

PUBERTAL TIMING AND INTERNALIZING PSYCHOPATHOLOGY OF ADOLESCENT
FEMALES: EVALUATING THE MATURATION DISPARITY HYPOTHESIS

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FEMALES: EVALUATING THE MATURATION DISPARITY HYPOTHESIS

The following faculty members have examined the final copy of this dissertation for form and content, and recommend that it be accepted in partial fulfillment of the requirement for the degree of Doctor of Philosophy with a major in Psychology.

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ABSTRACT

Previous research has demonstrated a robust link between early pubertal timing and increased risk for anxious and depressive symptoms among female adolescents. Although a number of explanations have been proposed, the most widely accepted—the maturation disparity hypothesis—has yet to be empirically tested. This hypothesis posits that the discrepancy between physical and psychological maturation among early physical developers increases risk for internalizing psychopathology, and empirically testing this hypothesis was the primary aim of this dissertation. Additional aims included replicating the commonly found association between early pubertal timing and psychopathology, as well as tracking the trajectory of adolescent egocentrism across adolescence. A community sample of 137 girls age 12 to 15 years old ($M = 14.32$, $SD = 1.03$) completed self-report measures on demographics, physical and social-cognitive development, and anxiety and depressive symptomatology. Results indicated that earlier pubertal timing was predictive of an increase in depressive symptoms, but was not associated with anxious distress. Also, contrary to expectations, imaginary audience ideation did not follow an inverted-U trajectory, as suggested by Elkind's (1967) theory and other previous research; however, the declining trajectory was consistent with Lapsley's (1993) "new look" model and accompanying literature. Partial support was found for the maturation disparity hypothesis. The difference between physical and psychological development significantly predicted depressive symptoms, while earlier pubertal timing and the interaction of pubertal timing by more advanced physical than psychological development were marginally significant predictors. Anxiety was also associated with a greater discrepancy between physical and psychological maturities, although neither pubertal timing nor the interaction term were significant. Clinical applications and directions for future research are highlighted.

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CHAPTER I

INTRODUCTION

Anxiety and depression are common psychological problems for youth. Lifetime prevalence rates for these disorders among adolescents are 31.9% and 14.3%, respectively, making internalizing conditions the most common form of psychopathology affecting young people (Merikangas et al., 2010). Not only are anxiety and depression widespread, but they also carry considerable economic, physical, and psychosocial costs. It is estimated, for example, that anxiety disorders cost the US between \$42.3 and \$46.6 billion annually (DuPont et al., 1996; Greenberg et al., 1999), while others argue that these figures vastly underestimate the actual cost (Kessler & Greenberg, 2002). Depression, on the other hand, is believed to be the most costly of all physical and mental disorders for young to middle-aged individuals, with annual US estimates at \$83.1 billion (Greenberg et al., 2003; Greenberg, Stiglin, Finkelstein, & Berndt, 1993; Kessler & Greenberg, 2002). In addition to being costly, anxiety and depression are related to the onset, persistence, and/or severity of a number of physiological conditions, including chronic pain, cancer, stroke, and cardiovascular disease (Evans & Charney, 2003; Lu et al., 2012; Roy-Byrne et al., 2008), and internalizing adolescents report a twofold increase in medical problems as young adults relative to their mentally healthy counterparts (Bardone et al., 1998). Adolescent anxiety and depression are also associated with a variety of negative psychological and social outcomes, such as substance use (O'Neil, Conner, & Kendall, 2011), poor academic outcomes (Ialongo, Edelsohn, Werthamer-Larsson, Crockett, & Kellam, 1995; Owens, Stevenson, Hadwin, & Norgate, 2012), as well as family and peer relationships characterized by greater dysfunction and conflict (Brumariu, Obsuth, & Lyons-Ruth, 2013; Sheeber, Hops, Alpert, Davis, & Andrews, 1997).

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Given the economic and human costs associated with anxiety and depression, it is fortunate that effective psychological and pharmacological treatments exist (e.g., cognitive-behavioral therapies, selective serotonin reuptake inhibitors; Kendall, 1994; Melvin et al., 2006; Walkup et al., 2008). Unfortunately, not all youth respond to these interventions, but with a greater understanding of the factors that bring about and maintain anxiety and depression, more effective prevention and treatment programs can be developed. One such factor related to youth internalizing disorders is pubertal timing, or the timing of puberty relative to one's peers (i.e., can be early, on-time, or late). The most robust and consistent finding is that early maturing females are at the greatest risk for internalizing outcomes compared to all other groups (e.g., later maturing girls, early maturing boys; Mendle, Turkheimer, & Emery, 2007; Negriff & Susman, 2011; Reardon, Leen-Feldner, & Hayward, 2009).

A number of explanations have been proposed to account for the link between pubertal timing and psychopathology. The most widely accepted explanation is the maturation disparity hypothesis. This explanation posits that a mismatch between physical and psychological maturities place early physical developers at an increased risk because these individuals are not psychologically prepared to successfully navigate the challenges associated with puberty (Allison & Hyde, 2013; Ge & Natsuaki, 2009). Despite its popularity, the maturation disparity hypothesis has never been directly tested. A primary challenge in evaluating this explanation has been the assessment of psychological maturation; however, adolescent egocentrism (Piaget, 1926, 1929; Piaget & Inhelder, 1956) may offer a needed assessment avenue. Specifically, adolescent egocentrism refers to the tendency for adolescents to believe that others are as preoccupied with their behavior and appearance as they are. Its two component parts—the imaginary audience and personal fable—refer to an adolescent's belief that they are the focus of

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others' attention and that they are special, unique, omnipotent, and invulnerable, respectively (Elkind, 1967). Therefore, these characteristic ways of thinking and viewing others can be conceptualized as reflecting adolescent social-cognitive development.

Purpose of the Study

The primary aim of the present study is to empirically evaluate the maturation disparity hypothesis with a community sample of adolescent females. Specifically, the project seeks to examine whether the gap between physical and psychological development places early physical maturers at an increased risk for anxiety and depression. In line with the maturation disparity hypothesis, adolescents with more advanced physical relative to psychological development should report greater levels of internalizing psychopathology than other groups (i.e., on-time physical and psychological developers, psychological development more advanced than physical development).

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CHAPTER II

LITERATURE REVIEW

The stages of childhood and adolescence are widely recognized as critical developmental periods. This stems from the notion that experiences during youth reverberate and influence across the lifespan, largely setting the stage for later development and adjustment. Adolescence in particular is characterized by a number of profound physical, psychological, and social changes that occur over a relatively short span of time. These novel and rapid changes can, unfortunately, place some individuals at increased vulnerability for an array of problems, ranging from breast cancer (Apter & Vihko, 1983; Houghton et al., 2014) and diabetes (He et al., 2010) to internalizing (Crockett, Carlo, Wolff, & Hope, 2013; Hayward & Sanborn, 2002) and externalizing disorders (Beltz, Corley, Bricker, Wadsworth, & Berenbaum, 2014; Dimler & Natsuaki, 2015). Even G. Stanley Hall (1904), a founding father of developmental psychology, recognized the turmoil that can characterize this stage of development when he coined the phrase ‘storm and stress’ to describe the changes that accompany adolescence. Considering the vulnerability and potential problems associated with this period in the lifespan, adolescence represents an optimal window for intervention. However, in order to develop and implement successful interventions, the processes by which psychological symptoms and disorders are engendered and maintained must be understood. With such an understanding, adolescents at the greatest risk can be identified and trajectories of maladjustment and psychopathology can be altered. Given the prevalence and outcomes associated with adolescent internalizing disorders, it is essential that this class of dysfunction be well understood.

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Adolescent Internalizing Disorders

Internalizing psychopathology includes anxiety and depressive symptoms and disorders and is contrasted with externalizing problems, which correspond to more overt behaviors and dysfunction (e.g., aggression, hyperactivity, delinquency). Anxiety and depression are common among children and adolescents and are often cited as being the most prevalent form of youth maladjustment. In a large and nationally representative study of adolescents, anxiety disorders were found to have the highest lifetime prevalence rate with 31.9% and mood disorders to have the third highest rate with 14.3% (Merikangas et al., 2010). In addition, females were more likely to meet criteria for these disorders, anxiety disorders were reported to have the earliest onset of any class with a median age of 6 years, and the rate of mood disorders increased with age, nearly doubling from the early to late adolescent years. Similar findings are found with 12-month and 30-day prevalence estimates, with anxiety and mood disorders being the first and third most common, respectively, and internalizing disorders being more prevalent among females (Kessler et al., 2012). Lifetime prevalence of specific anxiety and mood disorders indicate that specific phobia (20%), major depressive disorder (10.6%), and social phobia (8.6%) are most common and also occur more often in females than males (Kessler, Petukhova, Sampson, Zaslavsky, & Wittchen, 2012). Twelve-month prevalence rates are similar to lifetime prevalence, with specific phobia (16.3%) being the most common, but social phobia (7.9%) occurring slightly more often than major depressive disorder (7.4%). Additionally, anxiety and depression are highly comorbid among youth, with some estimates being as high as 62% to 74% comorbidity within clinical samples (Avenevoli, Stolar, Li, Dierker, & Merikangas, 2001; Brady & Kendall, 1992), and comorbidity is more common when depressive disorders are the primary problem (Cummings, Caporino, & Kendall, 2014). Taken together, these developmental

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prevalence estimates suggest that internalizing disorders are the most prevalent form of adolescent psychopathology, consistently found more frequently in females, and are highly comorbid.

Not only are internalizing problems common among adolescents, but they also carry immense financial, physical, and psychosocial costs. Although updated figures are needed, Greenberg and colleagues (1999) estimate the annual US cost of anxiety disorders to be \$42.3 billion while another team estimated this cost at \$46.6 billion (DuPont et al., 1996), and similar figures have also been reported in Europe (Andlin-Sobocki & Wittchen, 2002). The US estimates include psychiatric and non-psychiatric treatments, indirect workplace costs, and lost earnings potential due to mortality. However, Kessler and Greenberg (2002) argue that these figures grossly underestimate the actual cost of anxiety disorders, as they exclude costs associated with comorbidity as well as long-term unemployment and underemployment, the latter of which alone is estimated to be equivalent to an annual cost of over \$100 billion. Estimates related to depression range from \$43.7 billion to \$52.9 billion a year and, as a result, depression is generally considered to be the most costly of all physical and psychological conditions for people in the early to middle stages of life (Greenberg et al., 1993; Kessler & Greenberg, 2002). A more recent figure places the annual US cost of depression at \$83.1 billion, which includes direct treatment, workplace, and suicide-related costs (Greenberg et al., 2003). Although there are limitations to the above figures (e.g., dated estimates) and the economic impact related to internalizing disorders among children and adolescents specifically is needed (see Lynch & Clarke, 2006), it should nevertheless be clear that the societal cost stemming from anxiety and depression is substantial, emphasizing the importance of early intervention and prevention.

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In addition to a financial burden, anxiety and depressive disorders also influence and contribute to physiological problems. Both anxiety and depression affect medical illness in terms of onset, persistence, and severity. As identified in a review by Roy-Byrne and colleagues (2008), specific medical conditions associated with anxiety include chronic pain, irritable bowel syndrome, asthma, cancer, and cardiovascular disease. These are significant conditions given, for example, the negative implications of childhood asthma on adult health status and productivity (Fletcher, Green, & Neidell, 2010) as well as the high mortality rates associated with cancer and cardiovascular disease, with the latter being the leading cause of mortality in the US (Chang, Moonesinghe, Athar, & Truman, 2016; Jemal, Ward, Hao, & Thun, 2005). In a longitudinal study, Bardone et al. (1998) found that adolescent girls with an anxiety disorder reported twice the number of medical problems in young adulthood compared to healthy controls. Similar relationships exist between depression and physical illnesses, including chronic pain, asthma, cancer, stroke, and cardiovascular disease (Evans & Charney, 2003; Lu et al., 2012; Roy-Byrne et al., 2008), and, after experiencing depression as an adolescent, young adult females again reported double the number of medical problems as their psychologically healthy counterparts (Bardone et al., 1998). Depression also has implications for compliance with medical recommendations and treatment, as a meta-analysis demonstrated a threefold increase in noncompliance among depressed over nondepressed patients (DiMatteo, Lepper, & Croghan, 2000). Although there are shared risk factors and high comorbidity between internalizing disorders, which likely contribute to these similar physiological outcomes, evidence suggests that there are unique pathways between anxiety and depressive disorders and medical conditions (Sareen, Cox, Clara, & Asmundson, 2005). A complete discussion on the influence of anxiety and depression on physical illnesses is beyond the scope of the present report, but it should be

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noted that internalizing disorders affect medical conditions (and vice versa) in significant and lasting ways.

Psychological and social correlates of youth internalizing symptoms and disorders also illustrate the considerable impact of anxiety and depression, highlighting the amount of personal suffering that stem from these conditions. Both anxiety and depression, for example, are associated with later substance use and evidence suggests that internalizing problems precede substance use disorders (i.e., there is unidirectional risk; O'Neil et al., 2011). Woodward and Fergusson (2001) report that anxiety disorders during adolescence are predictive of suicidal behavior, early parenthood, and, not surprisingly, risks of anxiety disorders and major depression in late adolescence and emerging adulthood. Anxiety and depression are also associated with poorer academic achievement, in terms of lower academic competence and performance as well as higher dropout rates (Ialongo et al., 1995; Owens et al., 2012; Quiroga, Janosz, Bisset, & Morin, 2013; Van Ameringen, Mancini, & Farvolden, 2003). The social relationships of anxious and depressed youth tend to be marked by more dysfunction, both with peers and family members (Brumariu et al., 2013; Sheeber et al., 1997; Siegel, La Greca, & Harrison, 2009; Verduin & Kendall, 2008; Zimmer-Gembeck, Hunter, Waters, & Pronk, 2009). Unlike the association with substance use disorders, most of these social and psychological attributes function bidirectionally; that is, for instance, depression is both a predictor and outcome of decreased peer likeability (Zimmer-Gembeck et al., 2009).

Given the significant economic and human costs associated with youth internalizing psychopathology and their high prevalence rates, the development and dissemination of effective treatments is essential. Fortunately, both psychological and pharmacological interventions exist, such as cognitive-behavioral therapy (CBT; Barrett, Dadds, & Rapee, 1996; Ebert et al., 2015;

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Ishikawa, Okajima, Matsuoka, & Sakano, 2007; Kendall, 1994; Melvin et al., 2006; Walkup et al., 2008; Weisz, Hawley, & Doss, 2004) and selective serotonin reuptake inhibitors (SSRIs; Dubicka et al., 2010; Melvin et al., 2006; Walkup et al., 2008). However, these interventions are not perfect insofar as treatment nonresponders are not uncommon with children and adolescents (see, for example, Barrett et al., 1996; Kendall, 1994; Walkup et al., 2008). One avenue for improving these response rates is to better understand the risk factors and processes that contribute to the etiology and maintenance of youth internalizing disorders. With a more complete understanding, at-risk youth could be identified early and provided prevention services. More effective and efficient treatments could also be developed for youth already struggling with anxiety and depression, such as by implementing specific interventions designed to address particular classes of risk or maintenance factors. An area ripe for exploration and with potential for such clinical implications is pubertal timing.

Puberty and Internalizing Psychopathology

Pubertal development. Puberty involves a set of biological and hormonal processes that result in sexual maturation and marks the transition from childhood to adolescence. The physical changes that accompany puberty are categorized as either primary or secondary sex characteristics. The former involves development of body structures that are directly related to reproduction, including the ovaries and uterus in girls and scrotum and testes in boys. Secondary characteristics are changes that are not directly involved in sexual reproduction, such as breast development and widening of the pelvis in girls, facial hair and broadening of the shoulders in boys, as well as body hair and skin changes in both boys and girls (Kail & Cavanaugh, 2015). Puberty is first evident with breast development in girls and genital development in boys, which generally occur around 10 and 11.5 years of age, respectively (Euling et al., 2008). Most

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research suggests that there has been a secular trend towards earlier puberty for youth in developed countries since industrialization and that this trend leveled off during the second half of the 20th century (Chumlea et al., 2003; Karpati, Rubin, Kieszak, Marcus, & Troiano, 2002; Mul et al., 2001; Parent et al., 2003; Sun et al., 2002; Sun et al., 2005). Parent et al. (2003), for instance, identify that the mean age of menstruation onset in Western countries declined from 17 years in the mid-19th century to less than 14 years a century later. However, some debate exists as to whether there is enough evidence to support such trends, as studies differ in important respects (e.g., population, assessment methods), making across-study comparisons difficult (see Euling et al., 2008).

Furthermore, puberty is an endocrinological event that is primarily controlled by the hypothalamic-pituitary-gonadal (HPG) axis. The HPG axis is first active during gestation until a few months following birth, a period some have termed “minipuberty,” and then becomes largely dormant throughout childhood (Kuiri-Hanninen, Sankilampi, & Dunkel, 2014). Adolescent puberty, therefore, represents a reactivation of the HPG axis. This is initiated by the excitation of gonadotrophin releasing hormone (GnRH) in the hypothalamus (Delemarre-von de Waal, 2002) as well as additional metabolic, nutritional, and steroidal factors (Sorensen et al., 2012). Other hormones implicated in puberty include estrogen and the androgen hormone testosterone. The pituitary gland signals the release of estrogen by the ovaries and testosterone by the testes (Kail & Cavanaugh, 2015); however, estrogen and testosterone are involved in both female and male changes that accompany puberty (Kuhn & Muller, 1996). For example, testosterone is involved in pubic hair development in girls and both testosterone and estrogen are implicated in the pubertal growth spurt among boys.

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The surge of hormones during puberty not only culminates in sexual maturity and causes various co-occurring physiological changes, but the hormonal influx also results in a number of neurological changes. These include increases in white and grey matter, synaptogenesis in the prefrontal cortex, as well as growth in the amygdala and hippocampus (Blakemore, Burnett, & Dahl, 2010; Blakemore & Choudhury, 2006; Chavarria, Sanchez, Chou, Thompson, & Luders, 2014; Goddings et al., 2014; Ladouceur, Paper, Crone, & Dahl, 2012). Naturally, such profound neurological alterations have behavioral and psychological implications. Some suggest, for example, that changes in the brain's decision-making centers in concert with reconfiguration of areas that regulate emotion and reward explain the increase in risk-taking behaviors (e.g., substance use) often observed in adolescents (Bava & Tapert, 2010; Jacobus et al., 2013). The surge of hormones and considerable neurological changes that accompany puberty may contribute to psychological vulnerabilities for some adolescents. Variations in the timing when adolescents experience these changes may also exacerbate vulnerabilities, resulting in various increases in risk for psychological maladjustment.

Pubertal timing and internalizing outcomes. Puberty itself cannot be considered a risk factor for developmental outcomes insofar as a risk factor cannot be experienced by the entire population. Alternatively, the timing at which adolescents begin puberty relative to their peers—known as *pubertal timing*—has interindividual variability and therefore can be conceptualized as a risk (or protective) factor for various psychological and behavioral outcomes (Reardon et al., 2009). Pubertal timing can be early, on-time, or late in relation to peers' development and is categorically different from other puberty-related constructs as pubertal status and tempo. The former is an absolute measure that refers to an adolescent's current level of physiological development within the overall process of puberty (Dubas, Graber, & Petersen, 1991) and is

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often assessed with specific pubertal events, such as menarche in females, which is the onset of menstruation, and spermarche or oigarche in males, which refers to the first spontaneous emission of sperm-laden fluid and the first ejaculation, respectively (Reardon et al., 2009).

Pubertal tempo, on the other hand, is defined as the rate of intraindividual progression through pubertal stages, which can be either relatively fast or slow (Keenan, Culbert, Grimm, Hipwell, & Stepp, 2014; Mendle, Harden, Brooks-Gunn, & Graber, 2010). While some literature proposes that so called “off time” puberty (i.e., early or late) places individuals at risk for negative outcomes (see, for example, Graber, Lewinsohn, Seeley, & Brooks-Gunn, 1997; Hummel, Shelton, Heron, Moore, & van den Bree, 2013; Natsuaki, Biehl, & Ge, 2009), early developing adolescent females appear to be at the greatest risk for internalizing symptoms and disorders.

Research suggests that early maturing females are at the greatest risk for developing maladaptive anxiety. Using a large sample ($N = 5,692$) of archival data, Weingarden and Renshaw (2012) examined women’s perceived (i.e., retrospective report of) pubertal timing and anxiety disorder outcomes. Results indicated that early pubertal timing was predictive of having any lifetime diagnosis of an anxiety disorder, with increased risks for social anxiety disorder, posttraumatic stress disorder, and specific phobia, while late pubertal timing was only predictive of social anxiety disorder. Additional studies have also found incremental risks of social anxiety symptoms for early maturing females (Blumenthal, Leen-Feldner, Babson, et al., 2011; Blumenthal, Leen-Feldner, Trainor, Babson, & Bunaciu, 2009; Deardorff et al., 2007). Blumenthal and colleagues (2011), for instance, report that early maturing girls endorsed the highest level of social anxiety in comparison to all other groups (e.g., on-time and late maturing girls, early maturing boys) in a community sample. On-time and late maturing individuals did not differ in self-reported social anxiety symptoms as a function of gender nor did boys differ as

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a function of pubertal timing. Likewise, Deardoff and colleagues (2007) examined the interactive role of gender and found that only early maturing girls had increased social anxiety symptoms, while again no relationship was found between pubertal timing and social anxiety for boys. Looking at anxiety outcomes more generally, Zehr, Culbert, Sisk, and Klump (2007) found that undergraduate women and men with earlier pubertal timings reported higher state and trait anxiety levels than on-time or late maturers, with early developing women endorsing significantly greater anxiety than early developing men.

In an exhaustive literature review on the link between pubertal timing and anxiety, Reardon and colleagues (2009) identified the most prominent and consistent finding as increased risk of anxiety symptoms and disorders for early maturing females, suggesting that early pubertal development in girls has a persistent and clinically significant effect on anxiety outcomes. Late maturation among females was generally not associated with anxiety outcomes, although this finding is less consistent, and the literature on boys is relatively small and findings are largely inconsistent. Although strengths of the research reviewed include several large, representative longitudinal studies and the use of multiple informants, a number of limitations are also identified. Some studies, for example, used assessments of anxiety with questionable psychometrics (e.g., single-item measures) and similar problems exist with the assessment of pubertal timing (e.g., lack of multiple assessment points, retrospective reports). Other limitations include the lack of attention to possible moderators and mediators, despite theoretically-relevant variables, such as substance use, parent-adolescent relationship quality, as well as the emergence of romantic and sexual relationships (Cance, Ennett, Morgan-Lopez, Foshee, & Talley, 2013; Compian, Gowen, & Hayward, 2004; Hummel et al., 2013). This is a particularly salient limitation given that these third variables could help explain the processes and contexts

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associated with the link between pubertal timing and anxiety. These shortcomings notwithstanding, the current evidence suggests a reliable association between early pubertal timing and increased risk for anxiety problems in girls, with particularly strong findings for social anxiety, while showing less consistent findings for both later maturing females specifically, and males more generally.

Anxiety is not the only type of psychopathology that appears related to pubertal timing. Given the high comorbidity between youth anxiety and depressive disorders (Brady & Kendall, 1992; Cummings et al., 2014), it may not be surprising that depression also appears to be associated with variability in pubertal timing. In a recent longitudinal study with a large ($N = 2,450$) and diverse community sample of girls, early pubertal timing was predictive of depressive symptoms from early to late adolescence (Keenan et al., 2014). In a similar study, Mendle and colleagues (2010) also report that girls with an earlier onset of puberty endorsed greater levels of depressive symptoms throughout puberty compared with on-time and late maturing girls and with boys regardless of pubertal timing. With another large community sample of both girls ($N = 17,082$) and boys ($N = 15,992$), Kaltiala-Heino, Kosunen, and Rimpela (2003) found that early pubertal timing was associated with self-reported depression among both girls and boys, and late maturation was related to depression for boys. An additional study with the same sample showed that early maturing youth reported the highest level of depression compared to any other group (Kaltiala-Heino, Marttunen, Rantanen, & Rimpela, 2003). Although there does appear to be some gender similarities, early maturing girls tend to report the most severe levels of depression (Ge, Conger, & Elder, 2001) and these levels often persist into later adolescence, while depression levels in early maturing boys tend to subside earlier (Ge et al., 2003). One suggested process for explaining gender differences in severity and duration of depressive

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symptoms is emotional clarity, which is the ability to identify and understand emotional experiences (Hamilton, Hamlat, Stange, Abramson, & Alloy, 2014). In a recent study by Hamilton and colleagues (2014), although early pubertal timing and negative cognitive styles predicted depressive symptoms among both girls and boys, poor emotional clarity increased symptoms for girls only, suggesting that such emotional vulnerabilities may enhance the risk and expression of depression among early maturing girls.

Literature reviews on the link between pubertal timing and depressive affect during adolescence conclude that the most reliable finding is increased depressive symptoms among early maturing girls relative to both later developing girls and boys across the pubertal timing spectrum (Mendle, et al., 2007; Negriff & Susman, 2011). This finding remains even when considering various measures of puberty used across studies. Unlike the literature on females, both early and late maturation among boys appear to be risk factors for depression, but early maturation does seem to have incremental risk relative to late pubertal development for males. In a recent meta-analysis on the effects of pubertal timing on depressive symptoms and disorders in adolescent females, Galvao and colleagues (2014) conclude that early developing girls are at the greatest risk for depression, while late puberty was not associated with depressive disorders and may, alternatively, function as a protective factor against depressive symptoms. The authors identify a number of limitations in this body of literature, namely few available studies, differences in measurement of depression, as well as the use of retrospective reports of the age of menarche as a pubertal timing assessment, which may be subject to error when collected in middle age (Cairns et al., 2011; Cooper et al., 2006), but not all studies have supported such validity concerns (e.g., Bean, Leeper, Wallace, Sherman, & Jagger, 1979; Damon, Damon, Reed, & Valadian, 1969). However, self-report retrospective measures of pubertal timing that are

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short-term, or collected throughout adolescence, tend to be valid and reliable (Brooks-Gunn, Warren, Rosso, & Gargiulo, 1987; Caspi & Moffitt, 1991; Dubas et al., 1991; Graber et al., 1997). Nevertheless, these reviews and meta-analyses suggest that early maturing females are at an increased risk for depressive psychopathology.

Additional studies have examined the effects of race on the link between pubertal timing and internalizing psychopathology, although investigations of race and other moderators are less common than studies examining the direct or gender effects discussed above. In one large study of African American girls and boys, earlier developing youth were more likely to endorse symptoms of social anxiety, generalized anxiety, and major depression in a structured interview than on-time or late maturers (Ge, Brody, Conger, & Simons, 2006). Another study with a sample of African American girls found that adolescents' retrospective timing of having either early or late breast development was associated with higher teacher-rated internalizing problems (Carter, Jaccard, Silverman, & Pina, 2009). Carter (2015) recently examined the relationship between pubertal timing and specific anxiety symptoms among African American girls and boys and reports that early developing girls had higher levels of physical and harm avoidance symptoms, whereas late pubertal timing was associated with higher levels of anxiety for boys, including physical, harm avoidance, and social anxiety symptoms. In the Keenan et al. (2014) study mentioned above, earlier pubertal timing was associated with greater depressive symptoms for both African American and European American girls. African American girls also reported higher rates of depressive symptoms and were found to initiate puberty earlier than European American girls, both of which are consistent with other research (Biro et al., 2006; Chumlea et al., 2003; Franko, Striegel-Moore, Thompson, Schreiber, & Daniels, 2005; Garrison, Jackson, Marsteller, McKeown, & Addy, 1990; Mendle et al., 2007; Mendle et al., 2010). An additional

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study by Ge and colleagues (2003) again finds that early maturing African American girls report more depressive symptoms than their on-time and late developing counterparts. Although more research with minorities is needed, it appears as though African American and European American girls have similar internalizing outcomes as a result of pubertal timing, with early developers consistently being at the greatest risk.

Given this extant research, it should be clear that pubertal timing is related to risk for internalizing psychopathology and, in particular, girls with earlier puberties relative to their peers are at the greatest risk for these negative outcomes. This is the most consistent finding and is found with both anxiety and depressive symptoms and disorders and across at least some racial groups. Late pubertal timing in girls is usually not associated with these outcomes, although a few studies have found such a link (e.g., Carter et al., 2009; Weingarden & Renshaw, 2012), while the research on boys is more scarce and mixed with considerably less consistent findings across studies. Despite a number of notable strengths, such as several large longitudinal studies with repeated measures, this body of literature also has some salient limitations, including the use of single items to assess pubertal timing and few studies examining possible third variable and directional effects. Additionally, although a number of explanations have been proposed, direct tests of potential explanations for the link between pubertal timing and internalizing outcomes are needed. Empirical investigations into these explanations represent the next logical step among this avenue of research.

Explanations for the Early Pubertal Timing-Psychopathology Link

Maturation disparity hypothesis. Paradoxically, the most accepted yet infrequently tested explanation for the early pubertal timing-psychopathology link is the maturation disparity hypothesis (Allison & Hyde, 2013; Ge & Natsuaki, 2009), which has also been referred to as the

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early timing hypothesis, the developmental readiness hypothesis, and the stage termination hypothesis (e.g., Brooks-Gunn, Petersen, & Eichorn, 1985; Crockett et al., 2013; Hendrick, Cance, & Maslowsky, 2016; Negriff, & Susman, 2011). According to this hypothesis, “it is the gap between physical and psychological maturities that place early (physical) maturers at risk for developing psychopathology” (Ge & Natsuaki, 2009, p. 328). In other words, physical and neurological growth that accompany puberty develops prior to psychological developments in early maturing youth, and this discontinuity between systems results in an increased risk for developing behavioral and psychological problems. Allison and Hyde (2013) suggest that developmental events in childhood must be completed chronologically to promote normal adjustment; however, stemming from an abbreviated prelude to the changes associated with puberty, early maturers may be less prepared cognitively, affectively, and socially for the physical, psychological, and social challenges of puberty (Ge & Natsuaki, 2009). One such challenge, for example, may be an actual or perceived increase in social expectations by others placed on early maturers as a result of their older, more mature appearance (Vaughan, Van Hulle, Beasley, Rogers, & D’Onofrio, 2015). Although never mentioning this hypothesis explicitly, Blumenthal and others (2009) commenting on their work in the area stated:

This research has highlighted the particular importance of early maturation, suggesting that early developers are often faced with physiological development and social changes before they are prepared to manage them effectively, leaving them especially vulnerable to factors that may lead to the development of psychopathology. (p. 403)

In addition to accounting for the link between early pubertal timing and internalizing psychopathology among females, the research on structural and organizational changes in brain regions (e.g., prefrontal cortex, amygdala, hippocampus; see above) associated with puberty and

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pubertal hormones is also congruent with this hypothesis. Specifically, these structures, which are implicated in such abilities as decision making and emotional control, may not be adequately developed in early maturers to effectively manage the developmental challenges associated with puberty. There is also evidence from cognitive neuroscience that provides ancillary support for the maturation disparity hypothesis. Nelson, Leibenluft, McClure, and Pine (2005) assert that a mismatch between adolescents' emotional and cognitive regulatory systems may increase the risk for adverse psychological and behavioral outcomes among some youth. Additional evidence indicates that neural structures responsible for many higher-order functions (e.g., executive functioning, self-regulation) are still developing during adolescence, carrying implications for psychological and behavioral adjustment (Casey, Jones, & Hare, 2008; Casey, Tottenham, Liston, & Durston, 2005; Fjell et al., 2012; Spear, 2000). These findings imply that some brain structures—and therefore related psychological and cognitive abilities—may be particularly underdeveloped for early maturers and that this more pronounced mismatch might further increment risk for negative outcomes.

Despite its wide acceptance and popularity, there has been only one study (to the author's knowledge) that purports to directly test the maturation disparity hypothesis. In a recent longitudinal study from adolescence to emerging adulthood, Thompson, Hammen, and Brennan (2016) investigated the effect of pubertal synchrony—the level of concurrent development among morphological indicators of puberty—on depressive symptoms of a community sample of Australian females. However, when outlining the maturation disparity hypothesis, a broader and conflicting definition was used. Specifically, the authors described the maturation disparity hypothesis as suggesting “that girls who mature off-time relative to peers may feel abnormal and alienated from others, resulting in increased stress and a sense of isolation that can contribute to

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the development of psychopathology” (p. 494). Clearly this description is considerably different from those provided by Ge and Natsuaki (2009) and others (e.g., Allison & Hyde, 2013; Negri & Susman, 2011; Vaughan et al., 2015). This description, conversely, places both early *and* late developers at risk and makes no mention of physical and psychological discontinuity.

Furthermore, Thompson et al. (2016) examined only disconnects in physiological indicators of puberty—the difference between breast and pubic hair development—as the measure of pubertal synchrony. Consistent with their description of the maturation disparity hypothesis, results indicated that pubertal asynchrony was predictive of depressive symptoms in emerging adulthood (but not adolescence) with late pubertal timing moderating this relationship. Although examining an often under-researched aspect of puberty (i.e., pubertal synchrony) contributes significantly to the puberty-psychopathology literature, the notion that the Thompson et al. (2016) study offers a test of the maturation disparity hypothesis is questionable given (a) the authors’ description of this hypothesis and (b) the measure of pubertal synchrony. Had the authors examined the synchrony between physical and psychological development rather than just between physical indicators of puberty, the study would have provided a more informative and accurate test of the hypothesis as it is typically described.

The maturation disparity hypothesis is attractive because it integrates biological, psychological, and social forces associated with puberty. This hypothesis may also help explain some of the gender differences found in the pubertal timing-psychopathology literature. More specifically, although puberty is inherently biological, there are certainly social factors that accompany and influence pubertal change and these may be gender-specific. Early maturing girls are the first of their same- and opposite-sex counterparts to initiate puberty and develop outward signs of puberty—which carries social implications (see Williams & Currie, 2000)—

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leading some to identify early developing girls as pioneers among their peers (Caspi & Moffitt, 1993). Boys, on the other hand, tend to have later pubertal onsets, even among early developers, and undergo more nuanced changes, particularly in the earlier stages of puberty. This may help explain why the most robust finding between pubertal timing and internalizing psychopathology is characteristic of early developing girls and, importantly, offers a rationale for gender-specific explanations; that is, puberty differs by gender in many respects and, as a result, a unified theory to explain the pubertal timing-psychopathology link may not be possible. However, the maturation disparity hypothesis cannot account for the few studies reporting an increased risk for late developing adolescent females. Also, if the disconnect between biological and psychological systems places early maturers at an increased risk for psychological problems, then late developers (both females and males) should theoretically have the best adjustment outcomes, as they should be psychologically the best prepared for biological changes because they are the most psychologically mature at the onset of their puberty. Research consistently demonstrates, however, that on-time youth tend to have the best outcomes. Therefore, it may be that not only are gender-specific theories needed, but also specific explanations based on pubertal timing or the interaction between gender and pubertal timing. It is not inherently true that one theory must apply across either gender or pubertal timing, as unique processes may be responsible for increased risk as a function of gender and/or timing. Nevertheless, there has been no direct test (to the author's knowledge) of the maturation disparity hypothesis with internalizing disorders specifically, or of the hypothesis more generally. Given the acceptance of this explanation, such empirical investigations are clearly warranted and needed.

Hormonal influence hypothesis. A second hypothesis that aims to explain the link between early pubertal timing and psychopathology is the hormonal influence hypothesis, which

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maintains that the rise in pubertal hormones places earlier developing adolescents at an increased risk for developing internalizing symptoms and disorders (Ge & Natsuaki, 2009; Vaughan et al., 2015). Although these hormonal changes occur regardless of pubertal timing, this explanation posits that either one or both of the following specifically increases risk for earlier maturers. First, less developed (i.e., younger) brains may be especially sensitive to pubertal hormones. Indeed, some research with animals suggests that pubertal hormones are more powerful when introduced earlier in the pubertal transition (Schultz, Molenda-Figueira, & Sisk, 2009). A second, but not mutually exclusive, route that may increment risk is that early maturers secrete more hormones throughout puberty than later developing adolescents. For example, some evidence suggests that girls experiencing premature pubarche (appearance of pubic hair before age 8) have higher levels of pubertal hormones from pubertal onset to after puberty (Ibanez, Street, Potau, Carrascosa, & Zampolli, 1997).

There is no doubt that hormones play a central role in puberty and the neurological changes associated with puberty, making the hormonal influence hypothesis intuitively appealing. However, research supporting this hypothesis is limited and no study has demonstrated a link between greater sensitivity to and/or increased levels of pubertal hormones and psychopathology. Although the use of experimental designs with animal studies may offer one avenue for testing this hypothesis, large-scale correlational studies with human youth are ideal. This research is presently lacking and, despite the many challenges, hormone research may be an important key to understanding the pubertal timing-psychopathology link. If the hormonal influence hypothesis does prove to be at least partially successful in explaining this association, then early interventions could be specifically targeted for youths at greatest risk for developmental psychopathologies, such as those showing high hormone levels at young ages.

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Contextual amplification hypothesis. The contextual amplification hypothesis posits that pubertal timing and adverse social contextual factors interact to influence psychopathology (Allison & Hyde, 2013; Ge & Natsuaki, 2009). Specifically, the rapid physiological changes that accompany puberty in concert with negative contextual factors, such as interpersonal conflict and neighborhood dysfunction, interact to accentuate the negative outcomes associated with earlier puberties. Ge and Natsuaki (2009) suggest that contextual factors may serve to either exacerbate (if negative) or inhibit (if positive) the adverse effects stemming from early pubertal timing through standards, opportunities, and contingencies inherent in the environment. Indeed, the interaction between puberty and social and environmental factors has been demonstrated in the past, as such contextual forces can influence pubertal timing. Among girls, for example, antecedents of earlier pubertal timing include childhood physical and sexual abuse (Negriff, Blankson, & Trickett, 2015; Romans, Martin, Gendall, & Herbison, 2003), parental dysfunction and negative parental rearing practices (Belsky et al., 2007; Graber, Brooks-Gunn, & Warren, 1995; Tither & Ellis, 2008), as well as father absence (Bogaert, 2005; Romans et al., 2003; Quinlan, 2003), although this latter influence may be moderated by ethnicity and family income (Deardorff et al., 2011).

It appears clear that puberty and contextual factors interact; however, only a few studies have directly tested the contextual amplification hypothesis by examining contextual moderators of the relationship between pubertal timing and psychopathology. Rudolph and Troop-Gordon (2010) found support for this hypothesis in that recent maternal depression and family stress predicted later depression among both early maturing girls and boys, although neither lifetime maternal depression nor lifetime family adversity were reliable moderators. Ge, Conger, and Elder (1996) examined the role of peer group sex composition, deviant peers, and paternal

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hostility on the interaction between pubertal timing and psychological distress in adolescent females. Findings for the sex composition of friends supported the contextual amplification hypothesis, as early maturing girls with mixed-sex as opposed to same-sex friends had greater levels of self-, mother-, and father-reported psychological distress. Similar support was found for deviant peer associations and paternal hostility insofar as both increased vulnerability for psychological distress for early compared to later maturers. Additional research on pubertal timing and externalizing behavior has also found support for contextual influences. Evidence suggests, for instance, that early puberty is more predictive of problem behaviors (e.g., substance use, school truancy, defying parents) among girls in cultures where adolescent heterosexual relationships are more accepted (Skoog, Stattin, Ruiselova, & Ozdemir, 2013). Similarly, early maturing girls in mixed-sex relative to all-girl secondary schools have also been found to have greater rates of delinquency (Caspi, Lynam, Moffitt, & Silva, 1993). Collectively, these studies support the contextual amplification hypothesis because they find unique vulnerabilities among early maturers (mostly girls) for psychological and behavioral maladjustment as a function of contextual factors, such as more challenging home, peer, and school environments.

As described above, this hypothesis posits that these increased vulnerabilities stem from contextual factors that exacerbate the effects of earlier pubertal timing via standards, opportunities, and contingencies inherent within that context. For example, girls in mixed-sex settings—whether in their peer group or school—may have greater access to peers who violate norms, offer opportunities for delinquency, and reinforce deviant behavior. The contextual amplification hypothesis specifically postulates that such a context will differentially affect early relative to on-time and late developers. It is important to note, however, that this evidence specifically and the hypothesis more generally are not mutually exclusive from either of the two

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previously outlined hypotheses. In fact, the studies discussed here may indirectly support the maturation disparity hypothesis as it may be that the negative effects stemming from challenging contextual factors are the result of a disconnect between more advanced physical (which places individuals in these challenging settings) and less developed psychological (which makes successful navigation of these challenges less likely) systems. In other words, evidence in support of the contextual amplification hypothesis does not necessarily oppose the maturation disparity hypothesis and, conversely, may actually support it.

Accentuation hypothesis. A final explanation for the early pubertal timing- psychopathology link is the accentuation hypothesis. This hypothesis proposes that challenging transitional periods, such as early physical maturation, tend to amplify previous psychological and behavioral difficulties because these periods are marked by a high degree of novelty, ambiguity, and stress (Allison & Hyde, 2013; Caspi & Moffitt, 1991; Ge & Natsuaki, 2009). In this case, individual differences—whether that is vulnerability for emotional and behavioral maladjustment, for example—are magnified in response to a demanding life transition. The negative outcomes associated with early pubertal timing, then, represent an accentuation of previous difficulties.

There have been a couple empirical tests of this hypothesis. Caspi and Moffitt (1991) were the first to find support in that earlier menarche magnified prepubertal levels of behavioral problems. Early maturing girls with a predisposition to externalizing behaviors before puberty exhibited the greatest maladjustment throughout adolescence compared to any other group (i.e., later maturing girls with and without such childhood behavioral problems). In addition to finding support for the contextual amplification hypothesis (as discussed above), Rudolph and Troop-Gordon (2010) also tested the accentuation hypothesis by examining whether prepubertal

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personal risks strengthened the relationship between earlier maturation and later adolescent depression. Indeed, prior depression, a tendency to respond poorly to stress, and depressive personality traits among earlier maturers more strongly predicted their later levels of depression. Mensah and colleagues (2013) also provide evidence for this hypothesis in that girls and boys with earlier pubertal timings demonstrated both poorer parent-reported psychosocial outcomes at puberty as well as earlier in childhood. This pattern was also found for behavioral difficulties, but only among boys, which is inconsistent with the Caspi and Moffitt (1991) study that found such a relationship among girls. Nevertheless, congruent with the accentuation hypothesis, these studies suggest that previous psychological or behavioral difficulties are magnified following an early pubertal transition.

In summary, there are several proposed hypotheses that aim to explain the link between earlier pubertal timing and increased risk for psychopathology. Although each appears to have some empirical support, the maturation disparity hypothesis has yet to be directly tested. This is a notable exception given that, according to Ge and Natsuaki (2009), this hypothesis is the most widely accepted explanation for the link. In their paper reviewing these hypotheses, Ge and Natsuaki (2009) observe that the maturation disparity hypothesis has “more often been implied rather than directly tested” (p. 329). The authors speculate that the scarcity of direct empirical tests is partially due to methodological challenges in defining psychological development. The present study aims to address this salient limitation in the research on pubertal timing and developmental psychopathology, and address this methodological challenge by using adolescent egocentrism to assess level of psychological maturity.

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Adolescent Egocentrism

According to Piaget (1926, 1929; Piaget & Inhelder, 1956), egocentrism refers to a lack of differentiation between the subjective and objective. This differentiation failure results in predictable behaviors at various stages of cognitive development, such as difficulties with perspective taking during the preoperational stage (Piaget & Inhelder, 1956). Although adolescents recognize that others have different views, thoughts, and feelings than their own, adolescent egocentrism refers to the tendency to be self-interested, as adolescents often fail to differentiate between the focus of their and others' thoughts, assuming that others are as preoccupied as themselves with their behavior and appearance (Elkind, 1967). This tendency to believe that others are similarly preoccupied constitutes *adolescent egocentrism*. Elkind (1967) first described two characteristics or components of adolescent egocentrism: (a) *Imaginary audience*, which refers to the adolescent's belief that they are the focus of others' attention, and (b) *personal fable*, which references an adolescent's belief that they are special, unique, omnipotent, and invulnerable. These two characteristics account for a number of common adolescent behaviors and experiences, such as the tendency for adolescents to be overly self-conscious and the increase in risk-taking behavior that often accompanies adolescence (Alberts, Elkind, & Ginsberg, 2007; Arnett, 1990a, 1990b; Greene, Krcmar, Walters, Rubin, & Hale, 2000; Lechner & Rosenthal, 1984; Ryan & Kuczkowski, 1994). Therefore, adolescent egocentrism in general, as well as its two component parts in particular, can be conceptualized as a form or indicators of adolescent social-cognitive development.

Despite representing an adolescent's social-cognitive growth, adolescent egocentrism does not follow a typical upward trajectory across adolescence. Conversely, because adolescent egocentrism reflects failures in both social and cognitive domains that characterize adolescents,

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the trajectory of this construct should theoretically follow an inverted-U path across adolescence, with lower levels at the onset of adolescence, a rise during middle adolescence, and then decreasing during late adolescence. There is empirical evidence for this inverted U-shaped trajectory. Enright, Lapsley, and Shukla (1979) found that adolescent egocentrism (which was a composite variable of imaginary audience, personal fable, and self-focus measures) among girls and boys followed an inverted-U trend, rising from sixth grade to eighth grade and then falling below either previous level for college students. Another, more recent cross-sectional study examined the trajectory of the imaginary audience and personal fable across early, middle, and late adolescence (defined as ages 11-14, 15-17, and 18-21, respectively; Schwartz, Maynard, & Uzelac, 2008). Although there were no statistically significant differences among females, the imaginary audience score rose from early to middle adolescence and then fell below both earlier levels in late adolescence. The female personal fable score displayed the same trend, and levels in late adolescence fell significantly relative to both developmentally earlier scores. Similarly, Goossens, Seiffge-Krenke, and Marcoen (1992) tested levels of adolescent egocentrism (consisting of imaginary audience, personal fable, and self-focus subscales) and found an increase from 7th to 9th grade and then a decrease at 11th grade for both adolescent girls and boys. This quadratic trend was statistically significant, and girls reported significantly greater levels of adolescent egocentrism scores than boys.

Additional studies have examined the imaginary audience component specifically (see Table 1). Elkind and Bowen (1979) examined the cross-sectional progression of the imaginary audience at 4th, 6th, 8th, and 12th grade, with mean ages of 9.4, 11.9, 13.7, and 17.7 years, respectively. Among girls, the imaginary audience scores increased from 4th to 6th grade then peaked in 8th grade before decreasing to its lowest level in 12th grade. The two component

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scales (Abiding Self and Transient Self) also demonstrated this inverted-U trajectory for females. The progression for boys was more mixed, as one imaginary audience scale (Abiding Self) followed the inverted-U pattern, while the other scale (Transient Self) had highest scores in 4th grade and lowest in 12th, with 8th grade having higher scores than at 6th grade. Ryan and Kuczkowski (1994) also found the same general pattern as Elkind and Bowen. More specifically, female imaginary audience scores (as well as the composite scales) increased from 7th to 8th grade and then decreased at 9th and again at 12th grade. Additional studies suggest that there is another—albeit less prominent—spike in female imaginary audience scores at the start of college, which then declines again soon after (Peterson & Roscoe, 1991; Rycek, Stuhr, McDermott, Benker, & Swartz, 1998). In other words, it appears that there may be a slight and brief increase in female’s imaginary audience levels at the initiation of college, although these scores fail to reach levels of the overall peak during 8th grade. Alberts and colleagues (2007) also demonstrated an upward trend peaking at 8th grade for the personal fable, although scores were only assessed at 6th, 7th, and 8th grade, making it unclear whether the personal fable continues to increase past 8th grade, levels off, or declines like the imaginary audience.

Furthermore, gender differences are often found in adolescents’ reports of the imaginary audience and personal fable. Elkind and Bowen (1979) report that girls endorsed higher imaginary audience levels than did boys, which is consistent with other research (Galanaki, 2012; Goossens et al., 1992; Gray & Hudson, 1984; Greene, Rubin, Hale, & Walters, 1996; Hauck, Martens, & Wetzel, 1986; Montgomery, 2005; Richter, Reaves, Deaver, & Lacy, 1982; Ryan & Kuczkowski, 1994; Rycek et al., 1998). However, not all studies report gender differences in imaginary audience ideation (e.g., Enright et al., 1979; Jahnke & Blanchard-Fields, 1993). Personal fable, on the other hand, tends to be more characteristic of males (Alberts et al.,

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2007; Galanaki, 2012; Greene et al., 1996; Goossens, Beyers, Emmen, & van Aken, 2002; Lapsley, FitzGerald, Rice, & Jackson, 1989). Although Elkind (1967) did not postulate such gender differences, these findings are often explained in that the socialization of females leads to greater self-consciousness and that of males results in increased risk taking and beliefs of invulnerability (Galanaki, 2012; Richter et al., 1982).

Considering evidence from these studies, adolescent egocentrism and its two components appear to largely follow an inverted-U course across adolescence—peaking at 8th grade and with a possible slight increase in late adolescence that coincides with the start of college and decreasing shortly thereafter. These patterns seem to hold especially true for females. It should be noted, however, that any general claim may be less applicable to the personal fable insofar as there are fewer studies examining this component. With the exception of gender differences and the spike in late adolescence among freshman college students (Peterson & Roscoe, 1991), these findings are congruent with Elkind's (1967) theory. He posited that the imaginary audience should start to diminish by age 15 or 16, at which point formal operational thought is established, and that the personal fable decreases via intimate relationships, as shared confidences reveal that others also experience these cognitive misperceptions, leading to more realistic and accurate thinking styles. Despite evidence supporting the original theory, additional research has reported conflicting results. At least one study, for example, reports an increase in adolescent egocentrism past the expected peak and before the spike at college (Adams & Jones, 1981), while other research finds no relationship with age (Galanaki, 2012; Goossens, 1984; Gray & Hudson, 1984; Jahnke & Blanchard-Fields, 1993; Lapsley, Milstead, Quintana, Flannery, & Buss, 1986; Montgomery, 2005; Richter et al., 1982). Although the exact reason for the inconsistent findings is not clear, some of the contradictory results in these studies relative to the

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ones discussed earlier may be explained by different measurement tools, small sample sizes, and only using male participants.

Research on the relationship between adolescent egocentrism and either internalizing symptoms or puberty is fairly limited. Some evidence suggests that the imaginary audience is related to generalized anxiety, and to social anxiety in particular, among adolescent and adult populations (Kelly, Jones, & Adams, 2002; Ryan & Kuczkowski, 1994). This makes theoretical and conceptual sense given that the imaginary audience is associated with such anxiety-related constructs as fear of negative evaluation and self-consciousness (Frankenberger, 2000; Kelly et al., 2002; Muuss, 1982; Rapee & Heimberg, 1997; Ryan & Kuczkowski, 1994; Takishima-Lacasa, Higa-McMillan, Ebesutani, & Smith, 2014). However, not all studies have found a link between the imaginary audience and increased anxiety (e.g., Lechner & Rosenthal, 1984). Additional evidence suggests that imaginary audience is positively associated with depression, while uniqueness and invulnerability aspects of the personal fable are positively and negatively, respectively, related to both depression and loneliness among European adolescents (Goossens et al., 2002). Similarly, Cohn and colleagues (1988) report that adolescent egocentrism and the imaginary audience are related to depression, although correlations were relatively small, and the imaginary audience was associated with greater endorsement of personal trauma. The only other available study on adolescent egocentrism and psychopathology indicated that neither the imaginary audience nor personal fable were reliably predictive of internalizing or externalizing problems in adolescent boys with behavioral disorders (Beaudoin & Schonert-Reichl, 2006). However, for boys without behavioral disorders, imaginary audience was positively associated with externalizing behaviors and the invulnerability component of the personal fable was negatively related to the internalizing problems. Although these studies suggest there may be

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some relationship between adolescent egocentrism and internalizing psychopathology, this research is too limited to draw any firm conclusions.

There are only two studies (to the author's knowledge) that have examined adolescent egocentrism and pubertal development. This is a significant limitation insofar as Elkind (1967) identified that adolescent egocentrism and the resulting self-absorption occurs "*because* [emphasis added] of the physiological [sic] metamorphosis" (p. 1029) of puberty. Galanaki (2012) examined the relationship between pubertal timing and status and the imaginary audience and personal fable. Results suggested that late pubertal timing reliably predicted lower levels of imaginary audience ideation, but only among boys, while there was no difference for girls. However, a three-way interaction between gender, grade, and pubertal timing suggests that late maturation in 12th grade is associated with higher imaginary audience scores than early and on-time maturation. Galanaki (2012) also found that late maturing girls report lower omnipotence (a personal fable component) scores than early and late maturers, while late developing boys tended to have higher omnipotence scores than boys with early and on-time maturations. Boys with late pubertal timing reported higher speciality (also related to the personal fable) scores than did earlier developers. There were no other differences among pubertal timing and additional facets of the imaginary audience or personal fable. The only significant pubertal status relationship was on a personal fable scale related to risk taking and suggested that adolescents within the pubertal transition had higher scores than did adolescents that had already completed puberty. Similarly, Cohn and others (1988) did not find any relationship between pubertal status and imaginary audience when controlling for age. Therefore, given that only a few of the imaginary audience and personal fable scales were related to pubertal development (either timing or status) despite the many scales examined, neither of these studies are suggestive of strong

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relationships among these constructs. This appears to be particularly true for females, as even less reliable findings have been reported.

Present Study

Summarizing the above discussion, a number of points are clear. First, anxiety and depression are common and highly comorbid in adolescence, carrying considerable economic, psychosocial, and psychosocial costs. Internalizing symptoms and disorders are also more common among females than males. Second, pubertal timing has been identified as a risk factor for youth anxiety and depression, with early maturing adolescent females at greatest risk. Third, the maturation disparity hypothesis offers one explanation for the early pubertal timing- psychopathology link. Despite its popularity and acceptance, this hypothesis has never been directly tested. A major contributing factor for this limitation is methodological difficulties related to measuring psychological development, which is a central aspect of this explanation. Fourth, adolescent egocentrism may offer an avenue for assessing psychological maturity, permitting for an adequate evaluation of the maturation disparity hypothesis. Fifth, although there is some conflicting evidence, most studies suggest that adolescent egocentrism follows an inverted-U trajectory across adolescence. Last, the relationship between adolescent egocentrism and both internalizing problems and puberty is largely unexamined.

The present study aims to empirically evaluate the maturation disparity hypothesis with a sample of female adolescents. This study may significantly contribute to research on the link between pubertal timing and risk for psychopathology because this hypothesis has never been directly tested. This study also aims to address a number of limitations often associated with pubertal timing and internalizing psychopathology research, such as the use of retrospective measures and assessing constructs with single-item questions. The decision to include just

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females in the study was informed by previous research. The link between pubertal timing and anxiety and depression is far more consistent for females than any connection between pubertal timing and internalizing outcomes among males. Research on adolescent egocentrism is also more consistent for females than males. These findings suggest that females are better candidates for a direct test of the maturation disparity hypothesis. The exclusive focus on females can also be justified by their higher prevalence rates of internalizing problems during adolescence. Females also report higher levels of anxious and depressive distress and poorer outcomes as a result of this maladjustment than do males. Therefore, not only are females better suited for testing this hypothesis, but a greater understanding of the processes that give rise to and maintain these psychological problems may lead to prevention and intervention efforts with stronger and more lasting effects, highlighting the possible clinical implications of the present investigation.

In order to test the maturation disparity hypothesis, a community sample of 137 female adolescents completed self-report assessments on pubertal development and timing, adolescent egocentrism, and symptoms of anxiety and depression. The study hypotheses are as follows:

1. Consistent with past research, early maturing girls are expected to report the highest levels of internalizing symptoms relative to on-time and late developing girls.
2. In line with most of the adolescent egocentrism literature as well as Elkind's (1967) theory, imaginary audience and personal fable levels are expected to follow an inverted-U trajectory across the assessment age range, with a peak at approximately 13.5 years old, which is roughly equivalent to the 8th grade.
3. Congruent with the maturation disparity hypothesis, participants with the greatest disparity between physical and psychological maturation are expected to endorse

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increased internalizing symptomatology. More specifically, it is hypothesized that youth with the greatest difference between physical and psychological development—with physical being more advanced than psychological—will report greater levels of internalizing psychopathology relative to individuals with on-time physical and psychological development and individuals whose psychological development is more advanced than their physical development.

CHAPTER III

METHOD

Participants

One hundred and forty girls were recruited via flyers, targeted postcard mailing, information tables at community events, and informing key community members (e.g., teachers) of the study. Eligibility criteria were fairly broad, as participants simply had to be female and between 12 and 15 years old. Upon arrival at the laboratory, there was one participant who was age 11, one age 16, and one who self-identified as neither female nor male. These cases were removed from all analyses, leaving a community sample of 137 female adolescents from the southern US. The mean participant age was 14.32 years ($SD = 1.03$, range = 12.00-15.92). Most of the girls were in the 9th grade (36.5%) followed by 10th (22.6%), 8th (17.5%), 7th (14.6%), 6th (5.8%), and 11th (1.5%) grades. The girls primarily identified as Caucasian (73.0%), while others identified as African American (13.1%), Asian American (5.1%), or American Indian (3.6%), and approximately one-sixth of girls identified with one or more additional racial group. The sample was largely non-Hispanic/Latino (81.8%).

To get a sense of the girls' past psychological functioning, additional questions were asked about mental health history. Most of the girls denied having ever seen a mental health professional for psychological problems (78.1%), being hospitalized for reasons related to mental health (96.4%), or currently being prescribed psychotropic medication (92.7%). Of the 30 girls who endorsed having seen a mental health professional, 10 were currently in therapy. These mental health utilization rates are considerably lower than those reported by adolescents with psychological disorders (Essau, 2005; Merikangas et al., 2011).

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Procedure

After girls had expressed interest in the study, they and their parent(s)/guardian(s) were invited to the university-based research lab. Informed consent and assent were obtained and the study procedures were described. Participants were informed that the purpose of the study was to better understand adolescent development and experiences. Privacy procedures and limitations to confidentiality were reviewed. Assessments included a questionnaire packet, speech task, and an interview; however, only the questionnaires are relevant to the present study. For measures of physical development, participants were informed that they could ask to go to the restroom to examine their bodies if needed. Girls were compensated \$30 for completing the assessments.

Measures

Physical development: Pubertal status and timing. Youth physical development was assessed via two widely used measures for pubertal development and timing. The Tanner Stages (Tanner, 1962) involve presenting participants with schematic drawings of female secondary sex characteristics—breast and pubic hair, specifically—in various stages of maturity and asking participants to identify which stage best matches their current developmental level. Stages range from prepubertal (Stage I), showing no visible signs of pubertal development, to postpubertal (Stage V), which is consistent with adult maturation. Correlations between adolescent females' self-report of Tanner Stages and physical examinations, which are often considered the “gold standard” for the assessment of physical development, have demonstrated adequate convergence ($r = .52 - .88$ for breast ratings, $r = .58 - .91$ for pubic hair ratings; Brooks-Gunn et al., 1987; Dorn, Susman, Nottelmann, Inoff-Germain, & Chrousos, 1990; Morris & Udry, 1980; see Dorn,

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Dahl, Woodward, & Biro, 2006 for a review). The two Tanner items were determined to be internally reliable ($\alpha = .70$) in the present sample.

In addition to the Tanner Stages, youth physical maturation was also assessed via the Pubertal Development Scale (PDS; Petersen, Crockett, Richards, & Boxer, 1988). The PDS is a five-item self-report questionnaire on pubertal development. Participants are asked to rate changes in height, body hair, skin, and breast development and these items are rated on a 4-point scale (1 = *has not yet started changing*, 2 = *has barely started changing*, 3 = *changes are definitely underway*, 4 = *changes seem complete*¹). There is also a dichotomous yes-no item on whether girls have menstruated (coded dichotomously, 1 = *premenarcheal* or 4 = *postmenarcheal*). An overall pubertal development score is obtained by summing and averaging these five items (Petersen et al., 1988). Additionally, scores from the body hair, breast development, and menarche items (considered to be the most salient) can be used to categorize participants into one of five pubertal classifications (prepubertal, early pubertal, midpubertal, late pubertal, and postpubertal; Crockett, 1988; also see Carskadon & Acebo, 1993), which are similar to the five Tanner Stages. The PDS has demonstrated adequate psychometric properties (Carskadon & Acebo, 1993; Petersen et al., 1988; Robertson et al., 1992), and correlations between adolescent females' self-report PDS scores and physician-rated Tanner Stages ($r = .67$; Brooks-Gunn et al., 1987) demonstrate sufficient convergent validity. Cronbach's alpha on the

¹ In addition to these responses, participants could also select a fifth *I don't know* response, which has been included as an option in previous research with the PDS (e.g., Ellis, McFadyen-Ketchum, Dodge, Pettit, & Bates, 1999). The frequency of this response was fairly low ($n \leq 6$) for the body hair, skin changes, and breast development items. However, a sizable proportion of the sample ($n = 29$) selected this response for the growth spurt item. These responses were recoded as missing data in all analyses.

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four pubertal status items (on growth spurt, body hair, etc.) and the dichotomous menarche item was .71.

Participants also completed two items on pubertal timing. One item asked girls to rate the timing of their physical development relative to same-age peers, to which participants responded on a 5-point scale (1 = *much earlier*, 2 = *somewhat earlier*, 3 = *about the same*, 4 = *somewhat later*, 5 = *much later*). A second item asked participants to indicate the age at which they had their first menstrual cycle and there were six possible responses (1 = *10 years or younger*, 2 = *11 years*, 3 = *12 years*, 4 = *13 years*, 5 = *14 years*, 6 = *15 years or older*). Such pubertal timing items are common within the literature (e.g., Cance, Ennett, Morgan-Lopez, & Foshee, 2012; Carter, 2015; Caspi & Moffitt, 1991; Dubas et al., 1991; Graber et al., 1997; Harden, Mendle, & Kretsch, 2012; Hendrick et al., 2016; Kaltiala-Heino et al., 2003; Skoog et al., 2013). These two items were found to be internally reliable, as Cronbach's alpha was .67.

Social-cognitive development: Adolescent egocentrism. The Imaginary Audience Scale (IAS; Elkind & Bowen, 1979) and Personal Fable Scale (PFS; Alberts et al., 2007) were used to assess participants' level of adolescent egocentrism, which reflects their social-cognitive development. The IAS is a 12-item self-report measure that assesses adolescents' willingness to reveal aspects of the self to others and contains two subscales, each with six items: (a) the Abiding Self scale, which assesses reactions to situations in which self-revealing information may be disclosed (e.g., discussing a hobby in front of a class), and (b) the Transient Self scale, which assesses responses to potentially embarrassing situations (e.g., grease spot on clothing while at a party; Elkind & Bowen, 1979). Responses are coded on a 3-point scale from least egocentric reaction to most. Elkind and Bowen (1979) demonstrated reliability and construct validity for the IAS, and the measure was internally consistent in the present sample ($\alpha = .73$).

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Similarly, the PFS (Alberts et al., 2007) is a 12-item self-report measure with two subscales. The Invulnerability scale assesses the degree to which adolescents feel immune to negative consequences of potentially risky behaviors, while the Speciality scale relates to the uniqueness of adolescents' experiences. Example items from the former scale include: "Some kids don't worry about getting injured when they play sports" and "Some kids believe that even if they try drugs they will never get hooked on them." Example Speciality items include: "When my parents or friends tell me that they know how I feel, I don't believe that they really do" and "Sometimes when I see a good-looking girl/boy, I think that they are looking at me in a very admiring way." Participants respond to items on a 5-point Likert scale (1 = *This is never true for me* to 5 = *This is always true for me*). The PFS has demonstrated adequate internal reliability (Alberts et al., 2007). In the present sample, Cronbach's alpha for the 12 PFS items was .65.

Internalizing psychopathology. Two self-report measures were used to assess participants' internalizing psychopathology. The Revised Child Anxiety and Depression Scale (RCADS; Chorpita, Yim, Moffitt, Umemoto, & Francis, 2000) is a 47-item measure that assesses the frequency of youth anxiety and depression symptoms. The RCADS has six scales, each of which correspond to a specific diagnostic category, namely (a) major depressive disorder (e.g., "I feel sad or empty"), (b) social phobia (e.g., "I worry I might look foolish"), (c) panic disorder (e.g., "My heart suddenly starts to beat too quickly for no reason"), (d) separation anxiety disorder (e.g., "I worry about being away from my parents"), (e) obsessive-compulsive disorder (e.g., "I can't seem to get bad or silly thoughts out of my head"), and (f) generalized anxiety disorder (e.g., "I worry about things"; Chorpita et al., 2000). The anxiety scales can be combined to yield a Total Anxiety Scale while the Depression Scale can be used alone as a measure of depressive symptoms, and the sum of all scales yield a Total Internalizing Scale.

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Items are scored on a 4-point Likert scale (0 = *Never* to 3 = *Always*). The RCADS has been shown to be both valid and reliable, including convergent and discriminant validity with structured diagnostic interviews (Chorpita et al., 2000; Chorpita, Moffitt, & Gray, 2005; de Ross, Gullone, & Chorpita, 2002). The 47 RCADS items were internally reliable ($\alpha = .96$).

Participants also completed the Positive and Negative Affect Schedule for Children (PANAS-C; Joiner, Catanzaro, & Laurent, 1996; Laurent et al., 1999). The PANAS-C assesses positive and negative mood in children and adolescents. Specifically, participants are asked to indicate the extent to which they had experienced 30 single-word feelings and emotions (e.g., sad, frightened, excited, happy) during the past few weeks on a 5-point scale (1 = *Very slightly/Not at all* to 5 = *Extremely*). The measure yields both a Positive Affect and Negative Affect scale. This is an important differentiation given theory and research suggesting that both anxiety and depression are associated with elevated negative affect, but only depressed individuals also report low levels of positive affect (Clark & Watson, 1991; Watson, Clark, & Carey, 1988). The PANAS-C has demonstrated good reliability and construct validity (Joiner et al., 1996; Laurent et al., 1999; Wilson, Gullone, & Moss, 1998). The PANAS-C scales were internally reliable in the present sample, as Cronbach's alpha for the Positive and Negative Affect scales were .90 and .93, respectively.

Data Analysis

Missing data. Missing data was accounted for before measure scales were calculated, and a summary of missing data is presented in Table 2. Given that the Tanner Stages consists of only two items, if either of the items were missing, which occurred in one case, then Tanner Stage was not computed, nor was that participant's Tanner data included in the analyses. In addition, there were 15 cases with no Tanner data (i.e., missing both items). Of the 137

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participants, therefore, a total of 121 Tanner Stages were available for analysis. On the PDS, mean total scores were computed as long as three of the five PDS items were available. For example, if a participant selected the *I don't know* response and/or left an item blank, as long as 60% of the items (3 of 5) were available, then PDS mean scores were calculated from the available items. If less than 60% of the items were available for a participant, then the PDS mean score was not calculated and the participant's PDS data was not included in any analysis. There was a total of 33 cases with at least one missing PDS item and four cases with no PDS data. Of the 33 cases, only 1 case had less than 3 items available and, therefore, the PDS mean score was not calculated for this case. The PDS means were calculated for the remaining 32 cases based on available items, yielding a total of 132 cases available for analysis.

Given that IAS and PFS scales are computed by summing items (as opposed to item means), item scores were calculated for missing items as long as at least 60% of items were available. In other words, as long as 8 of the 12 IAS/PFS items were available, then the mean of the available items was entered as the value for the missing item before calculating sums. In total, there were no cases of partial IAS data, although there were 10 cases with no IAS data, leaving a total of 127 IAS total scores available for analysis. For the PFS, there was one case with partial data (missing one item) and the mean from the available 11 items was entered as the value for the missing item. There was a total of 17 cases in which either there was less than 60% of items available or there were no PFS items, yielding 120 cases for analysis.

The RCADS and PANAS-C also use sum scores in computing scale and total scores. However, given that specific scales will be used in analyses as opposed to only total scores, when entering means for missing items, means were calculated from only items on the same scale as the missing item. For instance, the RCADS Social Phobia scale has nine items and any

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missing item was computed as an average from the available items on the Social Phobia scale.

The same procedure was used for the other RCADS scales as well as for the PANSA-C Positive Affect and Negative Affect scales. As was the case with the previously discussed scales, a criterion of at least 60% of items were required to calculate missing item scores. For the RCADS, there were a total of nine cases with partial data (five cases with one missing item, three cases with two missing items, and two cases with three missing items) for which available item means from the same scale were calculated and entered in place of the missing item. There were also eight cases with no RCADS data, yielding a total of 129 cases for analysis.

Additionally, the PANAS-C had four cases with partial data, two of which were missing one item and these values were estimated by entering the mean from the available items from that scale (i.e., either the Positive Affect or Negative Affect scale). For the other two cases there were too few items available to calculate missing items, and there were also 21 cases with no PANAS-C data available. This left a total of 114 cases for use in analyses.

Analytic strategy. In order to test the first hypothesis—that early maturing girls will report greater internalizing symptoms than on-time and late developing girls—a regression analysis was used, and a Pubertal Timing variable had to be created. Although there are various ways in which this has been done in previous research (e.g., age at menarche, perceived pubertal timing item; see Dorn et al., 2006 for a comprehensive review), the Pubertal Timing variable was constructed by standardizing the level of physical maturation by age. This was accomplished by regressing pubertal status onto chronological age, yielding a continuous metric (with a mean of 0 and standard deviation of 1) in which higher scores indicate earlier pubertal timing. The Tanner Stages were used as the measure of pubertal status and were kept continuous; that is, if a participant selected Stage II of breast development and Stage III for pubic hair, for example, then

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the overall Tanner Stage was 2.50. Likewise, age as measured in years and months (e.g., an age of 15 years and 3 months would be converted to an age of 15.25 where 15 indicates the years and .25 is indicative of a quarter year or 3 months) was also kept as a continuous variable.

Standardizing pubertal status by age in this way has been a common approach to computing pubertal timing (e.g., Dorn, Susman, & Ponirakis, 2003; Ellis & Garber, 2000; Ellis et al., 1999; Rudolph & Troop-Gordon, 2010). However, additional studies (e.g., Blumenthal et al., 2011; Ge, Brody, Conger, Simons, & Murry, 2002; Ge et al., 2003, 2006; Kogan et al., 2015; Steinberg, 1987) have taken a further step and classified participants categorically by using scores above and below the mean by one standard deviation to categorize early and late developers, respectively. There were three reasons this was not done in the present study. First, this tends to be a fairly conservative approach, as only approximately 16% of participants are classified as early and 16% as late (assuming a normal distribution). As a result, other investigations (e.g., Brooks-Gunn & Warren, 1989; Stroud & Davila, 2008; Thompson et al., 2016) have used a slightly more liberal cutoff of ± 0.67 standard deviations for early and late classifications, which results in approximately 20% of the sample being early and 20% being late maturers. However, neither of these categorical approaches were used because, secondly, such thresholds are somewhat arbitrary and a continuous variable more accurately represents the phenomena (e.g., a girl could be slightly earlier or much earlier than her peers). Third, in order for Pubertal Timing to be used optimally within the proposed analysis, a continuous variable is required.

In creating the criterion variables for internalizing symptoms, theoretical and empirical information was considered. As mentioned above, both anxiety and depression are associated with elevated levels of negative affect, while only depression is associated with low levels of

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positive affect. The present data reflect this tendency (see Table 3 for correlations between internalizing scales), as the RCADS Depression Scale is most strongly related to both the Positive ($r = -.34, p < .001$) and Negative ($r = .70, p < .001$) Affect scales of the PANAS-C relative to RCADS anxiety subscales. Therefore, these three scales were converted into z-scores and averaged (with the Positive Affect scale reverse coded so that higher scores indicate less positive affect) to create a depression composite variable. Additionally, the RCADS Total Anxiety Scale, which is a sum of the five anxiety subscales from the RCADS, was used for the anxiety criterion variable. In testing the first hypothesis, therefore, Pubertal Timing was entered into two linear regression models as the predictor variable with the depression composite variable as one criterion variable and the RCADS Total Anxiety Scale as the other (hereafter referred to as the Depression and Anxiety variables, respectively).

The second hypothesis aims to evaluate the trajectory of adolescent egocentrism across the assessed age range (i.e., 12-15 years old). Given that IAS and PFS total scores were unrelated ($r = -.11, p = .24$, see Table 4 for correlations between IAS and PFS scales), the two measures were examined separately. Because adolescent egocentrism has been shown to follow an inverted-U pattern increasing to a peak in mid-adolescence (i.e., at 13.5 years of age) and then decreasing with age, analyses designed to model linear relationships would not be appropriate. Therefore, a quadratic regression was used to assess the curvilinear relationship between adolescent egocentrism and age. For the IAS, a two-step hierarchical linear regression was used with IAS total score entered as the predictor variable on the first step, and squared IAS total score entered on the second step. Age was set as the criterion variable. In addition to the regression, IAS trajectory was also assessed by using a scatter plot and checking for a quadratic line of best fit. Moreover, the PFS trajectory was not examined for three reasons. First, the

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correlation between IAS and PFS total scores was not significant, indicating that the measures are unrelated. This lack of significance suggests that the two are measuring different constructs as opposed to different aspects of the same construct (i.e., adolescent egocentrism). Second, PFS total scores were unrelated to age ($r = .10, p = .30$)—unlike the IAS total score ($r = -.19, p < .05$)—suggesting that developmental maturation may be unrelated to changes in the personal fable. Third, a large body of research indicates that imaginary audience ideation tends to be more characteristic and robust among females (Elkind & Bowen, 1979; Galanaki, 2012; Goossens et al., 1992; Gray & Hudson, 1984; Greene et al., 1996; Hauck et al., 1986; Montgomery, 2005; Richter et al., 1982; Ryan & Kuczkowski, 1994; Rycek et al., 1998), while males tend to have higher rates of personal fable ideation (Alberts et al., 2007; Galanaki, 2012; Greene et al., 1996; Goossens et al., 2002; Lapsley et al., 1989). This tendency suggests that imaginary audience scores may be a more accurate measure of adolescent egocentrism in females relative to males. For the above reasons, the trajectory of personal fable across the assessment age range was not assessed, and imaginary audience (measured via the IAS) was used as the sole measure of adolescent egocentrism.

Finally, linear regression models were used to test the maturation disparity hypothesis. Three predictor variables were entered into each regression model. The first was the Pubertal Timing variable described above. The second was a Maturation Discrepancy variable, which was a difference variable created in order to account for discrepancies between physical and psychological maturation. Given the correlation between age and IAS total scores ($r = -.19, p < .05$) indicated that older participants had lower IAS scores, the IAS total scores were reverse coded, making higher IAS scores suggestive of less adolescent egocentrism and greater psychological development. This reverse coding was conducted to align the direction of the IAS

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with the PDS (hence, higher scores on both the IAS and PDS reflect more advanced development) so that a difference score could be calculated. PDS mean scores and the reverse coded IAS sum scores were converted to z-scores to set them on equivalent scales, and then the IAS z-scores were subtracted from the PDS z-scores. This yielded a variable representing differences in physical and psychological development, with higher (and positive) scores indicating that physical development was more advanced than psychological development and lower (and negative) scores indicating the opposite. Finally, an interaction term was created by multiplying Pubertal Timing by Maturation Discrepancy. These three constructs—Pubertal Timing, Maturation Discrepancy, and their interaction—were force entered into two linear regression models as predictors, while the Anxiety and Depression variables (described above) were entered as criterion variables.

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CHAPTER IV

RESULTS

Sample Characteristics

Descriptive and distributional properties of measures and scales are presented in Table 5. The Tanner and PDS scores suggest that participants were, on average, fairly far along in the pubertal transition, as mean Tanner and PDS Stages were 3.95 ($SD = 0.76$) and 4.05 ($SD = 0.67$), respectively, although average PDS scores were slightly lower ($M = 3.23$, $SD = 0.63$). The perceived pubertal timing item suggested that a majority of girls considered their pubertal timing to be on-time ($M = 3.02$, $SD = 1.04$ on a scale in which 3 = *about the same* in reference to relative timing of maturation), while the age at menarche item indicated that most girls experienced menarche between 11 and 13 years old ($M = 2.93$, $SD = 1.11$ where 2 = 11 years, 3 = 12 years, and 4 = 13 years). On nearly every metric of physical development, the entire scale range was represented by the sample. For instance, on the four PDS items related to changes in height, body hair, skin, and breast development, participant responses ranged from *has not yet started changing* to *changes seem complete*, which encompasses the entire scale. This indicates that, although mean physical development indices suggest that participants had fairly advanced pubertal statuses, the sample reflects a wide spectrum of physical development within the pubertal transition. Additionally, RCADS means and standard deviations in the present sample are comparable to other adolescent community samples (Chorpita et al., 2000; de Ross et al., 2002) and, similarly, PANAS-C descriptives were also similar to those obtained from normative samples (Laurent et al., 1999; Wilson et al., 1998).

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Hypothesis 1: Pubertal Timing and Internalizing Outcomes

In order to test the first hypothesis—that early pubertal timing would be associated with greater levels of internalizing symptoms—Pubertal Timing was entered into two linear regression models with the Anxiety variable as one criterion and the Depression composite variable as the other. In other words, simple linear regressions were used to predict Anxiety and Depression scores from Pubertal Timing. The results of the first regression, with Anxiety as the criterion variable, was not statistically significant, $F(1, 117) = 0.83, p = .36$, with an R^2 of .01. The model was expressed as: $\text{Anxiety} = 1.75 * \text{Pubertal Timing} + 35.04$. Pubertal Timing did not significantly predict the RCADS Total Anxiety Scale, $t = 0.91, p = .36$. Therefore, earlier pubertal timing was not significantly associated with higher rates of anxiety psychopathology as predicted.

Moreover, the results of the second regression model using Depression as the criterion variable was marginally statistically significant, $F(1, 118) = 3.92, p = .05$, with an R^2 of .03. The regression equation was: $\text{Depression} = 0.14 * \text{Pubertal Timing} + 0.00$. Pubertal Timing was marginally predictive of the Depression composite variable (i.e., z-score sums of the RCADS Depression Scale and the PANAS-C Positive [reverse coded] and Negative Affect Scales), $t = 1.98, p = .05$. Thus, earlier pubertal timing was marginally predictive of the Depression composite variable. However, using only the RCADS Depression Scale as the criterion variable, a statistically significant regression was achieved, $F(1, 117) = 8.09, p < .01$, with an R^2 of .07. This equation can be expressed as: $\text{RCADS Depression Scale} = 1.54 * \text{Pubertal Timing} + 8.41$. Pubertal Timing, in this case, was found to be a significant predictor of the RCADS Depression Scale, $t = 2.84, p < .01$. It can be concluded, therefore, that the data provided mixed support for the first hypothesis. Specifically, earlier pubertal timing was not predictive of greater levels of

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anxiety symptoms, but was marginally predictive of the Depression composite variable and significantly predictive of the RCADS Depression Scale.

Hypothesis 2: Trajectory of Imaginary Audience

The second hypothesis predicts that IAS scores will follow an inverted-U trajectory across the assessment age range, peaking at approximately 13.5 years old. Mean IAS total scores as well as the Abiding Self and Transient Self subscales demonstrate an overall decreasing trajectory across both the assessment age and grade range (see Table 6). More specifically, scores peak at 12 years old, decline at 13 years old, and then show a slight decline at 14 and 15 years old. Similarly, scores peak at 6th grade, show a marked decrease at 7th grade, stay relatively stable between 8th and 10th grade, and then decline again at 11th grade (it should be noted, however, that limited data exists for 11th grade as $n = 2$).

Additionally, IAS total scores were entered into a two-step hierarchical linear regression with age entered as the criterion variable. In the first step, IAS total scores were entered as the predictor variable and age as the criterion variable. This yielded a regression model that was significant, $F(1, 125) = 4.68, p < .05$, with an R^2 of .04. The regression equation was: Age = $14.89 - 0.05$ (IAS). IAS total score was found to be a significant predictor of age, $t = -2.16, p < .05$. In the second step, IAS total scores were squared and entered as the predictor variable with age again as the criterion. The purpose for the second step is to explain additional variance above what is accounted for by the predictor variable in the original regression. Although approaching statistical significance, this regression model was not significant, $F(2, 124) = 2.33, p = .10$, with an R^2 of .04. The regression equation was: Age = $14.83 - .04$ (IAS) + 0.00 (IAS²). In this case, neither IAS ($t = -0.31, p = .76$) nor IAS² ($t = -0.12, p = .91$) were significant. There was also no change in R^2 between the two regression models. This suggests that no additional

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variance was accounted for in the second regression relative to the variance accounted for by the first model.

To further test this hypothesis, a scatter plot was created with age and IAS total scores entered as variables (see Figure 1). The quadratic R^2 was .06. Contrary to the hypothesis, the quadratic line of best fit demonstrated a U-shaped trajectory across the assessment age range with approximately 15 years of age representing the lowest point. This trajectory is inconsistent with the predicted trajectory as well as with the research outlined above.

Hypothesis 3: The Maturation Disparity Hypothesis

The final and primary hypothesis aims to assess the maturation disparity hypothesis as an explanation for the early pubertal timing-internalizing psychopathology link. Specifically, should the hypothesis be supported, a greater discrepancy between physical and psychological maturation—with physical being more advanced than psychological—should be predictive of greater internalizing symptoms. Again, two separate multiple regressions were used to examine the impact of this relationship on anxiety and depression separately. For the first multiple regression, Pubertal Timing, Maturation Discrepancy, and their interaction were entered as predictors. Entering the Anxiety variable as the criterion, a significant proportion of variance was accounted for, $F(3, 112) = 4.28, p < .01$, with an R^2 of .10. The model was expressed as: Anxiety = 33.81 – 0.21 (Pubertal Timing) + 5.25 (Maturation Discrepancy) + 2.24 (interaction term). Although the regression model was significant, only the Maturation Discrepancy variable was found to be a significant predictor, $t = 3.33, p < .01$, while Pubertal Timing ($t = -0.10, p = .92$) and the interaction term ($t = 1.70, p = .09$) were not significant, yet the latter did approach statistical significance. Therefore, this analysis indicates that a greater discrepancy between physical and psychological maturities—with physical being more advanced than

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psychological—was predictive of anxiety symptoms, although neither Pubertal Timing nor the interaction term were significant predictors of anxiety levels.

Next, a multiple regression analysis was run with the same three predictors as above, but the Depression composite variable was entered as the criterion rather than Anxiety. Again, a significant proportion of variance was accounted for, $F(3, 112) = 6.55, p < .001$, with an R^2 of .15. The regression formula was: Depression = 5.68 + 0.03 (Pubertal Timing) + 0.23 (Maturation Discrepancy) + 0.01 (interaction term). Despite the regression being significant, again only the Maturation Discrepancy variable was a significant predictor, $t = 3.91, p < .001$, while neither Pubertal Timing ($t = 0.38, p = .70$) nor the interaction term ($t = 0.25, p = .80$) were significant predictors. Furthermore, entering the RCADS Depression Scale as the criterion variable also yielded a significant regression, $F(3, 112) = 6.61, p < .001$, with an R^2 of .15. The model was expressed as: RCADS Depression Scale = 8.14 + 0.98 (Pubertal Timing) + 1.46 (Maturation Discrepancy) + 0.58 (interaction term). In this case, Maturation Discrepancy was again a significant predictor ($t = 3.28, p < .01$), while Pubertal Timing was marginally significant ($t = 1.72, p = .09$) and the interaction term approached statistical significance ($t = 1.56, p = .12$). This set of regressions suggests that the difference between physical and psychological development accounts for much of the variance in depression outcome scores. However, in the latter regression, both Pubertal Timing and the interaction score approached significance. This suggests that, in addition to Maturation Discrepancy being a significant predictor, both earlier pubertal timing and the interaction of pubertal timing by more advanced physical than psychological development predicted depression symptoms in the direction that supports the maturation disparity hypothesis.

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CHAPTER V

DISCUSSION

The primary purpose of the present study was to empirically evaluate the maturation disparity hypothesis. Additional aims included testing whether early maturing girls would report higher levels of internalizing symptoms compared with on-time and late developing girls, and assessing the trajectory of imaginary audience scores across the assessment age range. The maturation disparity hypothesis was partially supported and, in a similar vein, early pubertal timing was associated with greater levels of depression while not being significantly related to increases in reported anxiety. Finally, imaginary audience scores did not follow an inverted-U trajectory as expected.

The Maturation Disparity Hypothesis

The maturation disparity hypothesis attempts to explain the link between early pubertal timing and increases in psychopathology by positing that early maturers' physical development is more advanced than their psychological development, and it is this disconnect that places them at greater risk for adverse outcomes relative to either on-time or late developers. In order to test this explanation, it was hypothesized that participants with a greater difference between their physical and psychological development—with physical being more advanced than psychological—would report greater levels of anxiety and depression. For both anxiety and depression, results suggested that such a discrepancy was related to higher levels of internalizing symptoms; that is, those with more advanced physical relative to psychological development did report greater levels of anxiety and depression. Although this supports the maturation disparity hypothesis, pubertal timing was not related to anxiety outcomes, nor was pubertal timing predictive of the Depression composite variable. This finding in concert with physical and

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psychological discrepancy being a significant predictor of internalizing outcomes suggests partial support for the maturation disparity hypothesis. Not only should maturation discrepancy predict anxiety and depression, but pubertal timing should also be predictive because the hypothesis suggests that early developers are at an increased risk *because of* the discrepancy between their physical and psychological development. The present findings suggest that pubertal timing is not related to internalizing symptoms, while the discrepancy is a strong predictor of anxiety and depression.

Furthermore, additional support for the maturation disparity hypothesis was found when using the RCADS Depression Scale as the criterion variable as opposed to the composite Depression variable (consisting of scales from both the RCADS and the PANAS-C). Specifically, the maturation disparity variable (representing the difference between physical and psychological development) was again a significant predictor, while both pubertal timing and the interaction between pubertal timing and maturation disparity approached significance. This finding suggests that in addition to the variance accounted for by the maturation disparity variable, both pubertal timing and the interaction term may explain additional variance in depression outcomes (when measured by only the RCADS Depression Scale). It should be noted that had there been more participants in the present sample it is possible that both pubertal timing and the interaction would have achieved statistical significance, which would have supported the maturation disparity hypothesis in explaining the link between early pubertal timing and increases in depressive outcomes. Additionally, it is also possible that using a sample with a broader range of pubertal status, as the present sample was, on average, fairly far along in the pubertal transition (see Table 5), would have led to an increase in statistical power and statistical significance for all three terms.

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The reason for stronger findings with the RCADS Depression Scale as the criterion variable as opposed to the composite variable is unclear. As discussed, theory and research indicates that negative affect is associated with both anxiety and depression, but depressed individuals also report lower levels of positive affect while anxious individuals do not (Clark & Watson, 1991; Watson et al., 1988), suggesting that positive affect may be useful in differentiating anxious and depressive distress. The present study supported this notion, as the PANAS-C Positive Affect Scale was most strongly (negatively) correlated with the RCADS Depression Scale relative to any of the RCADS anxiety scales and the PANAS-C Negative Affect Scale (see Table 3). One potential explanation for the differences found between the RCADS Depression Scale and the Depression composite variable is that the former includes items on both physical and psychological changes associated with depression, whereas the composite variable relies more heavily on affective changes associated with depression.

Early Pubertal Timing and Internalizing Psychopathology

Congruent with previous research, it was also expected that early pubertal timing would be predictive of higher rates of anxiety and depression. However, earlier pubertal timing was not found to be associated with anxiety outcomes. This is inconsistent with a large number of studies that demonstrate such a link with females (e.g., Blumenthal, Leen-Feldner, Babson, et al., 2011; Blumenthal, Leen-Feldner, Trainor, et al., 2009; Deardorff et al., 2007; Weingarden & Renshaw, 2012; Zehr et al., 2007). Even in a literature review, Reardon and colleagues (2009) concluded that the most prominent and consistent finding among the pubertal timing and anxiety research is that early maturing females are at an increased risk for anxiety symptoms and disorders. The reason this finding was not replicated with the present sample is unclear.

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On the other hand, having an earlier puberty reliably predicted depressive symptoms. Specifically, when using the RCADS Depression Scale as the criterion variable, earlier pubertal timing was a significant predictor, and when using the Depression composite variable earlier pubertal timing was marginally significant ($p = .05$). In the latter case, it is again possible that a larger sample size would have yielded a statistically significant association. Likewise, it is also possible that participants representing a wider spectrum of pubertal status would have yielded a similar result. These results are consistent with a large body of empirical research indicating that early puberty among females increases risk for depressive outcomes (Ge et al., 2001; Ge et al., 2003; Keenan et al., 2014; Kaltiala-Heino, Kosunen, et al., 2003; Kaltiala-Heino, Marttunen, et al., 2003; Mendle et al., 2010) as well as the conclusions of two literature reviews (Mendle et al., 2007; Negriff & Susman, 2011) and a meta-analysis (Galvao et al., 2014). Given that these results are consistent with previous research—unlike the anxiety-related outcomes—this may suggest that depression, in this particular case, may be a more appropriate criterion variable for adequately testing the maturation disparity hypothesis, further strengthening the support for the hypothesis outlined above.

Although the maturation disparity hypothesis may be the most widely accepted (yet least tested) explanation for the early pubertal timing-psychopathology link (Ge & Natsuaki, 2009), there are additional explanations that have been proposed. In light of the strongest evidence provided by the present study for the maturation disparity hypothesis (i.e., model with RCADS Depression Scale as the criterion), it may be beneficial to discuss implications for these alternative explanations. The hormonal influence hypothesis states that the rise in pubertal hormones results in increased vulnerabilities to internalizing symptoms and disorders for early physical maturers as (a) less developed neurological systems may be more sensitive to these

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hormones and/or (b) more hormones are released during the pubertal transition for early relative to later developers (Ge & Natsuaki, 2009; Vaughan et al., 2015). The present findings do not necessarily contradict the hormonal influence hypothesis and, conversely, may add some support to this explanation. Specifically, it may be possible that individuals with more advanced physical than psychological development are especially sensitive to and/or produce more pubertal hormones across the pubertal transition. This may appear counterintuitive given that the maturation disparity hypothesis posits that physical development is *more advanced* than psychological among those at increased risk while one explanation of the hormonal influence hypothesis maintains that a *less advanced* neurological system may be more affected by pubertal hormones; however, the former hypothesis emphasizes more advanced outward physical development while the latter posits that internal physical structures are less advanced. This is an important distinction given that these internal structures—namely, the brain—give rise to one’s psychological functioning, which is less advanced for those at risk according to the maturation disparity hypothesis, so a less developed brain gives rise to less sophisticated psychological functioning (e.g., decision making, emotion regulation). Therefore, these two hypotheses may be particularly compatible, and may both be supported by the present findings, although stronger and more direct support exists for the maturation disparity hypothesis.

The contextual amplification hypothesis offers another explanation and postulates that increased risk of psychopathology stems from the interaction between early pubertal timing and challenging social contextual factors, such as interpersonal and environmental stress (Ge & Natsuaki, 2009). Like the hormonal influence hypothesis, the contextual amplification hypothesis can also be conceptualized as being consistent with the maturation disparity hypothesis as well as the present findings. The inability to successfully navigate challenging

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contextual factors may be the result of one's physical development being more advanced than psychological development, as the former may place individuals in these challenging scenarios while the latter makes it less likely to successfully manage these situations. For example, an early developing female may appear to be mature and, as a result, adults and peers may assign her increased responsibility, leading to challenging social situations that she is not psychologically prepared to handle and cannot successfully manage. The support for the maturation disparity hypothesis demonstrated by the present study—albeit with some caveats—is not incompatible with the context amplification hypothesis.

The final hypothesis proposed to explain the early pubertal timing-psychopathology link is the accentuation hypothesis and states that past maladjustment is amplified by challenging transitional periods, with one example being early physical development. This explanation is less consistent with the maturation disparity hypothesis and present findings insofar as it emphasizes previous psychological and behavioral difficulties as the source of future psychopathology and posits that early pubertal timing magnifies the former and results in the latter. It is less clear how a physical and psychological maturation disparity would be relevant or integrative with the accentuation hypothesis. Thus, the present findings do not appear to support or contradict this explanation.

The possibility that the maturation disparity hypothesis and the present findings may also be compatible with the hormonal influence hypothesis and the contextual amplification hypothesis is significant. The ways in which these hypotheses are compatible result in challenges when attempting to devise methodology and approaches aimed at evaluating them individually or concurrently. Additionally, it is also possible that each of these three explanations account for the increase in psychopathology associated with earlier puberty; in

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other words, they are not inherently mutually exclusive. That is, hormonal vulnerabilities among early developers (hormonal influence hypothesis) could be a precursor to the relevance of being more physically than psychologically developed (maturation disparity hypothesis), which could then result in an increase in negative outcomes associated with unsuccessfully managing challenging contextual factors (contextual amplification hypothesis). This possibility could help explain why there is now some empirical support for each of these hypotheses, given previous research in concert with the present study.

Trajectory of Imaginary Audience Ideation

The second hypothesis of the study aimed to assess the trajectory of adolescent egocentrism. It was determined, however, to focus on the imaginary audience component of adolescent egocentrism for reasons outlined above. Consistent with past research as well as Elkind's (1967) theory, it was expected that imaginary audience scores would follow an inverted-U course across the assessment age range, peaking at approximately 13.5 years old (8th grade). This hypothesis was not supported. Imaginary audience scores demonstrated an overall decline by both age (12-15 years old) and grade (6th-11th grade). Although the Abiding Self Subscale by age and the Transient Self Subscale by age and grade showed slight increases (for example, mean Transient Self scores were 4.55, 4.67, and 4.34 at 13, 14, and 15 years old, respectively; see Table 6), these differences are not reflected in total scores and so small that making any meaningful interpretation is questionable. Also, the regression and scatter plot did not support an inverted-U trajectory. These findings are inconsistent with Elkind's (1967) theory, which maintains that imaginary audience ideation should increase across adolescence, and then start to decrease at 15 or 16 years old. Some research supports this notion, demonstrating an inverted-U trajectory of imaginary audience scores across adolescence (Elkind

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& Bowen, 1979; Ryan & Kuckowski, 1994; see Table 1); however, Ryan and Kuckowski (1994) only found statistical significance between grades 7, 8, and 9 and 12th grade, while finding no significant differences between 7th, 8th, and 9th grade scores. Additional studies report that imaginary audience scores do not differ by age among females (Galanaki, 2012; Gray & Hudson, 1984; Jahnke & Blanchard-Fields, 1993; Lapsley et al., 1986; Montgomery, 2005; Richter et al., 1982). Adams and Jones (1981), on the other hand, found an increase in imaginary audience scores across adolescence, while Enright and colleagues (1979, 1980) as well as Lechner and Rosenthal (1984) found results similar to the present findings, as imaginary audience scores decreased from early to late adolescence.

Although inconsistent findings related to the developmental trajectory of imaginary audience ideation across adolescence may be partially explained by the use of different measurement tools or even generational effects, for example, additional problems exist for Elkind's (1967) theory of adolescent egocentrism. For instance, Elkind proposed that the onset of adolescent egocentrism coincides with the inception of formal operational cognitive abilities, while this egocentrism begins to decline once formal operations have become firmly established in mid- to late adolescence (at 15 or 16 years old). However, several investigations assessing the relationship between formal operational abilities and the onset or decline of adolescent egocentrism (or imaginary audience) have not found this tenet of Elkind's theory to be empirically supported (Goossens, 1984; Gray & Hudson, 1984; Jahnke & Blanchard-Fields, 1993; Kelly et al., 2002; Lapsley et al., 1986; O'Connor & Nikolic, 1990; Riley, Adams, & Nielsen, 1984; Rycek et al., 1998), although there are at least two studies that report partial support (Galanaki 2012; Gray & Hudson, 1984). Commenting on the shortcomings of Elkind's

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theory, Lapsley (1993) concludes that there are “grave doubts that formal operations have much to do with the formulation of adolescent personality” (p. 565).

In light of inconsistent findings and limited empirical support, alternative adolescent egocentrism theoretical frameworks have been proposed (see Vartanian, 2000 for a review). Lapsley and colleagues (Lapsley, 1993; Lapsley et al., 1989; Lapsley & Rice, 1988) offer the most prominent example with their “new look” model, which conceptualizes the imaginary audience and personal fable within the context of identity development. Specifically, separation-individuation, wherein the goal for an adolescent is to simultaneously maintain family relationships while also establishing an identity independent from the family, is aided by the imaginary audience and personal fable by conceptualizing these two ideation patterns as “interpersonally-oriented daydreaming” (Vartanian, 2000, p. 647). The form of this daydreaming may be fantasizing about being the focus of a social situation (imaginary audience), as this allows adolescents to preserve feelings of interpersonal closeness while distancing from family, or as unique, omnipotent, and invulnerable (personal fable), which highlights one’s individuality independent of family. Unlike Ekland (1967), therefore, the imaginary audience and personal fable are not failures of differentiation, but adaptive coping mechanisms that aid adolescents in separating from parents and achieving an individual identity. Various aspects of the “new look” theory have received empirical validation (Galanaki & Christopoulos, 2011; Goossens et al., 2002; Jahnke & Blanchard-Fields, 1993; Lapsley et al., 1989; O’Connor, 1995; O’Connor & Nikolic, 1990; Ryan & Kuczowski, 1994; Vartanian, 1997) and overcome major shortcomings of Elkind’s (1967) conceptualization, such as limited evidence related to the role of formal operations in adolescent egocentrism. These limitations are addressed by integrating Selman’s (1980) developmental theory of social perspective-taking. Specifically, Lapsley and

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Murphy (1985) suggest that the imaginary audience and personal fable may be consequences of cognitive developments first possible in Selman's Level 3 perspective taking, in which a third-party perspective is achieved, and then start to diminish at the onset of Level 4 perspective taking, wherein a generalized societal perspective is possible. As highlighted by Lapsley (1993), "separation-individuation will commence whenever an adolescent reaches a certain *social cognitive developmental* [emphasis added] understanding of the self, say, at Selman's (1980) Level 3" (p. 569). Importantly, and as emphasized here, this alternative model maintains the notion that the imaginary audience and personal fable are features of adolescent social cognitive development. Therefore, the imaginary audience is still appropriate for the present purposes—as a measure of social cognitive development or, in other words, the level of adolescent psychological maturation.

Although the trajectory of imaginary audience did not follow an inverted-U course as expected and present findings are inconsistent with Elkind's (1967) theory as well as some previous research (e.g., Elkind & Bowen, 1979; Ryan & Kuckowski, 1994), these findings are congruent with Lapsley's (1993) "new look" model. Specifically, the theory posits that the onset of imaginary audience and personal fable ideations coincide with Level 3 of Selman's (1980) social perspective-taking theory, while their decline is associated with Level 4. Given this framework, it would be expected for these ideation patterns to decline as adolescents become more socially and cognitively sophisticated. According to Selman (1980) and others (e.g., Gurucharri & Selman, 1982), youth that are approximately 10-15 years old are typically in Level 3 and individuals 12 years and older are generally considered to be in Level 4. Despite the fairly broad and overlapping age ranges, these figures fit well with the present findings and may help

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explain the rather pronounced decrease in imaginary audience scores between 12 and 13 year olds (see Table 6).

Clinical Application

As previously outlined, internalizing symptoms and disorders carry immense financial, physical, and psychosocial costs. For these reasons, it is crucial to have effective prevention, assessment, and treatments for these conditions. Although helpful assessments, such as the Beck inventories (i.e., Beck Anxiety Inventory, Beck Depression Inventory-II; Beck & Steer, 1990; Beck, Steer, & Brown, 1996), and effective psychological and pharmacological interventions (e.g., CBT, SSRIs; Dubicka et al., 2010; Ebert et al., 2015; Kendall, 1994; Melvin et al., 2006; Walkup et al., 2008) exist, assessments that identify at-risk individuals and more targeted, individualized treatments are needed. The present study illustrates the importance of physical and psychological development in identifying internalizing outcomes, with implications for prevention, assessment, and intervention. For example, a brief screener could be used in early-adolescence (or late childhood) to identify youth with more advanced physical than psychological developments with the aim of preventing or mitigating later maladjustment. Additionally, treatments specifically aimed at advancing an adolescent's psychological maturation may be helpful in preventing or reducing anxious and depressive distress. Given that the present study partially supports the maturation disparity hypothesis and this hypothesis maintains that physically mature but psychologically immature adolescents may not possess the psychological sophistication to successfully manage situations as a result of their advanced physical development, a treatment that promotes psychological development could be beneficial. Such an approach, for instance, might aim to increase social skills, emotion regulation abilities, and/or executive functioning. Existing therapies such as emotion regulation therapy (Mennin,

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2004; 2006) and dialectal behavior therapy adolescent skills groups (Rathus & Miller, 2015) may offer avenues for meeting these goals. Psychoeducation on the significance of discrepancies in physical and psychological development may also be advantageous. Such an understanding may help increase an adolescent client's buy-in to the therapeutic process, reducing attrition and increasing effectiveness. In sum, the present findings suggest that these clinical applications warrant further consideration and investigation.

Strengths and Limitations

The most notable strength of the present investigation is that it provides the first empirical test of the maturation disparity hypothesis. Although Thompson and colleagues (2016) recently claimed to have found support for the maturation disparity hypothesis through studying the effects of pubertal synchrony, reasons highlighted above suggest that their approach was inadequate. The fact that no other studies have directly evaluated this hypothesis is salient given its wide acceptance (Ge & Natsuaki, 2009). Thus, the present study contributes significantly to the pubertal timing-psychopathology literature, despite only finding partial support for the hypothesis. The primary reason others have not tested the maturation disparity hypothesis is due to challenges associated with measuring an adolescent's psychological maturation. The present investigation, therefore, also offers researchers a possible avenue for assessing adolescent psychological development within this context. Additional strengths include the use of concurrent measures of pubertal development (as opposed to retrospective) as well as multiple items and measures in the creation of constructs (e.g., Maturation Discrepancy variable).

The present study is also not without its limitations. Likely the most salient limitation is the inability of the present investigation to find support for one explanation for the early pubertal timing-psychopathology connection while also simultaneously refuting competing explanations.

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Although explicit (partial) support was found for the maturation disparity hypothesis (unlike the other hypotheses) and some integrative explanation cannot be ruled out, a research paradigm that evaluated each explanation, while being able to distinguish them or support an integrative framework, would be especially compelling. Additionally, and as previously mentioned, the sample size was somewhat small given the age range of interest and participants were on average fairly far along in the pubertal transition. Had the sample size been larger or participants represented a wider range of pubertal status, it is possible that marginally significant findings would have become statistically significant if the sample were appropriately powered. Another limitation is the exclusive reliance on self-report data collection. The design would have been strengthened had multiple informants and collection methods been used, such as physician ratings of pubertal development or parent-report of adolescent internalizing symptoms. The present sample was also quite racially and ethnically homogenous, as nearly three-fourths identified as Caucasian and over four-fifths as non-Hispanic/Latino. A more diverse sample would have improved the external validity of the findings. Finally, some research (e.g., Cohn et al., 1988; Goossens et al., 2002; Kelly et al., 2002; Ryan & Kuczkowski, 1994) suggests that the imaginary audience is related to increases in anxiety and depression. Such a relationship would confound the present findings; however, had imaginary audience scores had a strong influence on internalizing outcomes, it is unlikely that pubertal timing would have been marginally significant ($p = .09$) when testing the maturation disparity hypothesis with the RCADS Depression Scale as the criterion variable. That is, if imaginary audience scores accounted for large amounts of variance in anxiety and depression rates, then it is unlikely that terms other than the Maturation Disparity variable would have been marginally significant. Also, additional

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studies have not found a relationship between imaginary audience scores and internalizing outcomes (e.g., Beaudoin & Schonert-Reichl, 2006; Lechner & Rosenthal, 1984).

Future Directions

Future studies should aim to address the specific limitations of the present investigation. It will also be crucial to develop research designs that test the different explanations for the pubertal timing-psychopathology link. As mentioned, three of the four explanations can be integrated together and each may contribute to the increase in internalizing outcomes associated with earlier pubertal timing among female adolescents. An ideal research design, therefore, must be specific enough to validate one hypothesis while not simultaneously validating others; however, devising such an approach is challenging given the integrative possibilities of the hypotheses. Despite these challenges, results that successfully distinguish between explanations, or support an integrative model, would significantly advance this body of research and carry noteworthy implications for applied settings.

Research should also aim to further elucidate gender differences within the pubertal timing-psychopathology literature. Gender differences in pubertal timing outcomes are well established (see, for example, Graber et al., 1997; Graber, Seeley, Brooks-Gunn, & Lewinsohn, 2004; Mendle et al., 2007; Mendle & Ferrero, 2012). However, hypotheses that attempt to explain the link between early pubertal timing and psychopathology fail to discriminate between genders; that is, explanations aim to account for the link uniformly for girls and boys. Considering intrinsic gender differences in puberty (e.g., some primary and secondary sex characteristics, girls being pubertal pioneers, various gender-specific social implications), such explanations may be inadequate. Therefore, gender-specific explanations seem warranted. It is also important to note that these explanations are specifically for psychopathology associated

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with early pubertal timing. Given research suggesting that late maturing boys are at risk for adverse outcomes (Graber et al., 2004; Mendle & Ferrero, 2012) as well as some findings suggesting that late developing girls are at risk for internalizing symptoms (Carter et al., 2009; Weingarden & Renshaw, 2012), frameworks that aim to explain maladjustment as a result of late development are also needed. Finally, explanations may need to be gender- *and* timing-specific (i.e., gender X timing), as it is not inherently true that one model must unify all findings across both gender and pubertal timing. The level of inconsistency in the pubertal timing- psychopathology research may make such approaches especially justified.

An additional avenue for future study includes an exploration of alternative psychological development measures. Although the IAS appears to be one such measure and its brevity makes it suitable for applied settings, alternative approaches may offer additional empirical and clinical utility. Measures of emotion regulation and executive functioning, for example, may be integrated to assess an adolescent's psychological maturation, and then prevention and treatment approaches could be designed and implemented to specifically target areas of deficiency.

Conclusion

The present study provides the first direct, empirical test of the maturation disparity hypothesis for explaining the early pubertal timing-psychopathology link. Partial support was found for the hypothesis, and clinical application as well as study strengths and limitations are highlighted. There are a number of areas ripe for future investigation. Despite various conceptual and methodological challenges, pubertal research remains a domain worthy of attention and resources. Given the degree to which developmental change occurs during adolescence, this period offers unique, pragmatic, and opportunistic possibilities for prevention and intervention. Indeed, adolescence represents a pivotal crossroads between childhood and

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adulthood, and a more complete understanding of this often difficult transitional period could significantly improve the lives and wellbeing of countless individuals.

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Table 1

Summary of Mean Imaginary Audience Scale Scores for Females by Grade

	Imaginary Audience	Abiding Self Subscale	Transient Self Subscale
Elkind & Bowen (1979)			
4th Grade ($M = 9.40$ yo)	11.32	5.90	5.42
6th Grade ($M = 11.90$ yo)	12.00	6.53	5.47
8th Grade ($M = 13.70$ yo)	13.78	7.45	6.33
12th Grade ($M = 17.70$ yo)	10.48	5.73	4.75
Ryan & Kuczkowski (1994) ^a			
7th Grade	18.47	10.74	8.76
8th Grade	20.02	11.07	8.95
9th Grade	19.87	10.99	8.88
12th Grade	19.00	10.11	8.89
Peterson & Roscoe (1991) ^b			
College Freshman ($M = 17.6$ yo)	12.37	6.81	5.56
Rycek et al. (1998) ^b			
College Students ($M = 19.6$ yo)	11.21	6.19	5.11

Note. M = mean; yo = years old. These studies suggest that imaginary audience follows an inverted-U pattern across adolescence, with a peak in 8th grade and another, less prominent spike during the first year of college.

^a Ryan & Kuczkowski (1994) did not report mean participant ages.

^b The Peterson & Roscoe (1991) and Rycek et al. (1998) studies used a slightly modified version of the IAS that was suited specifically for college students.

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Table 2

Summary of Missing and Available Cases

Measure	Total Number of Cases	Number of Missing Cases	Number of Cases with Too Few Items to Estimate Missing	Total Number of Cases Available for Analysis
Tanner Stage	137	15	1	121
PDS	137	4	1	132
IAS	137	10	0	127
PFS	137	15	2	120
RCADS	137	8	0	129
PANAS-C	137	21	2	114

Note. PDS = Pubertal Development Scale; IAS = Imaginary Audience Scale; PFS = Personal Fable Scale; RCADS = Revised Child Anxiety and Depression Scale; PANAS-C = Positive and Negative Affect Schedule for Children.

MATURATION DISPARITY HYPOTHESIS

Table 3

Correlations between Internalizing Outcomes

Measure/Scale	1	2	3	4	5	6	7	8	9	10
1. RCADS SP	—									
2. RCADS PD	.63**	—								
3. RCADS MDD	.62**	.69**	—							
4. RCADS SAD	.61**	.69**	.57**	—						
5. RCADS GAD	.64**	.62**	.56**	.66**	—					
6. RCADS OCD	.63**	.62**	.66**	.66**	.66**	—				
7. RCADS Total Anxiety	.86**	.86**	.74**	.83**	.83**	.83**	—			
8. RCADS Total Internalizing	.85**	.86**	.84**	.81**	.81**	.83**	.99**	—		
9. PANAS-C Positive Affect	-.24*	-.16	-.34**	-.15	-.09	-.11	-.19	-.23*	—	
10. PANAS-C Negative Affect	.54	.57**	.70**	.52**	.46**	.46**	.61**	.66**	-.21*	—

Note. RCADS = Revised Child Anxiety and Depression Scale; SP = social phobia; PD = panic disorder; MDD = major depressive disorder; SAD = separation anxiety disorder; GAD = generalized anxiety disorder; OCD = obsessive-compulsive disorder; PANAS-C = Positive and Negative Affect Schedule for Children.

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

MATURATION DISPARITY HYPOTHESIS

Table 4

Imaginary Audience Scale (IAS) and Personal Fable Scale (PFS) Correlations

Measure/Scale	1	2	3	4	5	6
1. IAS Abiding Self	—					
2. IAS Transient Self	.34**	—				
3. IAS Total	.83**	.81**	—			
4. PFS Invulnerability	-.10	-.09	-.12	—		
5. PFS Speciality	-.17	.09	-.06	.37**	—	
6. PFS Total	-.16	-.02	-.11	.88**	.76**	—

Note. IAS = Imaginary Audience Scale; PFS = Personal Fable Scale.

** $p < .001$.

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Table 5

Descriptive and Distributional Properties of Predictor and Outcome Measures

Measure/Scale	<i>M</i> (<i>SD</i>)	Range	Skewness	Kurtosis
Tanner Stages				
Breast Development	3.82 (0.83)	2.00 – 5.00	-0.17	-0.65
Pubic Hair	4.09 (0.89)	1.00 – 5.00	-0.84	0.46
Tanner Stage	3.95 (0.76)	1.50 – 5.00	-0.58	0.20
Pubertal Development Scale (PDS)				
Growth Spurt	3.17 (1.04)	1.00 – 4.00	-1.05	-0.14
Body Hair	3.45 (0.81)	1.00 – 4.00	-1.37	1.03
Skin Change	3.03 (0.72)	1.00 – 4.00	-0.56	0.49
Breast Change	2.99 (0.76)	1.00 – 4.00	-0.21	-0.66
Menarche	3.48 (1.14)	1.00 – 4.00	-1.75	1.08
PDS Score	3.23 (0.63)	1.40 – 4.00	-0.99	0.18
PDS Stage	4.05 (0.67)	1.00 – 5.00	-0.68	2.37
Pubertal Timing				
Perceived Pubertal Timing	3.02 (1.04)	1.00 – 5.00	-0.20	-0.28
Age at Menarche ^a	2.93 (1.11)	1.00 – 5.00	-0.31	-0.84
Imaginary Audience Scale (IAS)				
Abiding Self Scale	6.97 (2.64)	0.00 – 12.00	-0.10	-0.51
Transient Self Scale	4.69 (2.51)	0.00 – 11.00	0.36	-0.53
Imaginary Audience Total	11.65 (4.22)	2.00 – 21.00	0.17	-0.69
Personal Fable Scale (PFS)				
Invulnerability Scale	15.38 (5.01)	6.00 – 29.00	0.16	-0.34
Speciality Scale	16.82 (3.62)	6.00 – 29.00	0.01	0.98
Personal Fable Total	32.19 (7.17)	13.00 – 51.00	-0.14	0.06
Revised Child Anxiety and Depression Scale (RCADS)				
Social Phobia	12.84 (6.52)	0.00 – 27.00	0.28	-0.68
Panic Disorder	6.24 (5.84)	0.00 – 26.00	1.33	1.44
Major Depression	8.20 (5.97)	0.00 – 24.00	0.76	-0.19
Separation Anxiety	3.54 (3.44)	0.00 – 17.00	1.39	2.02
Generalized Anxiety	6.94 (4.15)	0.00 – 18.00	0.85	0.05
Obsessive-Compulsive	5.08 (4.31)	0.00 – 17.00	0.70	-0.33
Total Anxiety Scale	34.64 (20.51)	0.00 – 97.00	0.78	0.01
Total Internalizing Scale	42.84 (25.24)	0.00 – 121.00	0.75	-0.07
Positive and Negative Affect Schedule for Children (PANAS-C)				
Positive Affect Scale	48.98 (11.82)	21.00 – 75.00	-0.27	-0.33
Negative Affect Scale	29.91 (12.83)	15.00 – 71.00	1.11	0.81

Note. *M* = mean; *SD* = standard deviation.

^a Indices coded on a 6-point scale: 1 = 10 years or younger, 2 = 11 years, 3 = 12 years, 4 = 13 years, 5 = 14 years, 6 = 15 years or older.

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Table 6

Descriptive Properties of Imaginary Audience Scores by Age and Grade

	Imaginary Audience	Abiding Self Subscale	Transient Self Subscale	<i>N</i>
Age				
12	14.47 (4.00)	8.76 (2.41)	5.71 (2.93)	17
13	11.40 (3.94)	6.85 (2.52)	4.55 (2.44)	20
14	11.31 (3.96)	6.63 (2.41)	4.67 (2.48)	49
15	11.02 (4.41)	6.68 (2.82)	4.34 (2.36)	41
Grade				
6th	16.25 (3.65)	9.50 (1.69)	6.75 (3.20)	8
7th	11.87 (4.16)	7.47 (2.72)	4.40 (2.20)	15
8th	11.57 (3.80)	6.86 (2.43)	4.71 (2.59)	21
9th	11.57 (4.16)	6.80 (2.51)	4.78 (2.59)	49
10th	10.80 (4.32)	6.60 (2.88)	4.20 (2.27)	30
11th	8.50 (2.12)	5.00 (1.41)	3.50 (0.71)	2

Note. Means are displayed outside of parentheses and standard deviations are in parentheses.

Total *N* for age (127) and grade (125) are different because grade was missing for one participant and another participant was homeschooled so selected “other” as her grade.

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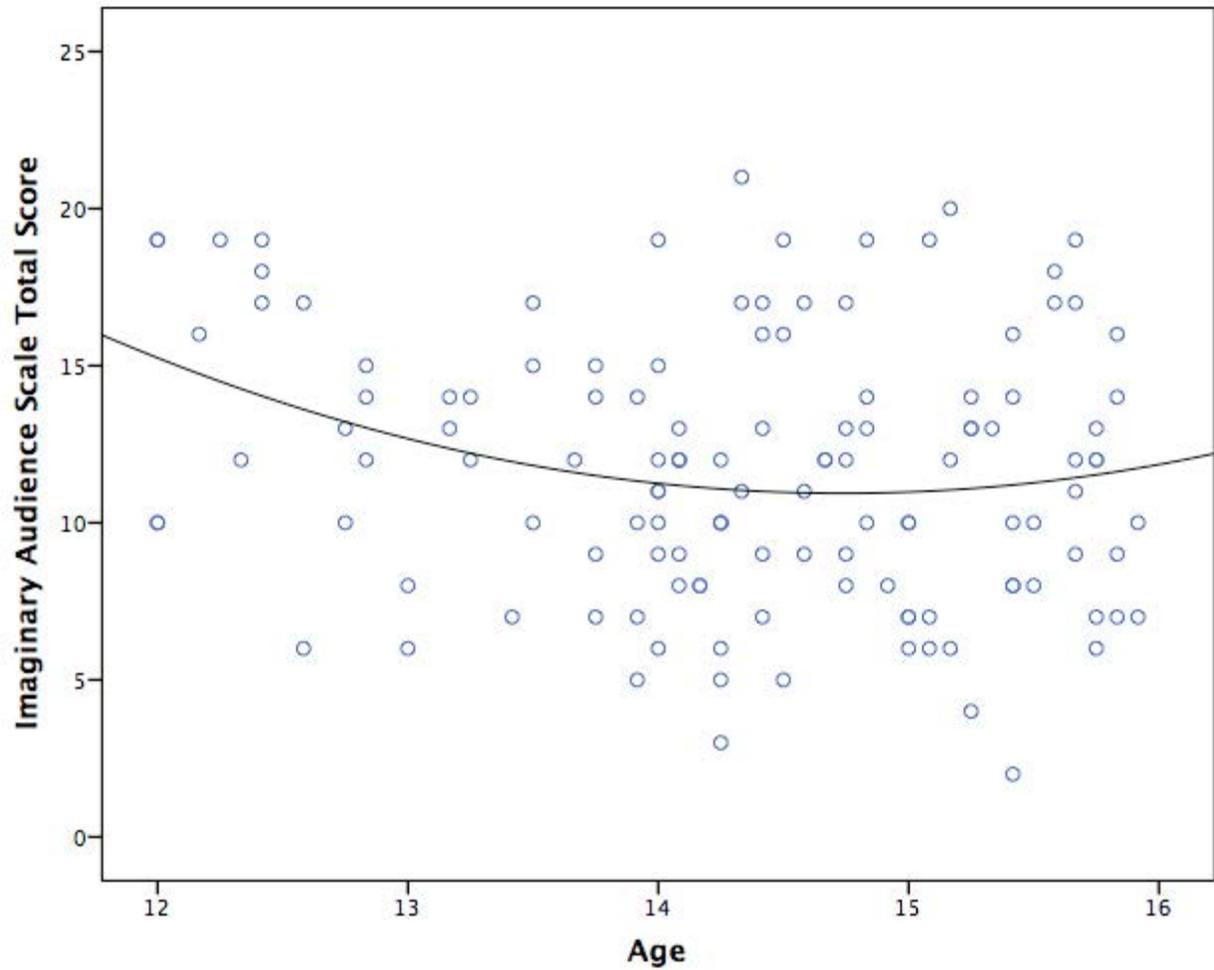


Figure 1. Trajectory of IAS total scores across the assessment age range of 12-15 years old. Quadratic line of best fit included to demonstrate overall trajectory, which does not support Elkind's (1967) theory of an inverted-U trajectory across adolescence.