Pubertal Maturation and Depressive Symptoms among Developing Young Women: Examining the Moderating Influence of Parent-daughter Relationship Quality.

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PUBERTAL MATURATION AND DEPRESSIVE SYMPTOMS AMONG DEVELOPING YOUNG WOMEN: EXAMINING THE MODERATING INFLUENCE OF PARENT-DAUGHTER RELATIONSHIP QUALITY

By

Courtney L. Whitt

A DISSERTATION

Submitted to the Faculty of the University of Miami in partial fulfillment of the requirements for the degree of Doctor of Philosophy

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PUBERTAL MATURATION AND DEPRESSIVE SYMPTOMS AMONG
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OF PARENT-DAUGHTER RELATIONSHIP QUALITY

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Given the heightened incidence of depression among girls of pubertal age, this study sought to examine the interrelationships between pubertal advancement, the nature and stability of parent-daughter relationship quality, and depressive symptomology. Grounded in an Integrated Life Course model, and drawing upon diathesis-stress, stress-buffering, and family life cycle perspectives, an essential aim of this research was to identify risks and resources in the parent-daughter relationship that may actualize or attenuate the emergence of depressive symptomology in puberty. Study hypotheses were examined in a diverse, nationally representative sample of girls, ages 12 to 16, who participated in the National Longitudinal Study of Adolescent Health. Measures of pubertal development status, parent relationship quality, and depressive symptomology were completed at two points separated by 12 months. Indices of change in pubertal development status and change in parental relationship quality were examined in relation to change in depressive symptoms. Contrary to study hypotheses, between-wave pubertal advancement was not associated with a concurrent increase in depressive symptomology. Neither baseline parent relationship quality nor relationship quality deterioration interacted with pubertal advancement to predict a rise in depressive symptoms as predicted. Findings are explored in the context of measurement limitations and extant research. Implications and directions for future research are also discussed.
DEDICATION

What has largely been an exercise of learning, overcoming, and mastering the self has also been an exercise of reflection, admiration, and gratitude for others. To my “others” – my loyal and loving family and friends who keep me grounded. For each of you – your unconditional acceptance, support, and humor – I am so grateful and owe so much. I love you immensely.
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A special acknowledgment is owed to Dr. Robert McMahon who chaired this dissertation research and whose contribution to my evolution as a clinician and researcher over the years has been profound. Thank you for your patience and understanding along the way, as well as for your wisdom and insights. I would also like to acknowledge Drs. Lydia Buki, Lissette Perez – Lima, and Robert Halberstein. I am grateful for each of you and your time, perspectives, and willingness to take a chance on me and serve on this committee. Acknowledgment is also owed to all the faculty and staff of the Counseling Psychology program and EPS department who have contributed to my learning and answered countless questions and provided a great deal of assistance over the years.

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CHAPTER I
AN INTRODUCTION AND REVIEW OF THE LITERATURE

Introduction

An estimated 11.7% of adolescents in the U.S. are reported to meet diagnostic
criteria for a DSM – IV depressive disorder (i.e., Major Depressive Disorder, Dysthymic
Disorder) at some point within this developmental period (ages 13 to 18), with 8.7%
exhibiting severe impairment (Merikangas et al., 2010). An examination by sex reveals
adolescent girls disproportionately carry the burden (lifetime prevalence: 15.9% females,
7.7% males, 11.7% combined-sex). Other epidemiological surveys have reported
adolescent girls to not only be nearly three times more likely than adolescent boys to
experience a major depressive episode (past year point prevalence rate: 12.4% females,
4.3% males), but also be three times more likely to experience a major depressive episode
falling within a severe impairment range (past year point prevalence rate: 9.2% females,
2.9% males), (Substance Abuse & Mental Health Services Administration [SAMHSA],
2009). Such a trend has been documented across cultures, observed in both community
and clinical population samples, and has been noted to extend to non-clinical, depressed
mood states and associated depressive symptomology (Allen, Barrett, Sheeber, & Davis,
2006; Cyranowski, Frank, Young, & Shear, 2000; Parker & Brotchie, 2010; Wilhelm,
Parker, & Asghari, 1998). These disparate rates do not exist from the outset of life,
however, as comparable rates of depressive disorders are found throughout childhood
(Allen et al., 2006); rather, sex differences have been found to arise in early adolescence
with the heightened incidence of depression among teen girls, and continue “for the next
35 to 40 years” of the human life cycle (Cyranowski et al., 2000, p. 21).
Aggregated findings from the National Surveys on Drug Use and Health, 2008 to 2010, indicate that an average of 1.4 million (12%) teen girls experienced an episode of major depression within the past year (SAMSHA, 2012). Relative to 12-year old females, young women 15 years of age were nearly three times more likely (15.2% vs. 5.1%) to experience a past year major depressive episode. Rates of depression were steadily higher across each advancing age group during early and middle adolescence. Differences were less sharp for young women in the later stages of adolescence (i.e., 12: 5.1%, 13: 8.5%, 14: 11.6%, 15: 15.2%, 16: 16.1%; 17: 14.6%). Rates of major depression and age trends for adolescent females from minority racial and ethnic backgrounds have been found to parallel those of non-Hispanic White teen girls (Rohde, Beever, Stice, & O’Neil, 2009). Overall, these epidemiological findings draw attention to, and raise questions concerning, developmental factors influencing the mental health of female adolescents.

It has been suggested that this increasing rate of depression among females during adolescence may be linked to pubertal development (Graber, Petersen, & Brooks-Gunn, 1996). Despite age and pubertal maturation status being closely intertwined and highly statistically correlated, there is some evidence to suggest that differences in rates of depression may be better explained by pubertal developmental status than age (Angold, Costello, & Worthman, 1998). In one large ($n = 2,927$), two-nation study (Washington State, U.S.; Victoria, Australia), Patton and colleagues (2008) found that 26% percent of girls, ages 10 to 15, exhibit high levels of depressive symptomology. An examination by pubertal maturation status revealed that young women in mid- and later stages of puberty had 1.4 (95% CI: 1.0 – 1.9) and 2.5 (95% CI: 1.7 – 3.6) greater odds, respectively, of reporting high levels of depressive symptoms relative to those in early stages of
development, after controlling for age and grade. Specifically, 14% percent of young women in early, 20% in mid-, and 33% in late stages of puberty reported elevated depressive symptomology. Unfortunately, data pertaining to pubertal maturation status is not routinely collected in nationwide epidemiological surveys; therefore, U.S. prevalence rates of clinically diagnosable depressive disorders stratified by pubertal development level are not readily available.

Findings from cross-sectional analyses of pubertal stage differences in levels of depressive symptomology or rates of depressive disorders among young women have been mixed (Angold & Costello, 2006; LeResche, Mancl, Drangsholt, Saunders, & Von Korff, 2005; Oldenhinkel, Verhulst, & Ormel, 2011). Results from longitudinal analyses have been less inconsistent, and provide evidence to suggest increases in depressive symptomology parallel advancing maturation (Angold et al., 1998; Paikoff, Brooks-Gunn, & Warren, 1991). For instance, controlling for age and grade level, Patton et al. (2008) found significantly higher rates of depressive symptomology 12 months later among young women who advanced in maturation (i.e., early to mid-, mid- to late) relative to those whose development was stable (i.e., remained early, mid-, or late pubertal) across study waves. The incidence of high depressive symptomology, suggestive of a potentially diagnosable depressive disorder, increased by 4% among those transitioning from early to mid-stages of development (i.e., from 13% to 17%), and by 8% among those advancing from mid- to late-stages of puberty (i.e., from 21% to 29%). Extensions of these analyses also linked advancing maturation with the onset and persistence of depressive symptoms.


Understanding the Association Between Puberty and Depression

A number of theories and empirical investigations have been generated across a multitude of disciplines in an effort to understand the association between puberty and depression among developing young women. This task has not been a simple one. The confluence of biological, psychological, and social changes occurring as a result of, and over the course of puberty present challenges to researchers seeking to define, measure, and isolate the specific features of pubertal development that may place teen girls at risk (Graber et al., 1996; Negriff, Fung, & Trickett, 2008).

Drawing upon theory and research from biomedical, behavioral, and social sciences, an Integrated Life Course model provides a broad framework through which the relation between advancing pubertal maturation and depression can be meaningfully conceptualized and examined (Harris et al., 2009; Harris, 2010; Singer & Ryff, 2001). From this perspective, health and well-being are multi-faceted and multi-determined. Both influence and are influenced by reciprocally interacting biological, behavioral, and socio-contextual forces throughout the lifespan. These influences may be non-linear and variable over time and context (Figure 1). The fact that there appears to be an elevated occurrence of depression at a time when developing young women are undergoing complex changes in these very domains provides further credence to an integrative developmental model for understanding factors that might influence the probability of depression emerging among teen girls in pubertal transition.

As discussed, puberty is a complex process of biological, psychological, and social origin and significance (Cyranowski et al., 2000; Graber et al., 1996; Slater, Guthrie, & Boyd, 2001). The fluctuation and release of pubertal hormones stimulates
physiological and anatomical changes characteristic of puberty (Colvin & Abdullatif, 2012; Graber et al., 1996). Reproductive organs further develop and mature, signaling the initiation of menstruation, and thus the capacity for reproduction. Fat redistributes, breasts develop, and the body becomes more curvaceous and womanly. Neuroendocrine processes give rise to further neural developments in the prefrontal cortex and limbic system of the brain, impacting the experience, expression, and regulation of cognition, emotion, and behavior (Angold, Worthman, & Costello, 2003; Spear, 2000, 2008; Steiner, Dunn, & Born, 2003). In turn, these physiological, neurological, and structural advances, occurring in a broader sociocultural context, may have meaningful implications for young women’s self-image and worth, value and needs in relationships, place and security in the world, and expectations and goals for the future.

The many changes of puberty may open young women up to a number of new and exciting opportunities, experiences, and perspectives; however, they too can be the source of considerable stress (Caspi & Moffitt, 1991; Cyranowski et al., 2000; Dahl, 2004). Stress does not inevitably lead to depression; however, when it accumulates, persists, or exceeds one’s perceived ability to cope, thoughts and feelings of helplessness and hopelessness and a loss of self-esteem and efficacy may ensue (Cohen & Wills, 1985). These thoughts and feelings may pose a considerable risk for depression, and are actually at the core of depression from a cognitive perspective (Abramson, Metalsky, & Alloy, 1989; Beck, 1974; Henkel, Bussfeld, Möller, & Hegerl, 2002). Further, puberty-driven biological states and changes may leave developing young women “highly sensitive” to environmental stressors. While physiological and neurological changes may not “cause” depression, they may set “stage” for its development (Frank & Young, 2000). It may be
only in the presence or absence of psychological and sociocultural stressors and resources that symptoms of depression would emerge (Dahl, 2004; Parker & Brotchie, 2010; Susman, Dorn, & Chrousos, 1991).

The Parent-Daughter Relationship: Opportunities and Risks

The family system has been described as “the natural context within which to frame individual identity and development” (Carter & McGoldrick, 1999, p. 1). Perhaps most critical within the family system and to individuals’ identity and development are parent figures. Their contribution may be especially important to consider at this developmental juncture, and may provide insight into specific risk or protective factors that materialize or modulate developing young women’s vulnerability to depression. The extensive body of research establishing the significance of the parental relationship along a number of dimensions (e.g., control and autonomy, communication and conflict, warmth and acceptance, etc.) as it relates to depression strengthens the argument for examining parent-daughter relationships and the function they may serve in protecting or placing developing young women at risk for the development of depression over the course of puberty (Hughes & Gullone, 2008; Papini & Roggman, 1992; Sagrestano, Paikoff, Holmbeck, & Fendrich, 2003; Stark, Banneyer, Wang, & Arora, 2012).

Ideally, as nurturers, protectors, educators, and the primary transmitters of culture, mothers and fathers play a crucial role in socializing their maturing daughters to the physical, relational, and cultural demands of womanhood (Bronstein, 2006; Crouter, Manke, & McHale, 1995; Papini & Sebby, 1987; Petersen, 1988). Their love, support, and guidance may be key in helping their maturing daughters adjust to the many rapid, intersecting, and at times confusing, changes associated with pubertal development.
Empirical findings have consistently established the protective function of high quality parental relationships in relation to a number of behavioral, psychological, educational, and health outcomes, including depression (Beardslee, Gladstone, & O’Connor, 2012; Cohen & Wills, 1985; Whiffen & Demidenko, 2006). Given this, and in considering the relatively high probability of risk for developing depression during puberty, perhaps depressive symptomology is more likely to emerge in adolescent young women who do not have a high quality parent-daughter relationship to attenuate, or “buffer” against, the many changes, stressors, and vulnerabilities associated with advancing development.

While the quality of the parent-daughter relationship may be a protective factor in assisting female adolescents in adjusting to the many changes, stressors, and potential vulnerabilities brought upon by pubertal maturation, this relationship may change and evolve over the course of development. During adolescence, and perhaps “signaled” by changes of puberty, family relationships must be renegotiated and evolve in order to meet the psychosocial needs, developmental milestones, and task resolutions necessary for optimal functioning and well-being (Erikson, 1968; Gilligan, 1982; McGoldrick & Carter, 1999; Papini & Sebby, 1987; Petersen, 1988). “Boundaries shift, psychological distance among members changes, and roles within and between subsystems [must be] redefined” (Carter & McGoldrick, 1999, p. 1). The exact nature of these adaptations may vary substantially, and are dependent upon a number of individual, family, sociocultural, and historical factors (McGoldrick & Carter). Although these developmental transitions are viewed as “stressors” within the Family Life Cycle model, they do not inevitably lead to distress or turmoil (Carter & McGoldrick). In fact, these adaptations may cause little disruption or change in the affective quality of the parent-adolescent relationship, and
may even be quite favorable, providing new opportunities for connection, growth, and support to effectively cope with maturational stressors and changes (Preto, 1999). It is perhaps when there is deterioration in emotional connection and communication that difficulties ensue (De Genna, Larkby, & Cornelius, 2011; Paikoff et al., 1991; Petersen, 1988; Preto, 1999; Steinberg, 1988).

Deterioration in the quality of the parent-daughter relationship over the course of pubertal development may pose a significant risk for depression. Specifically, in the context of puberty-driven biological vulnerabilities, losses within the parent-daughter relationship, such as the loss of emotional connection, acceptance, security, or the “stress-buffering” influence of the parental relationship, for example, may be the stress that places young women at risk for depression. Despite the extensive body of research examining the association of parental relationship quality and depression, as well as the important role of parents in adolescent young women’s development and adjustment, surprisingly few empirical studies have examined interactions between pubertal development, the parent-daughter relationship, and depression.

The Present Study

While a number of studies have documented an association between pubertal development status and indicators of depression (Angold & Costello, 2006; Patton et al., 2008), findings within the literature base have mixed, and studies have often been limited to small, geographically restricted, and racially and ethnically homogenous samples (Graber et al., 1996; Negriff et al., 2008). Further, longitudinal designs have been scarce, and there has been a virtual absence of studies examining parallel changes in development and depressive symptomology, therefore limiting the ability of researchers
to draw meaningful conclusions regarding the nature of the association between female pubertal maturation and depression. The present study is an initial step towards bridging this gap in the literature.

Negriff and colleagues (2008) argued that research on pubertal development needs to move beyond documenting associations between pubertal maturation and depressive symptoms, to investigating factors that underlie and/or moderate these associations. Grounded in an Integrated Life Course model of depression, and drawing upon diathesis-stress, stress-buffering, and family life cycle (Carter & McGoldrick, 1999; Cohen & Wills, 1985; Parker & Brotchie, 2010; Susman et al., 1991) perspectives, this research will also examine whether the quality of the parent-daughter relationship moderates the risk for depression among young women in pubertal transition. Further, this study will explore whether a deterioration in parent-daughter relationship quality over the course of pubertal transition moderates the association between advancing maturation and increasing depressive symptoms. Examining these associations among a large, nationally representative sample is essential for generalizing findings concerning protective resources and developmental and familial risks to the U.S.’s diverse female adolescent population.

Review of the Literature

This review will provide a theoretical and empirical framework within which proposed associations between female pubertal maturation, parent-daughter relationship quality, and depressive symptoms among adolescent young women will be conceptualized and examined. First, an overview of the female pubertal development process will be provided to summarize and contextualize the physiological,
morphological, and psychosocial changes of puberty. Empirical findings from studies examining associations between indicators of female pubertal development and depression will then be examined, and a transtheoretical framework for understanding these associations will be discussed. Next, and central to the present study, the role of parent-daughter relationships will be examined. Theory and findings pertaining to the protective function and potential risk of the parent-daughter relationship will be presented as it relates to depression in puberty. Justification for the present research will be outlined, and specific study hypotheses will be detailed.

Female Pubertal Development – An Overview

The biological nature of puberty and its development. While socially and culturally its definition may vary, as a biological event, puberty, simply stated, is a “period of biologic transition from childhood to adulthood” – two life phases distinguished, primarily, by reproductive potential (Colvin & Abdullatif, 2012, p. 1). While its function is simple, the transitional process of puberty itself is quite an “intricate and coordinated physiological phenomenon,” initiating “profound changes … throughout the body” (p. 13) and, ultimately, in the case of young women, actualizing the capacity to conceive, carry, and nurture offspring. Pubertal transition is initiated by the activation of the hypothalamic-pituitary-gonadal (HPG) axis in which small amounts of gonadotropin releasing hormone (GnRH), luteinizing hormone (LH), and follicle-stimulating hormone (FHS) are gradually released nocturnally by the hypothalamus and pituitary gland, acting in concert over the course of puberty to stimulate the physiological and anatomical changes characteristic of puberty. Most notably, the release of these hormones triggers the further structural development of the ovaries, as well as the stimulation of ovarian
estrogen and progesterone production, over time leading to ovulation and the first menstruation (Angold et al., 2003; Colvin & Abdullatif, 2012; Reiter & Grumbach, 1982). Menarche is often viewed as the hallmark of female pubertal development; however, it is only one of many influential changes (Graber et al., 1996).

In addition to ovulation and menstruation, the pelvis will also widen and tilt, allowing for the carrying of a child. Fat will cluster in the hips, thighs, and buttocks, creating a “store” of fats essential for fetal neural development (Colvin & Abdullatif, 2012; Lassek & Gaulin, 2008; Roemmich & Rogol, 1999). Collectively, these changes result in a more defined waist and broader hips, creating a more “curvy,” womanly silhouette. The secretion of estrogen by the ovaries facilitates thelarche, or breast development (Colvin & Abdullatif, 2012). Not only will the breasts become larger in size and change in shape, but these changes set the stage for future milk production. Albeit less directly tied to reproduction, adolescent young women in pubertal transition will also begin to grow pubic hair, an event known as pubarche, and experience a fairly significant increase in height and a strengthening of the circulatory and respiratory systems (Biro et al., 2001, Colvin & Abdullatif, 2012; Graber et al. 1996).

The onset, course, and tempo of female pubertal development. Defining the temporal and sequential boundaries of puberty is challenging given the complexity of physiological events, as well as individual and cultural differences in the process. In their seminal work, Marshall & Tanner (1969), building on the work of Tanner (1962), developed a rating system in which several secondary-sexual characteristics associated with puberty, specifically breast and pubic hair development, could be classified into one of five stages and monitored to determine pubertal status and other elements of pubertal
transition (i.e., timing, tempo, etc.). Age, height, and menarche status have also routinely been collected as part of the Tanner assessment. This rating system has been essential for the advancement of pubertal development research across disciplines.

The stage is set for pubertal development during utero. Subtle changes begin taking place as early as age 6 in normally developing girls, and some changes and growth continue into late adolescence or early adulthood (Colvin & Abdullatif, 2012). The primary changes that characterize female puberty, however, typically occur between the ages of 8 to 15 (U.S. Department of Health & Human Services [USDHHS], 2009). The beginning emergence of breasts is typically the first visible indicator of puberty. There are individual variations in age of onset, as well as onset variations among girls of different racial and ethnic backgrounds and nationalities. While some secular trends have been noted (Sun et al., 2002; Sun et al., 2005), the median age of thelarche in the U.S. is 10.4 years for non-Hispanic White girls, 9.5 years for non-Hispanic Black females, and 9.8 years for Mexican American girls, with full breast development typically achieved over an approximately 5.5 year period (i.e., ages 14 to 15.5; Sun et al., 2002). Pubarche has been documented to occur at a median age of 10.6 for non-Hispanic White females, 9.4 for non-Hispanic Black girls, and 10.4 for Mexican American females, with development being completed within a period of approximately 5 years; however, some slight secular trends have been noted (Sun et al., 2002; Sun et al., 2005). Less has been documented overall with regards to the age of onset and progression of fat redistribution and hip development; however, there is evidence that it parallels breast and pubic hair development (Hillman & Biro, 2010; Mihalopoulos et al., 2010).
Although menarche does not mark the culmination of puberty, given that physical changes such as breast, pubic hair, and hip development may continue well after the achievement of menarche, it often tends to occur later in the process and suggests more advanced maturation (Graber et al., 1996). In the U.S., the mean age of menarche is 12.43 years of age, with only 10% of girls menstruating by 11 and 90% of girls having reached menarche by 13.75 years of age (Chumlea et al., 2003). Despite some evidence for a secular trend towards a decreasing age of onset of menarche in the U.S., nearly one quarter of girls in the U.S. do not achieve menarche until after the age of 13 (Chumlea et al., 2003; McDowell, Brody, & Hughes, 2007). Some variation in this process is normal, including within- and between-individual “asynchronies” in the previously noted developmental events (Graber et al., 1996; Marceau et al., 2011).

**Neurocognitive and affective changes of pubertal development.** In addition to the reproductive and other physical changes of puberty, young women in pubertal transition undergo significant neurological development (Biro et al., 2001; Dahl, 2004; Spear, 2000). The release and fluctuation of pubertal hormones act on the central nervous system and trigger functional changes in the brain (Angold et al., 2003; Kuhn, 2006; Vigil et al., 2011). These changes are related to prefrontal cortical and limbic function, which act in concert, and underlie the experience, expression, and regulation of emotion, cognition, and behavior (Angold & Worthman, 1993; Dahl, 2004; Rasgon, Zappert, & Williams, 2006; Spear, 2008; Steiner et al., 2003).

Studies have linked puberty-related neuroendocrine markers (i.e., estrogen, testosterone, etc.) with structural and functional states in the developing adolescent brain (Angold, Costello, Erkanli, & Worthman, 1999; Shirtcliff, Dahl, & Pollak, 2009).
Associations between stage indicators of pubertal maturation and neurocognitive-affective activity have also been supported in the literature (Blanton et al., 2012; Forbes et al., 2010). This is not surprising given the interrelated aspects of pubertal development, and the role of pubertal hormones in the initiation of changes in the adolescent body and brain. Given the correlation among neuroendocrine, neurological, and stage ratings of secondary-sex characteristics, stage rating systems are frequently used as a proxy for the underlying physiological, neurological, and morphological changes of puberty (Angold et al., 1999; Blanton et al., 2012; Forbes et al., 2010; Shirtcliff et al., 2009).

**Psychosocial aspects of female maturation.** Puberty is often considered a primarily biological event; however, it is a complex process of biological, psychological, and social changes (Cyranowski et al., 2000; Edwards, Rose, Kaprio, & Dick, 2011; Graber et al., 1996; Slater et al., 2001). As described by Silbereisen and Kracke (1997), puberty requires adolescents “to integrate into their self-concepts their changing bodily appearance, their unprecedented feelings, and the as yet unknown reactions by people in their surroundings” (p. 85). It often signals the culmination of childhood and the recognition of one’s emerging adult status, and all the changes and responsibilities this entails (Benjet & Hernández-Guzmán, 2002; Erikson, 1968; Gilligan, 1982). Evolving cognitive-affective capacities may make adolescents more sensitive and attuned to the beliefs, behaviors, and expectations of others in their social worlds, particularly as it concerns their standing in relation to significant others, such as parents, peers, and romantic prospects (Collins & Steinberg, 2007; Dahl, 2004).

Collectively, the shifts, changes, and growths within multiple interacting domains have the potential to bring about biological, psychological, and social disequilibrium.
These challenges are not unique to adolescent young women; however, their occurrence in the larger, gendered sociocultural context is significant. In a society valuing women’s appearance, and “where the sexualization of women is [largely] cultivated and culturally condoned” (Szymanski, Moffitt, & Carr, 2011, p. 10), the physical changes of puberty may have meaningful implications for young women’s self-worth and concept, role and value in relationships, place and security in the world, and expectations and goals for the future. Whether innate or a factor of gender learning, women are often conceived as strongly relational beings, and therefore, the relational aspects of these developmental transitions may carry particular weight and influence (Abrams, 2002; Needham, 2008).

**Female Pubertal Development and Depressive Symptomology**

The confluence of biological, psychological, and social changes that occur over the course of pubertal development may individually or collectively be implicated in possible increases in levels or rates of depression that have been purported to occur during this period. Numerous studies have examined the associations between depression and various indicators of female pubertal development, and largely suggest a significant association between degree of pubertal development and depression (Angold et al., 1998; Angold & Costello, 2006; Brooks-Gunn & Warren, 1989; LeResche et al., 2005; Oldenhinkel et al., 2011; Patton et al., 1996, 2008; Wouters et al., 2011). 

Unfortunately, differences in the ways in which depression and pubertal development have been operationalized across studies make it difficult to draw general conclusions regarding the strength and validity of findings. For instance, some studies examine the frequency or intensity of self-reported depressive symptomology, whereas others categorically examine depression on the basis of clinical diagnostic criteria or
other established criteria for classification. Differences in the time frame for evaluating depression or depressive symptomology are also common (i.e., past two weeks, past year episode, etc.). Thus, differences in prevalence rates of depression across studies, for example, may be a function of variations in definitions of depression, established scale cut-offs for group classification, and symptom duration.

Similarly, cross-study differences in significantly correlated, yet different indicators of pubertal development (i.e., menarche status, neuroendocrine markers, secondary sex characteristics, pubertal timing, etc.), may also compromise efforts to draw general conclusions from the literature base (Graber et al., 1996; Negriff et al., 2008). For example, pubertal timing and maturation status are related yet distinct constructs, yielding meaningfully different information, despite often being used interchangeably in research (Ge et al., 2003; Graber et al., 1996; Negriff et al., 2008). Pubertal maturation status represents the particular stage of pubertal development an individual is in at any given time (i.e., Tanner stage, menarche status). Pubertal timing, on the other hand, is a relative variable, in which an individual’s pubertal development is defined in relation to developmental norms (i.e., early, on time, late). Both maturation status and timing (particularly “early” pubertal timing) have been found to be significantly associated with depression (Caspi & Moffitt, 1991; Ge, Brody, Conger, & Simons, 2006; Negriff & Susman, 2011; Patton et al., 2008; Stroud & Davila, 2008); however, pubertal maturation status is perhaps most relevant to understanding the high rates of depression that emerge over the period of time in which female pubertal development typically occurs (i.e., approximately age 12 to 16; SAMSHA, 2012). Further, whereas age is commonly controlled for in studies examining the associations between pubertal maturation and
depression, age and maturation status are not only highly correlated, but age is significant to the degree it provides a developmental context (i.e., physical state, cognitive capacities, psychosocial stage, etc.) for understanding the nature, onset, and the like of a particular variable of interest (Angold et al., 1999), such as that of pubertal development.

Sampling, design, and other methodological limitations only further complicate these issues. Raising these points is not to discredit the significance or implications of findings from studies exploring the association between female pubertal development and depression. Rather, it is important to bear these methodological concerns in mind when reviewing and drawing conclusions from the literature reviewed next.

**Cross-sectional examinations.** In a large nationally representative sample of racially and ethnically diverse U.S. children and adolescents ($n = 6,748$; 53% female, 47% male), grades 5 through 12, Hayward, Gotlib, Schraedley, and Litt (1999) examined mean differences in depressive symptoms as determined by a slightly modified version of the Children’s Depression Inventory (CDI; Kovacs, 1985) among males, pre-menarcheal girls, and post-menarcheal girls, stratified by racial-ethnic group (i.e., Caucasian, African-American, Hispanic/Latino). The CDI is comprised of 14 self-report items scored on a 3-point scale (i.e., 0 or “mild,” 1 or “moderate,” 2 or “severe”), with possible summed scores ranging from 0 to 28. Controlling for age and SES (i.e., income, parental education), Caucasian post-menarcheal girls in each age group were found to have significantly higher mean levels of depressive symptoms relative to pre-menarcheal females and males, $F(2, 1886) = 12.44, p < 0.001$. For example, among 10- and 11-year old Caucasian participants, mean levels of depressive symptoms were 5.7 ($SD = 6.6$) for post-menarcheal females, 3.7 ($SD = 4.2$) for pre-menarcheal girls, and 3.8 ($SD = 4.2$) for
male participants. No significant mean differences in depressive symptoms were found between Caucasian pre-menarcheal girls and boys, nor were age and SES significant predictors of depressive symptoms. A similar pattern of higher depressive symptoms among post-menarcheal females relative to pre-menarcheal females and their same-aged male counterparts did not emerge for African-American and Hispanic/Latino participants, even after controlling for age and SES. These findings must be interpreted in light of significant differences in comparison group sizes. Smaller group sizes limit the power to detect statistically significant group differences (Cohen, 1988). Further, although statistically significant group differences emerged in the Caucasian subgroup analysis, it levels of depressive symptomology were relatively low even among post-menarcheal girls. The mean CDI value in the post-menarcheal subgroups were between 5 and 6 for the 10 to 11, 12, and 13 to 14 age groups, which is lower than the established CDI cut-off of 12, which is indicative of high levels of depressive symptoms.

Ge and colleagues (2006) found self-reported pubertal maturation status, as determined by the Pubertal Development Scale (PDS; Petersen et al., 1988), among African American girls, ages 10 to 12, to be significantly positively correlated with levels of depressive symptoms ($r = .17, p < .01$). Depression level was established by the Diagnostic Interview Schedule for Children, Version 4 (DISC – IV; Shaffer, Fisher, Lucas, Dulcan, & Schwab-Stone, 2000). The Mood Disorder (MD) symptom count scale represents the number of reported dysthymia/major depression symptoms in the past year, with possible scores ranging from 0 to 21. The mean depression symptom score for all female study participants was 5.71 ($SD = 4.79$); however, between group differences in levels of depressive symptoms were found among female pubertal timing groups. Early
maturers had significantly higher levels of depressive symptoms ($M = 7.19, SD = 4.51$) relative to on-time ($M = 6.16, SD = 4.83$) and late maturers ($M = 5.04, SD = 4.85$). Late maturers had the lowest levels of depressive symptoms.

In the Great Smoky Mountains Study (Angold et al., 1998), one of the most referenced studies of pubertal development and depression, girls ($n = 1,552$) ages 9 to 16 from the public school system in western North Carolina completed 5-point pictorial Tanner ratings of their current pubertal status. Responses to two items assessing degree of breast and pubic hair development were averaged together to determine each participant’s current maturation stage (i.e., Tanner stage 1 to stage 5). The presence of a clinically significant depressive disorder (i.e., major depression, dysthymia, and depression-NOS) within the past three months was determined by child and parent-ratings on the Child and Adolescent Psychiatric Assessment (CAPA; Angold, Prendergast, et al., 1995) interview. Relative to developmentally “immature” girls (i.e., Tanner stage 1 and 2), participants at Tanner stages 3 and higher were three times more likely to meet DSM-IV criteria for a depressive disorder ($OR = 3.4, p = 0.005$). Compared to approximately 2% of girls in Tanner Stages 1 and 2, nearly 6% of those at Stage 3, 4% of those at Stage 4, and 11% of those at Stage 5 met diagnostic criteria for a depressive disorder.

Follow-up analyses (Angold et al., 1999) within the Great Smoky Mountains Study elaborated on these findings (Angold et al., 1998). Specifically, serum blood samples were also collected among 465 girls ages 9, 11, and 13, to examine levels of several gonadotropin and steroid hormones largely responsible for the initiation and regulation of female pubertal development. Given that neuroendocrine processes underlie the process of pubertal development, it is not surprising that Tanner-based ratings of
pubertal maturation status positively correlated with several of the primary pubertal hormones among this sample (i.e., FSH: $r = 0.19$; LH: $r = 0.42$; E$_2$: $r = 0.45$; T: $r = 0.44$). Participants with higher levels of pubertal hormones were found to exhibit higher rates of DSM–IV depressive disorders. With every stage increase in testosterone (T; OR = 4.0, $p = .0001$) and oestradiol (E$_2$; OR = 2.5, $p = .0006$), there was a significantly greater likelihood of young women meeting DSM-IV criteria for a depressive disorder (i.e., major depression, dysthymia, and depression-NOS). The effects for follicle stimulating (FSH) and luteinizing (LH) hormones were in the same direction, but did not achieve statistical significance (FSH: OR = 1.7, $p = .1$; LH: OR = 1.5, $p = .2$). These findings suggest the risk of depression may increase over the course of development, with those most advanced in pubertal maturation being at the greatest risk; however, they must be interpreted in light of the cross-sectional nature of the study. Additionally, the fact that neither age nor other potentially confounding variables (i.e., race, ethnicity, grade, SES, etc.) were statistically controlled for should be considered.

**Longitudinal examinations.** Paikoff and colleagues (1991) examined the association between estradiol, an estrogen hormone involved in pubertal maturation (i.e., organ and secondary sexual development, menstruation onset and cyclicity, etc.), and depression symptoms among a small ($n = 72$) sample of Caucasian girls, ages 10 to 14, from affluent backgrounds in the New York area. Time 1 symptoms were measured by the Depressed-Withdrawal scale of the Youth Behavior Profile (Achenbach & Edelbrock, 1983), an 11-item measure of depressed affect and behavior in which participants rate how true each statement is of them (e.g., 0 = *Not at all true*, 1 = *Somewhat true*, 2 = *Very true*). Possible scores range from 0 to 22. At Time 2, depressive symptoms were
measured with the 20-item Center for Epidemiologic Studies – Depression Scale (CES-D; Radloff, 1977). Each of the 20 items were rated on a 4-point (1 to 4) Likert scale and summed (range: 20 to 80), with higher scores indicative of greater symptom distress.

Paikoff et al. (1991) found no significant associations between estradiol levels and depressive symptoms at Time 1 were found. Controlling for age and Time 1 depression symptoms, girls with higher levels of Time 1 estradiol (E2), indicating more advanced maturation status, were found to exhibit significantly ($R^2 = .26, p < .05$) higher rates of depressive symptoms one year later relative to those with lower levels. Girls prepubertal at Time 1 ($E_2 < 25$ pg/ml) exhibited the lowest mean levels of CES-D depressive symptoms at follow-up ($M = 30.53, SD = 7.18$), and those whose estradiol levels suggest they have completed or are nearing the end of puberty ($E_2 > 75$ pg/ml) reported the highest level of CES-D depressive symptoms at follow-up ($M = 37.55, SD = 11.54$). Girls in the earlier ($25 > E_2 < 50$ pg/ml) and mid-to-later stages ($50 > E_2 < 75$ pg/ml) of pubertal transition reported significantly higher levels of CES-D depressive symptoms relative to prepubertal girls and significantly lower levels of CES-D depressive symptoms than those young women at the end or completion of pubertal development (Early: $M = 33.31, SD = 13.08$; Mid: $M = 33.08, SD = 9.70$). Unfortunately, sample and measurement limitations restrict the generalizability of these findings. For instance, estradiol samples were not collected at Time 2, and therefore could not be controlled for. Additionally, a different measure of depression was utilized at the second time point (i.e., CES-D; Radloff, 1977). Correlations between the depression measures were relatively low ($r = 0.47$), suggesting these measures may have been tapping into different constructs.
Longitudinal data were also collected in the Great Smoky Mountains Study (Angold et al., 1998). Participants at Tanner stages 3 and higher were three times more likely to meet DSM-IV criteria for a depressive disorder relative to those developmentally “immature” girls (i.e., Tanner stage 1 and 2); however, movement from “immature” (i.e., Tanner stage ≤ 2) to “mature” (i.e., Tanner stage ≥ 3) pubertal status over a 12-month period of time was not associated with a concurrent increase in rates of DSM – IV depressive disorders among developing young women as hypothesized (OR 1.25, p = 0.6). The longitudinal nature of this study also allowed the authors to examine whether rates of depression among developmentally “mature” girls at Wave 2 were attributable to the recency, rate, or timing of pubertal maturation. Recency was examined by comparing rates of depression among those who were “mature” at the last and previous data collection period (one year previously) to those who were “immature” at the previous observation and “mature” at the last. The rate of pubertal change was defined as the number of stages advanced over one year between waves. Pubertal timing was examined by comparing those who developed “early” (i.e., reaching Tanner Stage 3 or menarche before the age of 12) to those who were developmentally on time (i.e., “mature” status at age 12 or later). Neither recency, rate, nor timing of maturation were significantly associated with rates of depression among “mature” girls, leading the authors to conclude that simply reaching “mature” status (i.e., Tanner stage ≥ 3) increased the risk of developing depression irrespective of “when they got there, ‘how fast’ they got there, or once there, ‘how fast’ they subsequently matured” (p. 58).

Benjet & Hernández-Guzmán (2002), in a longitudinal study of Mexican students (M age = 12.3, SD = 0.96; n = 512 female), grades 5 through 7, found girls greater than 6
months post-menarche to have significantly higher mean levels of depression symptoms (as determined by the Spanish version of the Center for Epidemiological Studies – Depression Scale; CES – D; Radloff, 1977) at both the beginning (i.e., Time 1) and end (i.e., Time 2) of an academic school year relative to pre-menarcheal girls, after controlling for age and sex, $F(1, 933) = 6.7, p = 0.01$. A repeated measures ANOVA was then used to analyze changes in level of depressive symptoms across the time of an academic school year among menarche status/menarche recency groups.

No significant between-wave changes in depressive symptoms were noted among those who remained pre-menarche or were greater than 6 months post-menarche at Time 1, and therefore, greater than 1 year post-menarche at Time 2. An increase in depressive symptoms was found among young women who achieved menarche over the course of the study (i.e., Time 1: Pre-menarche, Time 2: Post-menarche); however, this increase did not reach statistical significance. Interestingly, there was a statistically significant increase (i.e., approximately one-third $SD$ or 3.75 mean increase) in depressive symptoms among young women who were less than 6 months post-menarche at Time 1, and therefore between 6 and 12-months post-menarche at Time 2. No effect for age was found across study analyses. At Wave 2, rates of “elevated depressive symptomology” were as follows: 14% for those who remained pre-menarche, 16% for those who transitioned from pre to post-menarche across study waves, 31% for those between 6 and 12 months post-menarche, and 25% for those greater than 12 months post-menarche.

“Elevated depressive symptomology” was established at one standard deviation above the sample mean, and therefore, such a classification is sample-dependent. Given this, as well as the fact that sample mean CES – D scores were not presented in the study
write-up, insight into the clinical significance of these rates cannot be determined, limiting cross-study comparisons. It is unlikely that actual clinical depression among the general public occurs at such high rates (SAMSHA, 2012). Such rates raise question as to the equivalence of select measures across linguistic and cultural groups; however, it must be noted that the Spanish translation of the CES – D has been found to be reliable and valid for use with Mexican children and adolescents (Benjet, Hernández-Guzmán, Tecero-Quintanilla, Hernández-Roque, & Chartt-Léon, 1999). The degree to which distributions of sample CES-D scores mirror normative data for Mexican youth was not discussed, and therefore more meaningful conclusions cannot be drawn. Additionally, relatively few participants \((n = 55)\) achieved menarche over the study period, therefore, limiting study power.

Despite these limitations, results corroborate previous findings that young women more pubertally advanced are more likely to exhibit higher levels of depressive symptomology (Angold et al., 1998; Angold & Costello, 2006; Brooks-Gunn & Warren, 1989; LeResche et al., 2005; Oldenhinkel et al., 2011; Patton et al., 1996, 2008). Findings also suggest a female puberty-related depression risk may not be culturally relative and limited to non-White Hispanic teen girls, but rather is a cross-cultural phenomenon, extending across borders and racial and ethnic group lines. Lastly, results further suggest that the post-menarcheal depression risk may not be immediate, but rather there may be a critical period between 6 and 12 months post-menarche in which developing young women may be at a particular risk. A failure to capture other aspects of pubertal development, however, precludes concluding that this risk is attributable to menarche
change alone, as other post-menarcheal morphological changes (i.e., breast growth, hip development, etc.) may be more likely to occur within this window of time.

More recently, Patton and colleagues (2008) examined associations between pubertal maturation and depression symptoms over three years (2002 – 2004) among a large, international sample (Wave 1: \( n = 2,885 \) in Washington and 2,884 in Victoria; Wave 3: \( n = 961 \) in Washington and 1,904 in Victoria), ages 10 to 15. Depressive symptoms were measured using the self-report Short Mood and Feelings Questionnaire (Angold, Costello, et al., 1995), and the presence of high levels of depressive symptomology was determined by a score of 11 or higher on this measure. This measure has demonstrated high reliability and moderate to high criterion validity as indicated by strong correlations with a similar child and adolescent self-report depression scales (e.g., Children’s Depression Inventory; Kovacs, 1985) and outpatient diagnostic interview (e.g., Diagnostic Interview Schedule for Children’s Depression Scale) (Daviss et al., 2006; Kent, Vostanis, & Feehan, 1997; Shaffer et al., 2000; Wood, Kroll, Moore, & Harrington, 1995); however, empirical nor clinical justification for the 11 or higher cut-off utilized was provided by study authors. Interpretations of the rates of high depressive symptomology must be made with this in mind.

At Wave 1, 26% of young women were classified as exhibiting high levels of depressive symptoms. Relative to girls early in pubertal transition (i.e., Tanner Stages 1 or 2), those in mid- (i.e., Tanner Stage 3) and later stages of pubertal development (i.e., Tanner Stages 4 or 5) had 1.4 and 2.5 greater odds, respectively, of reporting high levels of depressive symptoms at Wave 1 and a 1.7 and 3.6 greater odds at Wave 2, adjusting for age and academic grade. Because so few female participants were early pubertal at
Wave 3, mid-pubertal girls were used as the reference point. Specifically, relative to participants at mid-puberty at Wave 3, girls in later stages of development had a 1.7 greater odd of reporting high levels of depressive symptoms. Age, nor academic grade, significantly predicted high depressive symptoms at either wave. Half of female participants achieving the most advanced stage of development (i.e., Tanner Stages 4 or 5) reported significant depressive symptoms (i.e., ≥11) over the course of the study.

Change in pubertal development over a 12-month period was also examined in relation to change in rates of high depression symptomology over the same period. Female participants (n = 3,652) completed ratings of pubertal development status and depressive symptoms at two consecutive 12-month study waves (i.e., Wave 1 and Wave 2, or Wave 2 and Wave 3). Changes (i.e. from baseline to 12-month follow-up) in rates of high depressive symptomology were compared across five pubertal transition groups: (1) Remained Early, (2) Early to Mid-Puberty Transition, (3) Remained Mid-Puberty, (4) Mid- to Late Puberty Transition, (5) Remained Late Puberty. Approximately 5% (i.e., 4.6%, n = 168) of female participants were excluded from study analyses given reports of regressed pubertal development. The magnitude of change was determined by depressive symptom prevalence confidence intervals at Time 1 and Time 2 that do not overlap.

Within the Early Puberty Constant group (n = 125), 14% (95% CI: 7 – 20) at Time 1, and 12% (95% CI: 5 – 19) at Time 2 reported high rates of depressive symptomology. This represents a slight, albeit non-significant, between-wave decrease in prevalence of high depressive symptomology among this group. Relative to those who remained in the early stages of puberty at Time 2 (i.e., Early Puberty Constant), 17% (95% CI: 11–22) of participants transitioning from early to mid-puberty between Times 1
and 2 ($n = 242$) exhibited “high” levels of depressive symptomology at Time 2. This represents a 4% increase in the rate of high depressive symptomology among young women transitioning from early to mid-puberty across study waves (Time 1: 13%, 95% CI: 11–17). Among those transitioning from mid- to late puberty ($n = 513$), there was an 8% increase in prevalence of high levels of depressive symptoms between waves, with 29% reporting high levels of depressive symptoms at Time 2 (Time 1: 21%, 95% CI: 17–24; Time 2: 29%, 95% CI: 24–33). This increase did not occur among those remaining mid- ($n = 508$; Time 1: 19%, 95% CI: 15–22; Time 2: 17%, 95% CI: 13–22), or late pubertal ($n = 2,078$; Time 1: 35%, 95% CI: 32–37; Time 2: 35%, 95% CI: 32–37) stages at Time 2. Thus, while rates of depressive symptoms were higher among young women more pubertally developed, the increase in depressive symptoms appears to parallel the transition from earlier to later stages of puberty, with the greatest increase likely to occur from mid- to late-puberty.

Patton and colleagues (2008) then utilized logistic regression to prospectively examine whether pubertal maturation status prospectively predicted the onset of elevated depressive symptomology among the same paired-observation study sample. Specifically, Time 1 pubertal development status was evaluated in relation to change in depressive symptoms over the 12-months between study waves among participants reporting low levels (i.e., <11) of depressive symptoms at baseline (i.e., Time 1 = Wave 1 or Wave 2) and between-wave pubertal stability. Age, academic grade, geographic location, level of depressive symptoms at Time 1, and between-wave pubertal stage change were all adjusted for in this analysis. Relative to girls in earlier stages of puberty at Time 1, young women in mid- and late stages of puberty had 1.5 (95% CI: 0.95–2.3) and 1.8 (95% CI:
1.1–2.9) greater odds of reporting new onset high depressive symptomology 12-months later. Further, not only did maturation status predict the onset of new depressive symptoms, but it was also found to predict the persistence of depressive symptoms. More specifically, a separate, but identical, regression model was run among participants reporting high levels of depressive symptoms at two consecutive waves (12-months apart) to determine if pubertal maturation status predicted the persistence of depressive symptoms. Relative to girls in early stages of puberty at baseline (i.e., Time 1 = Wave 1 or Wave 2), young women in mid- and late stages of puberty were 2.2 (95% CI: 1.1–4.3) and 2.4 (95% CI: 1.2–4.7) times more likely to have persisting high symptoms 12 months later (i.e., Time 2 = Wave 2 or Wave 3).

Taken together, findings from this study revealed that advanced maturation predicted an increase in depressive symptoms among young women in pubertal transition. Not only did pubertal maturation status predict the onset of high depressive symptomology, with girls in late puberty being at a significantly elevated risk, but it was also linked with persistence of elevated depressive symptoms, particularly from mid- to late puberty. Wave 1 depressive symptoms were not found to be associated with the rate of pubertal development, suggesting that it was not high depressive symptoms that increase, or “accelerate,” pubertal maturation.

**Summary and limitations of this research.** Empirical evidence largely supports an association between pubertal development and depression, such that more advanced or advancing pubertal maturation is associated with higher rates of depression and increases in depressive symptomology (Angold et al., 1998; Angold & Costello, 2006; Brooks-Gunn & Warren, 1989; LeResche et al., 2005; Oldenhinkel et al., 2011; Patton et al.,
1996, 2008); however, findings have been mixed, interestingly, even across studies
within the same research program (Angold et al., 1998; Negriff et al., 2008; Paikoff et al.,
1991). The same is true among research participants with similar racial and ethnic
backgrounds (Ge et al., 2006; Hayward et al., 1999). For example, whereas Hayward and
colleagues (1999), as described previously, found no effect of menarche status on
depressive symptomology, Ge and colleagues (2006), also reviewed earlier, found a
significant positive correlation between pubertal development status and level of
depressive symptoms among African American girls.

This body of work is not without its limitations. The degree to which
inconsistencies in findings are attributable to differences in sampling, measurement, and
other methodological dissimilarities should be further considered. Notably, it is difficult
to make comparisons across studies, as well as draw firm conclusions, due to differences
in the ways in which pubertal development has been operationalized (i.e., neuroendocrine
markers, secondary sex characteristics, menarche status, pubertal timing, etc.) and
depression has been measured (i.e., diagnostic categorical vs. trait-dimensional,
dichotomous vs. continuous, cross-sectional vs. longitudinal, etc.) (Graber et al., 1996;
Negriff et al., 2008). Further, this body of work has largely been limited by small,
culturally homogenous and non-representative samples, as well as by cross-sectional
research designs. Few studies have examined these relationships longitudinally, and even
fewer have investigated parallel pubertal and depressive symptom change over time.

Although questions remain regarding the nature of associations between
indicators of female pubertal development and depression, findings across studies largely
suggest that there is likely “some aspect of puberty itself [that] is important in the
appearance of the female preponderance to depression” or depressive symptomology (Angold et al., 1998, p. 58). Negriff and colleagues (2008) argued that research on pubertal development needs to move beyond documenting associations between pubertal maturation and depressive symptoms, to investigating factors that underlie and/or moderate these associations.

**Pubertal Development and Depression Risk: An Integrated Life Course Model**

From an Integrated Life Course perspective, depression is multi-faceted, multi-determined, and non-linear (Harris et al., 2009; Harris, 2010). It involves the reciprocal interaction of biological, behavioral, and sociocultural variables in a developmental context (Figure 1). Diathesis-stress and stress-buffering models, both of which can be subsumed within this broader, Integrated Life Course framework, have garnered support across disciplines as it relates to understanding the occurrence of depression among young women of pubertal age (Cohen & Wills, 1985; Edwards et al., 2011; Forbes et al., 2010). From these perspectives, developing young women may have a biological predisposition (i.e., “diathesis”) to depression; however, this vulnerability may or may not be actualized depending on the presence or absence of psychological and sociocultural stressors and resources (Parker & Brotchie, 2010; Susman et al., 1991).

**The biological vulnerabilities of female puberty.** The myriad of neurological changes that underlie and result from the maturation process have been argued to leave young women biologically vulnerable to depression, and a number of empirical studies have found significant associations between biological variables (i.e., neuroendocrine and neurocognitive-affective markers) and depressive symptoms among adolescent young women in pubertal transition (Forbes et al., 2010; Hayward & Sanborn, 2002; Paikoff et
al., 1991; Patton et al., 2008). These associations are complex, and difficult to examine and confidently address through research. Relevant biological indicators have been difficult to isolate and challenging to study in association with environmental influences. However, there are several theories concerning these associations and their role in understanding elevated rates of depressive symptomology among girls of pubertal age.

From a pathophysiology perspective, the surge, withdrawal, or interaction of certain hypothalamic-pituitary-gonadal hormones involved in female puberty have been proposed to have a direct, “activational” effect on the central nervous system (CNS), resulting in mood and other vegetative symptoms characteristic of depression (Angold et al., 1999; Brooks & Warren, 1989, p. 50; Hayward & Sanborn, 2002; Paikoff et al., 1991). However, this conception of female pubertal depression has largely evolved in biomedical and psychological models. It is now commonly believed the hormonal environment during puberty is associated with a redistribution of excitatory and inhibitory neurotransmitters. “Such a ‘shift’ might affect the background ‘tone’ or reactivity of the CNS to environmental stimuli” (Hayward & Sanborn, 2002, p. 52).

As noted, pubertal hormonal processes also trigger functional changes in the adolescent brain (Angold et al., 2003; Forbes et al., 2010; Kuhn, 2006; Vigil et al., 2011). These puberty-driven “organizational effects” are in regions of the brain that have been associated with cognitive-affective and behavioral aspects of depression, such as the hypothalamus, amygdala, hippocampus, and cerebral cortex to name a few (Hayward & Sanborn, 2002, p. 52), and involve such neurological processes as socioemotional processing, reactivity and reward-motivated behavior, memory, and emotion regulation that have been implicated in depression (Blanton et al., 2012; Forbes et al., 2010). While
the adolescent brain becomes fully mature in terms of affective structures and capacities (i.e., subcortical), the prefrontal cortex, associated with cognition and the regulation of behavior, is among the last to develop (Jiao et al., 2011). Jiao and colleagues (2011) suggested that young, developing women may be vulnerable to depression as a result of “functional discrepancy … between (the frontal) cognitive control and (the subcortical) affective processing” (p. 1). This, coupled with a heightened affective tone and reactivity to intrapsychic and environmental stimuli, is argued to underlie a biological predisposition to depression (Edwards et al., 2011).

**Psychosocial stressors and depression risk.** The changes and evolving capacities young women endure as a result, and over the course of, puberty may open them up to a number of new opportunities, rich experiences, and ways of relating to themselves and others; however, such changes may also be the source of considerable stress. When stress accumulates, persists, or exceeds one’s perceived ability to cope, thoughts and feelings of helplessness and hopelessness and a loss of self-esteem and efficacy may ensue (Cohen & Wills, 1985). Such thoughts and feelings pose a risk for depression that may be further inflated by puberty-driven biological states and changes. These factors may leave developing young women “highly sensitive” to environmental stressors, and thus vulnerable to depression.

Stressors of an interpersonal nature have been noted to be especially significant to the health and well-being of developing young women, and have been associated with depressive symptomology (Rudolph, 2002; Rudolph, Flynn, & Abaied, 2008). Puberty-related changes in the amygdala involve socioemotional processing relevant to self-evaluation and relatedness to others (Blanton et al., 2012). Real or perceived social threat,
rejection, or interpersonal loss, for example, may frustrate or challenge a young woman’s fundamental need for belonging and meaningful interpersonal connection (Baumeister & Leary, 1995). This, coupled with heightened physiological reactivity, would significantly increase depression odds from a diathesis-stress conception of depression.

**Psychosocial resources and depression risk.** Not only can biological mechanisms influence the interpretation and reaction to environmental stimuli, there is some evidence to suggest interpersonal interactions, both positive and negative, can modulate physiological reactions (Eisenberger, Taylor, Gable, Hilmert, & Liberman, 2007). Positive interpersonal connection, for instance, has been found to attenuate affective tone and stress reactivity (Cohen & Wills, 1985; Uchino, 2006). Further, according to the stress-buffering hypothesis, social support systems may play an important role in modulating the impact of potential stressors at either or both the point of perception and coping (Cohen & Wills, 1985; Frey & Röthlisberger, 1996; Ge, Natsuaki, Neiderhiser, & Reiss, 2009; Printz, Shermis, & Webb, 1999). As described by Cohen & Wills (1985), “the perception that others can and will provide necessary resources may redefine the potential for harm … and/or bolster one’s perceived ability to cope” (p. 312). Such support may be especially important to young women navigating the new stressors, demands, opportunities, and unknowns brought on by pubertal development, and it has been suggested its importance may even hold among those with a vulnerability to depression (Wetter & Hankin, 2009).

**Puberty and Depression in Context: Exploring the Parent-Daughter Relationship**

Carter and McGoldrick (1999) described “the family life cycle [as] the natural context within which to frame individual identity and development,” (p. 1), including the
development, maintenance, and resolution of emotional and behavioral distress and difficulty. The family context can be one of opportunity or risk. This is particularly true of the parent-child relationship, the nature and stability of which may pose a risk or provide protection as it concerns depression during puberty (Hughes & Gullone, 2008; Papini & Roggman, 1992; Sagrestano et al., 2003; Stark et al., 2012).

**Parental relationship resources and depression risk in puberty.** Young women who lack a quality relationship with their mothers or fathers may have more difficulties navigating the changes of puberty, and be more susceptible to actualizing puberty-related depressive vulnerabilities (Dahl, 2004; Parker & Brotchie, 2010). On the other hand, young women with a strong, quality parent-daughter relationship may be protected from the biological, neurological, and sociocultural vulnerabilities brought on by puberty (Beardslee et al., 2012; Forehand et al., 1991; Frey & Röthlisberger, 1996; Printz et al., 1999; Wetter & Hankin, 2009).

Young women’s perception that their mothers and fathers are available for nurturance, companionship, advice, and other needs may feel better able to meet the demands of new stressors and opportunities, thus protecting them from thoughts of helplessness or hopelessness that may contribute to depression (Cohen & Wills, 1985; Wetter & Hankin, 2009). Additionally, parental support and emotional connection can “tranquiliz[e] the neuroendocrine system” (Cohen & Wills, 1985; Eisenberger et al., 2007; Uchino, 2006), which may be particularly important given the heightened affective tone and stress reactivity associated with pubertal neuroendocrine changes (Hayward & Sanborn, 2002). Thus, a strong quality parent-daughter relationship may directly attenuate the association between advancing maturation and depression risk, or “buffer”
against the impact of life stressors that increase the risk of depression (Cohen & Wills, 1985). Unfortunately, few studies have examined the associations among pubertal development and parent-daughter relationship quality as it relates to depression; however, the extensive research that has been conducted on relationship quality and depression provides a strong empirical basis to build upon (Hughes & Gullone, 2008; Sagrestano et al., 2003; Stark et al., 2012).

**Empirical review: Parent relationship quality and depression.** A sizable body of research has documented associations between depression and the parent-adolescent relationship, particularly along such dimensions of control and autonomy, communication and conflict, the affective quality of the relationship (i.e., closeness, care, warmth, etc.), and overall relationship satisfaction (Epkins & Heckler, 2011; Sheeber, Hops, & Davis, 2001; Stark et al., 2012; Vazsonyi & Belliston, 2006). Support for the linkage of parental and familial factors and youth depression is so robust that it has been argued that relational disturbances in the family (i.e., attachment problems, low cohesion and support, poor communication, high conflict, etc.) should be considered along with traditional criteria for a diagnosis of depression among children and adolescents (Epkins & Heckler, 2011; Kaslow, Broth, Arnette, & Collins, 2009). For instance, Stark and colleagues (2012) found the parent-child relationship among depressed, relative to non-depressed, youth to be characterized by poorer attachment, less and more negative communication, less support and cohesion, and little shared involvement in activities.

In a large ($n = 6,935$), cross-cultural study of adolescents ages 15 to 19 from four countries (i.e., U.S., Hungary, Netherlands, Switzerland), Vazsonyi & Belliston (2006) examined associations between adolescent-reported maternal and paternal closeness,
support, monitoring, communication, peer approval, and conflict (as measured by the Adolescent Family Process Measure; Vazsonyi, 2003) and internalizing symptoms (i.e., depression, anxiety) as measured by a shortened version of the Weinberger Adjustment Inventory; Weinberger, 1998, Weinberger & Schwartz, 1990) at a single-time point. Across cultures, paternal and maternal closeness, support, monitoring, communication, and peer approval were significantly negatively correlated with adolescent-reported depressive symptomology. Maternal conflict and paternal conflict were significantly positively associated with depressive symptomology. Controlling for family structure and demographics (i.e., age, gender, SES), parenting variables accounted for 18.6% to 23.3% of the variance in depression symptoms across countries. Both maternal and paternal support and parent-child conflict accounted for the most variance such that lower perceived support and higher conflict were associated with higher depressive symptoms.

Parent-child relationship quality has further been linked to depression trajectory. Brendgen and colleagues (2005) examined the predictive links between relations with parents (averaged across time points), based on several self-report items reflecting parental attachment, communication, and self-disclosure, and depression trajectory group status among a sample of 550 (46.9% female, 53.1% male) Canadian early adolescents ages 11 to 14. With respect to depression status, adolescents were classified into the following groups: (1) Consistently Low/Minimal Depression (47.8%, n = 214), (2) Consistently Moderately Depressed (30.3%, n = 119), (3) Increasingly Depressed (12.7%, n = 45), (4) Consistently Highly Depressed (9.3%, n = 36). Controlling for sex, family adversity (i.e., family structure, educational level and occupational status of parents, age of parents at birth of child), temperamental reactivity (averaged), and peer
popularity–rejection (averaged), adolescents with low quality parental relationships were found to have significantly ($p < .001$) greater odds of having consistently moderate (OR = 0.61), consistently high (OR = 0.33), and increasing depressive symptom trajectories (OR = 0.56) relative to adolescents with high quality parental relationships. Sex, family adversity, and temperamental reactivity were both significant predictors such that females, high family adversity, and temperamental reactivity individually contributed to odds of maintaining or developing high depressive symptomology. Peer relationship variables were not significant predictors in this model.

Stice, Ragan, & Randall (2004) conducted prospective analyses of perceived parent and peer support as predictors of depression status among 496 girls ages 11 to 15 in the U.S., over a 2-year period (i.e., baseline, 1 year, 2 years). Social support was assessed using the Network of Relationships Inventory (Furman, 1996), which evaluates such relationship dimensions as companionship, guidance, intimacy, affection, admiration, and reliable alliance. Depression was determined by a structured interview that was adapted from the original Schedule for Affective Disorders and Schizophrenia for School-Age Children (Puig-Antich & Chambers, 1983) in line with DSM – IV diagnostic criteria. Both measures have been found to demonstrate strong reliability and validity (Furman, 1996; Lewinsohn, Hops, Roberts, Seeley, & Andrews, 1993; Stice & Bearman, 2001). Among girls who were not depressed 12-months prior, lower parental support prospectively predicted the onset of depression 1 year later (OR: -0.46, $p = .001$), controlling for peer support. Peer support did not emerge as a significant predictor of depression. Further, baseline depression was not found to predict a decrease in parental support across waves, suggesting youth who are depressed are not more likely to perceive
the quality of their parental relationships to decline significantly over time. These findings suggest “deficits in parental support” may be a risk factor for depression, and further, suggest depression itself may not be deleterious to the parental relationship. However, it is important to note that although statistically significant, the effect was relatively small.

Further, while not concurrently examining maturational effects, studies suggest having a high quality, warm, and supportive relationship with mother and father figures may serve a protective function as it concerns the risk of depression among developing young women (Barber, Stoltz, & Olsen, 2005; Cornwell, 2003; Eshbaugh, 2008; Needham, 2008). In one study of racially and ethnically diverse female and male middle-school adolescents ($n = 636$) over the course of an academic school year, Rueger, Malecki, & Demaray (2010) examined the concurrent and predictive associations between perceived social support and indicators of adjustment and well-being (i.e., depression, academic performance, etc.). Self-report measures of social support (i.e., Child and Adolescent Social Support Scale, CASSS; Malecki, Demaray, Elliott, & Nolten, 1999; Social Support Scale for Children, SSSC; Harter, 1985) and behavior and adjustment (i.e., BASC Self-Report of Personality, SRP; Reynolds & Kamphaus, 2004) were collected within 1 month of beginning the school year, and behavior and adjustment measures were repeated again at 1 month prior to the completion of the school year. Grade point average was also drawn from school transcripts at Time 2. Social support (i.e., emotional support, informational support, instrumental support, appraisal support, and feeling understood) measures were only administered at baseline. Among female participants ($n = 325$), baseline social support (i.e., perceived support from parents,
teachers, classmates, friends, and school) was significantly ($p < .01$) associated with depressive symptoms at Time 1 and Time 2 (controlling for Time I depressive symptoms), accounting for 22% and 10%, respectively, of the variance in depressive symptoms at these two time points. Consistent with a protective effect, parental support emerged as the most significant predictor of depressive symptoms relative to other sources of social support (i.e., teacher, classmate, friend, and school support) at both Time 1 ($B = -.41, p < .01$; other support $B$ ranges: -.03 to .06) and Time 2 ($B = -.24, p < .01$; other support $B$ ranges: -.11 to .04). Unfortunately, effect sizes in the present study were relatively small. Additionally, despite the associations documented between pubertal maturation status and depression, developmental status was not taken into consideration.

Empirical review: Pubertal development, parent relationship quality, and depression. Patton et al. (2008), whose work was previously presented, examined whether levels of family attachment and conflict predicted the onset and persistence of high depressive symptomology over the 12-month period between study waves. Participants were 5,769 children and adolescents ages 10 to 15 from the U.S. (Washington State) and Australia (Victoria). Depressive symptoms were measured using the self-report Short Mood and Feelings Questionnaire (Angold, Costello, et al., 1995), and the presence of high levels of depressive symptomology was established by a score of 11 or higher. Adjusting for age, grade level, location, pubertal stage change, school connection, and bullying, participants with low (relative to high) family attachment and high (relative to low) family conflict had a 1.1 (95% CI: 0.9 – 1.5) and 1.3 (95% CI: 1.0 – 1.7) greater odds, respectively, of reporting onset elevated depressive symptomology 12
months later. Further, controlling for demographic variables (i.e., age, grade level, location), school/peer variables (i.e., school connection, bullying), pubertal stage change, and psychological variables (i.e., self-blaming coping style, emotional control, self-efficacy), participants with low (relative to high) family attachment and high (relative to low) family conflict had a 1.1 (95% CI: 0.8 – 1.5) and 1.6 (95% CI: 1.2 – 2.1) greater odds, respectively, of high depressive symptomology persisting over the 12 months between study waves.

Of the family context variables (i.e., family attachment, family conflict), only high family conflict was significant in relation to onset high depressive symptom risk and continuing high depressive symptom risk. Social context variables, which included family attachment and family conflict, among several other social variables (e.g., school attachment, bullying, etc.), were also examined. There was not a significant reduction in the association of pubertal maturation status and new onset high depressive symptomology, nor pubertal maturation status and persisting high depressive symptomology after controlling for family attachment and contact, as well as other potentially meaningful social context variables (e.g., bullying, etc.). This suggests pubertal maturation status and family attachment and conflict may have independent effects on depression risk. Unfortunately, the interaction of pubertal maturation status and family relationship variables were not examined, and therefore conclusions related to the protective and risk influence of parental/family relationships for young women in pubertal depression could not be drawn as it concerns depression risk.

Only one study was found that directly addressed the potential protective influence of the parent-daughter relationship as it relates to the association between
pubertal development and depression. Specifically, the study of early adolescent Mexican female students ($n = 503$) in 5th through 7th grade (age: $M = 12.3$ at Time 2) described extensively earlier, Benjet & Hernández-Guzmán (2002) examined changes in menarche status in relation to changes in depression symptomology over the course of an academic school year, as well as potential parenting factors that might moderate these associations (i.e., maternal and paternal affect, communication, and control). At both the beginning and end of the school year, young women reported whether and when they reached menarche, as well as completed a self-report measure of depressive symptomology (i.e., Spanish version of CES-D; Radloff, 1977). Study participants also responded to items related to maternal and paternal relationship quality (i.e., Relation with Mother and Relation with Father subscales of the Drug Risk Scale; Climent, Aragon, & Plutchick, 1989) at baseline. There is some evidence to suggest the Drug Risk Scale is a valid and reliable instrument for use with Mexican adolescents (Gonzalez-Forteza, 1996).

As noted, changes in levels of depressive symptoms between waves were examined among four menarche status/recency change groups (i.e., Pre/Pre, Pre/Post, < 6 months Post / > 6 Post, > 6 months Post/ > 12 Post). Findings revealed a significant increase in depressive symptoms over the course of the academic year for girls who were less than 6 months post-menarche at Time 1, and therefore greater than 6 months post-menarche at Time 2. While not reaching statistical significance, there was a trend suggesting increase in depressive symptoms noted among those who reached menarche over the course of the study (i.e., Pre/Post). There was no change among those who remained pre-menarche or those more than 6 months post-menarche at Time 1 (i.e., > 6 months Post/ > 12).
Baseline parenting variables (i.e., maternal and paternal affect, communication, and control) were each examined for their moderating influence on the association between menarche status/recency and depressive symptomology (Benjet & Hernández-Guzmán, 2002). The authors proposed “an increase in parental control may undermine an adolescent girl’s sense of competence and increase risk for depression whereas parental affect might buffer the challenges faced by adolescents during this transition” (p. 431). A significant Time by Menarche Status by Maternal Control interaction was found. Maternal control moderated the association between menarche status and change in depressive symptoms for those recently post-menarche at Time 1 (i.e., < 6 months at Time 1, 6 to 12 months post-menarche at Time 2), but not for the other 3 menarche status groups (i.e., Pre/Pre, Pre/Post, > 6 months Post/ > 12 Post). Specifically, between-wave depressive symptomology increased significantly among young women who were recently post-menarche at Time 1 (i.e., < 6 months at Time 2, between 6 and 12 months post-menarche at Time 2) and perceived their mothers as highly controlling. No such moderating effect was found for paternal control. This finding is consistent with theory and research based largely upon non-Hispanic White families in the U.S. related to the struggle between autonomy and control in child-parent relationships at this developmental juncture (Preto, 1999). Findings suggest such an effect may only be temporary, however. The authors do call for caution in the interpretation of findings due to limited group sizes along menarcheal change by parent variables.

Unexpectedly, no maternal or paternal influence of relationship affect quality and communication (general and specific to sexuality) were found. Though contrary to the expected protective function of parental relationships, such a hypothesis was drawn from
research conducted primarily with U.S., opposed to Mexican, adolescents (Herman-Stahl & Petersen, 1999; Houltberg, Henry, Merten, & Robinson, 2011; Hughes & Gullone, 2008). No comparable studies examining the affective quality of the parent-daughter relationship in association with depressive symptomology and pubertal advancement among developing young women from other, non-Mexican cultural backgrounds were located, however. Non findings raise question concerning possible cultural differences in risk and protective factors in the parent-daughter relationship.

Unfortunately, specific statistical findings were not reported and were only briefly mentioned in the context of the discussion. Further, between wave changes in the parent-daughter relationship over the course of puberty were not reported or controlled for, pubertal development was narrowly defined (i.e., menarche status), and the sample was limited to the study of Mexican youth (Benjet & Hernández-Guzmán, 2002). Additionally, only a small number of participants ($n = 55$) transitioned from pre- to post-menarche between study waves, limiting the power to detect an effect, if present.

Overall, there is a virtual absence of studies related to the potential risk or protective influence of the parent-daughter relationship as it relates to vulnerabilities and risks for depression during pubertal transition. This is true of both studies concerning the protective or deleterious effect of relationships with regards to depression and well-being (Eshbaugh, 2008). Despite this, the evidence relating to the potential protective or harmful influence of the parent-adolescent relationship in general is compelling, and further investigation into the association between parental relationship quality and depression in the context of adolescent young women’s pubertal development is warranted. Drawing upon theory and existing literature, pubertal advancement is
expected to be significantly associated with a rise in depressive symptoms for young women with low quality parental relationships but not for those who advance in development but perceive relationship quality to be high.

**The parent-daughter relationship: Pubertal change and contributing risks.**

Several researchers have documented associations, most small in magnitude, between female pubertal development status and various indicators of the parent-daughter relationship (Hill, Holmbeck, Marlow, Green, & Lynch, 1985; Holmbeck & Hill, 1991; Montemayor, Eberly, & Flannery, 1993; Papini & Sebby, 1987, 1988; Steinberg, 1988). Thus, while parent relationship quality may be a protective resource or risk for depression during pubertal transition, a decline in the relationship may pose an additional risk.

**Pubertal change: An individual and family metamorphosis.** Greater “conflictive engagement,” withdrawal of positive affect, and more negative affect has been observed in parents in interactions with their daughters in pubertal transition (Hill et al., 1985; Holmbeck & Hill, 1991; Montemayor et al., 1993; Patton et al., 2008; Steinberg, 1988). In their 3-year, two-nation (i.e., U.S., Australia) examination of the associations between pubertal maturation and depression (previously described in detail), Patton and colleagues (2008) aggregated data from the three waves and examined mean differences in levels of family conflict and family attachment among young women in different stages of pubertal transition (i.e., early, mid-, and late puberty; as determined by a modified version of the Pubertal Development Scale; Peterson et al., 1988). Young women late in development reported significantly lower levels of family attachment and higher levels of family conflict than those in early or mid-puberty, and those in early puberty reported the highest levels of family attachment and lowest levels of conflict.
Steinberg (1988) conducted a longitudinal study of 106 females ages 11 to 16, and their parents, in which adolescent and parent reports of relationship cohesion, conflict, and autonomy were examined in relation to pubertal maturation at two time points (i.e., baseline, 12-month follow-up). Controlling for relationship distance at baseline, Steinberg (1988) examined whether pubertal maturation status at Time 1 predicted parent-daughter relationship distance at Time 2. Overall, findings suggest young women perceive more arguments with their mothers, and fewer calm discussions and more intense conflicts with their fathers, as they mature (Steinberg, 1988). They also found developing young women may perceive a decline in paternal cohesion and less parental permissiveness at the “midpoint” or “apex” of puberty. These findings, like those just presented, also suggest mothers and fathers perceive differences in their relationships with their daughters as they mature, particularly in communication. Specifically, in this study, parents reported fewer calm discussions, as well as fewer arguments (fathers only), at the apex of puberty relative to earlier and later stages of development. It is important to note that age was not controlled for, the sample was predominately non-Hispanic White, and pubertal stage comparison groups were relatively small. Additionally, although significant, maturation status only explained a small amount of variance in relationship distance measures. Despite these limitations, Steinberg (1988) concluded that findings support the notion that pubertal maturation is associated with “distancing” in the parent-daughter relationship, suggesting puberty may not only be a time of physical and neurocognitive change, but also interpersonal change in relationships with parents.

From a family life cycle perspective, there is reciprocal influence among individual, family, sociocultural, and timing factors that have meaningful implications for
individual family members and the family system as a whole. Thus, the normative
developmental transitions individuals within a family go through as they mature
influence and are influenced by the broader family system (Carter & McGoldrick, 1999).
Such developmental shifts often bring about a state of disequilibrium and stress in the
system, requiring adaptation. “Boundaries shift, psychological distance among members
changes, and roles within and between subsystems [must be] redefined” (Carter &
McGoldrick, 1999, p. 1). There is a reciprocal influence on both the individual and family
system. During adolescence, family relationships must be renegotiated and evolve in
order to meet the psychosocial developmental milestones and task resolutions necessary
Preto (1999) argued, “adaptations in family structure and organization … are required to
handle the tasks of adolescence [and] are so basic that the family itself is transformed
from a unit that protects and nurtures young children to one that is a preparation center
for the adolescent’s entrance into the world of adult responsibilities and commitment” (p.
274). At the center of this “family metamorphosis” (Preto, 1999, p. 274) is the struggle
between “connectedness and separateness, belonging and individuation, accommodation
and autonomy,” an “optimal balance” of which must be developed to ensure “healthy
described the following:

“Adolescents feel the need to move toward independence, and to do that, they feel
compelled to turn away from their childish ways. Implicit in this task is the need
to transform the relationship with their parents. … It is a process that cannot be
avoided. This is complicated, however, because along with wanting to venture out
and become independent, there is also a part of them that pulls toward wanting parents to take care of them. They do not want to break the emotional bond they have with their parents. Instead, they want a different balance in the relationship that allows for validation of their changing selves.” (p. 279)

While developing young women may want “a different balance” in their relationships with their parents as they mature, they still want to maintain their “emotional bonds,” and desire “validation of their changing selves” and continued “care” (Preto, 1999, p. 279). They too depend on the conditional love, support, and guidance of their parents to navigate the physical, psychological, and social changes, challenges, and opportunities they will likely experience as a result of and over the course of maturation (Bronstein, 2006; Crouter et al., 1995; Papini & Sebby, 1987, 1988; Petersen, 1988). Thus, parents maintain an essential role and considerable influence in the lives and development of their daughters (Erikson, 1968; Gilligan, 1982; Petersen, 1988).

**Pubertal and parent-relationship quality change: Implications for depression risk.** The developmental changes of puberty may bring about a state of disequilibrium and stress in the family system, as theory and research suggests; however, such a “metamorphosis” may not be inherently damaging to the individual or family. In fact, many individuals and families are able to successfully navigate these normative changes (Preto, 1999). It has been further suggested that what is of greatest importance is not the actual change (e.g., increased parental monitoring and control, etc.) that occurs within the parent-adolescent relationship; rather, it is young women’s subjective experience of parental relationship change (e.g., feeling more controlled and perception of low parental trust, etc.) that may have the greatest implications for development and adjustment.
(Steinberg, 1988). It is perhaps when young women perceive there to be a breakdown in, or absence of, emotional connection, acceptance, communication, or support with their mothers and fathers that difficulties ensue (Papini & Sebby, 1987).

Consistent with a cognitive-interpersonal model of depression (Abela & Hankin, 2009; Auerbach & Ho, 2012; Prinstein, Borelli, Cheah, Simon, & Aikins, 2005), the attributions maturing young women make regarding the real or perceived changes in the affective quality of their parent relationships may put them at risk for developing depression. Thus, a developing young woman who perceives her mother or father as withdrawing positive affect, and experiences increasing negativity and conflict in her parental interactions, may develop “depressogenic attributions” about her self (i.e., “deficient, unlovable, and/or unlikable”) and the cause, controllability, and consequences (i.e., support, etc.) of these new interpersonal changes that make her vulnerable to developing depression (Auerbach & Ho, 2012, p. 794). Such interpersonal experiences and subsequent attributions may be the “stress” that actualizes puberty-driven biological vulnerabilities, leading to the emergence of depression (Dahl, 2004; Frank & Young, 2000; Susman et al., 1991). However, parent-daughter relationships are not necessarily precluded from improving and growing over the course of pubertal development; therefore, improving relationships may prevent puberty-related depression vulnerabilities from actualizing (Cornwell, 2003).

No empirical studies were found that examined the prospective order of change (i.e., pubertal advancement, parental relationship quality deterioration, emergence of depressive symptoms) or the associations of simultaneous change in pubertal maturation, parent-daughter relationship quality, and depressive symptomology. Additionally, no
studies were located that directly examine whether changes in the parent-daughter relationship over time parallel changes in levels of depressive symptomology or the onset or resolution of depression. Despite this, theory and related empirical findings suggest this may be an important area of investigation to better understand the occurrence of depression among young women in pubertal transition.

The Present Study

The elevated risk of occurrence of depression among adolescent young women of pubertal age raises questions concerning the possibility of pubertal maturational effects (SAMSHA, 2012). Several studies have documented increases in depression with advancing development (Angold & Costello, 2006; Patton et al., 2008); however, these investigations have frequently been limited to relatively small, geographically restricted, and racially and ethnically homogenous samples, and few have examined longitudinal change (Graber et al., 1996; Negriff et al., 2008). Given this, the present study will examine whether the association between advancing maturation and rising depressive symptomology holds longitudinally among a diverse sample of adolescent girls.

Understanding the factors underlying the association between female pubertal development and depression is a difficult task given that both puberty and depression involve complex, interacting biological, psychological, and social processes (Cyranowski et al., 2000; Graber et al., 1996; Slater et al., 2001). Evidence suggests the physiological and neurocognitive-affective changes of female pubertal development make developing young women vulnerable to depression (Edwards et al., 2011; Frank & Young, 2000). However, it is perhaps only in the presence or absence of psychological and sociocultural stressors and resources that symptoms of depression might emerge (Dahl, 2004; Parker &
Given the importance of parental support as it concerns health and well-being, the absence of a high quality parent-adolescent relationship, or a disruption or deterioration in the relationship, may be factors that carry a sizable weight as it relates to depression risk in pubertal transition (Dahl, 2004; Parker & Brotchie, 2010). On the other hand, a stable, high quality parent-adolescent relationship may be a strong, protective force in ameliorating puberty-related depression risk (Beardslee et al., 2012; Frey & Röthlisberger, 1996; Houlberg et al., 2011; Printz et al., 1999; Wetter & Hankin, 2009).

Developmental theories and research highlight the essential role of mothers and fathers in socializing their daughters to, as well as protecting them from, premature exposure to the physical, relational, and cultural demands of womanhood (Bronstein, 2006; Crouter et al., 1995; Papini & Sebby, 1987; Petersen, 1988). From infancy to adulthood, the relationships girls have with their parents have been found to significantly influence important aspects of development and well-being (Gilligan, 1982). The relationships young women have with their parents may be especially important during pubertal transition (Cyranowski et al., 2000; Rose & Rudolph, 2006). Whereas warm, loving, supportive parental relationships have been suggested to protect adolescent young women from excessive distress during this challenging period, the absence of this emotional connection and support, and the presence of parent-child relationship conflict have been consistently associated with depression risk in the literature (Beardslee et al., 2012; Cohen & Wills, 1985; Thoits, 1982; Parker & Brotchie, 2012). Real or perceived deterioration in the affective quality of parent-daughter relationships over the course of puberty may place developing young women at particular risk for depression (Auerbach & Ho, 2012, Parker & Brotchie, 2010; Preto, 1999). Despite the important role of parents
in the lives of developing young women, surprisingly little empirical research has examined the risk or protective functions of the parent-daughter relationship as it concerns the occurrence of depression among young women in pubertal transition. The present study represents an initial step towards bridging this gap in the literature.

Drawing upon existent theory and empirical findings as delineated previously, the present study will test the following research hypotheses:

**Hypothesis 1.** Young women who advance in pubertal development will have significantly greater odds of experiencing a rise in depressive symptoms relative to those whose development remains stable.

**Hypothesis 2.** Young women with strong quality parent-daughter relationships will be significantly less likely to develop depressive symptoms in the context of pubertal advancement relative to those reporting lower quality relationships.

**Hypothesis 3.** Young women reporting simultaneous maturation and relationship deterioration will have significantly greater odds of developing depressive symptoms relative to those who advance in development but whose parental relationships do not decline.
CHAPTER II

METHODS

This research will be conducted utilizing select secondary data from the *National Longitudinal Study of Adolescent Health (Add Health; Harris, 2009)*, a comprehensive, nationally-representative, multi-year study of the individual (i.e., biology, personality, beliefs, behaviors, etc.), interpersonal (i.e., family relationships, peer groups/social networks, romantic relationships), and other contextual (i.e., schools, neighborhoods/communities) risk and protective factors that influence emotional well-being, health behaviors, and health status from adolescence through early adulthood (Harris et al., 2009). From an integrative life course perspective, *Add Health* was founded on the assumption that “adolescents, like all humans, not only are affected by the contexts in which they are embedded but also select and shape the environments they are exposed to” (Udry & Bearman, 1998, p. 243).

**Participants**

*Add Health.* A core sample of 12,105 adolescents in grades 7 through 12 during the 1994-1995 school year completed in-home interviews at Wave 1. The demographic composition of this sample is reflective of the diversity of the U.S. population at the time the study was designed and conducted (Udry & Bearman, 1998). Specific subpopulations, such as non-Hispanic Black adolescents from well-educated families ($n = 1,547$), Puerto Ricans ($n = 633$), Cubans ($n = 538$), Chinese ($n = 406$), individuals with disabilities ($n = 957$), and genetic siblings, were oversampled, making the total sample 20,745 of the originally 90,118 students identified (Harris et al., 2009). The public-access data was restricted to a sample of 6,504 participants (Harris et al., 2009; Udry & Bearman, 1998).
Response rates for each wave of *Add Health* are as follows: Wave 1 (Grades 7 – 12), 79.0% (*n* = 20,745), Wave 2 (Grades 8 – 12), 88.6% (*n* = 14,738). If not among the biological sibling or disability classification subsamples, participants who were high school seniors at Wave 1 were not eligible to participate in Wave 2 interviews. With the exception of this, no notable systematic variables at Wave 1 were found to differentiate participants who responded in Wave 2 relative to those who did not complete Wave 2 (Harris et al., 2009). Nearly three quarters (74.3%, *n* = 4,834) of the public-access data subsample participated at Wave 2.

**The present study.** Of the 3,356 Wave 1 female adolescents surveyed in 1995 whose data were available for public use, 75.1% (*n* = 2,519) also participated at Wave 2. In the present study, only adolescent young women completing Waves 1 and 2 were included. Additionally, given epidemiological reports that rates of female depression increase over the course of early and mid-adolescence, the present sample was age-restricted, with only participants between the ages of 12 and 16 included (SAMHSA, July 2012). This yielded a total of 2,000 initial participants eligible for inclusion. The sizable number of participants retained from Wave 1 to Wave 2 allowed for more than adequate power for the analyses outlined below. For an overview of sample demographic and household parent structure characteristics, see Table 1.

**Procedure**

**Sampling and recruitment.** Of the 26,666 high schools in the U.S. at the time of study design, a stratified random sample of 80 high schools was selected for participation in the *Add Health* study based on the following five variables: percentage of non-Hispanic White students, school type, school size, level of urbanization, and census
region (Harris et al., 2009; Udry & Bearman, 1998). Fifty-two schools met the eligibility
criteria and agreed to participate. Twenty-eight additional schools matching the 28
schools who did not agree to participate on the above noted five variables, as well as
three additional variables (i.e., percentage of non-Hispanic Black students, grade span,
census division) were randomly selected, making the total number of high schools
sampled equal to 80. For each high school without a 7th or 8th grade, one middle/junior
high feeder school per high school was selected for inclusion, making the total number of
schools sampled equal to 144. Each of the selected schools then produced a student
roster; however, parental consent (active or passive depending on specific school
procedures) was required for a student’s name to be added to the roster and released to
study personnel (Harris et al., 2009; Udry & Bearman, 1998).

**Administration.** Written parental consent and adolescent assent was required for
adolescents to participate in in-home interviews at Waves 1 and 2 (Harris et al., 2009;
Udry & Bearman, 1998). Wave 1 took place in the Spring and Fall of 1995, and Wave 2
was conducted the following academic year. The in-home questionnaire, collecting
information on such topics as adolescent demographics, development (i.e., puberty),
health and health behaviors (i.e., depression, substance use, sexual behavior), the quality
and structure of family relationships, peer networks and romantic partners, and academic
environment and performance, to name a few, was primarily verbally administered by a
trained interviewer and through the utilization of a Computer-Assisted Personal Interview
(CAPI) in which responses were entered and questions were personalized based on
adolescent responses to previous items (i.e., age, sex, relationship status, etc.). Potentially
more sensitive information was collected utilizing an Audio Computer Assisted Self-
Interview (ACASI) to decrease participants’ discomfort and increase disclosure and honesty in the presence of the interviewer, and possibly, parent figures as well. Interviews ranged from 60 to 120 minutes. Information pertaining to interviewer characteristics, interviewer observations regarding participants’ comfort level and boredom or impatience, as well as adolescents’ belief in the value of the study, was collected as part of the in-home interview to control for potential measurement biases (Harris et al., 2009; Udry & Bearman, 1998). Information regarding the option to receive instruction and complete study questionnaires in a language other than English was not readily available.

**Security.** Add Health investigators went to great lengths to protect the confidentiality of Add Health participants while allowing for an ambitious 13-year longitudinal study where participants were tracked during adolescence and into adulthood. Although specific details of what is described as a “rigorous security system” are not readily available, investigators enlisted a third-part contractor to manage the security of the data. Most notably, identification numbers were assigned to each participant. These identification numbers are not available to the public for distribution, and the design ensures that participants’ identities cannot be matched to their responses (Harris et al., 2009; Udry & Bearman, 1998).

**Measures**

Due to the comprehensive examination of a multitude of individual, family, community, and institutional factors that potentially impact adolescent health, Add Health researchers restricted the number of items within each study domain (Udry, n.d.). Thus, a number of measures were referenced and consulted in the development of the Add Health
study, but few instruments remained intact and utilized in their entirety. (For a list of referenced studies and instruments, see http://www.cpc.unc.edu/projects/addhealth/data/guides/refer.pdf.) The aggregate of items surveyed in Add Health were piloted and modified in line with pilot findings. While the depth of any one particular construct may be limited as a result, the breadth of the Add Health measurement design allows researchers to examine many interacting associations over time and across a multitude of emotional, behavioral, relational, and physical health domains (Udry, n.d.).

**Depressive symptomology.** Depressive symptomology was measured at Waves 1 and 2 utilizing select intact and slightly modified items from the *Center for Epidemiologic Studies – Depression Scale* (CES-D; Radloff, 1977). The CES-D is a widely used measure of depressive symptomology tapping into the frequency (*never or rarely, sometimes, a lot of the time, most of the time or all of the time*) of symptoms experienced over the past week on a 4-point (“0” to “3”) continuum. The majority of items that comprise the CES-D are worded in the negative direction, whereas several items are worded in a positive direction and reverse coded (Radloff, 1977). In Add Health, the wording of items was slightly modified to allow for interviewer administration (i.e., use of the pronoun “you” instead of “I”). Further, the choice was made by the original investigators to reword several items, likely to make the items more clear and meaningful to the adolescent population; however, the content of these modified items remained much the same (i.e., “It was hard to get started doing things” opposed to the original “I could not get going.”) (Crockett, Randall, Shen, Russell, & Driscoll, 2005). Two items from the original CES-D related to restless sleep and crying spells were administered in a separate section of the interview, and some modifications
were made to the wording, as well as to response options and time period; however, these items were coded in a way that is comparable to the original scale. The construction of the scale in this manner has been supported in epidemiological studies and other Add Health investigations (Rushton, Forcier, & Schectman, 2002; Russell, Crockett, Shen, & Lee, 2008). For the scale utilized in this study, see Appendix A.

Participant responses were summed across the 20 items, with possible scores ranging from 0 to 60. Higher scores are indicative of greater symptom severity (i.e., higher number of symptoms experienced, greater frequency of symptom expression). Based on classification cut-offs commonly found in both the general depression and adolescent depression literatures (Radloff, 1977; Rushton et al., 2002; Russell et al., 2008), scores < 16 are generally considered “minimal,” and those ≥ 16 are suggestive of clinically significant distress. Whereas scores between 16 and 23 are reflective of “mild” depressive symptomology and suggestive of “possible depression,” scores ≥ 24 are reflective of “moderate/severe” symptomology and indicative of “probable depression” (Rushton et al., 2002, p. 200). Computational means (i.e., person-mean imputation) were substituted for participants missing data on 4 or fewer items, and those missing data on 5 or more items were excluded from data analyses (Radloff, 1977).

In its original form, the CES-D has been viewed as reliable (e.g., α = .85 to .93) and psychometrically sound (Cuijers, Boluijt, & van Straten, 2008; Radloff, 1977; Roberts, Lewinsohn, & Seeley, 1991). Similarly adequate internal consistency coefficients were obtained in the present sample (w1: .84, w2: .86). The CES – D has been commonly utilized to document the presence of depressive symptomology among diverse samples of adolescents (Cuijers et al., Radloff, 1977; Roberts et al., 1991;
Thomas, Jones, Scarinci, Mehan, & Brantley, 2001; Van Voorhees et al., 2008), and the “meaningful comparison” of adolescents’ CES-D scores over time has been supported (Motl, Dishman, Birnbaum, & Lytle, 2005, p. 90). Despite its wide use in the literature, it is important to note that CES – D items and instructions were not developed inline with the Diagnostic and Statistical Manual of Mental Disorders (DSM – IV – TR; American Psychiatric Association [APA], 2000) criteria for a Major Depressive Episode, and depression risk derived from this measure should be interpreted with this in mind. This scale cannot substantiate a clinical depression diagnosis; however, items consistent with the DSM – IV – TR (APA, 2000) criteria for a Major Depressive Episode are reflected in scale items including: depressed mood (e.g., “You felt depressed.”), anhedonia (e.g., “You enjoyed life;” reverse coded), appetite disturbance (e.g., “You didn’t feel like eating, your appetite was poor.”), psychomotor retardation/fatigue (e.g., “You felt that you were too tired to do things.”), worthlessness/guilt (e.g., “You thought your life had been a failure.”), and concentration difficulties (e.g., “You had trouble keeping your mind on what you were doing.”).

To examine between-wave change in depressive symptomology, sum scale scores from each wave were subtracted, yielding raw difference change scores. To establish a cut-off for classifying significant change, the Reliable Change Index (RCI) was computed as proposed by Jacobson & Truax (1991). This statistic helps to determine a reliable point in the data whereby significant change is expected to occur after controlling for measurement error. In the present study, a statistically significant change in symptomology was established as follows: Stable (RCI: > - 0.84 and < 0.84; raw score equivalent: -3 to 3), Decreased (RCI: ≤ - 0.84; raw score equivalent: ≤ 4), and Increased
(RCI: ≥ 0.84; raw score equivalent: ≥ 4). The RCI cut-off of +/- 0.84 was selected based on sample distribution and proposed recommendations for similar scales, and suggests with 80% confidence that actual change has occurred (Wise, 2004). Groups were dummy coded for study analyses, with those in the Stable or Decreased (coded: 0) groups serving as the reference group. This allowed for the odds of reporting a significant between-wave rise in depressive symptomology (i.e., Increased; coded: 1), the outcome variable of interest, to be determined.

**Pubertal development status.** Female participants at Waves 1 and 2 were asked to rate the size of their breasts and the curvaceousness of their body at the present time relative to when they were in grade school. Each question began with a contextualizing statement (i.e., “As a girl grows up her breasts develop and get bigger.” “As a girl grows up her body becomes more curved.”). They were then asked, “Which sentence best describes you?” Responses ranged from 1 to 5, with a response of “1” suggesting no or very little change (i.e., “My breasts are about the same size as when I was in grade school.” “My body is about as curvy as when I was in grade school.”), and a response of “5” suggesting a significant amount of change (i.e., “My breasts are a whole lot bigger than when I was in grade school, they are as developed as a grown woman’s breasts.” “My body is a whole lot more curvy than when I was in grade school.”). These items are strongly and significantly correlated. Among the present sample, the correlation between breast development and curvaceousness was .59 (p < .01) and .58 (p < .01) at Waves 1 and 2, respectively. These two items were averaged to represent pubertal development in terms of secondary-sex characteristics. Participants denying the emergence of any secondary-sex characteristics (i.e., secondary sex average = 1.00) by Wave 1 were
considered *Late Developing* given no signs of development by age 12, the youngest age group included in the sample (Chumlea et al., 2003). Participants were also asked at both waves whether they “ever had a menstrual period,” and if so, the age at which this occurred. Based on epidemiological findings, the following classifications were made with regards to timing and onset: *Normal Menarche* (onset: ages 10 – 15); *Early Menarche* (onset: ≤ age 9); *Late Menarche* (onset: ≥ age 16) (Chumlea et al., 2003). For the pubertal development scale utilized in this study, see Appendix B.

Although the above noted items do not comprise the specific items of commonly used pubertal development scales, there is a great deal of overlap in structure and content (Carskadon & Acebo, 1993; Petersen, Crockett, Richards, & Boxer, 1988). Responses to the above pubertal indicators were grouped in a similar manner as outlined by Carskadon & Acebo (1993) and Petersen et al. (1988) and coded. Pubertal development status classifications for the present study were as follows: *Pre-pubertal* (coded: 0; 1.00 and pre-menarche), *Early Pubertal* (coded: 1; 1.50 – 2.00 and pre-menarche), *Early-mid Pubertal* (coded: 2; 2.50 – 3.00 and pre-menarche), *Mid-pubertal* (coded: 3; 2.50 – 3.00 and post-menarche), *Mid-late Pubertal* (coded: 4; 3.50 – 4.00 and post-menarche), and *Late Pubertal* (coded: 5; 4.50 – 5.00 and post-menarche). A young woman classified as Pre-pubertal had yet to achieve menarche as well as denied the start of any breast or hip development, whereas a young women classified as Mid-pubertal had achieved menarche and reported both breast and hips to be “somewhat” more developed relative to when in grade school. Participants classified as Late Pubertal were post-menarcheal and perceived their breast and/or hip development to be “as developed as a grown woman.”
To examine change in pubertal development over the 12-months between waves, change in pubertal development status groups were created by subtracting Wave 1 from Wave 2 pubertal development status scores. Resulting change scores were grouped as follows: *Stable* (coded: 0; raw score: 0), *Advanced* (coded: 1; raw score: ≥ 1), and *Regressed* (coded: -1; raw score: ≤ -1). Participants reporting pubertal regression were excluded from study analyses.

**Parent-daughter relationship quality.** The quality of parent-daughter relationships was established through a series of questions in which participants were asked about their relationships with their resident mother and father figures at Waves 1 and 2. For the purposes of this study, a “resident mother” or “resident father” was defined as a woman or man who functions as a mother or father in the respondent’s household. This could include a biological mother or father, stepmother or father, adoptive mother or father, grandmother or grandfather, etc.

For both “resident mother” and “resident father” figures, participants responded to five items on a five-point scale related to perceived closeness (“*how close do you feel to your ...*”), care (“*how much do you think [she/he] cares about you?*”), warmth/love (“*Most of the time, your [mother/father] is warm and loving toward you*”), communication (“*You are satisfied with the way your [mother/father] and you communicate with each other.*”), and overall relationship satisfaction (“*Overall, you are satisfied with your relationship with your [mother/father].*”). For both the “closeness” and “care” items, participants were to respond on a scale from 1 to 5, with a rating of “1” indicating “not at all” and a rating of “5” indicating “very much.” For the remaining items that comprise the mother-daughter and father-daughter relationship scales (i.e.,
“warmth/love,” communication,” “overall satisfaction”), participants rated their degree of agreement on a scale of 1 to 5, with a response of “1” indicating “strongly agree” and a rating of “5” indicating “strongly disagree.” These remaining three items were reverse coded so that all items are worded in the same direction. For the parent-daughter relationship quality scale utilized in this study, see Appendix C.

Items across the mother-daughter and father-daughter quality items were averaged to create a scale of parent relationship quality. Thus, only sample participants indicating both a “resident mother” and “resident father” figure and who completed PRQ-items are included in analyses. Cronbach alpha coefficients at Waves 1 ($\alpha = .89$) and 2 ($\alpha = .87$) for the combined parent relationship quality scale (i.e., average of mother and father relationship quality items) indicate quite strong internal reliability (George & Mallery, 2010). Possible scores ranged from 1 to 5.

To examine change in relationship quality, Wave 1 parent relationship quality scores were subtracted from Wave 2 responses, yielding raw difference change scores. The Reliable Change Index (RCI) was computed and utilized for categorization as described above with change in depressive symptomology. In the present study, statistically significant change in relationship quality was established as follows: Stable (RCI: $> - 0.84$ and $< 0.84$), Deteriorated (RCI: $\leq - 0.84$), and Improved (RCI: $\geq 0.84$). Groups were dummy coded for study analyses, with those in the Stable or Improved (coded: 0) groups serving as the reference group. This allowed for the moderating influence of relationship deterioration (i.e., Deteriorated; coded: 1) to be determined.
Analytic Strategy

For an overview of study variables examined in statistical analyses, see Table 2. The analytic strategy for the present research was largely influenced by statistical models and procedures within Bannink and colleagues (2013) and Patton et al. (2008), and represents somewhat of a hybrid between the two related, yet separate, research questions. Moderation analyses followed the statistical procedures of Baron and Kenny (1986). Study hypotheses were examined utilizing a series of binary logistic regression analyses, with change in depressive symptomology (ΔDEP; 0 = Stable/Decreased, 1 = Increased) serving as the dependent variable. Model significance (i.e., $\chi^2$, $p$ with $df$) and strength (i.e., Nagelkerke’s $R^2$) for reliably distinguishing a statistically significant increase in depressive symptomology are reported for models of interest. Statistical significance was established at the .05 level, and heuristics offered by Keith (2006) for determining the magnitude of variance explained by variables in the model was referenced for interpretation (i.e., Negligible: < .05, Small but Meaningful: $\geq .05$ and < .10, Moderate: $\geq .10$ and < .25, and Large: $\geq .25$). Individual predictors were examined for their unique contribution by evaluating the size and significance of the Wald test statistic (i.e., Wald’s $\chi^2$, $p$). Odds ratios (i.e., OR) and associated confidence intervals (i.e., 95% CI: Lower, Upper) for reporting a statistically significant between-wave rise in depressive symptoms are reported, and the following heuristics were referenced to qualify the size of the effect: Small: < 1.5, Medium: $\geq 1.5$, and Large: $\geq 5$ (Chen, Cohen, & Chen, 2010).
CHAPTER III
RESULTS

Descriptives

Sample. Due to study exclusions aimed at improving measurement and reducing the potential influence of other confounding variables, there was a dramatic reduction in the size of the sample available for study analyses. Specifically, participants who achieved menarche early (i.e., ≤ 9; \( n = 76 \)) or who initiated menarche late (i.e., ≥ 16; \( n = 20 \)) and/or whose silhouette development was late (i.e., = 1.00; \( n = 21 \)) were excluded from study analyses (Chumlea et al., 2003). A sizable proportion of the sample (i.e., 18.6%; \( n = 265 \)) reported between-wave pubertal regression, and were therefore also excluded from study analyses. This yielded a subsample of 1,157 eligible for analysis related to the association between pubertal advancement and parallel increases in depressive symptomology (i.e., Hypothesis 1). Further, only 798 participants were eligible for moderation analyses (i.e., Hypotheses 2 and 3) due to participants with non-dual parent family structures being excluded (\( n = 894 \)).

As with the original total sample (\( M = 14.49, SD = 1.19 \)), participants in the analysis subsamples (i.e., pubertal development inclusion, parent structure inclusion) ranged in age from 12 to 16 (\( M = 13.99 – 14.57 \)). Of the approximately 99.0% of participants enrolled in school, the breakdown by grade at Wave 1 was as follows: 19.4% in 7th, 22.3% to 23.6% in 8th, 25.6% to 26.2% in 9th, 24.8% to 25.3% in 10th, and 5.5% to 6.4% in 11th. Changes in the racial and ethnic composition of the sample with the imposition of the before noted exclusion criteria are important to highlight. Specifically, whereas 41.2% (\( n = 824 \)) of the original total sample was from a diverse, non-White
background, there was a trend toward the sample becoming increasingly non-Hispanic White when participants with early/late or regressed development (i.e., 64.0%, \( n = 741 \)) were excluded, and when analyses were restricted to dual parent households (i.e., 69.7%, \( n = 556 \)). The sharpest decrease was among participants identifying as non-Hispanic Black (i.e., 22.2% to 18.9% to 13.4%). Despite this, participants from non-Hispanic White, non-Hispanic Black, Hispanic/Latina, Asian, Native American, and Other (i.e., multiracial, etc.) backgrounds were represented in study analyses. The majority of participants across samples (i.e., original total sample, pubertal development inclusion, household parent structure inclusion) were born in the U.S. (i.e., 94.5% – 96.2%) and spoke primarily English at home (i.e., 93.4% – 94.6%). For a more detailed overview of sample demographic and household parent structure characteristics, see Table 1.

**Depressive symptomology and between-wave change.** Mean depressive symptoms were 12.05 (w1DEP; \( SD = 7.43 \); Min = 0, Max = 41) at Wave 1 and 12.55 (w2DEP; \( SD = 7.68 \); Min = 0, Max = 49) at Wave 2 (Table 3). The magnitude of skew (w1: 0.90, w2: 0.81) and kurtosis (w1: 0.60, w2: 0.54) at both waves were within acceptable limits based on recommended heuristics provided by Curran and colleagues (1996; i.e., skew: \( \approx 2.00 = \text{moderate}, \approx 3.00 = \text{large} \); kurtosis: \( \approx 7.00 = \text{moderate}, \approx 21.00 = \text{large} \)). At Wave 1, 27.4% (\( n = 317 \)) of participants reported clinically significant depressive symptoms (i.e., *Clinical Risk*), 69.1% (\( n = 219 \)) and 30.9% (\( n = 98 \)) of which fell in the “mild” (i.e., 16 – 23; “possible depression”) and “moderate/severe” (i.e., ≥ 24, “probable depression”) ranges, respectively. Nearly 73% (\( n = 840 \)) of participants reported *Minimal* (i.e., < 16) depressive symptomology at Wave 1. Levels of depressive
symptomology were similar at both waves for the parent structure inclusion analysis sample (i.e., w1: 11.27 vs. 12.05, w2: 11.88 vs. 12.55).

Regarding statistically significant between-wave change in depressive symptomology (ΔDEP; i.e., RCI: +/- 0.84; Raw Score Equivalent: +/- 4), the outcome variable of interest, 45.7% (n = 529) exhibited no statistically significant between-wave change in depressive symptoms (i.e., Stable), whereas 29.4% (n = 340) endorsed a statistically significant rise in depressive symptomology (i.e., Increased) (Table 4). Approximately 25% (n = 288) of participants reported a statistically significant decline in depressive symptoms (i.e., Decreased) over the 12-months between waves. Only 39.1% (n = 133) of participants reporting a significant rise in depressive symptomology across waves (i.e., Increased; n = 340) reported the between-wave onset (i.e., Wave 1: < 16, Wave 2: ≥ 16) of potentially clinically significant symptomology. This is relative to 17.1% (n = 58) of participants who exhibited clinically elevated symptomology at Wave 1 (i.e., Wave 1: ≥ 16) and reported a worsening of symptom distress between waves.

Rates of between-wave symptom change were comparable for the parent structure inclusion analysis sample (i.e., n = 798; Stable: 48.9%, n = 390; Increased: 27.8%, n = 222; Decreased: 23.3%, n = 186); however, slightly fewer participants in dual parent households exhibited a rise in depressive symptoms (27.8% vs. 29.4%).

**Pubertal development status and pubertal change.** Mean pubertal development status was 3.51 (w1PDS; SD = 0.84; Min = 1, Max = 5) at Wave 1 and 3.88 (w2PDS; SD = 0.86; Min = 1, Max = 5) at Wave 2 (Table 3). Pubertal development status was slightly negatively skewed at both waves (w1: -0.39, w2: -0.59). Kurtosis for each scale was within normal limits (w1: 0.67, w2: 0.26). Sample distributions were similar at both
waves for the parent structure inclusion analysis sample (i.e., w1: 3.47 vs. 3.51, w2: 3.81 vs. 3.88). With regards to across-wave pubertal change (ΔPDS), the explanatory variable of interest, 61.5% (n = 711) of participants with available and included data reported pubertal stability (i.e., Stable) and 38.5% (n = 446) reported pubertal advancement (i.e., Advanced) over the 12-months between waves (Table 4). Rates were similar for participants in the parent structure inclusion analysis sample, though slightly less reported between-wave pubertal advancement (i.e., 36.3.8% vs. 38.5%).

**Parent-daughter relationship quality and relationship change.** Among sample participants with available data at Waves 1 and 2 and who reported both a mother and father figure in the household (n = 798), mean parent relationship quality was 4.33 (w1PRQ; SD = 0.62; Min = 1.5, Max = 5.0) at Wave 1 and 4.26 (w2PRQ; SD = 0.60; Min = 1.6, Max = 5.0) at Wave 2 (Table 3). Parent relationship quality scales at each wave were negatively skewed (w1: -1.28, w2: -1.03), but remained within acceptable limits. Kurtosis for each scale was also within normal limits (w1: 1.89, w2: 1.23). With regards to between-wave change in parent relationship quality (ΔPRQ), the moderating variable of interest, 49.6% (n = 373) of participants reported no change in relationship quality (i.e., Stable), whereas 32.4% (n = 244) reported deterioration (i.e., Deteriorated) (Table 4). Less than 20% (i.e., 18.0%, n = 135) of participants reported improvement (i.e., Improved) in the quality of the parent-daughter relationship.

**Preliminary Analyses**

**Depressive symptoms and significant between-wave change in symptomology.** Participant age (r = .11, p < .01) and grade (r = .10, p < .01) were significantly, but weakly, positively correlated with Wave 1 depressive symptoms (w1DEP). Further, there
was a significant effect of racial-ethnic group on Wave 1 depressive symptoms (w1DEP), $F(5, N = 1,137) = 4.26, p < .01$; however, no significant between-group differences emerged in post hoc analyses (Tukey HSD). Though not statistically significant, participants identifying as non-Hispanic White ($M = 11.39, SD = 7.33$) were noted to have the lowest mean level of depressive symptoms at Wave 1, while those of Asian ($M = 14.49, SD = 6.52$) and Native American ($M = 14.49, SD = 7.76$) backgrounds had the highest. Mean levels of baseline depressive symptoms were 12.53 ($SD = 7.36$) and 13.51 ($SD = 8.14$) for non-Hispanic Black and Hispanic/Latina participants, respectively. Results must be considered in light of the fact that there were substantial differences in group sizes (i.e., $n$ range: 35 to 731).

Neither age or grade were significantly associated with change in depressive symptoms ($\Delta$DEP), \textit{Age}: $X^2 (1, N = 1,138) = 0.03, p = .87$; \textit{Grade}: $X^2 (1, N = 1,138) = 0.01, p = .95$. Further, the odds of reporting a significant between-wave increase in symptoms, relative to stability or symptom reduction, across racial-ethnic group ranged from 0.28 (i.e., 22.0\% \textit{Increased}) to 0.46 (i.e., 31.5\% \textit{Increased}). This association was not statistically significant, however, $X^2 (5, N = 1,138) = 4.34, p = .50$. Baseline depressive symptomology (w1DEP) was significantly associated with between-wave change in depressive symptoms ($\Delta$DEP), $X^2 (1, N = 1,138) = 35.97, p < .01$. Participants with higher levels of Wave 1 symptomology were less likely to report a significant between-wave rise in depressive symptoms, $b = -0.06$, Wald’s $X^2 = 32.46, p < .01$, OR: 0.95 [95\% CI: 0.93, 0.96].

\textbf{Pubertal development status and pubertal change.} Wave 1 pubertal development status (w1PDS) was moderately and significantly positively correlated with
age \( (r = .33, p < .01) \) and significantly but weakly associated with grade \( (r = .07, p = .03) \).

No significant effect of racial-ethnic group on Wave 1 pubertal development status (w1PDS) emerged, \( F(5, N = 1,137) = 1.26, p = .28 \).

Age, but not grade, was significantly associated with change in pubertal development status (\( \Delta \text{PDS} \)), \( \text{Age: } X^2 (1, N = 1,138) = 9.11, p < .01; \text{Grade: } X^2 (1, N = 1,138) = 0.70, p = .80 \). Specifically, the odds of reporting pubertal advancement across waves, relative to developmental stability, decreased with advancing age, \( b = -0.16 \), Wald’s \( X^2 = 9.08, p < .01, \text{OR: } 0.85 \) [95% CI: 0.77, 0.95]. No significant effect of racial-ethnic group on change in pubertal development status (\( \Delta \text{PDS} \)) emerged, \( X^2 (5, N = 1,138) = 0.55, p = .99 \). Baseline pubertal development status (w1PDS) was significantly associated with between-wave change in pubertal development status (\( \Delta \text{PDS} \)), \( X^2 (1, N = 1,138) = 66.50, p < .01 \). Specifically, participants more pubertally advanced at Wave 1 were less likely to report pubertal advancement across waves, \( b = -0.62 \), Wald’s \( X^2 = 60.37, p < .01, \text{OR: } 0.54 \) [95% CI: 0.46, 0.63].

**Parent-daughter relationship quality and relationship change.** Age \( (r = -.16, p < .01) \) and grade \( (r = -.08, p = .02) \) were significantly, but weakly, negatively correlated with Wave 1 parent relationship quality (w1PRQ). Participants identifying as Hispanic/Latina endorsed the lowest quality parent-daughter relationships \( (M = 4.05, SD = 0.61) \), whereas non-Hispanic White participants reported the highest \( (M = 4.36, SD = 0.63) \). Mean group differences were trivial in size, and no significant effect of racial-ethnic group on Wave 1 parent relationship quality emerged (w1PRQ), \( F(5, N = 792) = 1.87, p = .10 \).
Neither age, grade, or race-ethnicity were significantly associated with change in relationship quality ($\Delta PRQ$). \textit{Age}: $\chi^2 (1, N = 746) = 0.49, p = .49$; \textit{Grade}: $\chi^2 (1, N = 746) = 0.00, p = .98$; \textit{Race-ethnicity}: $\chi^2 (5, N = 746) = 0.19, p = .99)$. Baseline parent relationship quality (w1PRQ) was significantly associated with between-wave change in parent relationship quality ($\Delta PRQ$), $\chi^2 (1, N = 746) = 31.89, p < .01$. Specifically, participants with higher quality parent-daughter relationships at Wave 1 were significantly more likely to report relationship deterioration between waves $b = 0.80$, Wald’s $\chi^2 = 27.42, p < .01$, OR: 2.23 [95% CI: 1.65, 3.02].

For a summary of correlations between select demographic variables and Wave 1 and Wave 2 scales, see Table 5.

**Primary Analyses**

**Assumptions.** As a nonparametric statistical analysis, binary logistic regression is not bound to the assumptions of normality, linearity, or homoscedasticity, as is the case with linear regression; however, meeting the standard of these assumptions, particularly normality, may increase statistical power (Garson, 2012; Spicer, 2004). Overall, and within each level of the dependent variables (i.e., $\Delta DEP$; i.e., \textit{Stable/Decreased} vs. \textit{Increased}), no significant departures from normality were observed among continuous independent variables (Curran et al., 1996). Further, all Cook’s distance values were less than 1, therefore, ruling out the potential influence of outliers on model fit (Garson, 2012). While linearity in the traditional sense is not applicable to logistic regression, there is a requirement that there be a linear association between continuous independent variables and log odds for the dependent variable (Garson, 2012). This assumption was tested by using the Box-Tidwell test in which the interaction between each continuous variable and
its log function is examined in relation to the binary outcome. A non-significant interaction term provides support in favor of upholding this assumption (Garson, 2012). Interaction terms were non-significant at the .05 level; thus, this assumption was upheld.

As with multiple linear regression, multivariate logistic regression requires there not be high inter-correlations between independent variables, as this may distort coefficient estimates in the model (Garson, 2012; Spicer, 2004). To this end, multicollinearity diagnostics among continuous independent variables to be included in study analyses were examined, with particular attention to tolerance and variance inflation factors (VIF) values. Tolerance values less than .20, and VIF values greater than 5 are indicative of a problematic degree of multicollinearity (Cohen, Cohen, & West, 2003; Garson, 2012). No significant multicollinearity threats were observed (i.e., Tolerance values: .22 to .88; VIF values: 1.14 to 4.56), although age and grade, not surprisingly, were strongly related (i.e., Pearson correlation: .87; Tolerance: .22; VIF: 4.47). Given this, and in the interest of fitting the most parsimonious model, the decision was made to exclude grade from regression analyses; participant age, however, was maintained and included as a control given its association with baseline measures (e.g., w1DEP, w1PDS, w1PRQ).

The sample size of the present study was determined to be adequate for study analyses. A sample of over 100 and a minimum of 50 cases per independent variable is required to have sufficient power for meaningful findings in logistic regression, and these requirements were met (Pampel, 2000; Garson, 2012; Spicer, 2004; Wright, 1995). Another rule of thumb requires 5 or more counts for each categorical independent by dependent variable cross-tabulation cell, and this guideline too was met by the present
sample composition (Garson, 2012). Lastly, cases were not duplicated in the dataset, and no participants were included in more than one group within each independent variable or the dependent variable; therefore, the requirement for independence of observations was also upheld (Garson, 2012).

**Hypothesis 1. Predicting rise in depressive symptoms: Pubertal advancement and depressive symptomology.** Hypothesis 1 was tested utilizing a binary logistic regression analysis. Change in depressive symptomology (ΔDEP; 0 = Stable/Decreased, 1 = Increased) served as the dependent variable. Age, baseline depressive symptomology (w1DEP), and Wave 1 pubertal development status (w1PDS) were entered as controls into Block 1, along with change in pubertal development status (ΔPDS; 0 = Stable, 1 = Advanced), the explanatory variable of interest. Results are summarized in Table 6.

Model variables combined to explain a statistically significant, but “negligible” (i.e., 4.6%; Keith, 2006), amount of variance in change in depressive symptomology (ΔDEP) odds, $\chi^2(4, N = 1,138) = 37.80, p < .01$. Neither age or Wave 1 pubertal development status emerged as significant predictors in the model. \textit{Age: }$b = 0.07$, Wald’s $X^2 = 1.17, p = .28$; \textit{w1PDS: }$b = -0.05$, Wald’s $X^2 = 0.35, p = .56$; however, consistent with preliminary analyses, baseline depressive symptomology (w1DEP) was statistically significant and explained the most variance in change in depressive symptomology (ΔDEP) odds, $b = -0.06$, Wald’s $X^2 = 33.38, p < .01$, OR: 0.94 [95% CI: 0.93, 0.96].

Pubertal advancement (ΔPDS) was not associated with a significantly greater likelihood of reporting a between-wave increase in depressive symptomology (ΔDEP), $b = 0.10$, Wald’s $X^2 = 0.50, p = .48$, OR: 1.10 [95% CI: 0.84, 1.45]. This stands in contrast to the
hypothesized association; therefore, there is insufficient evidence to reject the null in favor of Hypothesis 1.

**Hypothesis 2. Predicting rise in depressive symptoms: Pubertal advancement and the moderating influence of parent relationship quality.** Hierarchical binary logistic regression was utilized to examine Hypothesis 2. Age, baseline depressive symptoms (w1DEP), and Wave 1 pubertal development status (w1PDS) were entered as controls into Block 1, along with Wave 1 parent relationship quality (w1PRQ; centered). Change in pubertal development status (ΔPDS) was entered into the next step, followed by the interaction between pubertal maturation and baseline parent relationship quality (ΔPDS ° w1PRQ) in Block 3. Results are summarized in Table 7.

Change in pubertal development (ΔPDS) remained a statistically non-significant predictor of between-wave increase in depressive symptomology (ΔDEP) when the influence of baseline parent relationship quality (w1PRQ) was accounted for in the model (Block 2), $b = -0.10$, Wald’s $X^2 = 0.30$, $p = .59$, OR: 0.91 [95% CI: 0.65, 1.28]. This was in spite of the fact that Wave 1 parent relationship quality (w1PRQ) did not emerge as a significant explanatory variable in the model, $b = -0.11$, Wald’s $X^2 = 1.34$, $p = .25$.

Further, results did not support the hypothesized (i.e., Hypothesis 2) moderating effect of parent relationship quality (w1PRQ) as the inclusion of the pubertal change by parent relationship quality interaction term (ΔPDS ° w1PRQ) in Block 3 did not reach statistical significance, $b = 0.15$, Wald’s $X^2 = 0.66$, $p = .42$, OR: 1.16 [95% CI: 0.82, 1.64].

**Hypothesis 3. Predicting rise in depressive symptoms: Pubertal advancement and parent relationship quality deterioration.** Hypothesis 3, which predicted a significant interaction between pubertal and parent relationship quality change, was
examined utilizing a hierarchical binary logistic regression. As above, control variables were entered into Block 1. Change in pubertal development status (ΔPDS) and change in parent relationship quality (ΔPRQ) were entered in Block 2, and the interaction between the two (ΔPDS ° ΔPRQ) was entered in Block 3. Results are summarized in Table 8.

The interaction model (i.e., Block 3) failed to reach statistical significance at the .05 level, Block: $X^2(1, N = 746) = 2.83, p = .09$, $\Delta R^2 < .01$; Model: $X^2(7, N = 746) = 38.45, p < .01$, $R^2 = .07$. Participants with simultaneous advancement and relationship deterioration had comparable depression symptom odds as those who matured but whose relationships improved or remained stable, $X^2(1, N = 281) = 1.11, p = .29$, OR: 1.26. Thus, Hypothesis 3 was not supported. As above, pubertal advancement (ΔPDS) remained statistically non-significant (Block 2), $b = -0.03$, Wald’s $X^2 = 0.03, p = .85$, OR: 0.97 [95% CI: 0.68, 1.38]. A statistically significant and positive main effect of relationship quality deterioration (ΔPRQ) did emerge (Block 2), $b = 0.68$, Wald’s $X^2 = 14.41, p < .01$, OR: 1.97 [95% CI: 1.39, 2.79]. Lastly, as was the case with all prior models tested, baseline depressive symptoms (w1DEP) was significant, $b = -0.07$, Wald’s $X^2 = 19.00, p < .01$, OR: 0.94 [95% CI: 0.91, 0.96].
CHAPTER IV
DISCUSSION

A number of studies to date have established significant associations between indicators of female pubertal development and depression (Angold et al., 1998, 1999; Ge et al., 2006; LeResche et al., 2005; Oldenhinkel et al., 2011; Paikoff et al., 1991; Patton et al., 2008). These findings suggest the risk of depression may increase with advancing development; however, the notion of a puberty-related depression risk has largely been inferred from studies limited by their cross-sectional designs. Despite change being central to the argument of a puberty-depression link, the association between concurrent pubertal advancement and the development of depressive symptoms has rarely been examined (Angold et al., 1998; Paikoff et al., 1991; Patton et al., 2008). The present research aimed to add to the literature by examining the association between within-individual pubertal and symptom change among adolescent girls who participated in the National Longitudinal Study of Adolescent Health (Add Health; Harris, 2009).

While the complex physiological, neurocognitive-affective, and psychosocial changes of puberty are argued to leave developing young women vulnerable to depression, a central premise of this research was that it is in the presence or absence of sociocultural stressors and resources that vulnerabilities are actualized and depressive symptoms emerge (Dahl, 2004; Frank & Young, 2000; Parker & Brotchie, 2010; Susman et al., 1991). Despite the dynamic changes theorized to occur in the family system paralleling normative development (Carter & McGoldrick, 1999; Preto, 1999), as well as the significant influence of parents in the development and well-being of adolescents (Hughes & Gullone, 2008; Stark et al., 2012), limited research to date has examined...
risks and resources within the parent-daughter relationship as it relates to emerging depressive symptomology at this developmental juncture. Grounded in an Integrated Life Course model of health and well-being (Harris et al., 2009; Harris, 2010), and drawing from diathesis-stress, stress-buffering, and family life cycle perspectives (Carter & McGoldrick, 1999; Cohen & Wills, 1985; Parker & Brotchie, 2010), this research further sought to examine interrelationships among pubertal advancement, the nature and stability of the parent-daughter relationship, and emerging depressive symptomology.

**Study Hypotheses and Summary of Findings**

Young women who advanced in development between waves were hypothesized to be significantly more likely to develop depressive symptoms over the same period relative to those whose development remained stable (*Hypothesis 1*). Pubertal advancement in the context of lower quality parent-daughter relationships was predicted to be a risk factor for developing depressive symptoms, whereas higher quality relationships were hypothesized to attenuate this risk (*Hypothesis 2*). Deterioration in relationship quality in the context of pubertal advancement was predicted to be associated with a heightened risk of developing depressive symptoms (*Hypothesis 3*).

An effect of pubertal advancement on developing depressive symptomology did not emerge in the present study (*Hypothesis 1*). Participants reporting between-wave maturation were no more likely to endorse a rise in depressive symptoms than those who perceived stable development. This was true after controlling for the potential influence of age, initial development status, and baseline depressive symptomology, and remained so after parent-daughter relationship quality variables were included in the model. Further, the hypothesized moderating effects of parent-daughter relationship quality were not
supported. Specifically, a buffering effect was not found, and pubertal advancement in the context of low quality parental relationships was not associated with greater odds of developing depressive symptoms (*Hypothesis 2*). The likelihood of developing depressive symptoms was comparable among participants reporting between-wave pubertal advancement, irrespective of whether they perceived deterioration, stability, or improvement in relationship quality (*Hypothesis 3*).

**Review of Study Findings**

Research on the intersection of pubertal advancement, parent-daughter relationship quality, and depressive symptoms to date has been limited, making the placement of findings in an empirical context difficult. In fact, only one comparable study was located. As was the case in the present study, Benjet & Hernández-Guzmán (2002) identified no moderating effects of either baseline mother-daughter or father-daughter relationship affect and communication quality on menarche onset in relation to the development of depressive symptoms in their longitudinal (i.e., baseline, 6-month follow-up) study of Mexican female students (*M* age = 12.30, *SD* = 0.96; *n* = 512), grades 5 through 7. Several important differences in sampling (i.e., age range, ethnic background) and measurement (i.e., indicators of pubertal development, examination of change in relationship quality, etc.) exist between the referenced study and current research; however, both failed to establish an association between pubertal advancement and the development of depressive symptoms.

As described, girls more advanced in development have been found to exhibit more symptom distress, higher rates of clinically significant symptomology, and greater odds of onset depressive symptoms relative to those in earlier stages of development.
(Angold et al., 1998, 1999; Ge et al., 2006; LeResche et al., 2005; Oldenhinkel et al., 2011; Paikoff et al., 1991; Patton et al., 2008). However, few empirical investigations have directly examined between-wave pubertal advancement in association with symptoms of depression. Of the three that were located, all differed from the current research in meaningful ways (Angold et al., 1998; Benjet & Hernández-Guzmán, 2002; Patton et al., 2008). Only one (Benjet & Hernández-Guzmán, 2002) examined within-individual pubertal change over time in association with parallel changes in depressive symptomology, as was the case in the present study; however, as suggested, their study was restricted to the measurement of menarche only, not capturing other aspects of development and pubertal change, and the sample was younger (i.e., $M$ age = 12.3, $SD = 0.96$) and less diverse (i.e., Mexican only) than participants within the current research.

Although not directly analogous in sampling and design, Patton et al. (2008) found active pubertal change to be associated with the development of depressive symptoms, irrespective of where young women were in the development process. More specifically, Patton and colleagues identified a pattern in which prevalence rates of “elevated depressive symptomology” among female participants from the U.S. and Australia, ages 10 to 15, increased from baseline to 12-months follow-up for those reporting between-wave pubertal stage progression (i.e., change in rates between-waves by pubertal transition group; “early to mid-”: $+4\%$; “mid- to late-”: $+8\%$). A similar pattern did not emerge among developmentally stable groups for which rates were stable or slightly decreased (i.e., change in rates between-waves by pubertal constant group; “early puberty constant:” $-2\%$; “mid-puberty constant:” $-2\%$; “late puberty constant:” $0\%$). This finding is inconsistent with that of the present research in which young women
advancing in development were no more likely to report an increase in symptoms than those whose development remained stable. However, it is important to note that neither age, relative pubertal timing (i.e., early, late), or baseline symptomology were controlled for, as was done in the current research. Further, whereas intra-individual change in depressive symptomology was the primary point of analysis in both Benjet & Hernández-Guzmán (2002) and the present study, the referenced study was limited to the comparison of group (e.g., “remained mid-puberty” vs. “mid- to late puberty transition,” etc.) differences in rates of elevated symptoms at 12-months follow-up relative to baseline.

The absence of a significant association between female pubertal advancement and parallel development of depressive symptoms in the present study suggests that such an association does not hold when within-individual pubertal and symptom change are examined concurrently over time in a large, diverse sample, and when the effects of age and normatively early or late development are controlled for. However, there are several considerations, both unique to and extending beyond the current research, that are important to keep in mind prior to drawing definitive conclusions.

**Study-specific considerations and limitations: Implications for findings.**

**Parent-daughter relationship quality.** The fact that pubertal change was not a risk factor for increasing depressive symptomology could, in part, be explained by the context of relatively strong quality relationships that characterized the majority of sample participants. Study participants at both waves overwhelmingly endorsed high quality relationships with their mother and father figures (w1: $M = 4.33$, $SD = 0.62$, Min/Max: 1.5, 5.0; w2: $M = 4.26$, $SD = 0.60$, Min/Max: 1.6, 5.0). Even among the 32% ($n = 244$) who perceived deterioration in relationship quality across waves, average to high average
quality parental relationships were largely maintained (w2: $M = 3.90$, $SD = 0.57$, Min/Max: 2.00, 4.70). Although scale distributions and group sizes were within acceptable limits for purposes of analysis, the fact that lower quality (i.e., baseline) and more significantly declining (i.e., between-wave deterioration) parent-daughter relationships were not captured may have limited opportunities for detecting moderation effects. It is perhaps only when relationships are or become of low quality that pubertal advancement is associated with a heightened risk of emerging depressive symptomology (Hypothesis 2, 3). These points must be considered in light of limitations and other relevant considerations pertaining to the sampling and measurement of depressive symptomology, pubertal development status, and pubertal and symptom change.

**Depressive symptomology.** Intra-individual change in depressive symptomology was the primary point of analysis in the present study. The way depression is operationalized and measured has significant implications for the findings yielded in any given study. Examining the frequency or intensity of self-reported depressive symptomology at a given point in time may be suggestive of but is not definitely indicative of a clinically diagnosable mood disorder. This holds true for the measurement of symptom change as well. A between-wave increase in depressive symptoms in this study should not be interpreted as the onset of a clinically significant, diagnosable depressive disorder. In fact, of the 29% ($n = 340$) participants exhibiting a significant rise in depressive symptomology across waves, only 39% ($n = 133$) reported the onset (i.e., Wave 1: < 16, Wave 2: ≥ 16) of potentially clinically significant distress.

Change was established based upon the Reliable Change Index (RCI; Jacobson & Truax, 1991), which draws upon the test-retest reliability of a measure to determine a
point in the data in which statistically significant, reliable change can be expected to have occurred. In the present study, the RCI cut-off was established to reflect 80% confidence actual change has occurred (Wise, 2004). An 80% cut-off has been used with similar scales; however, there was a limited distribution of symptom change within the study sample that restricted a higher confidence criterion (i.e., 90% or 95%) from being utilized. Notably, even with a lower confidence criterion, nearly half (i.e., 46%, n = 529) the study sample exhibited stable depressive symptomology. This is relative to 29% (n = 340) who reported a significant rise in level of depressive symptoms (i.e., Increased) between waves. Although group sizes were judged to be adequate for study analyses, the fact that rates of increasing symptomology were not higher among the sample may have limited the ability to detect the hypothesized main effect and moderation associations.

The categorization of individuals along a number of dimensions (e.g., pubertal, relationship quality, depressive symptom change) and the collective examination of these variables may have limited study power. For instance, in spite of an initially moderate sized sample, only 85 participants with eligible data fell into one primary risk group of interest (i.e., simultaneous pubertal advancement and relationship quality deterioration), 25 of which reported a rise in depressive symptoms. This is relative to 126 participants whose development, relationship quality, and level of symptom distress remained stable. It is important to note that while potentially problematic from a statistical perspective, the fact only 85 participants fell within this risk group suggests this may be a low frequency phenomenon, not reflective of the general population of developing young women’s experience. Further, given only 29% (n = 25) of this risk group reported developing or worsening depressive symptomology suggests maturation and relationship deterioration
may not be a significant risk factor for depression. As such, parent-daughter relationship quality deterioration in the context of pubertal advancement would be unlikely to explain any heightened incidence of depression among adolescent females of pubertal age.

**Pubertal-development status.** The present study only captured a relatively small segment of the pubertal development process. The majority of participants endorsed development that was well underway at the initiation of the study (i.e., w1: $M = 3.61$, $SD = 0.92$; 35% Mid-Pubertal, 45% Mid-Late Pubertal, 16% Late Pubertal), with only 11% ($n = 198$) of study participants pre-menarche at baseline. The limited distribution of participants along indicators of pubertal development limits the generalizability of study findings to girls in mid- to later stages of development. This fact has significant implications for detecting simultaneous pubertal change and emerging depressive symptoms. For instance, 16% ($n = 251$) of sample participants reported their development to be complete at the outset of this study. As such, this subgroup (w1: $n = 251$; listwise: $n = 236$) was at the “ceiling” of development, and therefore, was unable to advance. The decision was not made to exclude this subgroup from study analyses; however, 41% ($n = 97$) were excluded from study analyses for reporting pubertal regression across waves. While 31% ($n = 446$) of participants from the original total sample evidenced pubertal stage advancement between waves, 50% ($n = 711$) perceived development that was stable. There may have been greater opportunities to detect pubertal progression effects if girls in earlier stages of development were more represented in the study sample.

Whereas breast development and menarche status are commonly utilized and established indicators of pubertal development stage, the current research included degree
of hip development to create a composite pubertal development stage index instead of the more commonly used indicator of pubic hair development. There is evidence that fat redistribution and hip development parallels breast and pubic hair development (Hillman & Biro, 2010; Mihalopoulos et al., 2010), but comparable stage specifications have not been established in research as is the case with breast, pubic hair development, and menarche status (Carskadon & Acebo, 1993; Marshall & Tanner, 1969; Petersen et al., 1988; Tanner, 1962). As such, it is possible that the stages defined in this research do not parallel those from studies in which a pubertal stage by depression link has emerged.

Further, item stems related to breast and hip development simultaneously presented two separate bases upon which participants were to rate their development. Whereas one item stem is consistent with the construct of interest (i.e., pubertal development status – development relative to a fully developed woman’s), the other reflects perceptions of retrospective change (i.e., development at present relative to when in grade school) (Appendix B). Not only may participants have differed in which item clause they attended to in their responses (i.e., self relative to developed woman versus current development relative to development in grade school), but it is conceivable that some interpreted these items differently at each wave, confounding the measurement of pubertal change. This may explain the unexpected occurrence of pubertal regression, which 19% of total sample participants reported (n = 265).

The occurrence of pubertal regression in the present study highlights the potential limitation of self-report measures of pubertal advancement in spite of arguments for their validity (Graber et al., 1996). No subjects reported between-wave pubertal regression in menarche status; rather, pubertal regression was only reported on anchored Likert-scale
items related to perceived degree of breast and hip development. Given this, pubertal change in the present study may be related to physical development, but it may also reflect changes in perception unrelated to such development (Dorn, Dahl, Woodward, & Biro, 2006). Although not unique to the present study, this is of significance and might have limited the ability to detect an effect of pubertal and symptom change.

**General considerations and limitations: Implications of study findings.** As suggested, the notion that pubertal advancement is associated with a heightened risk of developing depressive symptomology has largely been inferred from cross-sectional research designs. While significant positive associations between indicators of female pubertal development and depression have been found in numerous single-time point and longitudinal studies, it is important to note that empirical support has not been consistent, and effect sizes yielded have at times been surprisingly small in magnitude relative to the theorized effects (Angold et al., 1998, 1999; Angold & Costello, 2006; Brooks-Gunn & Warren, 1989; Graber et al., 1996; Negriff et al., 2008; Paikoff et al., 1991). Further, effects have not always been “over and above [the] effects of age or relative pubertal timing,” and studies have commonly been limited by “small and nonrepresentative samples” (Angold & Costello, 2006, p. 925). Thus, the present study’s failure to establish a significant association between pubertal advancement and the development of depressive symptoms is not unprecedented.

In spite of these points, findings across a number of studies suggest there is likely “some aspect of puberty itself [that] is important in the appearance of the female preponderance to depression” (Angold et al., 1998, p. 58); however, reliance on cross-sectional research has limited understanding of the nature and course of this association.
For instance, differences in prevalence rates of elevated depressive symptomology or DSM–IV depressive disorders could be associated with a puberty-driven vulnerability for the development of depression. However, the fact that few studies have examined within-individual pubertal change and the parallel development of depressive symptomology raises the possibility that observed findings are attributable to other, potentially related but confounding, aspects of female adolescent development. Findings of the current research in which within-pubertal advancement was not associated with parallel increases in depressive symptoms suggests this may be a reasonable conclusion; however, this point must also be considered in light of the sampling and measurement limitations previously discussed.

Further, whereas findings from a number of studies suggest the risk of depression or symptoms of distress rise with each advancing level of development (Angold & Costello, 2006; Patton et al., 2008), others have raised the possibility of a puberty-related “critical period” for depression over the course of pubertal transition (Edwards et al., 2011; Obradovic & Hipwell, 2010). For example, in an early single-time point, cross-sectional study on the topic, Brooks & Warren (1989) found the association between serum estradiol level, an indicator of pubertal development status, and depressive symptoms to be curvilinear in nature, after controlling for age. Specifically, the authors observed a significant trend in which mean levels of depressive affect increased across three consecutive categorical levels of estradiol (2 – 25 pg/ml, 26 – 50 pg/ml, 51 – 74 pg/ml), but then decreased when estradiol levels (75-100 pg/ml) reached a point in late puberty where secondary sex characteristics can be expected to be well developed and menarche already achieved. The present study did examine within-individual pubertal
stage advancement; however, pubertal stage by stage change interaction effects were not examined. It is possible pubertal advancement would be associated with a rise in depressive symptoms for girls in earlier stages of the development process; however, given the limited distribution of participants on indicators of pubertal development as described, the present study was not designed to capture such a “critical period” effect.

Additionally, the measurement of pubertal development status is a difficult task. A number of proxy indicators have been developed that correlate with puberty-related hormone markers and structural and functional states in the adolescent brain (Angold et al., 1998, 1999; Forbes et al., 2010; Marshall & Tanner, 1969; Shirtcliff et al., 2009); however, defining, categorizing and statistically controlling potentially “confounding” aspects of development remains a significant challenge to researchers (Angold & Costello, 2006; Dorn et al., 2006; Graber et al., 1996; Negriff et al., 2008; Reynolds & Juvonen, 2012). The present study took a stage approach to the measurement of pubertal development. There is precedence for examining pubertal development in this manner, and a stage approach has been the most widely used in research. However, some have argued that continuous measures of pubertal development are better indicators of maturational processes. This is because puberty “is a continuous process and not a single event” (Sadeh, Dahl, Shahar, & Rosenblat-Stein, 2009, p. 1603), and within- and between-individual variations in the course or sequence of pubertal development exist (Colvin & Abdullatif, 2012; Graber et al., 1996; Marceau et al., 2011).

The operationalization and measurement of pubertal development status has significant implications for defining and categorizing pubertal change, as well as examining the impact of such change in the lives of developing young women. Benjet &
Hernández-Guzmán’s (2002) study of Mexican female students speaks to this empirical challenge. Specifically, menarche onset over the 6 months between study waves was not associated with a significant change in depressive symptoms; however, girls less than 6 months post-menarche at Wave 1, and therefore between 6 and 12 months post-menarche at Wave 2, did exhibit a significant rise in depressive symptoms. Such a finding could represent a critical period of depression risk in puberty; however, it may also highlight the limitation of relying on proxy indicators of pubertal change processes. Menarche is one pivotal component of female puberty; however, other meaningful changes in development may be underway.

Similarly, it is possible that established pubertal change groups in the present research were not completely distinct. Young women who did not move between defined pubertal stages across waves, and therefore were categorized as Stable, may have reported the same amount of actual change in breast or hip development as someone categorized as Advanced whose perceived change moved her to a more advanced stage of development. Such a limitation could have limited this research in detecting a link between pubertal change and change in depressive symptomology. Although not a unique limitation, differences in indicators comprising the pubertal development stage index utilized in this research make this point important to carefully consider.

**Implications of Study Findings**

The reliance on cross-sectional study designs and the relative dearth of longitudinal studies examining within-individual changes in depressive symptoms may have limited researchers in developing a clearer understanding of possible puberty-driven vulnerability. This makes understanding the implications of non-findings within the
current research a challenge. Notable limitations in sampling and measurement within the
current research further complicate this task.

If in fact valid and reliable, study findings indicate pubertal advancement is not a
risk for emerging depressive symptomology, and other variables not included in the
present study are responsible for the significant, between-wave increase in depressive
symptoms among 29% \((n = 340)\) of sample participants. As such, the significant
association between pubertal development and depression established in a number of
existing studies limited by their cross-sectional nature may be an artifact of group
differences along related, but unexamined, confounding variables.

From a family life cycle perspective, maintaining “emotional bonds,” securing
“validation of [one’s] changing [self],” and receiving continued “care” in the midst of
developmentally-normative personal and family adaptation is considered an essential
need among developing adolescents (Preto, 1999, p. 279). The inability to do so is argued
to be a risk for maladjustment and distress (Papini & Sebby, 1987; Steinberg, 1988). The
low representation (i.e., \(n = 85\)) of participants reporting simultaneous pubertal
advancement and relationship quality deterioration was problematic in terms of
addressing Hypothesis 3; however, it suggests the majority of families are able to
navigate the changes of young women’s development without disrupting the affective
quality of parent-daughter relationships. The fact that sample participants were
predominately in mid- to later stages of development from the outset of the study and
parent-daughter relationship quality was largely perceived to be strong among the study
sample only speaks further to this point. Additionally, 18% \((n = 135)\) of participants
perceived a strengthening of the parent-daughter relationship across waves, suggesting
there may even be new opportunities for connection, growth, and support within this developmental period.

**Additional Limitations and Future Directions**

Pubertal advancement did not emerge as a risk factor for the development of depressive symptomology in this research. Despite this, future research on the topic is warranted given study-specific sampling and measurement limitations prevented definitive conclusions from being drawn. The fact so few studies have examined concurrent, within-individual pubertal and symptom change is a weakness in the literature. Additional longitudinal research on the topic may help to clarify the nature of associations that have been established between pubertal development and depression in cross-sectional studies. It may also serve to rule-out a female puberty-depression link if this is in fact the case.

**Pubertal development status.** As described, development was well underway at the outset of this study for a sizable portion of the sample. The implications of this for detecting between-wave pubertal change (i.e., main effect, pubertal stage by advancement interaction) and for the generalizability of study findings were noted. Given the maturation process can span several years, it has been suggested it would take 10 or more years to capture the pubertal processes of any given cohort (Graber et al., 1996; Petersen, 1983). Future research would benefit from a longitudinal model in which development and depressive symptoms are tracked at multiple points over an extended period of time.

Limitations related to the measurement of pubertal development status and pubertal change, both unique to and extending beyond the present study, should be addressed in future research. Using validated indicators of pubertal development is
particularly important if such measures are intended to serve as proxies for the physiological and neurocognitive-affective changes of female puberty. Improvement in such measures should also allow for more meaningful comparisons between study findings and existing research. Attempts should also be made at reducing perceptual biases, as change noted within a given study may be due to actual change or changes in perception regarding physical maturation. Although a young women’s perception of her development may be important to consider, future research would be strengthened by obtaining collateral, trained observer-ratings of development along a systematic measure of development, such as that developed by Tanner (Marshall & Tanner, 1969).

**Parent-daughter relationship quality.** The limited range of scores among indicators of parent-daughter relationship quality, as well as the small degree of between-wave relationship deterioration, had significant implications for examining the nature, stability, and influence of parental relationship quality in interaction with pubertal progression. Future research may be strengthened by sampling from a population, such as a clinical one, that includes greater initial variability in parent-daughter relationship quality and greater numbers showing meaningful decreases in quality over time. However, it is important to note that doing so would limit the generalizability of study findings. Additionally, the measurement of relationship quality was solely based on participant self-report. “Our understanding of ‘parent-adolescent relationships may be incomplete if we rely on a single reporter to represent a relational property” (McWey, Claridge, Wojciak, & Lettenberger-Klein, 2015, p. 257). Future research would be strengthened by the examination of both self-report and collateral ratings of relationship quality (Bogenschneider & Pallock, 2008).
Although items were highly correlated, the present study involved examination of an averaged value reflecting mother-daughter and father-daughter relationship quality. There is a potential that meaningful differences exist between mother-daughter and father-daughter relationships as it concerns pubertal advancement and depressive symptomology (Steinberg, 1988; Videon, 2002). Future research might examine the ways in which mother-daughter and father-daughter relationships change in the context of female pubertal development, as well as the implications of such change. Further, while the present study considered both biological and non-biological parent figures, it did not include single parent or same-sex households despite trends away from the “nuclear” or “traditional” family structure (Child Trends Data Bank, 2015). The decision not to consider single parent families led to a dramatic reduction in sample size, reducing study power. Future research should be more representative of U.S. family structures.

Cultural limitations and future directions. The present study sample was nationally representative at the time of data collection (i.e., 1995 – 1996); however, adolescents have become the most diverse segment of the U.S. population (Colby & Ortman, 2015). According to the U.S. Census Bureau (2009), of the nearly 20 million adolescent girls in the U.S. at the time, an estimated 42% were reported to be of color, and this was projected to increase. Among the original total research sample, 41% of participants described themselves as being non-Hispanic White. While comparable to the 42% estimate, important differences exist in the composition of racial-ethnic minority subgroups currently. Most notably, whereas 24.4% of children and adolescents in the U.S. were Hispanic or Latino in 2014 (Colby & Ortman, 2015), only 5% of sample participants identified as Hispanic/Latina in the study cohort. Additionally, as of 2013,
24% of children and adolescents in the U.S. were from first or second generation immigrant families (ChildStats.gov, 2014). In 2012, 22% of children and adolescents predominately spoke a language other than English at home (ChildStats.gov, 2014). This is relative to 6.7% in the original total sample. These facts highlight the necessity of examining the intersection of development, parent-daughter relationships, and depression within a cultural context. Doing so not only ensures study findings will have relevance to the diverse adolescents and family systems with the U.S., but also ensures the potential screening and intervention efforts that follow will be culturally sensitive and meaningful.

Preliminary analyses within the present study further highlight the necessity of examining study variables in a cultural context. Specifically, an effect, albeit small, of racial-ethnic group on baseline depressive symptomology was observed, with those identifying as non-Hispanic White having the lowest mean level of depressive symptoms at Wave 1, and those of Asian and Native American backgrounds having the highest. Further, while no significant effect of racial-ethnic group on Wave 1 parent relationship quality emerged, significant differences in racial-ethnic group sizes and the relative limited representation of racial-ethnic minorities overall may have precluded an effect from being identified if there is in fact one. Given this, racial and ethnic group differences may be important to consider more fully in future research. Doing so would require a study more representative of the U.S. adolescent population and with larger, more representative group sizes.

While the role and potential influence of ethnicity should be examined in future studies, ethnicity in itself is a complex construct and “intersects with class, religion, politics, geography, the length of time a group has been in this country, the historical
cohort, and the degree of discrimination the group has experienced” (Hines Almeida, Preto, Weltman, & McGoldrick, 1999, p. 69). As such, the intersection of other associated cultural variables should also be included in future research. Cultural differences that may exist within the family system itself (e.g., level of acculturation, etc.) may also be important to consider at this developmental juncture as opposing cultural values, traditions, and role expectations may become a particular point of contention and have meaningful implications for a young women’s well-being and adjustment (de las Fuentes & Vasquez, 1999; Denner & Guzmán, 2006; Juang & Cookston, 2009; Nicolas, DeSilva, Prater, & Bronkoski, 2009).
REFERENCES


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*Note. Initial Sample: n = 2,000. Pubertal Development Inclusion: n = 1,157; excludes participants with Early or Late Development, as well as between-wave Regressed PDS. Parent Structure Inclusion: n = 798; excludes participants with single parent or no parent households.*
Table 2

Overview of Primary Study Variables

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<th>Depressive Symptomology Variables</th>
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<td>Items/Factors: Depressed Affect, Positive Affect, Somatic Symptoms, Interpersonal</td>
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<th>Pubertal Development Status Variables</th>
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<td>Items: Breast Development, Silhouette/Hip Development, Menarche Status</td>
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<td>Values (Codes)</td>
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<td>-----------------------------------</td>
<td>------------</td>
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<td>w1PDS, w2PDS</td>
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<th>Parent Relationship Quality Variables</th>
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<td>Items: Closeness, Perceived Care, Warmth/Love, Communication Satisfaction, Overall Relationship Satisfaction</td>
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Table 3

*Measure Distributions by Sample*

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*Note. Initial Sample: n = 2,000. Pubertal Development Inclusion: n = 1,157; excludes participants with Early or Late Development, as well as between-wave Regressed PDS. Parent Structure Inclusion: n = 798; excludes participants with single parent or no parent households.*
Table 4

*Frequencies of Between–Wave Change in Primary Study Variables*

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<tr>
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<td></td>
<td>n</td>
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<table>
<thead>
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<th>Pubertal Development Inclusion</th>
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<td>Improved</td>
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*Note.* **Initial Sample:** $n = 2,000$. **Pubertal Development Inclusion:** $n = 1,157$; excludes participants with *Early* or *Late Development*, as well as between-wave Regressed PDS. **Parent Structure Inclusion:** $n = 798$; excludes participants with single parent or no parent households. **Change in Depressive Symptomology (ΔDEP):** Determined by 80% RCI (+/- 4). **Change in Pubertal Development Status (ΔPDS):** Determined by absolute changes in PDS stage between waves. **Change in Parent-daughter Relationship Quality (ΔPRQ):** Determined by 80% RCI.
Table 5
Correlations among Study Variables

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Note: ns = non-significant, * p < 0.05, ** p < 0.01, *** p < 0.001.
Table 6

*Predicting Rise in Depressive Symptoms: Associations with Pubertal Advancement*

<table>
<thead>
<tr>
<th>Model</th>
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<th>$p$</th>
<th>Nagelkerke $R^2$</th>
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</tr>
<tr>
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Note. Results of logistic regression analysis for Change in Depressive Symptomology ($\Delta$DEP; $0 =$ Stable or Decreased, $1 =$ Increased). $n = 1,138$. w1DEP: Wave 1 Depressive Symptomology. w1PDS: Wave 1 Pubertal Development Status. ΔPDS: Change in Pubertal Development Status ($0 =$ Stable, $1 =$ Advanced). Constant included in model, but not shown in table. $ns =$ non-significant, $* p < .05$, $** p < .01$, $*** p < .001$. 
Table 7

Predicting Rise in Depressive Symptoms: Pubertal Advancement and the Moderating Influence of Parent Relationship Quality

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<td>Nagelkerke $R^2$</td>
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<td>$ns$</td>
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Note. Results of hierarchical logistic regression analysis for Change in Depressive Symptomology (ΔDEP; 0 = Stable or Decreased, 1 = Increased). $n = 793$. w1DEP: Wave 1 Depressive Symptomology. w1PDS: Wave 1 Pubertal Development Status. w1PRQ: Wave 1 Parent Relationship Quality (centered). ΔPDS: Change in Pubertal Development Status (0 = Stable, 1 = Advanced). ΔPDS $\times$ w1PRQ: Change in Pubertal Development Status (ΔPDS) by Wave 1 Parent Relationship Quality (w1PRQ; centered) interaction term. Constant included in model, but not shown in table. $ns = $ non-significant, $^* p < .05$, $^{**} p < .01$, $^{***} p < .001$. 
Table 8

Predicting Rise in Depressive Symptoms: Pubertal Advancement and Parent Relationship Quality Deterioration

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<th>( \chi^2 (df) )</th>
<th>p</th>
<th>Nagelkerke ( R^2 )</th>
<th>( \chi^2 (df) )</th>
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<td>2.83(1)</td>
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<td>2</td>
<td>21.14(4)</td>
<td>***</td>
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<td>35.63(6)</td>
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<td>38.45(7)</td>
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<table>
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<th>3</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>b</td>
<td>p</td>
<td>OR</td>
</tr>
<tr>
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<td>-0.06</td>
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</tr>
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<td>ns</td>
<td>1.04</td>
</tr>
<tr>
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</tr>
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<td>ΔPDS</td>
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<td>-</td>
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</tr>
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<td>ΔPRQ</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>ΔPDS * ΔPRQ</td>
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<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

Note. Results of hierarchical logistic regression analysis for Change in Depressive Symptomology (ΔDEP; 0 = Stable or Decreased, 1 = Increased). \( n = 746 \). w1DEP: Wave 1 Depressive Symptomology. w1PDS: Wave 1 Pubertal Development Status. w1PRQ: Wave 1 Parent Relationship Quality (centered). ΔPDS: Change in Pubertal Development Status (0 = Stable, 1 = Advanced). ΔPRQ: Change in Parent Relationship Quality (0 = Stable or Improved, 1 = Deteriorated). ΔPDS \* ΔPRQ: Change in Pubertal Development Status (ΔPDS) by Change in Parent Relationship Quality (ΔPRQ) interaction term. Constant included in model, but not shown in table. ns = non-significant, * \( p < .05 \), ** \( p < .01 \), *** \( p < .001 \).
Figure 1

**Add Health Integrative Life Course Theoretical Framework**

*Note:* Figure from Harris et al. (2009).
These questions will ask about how you feel emotionally and about how you feel in general. How often was each of the following things true during the past week (seven days)?

0  Never or rarely
1  Sometimes
2  A lot of the time
3  Most of the time or all of the time

1. You were bothered by things that usually don’t bother you.
2. You didn’t feel like eating, your appetite was poor.
3. You felt that you could not shake off the blues, even with help from your family and your friends.
4. You felt that you were just as good as other people.
5. You had trouble keeping your mind on what you were doing.
6. You felt depressed.
7. You felt that you were too tired to do things.
8. You felt hopeful about the future.
9. You thought your life had been a failure.
10. You felt fearful.
11. Trouble falling asleep or staying asleep.
12. You were happy.
13. You talked less than usual.
15. People were unfriendly to you.
17. Frequent crying. *

18. You felt sad.

19. You felt that people disliked you.

20. It was hard to get started doing things.

* Modified CES – D items as described in measurement section. Prompt questioned frequency of occurrence over the past 12 months, with the following response options: Never; Just a Few Times; About Once a Week; Almost Every Day. Items were re-coded consistent with the remaining 18 CES – D items as done in previous studies.
APPENDIX B

PUBERTAL DEVELOPMENT STATUS MEASURE

As people reach adolescence, their bodies begin to change. This next set of questions ask about those changes.

1. As a girl grows up her breasts develop and get bigger. Which sentence best describes you?

   1. My breasts are about the same size as when I was in grade school.
   2. My breasts are a little bigger than when I was in grade school.
   3. My breasts are somewhat bigger than when I was in grade school.
   4. My breasts are a lot bigger than when I was in grade school.
   5. My breasts are a whole lot bigger than when I was in grade school, they are as developed as a grown woman’s breasts.

2. As a girl grows up her body becomes more curved. Which sentence best describes you?

   1. My body is about as curvy as when I was in grade school.
   2. My body is a little more curvy than when I was in grade school.
   3. My body is somewhat more curvy than when I was in grade school.
   4. My body is a lot more curvy than when I was in grade school.
   5. My body is a whole lot more curvy than when I was in grade school.

3. Have you ever had a menstrual period (menstruated)?

   1. No
   5. Yes

4. How old were you when you had your very first menstrual period?

   7. Seven years old and younger
   8. Eight years old
   9. Nine years old
   10. Ten years old
   11. Eleven years old
   12. Twelve years old
   13. Thirteen years old
   14. Fourteen years old
   15. Fifteen years old
   16. Sixteen years old
   17. Seventeen years old and older
APPENDIX C

PARENT-DAUGHTER RELATIONSHIP QUALITY MEASURE

Mother Figure

1. How close do you feel to your {MOTHER/ADOPTIVE MOTHER/ STEPMOTHER/ FOSTER MOTHER/etc.}?
   
   0 Not at all
   1 Very little
   2 Somewhat
   3 Quite a bit
   4 Very much

2. How much do you think she cares about you?
   
   0 Not at all
   1 Very little
   2 Somewhat
   3 Quite a bit
   4 Very much

3. Most of the time, your mother is warm and loving toward you.
   
   0 Strongly agree
   1 Agree
   2 Neither agree nor disagree
   3 Disagree
   4 Strongly disagree

4. You are satisfied with the way your mother and you communicate with each other.
   
   0 Strongly agree
   1 Agree
   2 Neither agree nor disagree
   3 Disagree
   4 Strongly disagree

5. Overall, you are satisfied with your relationship with your mother.
   
   0 Strongly agree
   1 Agree
   2 Neither agree nor disagree
   3 Disagree
   4 Strongly disagree
Father Figure

1. How close do you feel to your {FATHER/ADOPTIVE FATHER/ STEPFATHER/ FOSTER FATHER/etc.}?

<p>| | | | | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>Not at all</td>
<td>1</td>
<td>Very little</td>
<td>2</td>
</tr>
<tr>
<td>3</td>
<td>Quite a bit</td>
<td>4</td>
<td>Very much</td>
<td></td>
</tr>
</tbody>
</table>

2. How much do you think he cares about you?

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<thead>
<tr>
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<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>Not at all</td>
<td>1</td>
<td>Very little</td>
<td>2</td>
</tr>
<tr>
<td>3</td>
<td>Quite a bit</td>
<td>4</td>
<td>Very much</td>
<td></td>
</tr>
</tbody>
</table>

3. Most of the time, your father is warm and loving toward you.

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<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
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</thead>
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<td>1</td>
<td>Agree</td>
<td>2</td>
</tr>
<tr>
<td>3</td>
<td>Disagree</td>
<td>4</td>
<td>Strongly disagree</td>
<td></td>
</tr>
</tbody>
</table>

4. You are satisfied with the way your father and you communicate with each other.

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<table>
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<tr>
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<td>Agree</td>
<td>2</td>
</tr>
<tr>
<td>3</td>
<td>Disagree</td>
<td>4</td>
<td>Strongly disagree</td>
<td></td>
</tr>
</tbody>
</table>

5. Overall, you are satisfied with your relationship with your father.

<p>| | | | | |</p>
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