LEE, JESSICA M. Ph.D. Growing Up Too Fast? An Examination of Pubertal Timing and Childhood Social Wariness in the Development of Adolescent Depression. (2023)
Directed by Dr. Susan Keane. 79 pp.

Children are vulnerable to experiencing increases in depressive symptoms by late adolescence, as they are not yet fully equipped with effective cognitive and emotional resources to successfully navigate the adolescent transition. Biological factors (i.e., earlier pubertal timing) and social characteristics (i.e., childhood social wariness) have been associated with increased risk of depressive symptoms in adolescence. While the association between earlier pubertal timing and adolescent depressive symptoms has been well-established, no research has yet examined whether childhood social wariness is associated with depressive symptoms beyond childhood. In addition, the contextual amplification hypothesis suggests that the negative effects of earlier pubertal timing may be exacerbated by an earlier vulnerability to predict increases in depressive symptoms. To date, no study has yet integrated the pubertal timing and social wariness literatures to examine how a childhood vulnerability may exacerbate the negative effects of earlier pubertal timing to predict adolescent depression. The present study sought to examine the main effects and interaction between pubertal timing at age 12 and childhood social wariness at ages 2, 4, 5, and 7 in predicting increases in depressive symptoms at age 17 in adolescent boys and girls, controlling for depressive symptoms at age 10. As part of an ongoing longitudinal study, mothers completed a pubertal development measure, social fear and shyness measures and children/adolescents completed self-report measures of depressive symptoms. A regression analysis was conducted to test main effects and a multigroup analysis was conducted to test the three-way interaction. As expected, pubertal timing and social wariness, independently, predicted increases in adolescent depressive symptoms. Contrary to hypotheses, the proposed three-way interaction was not found. Findings of the current study are discussed.

GROWING UP TOO FAST? AN EXAMINATION OF PUBERTAL TIMING AND CHILDHOOD SOCIAL WARINESS IN THE DEVELOPMENT OF ADOLESCENT DEPRESSION

by

Jessica M. Lee

A Dissertation
Submitted to
the Faculty of The Graduate School at
The University of North Carolina at Greensboro
in Partial Fulfillment
of the Requirements for the Degree
Doctor of Philosophy

Greensboro

2023

Approved by

Dr. Susan Keane
Committee Chair

APPROVAL PAGE

This dissertation written by Jessica M. Lee has been approved by the following committee of the Faculty of The Graduate School at The University of North Carolina at Greensboro.

Committee Chair

Dr. Susan Keane

Committee Members

Dr. Gabriela Livas-Stein

Dr. Laurie Gold

Dr. Levi Baker

June 5, 2023

Date of Acceptance by Committee

May 22, 2023

Date of Final Oral Examination

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

CHAPTER I: INTRODUCTION

Adolescence is a time of marked biological, psychological, and social transformations. As many early adolescents are not yet fully equipped with adaptive cognitive and emotional resources (Evans, Borriello, & Field, 2018; Felner et al., 1981), these challenges associated with the adolescent transition can contribute to increases in depressive symptoms by late adolescence. In fact, we know that increases in depressive symptoms typically occur from ages 12-16 (Hankin, Mermelstein, Roesch, 2007; Merikangas et al., 2010). These increases have been linked to a variety of detrimental consequences, including interpersonal difficulties, poor academic functioning, suicidal ideation, (Lewinsohn, Gotlib, Lewinsohn, Seeley, & Allen, 1998; Masten et al., 2005), and clinical depression (Miloseva et al., 2017) and may persist into young adulthood (Graber et al., 2004; Steiger et al., 2014). If left untreated, adolescent depressive symptomatology impacts myriad domains, making it an important target for research.

Utilizing a developmental psychopathology framework, researchers have investigated multiple risk and resilience factors to better understand the etiological processes that may contribute to adolescent depression. To inform prevention and intervention programs, considering how biological, psychological, and social processes involved in adolescent development are interrelated is important in identifying whether children are at risk for increases in depressive symptoms over time. One salient biological developmental milestone that is a key risk factor for adolescent depression is pubertal timing.

Conceptualizing Pubertal Timing as a Risk Factor

Pubertal timing is conceptually defined as the age of onset of puberty, relative to same-aged, same-sex peers. The average age of pubertal onset is approximately 12.34 years (Chumlea et al., 2003; Dorn et al., 2013), and 15%-20% of youth enter puberty either earlier or later than

their same-aged, same-sex peers (Chumlea et al., 2003; Biro et al., 2011). Girls tend to enter puberty approximately one year earlier than boys (Chumlea et al., 2003; Biro et al., 2011). At approximately 6-8 years of age, the onset of puberty is stimulated by the hypothalamus releasing gonadotropin-releasing hormones (GnRH), which triggers the pituitary gland to secrete several gonadotrophins (luteinizing hormone [LH], follicle-stimulating hormone [FSH]), resulting in the production of estradiol and testosterone from the ovaries and testes, respectively. This stimulates gonadarche which contributes to the growth and development of primary sexual characteristics (e.g., breast and testicles) and adrenarche which contributes to the growth of secondary sexual characteristics (e.g., pubic and axillary hair; Hayward, 2003). A child's pubertal timing is captured by determining the stage of development for each sexual characteristic, then comparing the child's overall pubertal development to their same-age, same-sex peers.

Pubertal timing is operationally defined by using either a physical exam or a self-report measure to assess the child's pubertal development relative to same-sex, same-age peers. The gold-standard for pubertal timing assessment is a physical exam, conducted by a nurse or physician, that uses the Tanner Scale to rate the current stage of each primary and secondary sexual characteristic. The Tanner Scale is a five-stage scale (Tanner Stage 1 to Tanner Stage 5) assessing for pubic hair and breast development for girls, and pubic hair and genital development for boys (Marshall & Tanner, 1970). Despite the accuracy of physical examinations as a measure of pubertal timing, it is more common for researchers to use self-report measures of sexual characteristic development (i.e., self-report version of the Tanner Scale [Marshall & Tanner, 1970], the Pubertal Development Scale [Petersen & Taylor, 1980]), which are subjective reports based on a reporter's observations (e.g., mother, child), standardized separately for boys and girls, to establish pubertal timing. Not only do pubertal timing researchers often opt for self-

report measures over physical examinations due to the invasiveness of the latter, but this format allows one to glean a better understanding of how individuals within the child's social environment perceives their biological changes.

It must be noted that chronological age is a confounding variable: as age increases, so does pubertal maturation. Thus, age is controlled for when calculating pubertal timing. Pubertal timing is most commonly calculated by regressing pubertal status on chronological age to create a standardized residualized score; this score is calculated separately for girls and boys (Ge et al., 2001). Children whose primary and secondary sexual characteristics are more developed (i.e., advanced underarm and pubic hair growth) than their same-sex, same-age peers are considered *earlier maturers,* while children who show relatively little to no development (i.e., no changes in height) when compared to their same-sex, same-age peers are considered *later maturers*. Given that earlier and later maturers experience the onset of puberty at atypical ages, they represent a deviation from the norm (Rapkin et al., 2006).

Prior research suggests that off-time (i.e., earlier or later) pubertal timing increases risk for adolescent depression, given that any deviance from typical development is inherently stressful (e.g., maturational deviance hypothesis; Petersen & Taylor, 1980). Several pioneering studies found that, when compared to *on-time* developing children, earlier and later maturers were at increased risk of having internalizing symptoms (e.g., Ge et al., 1996; Silbereisen & Kracke, 1997). However, evidence has emerged over the recent decades indicating that earlier pubertal timing contributes to greater depressive symptoms than later pubertal timing, both concurrently and longitudinally (e.g., Black & Klein, 2012; Ge et al., 1997; Graber et al., 2006; McGuire et al., 2019; Natsuaki et al., 2009; Reynolds & Juvonen, 2011), such that more pubertal

theories specifically hypothesize the detrimental impact of earlier puberty (e.g., contextual amplification hypothesis, pubertal mediation hypothesis; Ge & Natsuaki, 2009; Rudolph, 2014).

Developmental scientists propose several explanations as to why earlier pubertal timing significantly impacts children when compared to later pubertal timing. One explanation is how the physical changes associated with earlier maturation evoke reactions from the child's social environment, that is, the "social stimulus value" of earlier maturation (Petersen & Taylor, 1980, p. 137). Adults (e.g., parents and teachers) may perceive earlier maturing children to be older than their chronological age, to be more cognitively and emotionally mature, and treat them more like adults by increasing their responsibilities and duties at home and at school (Ge et al., 1996; Natsuaki, Samuels, & Leve, 2015). As peers notice earlier maturers' physique, they may identify earlier maturers as not "fitting in," given physical dissimilarities to other same-aged, same-sex peers, which may make them targets of peer teasing and rumors (Reynolds & Juvonen, 2011). Moreover, as peer group conformity and the salience of peer relationships heightens during the transition into adolescence (Rudolph, 2002; Rudolph, 2007), earlier maturing children may feel self-conscious and uncertain of the social meaning of their mature physique. This likely elicits feelings of discomfort and awkwardness, especially as overt sexual characteristics (e.g., breast development, hair growth, increased body fat) are noticeable and difficult to hide (Rudolph, 2014). Although later maturing children may also feel discomfort and awkwardness for still exhibiting a child-like physique, researchers have found that, in most cases, distress is short-lived, as later maturing children eventually catch-up with their peers and begin to become more physically similar to their same-aged, same-sex peers (Conley & Rudolph, 2012; Ullsperger & Nikolas, 2017). Taken together, off-time pubertal timing – particularly earlier pubertal timing –

comes with myriad challenges that increase the risk of adolescent depression incidence and severity.

Sex Differences in the Pubertal Timing and Depression Link

Earlier Pubertal Timing and Depression

Given earlier maturing girls typically have a longer disparity period from their peers than earlier maturing boys (Natusaki, Samuels, & Leve, 2014), researchers examining sex differences in the link between pubertal timing and depression first hypothesized that earlier maturing girls were at the greatest risk for depression (e.g., Graber, 1997). The pubertal timing literature has since expanded to comprise extensive longitudinal data with mixed-sex samples, and alternative interpretations of sex differences in pubertal timing and depression suggest that both earlier maturing girls *and* boys may be at heightened risk for adolescent depression (e.g., Mendle et al., 2010). Thus, a brief review the literature and the progress in interpretations of sex differences of earlier and later pubertal timing predicting adolescent depression is warranted.

Early empirical studies exploring pubertal timing and depression with mixed-sex samples consistently found that earlier maturing girls reported more depressive symptoms than earlier maturing boys, both concurrently and over time. Cross-sectional work by Siegel et al. (1999) indicated that self-reported earlier maturing girls reported higher depressive symptoms compared to on-time and later maturing girls and earlier, on-time, and later maturing boys. Among early longitudinal studies, Natsuaki et al. (2009a) found that by age 15, self-reported earlier maturing girls at age 12 had higher levels of depressive symptoms compared to earlier maturing boys at age 12, controlling for earlier levels of depressive symptoms. Mendle et al. (2010) also found that maternal-reported earlier maturing girls at age 9 predicted increases in depressive symptoms by age 14 compared to earlier maturing boys, after controlling for earlier levels of symptoms. It

is nonetheless important to acknowledge that, when closely examining both longitudinal studies, there was only a slightly greater effect in girls than in boys (e.g., β=0.15; Mendle et al., 2010), which supports that earlier maturing boys may also experience depressive symptoms, albeit, at slightly lower levels than girls. While examining sex differences can determine whether girls are impacted more by earlier maturity compared to boys, this limits interpretations of the data as discussions tend to focus on earlier maturing girls rather than addressing whether and how earlier puberty contributes to boys' adolescent depression.

Given that prior research focused on earlier pubertal maturation in girls and that the studies focusing on sex differences found lesser detrimental impact for boys, the effect of pubertal timing in boys has been less frequently studied and less well-understood (Mendle & Ferraro, 2012). Historically, earlier maturing boys were thought to have more advantageous outcomes, such as more positive body image (Benjet & Hernàndez-Guzmàn, 2002) and greater popularity (Clausen, 1975). However, although boys may be satisfied with their desired masculine physique, researchers are now recognizing that boys may also experience negative effects of maturing earlier and may also experience similar stressors and psychological consequences as earlier maturing girls (for a review, see Mendle & Ferraro, 2012). In a mixed-sex sample, Rudolph et al. (2014) did not find sex differences; both maternal and child reported earlier maturing girls and boys reported increases in depressive symptoms from ages 12-13 to ages 15-16, controlling for baseline levels. Hamlat et al. (2014) also did not find sex differences as self-reported earlier pubertal timing at age 12 predicted depressive symptoms nine-months later for both boys and girls. Although there were no sex differences found in any of the above studies, it was, again, noted that the effect was slightly larger in girls than boys. These findings challenge interpretations made of early work – namely, that earlier maturing boys are not at an

increased risk of depression when compared to earlier maturing girls – and support that earlier pubertal maturation impacts both boys' and girls' depressive symptoms.

Inconsistencies in the sex difference finding may be attributable to 1) differences in sample characteristics, 2) the historical precedent of female-centric interpretations – as aforementioned – and 3) the shift in the social meaning of early puberty. First, regarding differences in sample characteristics, pubertal onset can be accelerated by biological factors (e.g., obesity; Dockray et al., 2009), and other psychological adversities (childhood sexual abuse; Noll et al., 2017). Because the pubertal timing construct typically utilizes the sample as the reference group, rather than a standardized reference group, it is possible that, for example, studies with more overweight girls may have a larger number of earlier maturing girls compared to other studies with no or fewer participants with any conflating factors (e.g., obesity, childhood sexual abuse, etc.). Furthermore, since girls naturally enter puberty earlier than boys there may be a slightly larger number of same-aged, earlier maturing girls than earlier maturing boys at a given assessment point which may make it difficult to detect an effect in boys (Mendle, Turkheimer, & Emery, 2007).

Second, the historical nature of the research on puberty has primarily focused on girls, as early work from researchers typically used all-female samples and utilized the age of menarche as a means of identifying pubertal timing (e.g., Ge et al., 1996; Rierdon & Kov, 1991). This naturally propelled the discussion on earlier maturing girls being at the greatest risk for depressive symptoms to the forefront of pubertal timing literature. However, research on pubertal measurements (Dorn et al., 2011) and the biological process of puberty indicates that these early studies may not have even accurately assessed pubertal timing, as menarche happens years later from when overt signs of puberty (e.g., breast budding) are visible (Parent et al., 2003).

Consequently, all-female sample studies from the 90s may have overestimated the relation between pubertal timing and depression, with the inflated number of all-female studies advancing the idea that earlier maturing girls were at the greatest risk.

Third, the social meaning of early puberty has also been increasingly examined in recent decades, in conjunction with the recognition that earlier pubertal timing also has a negative impact in boys. More recently, psychosocial theories have explored and defined how earlier pubertal timing interacts with a contextual factor (e.g., peer stress; Conley & Rudolph, 2012; peer rejection; Teunissen et al., 2011) to promote risk or resilience to predict adolescent depression (Ge & Natsuaki, 2009; Mendle, 2019). Several psychosocial theories on earlier pubertal maturation and depression risk highlight the importance of exploring sex-specific nuances associated with gender role adherence. The gender intensification hypothesis proposes that, as children grow older, their developing physiques elicit social environments that pressure them to conform to prescribed gender roles (by engaging in increasingly gender-differentiated behaviors and attitudes; Hill & Lynch, 1983). Gender socialization processes may uniquely lead girls and boys to experience and perceive similar situations in different ways (Natsuaki, Samuels, & Leve, 2014). For example, in the transition from childhood to adolescence, peer relationships are increasingly valued, particularly for girls (Natsuaki, Samuels, & Leve, 2014; Negriff & Susman, 2011). For earlier maturing girls, the increased salience of peer relationships may increase sensitivity to negative peer reactions (e.g., peer teasing) to their physique, whereas for earlier maturing boys, their more developed physique may lead to peer pressure for the youth to behave in masculine ways (i.e., participating in masculine activities) and to peer criticism when they do not exhibit these masculine behaviors (Natsuaki, Samuels, & Leve, 2014; Negriff & Susman, 2011). Thus, although boys and girls may be similarly impacted by earlier pubertal

timing, it is imperative to discuss whether and how earlier pubertal timing impacts both boys and girls to understand these etiological processes at a deeper level.

Later Pubertal Timing and Depression

Researchers initially posited that later pubertal timing would heighten children's depression risk, in both boys and girls, given deviations from the social norm (Ge & Natsuaki, 2009). However, the evidence for later maturation impacting depression risk is comparatively scarce. For girls, a few studies suggest that earlier *and* later maturing girls are at risk for depressive symptoms compared to on-time peers, with most researchers implicating earlier maturing girls being at the greatest risk for adolescent depression compared to later and on-time maturing girls (Galvao et al., 2013; Natsuaki et al., 2009a; Rudolph et al., 2014). For boys, there is research to indicate that earlier and later pubertal timing confer risk for depressive symptoms. For example, cross-sectional studies using a self-reported pubertal timing variable, find that earlier and later maturing boys at ages 12-13 reported higher depressive symptoms compared to on-time boys (Conley & Rudolph, 2009; Graber et al., 1997; Katalia-Heino et al., 2003b). In addition, in two mixed-sex longitudinal studies, Natsuaki et al. (2009a) and Conley & Rudolph (2009) also found a curvilinear association in which both self-reported earlier and later maturing girls and boys at age 12 predicted depressive symptoms at ages 15-16, controlling for prior depressive symptoms. However, in both studies earlier maturation was more predictive of depressive symptoms than later maturation. Interestingly, Rudolph et al. (2014) found that self-reported later pubertal timing in boys at age 12 predicted higher initial depressive symptoms that declined over time, and earlier pubertal timing predicted lower initial depressive symptoms that increased by ages 15-16, controlling for prior depressive symptoms. Furthermore, in a meta-analysis, Ullsperger & Nikolas (2017) did not find a significant association between later

pubertal timing measured between ages 12-13 and depressive symptoms at ages 15-16 compared to on-time peers for girls and boys. Overall, earlier pubertal maturation seems to have detrimental and persistent effects whereas later pubertal maturation contributes to initial or short-term effects that may dissipate over time.

When looking to understand the weaker association between later pubertal timing and depressive symptoms, when compared to earlier pubertal timing, we can examine: 1) methodological limitations, and 2) different theoretical explanations. First, regarding methodological issues, most studies on later maturation have relied on cross-sectional designs whereas studies indicating earlier puberty to be a risk factor utilized longitudinal designs (Rudolph, 2014). Second, one proposed explanation as to why later maturing girls and boys may not experience as many detrimental depressive effects is that – being the last to enter puberty within their peer groups, having seen their peers experience puberty – later maturers may be more cognitively and emotionally prepared for the pubertal process (Mendle & Ferraro, 2012; Rudolph, 2014; Ullsperger & Nikolas, 2017). Another explanation may be that later maturers have a shorter disparity period between average age of onset of puberty (age 12) to when later maturers most frequently begin puberty (age 13; Rudolph, 2014). Though pubertal timing disparities may cause distress in later maturers while their physiques remain child-like, this distress is resolved through their eventual pubertal onset and physical maturation, such that they again blend with their peer group. Thus, although earlier maturing children also begin to blend with their peer group around the average age of pubertal onset (e.g., 12 years old), the period of ages 10-12 may be a sensitive window as the initial distress earlier maturers experience at pubertal onset may be more salient given it tends to persist into the adolescent transition contributing to increases in depressive symptoms (Barendse et al., 2022; Rudolph et al., 2014).

Considered together, the literature suggests that the persistent negative effects of earlier pubertal timing may increase the risk for depressive symptoms by mid- to late adolescence. However, other variables, in addition to pubertal timing, are likely implicated in adolescent depression. As children begin to ascend into this developmental and pubertal transition, it is important to also consider a childhood predisposing factor that may exacerbate the risk for adolescent depression.

Childhood Predisposing Vulnerability

In addition to identifying earlier pubertal timing as an individual risk factor for adolescent depression, researchers began to explore other individual factors that might interact with pubertal timing effects to predict increases in depressive symptoms. The contextual amplification hypothesis suggests that a child's individual vulnerability may serve to exacerbate or ameliorate the negative effects of earlier pubertal timing to predict emotional and/or behavioral problems (Ge & Natsuaki, 2009). This theory is guided by the diathesis-stress model as it utilizes the person-by-interaction perspective and focuses on the interaction between earlier puberty and predisposing vulnerability as contributors to adolescent psychopathology (Ge et al., 2011; Rudolph, 2014). The present study incorporates this theoretical perspective to determine whether a relevant and specific childhood vulnerability exacerbates or buffers the negative effects of earlier pubertal timing on adolescent depression.

Among the few studies that have explored this interaction, vulnerability factors have included behavioral (e.g., antisocial problems; Caspi & Moffit, 1991) and emotional problems (e.g., nervousness, worry; Benoit et al., 2018) in children between the ages of 9-11. To the author's knowledge, no studies exist which examine childhood factors in children younger than 9-years old, or before the age of onset of puberty. This gap in the literature may be attributable to

the lack of longitudinal studies addressing this question that span multiple developmental periods, because they are time-intensive, labor-intensive, and expensive to conduct (Ge & Natsuaki, 2009). This highlights the value of studies that are able to address how an early childhood characteristic, such as a temperamental disposition, might make earlier maturers more vulnerable to increases in depressive symptoms by late adolescence.

There are several reasons why it is important to consider how an early childhood factor might exacerbate the negative effects of earlier pubertal timing. One reason is that this can help to better understand how a child with an underlying predisposing vulnerability has an increased risk of developing depressive symptoms, providing predictive information that can inform effective preventative care. Another reason is the increased social salience that occurs during the transition from childhood to adolescence. Research examining the interaction of early childhood factors and pubertal timing may provide context as to how socially relevant factors, identified in early childhood – such as reticence with peers or being more of a "wallflower" – may impact how earlier maturers react to different social responses (e.g., from parents, peers, or teachers, Natsuaki, Samuels, & Leve, 2014). For example, earlier maturers who exhibit socially reluctant behaviors in early childhood could face multiple challenges when experiencing increased attention or scrutiny from peers, or increased expectations of social maturity from adult figures (e.g., parents, teachers). Thus, an earlier maturing child who exhibits a childhood vulnerability that is social in nature may experience heightened levels of adolescent depression compared to non-socially vulnerable earlier maturing children.

Childhood Social Wariness

Childhood social wariness is one social vulnerability in early childhood that could be a predisposing risk factor. *Social wariness* is conceptually defined as socially reluctant behavior

that often emerges in novel or unpredictable social situations (Rubin et al., 2009; Rubin et al., 2018). Maternal-reported social wariness is moderately stable from toddlerhood to middle childhood (Rubin et al., 1999; Degnan et al., 2008; Jarcho et al., 2019). A socially wary child may hesitate to initiate and socially engage with both familiar and unfamiliar peers, typically avoids social interactions or waits for others to initiate interactions with them (Rubin et al., 1999). When they do engage with others, socially wary children may be unsure if these interactions will be successful, which can cause distress. For example, a socially wary child is less likely to initiate a social interaction when standing near a group of children due to the fear of experiencing an unsuccessful and, therefore, stressful interaction. As the socially wary child is timid, successfully navigating a social interaction may overtax their self-regulatory abilities (Rubin et al., 1999).

Social wariness has been operationalized utilizing a variety of measurements, including parent and/or teacher self-report measures and behavioral observation tasks. Researchers using informant measures typically utilize a toddler temperament measure that assesses social fear (e.g., Toddler Behavior Questionnaire; Goldsmith, 1996) in toddler-aged children, and a school-aged temperament measure that assesses shyness (e.g., Child Behavior Questionnaire; Rothbart, Ahadi, Hershey, & Fisher, 2001) in school-aged children. Behavioral observation tasks (e.g., Play Observation Scale; Rubin, 1989) provide objective measurements using real-time observations of a child's social wariness within a controlled lab setting. These behavioral observation tasks typically capture the child's frequency of "onlooking" and "unoccupied behaviors" with unfamiliar, same-aged peers (Rubin, 2001). To fully capture social wariness across the developmental periods, many researchers have created a composite score averaging the scores from mother reported measures of social fear in toddlers and shyness in young

children (e.g., Rubin et al., 1999; Jarcho et al., 2019; Mills et al., 2012; Natsuaki et al., 2013). Others create composite scores derived from the behavioral observation task at one time-point and temperament measures assessing shyness at ages 2-7 (e.g., Degnan et al., 2008; Guyer et al., 2015; Hastings & Kahle, 2014; Henderson, Fox, Rubin, 2001). Utilizing maternal reports of toddler and child temperament of social fear and shyness across both developmental periods has proven to be advantageous, given that this approach captures observations of social wariness by a reliable same reporter (e.g., mother), who has high familiarity with the child. Consistent with Rubin et al. (1999) and Jarcho et al. (2019), the current study conceptually and operationally defines social wariness as comprising social fear in toddlerhood and shyness in early-to-middle childhood and explores whether socially wary behavior in childhood presents as an individual and interactive vulnerability to adolescent depression.

Social Wariness and Depression

Socially wary children are more likely to develop concurrent and subsequent depressive symptoms as they experience frequent distress during social interactions (Brooker et al., 2016). These children may also develop behavioral tendencies outside the social norm (e.g., inconsistent eye contact, stuttering) and display poorer social skills (Doey, Coplan, & Kingsbury, 2014) while anticipating negative feedback (e.g., teasing, ignoring, ostracizing) from peers (Rubin et al., 2009; Guyer et al., 2015). As a result, socially wary children may tend to avoid certain people or types of social settings (i.e., group activities) and engage in fewer social interactions with peers to reduce their levels of distress (Mills et al., 2012). Short-term longitudinal studies have yielded significant associations for children with maternal-reported social wariness and depressive symptoms in toddlerhood after a 5-month follow-up (Mills et al., 2012) and in early childhood (Coplan et al., 2008; Hastings & Kahle, 2014) as well as internalizing symptoms in middle

childhood (e.g., Brooker et al., 2016). Taken together, although past research has examined the negative impact of childhood social wariness subsequently in childhood, no research has yet examined whether these effects persist into adolescence. As the enduring effects of social wariness may lead to detrimental consequences in adolescence, such as poor social and academic competence and internalizing symptoms (Rubin et al., 1999, Zeytinoglu et al., 2022), the current study will also examine whether childhood social wariness is associated with adolescent depressive symptoms.

Social Wariness and Earlier Pubertal Timing

Social wariness as a childhood vulnerability may predispose an emerging adolescent to have a heightened response to earlier puberty. A socially wary earlier maturer, not otherwise used to receiving significant attention, must manage the social responses elicited by their more advanced physique at an increasing frequency that may feel overwhelming to them. This can lead to the emerging adolescent avoiding people (e.g., peers who tease) or types of social settings (e.g., group activities) to reduce distress, which limits the quantity and quality of peer relationships during a developmental period when peer relationships are highly salient and valuable (Mills et al., 2012). Based on the literature differentiating boys' and girls' experiences of earlier pubertal maturation, it is possible that earlier maturing girls and boys who were socially wary may also have different experiences of associated psychosocial stress. Girls may be especially sensitive to overt negative social reactions (e.g., peer teasing) in response to their more advanced physique, and they may struggle to initiate and maintain highly-valued positive social relationships with peers. Boys, on the other hand, may experience distress in response to the heightened expectations of masculine role adherence, associated with a more mature physique. Thus, the effects of the earlier pubertal timing, combined with childhood social

wariness, could elicit more social challenges (e.g., peer ostracizing, a child having difficulty advocating for themselves), and given girls typically highly value and are more sensitive to negative social interactions (Rose & Rudolph, 2002), earlier maturing girls who were socially wary as children may report slightly greater increases in depressive symptoms, over time, than boys.

Control Variable

Earlier depressive symptoms are robust predictors of later depressive symptoms (Black & Klein, 2012; Klein et al., 2009; Luby et al., 2009). The current study will examine, as a potential control variable, depressive symptoms at age 10, given that symptoms typically emerge in middle childhood (Hankin, Mermelstein, Roesch, 2007; Merikangas et al., 2010).

Goals and Hypotheses

The current study has three goals. Prior research has established earlier pubertal timing as a risk factor for adolescent depressive symptoms. Previous research has also established childhood social wariness as a risk factor for concurrent or subsequent depressive symptoms in short-term longitudinal studies (e.g., 3-years). However, no study has yet examined the long-term impact of social wariness on adolescent depression. Thus, the first goal of the current study is to examine whether pubertal timing and childhood social wariness, independently, predict increases in adolescent depressive symptoms at age 17.

Second, prior literature has examined concurrent or later behavioral and emotional factors as moderators of the relation between pubertal timing and adolescent depression, calling upon the contextual amplification hypothesis framework. However, thus far, no study has yet integrated the childhood social wariness and pubertal timing literatures to investigate how a childhood social vulnerability may make earlier maturers more susceptible to increases in

adolescent depression. The second goal of this study is to examine whether an early childhood factor (i.e., childhood social wariness) heightens the negative effects of earlier pubertal timing to predict increases in depressive symptoms by late adolescence.

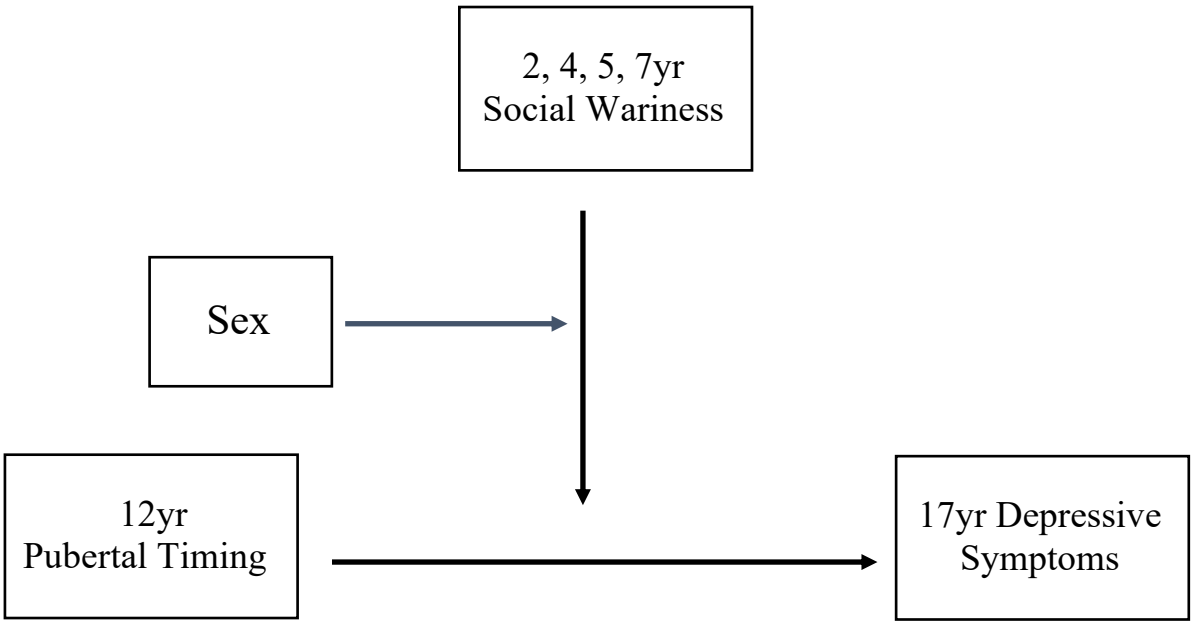
Third, in light of the inconsistent sex difference finding in the relation between earlier pubertal timing and adolescent depressive symptoms with recent evidence indicating that both earlier maturing girls *and* boys are at risk for depressive symptoms – with a slightly more robust effect observed in girls (e.g., Rudolph et al., 2014) – it is imperative to continue to determine whether there is a small discrepancy between earlier maturing boys' and earlier maturing boys' increases in depressive symptoms within the context of an individual vulnerability to provide further evidence that they both do, indeed, experience detrimental outcomes. Thus, this study seeks to expand the research on sex differences in earlier pubertal timing, specifically following the contextual amplification hypothesis and diathesis stress model, by examining sex as a second moderator in the relation between the interactive effects of earlier pubertal timing and social wariness predicting increases in depressive symptoms in late adolescence.

Author's hypotheses were as follows:

1. Pubertal timing at age 12 and childhood social wariness (a latent variable based on maternal report at ages 2, 4, 5, and 7), separately, will predict higher depressive symptoms at age 17, controlling for earlier depressive symptoms at age 10.
2. Childhood social wariness will moderate the relation between pubertal timing at age 12 and depressive symptoms at age 17, controlling for earlier depressive symptoms at age 10. More specifically, it was hypothesized that earlier maturing children who were socially wary will demonstrate increases in depressive symptoms at age 17, controlling for prior levels of depressive symptoms at age 10.

3. Childhood social wariness and sex will moderate the relation between pubertal timing and depressive symptoms, such that earlier maturing girls at age 12 with higher levels of childhood social wariness at ages 2, 4, 5, and 7 will predict increases in depressive symptoms by age 17, controlling for prior levels of depressive symptoms at age 10 (see Figure 1).

Figure 1. Conceptual Model



Note. The moderating role of sex on the interaction between pubertal timing and social wariness predicting depressive symptom.

CHAPTER II: METHOD

Participants

The current study utilized data from three cohorts of children who are part of an ongoing longitudinal study of social and emotional development. The goal for recruitment was to obtain a sample of children who were at risk for developing future externalizing behavior problems, and who were representative of the surrounding community in terms of race and socioeconomic status (SES). All cohorts were recruited through child day care centers, the County Health Department, and the local Women, Infants, and Children (WIC) program. Potential participants for Cohorts 1 and 2 were recruited at 2-years of age (Cohort 1: 1994-1996 and Cohort 2: 2000-2001) and screened using the Child Behavior Checklist (CBCL 2-3; Achenbach, 1992), completed by the mother, in order to over-sample for externalizing behavior problems. Children were identified as being at risk for future externalizing behaviors if they received an externalizing T-score of 60 or above. Efforts were made to obtain approximately equal numbers of males and females. This recruitment effort resulted in a total of 307 children. Cohort 3 was initially recruited when infants were 6 months of age (in 1998) for their level of frustration, based on laboratory observation and parent report, and were followed through the toddler period (see Calkins, Dedmon, Gill, Lomax, & Johnson, 2002, for more information). Children from Cohort 3 whose mothers completed the CBCL at two-years of age ($N = 140$) were then included in the larger study. Of the entire sample ($N = 447$), 37% of children were identified as being at risk for future externalizing problems. There were no significant demographic differences between cohorts with regard to gender, $\chi^2(2, N = 447) = .63$, $p = .73$, race, $\chi^2(2, N = 447) = 1.13$, $p = .57$, or two-year SES, $F(2, 444) = .53$, $p = .59$.

Of the 447 originally selected participants, six were dropped because they did not participate in any data collection at 2 years old. An additional 12 families participated at recruitment, did not participate at two-year, but did participate at later years. At age 4, 399 families participated. Families lost to attrition included those who could not be located, moved out of the area, declined participation, or did not respond to phone and letter requests to participate. There were no significant differences between families who did and did not participate at age four in terms of gender, χ^2 (1, N = 447) = 3.27, p = .07, race, χ^2 (1, N = 447) = .65, p = .42, two-year SES, t (432) = -.92, p = .36, or 2-year externalizing T score, t (445) = .45, p = .65. At age 5, 365 families participated, including four that did not participate in the four-year assessment. Again, there were no significant differences between families who did and did not participate in terms of gender, χ^2 (1, N = 447) = .76, p = .38, race, χ^2 (1, N = 447) = .14, p = .71, 2-year SES, t (432) = -1.93, p = .06, and 2-year externalizing T score, t (445) = 1.39, p = .17. At 7 years of age, 350 families participated, including 19 that did not participate in the 5-year assessment. Again, there were no significant differences between families who did and did not participate in terms of gender, χ^2 (1, N = 447) = 2.12, p = .15, race, χ^2 (1, N = 447) = .19, p = .67, and two-year externalizing T score, t (445) = 1.30, p = .19. Families with lower 2-year SES, t (432) = -2.61, p < .01, were less likely to participate in the 7-year assessment. At age 12, 222 families participated, including 13 families that did not participate in the 7-year assessment. There were no significant differences between families who did and did not participate in the 7-year assessment in terms of race χ^2 (3, N = 447) = 7.49, p = .06; 2-year SES t(432) = -1.82, p = .07; or 2-year externalizing T score t (445) = 1.92, p = .06. Boys were less likely to participate in the 12-year assessment χ^2 (1, N = 447) = 5.86, p = .01. At age 17, 307 families participated, including 112 families that did not participate in the 12-year assessment.

There were no significant differences between families who did and did not participate in the 7-year assessment in terms of 2-year SES $t(432) = .99$, $p = .32$ or 2-year externalizing *T* score $t(445) = 1.28$, $p = .20$. Non-White individuals χ^2 (3, $N = 447$) = 7.99, $p = .04$ and boys were less likely to participate in the 17-year assessment χ^2 (1, N = 447) = 3.89, p = .04. In addition, four participants were dropped from the current study due to developmental delays.

Of the 447 original participants, 20 of them were removed as they had missing data on pubertal development at age 12, social wariness at ages 2, 4, 5, and 7, *and* depressive symptoms at ages 10 and 17. Thus, the sample for the current study included 427 children/families (203 males, 224 females) given they had data on pubertal development at age 12, social wariness at ages 2, 4, 5, and 7 *and/or* depressive symptoms at ages 10 and 17. The sample was diverse with 66.9% White/European American, 27.1% Black/African American, 5.9% mixed or other. Families were economically diverse based on Hollingshead (1975) scores at the 2-year assessment, with a range from 14 to 66 ($M = 39.61$, SD = 11.12), thus representing families from each level of social strata typically captured by this scale. Hollingshead scores that range from 40 to 54 reflect minor professional and technical occupations considered to be representative of middle class. However, to address missing data and increase power, Full Information Maximum Likelihood (FIML) was used in Mplus 8 (Muthén & Muthén, 2017) as it included all available information on all participants and estimated data for missing cases. This was the least biased method of including the maximum amount of data as it calculated the covariance and correlation matrices derived from the data from each study variable.

Procedures

Children and their mothers participated in an ongoing longitudinal study beginning at age 2. The current analysis used the data collected from several time-points: ages 2, 4, 5, 7, 10, 12,

and 17. Maternal reports of their child's behavior was obtained at ages 2, 4, 5, 7 and 12. Children self-reported on depressive symptoms at ages 10 and 17. Assent and consent from the child and the caregiver were obtained prior to the start of any data collect at each time point. At the age of 2, the child and the mother came to the laboratory where the mother and child completed a series of laboratory tasks designed to elicit emotional and behavioral responses and completed parent measures. At the ages of 4, 7, 10, and 17, mothers and children returned to visit the lab to complete another series of tasks as well as self-report measures. At the age of 12, self-report measures were mailed to the family and were requested to be mailed back to the university lab. The families were compensated for their participation after each visit. Only the measures relevant for the current study are reported here.

Measures

Pubertal Timing at Age 12

Data for the 6-item mother report of pubertal status using the Pubertal Developmental Scale (PDS; Peterson et al., 1988) was used to assess pubertal timing. The PDS measures the current development of primary and secondary sexual characteristics for both girls and boys on a Likert rating scale of 1 (*no*) to 4 (*development completed*) for each item. For both sexes, the PDS assessed maternal-reported body hair development, height, and skin changes. For girls, development of breasts and the age of first menarche were also reported. For boys, the development of facial hair and voice changes was included. The PDS yielded a composite score by averaging the six items within each sex; higher scores indicated more advanced pubertal development.

A pubertal timing variable was created following the work of Ge and colleagues (Ge, Conger, & Elder, 2001). Pubertal timing accounts for the effects of age by regressing pubertal

status on same-age, same-sex which creates a standardized residual to capture pubertal timing on a continuous level (M=0, SD=1; Ge, Conger, & Elder, 2001). Using this standardized score, pubertal timing is operationalized as either a dichotomous variable (early vs not early), categorical (early, on-time, late) or continuous (earlier vs later; Dorn et al., 2006; Dorn & Biro, 2013). Studies using a dichotomous variable typically define early maturers as children with a score of +1 (i.e., one standard deviation above the mean) whereas anyone less than +1 (i.e., less than one standard deviation above the mean) is defined as not early (e.g., Angold et a., 1999). Studies using a categorical variable often define on-time as children who score between -1 and +1 standard deviation from the mean, early as children who score more than 1 standard deviation above the mean, and late as children who score less than 1 standard deviation below the mean (e.g., Graber, 1997). Studies using a continuous or dimensional variable do not include truncated cutoffs to reflect a continuum of earlier to later pubertal timing scores (e.g., Rudolph et al., 2014). Using a continuous variable is advantageous given it increases variability and power as it maximizes the full range of values and is not truncated into smaller groups (Dorn & Biro, 2011; Dorn et al., 2006). Thus, the most common and advantageous method of measuring pubertal timing is operationalizing it as a continuous variable. The current study used a continuous measure of pubertal timing. Higher scores indicated earlier pubertal timing.

Social Wariness at Ages 2, 4, 5 and 7

Consistent with Rubin et al. (1999) and Jarcho et al. (2019), social wariness was captured across time using a latent variable of social wariness based on maternal report of the social fear subscale at age 2 on the Toddler Behavior Assessment Questionnare (TBAQ; Goldsmith, 1996) and maternal report of the shyness subscale at ages 4 and 5 on the Children's Behavior Questionnaire Long Form (CBQ-LF; Rothbart, Ahadi, Hershey, & Fisher, 2001) and at age 7 on

the Children's Behavioral Questionnaire Short Form (CBQ-SF; Rothbart, Ahadi, Hershey, & Fisher, 2001) as this method of measurement provided a consistent measure using developmentally appropriate temperament measures and a constant reporter who was highly familiar with the child across time. The TBAQ is a 111-item measure designed to assess toddler temperament, specifically for children between 16 and 36 months. Parents rated their toddler's behavior on a scale from 1 to 7, with an additional option to select "NA" for "Does not apply," with "1" indicating "Never," "2" indicates "Very Rarely," "3" indicates "Less than half the time," "4" indicates "About half the time," "5" indicates "More than half the time," "6" indicates "Almost always," and "7" indicates "Always." The TBAQ yields 5 subscales: activity level, anger proneness, social fear, pleasure, and interest/persistence. The 19-item social fear subscale was utilized to measure the frequency of behaviors indicating distress, inhibition, or shyness in novel or uncertainty-provoking situations of a social nature.

The maternal report of the CBQ-LF at ages 4 and 5 and CBQ-SF at age 7 captures developmentally appropriate measures of social wariness through the Shyness subscale. The CBQ-LF and CBQ-SF is a 195-item and 94-item measure, respectively, designed to assess temperament, defined as a biologically-based measure of differences in levels of reactivity, or arousability of various response systems, and self-regulation, or processes that modulate reactivity (Putnam & Rothbart, 2006). The parent was asked to read the items about their child's reaction to a variety of situations and decide to what extent each item is true or untrue. Each item is rated on a scale from 1 to 7, with the additional option of selecting "N/A" or "Not Applicable." A response of "1" indicates "Extremely Untrue," a response of "4" indicates "Neither True nor Untrue," and a response of "7" indicates "Extremely True." The CBQ-LF and CBQ-SF yields 15 subscales: activity level, anger/frustration, attentional focusing, attentional shifting, discomfort,

fear, high intensity pleasure, impulsivity, inhibitory control, low intensity pleasure, perceptual sensitivity, approach/positive anticipation, sadness, shyness, smiling/laughter, and soothability. Since social fear is not considered on this measure due to developmental considerations at this age, the CBQ-LF 13-item and CBQ-SF 7-item shyness subscale was utilized to measure the frequency of behaviors indicating the extent to which a slow or inhibited approach occurs in social situations involving novelty or uncertainty. An example is "Rarely enjoys being talked to," "Gets embarrassed when strangers pay a lot of attention to her/him."

Depressive Symptoms at Age 17

The 12-item depression subscale within the 176-item adolescent self-report of the Behavioral Assessment Scale for Children, Fourth Edition (BASC-SRP-A; Reynolds & Kamphaus, 1992) was used to assess the level of depressive symptoms. The BASC-SRP-A is a multidimensional measure that assesses the adaptive and maladaptive behavior of adolescents from ages 12 to 21 on a rating scale from 0 to 3, with "0" indicating "never", "1" indicating "sometimes", "2" indicating "often", and "3" indicating "almost always." It contains 5 composites: Emotional Symptom Index, Inattention/Hyperactivity, Internalizing Problems, Personal Adjustment, and School Problems. Within the Internalizing Problems Composite, the depression subscale measures the frequency of experiencing depressive symptoms, such as the adolescent's feelings of sadness. An example item is "Nothing is fun anymore," or "I feel like my life is getting worse and worse." A general raw score was utilized rather than a T-score as the sample was a community one and clinically significant levels of reported depression were rare.

Depressive Symptoms at Age 10

The 12-item depression subscale within the 139-item child self-report of the Behavioral Assessment Scale for Children, Fourth Edition (BASC-SRP-C; Reynolds & Kamphaus, 1992)

was used to assess the level of depressive symptoms. The BASC-SRP-C is a multidimensional measure that assesses the adaptive and maladaptive behavior of children from ages 8 to 11on a rating scale from 0 to 3, with 0 (never), 1 (sometimes), 2 (often), and 3 (almost always). It contains 5 composites: Emotional Symptom Index, Inattention/Hyperactivity, Internalizing Problems, Personal Adjustment, and School Problems. Within the Internalizing Problems Composite, the depression subscale measures the frequency of experiencing depressive symptoms, such as the adolescent's feelings of sadness. An example item is "Nothing is fun anymore," or "I feel like my life is getting worse and worse." Following the logic above, a general raw score was utilized rather than a T-score as the sample was a community one.

CHAPTER III: DATA ANALYTIC PLAN

First, a latent variable of social wariness was created in Mplus 8 (Muthén & Muthén, 2017) to ensure that the selected items loaded onto this underlying construct and that the construct existed within the current sample. This minimized measurement error, as the assumption that all items loaded on to the construct equally was removed. A second-order confirmatory factor analysis (CFA) was then conducted to verify that items loaded onto first-order factors that, in turn, loaded onto the second-order or primary factor which was the theorized construct. The CFA was created using the weighted least squares with means and variances (WLSMV; Muthén & Muthén, 2007) as it was designed for categorical variables with non-normal distributed data making it the least biased method (Li, 2016).

In order to identify a CFA model, fit indices were evaluated. If one or more fit indices indicated poor fit, modification indices were calculated to attain improved fit by examining whether one or more items did not load onto the social wariness construct. Item deletion criteria included any items with a factor loading of less than 0.4 and an R-square value of less than 0.2. Given the initial CFA model had one or more fit indices that indicated poor fit, an exploratory factor analysis (EFA) was conducted to determine whether items fell into one or more factors that best represented the latent social wariness variable. Once the appropriate EFA model was identified, it was tested using a CFA to confirm that the first- and second-order factors, and their respective items, loaded onto social wariness. A chi-square difference test was utilized to determine whether each new model generated by the EFA fit the data better than the original model by calculating the difference between chi-square values and observing whether the difference surpassed the critical value until one model was identified with the best fit and the most parsimonious.

Preliminary analyses were conducted using SPSS version 23 (IBM corp, 2021) including correlations, *t*-tests, and the descriptive statistics for all study variables, including the latent variable social wariness. Independent-samples *t*-tests were calculated to indicate whether there were significant mean differences between females and males.

Mplus 8 (Muthén & Muthén, 2017) was also utilized to conduct the main analyses in the full sample. Given Mplus 8 does not calculate hierarchical regression analyses, rather, it simultaneously computes the regression analyses included in the model, separate regression analyses were conducted in Mplus to test whether social wariness and pubertal timing were associated with depressive symptoms at age 17, controlling for earlier depressive symptoms at age 10. Fit indices were evaluated.

To test the two- and three-way interactions, a product-term regression and multigroup analysis, respectively and separately, were conducted by including relevant control variables, predictor variables, and the interaction simultaneously into the model. Fit indices were evaluated for each model. In order to test for moderation in the multigroup analysis, two models were compared using the chi-square difference test: an *unconstrained* model in which all paths between males and females were freely estimated across sex and a *constrained* model in which all structural weights were set and estimated to equal across both sexes. A chi-square difference test was used to test whether the unconstrained model fit better than the constrained model. Bootstrapping is a non-parametric method that estimates the sampling distribution of the parameter estimates based on resampling with replacement from the current data set. Given bootstrapping does not assume normality and reduces Type I error, it was used to infer the effect of the 95% bias-corrected confidence intervals to determine the significance of the conditional

effects (MacKinnon, Lockwood, & Williams, 2004). A bootstrapped 95% bias-corrected confidence interval that does not include zero indicated a statistically significant effect.

An a priori alpha level of .05 was used to determine the significance of all tests.

CHAPTER IV: RESULTS

Social Wariness Latent Variable Analyses

Confirmatory Factor Analysis (CFA)

Based on Jarcho et al. (2019) and others' (e.g., Brooker et al., 2016; Rubin et al., 1999) factor structure of social wariness consisting of items of social fear and shyness from the same or similar measures (e.g., Colorado Child Temperament Inventory; Buss & Plomin, 1984), a second-order confirmatory factor analysis (CFA) was conducted to determine whether the items from the 2-year social fear subscale from the TBQ and the 4-year, 5-year, and 7-year shyness subscale from the CBQ load onto the construct called social wariness. The fit of the CFA was tested relative to alternative factor structures to determine the best factor structure, along with theory, that best represented the data. The model was considered to be a good fit when the comparative fit index (CFI; Marsh & Hau, 2007) and the Tucker-Lewis index (TLI; Bentler, 1990) values were close to or greater than .95, the standardized root mean square residual (SRMR) value was less than or equal to .08., and the root mean square error of approximation (RMSEA; Cole & Maxwell, 2003) value was less than or equal to .08. Three competing models or factor structures were created. A one-factor (Model 1), where all items were regressed on a single factor (e.g., Social Wariness), a second-order factor model (Model 2) with four first-order factors (e.g., TBQ at Age 2, CBQ at Age 4, CBQ at Age 5, CBQ at age 7), where items from each subscale were regressed on its respective factor and these first-order factors were regressed on the second-order factor (e.g., Social Wariness), and, lastly, a second-order factor model (Model 3) with eight first-order factors (e.g., 1) TBQ doctor's office, 2) TBQ unfamiliar adult, 3) TBQ parent leaves the home, 4) TBQ unfamiliar adult, 5) TBQ unfamiliar adult, 6) CBQ at Age 4, 7) CBQ at Age 5, and 8) CBQ at age 7) were compared using the chi-square difference test

(see Table 1). Evaluation of fit indices and factor structure development for all three models are discussed below.

Table 1. Second-Order Confirmatory Factor Analysis Structure of Model 1, Model 2, and Model 3

Model 1			Model 2			Model 3		
Social Wariness	2YR TBQ 4YR CBQ 5YR CBQ 7YR CBQ	T4R, T66, T67R, T68, T73R, T74R, T75R, T81, T82, T83R, T84, T85, T86R, T102R, T103R, T104R, T105R, T106, T107, 4C7, 4C37, 4C74, 4C89, 4C106, 4C143, 4C17, 4C23, 4C57, 4C45, 4C119, 4C129, 4C158, 5C7, 5C37, 5C74, 5C89, 5C106, 5C143, 5C17, 5C23, 5C57, 5C45, 5C119, 5C129, 5C158, ,7C37, 7C42, 7C52, 7C70, 7C11, 7C60.	Social Wariness	2YR TBQ	T4R, T66, T68, T73R, T74R, T75R, T81, T82, T83R, T84, T85, T86R, T104R, T105R, T106, T107	Social Wariness	2YR TBQ 1	T66, T67R, T68.
				4YR CBQ	4C7, 4C37, 4C74, 4C89, 4C106, 4C143, 4C17, 4C23, 4C57, 4C45, 4C119, 4C129, 4C158.		2YR TBQ 2	T81, T82, T83R.
				5YR CBQ	5C7, 5C37, 5C74, 5C89, 5C106, 5C143, 5C17, 5C23, 5C57, 5C45, 5C119, 5C129, 5C158.		2YR TBQ 3	T84, T85, T86R.
				7YR CBQ	7C37, 7C42, 7C52, 7C70, 7C11, 7C60.		2YR TBQ 4	T73R, T74R, T75R.
							2YR TBQ 5	T105R, T106, T107.
							4YR CBQ	4C7, 4C37, 4C74, 4C89, 4C106, 4C143, 4C17, 4C23, 4C57, 4C45, 4C119, 4C129, 4C158.
							5YR CBQ	5C7, 5C37, 5C74, 5C89, 5C106, 5C143, 5C17, 5C23, 5C57, 5C45, 5C119, 5C129, 5C158.
							7YR CBQ	7C37, 7C42, 7C52, 7C70, 7C11, 7C60.

Model 1 provided a poor fit to the data [$\chi^2(1224) = 5402.945$, $p < .0001$, RMSEA = .09, CFI = .591, SRMR = .09] with all 51 items. Removing items according to the deletion criteria (e.g., items with a factor loading less than 0.4) would have taken a significant number of items from the TBQ alone (see Table 2).

Table 2. Confirmatory Factor Analysis for Model 1

	Factor Loading	R-square		Factor Loading	R-square		Factor Loading	R-square
T4R	.360	.129	4C17R	.707	.500	7C37	.544	.296
T66	.230	.053	4C23R	.776	.576	7C42	.596	.356
T68	.211	.045	4C89	.615	.379	7C52	.691	.477
T67R	.232	.054	4C57R	.768	.590	7C70	.653	.426
T81	.225	.051	4C106R	.745	.555	7C117R	.694	.481
T82	.243	.059	4C143R	.654	.427	7C607R	.430	.185
T83R	.223	.050	4C45R	.679	.461			
T84	.303	.092	4C119R	.522	.272			
T85	.364	.132	4C129R	.815	.664			
T86R	.373	.139	4C158R	.746	.556			
T73R	.418	.175	5C7	.525	.276			
T74R	.363	.132	5C17R	.646	.417			
T75R	.355	.126	5C23R	.776	.602			
T102R	-.318	.101	5C37	.645	.416			
T103R	.169	.029	5C57R	.744	.553			
T104R	.340	.115	5C74	.627	.393			
T105R	.422	.178	5C89	.618	.383			
T106	.333	.111	5C106	.762	.581			
T107	.363	.132	5C119R	.585	.342			
4C7	.504	.254	5C129R	.816	.666			
4C37	.593	.352	5C143	.740	.548			
4C74	.525	.276	5C158	.738	.545			

Thus, Model 2 was constructed so that the four first-order factors represented the subscales from the TBQ and CBQ and these first-order factors loaded onto one second-order factor, social wariness. Model 2 provided an improvement in fit [$\chi^2(1070) = 3252.159$, $p < .0001$, RMSEA = .07, CFI = .910, SRMR = .08] after removing items that met the deletion criteria (see Table 3).

Table 3. Confirmatory Factor Analysis for Model 2

	Factor Loading	R-square		Factor Loading	R-square		Factor Loading	R-square
2YR TBQ	**.451**	**.203**	45R	.732	.536	**7YR CBQ**	**.845**	**.714**
66	.595	.354	74	.585	.342	37	.515	.517
68	.555	.308	7	.542	.294	42	.516	.531
4R	.455	207	89	.669	.448	52	.761	.781
81	.851	.724	119R	.566	.321	70	.652	.670
82	.862	.743	106	.829	.688	117R	.713	.738
83R	.538	.290	129R	.877	.780	607R	.311	
84	.456	.208	143	.740	.644			
85	.530	.281	158	.802	.642			
86R	.607	.369	**5YR CBQ**	**.947**	**.896**			
73R	.626	.391	7	.597	.357			
74R	.546	.298	17R	.708	.501			
75R	.539	.291	23R	.842	.709			
104R	.487	.237	37	.687	.472			
105R	.668	.447	57R	.804	.646			
106	.452	.204	74	.690	.475			
107	.560	.314	89	.657	.431			
4YR CBQ	**.899**	**.809**	106	.839	.704			
17R	.770	.593	119R	.674	.454			
23R	.815	.664	129R	.883	.780			
37	.638	.408	143	.802	.644			
57R	.818	.669	158	.801	.642			

The chi-square difference test was significant [$\Delta\chi^2(154) = 2150.786$, $p < .0001$] which indicated that Model 2 fit the data better than Model 1. However, the social fear first-order factor did not have a strong loading, as it fell near the minimum cutoff of 0.4, onto the social wariness second-order factor compared to 4-year, 5-year and 7-year shyness factors (see Table 4).

Table 4. Model 1, Model 2, and Model 3 Fit Indices

	χ^2	p-value	RMSEA	CFI	SRMR
Model 1	5402.945	p< .0001	.09	.591	.09
Model 2	3252.159	p< .0001	.07	.910	.08
Model 3	2114.165	p< .0001	.06	. 942	.06

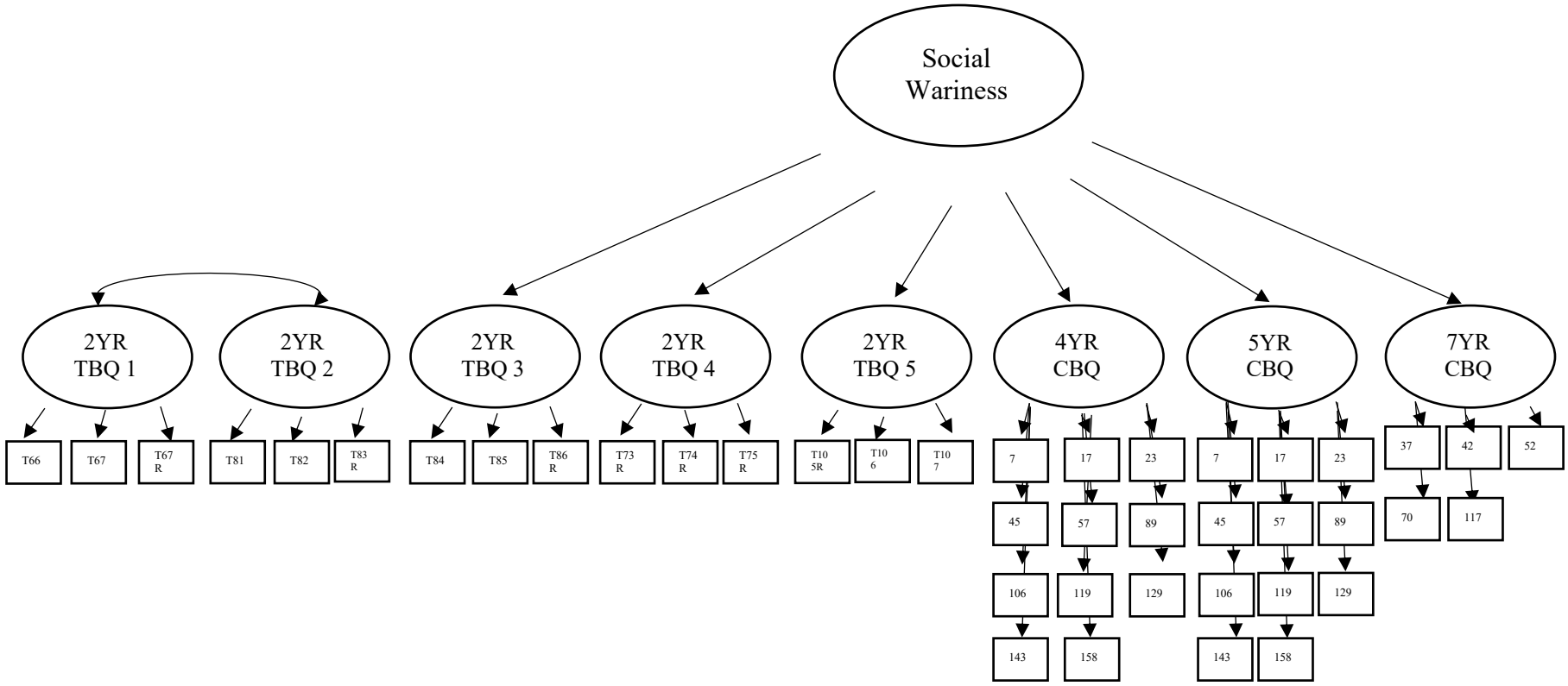
This suggests that the items were not loading onto the social fear first-order factor well. Thus, to examine the factor structure of the social fear latent variable, an exploratory factor analysis (EFA) was performed.

Exploratory Factor Analysis (EFA)

An EFA was conducted to determine whether items fell onto one or more factors that best represented the latent social wariness variable. First, it was necessary to establish the most appropriate number of factors to be extracted. Four to eight factor structures were examined. The four-factor model demonstrated poor fit [$\chi^2(1077) = 3172.335$, $p < .0001$, RMSEA = .06, CFI = .795, SRMR = .05] and was not better than the five-factor model [$\chi^2(1030) = 2730.406$, $p < .0001$, RMSEA = .06, CFI = .833, SRMR = .04; $\Delta\chi^2(47) = 441.929$, $p < .0001$]. The six-factor model was better than the five-factor model [$\chi^2(984) = 2390.896$, $p < .0001$, RMSEA = .05, CFI = .862, SRMR = .04; $\Delta\chi^2(46) = 339.420$, $p < .0001$]. The seven-factor model was better than the six-factor model [$\chi^2(939) = 2115.796$, $p < .0001$, RMSEA = .05, CFI = .885, SRMR = .03; $\Delta\chi^2(45) = 275.100$, $p < .0001$]. Lastly, the eight-factor model was better than the seven-factor model [$\chi^2(895) = 1866.373$, $p < .0001$, RMSEA = .05, CFI = .905, SRMR = .03; $\Delta\chi^2(44)$

=249.423, p< .0001]. A nine-factor model was not tested because, in order for a model to be identified, each factor must have at least three items and a nine-factor model would have resulted in at least one factor having less than three items. Thus, the eight-factor structure was utilized (see Figure 2). However, several items cross-loaded onto other factors or yielded low factor loadings and were removed.

Figure 2. Exploratory Factor Analysis Eight-Factor Model Structure



Furthermore, although the EFA resulted in the eight-item factor, some items did not theoretically load onto their respective factor as it appeared the items were capturing a different aspect of the construct. More specifically, items from the social fear subscale loaded onto factors that were consistent in the settings the behaviors occurred (e.g., doctor's office, interacting with an unfamiliar adult) whereas items from 4-year, 5-year and 7-year shyness loaded onto their own respective factors, for the exception of 1-item from 7-year shyness (item 607). Guided by theory and how the measurement was originally constructed (Goldsmith, 1996), social fear items that captured behaviors in the same setting (e.g., unfamiliar adult) were put into their own factors which is consistent with the eight-item factor generated by the EFA. Based on these theory-and data-driven approaches, single items that were not grouped within a setting (e.g., item 4R) and a three-item factor that included items that cross-loaded and had weak loadings (e.g., items 102R, 103R, 104R) were removed. Thus, a total of 4 items were removed leaving the social fear subscale with 15 items. Lastly, to address the concern regarding the low factor loading the latent social fear factor produced on social wariness factor, correlations between the social fear first-order factors were examined. Among the 15-item social fear subscale, five first-order factors were constructed from the social fear subscale: 1) doctor's office, 2) unfamiliar adult, 3) parent leaves the home, 4) unfamiliar adult, and 5) unfamiliar adult. First-order factors 1 and 3 were highly correlated with each other (r=.530, p<.0001) as these items captured a toddler's behavior in a new setting whereas factors 2, 4 and 5 captured a toddler's behavior when interacting with an unfamiliar person. Given the CBQ items also captured a child's behavior when interacting with unfamiliar people, factors 1 and 3 were not loaded onto the second-order factor, social wariness, but were instead correlated in the model. As a result, first-order factors 2, 4, 5, and 4-year, 5-year and 7-year shyness first-order factors were loaded onto the second-order factor.

Thus, a CFA was performed to test whether this six-factor model, including factors 1 and 3 correlated into the model, loaded onto the second-order factor, social wariness, or Model 3 (see Table 5).

Table 5. Confirmatory Factor Analysis for Model 3

	Factor Loading	R-square		Factor Loading	R-square		Factor Loading	R-square
2YR TBQ 1			**4YR CBQ**	**.894**	**.800**	**7YR CBQ**	**.831**	**.691**
66	.922	.849	17R	.769	.591	37	.719	.517
68	.829	.687	23R	.827	.683	42	.729	.531
67R	.619	.383	37	.643	.413	52	.884	.781
2YR TBQ 2			57R	.816	.666	70	.819	.670
81	.919	.844	89	.685	.469	117R	.859	.738
82	.953	.908	106	.842	.708			
83R	.680	.463	129R	.881	.776			
2YR TBQ 3	**.505**	**.255**	143	.754	.569			
84	.672	.451	158	.796	.634			
85	.789	.623	**5YR CBQ**	**.915**	**.838**			
86R	.791	.626	7	.577	.332			
2YR TBQ 4	**.555**	**.308**	17R	.703	.494			
73R	.888	.788	23R	.847	.718			
74R	.828	.685	37	.687	.472			
75R	.680	.462	57R	.806	.650			
2YR TBQ 5	**.594**	**.353**	74	.689	.474			
105R	.803	.645	89	.666	.443			
106	.506	.256	106	.847	.718			
107	.678	.460	119R	.648	.420			
			129R	.889	.790			
			143	.809	.654			
			158	.801	.642			

Note. TBQ1 was correlated with TBQ2 (r=.530, p=.000). SW was correlated with TBQ1 (r=.240, p=.000). SW was correlated with TBQ2 (r=.235, p=.000).

Model 3 provided good fit [$\chi^2(870) = 2114.165$, $p < .0001$, RMSEA = .06, CFI = .942, SRMR = .06; $\Delta\chi^2(260) = 1137.994$, $p < .0001$] and was a better fit to the data than Model 2. Thus, Model 3's factor scores were extracted to use in the preliminary and main analyses.

Preliminary Analyses

Descriptive statistics for all study variables are presented in Table 6 and descriptive statistics split by sex are presented in Table 7**.**

Table 6. Descriptive Statistics in Overall Sample

Variable	N	Mean	SD	Min-Max	Skew	Kurtosis
Pubertal Timing	187	0.00	0.99	-2.54-2.25	-.481	-.458
Social Wariness	420	0.00	0.31	-0.85-0.91	.012	-.062
Dep at age 17	300	3.66	4.13	0.00-26.00	2.68	6.29
Dep at age 10	288	2.98	4.66	0.00-27.00	2.53	6.20

Note. Total Study $N = 427$ (Girls $N = 224$; Boys $N = 203$). Dep = Depressive Symptoms

Table 7. Descriptive Statistics Split by Sex

	Girls						Boys					
Variable	N	Mean	SD	Min-Max	Skew	Kurtosis	N	Mean	SD	Min-Max	Skew	Kurtosis
Pubertal Timing	111	0.34	0.88	-2.53-2.25	-.602	-.198	76	- 0.51	0.94	-2.01-1.57	-.300	-.853
Social Wariness	222	0.00	0.30	-0.85-0.91	.176	.222	198	0.00	0.32	-0.85-0.73	-.135	-.325
Dep at age 17	169	3.25	4.40	0.00-26.00	2.43	6.98	131	2.64	3.73	0.00-21.00	2.19	5.61
Dep at age 10	161	3.88	5.18	0.00-27.00	2.41	5.78	127	3.38	3.90	0.00-24.00	2.50	6.82

N = 427 (Girls *N* = 224; Boys *N* = 203). Dep = Depressive Symptoms.

All study variables were assessed for normality and there were no issues with skewness or kurtosis. Higher scores for all study variables indicated earlier pubertal timing, higher social wariness, and higher depressive symptoms. However, consistent with studies using community samples, the BASC depression weighted raw score at ages 17 and 10 (M_{17}=3.66, SD_{17}=4.66, M_{10}=2.98, SD_{10}=4.13, respectively), indicated that children at age 10 and adolescents at age 17 endorsed few depressive symptoms. One relevant implication of this, is that a floor effect may have limited measurement variability. This will be further discussed below.

T-tests were calculated to examine whether there were sex differences between the primary study variables and are presented in Table 8.

Table 8. Independent Samples T-test

	Girls			Boys			
Variable	N	Mean	SD	N	Mean	SD	t test
Pubertal Timing	111	0.34	0.88	76	- 0.51	0.94	- 6.381**
Social Wariness	222	0.00	0.30	198	0.00	0.32	0.046
Dep at age 17	169	3.25	4.14	131	2.64	3.74	-1.262
Dep at age 10	161	3.88	5.18	127	3.38	3.90	0.077

**p <.01.

Independent-samples *t*-test revealed sex differences in pubertal timing, such that mothers of girls reported earlier pubertal timing (M=0.34, SD=0.88) than boys (M= - 0.52, SD=0.94), $t(187) = -6.38$, p<.001. Consistent with the literature, there were no sex differences for social wariness between boys (M= - 0.004, SD=0.32) and girls (M= -0.006, SD=0.30), $t(420) = 0.04$, p=.97. Contrary to expectations, there were no sex differences for depressive symptoms at age 17 between (M=2.64, SD=3.74) and girls (M=3.25, SD=4.41), $t(300) = -1.26$, p=.208.

Partial correlations, controlling for earlier depressive symptoms at age 10, and partial correlations by sex are reported in Tables 9 and 10, respectively.

Table 9. Partial Correlations among Study Variables Controlling for Earlier Depressive Symptoms at Age 10

Variable	*1*	*2*	*3*
1. Pubertal Timing	—	- .141	.229*
2. Social Wariness		—	.018
3. Dep at age 17			—

Note. Dep = Depressive Symptoms.

*p < .05. **p <.01.

Table 10. Partial Correlations Split by Sex

Variable	*1*	*2*	*3*
1. Pubertal Timing	—	- .178	.250*
2. Social Wariness	-.080	—	.108
3. Dep at age 17	.191	-.190	—

Note. Above diagonal = Girls. Below Diagonal = Boys. Dep = Depressive Symptoms.

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

As expected, pubertal timing at age 12 was positively correlated with depressive symptoms at age 17 (r=.229, p=.008). Contrary to expectations, there was no association between social wariness and depressive symptoms at age 17 (r= .018, p=.834). Sex differences in partial correlations were assessed using Fisher's R to Z transformation. There were no sex differences in the correlations between pubertal timing at age 12 and depressive symptoms at age 17 (r_{Girls}=.250, z_{Girls}=.255 p_{Girls}<.05; r_{Boys}=.191, z_{Boys}=.193 p_{Boys}=.199) and between social wariness and depressive symptoms at age 17 (r_{Girls}=.108, z_{Girls}=.108, p_{Girls}=.332; r_{Boys}= - .190, z_{Boys}=.192, p_{Boys}=.202).

Bivariate correlations in the full sample are reported in Table 11.

Table 11. Bivariate Correlations among Study Variables

Variable	*1*	*2*	*3*	*4*
1. Pubertal Timing	—	- .101	.245**	- .002
2. Social Wariness		—	.013	.019
3. Dep at age 17			—	.406**
4. Dep at age 10				—

Note. Dep = Depressive Symptoms.

$^{*}p < .05$. $^{**}p < .01$.

As expected, since depressive symptoms at age 10 was positively correlated with depressive symptoms at age 17 (r=.212, p<.001), depressive symptoms at age 10 was included as a control variable in the final model to account for earlier levels of symptoms prior to the impact of pubertal timing. This is also consistent with the literature, which shows children who were depressed in childhood tend to experience depressive symptoms into adolescence (Hankin, Mermelstein, Roesch, 2007). Thus, depressive symptoms at age 17 represented a change score (e.g., decreased depressive symptoms above age 10).

Main Analyses

Main Effects

The first hypothesis examined whether pubertal timing and social wariness, separately, contributed to increases in depressive symptoms at age 17, controlling for earlier depressive symptoms at age 10. A regression analysis was conducted in Mplus to test these hypotheses; results are presented in Table 12.

Table 12. Parameter Estimates for Two-way Interaction between Pubertal Timing and Social Wariness Predicting Depressive Symptoms in Overall Sample Controlling for Earlier Levels of Depressive Symptoms

Full Model (N=427)	B	β	SE_ β	*p*	R^2
Dep at Age 10	.321	.362	.081	.000**	
Pubertal Timing	.825	.199	.077	.010**	
Social Wariness	1.13	.183	.073	.012*	
PT X SW	-2.22	-.187	.124	.131	
					.244

Note. Model fit ($\chi 2$ (0) = .000, p = .00; CFI = 1.00; TLI = 1.00; RMSEA = .000). R^2 in MPLUS reflects the overall variance explained in a dependent variable by the set of independent variables entered altogether. Bootstrapped with 5000 draws.

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

Results yielded a just-identified model for both analyses: χ^2=0.000 (0), p=.000, RMSEA=0.000, CFI=1.000, TLI=1.000, SRMR=0.000, which means that the number of free parameters equals the number of known values (i.e., a model with zero or one degrees of freedom). There was a significant effect for pubertal timing such that earlier pubertal timing predicted increases in depressive symptoms by age 17, controlling for depressive symptoms at age 10 (β = 0.199, SE = 0.07, p = 0.010, 95% CI [0.046-0.329]). There was also a significant association between social wariness and depressive symptoms at age 17, controlling for earlier depressive symptoms at age 10 (β = 0.183, SE = 0.073, p = 0.012, 95% CI [0.041-0.318]). Thus, this suggests higher social wariness predicted increases in depressive symptoms by late adolescence.

Moderation

A two-way product-term interaction was conducted to examine the relation between pubertal timing and social wariness on depressive symptoms (without regard for sex), controlling for earlier levels of depressive symptoms (for results, see Table 12) with and without 95% bias-corrected bootstrapped confidence intervals to infer the significance of the parameter estimates while reducing Type 1 error and increasing the generalizability of the results. Results showed a just-identified model: χ^2=2.921 (3), p=.404, RMSEA=0.000, CFI=1.000, TLI=1.000, SRMR=0.03. There was a significant interaction (β = -0.237, SE = 0.077, p = 0.002), however, after running the model with the 95% bias-corrected bootstrapped confidence intervals (200, 500, 1000, 5000 draws), the interaction effect was no longer significant and zero was present in the 95% confidence intervals (β = -0.237, SE = 0.143, p = 0.09, 95% CI [-0.502-0.021]). This suggests that, from the many sampling distributions that were generated, there is the possibility

that the interaction effect is not different from zero. Thus, social wariness did not moderate the relationship between pubertal timing and depressive symptoms.

Multigroup Analysis: Sex differences

The third hypothesis examined the moderating role of social wariness on the relation between pubertal timing and depressive symptoms in girls and boys, controlling for earlier depressive symptoms. More specifically, it was predicted that maternal-reported earlier maturing girls at age 12 with higher levels of social wariness at ages 2, 4, 5, and 7 would report increases in depressive symptoms at age 17, controlling for earlier depressive symptoms at age 10. A multigroup analysis was conducted to examine this prediction; these results are presented in Table 13.

Table 13. Parameter Estimates for Three-way Interaction between Pubertal Timing, Social Wariness, and Sex Predicting Depressive Symptoms Controlling for Earlier Levels of Depressive Symptoms

	Male Sample					Female Sample				
Full Model (N=427)	B	β	SE_ β	*p*	R^2	B	β	SE_ β	*p*	R^2
Dep at Age 10	1.53	.225	.108	.038*		2.77	.332	.073	.000**	
Pubertal Timing	1.45	.392	.130	.003**		0.73	.164	.092	.050*	
Social Wariness	1.43	.124	.145	.200		0.84	.058	.072	.423	
PT X SW	2.00	.205	.145	.157		-3.31	-.284	.159	.096	
					.217					.304

Note. Model fit ($\chi 2$ (1) = 1.342, *p* = .246; CFI = 0.99; TLI = 0.93; RMSEA = .04; SRMR = .01). R^2 in MPLUS reflects the overall variance explained in a dependent variable by the set of independent variables entered altogether. Bootstrapped with 5000 draws.

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

Results showed that the unconstrained model was a just-identified model: χ^2=0.654 (1), *p*=.418, RMSEA=0.000, CFI=1.000, TLI=1.000, SRMR=0.012. The constrained model was also just-

identified: χ^2=0.170 (2), *p*=.912, RMSEA=0.000, CFI=1.000, TLI=1.000, SRMR=0.008. In order to test for moderation, a chi-square difference test was conducted to determine whether the unconstrained model was a better fit than the constrained model. The chi-square difference test revealed that there was no difference between the unconstrained and constrained models [$\Delta\chi^2(1)$ = 0.484, p=0.487].

As a follow-up to the traditional chi-square difference omnibus test, a theory-driven unconstrained model was tested by constraining all main effect paths to equality and freeing the interaction path. This was conducted to see if there are sex differences in the interaction path. Results showed that the theory-driven model showed a just-identified model: χ^2=0.170 (2), *p*=.918, RMSEA=0.000, CFI=1.000, TLI=1.000, SRMR=0.008. The model was run using bias-corrected bootstrapped confidence intervals (5,000 draws) to determine whether zero fell within the 95% confidence intervals. Results revealed that the interaction paths for both males and females were not significant (β = -0.297, SE = 0.345, p = 0.389, 95% CI [-1.091-0.312]; β = -0.210, SE = 0.242, p = 0.384, 95% CI [-.747-.222], respectively). Thus, contrary to the study hypothesis, sex did not moderate the link between pubertal timing and social wariness on depressive symptoms.

CHAPTER V: DISCUSSION

Adolescence is a challenging developmental period with a myriad of biological, social, and psychological changes that may overtax emerging adolescents' emotional and cognitive resources. The adolescent transition places youth at heightened risk for depressive symptoms highlighting the demand for researchers to investigate the etiological processes contributing to these symptoms to inform clinical prevention and intervention programs. Thus, guided by the contextual amplification and diathesis stress models, the current study contributes to our understanding of how an early individual childhood factor and the universally experienced stressors related to pubertal development, independently and jointly, contribute to increasing depressive symptoms during the adolescent period. The study also challenges prior interpretations of sex differences in the relation between pubertal timing and social wariness on depressive symptoms emphasizing the similar yet, nuanced, differences in girls' and boys' experiences.

As hypothesized, some socially wary children were more likely to experience increases in depressive symptoms at age 17, even after controlling for earlier depressive symptoms. This finding extends the social wariness literature as this is the first study, to the author's knowledge, to examine whether negative effects of childhood social wariness persist beyond childhood and into adolescence. Given social wariness is relatively stable across time (Rubin et al., 1999; Degnan et al., 2008; Jarcho et al., 2019), the persistent effects of childhood social wariness make it more challenging for some emergent adolescents to successfully navigate the increased frequency of social situations. Socially wary children may find it difficult to form meaningful relationships during the adolescent transition as they tend to be more socially inhibited (Zeytingoglu et al., 2022) and may not have sufficient experience in communication skills. In

addition, analyses revealed no sex differences in the social wariness mean and partial correlation between social wariness and depressive symptoms. This finding is also consistent with the literature, given boys and girls are likely to be somewhat wary when interacting with familiar and unfamiliar individuals in social situations (Rubin et al., 2009; Tang et al., 2017). Thus, socially wary girls and boys are more likely to feel lonely and sad about their limited social interactions compared to their non-socially wary peers in adolescence.

Further, EFA and CFA results generated a latent social wariness construct in which not all of the TBQ items at age 2 strongly loaded onto the construct and some were correlated into the model. One reason as to why the TBQ items at age 2 did not strongly load onto the latent social wariness construct may be partially due to a child's wary behaviors as a result of insecure or anxious/avoidant attachment. Consistent with this developmental period and according to attachment theory, a young toddler with an insecure or anxious/avoidant attachment to their parent/caregiver may experience some distress (i.e., crying, clinginess), or separation anxiety, when their parent/caregiver attempts to leave them or when they are in the presence of an unfamiliar adult (Bowlby, 1969; Cassidy & Berlin, 1994). On the other hand, children ages 4 and older may either have had a natural reduction in separation anxiety or were securely attached to their parent/caregiver such that they do not exhibit these behaviors. Thus, given young toddlers' earlier socially wary behaviors were likely related to an insecure or anxious/avoidant attachment and were not controlled for, future studies might consider removing the TBQ items or controlling for these attachment behaviors or separation anxiety at age 2.

Moreover, the social wariness latent variable primarily describes a child's distress when interacting with a familiar (e.g., parent's friend) and unfamiliar adult (e.g., stranger at the store). These interactions with adults are developmentally appropriate given toddlers and young

children typically spend time with people who are monitoring them (e.g., parents/caregivers, daycare providers, sitters; Rubin et al., 1999). When these children interact with adults, they may struggle to speak easily, feel embarrassed by the attention, anticipate negative feedback (e.g., laughing, ignoring), and wish to blend into the background. As a result, they may feel dissatisfied with the social outcome which likely contributes to feelings of sadness. Although the present study did not include many items describing whether a child hesitated to engage with familiar and unfamiliar peers, it is likely these socially wary behaviors may also apply with peers as prior research indicates these children are wary with unfamiliar and familiar adults and peers (e.g., Rubin et al., 1999; Degnan et al., 2008; Jarcho et al., 2019). Thus, creating a latent variable incorporating a spectrum of social interaction items would capture a richer picture of the child's social wariness behavior across settings (e.g., school, community events) and time, especially as they begin to spend more time with same-aged peers.

As expected, results reinforce the role of pubertal timing in adolescent depressive symptoms, as some earlier maturing children at age 12 were more likely to experience increases in depressive symptoms at age 17, controlling for earlier depressive symptoms. This finding is consistent with literature that demonstrates the negative effects of earlier pubertal timing increasing the risk for depressive symptoms in adolescence (Mendle, Turkheimer, & Emory, 2007; Mendle, 2020). Partial correlational analyses split by sex showed that, for girls only, pubertal timing was positively associated with depressive symptoms. However, a Fisher's Z test between the girls' and boys' pubertal timing and depressive symptoms association was not significantly different. This finding is also consistent with literature suggesting that earlier maturing girls and boys are more likely to experience depressive symptoms compared to later maturing children, with a slightly more robust effect observed in girls (e.g., Rudolph et al., 2014;

Hamlat et al., 2014). However, more research in this area is needed as the present sample size of boys was small (n=76). In addition, with girls entering puberty earlier in development, sample sizes typically include larger samples of identified earlier maturing girls than boys. Thus, these results suggest that *both* earlier maturing girls and boys are at risk for increases in adolescent depressive symptoms; however, this finding merits replication in a larger sample.

Nonetheless, the lack of sex differences in the link between pubertal timing and adolescent depressive symptoms contributes to the growing body of literature suggesting that earlier conceptualizations of sex differences in this link may need to be tempered. Guided by the gender intensification hypothesis (Hill & Lynch, 1983), earlier maturing girls and boys may encounter differing social responses, such as earlier maturing girls receiving comments about their physiques – leading to potential dissatisfaction with their bodies – and earlier maturing boys feeling pressured by others to engage in more "masculine"- valanced activities (e.g., football, contact sports). Moreover, the literature on social wariness also notes that there is little evidence for sex differences in social wariness that predicts depressive symptoms (Rubin et al., 1999; Mills et al., 2012). Socially wary girls may find it difficult to become part of a peer group, at a time when peer relationships are increasingly valued, and socially wary boys may receive negative comments from their male peers for not subscribing to stereotypical masculine roles (Doey et al., 2014; Tang et al., 2017). Taken together, although earlier maturing and socially wary girls and boys may be on the same pathway to experiencing depressive symptoms, their specific experiences on this pathway may be different.

Surprisingly, the interaction between pubertal timing and social wariness predicting increases in depressive symptoms, controlling for earlier depressive symptoms, was not significant. One possible explanation for this null finding could be measurement limitations. A

parent/caregiver is likely the best reporter for capturing social wariness behaviors at age 2, given that, at these ages, children typically spend most of their time with them. A parent/caregiver and teacher may serve as the best reporters of a child's social behaviors across settings during this early developmental period when a child is enrolled in a daycare center and then school. Calculating a combined social wariness score, based on parent and teacher reports, would potentially provide more insight into a child's social wariness behaviors, particularly in contexts with same-aged peers. In addition, social wariness was determined based on items drawn from self-report measures delivered to mothers only and did not incorporate the perspectives of other informants or data drawn from behavioral observation tasks (e.g., free play). Thus, latent social wariness construct is subject to mono-method and single reporter biases.

Another reason for the null findings may be the large chronological gap in measuring pubertal timing at age 12 and depressive symptoms at age 17. During the 5-year gap between measurements, postpubertal factors (e.g., peer teasing) that also may exacerbate the negative effects of being an earlier maturer and a socially wary child to increase the risk of depressive symptoms in adolescence went unmeasured. Conversely, potential protective factors (social support, close friend) that may buffer these negative effects also went unmeasured. As children with similar phenotypes and behavioral characteristics (homophily) are likely to become friends (Jänsch & Pupeter, 2017), socially wary children may befriend other socially wary/shy children, forming a relationship that may buffer social stressors. Thus, it is unclear whether other important social factors may have impacted these findings.

The third hypothesis was also not supported. There were no significant sex differences in the interaction for pubertal timing and social wariness predicting depressive symptoms, controlling for earlier depressive symptoms. Notably, the two- and three-way interaction

analyses had low power, which could have made it challenging to detect an effect. Post-hoc power analyses were calculated via G*Power (Version 3.1; Faul, Erdfelder, Lang, & Buchner, 2007) to determine whether the current sample size was sufficient to determine an effect for the three- and two-way interactions. First, a post-hoc power analysis was conducted for the three-way interaction using the relevant parameters to detect differences between the two group's (i.e., males and females) slopes for a bivariate linear regression model with a two-way interaction or the multigroup analysis. According to Giner-Sorolla (2018), in order to determine power for an interaction, it is recommended to first identify the shape of the simple slopes effect (e.g., knockout, reversal effect, attenuation) based on the researcher's prediction and then divide the sample sizes by the respective number recommended by Simohnson (2014) for each moderator variable in the analysis. Thus, for the current multigroup analysis, given an attenuation effect (i.e., simple slopes effects were in the same direction, but one was predicted to be weaker than the other) was predicted for both moderating effects (i.e., sex and social wariness), it was recommended to divide each group's sample size by 14 twice (Original: $n_{girls} = 224$, $n_{boys} = 203$, New: $n_{girls} = 2$, $n_{boys} = 1$; Giner-Sorolla, 2018, Simohnson, 2014). In addition, a standard residual σ of 0.35, standard deviation σ_x_{girls} of 0.27 and standard deviation σ_x_{boys} of 0.23 was used to estimate the power value of 0.12 at an alpha level of 0.05. Using these metrics, the three-way interaction was underpowered.

Then, a second post-hoc power analysis (i.e., linear multiple regression fixed model R^2 deviation from zero) was conducted to estimate the power value for the two-way interaction or moderation analysis. Given social wariness was a continuous moderator, a product term was created in which depressive symptoms at age 17 were regressed on the following predictors: pubertal timing, social wariness, pubertal timing x social wariness, depressive symptoms at age

10. Per Giner-Sorolla's (2018) and Simohnson's (2014) recommendations, an effect size of 0.11 (R^2) and a sample size of 31 was used (i.e., 427 divided by 14), given that social wariness was expected to produce an attenuated moderating effect. Power was estimated to be 0.23, with 4 predictors at an alpha level of 0.05. This analysis was also underpowered.

A sensitivity analysis computes an effect size utilizing the power value and sample size to determine what the smallest effect size is that the analysis is able to detect. However, given the multigroup (two moderators; sex and social wariness) and the two-way moderation (one moderator; social wariness) analyses were null, the sensitivity analysis was not computed.

Thus, taken together, we cannot confirm whether the null results are a true or false negative. Nevertheless, considering the burgeoning research suggesting earlier maturing girls and boys are at risk for depressive symptoms, it is essential to examine sex differences in the interaction between pubertal timing and a childhood contextual factor to pursue the question of whether and how these etiological processes impact both boys and girls, similarly and differently.

In summary, the findings have pushed the field forward by supporting the independent roles of pubertal timing and, for the first time, social wariness in increasing the risk for adolescent depressive symptoms. However, it is also important to note that the level of depressive symptoms observed is subclinical. In other words, while we see increases in depressive symptoms, socially wary and earlier maturing boys and girls, separately, are more likely to experience *some* depressive symptoms (e.g., loneliness) by age 17. Although the depressive symptoms level is subclinical, this predisposes late adolescents at greater risk for clinically significant symptoms as they transition into young adulthood. However, this may not be the case for everyone. Individual vulnerability factors (e.g., less effective coping strategies)

may maintain some adolescents' subclinical depressive symptoms and increase the risk for clinical depression. Conversely, protective factors (e.g., social support) may increase the likelihood that these subclinical depressive symptoms dissipate over time. Thus, although some socially wary and earlier maturing children may experience subclinical depressive symptoms, intervention programs emphasizing adaptive cognitive and emotional resources can help prevent clinically significant depressive symptoms in young adulthood.

Strengths

The current study has several strengths. To the author's knowledge, this is the first study that developed a latent social wariness variable. Most researchers have created an average composite score, typically including mother-reported scores from the TBQ and a measure that assessed for shyness (e.g., CBQ or Colorado Child Temperament Inventory), and/or using scores from a behavioral observation task (e.g., free play). In conducting a factor analysis, items from the TBQ social fear and CBQ shyness subscales strongly loaded onto social wariness and confirmed that this latent construct captured children who hesitate or experience distress when attempting to socially engage with adults. Moreover, consistent with the literature, similar time points (e.g., ages 2-7) were captured from toddlerhood to middle childhood, and factor scores loaded on the social wariness construct from each time point. This measurement design allowed for a longitudinal assessment of childhood social wariness across developmental periods.

Another study strength was that it used a prospective design with a large community sample of children and adolescents over time. The longitudinal nature of the study was able to address the temporal patterns in the relation between pubertal timing and depressive symptoms over a 5-year period. This is the first study, to the author's knowledge, that considered a contextual factor measured before pubertal development, as prior studies have only examined

factors measured at or near the pubertal onset age (e.g., age 10 or 12; Ge & Natsuaki, 2009). This is the first longitudinal study in this area of literature to span from toddlerhood to late adolescence.

Limitations

Several limitations were noted. Descriptive analysis indicated that all study variables were normally distributed. However, depressive symptoms at ages 17 and 10 were just under the minimum cutoff value for skewness. This suggests that most children at these ages reported little to no depressive symptoms, which is typical of a community sample. In addition, analyses revealed no sex differences for depressive symptoms. This finding was unexpected, given that the literature shows that depressive symptoms appear twice as much in adolescent girls than adolescent boys by the age 13-15 with the emergence of the sex differences in depression rates revealed between the ages of 12 and 14 (Khesht-Masjedi et al., 2017);-Merikangas et al., 2010). The reason for these sex differences in depressive symptoms is still unclear. However, researchers theorize that adolescent girls are more likely to exhibit negative cognitive styles (e.g., self-criticism, rumination; Khesht-Masjedi et al., 2017). Thus, the nonsignificant finding of sex differences in depressive symptoms may be due to low variability in this community sample.

Additional measurement limitations included the low sample size for pubertal timing at age 12 and consideration for factors accelerating the onset of puberty. Although FIML was utilized to estimate pubertal timing value for participants with missing data, it estimated values for more than half of the sample (240 out of 427). Even though FIML maintained the original descriptive statistic values (e.g., mean, skewness), it may not have accurately represented all 427 participants' pubertal timing sampling distribution. This may be especially true at the individual level when considering each participant's pubertal timing, social wariness, and the interaction

between both constructs in relation to depressive symptoms. In addition, obesity and adverse childhood experiences (ACE; e.g., childhood maltreatment) may have contributed to accelerated onset of puberty and, potentially, precocious puberty (Natsuaki, Samuels, & Leve, 2014). This may have increased the number of earlier maturers, especially in girls. Thus, the smaller pubertal timing sample size may limit the accuracy of the pubertal timing sample distribution and factors that may impact the onset of puberty should be considered when assessing for pubertal timing.

A statistical limitation that may undermine the childhood social wariness and adolescent depressive symptoms association was the nonsignificant partial correlation, controlling for earlier depressive symptoms, between childhood social wariness and adolescent depressive symptoms at age 17. This lack of finding in the partial correlation results may be due to the smaller sample size generated by the correlation analysis in SPSS (n=300) compared to the larger sample size generated by the regression analysis in Mplus (n=427), which utilized FIML. In addition, while the partial correlation indicated a weak relation, the regression indicated a small linear association. Thus, given the discrepancy between the partial correlation and regression results, it is even more important for studies to further examine this relation in adolescence to determine that this was not a spurious finding.

Lastly, the low power may have made it difficult to detect an effect. In response to the replication crisis, researchers are urging others to consider how power may limit the generalizability of results, regardless of whether the null hypothesis was rejected or not. Given the null findings, it is essential to recognize that the sample size was likely too small to detect an effect for the three-way and two-way interactions. One reason for this is that, although there may have been a larger group with at least one moderating effect (e.g., higher versus lower social wariness or girl versus boy), there may have been an even smaller group with one or more

moderating effects (e.g., earlier maturing girl who was socially wary). Thus, the respective sample group size for the interactions (e.g., *n* for each two-way or three-way interaction; high social wariness and girl) may not have been large enough to detect an interactive effect, especially for the three-way interaction. As a result, if there was an effect, it likely would not generalize to other samples as it could be the result of a spurious finding.

Further, with researchers encouraging increasing standards for methodological rigor, it is also important to consider and discuss a sensitivity power analysis and whether the smallest effect size of interest is meaningful. An increasing number of researchers have been conducting sensitivity analyses to infer the smallest effect size by inputting the desired or acquired power value and sample size. In this case, hypothetically, if a minimum power value of 0.8 was achieved, then the smallest effect size would be approximately 0.05. Although this small effect size may not be of societal importance, it is important on an individual level for children who are earlier maturers and socially wary in childhood because it may place them at greater risk for experiencing increases in depressive symptoms over time. Clinical interventions can be adapted to effectively address the interplay between the negative effects of being an earlier maturer and socially wary, equipping adolescents with coping strategies to reduce present or developing symptomatology. Taken together, even if the smallest effect size is small, the psychological impact on the adolescent is significant as adolescent depressive symptoms may lead to long-term consequences (e.g., clinical depression, suicidal risk), underlining this research topic as meaningful to investigate.

In summary, while the power is low, the null findings and theoretical implications are important to consider as it can inform future research by promoting a better understanding of the methodological limitations that may have minimized generalizability. By publishing findings

regardless of whether they are null, researchers can avoid the "file-drawer problem" to increase the efficient use of time and resources to make ethical contributions to the literature and prevent another replication crisis.

Future Directions

Researchers may benefit from the following methodological and theoretical considerations to inform future research. In regard to pubertal timing measurement, future studies would benefit from utilizing hormonal and neuroanatomical measurements to better understand the underlying physiological process in pubertal development. Given the dearth of research on pubertal hormones and depression, researchers may collect yearly hormonal assays pre-, mid-, and post-puberty, controlling for menstrual cycle in girls, to determine whether specific hormone(s) (e.g., testosterone; Copeland et al., 2019) are more likely to contribute to increases in depressive symptoms in adolescence. Moreover, increases in pubertal hormones (e.g., estradiol, DHEA) are associated with neuroanatomical development, such as the activation of the left anterior temporal cortex, which is involved in socioemotional processing during emotionally arousing situations, and increases in amygdala volume (Goddings et al., 2012; Goddings et al., 2014). Taken together, future longitudinal research is necessary to determine whether the effects of specific pubertal hormones, and potentially neuroanatomical development associated with socioemotional processing challenges, interacts with a childhood vulnerability to place earlier maturers at greater risk for depressive symptoms.

Moreover, future research may also benefit from examining a curvilinear association and capturing stressors associated with off-time pubertal timing at age 12 to better understand the pubertal timing and depressive symptoms relation. Given most studies within the puberty literature examined linear associations between pubertal timing and depressive symptoms, few

longitudinal studies yielded results suggesting that the negative effects of earlier pubertal timing persisted from initial levels of depressive symptoms at pubertal onset to mid-adolescence whereas the negative effects of later pubertal timing contributed to initial levels of depressive symptoms, but dissipated over time (e.g., Conley & Rudolph, 2009). However, more longitudinal research extending from childhood to late adolescence is needed to determine whether this is, indeed, the case and whether these curvilinear associations differ for girls and boys. In addition, risk factors associated with being an earlier maturer, like lower body satisfaction or fear of social rejection, should also be considered as potential moderators or mediators within the pubertal timing and depressive symptoms relation, given earlier maturers are simultaneously experiencing these psychosocial stressors in reaction to their social environment. Taken together, future studies examining curvilinear associations in the pubertal timing and depressive symptoms relation as well as considering puberty-related stressors at age 12 would provide more insight into the experiences of earlier versus later maturers and how these pubertal stressors impact their developmental trajectory.

Beyond adolescent depression, pubertal timing is also a risk factor for externalizing problems and a transdiagnostic risk for the common psychopathology factor or "*p*" factor, including externalizing and internalizing factors. Given earlier maturing children are perceived to be older by their social environment, they may affiliate with older peers who engage in externalizing behaviors, developing these behaviors as a method of coping with social pressures to act "adult-like" (Dhillon & Kanwar, 2018; Dimler & Natsuaki, 2015). Moreover, recent research has examined earlier pubertal timing to be a transdiagnostic risk factor for psychopathology (e.g., social anxiety, ODD, depression), with effects observed in earlier maturing boys and girls (Hamlat et al., 2019; Mendle et al., 2020). As earlier maturing children

are at greater risk for experiencing internalizing and externalizing symptoms, independently, and the *p* factor, future research may benefit from investigating whether an early vulnerability before the age of puberty may exacerbate the negative effects of earlier pubertal timing to increase the risk for one or more of these three outcomes emphasizing the multifinality in the development of adolescent psychopathology.

Future research may also examine other childhood social factors that may exacerbate or buffer the negative effects of pubertal timing. Given that earlier maturers' experiences occur within social contexts (e.g., peer rejection, body shaming) and peer relationships become increasingly salient, researchers should consider how a child's social interactions and/or social characteristics may impact the quality of their peer relationships. Thus, both positive (e.g., social support, peer acceptance) and negative (e.g., cyberbullying, peer rejection) social characteristics should be examined to inform clinical interventions for earlier maturing youth.

Furthermore, as toddlers and young children heavily rely on and depend on their parents, parenting factors may also serve as an earlier vulnerability that may exacerbate the effects of earlier pubertal timing to increase risk for adolescent depressive symptoms. Parents who are overcontrolling and provide less structure at home may send messages to their child that participating in seemingly stressful social situations is a threat which may prevent them from building their social confidence and learning how to successfully navigate these situations (Natsuaki et al., 2013). Poor parent-child relationships (i.e., insecure attachment) may also make it challenging for an earlier maturer to trust parents or adult figures to provide support during times of need and resort to less effective coping strategies (Kanwar, 2020). Taken together, parenting factors may amplify the stressors inherent in earlier pubertal development to increase the risk of experiencing depressive symptoms in adolescence.

The current sample was mostly White and, thus, did not generalize to other minority group populations. Although approximately 30% of children in this sample identified as Black, cultural comparisons between White and Black youth were not analyzed due to the unequal sample size, which could have increased the likelihood of a Type I error. Future studies must consider the consequences of earlier pubertal timing in racial/ethnic and gender minorities, especially in considering the intersectionality of these identities. Racial/ethnic socialization may play a key role in diverse youths' perceptions of pubertal development and how their pubertal timing compares to other children from similar and dissimilar racial/ethnic backgrounds. This phenomenon may be particularly pertinent for Black and Latine youth who mature earlier than their non-Black/Latine same-aged peers (Ramnitz & Lodish, 2013; Seaton & Carter, 2019). An adolescent's pubertal socialization experiences may be challenging if a youth from a minority group attends a school predominantly comprised of majority group youth. For example, Black children who attend predominantly White schools may have few or no Black children in their lives to use as developmental comparisons. The absence of a social comparison may increase the saliency of a child's experienced physical differences and, in turn, may increase the risk of depressive symptoms as they perceive themselves as not fitting in their social environment (Carter, Blazek, & Kwesele, 2020).

Similarly worth exploring is how sexual and gender minority youth may feel increased rates of anxiety and sadness in response to their bodies developing sex-specific sexual characteristics (Olson-Kennedy et al., 2016). These emerging adolescents may experience not only body dissatisfaction and confusion with their maturing physiques, but also uncertainty about how their developing cultural, gender, and sexual identities fit into their values, traditions, and beliefs (Mendle & Koch, 2019). Sexual minority youth (LGBTQ+) may feel emotionally and

cognitively unprepared for feelings of sexual arousal and romantic relationships with same-sex peers, given the stigma associated with same-sex orientation (Deardoff et al., 2019). Gender minority youth (e.g., transgender) may experience internal conflict with their gender identity being inconsistent with their preferred body shape as they also cope with societal pressures to conform to binary gender roles (Deardoff et al., 2019; Mendle & Koch, 2019). It is crucial that future studies consider how the experience of being an earlier maturer may impact diverse youths' psychological well-being across different social and cultural contexts.

Research and Clinical Implications

To bridge the scientist-practitioner gap, research findings examining the contextual amplification hypothesis and diathesis stress model should be applied to inform school-based interventions as children undergo puberty. At the macro level, health education courses in middle and high school could include discussions on the biology of puberty and the potential psychosocial consequences of earlier pubertal timing. At the micro level, interventions at middle schools (when pubertal onset typically begins) can target earlier maturing girls and boys who are identified as feeling physically different, having lower body image satisfaction, and/or having lower self-esteem. This research can inform clinical interventions, with clinicians implementing cognitive-behavioral skills to equip children and adolescents with cognitive and emotional resources to improve self-perception and reduce subsequent internalizing symptoms. Given that the consequences of social wariness and earlier pubertal timing include experiencing stressful peer contexts, teaching interpersonal effectiveness and social skills may help individuals to navigate negative peer interactions and promote positive ones. This intervention can also help youth to create and maintain close friendships, which may serve as a protective factor (Jänsch & Pupeter, 2017). Lastly, parents may learn to provide emotional and interpersonal support to their

child by attending parent workshops informed by this research that provide psychoeducation and parent training skills.

Conclusion

This study integrated the social wariness and pubertal timing literatures to investigate how these variables operated, independently and jointly, in placing children at risk for increases in depressive symptoms in late adolescence. The present findings suggest that socially wary children are more likely to experience subclinical depressive symptoms in adolescence when interacting with unfamiliar and familiar adults. These findings extend the literature indicating the negative effects of childhood social wariness likely extends beyond childhood. The current study also adds further evidence supporting the idea that earlier maturing girls and boys are at risk for experiencing increases in subclinical depressive symptoms in adolescence. Researchers are encouraged to discuss how prescribing to gender roles and stereotypes may influence earlier maturing girls' and boys' nuanced experience of the pubertal process. Lastly, substantial research has supported the contextual amplification hypothesis; however, childhood social wariness did not exacerbate the negative effects of pubertal timing, and the interactive path did not differ between boys and girls. Rather, other social characteristics (e.g., social anxiety) or peer stressors (e.g., low peer support) may exacerbate earlier pubertal timing effects creating a more stressful environment during the adolescent transition. The sample size and resultant low power may have also made it challenging to detect an effect. Thus, more work is needed to examine how the social context increases the vulnerability for adolescent depressive symptoms among earlier maturing children and whether these contexts operate differently for boys versus girls.

REFERENCES

Achenbach, T. M., Dumenci, L., & Rescorla, L. A. (2001). *Ratings of relations between DSM-IV diagnostic categories and items of the CBCL/6-18, TRF, and YSR*. Burlington, VT: University of Vermont, 1-9.

Angold, A., Costello, E. J., Erkanli, A., & Worthman, C. M. (1999). Pubertal changes in hormone levels and depression in girls. *Psychological Medicine*, 29(5), 1043–1053. https://doi-org.libproxy.uncg.edu/10.1017/S0033291799008946

Biro, F. M., Greenspan, L. C., & Galvez, M. P. (2012). Puberty in girls of the 21st century. *Journal of Pediatric and Adolescent Gynecology*, 25(5), 289-294.

Black, S. R., & Klein, D. N. (2012). Early menarcheal age and risk for later depressive symptomatology: The role of childhood depressive symptoms. *Journal of Youth and Adolescence*, 41(9), 1142–1150. https://doi-org.libproxy.uncg.edu/10.1007/s10964-012-9758-7

Bowlby, J. (1969). Attachment and loss: Vol. 1. *Attachment*. London: Hogarth Press.

Brooks-Gunn, J. (1991). How stressful is the transition to adolescence for girls. *Adolescent stress: Causes and consequences*, 131-149.

Brooks-Gunn, J., & Petersen, A. C. (1991). Studying the emergence of depression and depressive symptoms during adolescence. *Journal of Youth and Adolescence*, 20(2), 115-119.

Brooker, R. J., Kiel, E. J., & Buss, K. A. (2016). Early social fear predicts kindergarteners' socially anxious behaviors: Direct associations, moderation by inhibitory control, and differences from nonsocial fear. *Emotion*, 16(7), 997–1010. https://doi-org.libproxy.uncg.edu/10.1037/emo0000135

Carter, R., Silverman, W. K., & Jaccard, J. (2013). Race and perceived pubertal transition effects on girls' depressive symptoms and delinquent behaviors. *Journal of Youth and Adolescence*, 42(8), 1155-1168. doi:10.1007/s10964-012-9885-1

Carter, R., Seaton, E. K., & Blazek, J. L. (2020). Comparing Associations Between Puberty, Ethnic–Racial Identity, Self-Concept, and Depressive Symptoms Among African American and Caribbean Black Boys. *Child Development*, 91(6), 2019-2041.

Cassidy, J., & Berlin, L. J. (1994). The insecure/ambivalent pattern of attachment: Theory and research. *Child Development*, 65(4), 971–981. https://doi.org/10.2307/1131298

Caspi, A., & Moffit, T. (1991, April). Puberty and deviance in girls. In Symposium paper read at the Biennial Meeting of the Society for Research in Child Development, Seattle.

Chumlea, W. C., Schubert, C. M., Roche, A. F., Kulin, H. E., Lee, P. A., Himes, J. H., & Sun, S. S. (2003). Age at menarche and racial comparisons in U.S. girls. *Pediatrics*, 111, 110 – 113.

Clausen, J. A. (1975). *The Social Meaning of Differential Physical and Sexual Maturation*.

Coplan, R. J., Rubin, K. H., Fox, N. A., Calkins, S. D., & Stewart, S. L. (1994). Being alone, playing alone, and acting alone: Distinguishing among reticence and passive and active solitude in young children. *Child Development*, 65(1), 129-137.

Coplan, R. J., & Rubin, K. H. (2010). Social withdrawal and shyness in childhood: History, theories, definitions, and assessments. In K. H. Rubin & R. J. Coplan (Eds.), *The Development of Shyness and Social Withdrawal*. (pp. 3–20). The Guilford Press.

Coplan, R. J., Ooi, L. L., Xiao, B., & Rose, K. L. (2018). Assessment and implications of social withdrawal in early childhood: A first look at social avoidance. *Social Development*, 27(1), 125–139. https://doi-org.libproxy.uncg.edu/10.1111/sode.12258

Costello, E. J., Mustillo, S., Erkanli, A., Keeler, G., & Angold, A. (2003). Prevalence and development of psychiatric disorders in childhood and adolescence. *Archives of General Psychiatry*, 60(8), 837-844.

Cuijpers, P., & Smit, F. (2004). Subthreshold depression as a risk indicator for major depressive disorder: a systematic review of prospective studies. *Acta Psychiatrica Scandinavica*, 109(5), 325-331.

Cummings, C. M., Caporino, N. E., & Kendall, P. C. (2014). Comorbidity of anxiety and depression in children and adolescents: 20 years after. *Psychological Bulletin*, 140(3), 816.

Degnan, K. A., Henderson, H. A., Fox, N. A., & Rubin, K. H. (2008). Predicting social wariness in middle childhood: The moderating roles of childcare history, maternal personality and maternal behavior. *Social Development*, 17(3), 471–487. https://doi-org.libproxy.uncg.edu/10.1111/j.1467-9507.2007.00437.x

Doey, L., Coplan, R. J., & Kingsbury, M. (2014). Bashful boys and coy girls: A review of gender differences in childhood shyness. *Sex Roles: A Journal of Research*, 70(7–8), 255–266. https://doi-org.libproxy.uncg.edu/10.1007/s11199-013-0317-9

Dorn, L. D., Susman, E. J., & Ponirakis, A. (2003). Pubertal timing and adolescent adjustment and behavior: Conclusions vary by rater. *Journal of Youth and Adolescence,* 32, 157–167.

Dorn, L. D., Sontag-Padilla, L. M., Pabst, S., Tissot, A., & Susman, E. J. (2013). Longitudinal reliability of self-reported age at menarche in adolescent girls: Variability across time and setting. *Developmental Psychology*, 49(6), 1187-1193. doi:10.1037/a0029424

Galvao, T. F., Silva, M. T., Zimmermann, I. R., Souza, K. M., Martins, S. S., & Pereira, M. G. (2014). Pubertal timing in girls and depression: A systematic review. *Journal of Affective Disorders*, 155, 13–19. https://doi-org.libproxy.uncg.edu/10.1016/j.jad.2013.10.034

Ge, X., Conger, R. D., & Elder Jr, G. H. (1996). Coming of age too early: Pubertal influences on girls' vulnerability to psychological distress. *Child Development*, 67(6), 3386-3400.

Ge, X., Elder, C. J., Regnerus, M., & Cox, C. (2001). Pubertal transitions, perceptions of being overweight, and adolescents' psychological maladjustment: Gender and ethnic differences. *Social Psychology Quarterly*, 64(4), 363-375. doi:10.2307/3090160

Ge, X., & Natsuaki, M. N. (2009). In search of explanations for early pubertal timing effects on developmental psychopathology. *Current Directions in Psychological Science*, 18(6), 327-331. doi:10.1111/j.1467-8721.2009.01661.x

Goldsmith, H. H. (1996). Studying temperament via construction of the Toddler Behavior Assessment Questionnaire. *Child Development*, 67(1), 218-235.

Graber, J. A., Lewinsohn, P. M., Seeley, J. R., & Brooks-Gunn, J. (1997). Is psychopathology associated with the timing of pubertal development? *Journal of the American Academy of Child & Adolescent Psychiatry*, 36(12), 1768–1776. https://doi-org.libproxy.uncg.edu/10.1097/00004583-199712000-00026

Graber, J. A., Seeley, J. R., Brooks-Gunn, J., & Lewinsohn, P. M. (2004). Is Pubertal Timing Associated with Psychopathology in Young Adulthood? *Journal of the American Academy of Child & Adolescent Psychiatry*, 43(6), 718-726. doi:10.1097/01.chi.0000120022.14101.11

Graber, J. A., Brooks-Gunn, J., & Warren, M. P. (2006). Pubertal Effects on Adjustment in Girls: Moving from Demonstrating Effects to Identifying Pathways. *Journal of Youth and*

Adolescence, 35(3), 413–423. https://doi-org.libproxy.uncg.edu/10.1007/s10964-006-9049-2, 43(5), 824–833. https://doi-org.libproxy.uncg.edu/10.1007/s10964-013-0015-5

Hamilton, J. L., Potter, C. M., Olino, T. M., Abramson, L. Y., Heimberg, R. G., & Alloy, L. B. (2016). The temporal sequence of social anxiety and depressive symptoms following interpersonal stressors during adolescence. *Journal of Abnormal Child Psychology*, 44(3), 495–509. https://doi-org.libproxy.uncg.edu/10.1007/s10802-015-0049-0

Hankin, B. L., Mermelstein, R., & Roesch, L. (2007). Sex Differences in Adolescent Depression: Stress Exposure and Reactivity Models. Child Development, 78(1), 279-295. doi:10.1111/j.1467-8624.2007.00997.x

Hayward, C. (2003). *Gender Differences at Puberty*. New York, NY, US: Cambridge University Press. doi:10.1017/CBO9780511489716

Hill, J. P., & Lynch, M. E. (1983). The intensification of gender-related role expectations during early adolescence. In *Girls at Puberty* (pp. 201-228). Springer, Boston, MA.

Jänsch, A., & Pupeter, M. (2017). Friendships Among Peers. In Well-being, *Poverty and Justice from a Child's Perspective* (pp. 135-147). Springer, Cham.

Jarcho, J. M., Grossman, H. Y., Guyer, A. E., Quarmley, M., Smith, A. R., Fox, N. A., Leibenluft, E., Pine, D. S., & Nelson, E. E. (2019). Connecting childhood wariness to adolescent social anxiety through the brain and peer experiences. *Journal of Abnormal Child Psychology*, 47(7), 1153–1164. https://doi-org.libproxy.uncg.edu/10.1007/s10802-019-00543-4

Kagan, J., Reznick, J. S., Snidman, N., Gibbons, J., & Johnson, M. O. (1988). Childhood derivatives of inhibition and lack of inhibition to the unfamiliar. *Child Development*, 1580-1589.

Kagan, J., Snidman, N., Kahn, V., Towsley, S., Steinberg, L., & Fox, N. A. (2007). The preservation of two infant temperaments into adolescence. *Monographs of the Society for Research in Child Development*, i-95.

Kaltiala-Heino, R., Marttunen, M., Rantanen, P., & Rimpelä, M. (2003). Early puberty is associated with mental health problems in middle adolescence. *Social Science & Medicine*, 57(6), 1055–1064. https://doi-org.libproxy.uncg.edu/10.1016/S0277-9536(02)00480-X

Khesht-Masjedi, M. F., Shokrgozar, S., Abdollahi, E., Golshahi, M., & Sharif-Ghaziani, Z. (2017). Comparing depressive symptoms in teenage boys and girls. *Journal of family medicine and primary care*, *6*(4), 775–779. https://doi.org/10.4103/jfmpc.jfmpc_129_17

Klein, D. N., Shankman, S. A., Lewinsohn, P. M., & Seeley, J. R. (2009). Subthreshold depressive disorder in adolescents: Predictors of escalation to full-syndrome depressive disorders. *Journal of the American Academy of Child & Adolescent Psychiatry*, 48(7), 703–710. https://doi-org.libproxy.uncg.edu/10.1097/CHI.0b013e3181a56606

Koch, M. K., Mendle, J., & Beam, C. (2020). Psychological distress amid change: Role disruption in girls during the adolescent transition. *Journal of Abnormal Child Psychology*. https://doi-org.libproxy.uncg.edu/10.1007/s10802-020-00667-y

Lewis, G., Ioannidis, K., van Harmelen, A. L., Neufeld, S., Stochl, J., Lewis, G., Jones, P. B., & Goodyer, I. (2018). The association between pubertal status and depressive symptoms and diagnoses in adolescent females: A population-based cohort study. *PloS One*, 13(6), e0198804. https://doi.org/10.1371/journal.pone.0198804

Lewinsohn, P. M., Gotlib, I. H., Lewinsohn, M., Seeley, J. R., & Allen, N. B. (1998). Gender differences in anxiety disorders and anxiety symptoms in adolescents. *Journal of Abnormal Psychology*, 107(1), 109.

Marshall, W. A., & Tanner, J. M. (1969). Variations in pattern of pubertal changes in girls. *Archives of Disease in Childhood*, 44(235), 291.

Masten, A. S., Roisman, G. I., Long, J. D., Burt, K. B., Obradović, J., Riley, J. R., ... & Tellegen, A. (2005). Developmental cascades: linking academic achievement and externalizing and internalizing symptoms over 20 years. *Developmental psychology*, 41(5), 733.

McGuire, T. C., McCormick, K. C., Koch, M. K., & Mendle, J. (2019). Pubertal maturation and trajectories of depression during early adolescence. *Frontiers in Psychology*, 10. https://doi-org.libproxy.uncg.edu/10.3389/fpsyg.2019.01362

Mendle, J., Turkheimer, E., & Emery, R. E. (2007). Detrimental psychological outcomes associated with early pubertal timing in adolescent girls. *Developmental Review*, 27(2), 151-171. doi:10.1016/j.dr.2006.11.001.

Mendle, J., Harden, K. P., Brooks-Gunn, J., & Graber, J. A. (2010). Development's tortoise and hare: Pubertal timing, pubertal tempo, and depressive symptoms in boys and girls. *Developmental Psychology*, 46, 1341–1353.

Mendle, J., & Ferrero, J. (2012). Detrimental psychological outcomes associated with pubertal timing in adolescent boys. *Developmental Review*, 32(1), 49-66. doi:10.1016/j.dr.2011.11.001

Mendle, J., & Koch, M. K. (2019). The psychology of puberty: What aren't we studying that we should? *Child Development Perspectives*. https://doi-org.libproxy.uncg.edu/10.1111/cdep.12333

Merikangas, K. R., He, J. -p, Burstein, M., Swanson, S. A., Avenevoli, S., Cui, L., ... Swendsen, J. (2010). Lifetime prevalence of mental disorders in US adolescents: Results from the national comorbidity survey replication–adolescent supplement (NCS-A). *Journal of the American Academy of Child and Adolescent Psychiatry*, 49(10), 980–989.

Mills, R. S. L., Hastings, P. D., Helm, J., Serbin, L. A., Etezadi, J., Stack, D. M., Schwartzman, A. E., & Li, H. H. (2012). Temperamental, parental, and contextual contributors to early-emerging internalizing problems: A new integrative analysis approach. *Social Development*, 21(2), 229–253. https://doi-org.libproxy.uncg.edu/10.1111/j.1467-9507.2011.00629.x

Miloseva, L., Vukosavljevic-Gvozden, T., Milosev, V., & Davis III, T. (2018). Can we predict and prevent subclinical depression in adolescents?. *The Journal of Nervous and Mental Disease*, 206(2), 102-107.

Muthén, L. K., & Muthen, B. (2017). *Mplus User's Guide: Statistical Analysis with Latent Variables, User's Guide*. Muthén & Muthén.

Natsuaki, M. N., Leve, L. D., Harold, G. T., Neiderhiser, J. M., Shaw, D. S., Ganiban, J., Scaramella, L. V., & Reiss, D. (2013). Transactions between child social wariness and observed structured parenting: Evidence from a prospective adoption study. *Child Development*, 84(5), 1750–1765. https://doi-org.libproxy.uncg.edu/10.1111/cdev.12070

Natsuaki, M. N., Samuels, D., & Leve, L. D. (2015). *Puberty, Identity, and Context: A Biopsychosocial Perspective on Internalizing Psychopathology in Early Adolescent Girls*. In K. C. McLean, M. Syed, K. C.

Negriff, S., & Susman, E. J. (2011). Pubertal timing, depression, and externalizing problems: A framework, review, and examination of gender differences. *Journal of Research on*

Adolescence, 21(3), 717–746. https://doi-org.libproxy.uncg.edu/10.1111/j.1532-7795.2010.00708.x

Parent, A. S., Teilmann, G., Juul, A., Skakkebæk, N. E., Toppari, J., & Bourguignon, J. P. (2003). The timing of normal puberty and the age limits of sexual precocity: Variations around the world, secular trends, and changes after migration. *Endocrine Reviews*, 24, 668–693.

Petersen, A. C., & Taylor, B. (1980). The biological approach to adolescence. In J. Adelson (Ed.), *Handbook of adolescent psychology* (pp. 117-155). New York, NY: Wiley.

Rapkin, A. J., Tsao, J. C., Turk, N., Anderson, M., & Zeltzer, L. K. (2006). Relationships among self-rated tanner staging, hormones, and psychosocial factors in healthy female adolescents. *Journal of pediatric and adolescent gynecology*, 19(3), 181–187. https://doi.org/10.1016/j.jpag.2006.02.004

Rierdan, J., & Koff, E. (1991). Depressive symptomatology among very early maturing girls. *Journal of Youth and Adolescence*, 20(4), 415–425. https://doi-org.libproxy.uncg.edu/10.1007/BF01537183

Rothbart, M. K., Ahadi, S. A., Hershey, K. L., & Fisher, P. (2001). Investigations of temperament at three to seven years: The Children's Behavior Questionnaire. *Child development*, 72(5), 1394-1408.

Rubin, K. H., Hymel, S., & Mills, R. S. (1989). Sociability and social withdrawal in childhood: Stability and outcomes. *Journal of personality*, 57(2), 237-255.

Rubin, K. H., Nelson, L. J., Hastings, P., & Asendorpf, J. (1999). The transaction between parents' perceptions of their children's shyness and their parenting styles. International

Journal of Behavioral Development, 23(4), 937–958. https://doi-org.libproxy.uncg.edu/10.1080/016502599383612

Rubin, K. H., & Burgess, K. B. (2001). Social withdrawal and anxiety. *The developmental psychopathology of anxiety*, 407-434.

Rubin, K. H., Coplan, R. J., & Bowker, J. C. (2009). Social withdrawal in childhood. *Annual Review of Psychology*, 60, 141–171. https://doi-org.libproxy.uncg.edu/10.1146/annurev.psych.60.110707.163642

Rubin, K. H., & Coplan, R. J. (2010). *The development of shyness and social withdrawal* (K. H. Rubin & R. J. Coplan (Eds.)). The Guilford Press.

Rubin, K. H., Root, A. K., & Bowker, J. (2010). Parents, peers, and social withdrawal in childhood: A relationship perspective. In H. Gazelle & K. H. Rubin (Eds.), *Social anxiety in childhood: Bridging developmental and clinical perspectives.* (Vol. 2010, Issue 127, pp. 79–94). Jossey-Bass.

Rubin, K. H., Bowker, J. C., Barstead, M. G., & Coplan, R. J. (2018). Avoiding and withdrawing from the peer group. In W. M. Bukowski, B. Laursen, & K. H. Rubin (Eds.), *Handbook of peer interactions, relationships, and groups., 2nd ed*. (pp. 322–346). The Guilford Press.

Rudolph, K. D., & Troop-Gordon, W. (2010). Personal-accentuation and contextual-amplification models of pubertal timing: Predicting youth depression. *Development and Psychopathology*, 22(2), 433-451.

Rudolph, K. D. (2014). Puberty as a developmental context of risk for psychopathology. In Handbook of developmental psychopathology (pp. 331-354). Springer, Boston, MA.

Rudolph, K. D., Troop-Gordon, W., Lambert, S. F., & Natsuaki, M. N. (2014). Long-term consequences of pubertal timing for youth depression: Identifying personal and contextual pathways of risk. *Development and psychopathology*, 26(4pt2), 1423-1444.

Seaton, E. K., & Carter, R. (2018). Pubertal timing, racial identity, neighborhood, and school context among Black adolescent females. *Cultural diversity and ethnic minority psychology*, 24(1), 40.

Sullivan, H. S. (1953). The interpersonal theory of psychiatry. New York: Norton

Tang, A., Van Lieshout, R. J., Lahat, A., Duku, E., Boyle, M. H., Saigal, S., & Schmidt, L. A. (2017). Shyness trajectories across the first four decades predict mental health outcomes. *Journal of Abnormal Child Psychology*, 45(8), 1621–1633. https://doi-org.libproxy.uncg.edu/10.1007/s10802-017-0265-x

Ullsperger, J. M., & Nikolas, M. A. (2017). A meta-analytic review of the association between pubertal timing and psychopathology in adolescence: Are there sex differences in risk?. *Psychological Bulletin*, 143(9), 903-938. doi:10.1037/bul0000106

Winer, J. P., Parent, J., Forehand, R., & Breslend, N. L. (2016). Interactive effects of psychosocial stress and early pubertal timing on youth depression and anxiety: Contextual amplification in family and peer environments. *Journal of Child and Family Studies*, 25(5), 1375–1384.

Zeytinoglu, S., Neuman, K. J., Degnan, K. A., Almas, A. N., Henderson, H., Chronis-Tuscano, A., ... & Fox, N. A. (2022). *Pathways from maternal shyness to adolescent social anxiety. Journal of Child Psychology and Psychiatry*, 63(3), 342-349.

APPENDIX A: PUBERTAL DEVELOPMENT SCALE

Girls' Pubertal Development Scale (Parent Report)

For each of the following questions, please circle the answer that best describes your daughter's physical development right now. If you feel you do not know enough to answer a particular question, then just choose "Don't Know" and go on to the next one.

1 No	2 Yes, barely	3 Yes, definitely	4 Developmentally Completed	5 Don't Know

1. Would you say that your daughter's growth spurt (in **height**) has started yet? (A growth spurt is defined as growth in height that is faster than usual.)	**1 2 3 4 5**
2. Would you say that growth of her underarm and pubic hair has started yet?	**1 2 3 4 5**
3. Would you say that her breasts have started to grow:	**1 2 3 4 5**
4. Have you noticed any changes in her skin, especially pimples?	**1 2 3 4 5**

5. Has she had her first menstrual period? Yes No

   If yes, how old was she when she had her first period? ____ years, ____ months

6. Compared with other girls her age, would you say your daughter's physical development is:

   1 much earlier than the other girls.
   2 somewhat earlier than the other girls.
   3 about the same as the other girls.
   4 somewhat later than the other girls.
   5 much later than the other girls.

Boys' Pubertal Development Scale (Parent Report)

For each of the following questions, please circle the answer that best describes your son's physical development right now. If you feel you do not know enough to answer a particular question, then just choose "Don't Know" and go on to the next one.

1 No	2 Yes, barely	3 Yes, definitely	4 Developmentally Completed	5 Don't Know

1. Would you say that your son's growth spurt (in **height**) has started yet? (A growth spurt is defined as growth in height that is faster than usual.)	**1 2 3 4 5**
2. Would you say that growth of his underarm and pubic hair has started yet?	**1 2 3 4 5**
3. Have you noticed any changes in his skin, especially pimples?	**1 2 3 4 5**
4. Have you noticed a deepening of his voice?	**1 2 3 4 5**
5. Has he started to grow hair on his face?	**1 2 3 4 5**

6. Compared with other boys his age, would you say your son's physical development is:

1 much earlier than the other boys.
2 somewhat earlier than the other boys.
3 about the same as the other boys.
4 somewhat later than the other boys.
5 much later than the other boys.

.

APPENDIX B: TODDLER BEHAVIOR ASSESSMENT QUESTIONNAIRE

Toddler Behavior Assessment Questionnaire (Parent Report)

As you read each description of the child's behavior below, please indicate how often the child did this during the last month by circling one of the numbers in the left column. These number indicate how often you observed the behavior described during the last month.

1 Never	2 Very Rarely	3 Less than half the time	4 About half the time	5 More than half the time	6 Almost always	7 Always	NA Does not apply

4. When s/he saw other children while in the park or playground, how often did your child approach and immediately join in play?	**1 2 3 4 5 6 7 NA**
66. When at the doctor's office, how often did your child cling to the parent?	**1 2 3 4 5 6 7 NA**
67. When at the doctor's office, seem unconcerned and comfortable?	**1 2 3 4 5 6 7 NA**
68. When at the doctor's office, cry or struggle when the doctor tried to touch her/him?	**1 2 3 4 5 6 7 NA**
73. When first meeting a stranger coming to visit in the home, how often did your child allow her/himself to be picked up without protest?	**1 2 3 4 5 6 7 NA**
74. When first meeting a stranger coming to visit in the home, how often did your child abandon the parent to go to the stranger?	**1 2 3 4 5 6 7 NA**
75. When first meeting a stranger coming to visit in the home, how often did your child "warm up" to the stranger within 10 minutes?	**1 2 3 4 5 6 7 NA**
81. When the child knew the parents were about to leave her/him at home, how often did your child cry?	**1 2 3 4 5 6 7 NA**
82. When the child knew the parents were about to leave her/him at home, how often did your child clint to the parent?	**1 2 3 4 5 6 7 NA**
83. When the child knew the parents were about to leave her/him at home, how often did your child show no evidence of distress?	**1 2 3 4 5 6 7 NA**

84. When one of the parents' friends who does not have daily contact with your child visited the home, how often did your child check with parent for assurance?	**1 2 3 4 5 6 7 NA**
85. When one of the parents' friends who does not have daily contact with your child visited the home, how often did your child talk much less than usual?	**1 2 3 4 5 6 7 NA**
86. When one of the parents' friends who does not have daily contact with your child visited the home, how often did your child enthusiastically greet them?	**1 2 3 4 5 6 7 NA**
102. When first visiting a babysitting co-op, daycare center, or church nursery, how often did your child cry when not being held by the parent and resist being put down?	**1 2 3 4 5 6 7 NA**
103. When first visiting a babysitting co-op, daycare center, or church nursery, how often did your child feel at ease within 10 minutes?	**1 2 3 4 5 6 7 NA**
104. When first visiting a babysitting co-op, daycare center, or church nursery, how often did your child immediately begin to explore?	**1 2 3 4 5 6 7 NA**
105. When your child was being approached by an unfamiliar adult while shopping or out walking, how often did your child babble or talk?	**1 2 3 4 5 6 7 NA**
106. When your child was being approached by an unfamiliar adult while shopping or out walking, how often did your child show distress or cry?	**1 2 3 4 5 6 7 NA**
107. When your child was being approached by an unfamiliar adult while shopping or out walking, how often did your child avoid possible danger by looking to parent for assurance?	**1 2 3 4 5 6 7 NA**

Footnote: Items from the social fear subscale are presented only.

APPENDIX C: BEHAVIOR ASSESSMENT SYSTEM FOR CHILDREN, SECOND EDITION

Behavior Assessment System for Children, Second Edition (Self-Report Child)

This booklet contains sentences that tell how some boys and girls think or feel or act. Read each sentence carefully. For the first group of sentences, you will have two answer choices: T or F. For the second group of sentences, you will have four answer choices: N, S, O, and A.

T True	F False

4. Nothing ever goes right for me.	**T F**
14. I used to be happier.	**T F**
17. Nothing goes my way.	**T F**
19. I have too many problems.	**T F**
29. Nobody ever listens to me.	**T F**
33. Nothing is fun anymore.	**T F**
43. I don't seem to do anything right.	**T F**
46. Nothing about me is right.	**T F**
48. I just don't care anymore.	**T F**

N Never	S Sometimes	O Often	A Almost aways

61. I feel depressed.	**N S O A**
71. No one understands me.	**N S O A**
74. I feel sad.	**N S O A**
76. I feel like my life is getting worse and worse.	**N S O A**

Footnote: Items from the depression subscale are presented only.

Behavior Assessment System for Children, Second Edition (Self-Report Adolescent)

This booklet contains sentences that tell how some boys and girls think or feel or act. Read each sentence carefully. For the first group of sentences, you will have two answer choices: T or F. For the second group of sentences, you will have four answer choices: N, S, O, and A.

T True	F False

3. Nothing goes my way.	**T F**
8. I used to be happier.	**T F**
21. Nothing is fun anymore.	**T F**
33. Nobody ever listens to me.	**T F**
38. I just don't care anymore.	**T F**
51. I don't seem to do anything right.	**T F**
53. I have attention problems.	**T F**
68. Nothing about me is right.	**T F**

N Never	S Sometimes	O Often	A Almost aways

81. I feel like my life is getting worse and worse.	**N S O A**
93. I feel depressed.	**N S O A**
98. No one understands me.	**N S O A**
111. I feel sad.	**N S O A**

Footnote: Items from the depression subscale are presented only.