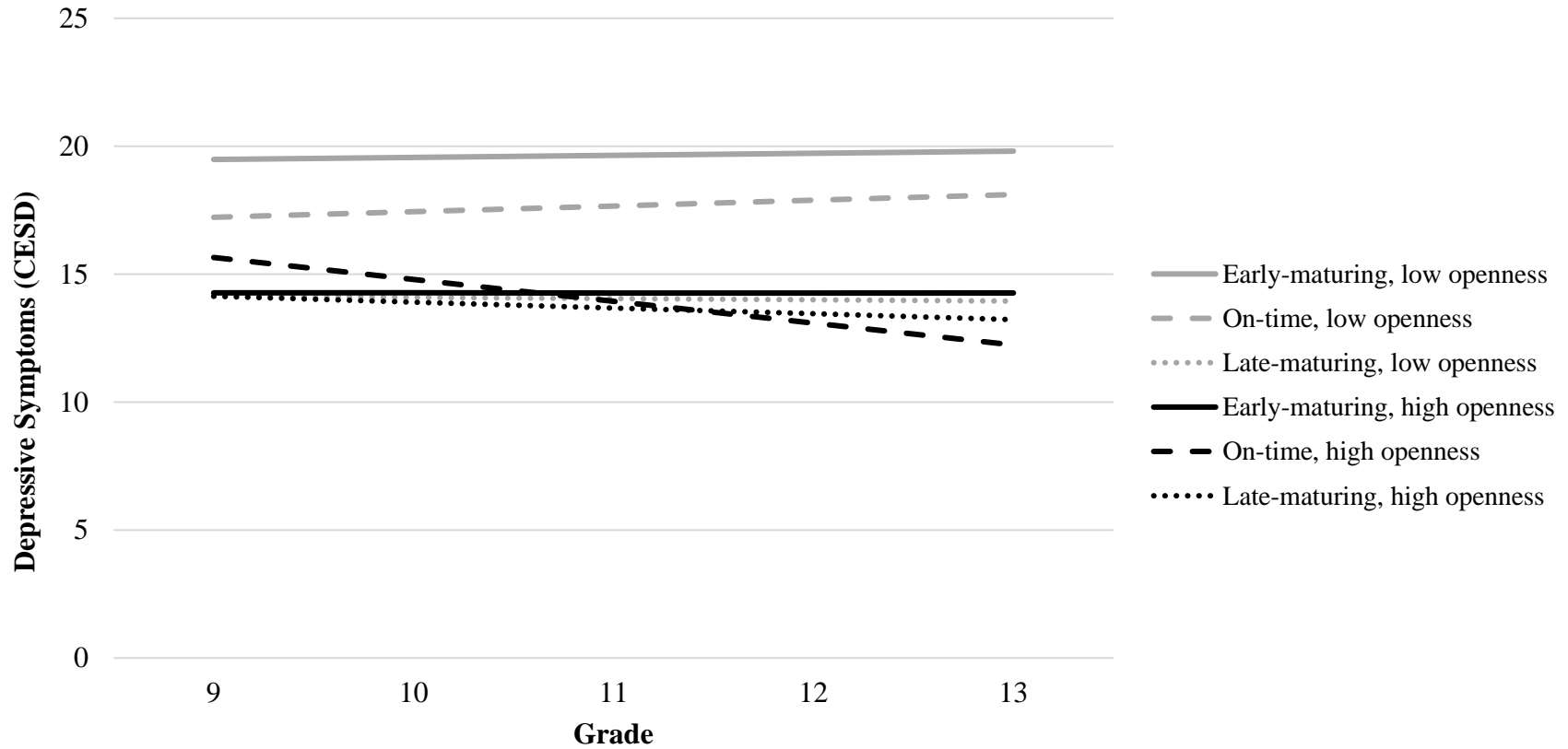


Figure 11. Significant interaction between quadratic pubertal timing and paternal conflict in the prediction of the latent linear slope of depressive symptoms among females from grade 9 through one year post-high school (Study 1). Early and late pubertal timing represent +/- 1.5 SD from the mean. High and low levels of paternal conflict represent +/- 1 SD from the mean.

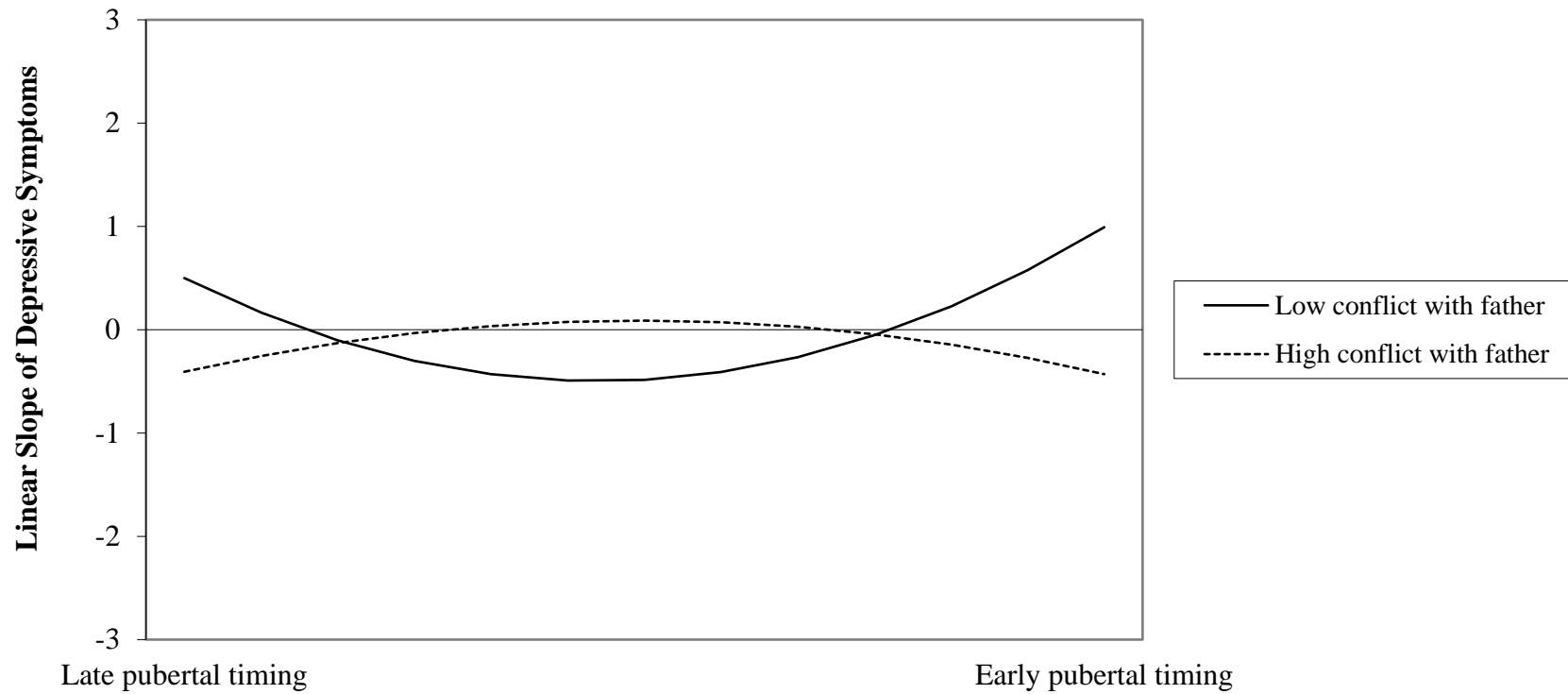


Figure 12. Depressive symptoms among females from grade 9 through one year post-high school by pubertal timing and paternal conflict, adjusted for childhood depressive symptoms (Study 1). Early and late maturation reflect +/- 1 SD from the mean of pubertal timing. High and low levels of paternal conflict represent +/- 1 SD from the mean.

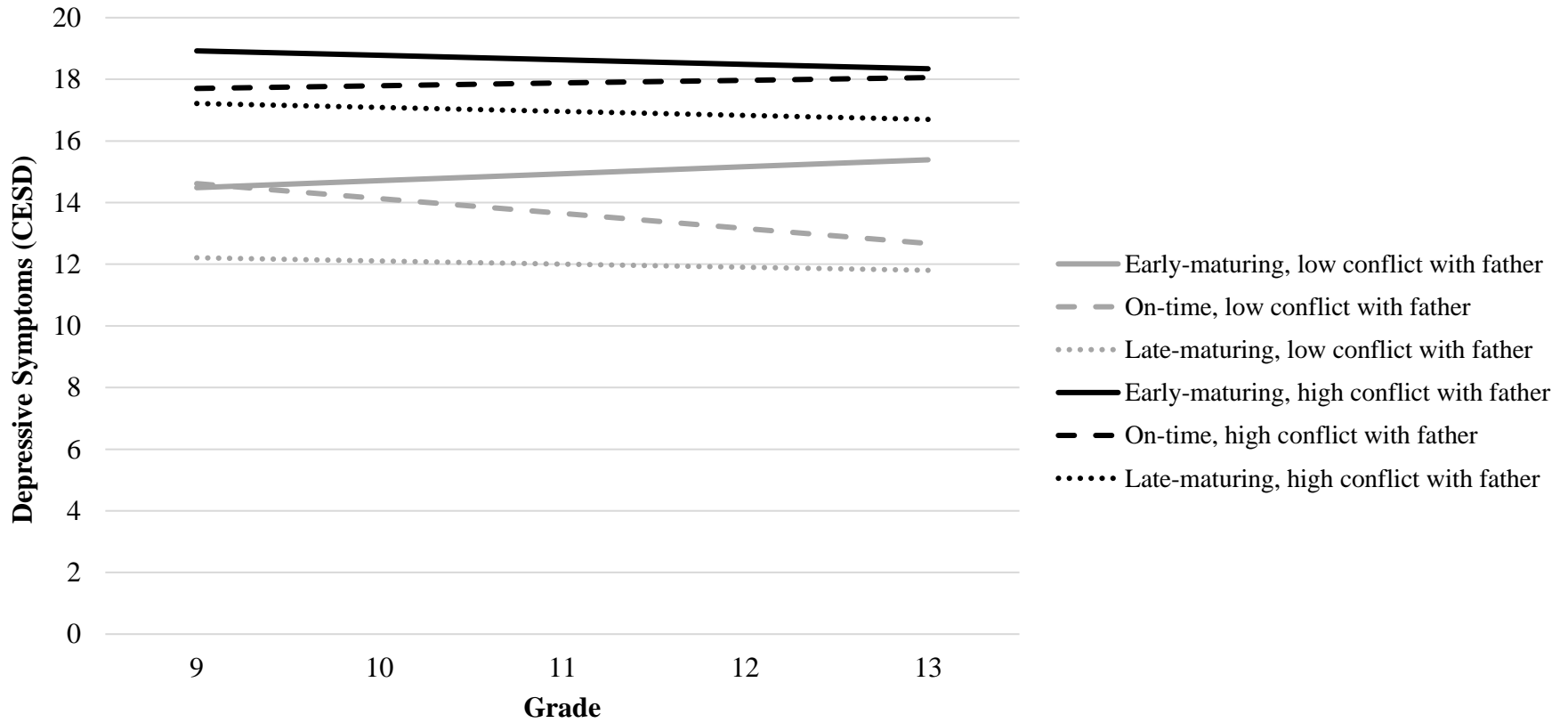


Figure 13. Significant interaction between quadratic pubertal timing and neighborhood stress in the prediction of the latent linear slope of depressive symptoms among females from grade 9 through one year post-high school (Study 1). Early and late pubertal timing represent +/- 1.5 SD from the mean. High and low levels of neighborhood stress represent +/- 1 SD from the mean.

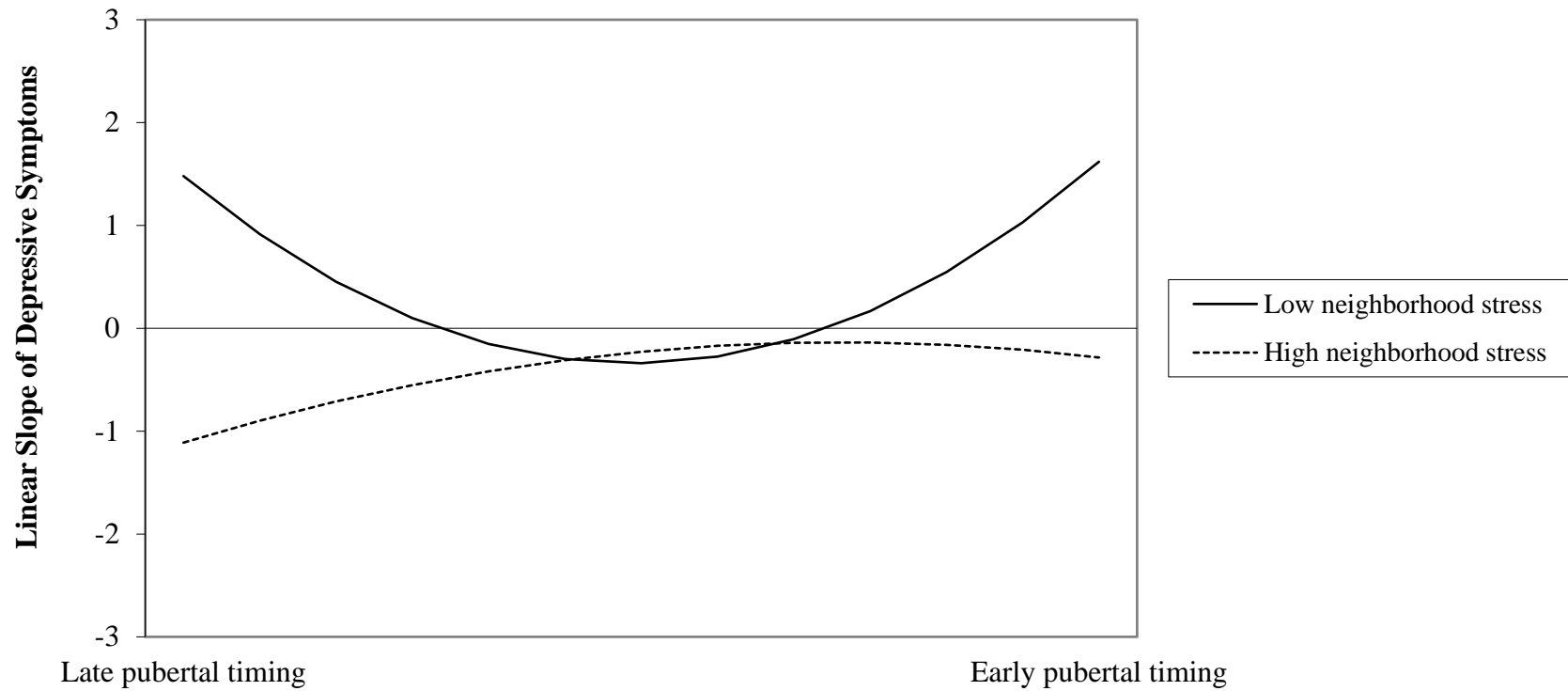


Figure 14. Depressive symptoms among females from grade 9 through one year post-high school by pubertal timing and neighborhood stress, adjusted for childhood depressive symptoms (Study 1). Early and late maturation reflect +/- 1 SD from the mean of pubertal timing. High and low levels of neighborhood stress represent +/- 1 SD from the mean.

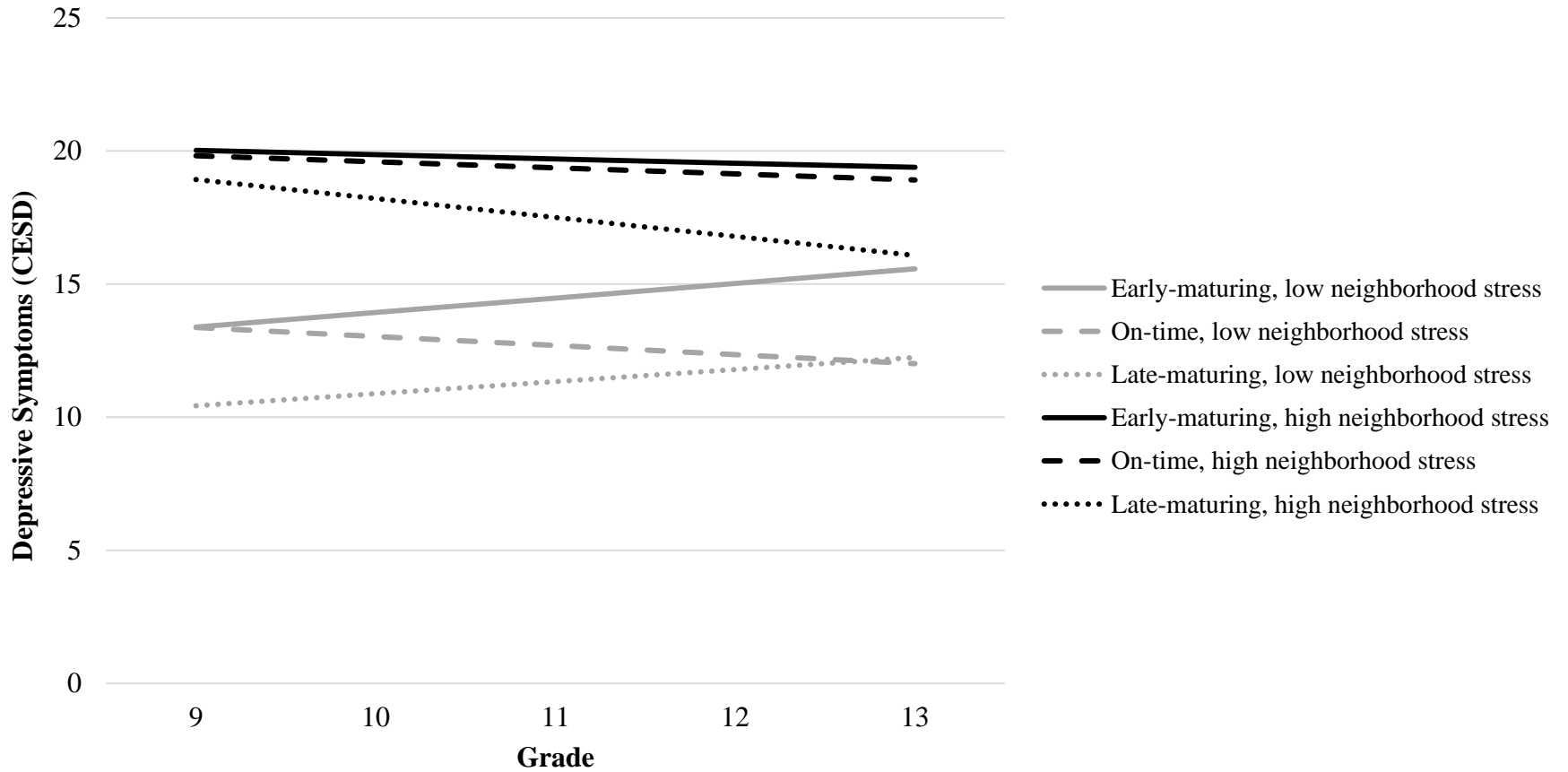


Figure 15. Three-way interaction between gender, pubertal timing, and chronic peer stress at age 15 in the prediction of depressive symptoms at age 15, adjusted for maternal depression history and prior youth depressive symptoms (Study 2). High and low levels of chronic peer stress represent +/- 1 SD from the mean. Early and late pubertal timing represent +/- 1.5 SD from the mean.

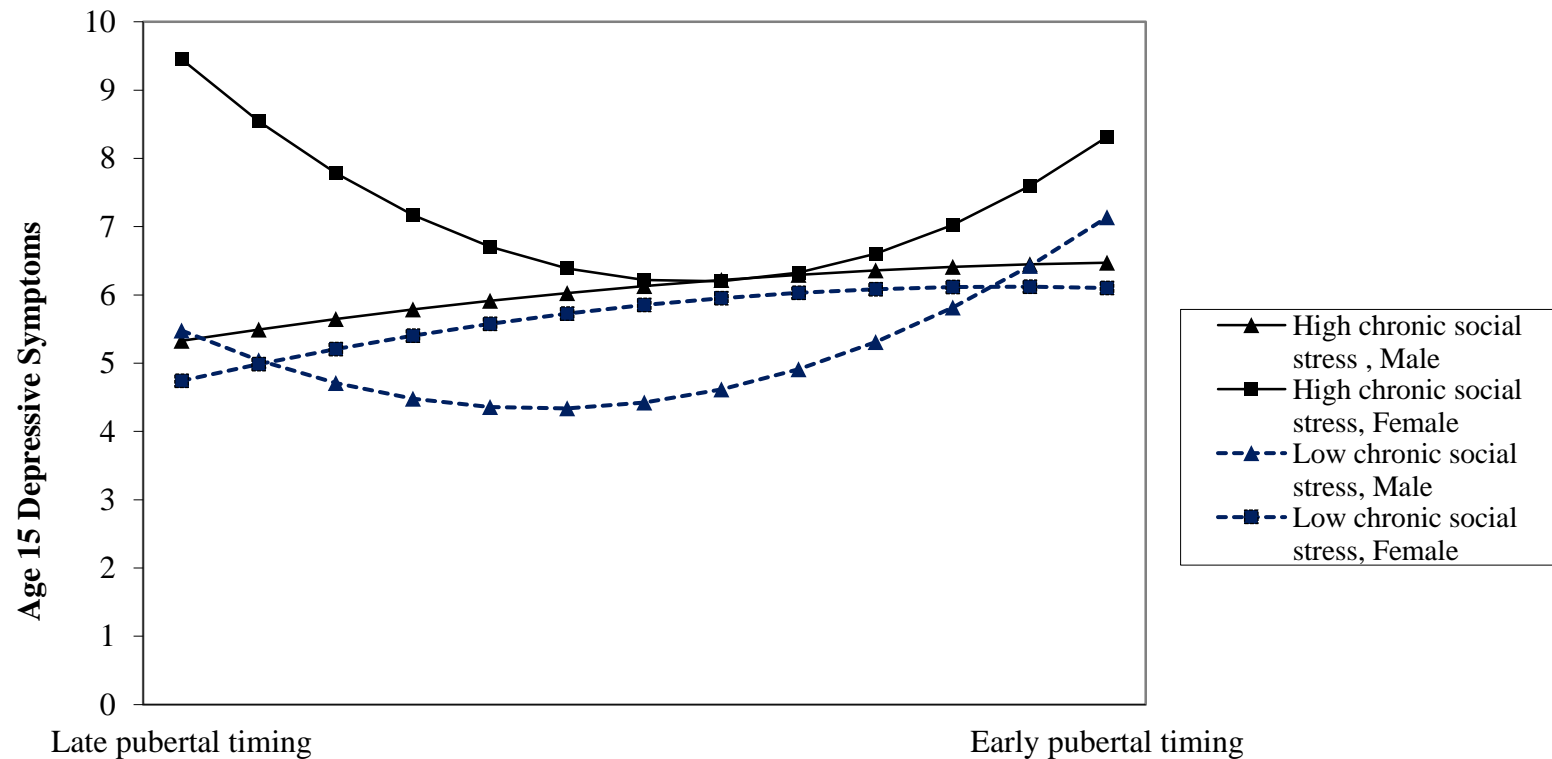


Figure 16. Three-way interaction between gender, pubertal timing, and chronic family stress at age 15 in the prediction of depressive symptoms at age 15, adjusted for maternal depression history and prior youth depressive symptoms (Study 2).

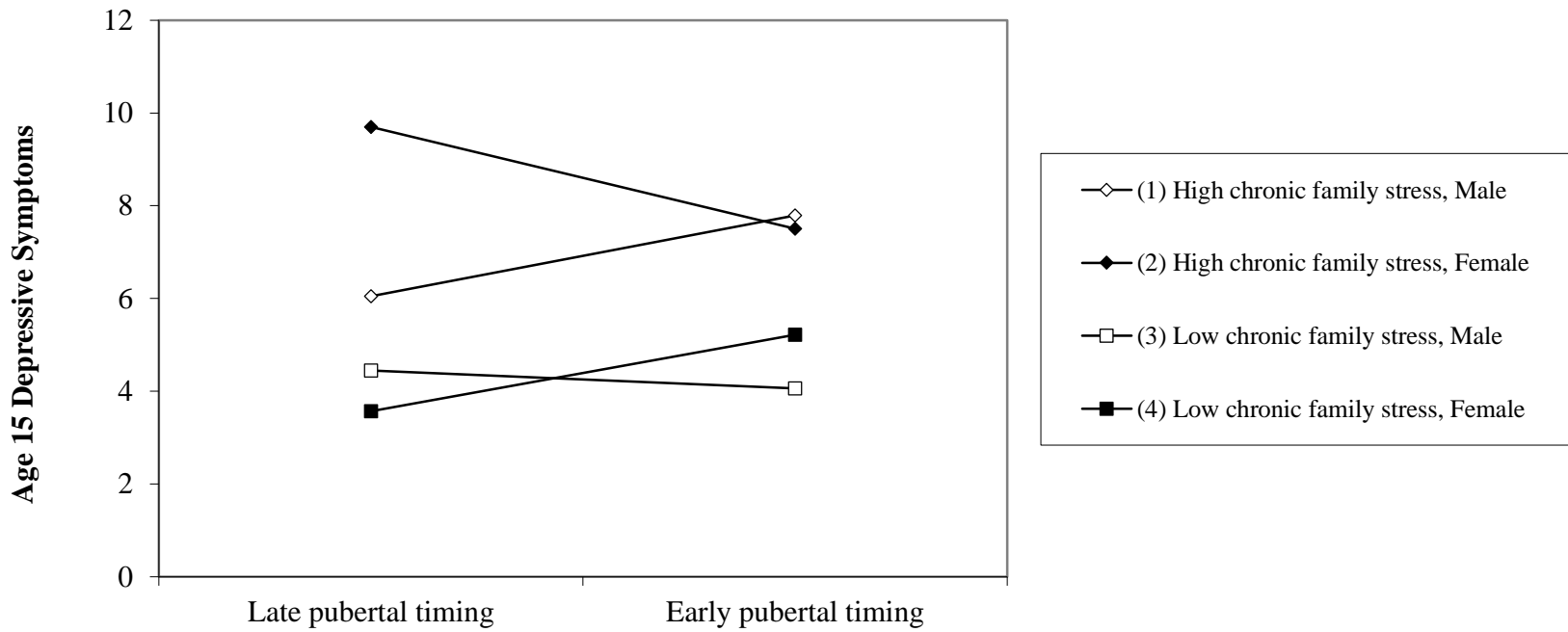


Figure 17. Two-way interaction between pubertal timing and dependent episodic stress at age 15 in the prediction of depressive symptoms at age 15 among males, adjusted for maternal depression history and prior youth depressive symptoms (Study 2).

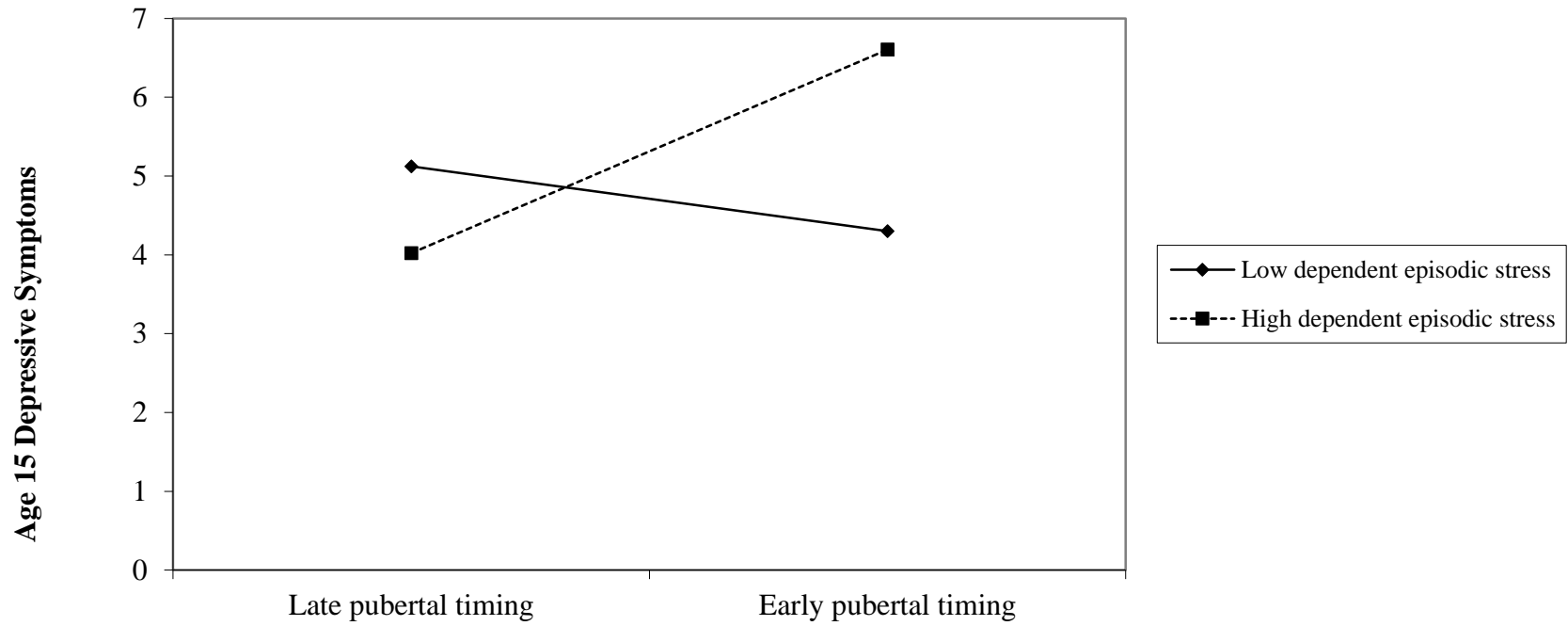


Figure 18. Group differences in depressive symptoms at age 20 by pubertal synchrony, adjusting for maternal depression history, maternal education status, and age 15 depressive symptoms (Study 3). Error bars represent the standard error of the mean.

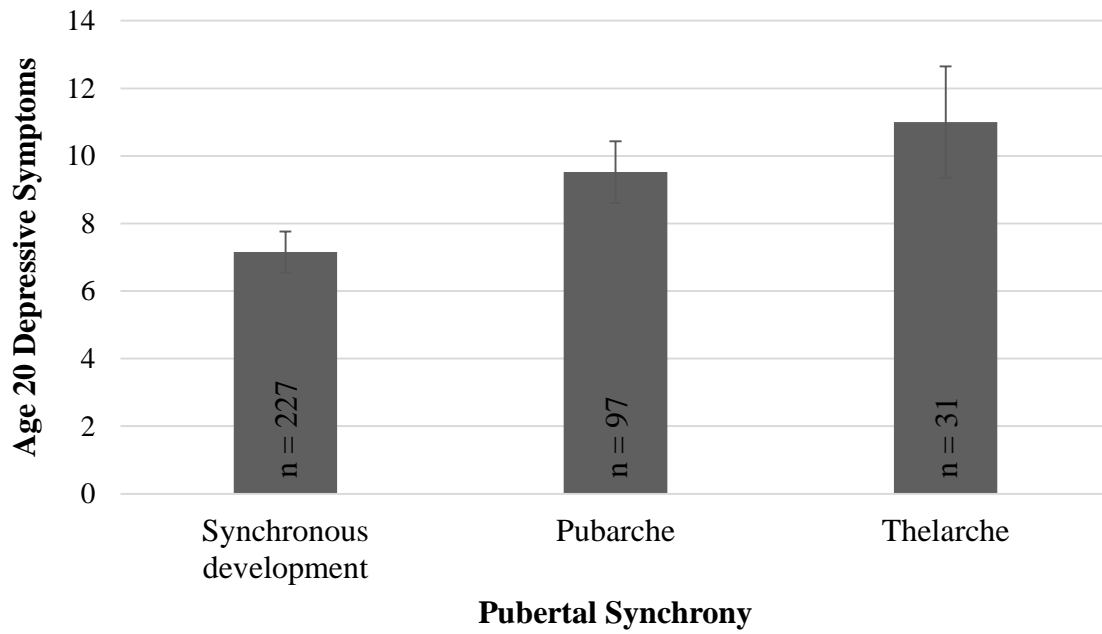
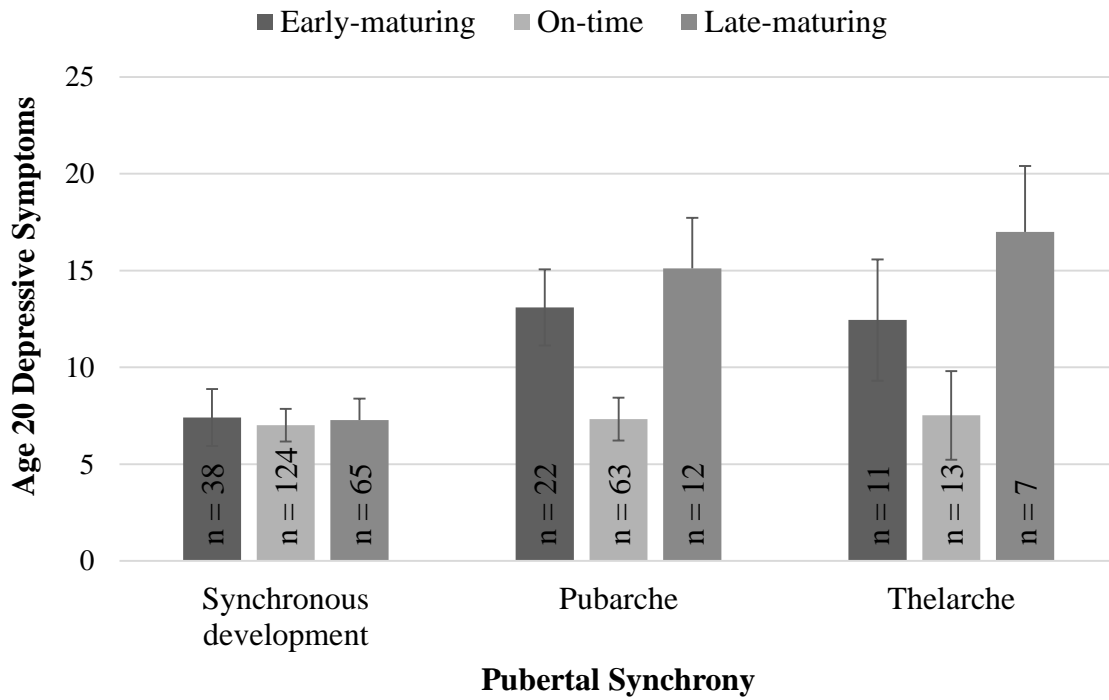


Figure 19. Group differences in age 20 depressive symptoms by pubertal synchrony and pubertal timing, adjusting for maternal depression history, maternal education status, and age 15 depressive symptoms (Study 3). Error bars represent the standard error of the mean.



Appendix: Study 1 Measures

Pubertal Development Scale

All boys/girls change and develop physically, mentally, and emotionally in the process of “growing up.” The growth and development of his/her body is an especially important part of this process. It is normal for different boys/girls to go through this development at different ages. While answering questions about your son’s/daughter’s development, it is important to remember that no one will see these answers other than the researchers doing the study. Please be as honest as possible; honest answers will help us learn about boys/girls your son’s/daughter’s age.

**The following response options were provided and adapted to each question (e.g., Has not yet begun to spurt, Has not yet begun showing any changes). The item assessing the initiation of menstruation among females allowed only two response options: yes or no.*

Has not yet started——Has barely started——Is definitely underway——Seems completed

Questions about Males

1. Would you say his growth in height...
2. And how about the growth of his body hair (e.g., underarm or pubic hair)? Would you say that his...
3. Have you noticed any skin changes, especially pimples?
4. Have you noticed a deepening of his voice?
5. Has he begun to grow hair on his face?

Questions for Females

1. Would you say her growth in height...
2. And how about the growth of her body hair (e.g., underarm or pubic hair)? Would you say that her...
3. Have you noticed any skin changes, especially pimples?
4. Have her breasts begun to grow?
5. Has she begun to menstruate? *Yes/No*

Center for Epidemiological Studies Depression Scale (CESD)

Fill in the circle for the statement which best describes how often you felt this way during the past week.

Rarely or None of the Time——Some or a Little of the Time——Occasionally or a Moderate Amount of Time——Most or All of Time

- a. I was bothered by things that usually don’t bother me.
- b. I did not feel like eating; my appetite was poor.
- c. I felt that I could not shake off the blues even with help from my family or friends.
- d. I felt that I was just as good as other people.

- e. I had trouble keeping my mind on what I was doing.
 - f. I felt depressed.
 - g. I felt that everything I did was an effort.
 - h. I felt hopeful about the future.
 - i. I thought my life had been a failure.
 - j. I felt fearful.
 - k. My sleep was restless.
 - l. I was happy.
 - m. I talked less than usual.
 - n. I felt lonely.
 - o. People were unfriendly.
 - p. I enjoyed life.
 - q. I had crying spells.
 - r. I felt sad.
 - s. I felt that people disliked me.
 - t. I could not get “going.”
-

Coping

Below are some things that people may or may not do when they have problems at school or at home, or something happens that they don't feel good about. Read each one and mark an answer to show what you do. For each one, think about whether you never do this, occasionally, sometimes, often, or always do this.

Never——Occasionally——Sometimes——Often——Always

When I have a problem or something happens that I don't feel good about, I:

- 1. Look for a person who might understand my problem.
 - 2. Find someone to share my problem with.
 - 3. Talk with one of my friends.
 - 4. Organize my life and what I have to do.
 - 5. Try to think of the good things in my life.
 - 6. Try to make my own decisions.
-

Major Life Events Checklist

Have any of the following things happened to you in the past 12 months? If something has happened, answer YES; if it has not happened, answer NO.

Yes——No

- a. I broke up with my boyfriend or girlfriend.
- b. I began dating, going out, or going steady with someone.
- c. A brother or sister left home.

- d. I did not get into a club or sport I really wanted to be involved in.
 - e. There was a change in the amount of money my family has.
 - f. One of my close friends died or became seriously ill.
 - g. One of my grandparents died.
 - h. My mother began working.
 - i. I was suspended from school.
 - j. I became involved in some new religious activities.
 - k. One of my parents became seriously ill or was hospitalized.
 - l. I moved to a new school district.
 - m. One of my parents changed jobs.
 - n. I thought that I or my girlfriend was pregnant.
 - o. One of my parents lost his or her job.
 - p. A new baby was born in our family.
 - q. My parents were divorced or separated.
 - r. One of my parents remarried.
 - s. My mother or father died.
 - t. I became seriously ill or was hospitalized.
-

Abbreviated Offer Self-Image Questionnaire

Please read each statement below. Indicate how well that statement DESCRIBES YOU, using the scale.

Describes me very well——Describes me well——Describes me fairly well——Does not quite describe me——Does not describe me at all

- 1. The picture I have of myself in the future satisfies me.
 - 6. I am proud of my body.
 - 7. I seem to be forced to imitate (or act like) the people I like.
 - 8. Very often I think that I am not at all the person I would like to be.
 - 9. I frequently feel ugly and unattractive.
 - 10. I feel strong and healthy.
-

Modified Self-Perception Profile for Adolescents

Below is a list of statements that describe teens. We want to know what you are like. First read both sides of the statement. Next, choose which side of the statement best describes you. Now, decide whether that is sort of true for you, or really true for you, and fill in one circle that best describes you.

- 1. Some teenagers find it hard to make friends BUT for other teenagers, it is pretty easy.
- 2. Some teenagers feel like they are just as smart as other kids their age BUT other teenagers aren't so sure and wonder if they are smart.

3. Some teenagers are pretty slow in finishing their schoolwork BUT other teenagers can do their schoolwork more quickly.
 4. Some teenagers have a lot of friends BUT other teenagers don't have very many friends.
 5. Some teenagers do very well at their classwork BUT other teenagers don't do very well at their classwork.
 6. Some teenagers are kind of hard to like BUT other teenagers are really easy to like.
 7. Some teenagers have trouble figuring out the answers in school BUT other teenagers can almost always figure out the answers.
 8. Some teenagers are popular with others their age BUT other teenagers are not very popular.
 9. Some teenagers feel that they are pretty intelligent BUT other teenagers question whether they are intelligent.
 10. Some teenagers feel that they are socially accepted BUT other teenagers wished that more people their age accepted them.
-

Abbreviated Mini-Markers Personality Assessment

Using the adjectives below, describe yourself as you honestly see yourself. Compare yourself to others of your same sex and age. Please fill in a circle for each item.

False——Somewhat False——Neither false nor true——Somewhat true——Very true

- a. sociable, outgoing
 - b. tidy
 - c. creative
 - d. energetic
 - e. helpful
 - f. plan ahead
 - g. friendly
 - h. moody
 - i. intelligent
 - j. anxious
-

Abbreviated CESD (Parents)

Listed below are ways that people sometimes feel. Please indicate how many times you felt this way during the past week.

Rarely or None of the Time——Some or a Little of the Time——A Moderate Amount of the Time——Most or All of the Time

- a. I did not feel like eating; my appetite was poor.
- b. I felt that I could not shake off the blues, even with help from my family or friends.
- c. I felt that I was just as good as other people.
- d. I felt depressed.

- e. My sleep was restless.
 - f. I felt happy.
 - g. I felt lonely.
 - h. I felt sad.
 - i. I felt that people disliked me.
 - j. I could not “get going.”
-

Abbreviated Conflict Behavior Questionnaire

Instructions for Youth: For the following questions think back over the last four weeks at home. Read the statements below and decide if it is MOSTLY TRUE or MOSTLY FALSE, first, for your Mother/Stepmother and then for your Father/Stepfather. Fill in T for MOSTLY TRUE or F for MOSTLY FALSE for each parent. Remember: When we ask about parents, we are asking about the parents or adults that you live with and spend the most time with. If you live with one parent, please answer only for that parent.

Mostly True——Mostly False

- 1. We joke around often.
 - 2. We do a lot of things together.
 - 3. I enjoy the talks we have.
 - 4. We never have fun together.
 - 5. We have big arguments about little things.
 - 6. At least once a day we get angry at each other.
 - 7. At least three times a week, we get angry at each other.
-

Community Violence Scale

In the last year, have you witnessed or experienced any of the following? Please fill in the circle below your response.

Never——Once——More Than Once

- 1. Have you seen anyone being beaten, shot or really hurt by someone?
- 2. Have you seen someone get arrested?
- 3. Have you been afraid to go outside, or have your parents made you stay inside, because your neighborhood is dangerous?
- 4. Have you heard guns being shot (other than for hunting)?
- 5. Has someone threatened to beat or attack you?
- 6. Have you been beaten up, robbed or attacked?
- 7. Has someone you know been beaten up, robbed or attacked?

Modified Reynolds Child Depression Scale

Here are some sentences about how you may have been feeling for the past two weeks or so. Read each sentence and decide how often you feel this way. Decide if you feel this way ALMOST NEVER, SOMETIMES, A LOT OF THE TIME, or ALL THE TIME. Fill in the circle under the answer that best describes how you really feel. There are no right or wrong answers. Just choose the answer that tells how you have been feeling for the past two weeks.

Almost Never——Sometimes——A Lot of the Time——All the Time

- a. I feel lonely.
- b. I feel my parents don't like me.
- c. I feel like hiding from people.
- d. I feel sad.
- e. I feel like crying.
- f. I feel that no one cares.
- g. I feel like running away.
- h. I feel like hurting myself.
- i. I feel that other kids don't like me.
- j. I feel upset about things.
- k. I feel I am bad.
- l. I feel that I am no good.
- m. I feel like nothing I do helps anymore.

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