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Associations of Childhood Family Adversity and Pubertal Timing with Depressive Symptomatology in Adulthood

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ASSOCIATIONS OF CHILDHOOD FAMILY ADVERSITY AND PUBERTAL
TIMING WITH DEPRESSIVE SYMPTOMATOLOGY IN ADULTHOOD

A Thesis Presented

by

JEFFREY P. WINER

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ABSTRACT

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MAY 2013

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To date, no prior research has examined the combined roles of childhood family adversity and pubertal timing in longitudinal pathways to depressive symptomatology in adulthood. The present study was conducted with 225 men and 225 women to explore the unique and combined roles of childhood family adversity and pubertal timing on depressive symptoms in a community sample of married adults. Results for both men and women indicated significant main effects of a cumulatively risky family environment on depressive symptoms, as well as main effects of families with higher levels of abuse and neglect, chaos and disorganization, and interpersonal family conflict. A significant moderating relation was found for women with earlier pubertal timing and higher levels of childhood interpersonal family conflict on greater levels of depressive symptoms in adulthood. No other significant relations were determined in other moderation and mediational analyses. This project furthers our understanding of how the combined roles of pubertal timing and childhood family experiences can clarify the developmental, evolutionary, and clinical theories that link childhood and adolescent experiences to depression in adulthood. Specifically, childhood home environments defined by frequent interpersonal conflict (quarreling, arguing, and shouting), combined with early pubertal

development, may play an important role in predicating depressive symptomatology among adult women.

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CHAPTER 1

INTRODUCTION

Introduction and Specific Aims

Depression in adulthood is an etiologically complex and multifaceted disorder that causes enormous harm to individuals, families, and society. Continued research on the developmental antecedents of depression is essential to ameliorate the devastating impact depression has on communities around the world. In the field of developmental psychopathology, a significant literature indicates that family conflict, abuse, and neglect (i.e., “risky family factors”) in a child’s home environment are strongly associated with depressive symptomatology in adolescence and adulthood (Repetti, Taylor & Seeman, 2002). Research on the developmental antecedents of depression in female adolescents also underscores a relation between depression and early pubertal timing—experiencing pubertal changes earlier than same-sex peers (Ellis, 2004). Early pubertal timing in girls is also linked to adverse childhood family experiences (i.e., neglect; conflict), and later pubertal development is associated with greater familial warmth (Ellis & Garber, 2000; Ellis, 2004). For boys, early pubertal development may predict externalizing behavior problems during adolescence, and later pubertal development may be associated with lower self-esteem in adolescence (Belsky et al., 2007).

These factors noted, no prior research has been conducted to examine the combined roles both childhood family adversity and pubertal timing play in longitudinal pathways to depressive symptomatology in adulthood. The current study explored a series of etiological models to examine: 1) whether childhood family adversity and pubertal timing have independent main effects on adult depressive symptomatology, 2)

whether childhood family adversity moderates the relation of pubertal timing and adult depressive symptoms, and 3) whether pubertal timing mediates the association of childhood family adversity with depressive symptoms in adulthood.

The present study was conducted to examine the roles and relations among risky childhood family factors, pubertal timing, and depressive symptoms in a community sample of married opposite-sex couples. In this study we extend literature from clinical, developmental, and evolutionary theories to advance our understanding of family adversity in childhood and pubertal timing as important factors in unraveling the social, biological, evolutionary, and sex differences that may link childhood family experiences to depression later in life.

While nearly all proposed long-term developmental pathways to depression implicate childhood family adversity (neglect, abuse, and conflict) as a potent etiological variable, there is significantly less specific evidence on how the transition from childhood to adolescence (“going through puberty”) can contribute to our understanding of childhood adversity’s effects on later depression. This review of the literature will outline four areas of research: 1) depression as a significant public health problem and etiologically complex disorder, 2) childhood family adversity as a risk factor for depression, 3) “off-time” pubertal timing as a risk factor for depression, especially for women, and 4) the role childhood stress may play in the timing of pubertal development.

Depression in Adulthood

Depression in adulthood has an enormous impact on individuals, families, communities, and societies as a whole. In the United States alone, it is estimated that 17% of all adults will experience major depression during their lifetime (Kessler, 2002).

Depression, defined by its two cardinal symptoms of depressed mood and anhedonia (loss of pleasure in activities that were once enjoyable) leads to social withdrawal, interpersonal misery, and enormous decreases in occupational productivity (Simon, 2003). Experiencing depression places individuals at risk for a host of other psychological illnesses, including anxiety disorders and substance use disorders (Kessler, 2002). Depression is often chronic, relapses can be frequent, and the disease is associated with an increased risk of self-injurious behavior, suicide, and earlier mortality (Irwin, 2002; Kessler, 2002). Depression is one of the most commonly diagnosed mental disorders in adulthood, and according to the United States Center for Disease Control, antidepressants are the most commonly prescribed medications in the United States (Kendler, Gardner, & Prescott, 2002, 2006; Kessler, 2002).

The above noted, depression is an enormous public health problem, and with rates of depression increasing every decade, researchers continue to search for etiological explanations for this complex disorder (Kendler et al., 2006; Kendler, 2002). To date, depression has been linked to a myriad of factors, which include, but are not limited to: genetic influences, exposure to disturbed family environment, sexual abuse in childhood, premature parental loss, childhood onset anxiety or conduct disorders, individual differences in personality, exposure to major environmental adversities or traumatic events, low social support, substance abuse, interpersonal problems, recent life stressors, and prior history of mood disorder (Kendler et al., 2002, 2006; Kessler et al., 2002). Given this multitude of possible influences, one common ingredient across many factors is their origin in the context of the childhood family ecosystem. In order to contribute to the etiological narrative of depression, the focus of this research project is to explore

developmental experiences and potential risk factors during childhood and adolescence that may contribute to this broader array of risk. Of particular interest and importance for the current research are children who grow up in a “risky family environment.”

Childhood Family Adversity as Risk Factor for Depression

While the term itself may be misleading, the label “risky family” is not intended as a pejorative term towards certain families. Rather, the term is used to indicate families and family environments that may place children at increased risk for mental health problems later in life (Repetti, Taylor, & Seeman, 2002). A compelling literature indicates that children who grow up in highly stressful environments, particularly those environments defined by familial conflict, anger and aggression, relationships that lack warmth and support, and neglect of both immediate and secondary needs, are at increased “risk” for emotion and behavior problems (Repetti et al., 2002; Taylor, Lerner, Sage, Lehman, & Seeman, 2004). Numerous studies have shown graded relations between childhood adversity and both the presentation of depressive symptoms and whether suicide had ever been attempted (Felitti et al., 1998; Repetti et al., 2002; Taylor et al., 2004). These findings dovetail with evidence that the relation between childhood adversity in the family environment and depression later in life is present in both prospective and retrospective studies (Repetti et al., 2002; Weich, Patterson, Shaw, & Stewart-Brown, 2009). Thus, “a risky family” may be conceptualized as a latent construct that predisposes a child to later adversity.

A variety of mechanisms have been posited to unravel the relation between a child’s experience in a risky family environment and greater prevalence of psychological illness, particularly depression, in adulthood (Repetti et al, 2002). These may be

summarized as: 1) in a risky family environment, children experience consistent and immediate threats to their livelihood and safety and thus developing physiological and neuroendocrine systems are repeatedly activated. This over-activation of the stress network (including the hypothalamic-pituitary-adrenal axis) may contribute to a buildup of allostatic load, premature physiological wear and tear on the body (McEwen, 1993; McEwen, 2003). 2) Risky families may fail to provide children with essential self-regulatory skills, which may lead to deficits in abilities to regulate emotion in arousing interpersonal situations and/or an inability to seek proper social support in times of stress (Repetti et al., 2002; Taylor et al., 2004). 3) Risky families may increase vulnerability to substance abuse and behavior problems, either through direct exposure, or secondary effects of neglect and abuse (Repetti et al., 2002). These substance abuse and externalizing behavior problems are highly comorbid with clinical and subclinical levels of depression in adulthood (Kendler, Gardner & Prescott, 2002; Kendler, Gardner, & Prescott, 2006). As outlined in these three processes, the notion of excessive stress in childhood appears to be a major component in the development and long-term maintenance of depressive symptoms in adulthood.

Although risky families may include extreme cases of neglect and abuse, varying levels of risky family dysfunction are present across most families in the United States (Taylor et al, 2004). As a result, studying these variables in a community sample, rather than specifically targeting families from highly risky environments (e.g. clinical samples) allows researchers to explore family dynamics and common family dysfunction across high and lower “risk” families. In addition to the normative distribution of risky components across families, normative developmental milestones are essential to account

for across families. An emerging area of research points to the importance of uncovering variables related to one of the most significant developmental milestones, pubertal development, and the timing of this transition from childhood into adolescence as a potent etiological variable in the life course of depression.

Of particular note, research indicates that while sex differences in depression during pre-adolescence are relatively insignificant, beginning in the transition from childhood to adolescence, and throughout adolescence and into adulthood, a significant increase in depressive symptomatology occurs in women; approximately a 2:1 increase as compared to men (Hankin, Mermelstein, & Roesch, 2007; Kendler, 2002). Furthermore, not only does this sex difference occur in early adulthood but it appears to persist across the life span (Hankin et al., 1998, 2007). While this sex difference may be accounted for through a variety of biological, social, and individual psychological factors, studying the relative timing of the transition to and through adolescence may provide a unique vantage point in uncovering important etiological components of the sex differences found in depression (Kendler et al., 2002).

Pubertal Timing

Puberty is viewed as a particularly sensitive transitional period of development because of major changes in the neuroendocrine system, as well as the growth and physical maturation that contribute to the change of child into adolescent (Ellis & Garber, 2000; Ellis, 2004; Marshall & Tanner, 1969, 1970; Smith & Powers, 2009). As a result, puberty is a particularly salient window into the transition from childhood to later development, and a time of enormous change and potential sensitivity (Smith & Powers, 2009). While the sequence of pubertal changes in adolescence is predictable, for

example, stages of breast development (size and shape) or pubic hair growth (area and density), the chronological and/or relative timing of puberty (i.e., at what age a certain stage begins) is variable and difficult to predict (Belsky, Steinberg, & Draper, 1991). Pubertal timing, as defined here, refers to the age at which adolescence begins, and when the different stages of physical and sexual maturity are begun and completed, as compared to one's same-sex peers (Marshall & Tanner, 1969, 1970; Ellis, 2004). Specifically, this refers to the relative timing (as compared to same-sex peers) of the development of secondary sex characteristics--genitals for boys, breasts for girls, and public hair for both sexes (Marshall & Tanner, 1969, 1970). Additionally, age of menarche (first menstrual period) for girls and the growth of facial hair and deepening of voice for boys are used as markers of pubertal timing and developmental transition (Ellis, 2004).

A substantial body of evidence indicates that "off-time" pubertal timing (developing earlier or later than same-sex peers) is of great developmental significance, yet it is often underrepresented in literature on the developmental pathways to physical and mental health in later life. Earlier pubertal development for girls (developing earlier than same-sex peers) is associated with earlier age of first dating, first kissing, first genital petting, earlier age at first sexual intercourse, and higher rates of adolescent pregnancy (Belsky, Steinberg, Houts, & Halpern-Felsher, 2010; Ellis, 2004; Graber, Seeley, Brooks-Gunn, & Lewinsohn, 2004). Additionally, early pubertal development in girls has been identified as a risk factor for breast cancer, cardiovascular disease, unhealthy weight gain, and an increase likelihood of giving birth to underweight babies (Ellis, 2004; Feng et al., 2008; Frontini, Srinivasan, & Berenson, 2003). In reference to

maladaptive psychological outcomes, earlier maturing girls are at increased risk for depression, as well as disturbances in body image and anxiety, and they may be more likely to engage in promiscuous sexual behaviors and binge alcohol consumption (Belsky et al., 2007, 2010; Deardorff et al., 2011; Ellis, 2004). With regards to broader family impacts, longitudinal analyses demonstrate that families with early developing adolescents are associated higher levels of family conflict and decreases in family cohesion (Coakley, Holmbeck, Friedman, Greenley, & Thill, 2002).

While strong evidence indicates that early puberty timing in adolescence is linked to adolescent depression, and that adolescent depression is a major risk factor for depression in adulthood (Shankman et al., 2009), less is known as to what mechanisms of “early pubertal timing” influence adolescent depression. Some evidence suggests that neurobiological mechanisms are partly responsible for the relation of early pubertal timing and depression. One current theory posits that an enlarged pituitary gland in adolescents with early pubertal timing might be associated with hyperactivation of the hormonal stress response, leading to an increased susceptibility to environmental stressors, and subsequent presentation of depressive symptomatology (Whittle et al., 2011). This noted, significant results for pituitary gland size as a partial mediator between early pubertal timing and depression has only been documented in female adolescents in early and mid-stages of adolescent development, rather than cross-sectionally across the entire adolescent period (Whittle et al., 2011).

Other accounts of the relation between early pubertal development in girls and depressive symptoms highlights the maturation disparity hypothesis, that posits that the gap between physical and psychosocial maturity is what places early (physically)

developing adolescents at risk for depression (Ge & Natsuaki, 2009). Research documents that girls who develop earlier than same-sex peers are perceived by others as older and more mature than they actually are (Sontag, Graber, & Clemans, 2011). As a result these girls may be socially or sexually targeted, and/or may be placed in situations that are less appropriate for their cognitive and emotional development (Ge & Natsuaki, 2009). This constellation of accelerated social influence (i.e., being treated older than one's chronological age) puts early developing girls at increased risk for a host of risky behaviors, including unwanted sexual activity and substance misuse (Ellis, 2004). Additionally, other research notes that negative adolescent reputation, in the form of rumors and gossip, in addition to false perceptions of maturity, may act as partial mediators between off-time physical development and internalizing symptoms (Reynolds & Juvonen, 2011).

While this account is compelling for early pubertal timing as a meaningful variable in female adolescent depression, for boys, effects of early pubertal maturation are much more mixed in terms of both positive outcomes (e.g., increased self confidence and popularity among peers) and negative outcomes (e.g., externalizing behavior problems and delinquency) (Belsky et al., 2007; Susman et al., 2012). Additionally, while some studies have produced evidence that later developing boys are at increased risk for internalizing symptoms (depression and anxiety), the published data on this topic are much more heterogeneous than the literature on female somatic development and depression (Belsky et al., 2007; Marceau, Dorn, & Susman, 2012; Marceau, Ram, Houts, Grimm, & Susman, 2011). As a result, the majority of the theory presented below is focused on female development.

Pubertal Timing and Childhood Family Adversity

Pubertal maturation is controlled by a multiplicity of complex interactions between and among biological and environmental factors (Belsky et al., 1991; Ellis, 2004; Marceau et al., 2012; Matchock & Susman, 2006). The timing of puberty may be influenced by, but is not limited to, genetic factors, socioeconomic status, environmental toxins, diet, exercise, pre-pubertal fat and body weight, and the presence of chronic illness and stress (Belsky et al, 2007). This link between stress and pubertal timing is both intriguing and complicated (Belsky et al., 2007). While high levels of chronic and severe stress (i.e., nutritional deprivation, extreme exercise regimens) have been linked to later pubertal timing (Ellis, Figueredo, Brumbach, & Schlomer, 2009), research that originally hatched from the catalyst publication of Belsky, Steinberg, & Draper (1991) on individual differences in pubertal development, has shown that the childhood familial environment may be one of the largest factors that impacts girls' pubertal timing. Specifically, a growing body of research indicates family environment (and adversity) is most strongly associated with *earlier*, rather than *later*, pubertal development (see Ellis, 2004 for a review). Cohesive family relationships and higher frequency of contact with biological parents are, accordingly, associated with later pubertal timing in girls and environments defined by neglect (low parental investment), abuse, and general chaos are associated with earlier pubertal timing (Ellis, 2004).

The broad construct behind this theory of environmental influence on pubertal timing, grounded in evolutionary psychology (Ellis, 2004; Ellis & Boyce, 2005), is that earlier female pubertal development may increase the likelihood of finding and obtaining potential mates (pair-bonds) and thus increase the window in which offspring can be

produced. In this model, early pubertal development is conceived as an evolved and adaptive response to childhood family adversity, as earlier onset of sexual maturity increases the timeline in which a female can mate and bear children across the lifespan (Saxbe & Repetti, 2009).

Furthermore, across the literature on pubertal development and relative pubertal timing in girls, a consistent literature points to the relation between pubertal timing in girls and the absence or presence of a father in the home environment. Building on paternal investment theory and its relation to the development of female reproductive strategies, evolutionary theory posits that the physiological and motivational systems underlying variation in timing of girls' sexual development are especially sensitive to the father's role in the early childhood environment (Belsky et al., 1991; Boyce & Ellis, 2005). Specifically, experiences associated with early father absence and relational distance are hypothesized to evoke the development of reproductive strategies that match low male parental investment (Belsky et al., 2007). Girls in this context are predicted to develop in a manner that speeds up the rate of pubertal maturation, accelerates onset of sexual activity, and orients the individual toward relatively unstable pair bonds (Belsky et al., 2007; Deardorff et al., 2011; Ellis, 2004). On the other hand, experiences associated with early father presence and relational closeness are hypothesized to evoke the opposite pattern of sexual development. Regardless of the paternal role, the developmental trajectory that unfolds was likely to have promoted reproductive success across our ancestral past (Belsky et al., 1991; Deardorff et al., 2011; Ellis, 2004; Graber, Brooks-Gunn, & Warren, 1995).

While the specific mechanisms (e.g., pheromones, stress reactivity) of how the evolved components of earlier pubertal development occurs (i.e., upregulation or downregulation of certain biological and reproductive processes) are not yet fully defined in the literature, enough evidence has been provided to emphasize that pubertal timing is an important developmental factor in etiological pathways to depression, and warrants further research.

To date, no research has specifically explored both childhood family adversity and variations in pubertal timing and their combined relation to depression in adulthood. Specifically, no research has been conducted that incorporates theory grounded in both developmental psychopathology, which typically frames childhood experiences as potential risk factors for adaptive and maladaptive development, and evolutionary psychology, which typically frames developmental events as adaptive or maladaptive strategies as inherited processes of a deep evolutionary past.

Statement of Research Questions and Hypotheses

The aim of the current study is to explore if pubertal timing plays a combined, interactive, or mediating role with childhood family adversity and depressive symptomatology in adulthood for women and men.

Research Question 1. Do childhood family adversity and pubertal timing independently (through main effects) predict depressive symptomatology for women and for men?

We hypothesize that main effects will exist between childhood family adversity and depressive symptomatology for both sexes. We hypothesize that main effects will exist between off-time pubertal development and depression for women but not for men.

Research Question 2. Does childhood family adversity moderate the relation between pubertal timing and depressive symptomatology for women and for men?

We hypothesize that childhood family adversity will moderate the relation between pubertal timing and depressive symptomatology, and that sex differences will emerge presenting unique developmental pathways. We anticipate childhood family adversity will strengthen the effect of early pubertal development on women's depressive symptoms in adulthood. We anticipate that if a significant moderating relation unfolds for men, childhood family adversity will increase the effect of later, not early, pubertal development on men's depressive symptoms in adulthood.

Research Question 3. Does a mediation model emerge, in so far as, the relation between childhood family adversity and depressive symptoms in adulthood is partially mediated through off-time pubertal development?

We hypothesize that some amount of variance for women who experience childhood family adversity may be mediated through off-time pubertal timing. We do not anticipate this to be a complete mediational model, or to account for the majority of the variance. This noted, as evolutionary theory links childhood family adversity to pubertal acceleration in girls, we will explore this hypothesis for women. We do not hypothesize a significant mediational relation among these variables for men.

CHAPTER 2

METHOD

Participants

The current data were obtained from a larger study entitled the Growth in Early Marriage (GEM) project. Opposite-sex married couples ($n = 225$) living in western Massachusetts (men, $M_{age} = 29.06$, $SD = 5.23$, 96% White; women, $M_{age} = 27.70$, $SD = 4.80$, 92% White) were recruited from marriage license records to participate in a longitudinal project examining evolving marital relationships and mental health. The current data were derived from the first of three laboratory visits in which spouses answered a series of computerized questionnaires and participated in two interpersonal negotiation tasks. All couples used in the sample (from Time 1) were in the first seven months of their marriage. For all couples this was their first marriage and neither spouse had children (together, or with a previous partner). Neither spouse had been diagnosed with an endocrine disorder, which could disrupt stress hormone analysis, which is part of the larger GEM project.

Procedures

Participants completed a telephone screen and if qualified based on standardized criteria, were scheduled to report to the GEM laboratory at the University of Massachusetts in Amherst, MA for the first of three lab visits. Over the course of the three-hour experimental session, couples separately answered a series of computerized questionnaires. Couples were seated in a comfortable, but stimulus-neutral room monitored by a research assistant. Couples sat at desktop computers with a large cubicle divider to prevent communication during the questionnaire answering process. Over the

course of the study visit, participants each provided seven saliva samples for stress hormone analysis and participated in two-filmed interaction tasks (one 15 minute conflict negotiation task, and one resolution/positive interaction task), but only questionnaire responses were used in the current analyses.

Measures

Risky Families Questionnaire. The 13-item Risky Families Questionnaire (RFQ) (Taylor et al., 2004) was used to retrospectively assess abuse, neglect (parental investment), and family conflict during ages 5 through 15, and has been reliably correlated with adverse mental health outcomes in adulthood, including depression (Felitti et al., 1998; Taylor et al., 2004). The RFQ was originally adapted from a larger instrument developed to assess the relation of childhood experiences, including family stress, to mental and physical health outcomes in adulthood (Felitti et al., 1998). In previous research, the RFQ has been validated against clinical interviews of individuals' experiences during childhood conducted and coded by trained clinical interviewers (Taylor et al., 2004; Taylor et al., 2006). This dual assessment methodology of childhood adversity through questionnaire and interview techniques has demonstrated high agreement and reliability of the RFQ (Taylor et al., 2004).

On the RFQ, participants rated aspects of their childhood family environment on a series of 5-point scales ranging from 1 (not at all) to 5 (very often), with items related to *neglect* (i.e., “How often would you say you were neglected while you were growing up, that is, left on your own to fend for yourself?”), *abuse* (i.e., “How often did a parent or other adult in the household push, grab, shove, or slap you?”), *family conflict* (i.e., “How often would you say there was quarreling, arguing, or shouting between your parents?”),

and *household (dis)organization* (“Would you say the household you grew up in was chaotic and disorganized?”). Positively worded items were reverse coded (i.e., “How often did a parent or other adult in the household make you feel that you were loved, supported, and cared for?”). Scores range from 13 to 65. In our current sample Cronbach’s alpha was measured at .86 (men $\alpha = .85$, women $\alpha = .87$).

In an effort to further develop an understanding of what components of childhood adversity, as assessed by the RFQ, lead to maladaptive outcomes of depression, we used factor analytic techniques to develop, based on theory, several subscales of the RFQ. We used a principal-components factor analysis with Varimax rotation across men and women separately. Following this examination, three distinct subscales emerged.

While the RFQ Total (all 13 items) indicates an overall environment that places the developing child, and later adult, at greater risk for psychopathology, three unique subscales were identified: Childhood Family Interpersonal Conflict (RFQ-IC = items 8, 9, 10, and 11), Childhood Family Abuse and Neglect (RFQ-AN = items 2, 4, 7, 13, and items 1 & 3 reverse scored), and Childhood Family Chaos and Disorganization (RFQ-CD = items 5 and 12, and item 6 reverse scored).

Inventory of Depressive Symptomatology: Self-Report. To assess for current symptoms of depression, participants answered the 30-item Inventory of Depressive Symptomatology: Self-Report (IDS-SR). The IDS-SR (Rush, Gullion, Basco, Jarrett, & Trivedi, 1996) evaluates all symptoms required for a DSM-IV-TR diagnosis of a major depressive episode. The IDS-SR improves on other standard self-report measures of depressive symptomatology because each item assesses a single symptom only and all items are equally weighted. The IDS-SR is reliable (30 items; $\alpha = .93$) and correlates

highly with the Hamilton Rating Scale for Depression ($r = .88$), the Beck Depression Inventory ($r = .93$), and the clinician-rated version, the IDS ($r = .91$) (Biggs et al., 2000; Rush et al., 1996; Rush, Carmody, & Reimitz, 2000). Scores range from 0 to 84; scores of 14-25 are associated with clinically significant *mild* symptoms of depression, scores of 26-38 are associated with clinically significant *moderate* symptoms of depression, and scores of 39 and above are associated with clinically significant symptoms in the *severe* range. Each individual item is rated on a 0-3 scale (higher numbers indicating greater symptom severity). On two items of the measure, participants are asked to answer one of two questions, thus only 28 questions are answered and scored.

Pubertal Timing. Pubertal timing (PT) was assessed by retrospective self-report, which has been determined an effective measure of pubertal changes (Dubas, Graber, & Petersen, 1991; Smith & Powers, 2009). Men and women were asked to assess their pubertal timing relative to those of their same-sex peers at the time of puberty. The specific item reads, “Please try to remember when the following occurred: Compared to your same-sex peers (age-mate peers), when would you say you began to experience changes due to puberty, including changes in physical development?” This assessment was measured from 1 (“much earlier than most of my peers”) to 5 (“much later than most of my peers”). This assessment of pubertal timing is used as a continuous measure of pubertal timing from earlier to later maturation. Of the current sample, 31% of women and 17% of men experienced timing that was a little or much earlier than most of their peers (1 or 2), 44% of women and 61% of men experienced pubertal timing at about the same time as their peers (3), and the remaining 25% of women and 22% of men experienced pubertal timing that was a little or much later than their peers (4 or 5). This

distribution is consistent with other work that has assigned categories of early/on time/late pubertal timing to their samples (Brooks-Gunn & Warren, 1989; Ge, Conger, & Elder, 2001; Smith & Powers, 2009).

Childhood Socio-Economic Status (Childhood SES). Childhood socio-economic status (SES) has been determined to play a significant role in risky family environments (Repetti et al., 2002). SES itself may be thought of as a potential marker for the chronic stressfulness of a given environment. As chronic stress takes its toll on relationships, including those in the family, SES is an essential variable to account for in an assessment of childhood family adversity. Previous research indicates that low SES has been tied to all of the risky family characteristics discussed above (neglect, abuse, family conflict, and (dis)organized home environment etc.), and reductions in SES have been associated with an increase in risky family characteristics (Repetti et al., 2002). Children living in lower SES families are at heightened risk for physical mistreatment or abuse and are more likely to be in family relationships lacking warmth and support (Lehman, Taylor, Kiefe, & Seeman, 2005). Continued poverty and economic problems tend to move parenting strategies to more harsh, punitive, irritable, inconsistent, and coercive patterns (Repetti et al., 2002).

Consistent with this view of risky families, we used SES as a covariate in our regression analyses to control for variation across the RFQ based on SES. While there are many ways to operationalize and retrospectively assess estimated SES during childhood, in the present study we asked participants to report their parent's highest level of education. The higher of the two parent's education level was used as a marker for a participant's SES during childhood. If an individual lived in a single parent home, or

only one parent's education was reported, that value was used. To assess education, a 10-point scale was used whereas, 1 = Grade school, or no high school, 2 = Some high school, 3 = High school diploma, 4 = G.E.D., 5 = Associate's degree 6 = Vocational degree, 7 = Bachelor's degree, 8 = Master's degree, 9 = Ph.D., M.D., J.D., and 10 = Unknown (and dropped from our analyses). Analysis of our sample showed that RFQ Total and the RFQ subscales were not correlated to Childhood SES for women, but RFQ Total, RFQ-AN, and RFQ-CD presented weak negative correlations to Childhood SES in men. *See Table 1.* With this knowledge and significant theory linking SES and childhood family risk, we opted to include "Childhood SES" as a control variable for both men and women in our regression models.

Protection of Human Subjects

The Growth in Early Marriage (GEM) project is currently approved by the Institutional Review Board (IRB) of the University of Massachusetts Amherst and has been approved every year during its annual continuing review since initial approval. All participant couples receive a unique subject ID number when enrolled in the study and all data are identified solely with this unique and non-identifying code. During the initial informed consent process, participants are told that they may terminate their participation at any time, for any reason, and that they have the permission to contact the study principal investigators, Dr. Paula Pietromonaco and Dr. Sally Powers, if they have questions or concerns about their experiences as a participant.

CHAPTER 3

DATA ANALYSIS AND RESULTS

In order to examine the relations between our three major constructs, childhood family adversity, pubertal timing, and depression in adulthood, we used a series of hierarchical multiple regressions to explore several statistical models. We explored a 1) Main Effects Model, 2) Moderation Model, and 3) Mediation Model. While running both moderation and mediation analyses within the same research model often indicates a lack of guidance by theory, the current project indicated divergent theory behind our primary research questions, and thus required both moderation and mediation analyses to fully explore our hypotheses. Additionally, as the present data were at risk for non-independence of error because the participants are husbands and wives, not just men and women, we explored our questions about men and women independent of one another (building models for men and women, but not statistically comparing differences).

A power analysis of the sample (225 women, and 225 men) revealed that the detectable r^2 for women and men individually is 0.042 for power of 0.80. This indicates that each predictor must account for at least 4.20% of the variance to be detectable; therefore, the proposed study is highly powered to detect significant relations. All variables in our analyses were centered at their means. To account for any missing data, participants who had not answered one or two items on a given questionnaire were not dropped; their total score was calculated using the average of all questions answered.

Descriptive Analyses

Correlations among all variables for men and women are included in *Table 1*. Descriptive analyses revealed that the average IDS-SR depression scores in our sample

were 10.14 ($SD = 6.00$) for men (range 0 – 38) and 11.77 ($SD = 7.60$) for women (range 0 – 45). With regards to depressive symptoms, 52 men presented with symptoms associated with depression (mild = 48, moderate = 4, severe = 0). Among women, 71 women presented with symptoms associated with depression (mild = 56, moderate = 11, severe = 4). Thus, 23% (52 of 226) of men presented profiles associated with clinically meaningful depression and 31% (71 of 226) of women presented with profiles associated with clinically meaningful depression. Average scores on our PT measure were 2.92 ($SD = .96$) for women (range 1 - 5) and 3.04 ($SD = .80$) for men (range 1 - 5), which is consistent with previous literature (Brooks-Gunn & Warren, 1989; Ge et al., 2001; Smith & Powers, 2009). For full descriptive statistics, including those for the RFQ and subscales see *Table 2*.

Main Effects Model (Research Question 1)

In the first series of analyses we explored simple main effects between the RFQ and IDS-SR, and main effects between PT and the IDS-SR for men and women separately using a hierarchical regression with Childhood SES included as the first step, RFQ and PT as the second step, and with no interactions included. The rationale for these initial analyses is based upon the idea that in prior research depression has been correlated with childhood adversity and pubertal timing, and that the effects of childhood adversity on depression, and the effects of pubertal timing on depression may be primarily independent of one another. See *Figure 1*.

RFQ Total was significantly positively correlated with depressive symptoms for both men and women. See *Table 1*. Additionally, hierarchical regressions indicated main effects of the RFQ Total and the three Risky Families Subscales (RFQ Interpersonal

Conflict (RFQ-IC), RFQ Abuse & Neglect (RFQ-AN), and RFQ Chaos & Disorganization (RFQ-CD)) on depressive symptoms in women. For men, hierarchical regressions also indicated main effects of the RFQ Total and two of the RFQ subscales (RFQ-AN and RFQ-DC) on depressive symptoms. We did not find significant main effects for women's PT on depression, and we did not find significant main effects of men's PT on depression. *See Table 3.*

Moderation Model (Research Question 2)

In the second set of regression analyses we explored a series of moderation models. In these moderation analyses we used PROCESS (Hayes, 2012) to assess statistical moderation. We regressed IDS-SR total scores on the RFQ, PT, and the interaction of these two independent variables for both men and women separately. All variables were centered at their mean and Childhood SES served as the single covariate in all analyses. *See Figure 2.*

A moderation model for the RFQ Total was not significant for men or for women. To further explore the RFQ, and potential "active ingredients" of risky families that link pubertal timing and depression, we ran similar moderation models with the three RFQ subscales separately. Following these analyses, a significant interaction emerged when the IDS-SR was regressed on RFQ-IC, PT, and the interaction of these two terms, when controlling for Childhood SES: $b = -1.33$ $t(218) = -2.01$, $p < .045$. The RFQ-IC * PT interaction term accounted for 1.7% of the variance in depressive symptoms in women above and beyond RFQ-IC and Childhood SES ($R^2\Delta = .017$, $F(1, 218) = 4.05$, $p < .05$). The direction of the RFQ-IC * PT interaction indicates that earlier pubertal timing coupled with higher levels of interpersonal family conflict (quarrelling, arguing, and

shouting among family members), has a combined predictive ability on depressive symptoms in adult women. *See Table 4.* No significant moderation models with the RFQ subscales were found for men.

Mediation Model (Research Question 3)

In our third set of regression analyses we explored a series of mediation models, in which we attempted determine the proportion of variance of childhood family adversity that is mediated through pubertal timing in its relation to depression in adulthood. This was run for both men and women separately. *See Figure 3.*

We ran a series of exploratory mediational analyses using PROCESS (Hayes, 2012) and whereas main effects emerged for RFQ Total, and the three RFQ subscales on depressive symptoms in adulthood, no mediation model was significant for either men or women. RFQ Total and its subscales were not related to PT, and PT was not related to depressive symptoms, so mediation was not statistically viable. Childhood SES was used as the single covariate.

CHAPTER 4

DISCUSSION

Implications

While the effects of childhood adversity on depression in adulthood are extensively demonstrated across the clinical and developmental literature (Kendler et al., 2006; Kendler, 2002; Taylor et al., 2004), the present study underscores the core construct validity of the RFQ as a measure strongly associated with symptoms of depression in adulthood (Repetti et al., 2002; Taylor et al., 2004). Furthermore, to our knowledge, this is the first study to explore and identify several unique RFQ subscales of childhood family adversity (Interpersonal Conflict (RFQ-IC), Abuse & Neglect (RFQ-AN), and Disorganization & Chaos (RFQ-DC)) that may be differentially predicative of depressive symptoms in adulthood. As noted above, hierarchical regressions indicated a significant positive relation between RFQ Total, RFQ-AN, and RFQ-CD with depressive symptoms in men and in women. Thus the current data demonstrate that the RFQ Total and RFQ subscales highlight specific aspects of family function that are individually predictive of depressive symptoms in adulthood.

In contrast to the RFQ, we did not find main effects of pubertal timing on depressive symptoms for either men or women. Whereas some work has demonstrated main effects of off-time pubertal development on maladaptive outcomes in adolescent girls (Wasserman, Holmbeck, Lennon, & Amaro, 2012), most research on off-time pubertal development notes that the negative psychosocial outcomes associated with off-time pubertal development, like depression, are heavily influenced by context-specific factors (Ellis, 2004; Ge et al., 2001; Graber et al., 2004; Lynne-Landsman, Graber, &

Andrews, 2010; Rudolph & Flynn, 2007; Sontag et al., 2011). It is likely, therefore, that social context (family, peer, and community) strongly impacts pubertal timing's effects on internalizing symptoms, especially depression (Belsky et al., 2007, 2010; Ge et al., 2001). In order to operationalize the social context that may play a key role in pubertal timing's impact on mood disorder, we developed a statistical moderation model of childhood family adversity and pubertal timing.

In the assessment of this model, a compelling result emerged. We found a significant moderation effect of women's pubertal timing and childhood family interpersonal conflict (RFQ-IC) on depressive symptoms. Specifically, women with early pubertal timing, who grew up in homes with higher levels of interpersonal family conflict (i.e., quarreling, arguing, and shouting), presented a unique pathway to depressive symptoms in adulthood.

The significant moderation model among women indicates the role of context-specific interpersonal conflict and off-time pubertal development on depression. This context-specific stress activation can be considered a form of "contextual-amplification," a theory in developmental psychopathology which posits that the interactive process of off-time pubertal development and stressful social contexts is what links pubertal timing to negative affect and behavior (Ge & Natsuaki, 2009; Rudolph & Troop-Gordon, 2010). As such, our significant finding demonstrates that experiencing early pubertal timing may trigger or exacerbate interpersonal family conflict, early developing girls may be more vulnerable to the negative aspects of interpersonal family conflict, or both could simultaneously co-occur.

Model 3, our mediational analysis, did not indicate a significant mediational role for pubertal timing in explaining the relation of childhood family adversity to depressive symptoms in men or women. While the model for men was exploratory in nature and we were not surprised that the data produced null results, the mediational model for women was based on life history theory and evolutionary-developmental psychology that has demonstrated efficacy and interest in the child development literature linking early adversity and pubertal development in women (Belsky et al., 2007, 1991, 2010; Boyce & Ellis, 2005; Ellis, 2004; Ellis et al., 2012; Ellis, Essex, & Boyce, 2005; Ellis, Shirtcliff, Boyce, Deardorff, & Essex, 2011).

The current data do not support the notion that early childhood family adversity has direct effects on the timing of puberty in women, a pathway through which off-time development could have significant implications for the development of depressive symptoms in adolescence and adulthood. It is possible, however, that the manner in which the current study operationalized childhood family stress and adversity was inconsistent with the true active components that previous researchers have targeted. While we used the RFQ as our measure of early stress, we did not include such variables as father absence, stepfather presence, or specific maternal parenting practices that have been linked as psychosocial antecedents to early pubertal development in women (Belsky et al., 2010; Deardorff et al., 2011; Ellis & Garber, 2000).

Limitations

While significant effects were revealed in the current analyses, there are a number of limitations to the current study. First, two of our three primary measures, the RFQ and our assessment of pubertal timing are both retrospective. While both measures are

designed to be used retrospectively, possess high reliability with related prospective assessment procedures, and are considered valid measures of our constructs of interest, (Ellis, 2004; Smith & Powers, 2009; Taylor et al., 2004), caution must be exercised in their interpretation.

Second, our assessment of current depressive symptomology is obtained through questionnaire rather than researcher-participant interview. While the IDS-SR has been well validated alongside researcher-participant interviews (Biggs et al., 2000), the current assessment measures symptoms, *not* impairment or distress. As a result, some caution should be taken when generalizing our sample's depressive symptomatology to broader impairment via depression.

Third, as we cannot account for the temporal precedence of specific childhood and adolescent experiences in our model, we cannot be sure of the direction of effect. Our questionnaire methods produce results that may reflect pubertal timing's impact on childhood family adversity, vice versa, or their simultaneous occurrence.

Fourth, data used in the present study are from individuals who are legally married and in opposite-sex relationships. This sample does not allow for variance among individuals with different adult partnering patterns and does not include individuals who are in same-sex relationships, or identify as a gender other than "male" or "female." Additionally, while our sample is homogeneously individuals in opposite-sex relationships, it is also relatively homogenous with regard to race and ethnicity (over 90% of participants are White European-Americans). As race and ethnicity may play an important role in childhood family adversity, childhood SES, pubertal timing, and

depression in adulthood, our study cannot effectively be generalized to populations beyond White European-Americans.

Fifth, we do not include specific biological (genetic or stress hormone) assessment. This is of note because of the increasing knowledge in the developmental sciences that individuals may possess differential susceptibility patterns (rooted in *a priori* biological factors) to maladaptive outcomes like depression (Boyce & Ellis, 2005; Ellis & Boyce, 2011; Ellis et al., 2005). As a result, associations between individuals' early adversity and later functioning are not necessarily constructed in a linear pattern, but rather, a curvilinear relationship may emerge, in which some individuals are inherently more susceptible to environmental factors than others. Consequently, a U-shaped relation between early exposures to adversity and maladaptive outcomes may emerge in some samples, with high maladaptive phenotypes disproportionately emerging within both highly stressful and highly protected childhood environments (Boyce & Ellis, 2005). While this model is beyond the scope of this project, it provides compelling theory for future research on this topic.

These limitations noted, this study does expand the literature related to child and adolescent developmental antecedents to depression in adulthood. First, we have confirmed that the Risky Families Questionnaire is strongly predictive of depressive symptoms in adulthood among a community sample of adults. Second, we have provided evidence for the presence of several unique subscales within the Risky Families Questionnaire (Abuse and Neglect, Chaos and Disorganization, and Interpersonal Conflict) that have presented differential moderating effects on depressive symptoms in adults. Third, we have documented that in our sample, early pubertal timing predicts

women's depressive symptoms only in the context of childhood family conflict. Finally, these conclusions lend themselves towards the importance of future research that clarifies the specific roles of childhood family interpersonal conflict and early pubertal timing on depressive symptoms in adulthood

Tables

Table 1. Correlation Table of Risky Families Total Score, Risky Families Subscales, pubertal timing, depressive symptoms, and Childhood SES (N = 225). Women are below the diagonal.

Variables	1	2	3	4	5	6	7
1. RFQTotal	–	.763**	.893**	.819**	.031	.206**	-.170*
2. RFQ-IC	.815**	–	.515**	.458**	.072	.106	-.028
3. RFQ-AN	.911**	.609**	–	.645**	-.025	.210**	-2.01**
4. RFQ-CD	.760**	.456**	.558**	–	.044	.212**	-.169*
5. PT	.004	-.087	.046	.035	–	.047	.149*
6. IDS-SR	.274**	.239**	.278*	.141*	-.052	–	-.006
7. SES	-.073	.035	-.118	-.079	.051	-.002	-

Note: RFQ Total = Risky Families Questionnaire Total Score (Taylor, 2004). RFQ-IC = RFQ subscale, Interpersonal Conflict. RFQ-AN = RFQ subscale, Abuse & Neglect. RFQ-CD = RFQ subscale, Chaos & Disorganization. PT = pubertal timing single item. IDS-SR = Inventory of Depressive Symptomatology: Self-Report (Rush, 1996). SES = Childhood SES estimated from participant's parents highest level of education.

* $p < .05$. ** $p < .01$. *** $p < .001$

Table 2. Descriptive statistics of variables used in regression analyses.

Variables	Men				Women			
	<i>M</i>	<i>SD</i>	<i>n</i>	range	<i>M</i>	<i>SD</i>	<i>n</i>	range
RFQ Total	2.07	.663	225	1 - 4.31	2.15	.748	226	0 - 4.23
RFQ-IC	2.61	.810	226	0 - 5	2.85	.859	226	0 - 5
RFQ-AN	1.76	.712	226	0 - 4.17	1.75	.833	226	0 - 4.67
RFQ-CD	1.96	1.00	226	0 - 5	1.99	.977	226	0 - 4.67
PT	3.04	.801	225	1 - 5	2.92	.956	225	1 - 5
IDS-SR	10.14	6.00	226	0 - 38	11.77	7.60	226	0 - 45
SES	5.95	2.12	222	1 - 9	6.14	2.09	223	1-9

Note: RFQ Total = Risky Families Questionnaire Total Score (Taylor, 2004). RFQ-IC = RFQ subscale, Interpersonal Conflict. RFQ-AN = RFQ subscale, Abuse & Neglect. RFQ-CD = RFQ subscale, Chaos & Disorganization. PT = pubertal timing single item. IDS-SR = Inventory of Depressive Symptomatology: Self-Report (Rush, 1996). SES = Childhood SES estimated from participant's parents highest level of education.

Table 3. Main effects model of Risky Families Questionnaire (RFQ) and pubertal timing (PT) on depressive symptoms in adulthood (IDS-SR). Childhood SES included as covariate.

	Men		Women		
Variables	β	SE	Variables	β	SE
SES	.079	.193	SES	.080	.238
RFQTotal	2.14***	.629	RFQTotal	2.99***	.682
PT	-.265	.499	PT	-.398	.517
SES	-.026	.194	SES	-.033	.240
RFQ-IC	.792	.528	RFQ-IC	2.247***	.597
PT	.305	.511	PT	-.206	.524
SES	.096	.194	SES	.133	.238
RFQ-AN	2.054***	.597	RFQ-AN	2.698***	.600
PT	.358	.498	PT	-.497	.516
SES	.077	.193	SES	.047	.245
RFQ-CD	1.367***	.412	RFQ-CD	1.147*	.528
PT	.245	.500	PT	-.426	.534

Note: RFQ Total = Risky Families Questionnaire (Taylor, 2004). RFQ-IC = RFQ subscale, Interpersonal Conflict. RFQ-AN = RFQ subscale, Abuse & Neglect. RFQ-CD = RFQ subscale, Chaos & Disorganization. PT = pubertal timing single item. IDS-SR = Inventory of Depressive Symptomatology, Self-Report (Rush, 1996). SES = Childhood SES estimated from participant's parents highest level of education.

* $p < .05$. ** $p < .01$. *** $p < .001$

Table 4. Moderation model of women’s childhood family interpersonal conflict (RFQ-IC) and pubertal timing (PT) on depressive symptoms in adulthood (IDS-SR). Childhood SES included as covariate.

Variables	β	SE
SES	-.045	.238
RFQ-IC	2.22***	.593
PT	-.140	.522
RFQ-IC * PT	-1.33*	.661

Note: RFQ-IC = RFQ subscale, Interpersonal Conflict. PT = pubertal timing single item. SES = Childhood SES estimated from participant’s parents highest level of education.

* $p < .05$. ** $p < .01$. *** $p < .001$

Figures

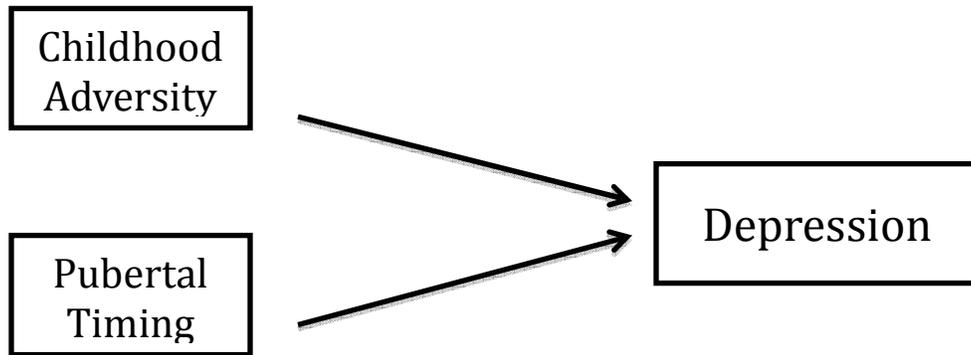


Figure 1. Main Effects Model. The main effect of childhood adversity on depression, and the main effect of pubertal timing on depression.

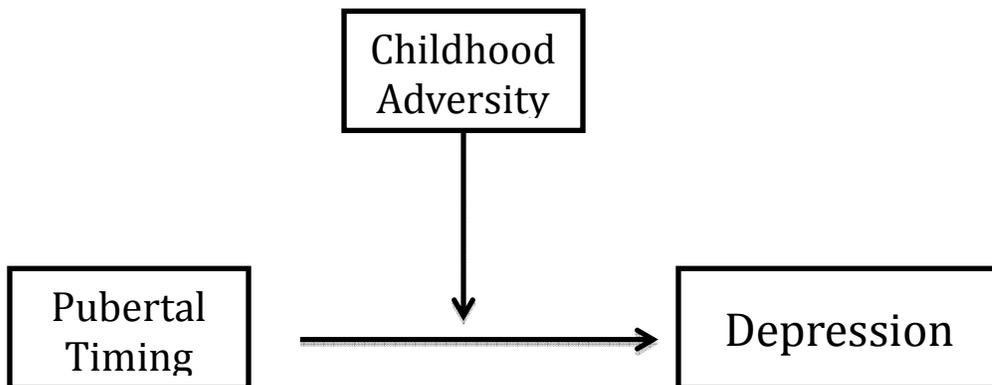


Figure 2. Moderation Model. Childhood adversity moderates the relation between pubertal timing and depression.

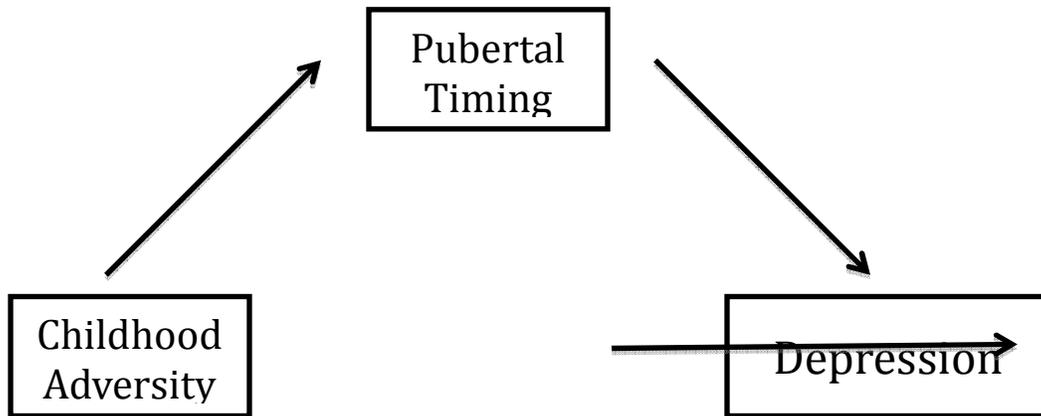


Figure 3. Mediation Model. Pubertal timing mediates the relation between childhood adversity and depression.

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