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A Critical Review of the Empirical Literature on the Relation between Anxiety and Puberty

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Abstract

The current paper critically reviews the empirical literature focused on the association between puberty and anxiety. A detailed review of more than 45 empirical articles is provided. There is some evidence that among girls, but not boys, a more advanced pubertal status (controlling for age) is associated with higher reported anxiety symptoms. Also among girls, earlier pubertal timing is linked to higher anxiety scores. It is unclear whether early puberty may lead to increased anxiety or if high anxiety influences pubertal timing. With respect to hormones, there were relatively few significant associations for girls, although this literature is very small. Among boys, several studies reported positive associations between both gonadal and adrenal hormones and anxiety. The direction of effect for these finding is also unstudied. The primary limitation of the hormone-anxiety literature pertains to the absence of pubertal measures in samples of youth in which hormones are measured. The paper concludes with a comprehensive examination of the methodological strengths and weaknesses of the literature and recommendations for future work.

Keywords

pubertal status; timing; hormones; anxiety; psychopathology; adolescence

Anxiety disorders are among the most common forms of psychopathology in adolescence (Costello, Mustillo, Erkanli, Keeler, & Angold, 2003) and across the lifespan, with lifetime prevalence of anxiety disorders estimated to be 29% (Kessler, Berglund, Demler, Jin, & Walters, 2005). These rates are alarming, in light of the fact that anxiety psychopathology negatively impacts functioning across multiple domains (e.g., McGee & Stanton, 1990), maintains a chronic course for a significant proportion of youth affected (Orvaschel, Lewinsohn, & Seeley, 1995), and increases the risk for other types of disorders (Cole, Peeke, Martin, Truglio, & Serocynski, 1998).

The period of adolescence appears to be a particularly high-risk phase in terms of the onset and intensification of anxiety problems; for instance, panic attacks (Macaulay & Kleinknecht, 1989; Warren & Zgourides, 1988), social phobia (Inderbitzen & Hope, 1995), and obsessive-compulsive disorder (Rasmussen & Eisen, 1990) commonly emerge during this stage. Accordingly, researchers have called for developmentally sensitive etiologic models of anxiety

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among youth (e.g., Cartwright-Hatton, McNicol, & Doubleday, 2006). Current models do not consider the potential role of puberty in anxiety development, despite the significance of this developmental milestone and its association with other types of psychopathology (e.g., depression [Angold, Costello & Worthman, 1998]; eating disorders [Killen et al., 1992]). The current review is therefore timely, as it culls out the association between puberty and anxiety and provides a foundation for more systematically integrating puberty into theoretical and empirical work on the developmental psychopathology of anxiety. The objective of this paper is to exhaustively review the extant puberty-anxiety literature, uniquely extending earlier and more broad-based reviews of adolescent moods and behavior (i.e., Buchanan, Eccles, & Becker, 1992) and the impact of pubertal maturation on psychosocial development (i.e., Alsaker, 1995, 1996).

Definition and Measurement of Theoretically-Relevant Constructs and Study Selection Criteria

As a conceptual backdrop for the following review, approaches to defining puberty and anxiety are discussed below, along with study selection criteria. The concept of sex/gender as well as a brief discussion of how puberty fits into contemporary conceptualizations of risk is also touched upon for purposes of clarification.

Puberty

Puberty is a relatively brief (i.e., two to four-year) period in which youth experience extensive physical development, including reaching skeletal maturity (i.e., growth spurt), developing primary and secondary sexual characteristics (e.g., breast and penis growth), and attaining reproductive capability (Rogol, Roemmich, & Clark, 2002; Sheehy, Gasser, Molinari, & Largo, 1999). When theorizing about the characteristics of puberty that may confer risk for psychopathology, researchers typically highlight three aspects: pubertal status, pubertal timing, and puberty-related hormonal changes, all of which are conceptualized to be in dynamic relation with one another and the larger social context (Graber, Brooks-Gunn, & Warren, 2006).

Pubertal status refers to an adolescent's current level of morphologic development (Dubas, Graber, & Petersen, 1991) and can be indexed by asking participants to self-report their development in terms of their experience of particular pubertal events such as spermatarche (first emission of spermatozoa), oigarche (first ejaculation), and menarche (first menstrual period or bleeding) or specific physical indicators (e.g., using the Pubertal Development Scale [PDS] which inquires about adolescents' degree of development across a range of indicators, such as height, pubic hair growth, and skin changes; Petersen, Crockett, Richards, & Boxer, 1988). The most commonly used measure of pubertal status is the Tanner staging system (Tanner, 1962), a schematic depiction of secondary sexual characteristics (i.e., breasts and pubic hair for girls; genitalia and pubic hair for boys) that allows for classification in one of five Tanner stages.

Pubertal timing refers to the timing of pubertal onset relative to peers (i.e., early, on-time, or late). In general, girls and boys who are "on time" experience development in secondary sexual characteristics around age 12 and 13 years, respectively (Tanner, 1962). While the majority of studies reviewed below focus on the effects of pubertal timing on anxiety-type symptoms, there is theoretical work suggesting anxiety in early life could affect the timing of puberty (e.g., via changes to the HPA-axis secondary to childhood stress: Van Voorhees & Scarpa, 2004; and/or effects on reproductive strategies: Belsky, Steinberg, & Draper, 1991). Accordingly, researchers also have examined the effects of childhood anxiety on the timing of puberty (e.g., do anxious children experience early onset or delayed puberty?). These studies also are

included in the review. There are a number of methods for assessing pubertal timing, including 1) comparing participants' status on a conceptually-relevant indicator (e.g., menarche; spermarche) with sample- or epidemiologically-defined norms (Dubas et al., 1991), 2) classifying participants on the basis of established standards of physical development (e.g., Todd standards of skeletal maturity), 3) using self-reported Tanner stage across multiple assessments to calculate pubertal timing within a given sample (Wilson et al., 1994), 4) studying youth who experience precocious puberty (e.g., Sonis et al., 1985), a relatively rare condition where signs of puberty are observable abnormally early (i.e. prior to age 10 years in boys and 9 years in girls; Rosenfield, 1982) as well as the more acute condition of premature adrenarche (i.e., girls with evidence of pubic hair before age 8 years [9.5 years in boys]; Dorn, Hitt, & Rotenstein, 1999) or 5) examining self-reported *perceptions* of pubertal timing relative to peers (Dubas et al., 1991).

There are at least three sets of hormonal changes associated with puberty (Dahl, 2004). First, growth hormone (GH) drives the rapid physical growth (e.g., height changes) and contributes to the appearance of secondary sexual characteristics during this phase. Second, adrenarche, which begins between ages 5 and 9 years in girls (approximately one year later in boys; Grumbach & Styne, 2003), involves the release of adrenal hormones including androstendione (Δ^4 -A), dehydroepiandrosterone (DHEA), its sulfate (DHEAS), and cortisol (Parker, 1999). Finally, gonadarche, which begins at approximately age 9–10 years in girls, is characterized by the release of luteinizing hormone (LH) and follicle stimulating hormone (FSH) by gonadotropes in the anterior pituitary (Johnson & Everitt, 2000). These hormones stimulate the enlargement of the gonads, which in turn release testosterone (T) and estrogen (E)/estradiol (E_2) that promote the development of breasts and genitalia.

In terms of study selection criteria for the construct of puberty, studies of *pubertal status* included any assessment (e.g., self-report; physician-administered) of participants' current level of pubertal development. In terms of *pubertal timing*, studies using objective indices of early pubertal onset and/or participants' perception of whether their pubertal development was early, on-time, or late compared to peers were reviewed. Finally, in terms of *hormones*, studies were included if any pubertal hormone and any other index of puberty (e.g., status) were evaluated. It is important to note in this context that, despite a significant correlation between the two variables, puberty is not redundant with chronological age. For example, age does not necessarily reflect pubertal status because the events of puberty vary across individuals (e.g., breast development typically begins anywhere from ages 8 to 13; Tanner, 1962). Accordingly, the treatment of age in studies reviewed will be explicitly discussed. For instance, in many studies, researchers statistically adjust for age whereas a number of investigators methodologically control for the effects of age by examining pubertal indices within specific age groups at multiple points during a longitudinal study. To avoid redundancy, if age is not analytically or methodologically addressed, it will not be mentioned in the study description.

Anxiety

In terms of the outcome, we elected to focus specifically on anxiety for two key reasons. First, the anxiety disorders are a specific class of psychopathology characterized by future-oriented apprehension (Barlow, 2002) and elevated threat value associated with particular classes of stimuli (e.g., physical, social, and mental stimuli; Craske, 2003). Despite high rates of co-occurrence, anxiety disorders are conceptually and empirically distinct from depressive disorders; the Revised Integrative Hierarchical Model for the anxiety and mood disorders suggests they can be decomposed into three subclasses: distress disorders characterized by negative affectivity (e.g., depression), fear disorders reflecting anxious arousal (e.g., panic disorder) and the bi-polar disorders (see Mineka, Watson, & Clark, 1998; Watson, 2005; Weinstock & Whisman, 2006 for reviews). Although not uniform (e.g., GAD may be

characterized as a distress disorder; Watson, 2005), the fact that the many of the anxiety disorders tend to “hang together” in a cluster that can be distinguished from the depressive-type disorders suggests it may be useful to clarify developmental trajectories and risk factor constellations (including the role of puberty) for anxiety disorders, specifically. Second, the current review is lengthy, covering more than 45 published articles; expanding the focus to include depressive disorders would negatively impact the degree to which the puberty-anxiety association is examined in a detailed and integrative manner. For these reasons, we constrained the current review to anxiety-relevant outcomes. Nonetheless, we acknowledge that anxiety and depression are highly co-morbid; in an effort to cull out the unique variance puberty may share with anxiety (versus depression), findings are presented for both outcomes when available.

Anxiety can be measured in a number of ways, including self and other-reports as well as self or observer ratings in response to anxiety-relevant laboratory-based challenges. Structured clinical interviews generally are conceptualized as the “gold standard,” as the format decreases errors related to misinterpretation of questionnaire items. Puberty studies that included any of these indices of anxiety were included in the review.

Biological Sex/Gender

Much of the literature reviewed utilizes the terms sex and/or gender, with no effort to distinguish between these terms. The Institute of Medicine (IOM; 2001) defines biological sex as “the classification of living things, generally as male or female according to their reproductive organs and functions assigned by the chromosomal complement” (p. 1), a characterization that focuses on the biological distinctions between males and females. Gender, by comparison, is conceptualized as psychosocial in nature, defined as “a person’s self-representation as male or female, or how that person is responded to by social institutions on the basis of the individual’s gender presentation. Gender is shaped by environment and experience” (p. 1). Given the measurement approach taken by the studies reviewed below likely captures biological sex (e.g., participants are asked a one-item question, such as “are you a boy or a girl?”), the term sex is employed throughout the paper when similarities and differences between boys and girls are discussed.

Puberty as a “Risk Factor”

Integrating puberty into models of etiology and maintenance of anxiety psychopathology requires clarification of how the various aspects of puberty fit into contemporary conceptualizations of risk. Helena Kraemer and her colleagues (Kazdin, Kraemer, Kessler, Kupfer, & Offord, 1997; Kraemer, Stice, Kazdin, Offord, & Kupfer, 2001; Kraemer et al., 1997) have developed a standardized nomenclature for operationalizing risk factor processes in which a risk factor is defined as characteristic, experience, or event that indicates the relative likelihood of a given outcome among individuals within a population. Importantly, if the entire population is exposed to the variable in question, it is not a risk factor. According to this conceptualization, puberty *per se* (i.e., pubertal status; hormonal changes) is not a risk factor; all youth are exposed to the morphologic and hormonal changes of puberty. Puberty generally, then, is more accurately described as a “sensitive period” (Romeo, 2003), or a developmental epoch during which experiences (e.g., normative and non-normative stressors) exert particularly potent effects on brain development and related behavioral/physiological sequelae (e.g., Andersen & Teicher, 2008; see also Knudson, 2004). As such, the typical bodily and hormonal changes of puberty likely make it a high-risk phase for vulnerable youth, rendering puberty an important time for screening and intervention.

In contrast to pubertal stage and hormones, pubertal timing can be described using Kraemer and colleagues’ nomenclature. Kraemer and her co-authors suggest that a characteristic can be

classified as a *risk factor* (i.e., a characteristic that temporally precedes, and is correlated with, an outcome) or a *causal risk factor* (i.e., a characteristic that temporally precedes and, when manipulated, produces systematic change in an outcome). Risk factors can be further sorted according to whether they are malleable; if the characteristic changes spontaneously (e.g., age) or can be manipulated (e.g., skill set) it is classified as a *variable risk factor*, whereas if the characteristic is non-modifiable (e.g., sex), it is referred to as a *fixed marker*. Pubertal timing is a feature of puberty that reflects individual differences in an otherwise universal human experience and as such lends itself to classification within the Kraemer model. Specifically, pubertal timing describes particular sub-groups of youth at risk for clinically-relevant outcomes (e.g., early-maturing females evidence increased risk of substance use problems; Dick, Rose, Viken, & Kaprio, 2000). Although the timing of puberty has evidenced a secular trend (earlier onset across generations), altering the timing of puberty is not currently a reasonable target of intervention. Pubertal timing is therefore non-malleable and classified as a *fixed marker*.

Study Identification Method and Layout of Review

A two-part strategy was used to identify studies with potential relevance to the current review. First, a search of the PsycINFO database was conducted using all possible two-term pairings of the following keywords: puberty, pubertal, status, timing, maturity, Tanner, development, anxiety, problems, ratings, symptoms, worry, post-traumatic stress disorder, phobia, panic, obsessive-compulsive, internalizing, hormone, estrogen, estradiol, luteinizing, follicle stimulating, testosterone, gonadotropin-releasing, progesterone, oxytocin, dehydroepiandrosterone, dehydroepiandrosterone sulfate, androstenedione, and cortisol. This procedure was then repeated using the MEDLINE database. From the lists generated, we selected articles in accordance with our selection criteria and of these, excluded only dissertations and articles printed in a language other than English. Next, we conducted a backward literature search; reference lists from all articles gathered during the database searches were reviewed by hand – any article with conceptual relevance was obtained and evaluated for inclusion in the review. This process was repeated on an ongoing basis as new studies were identified.

The manuscript is divided into four main sections. The first three sections review the available research focused on anxiety and each aspect of puberty noted above. Relevant naturalistic and, when available, laboratory-based studies are reviewed and study-specific strengths and challenges are highlighted. In light of the distinct hormonal processes and psychosocial effects that characterize development among boys and girls (Brooks-Gunn, 1984), studies are organized in terms of the sample sex characteristics (i.e., all-female samples; all-male samples; mixed sex samples). In an effort to delimit the overall length of this review, an alphabetical listing of individual studies' key methodological characteristics and findings are provided in a separate table for each section. Whenever possible (e.g., provided by authors or extrapolated from available data), specific data (e.g., means; frequencies) as well as formal indices of effect size are provided to illustrate the nature and magnitude of findings. Standard recommendations for describing effect sizes are adopted (e.g., Cohen, 1988). Importantly, the review is specifically organized according to studies' capacity to provide direct and indirect evidence regarding the relation between anxiety and puberty. *Direct evidence* constitutes studies that examine specific anxiety outcomes (e.g., anxiety symptom checklist). *Indirect evidence* refers to studies that utilize more broad-based indices that include anxiety as part of a larger construct (e.g., internalizing problem scales). An integrative summary of findings is provided at the conclusion of each section (also organized by sex). The final section of the paper provides a description of the overarching strengths and limitations of the literature as well as specific recommendations for future research.

Anxiety and Pubertal Status

Direct Evidence

See Table 1 for a brief summary of key characteristics of each of the investigations reviewed (e.g., sample size, mean age, variables assessed, findings). In terms of direct evidence for the pubertal-status-anxiety linkage, three studies employed all-female samples, and none utilized a strictly male sample to examine this relation. In one of the earliest investigations, Stone and Barker (1939) examined differences in fear and worry (e.g., afraid/not afraid of suffocating; fear of dogs) between pre- and post-menarcheal girls of the same chronological age. Small differences in the tendency to endorse fear and worry were observed across groups, a pattern interpreted by authors as a “very slight tendency for the pre-menarcheal girls to indicate more fears than the post-menarcheal girls” (p. 57). Although a notable strength of this study was its large sample size ($N = 951$), one significant limitation was the use of an unstandardized measure of anxiety. Hayward and colleagues (1992), by contrast, utilized a gold-standard structured clinical interview to examine panic attack history in relation to pubertal status within a large school-based sample of 6th and 7th grade girls to evaluate the relations among chronological age, pubertal status, and panic attack history. Findings indicated a two-fold increase in the likelihood of having had a panic attack for each one-point change in Tanner stage, after adjusting for age (OR: 2.3). Indeed, none of the girls at Tanner stages I or II reported having had a panic attack. Unfortunately, the design of the study did not specifically allow for determination of when the girls’ first panic attack was experienced (i.e., during or prior to puberty). Finally, Huerta and Brizuela-Gamiño (2002) examined pubertal status, trait anxiety, and depressive symptoms in a large sample of Mexican adolescents. Pubertal stage correlated positively with both trait anxiety and depressive symptoms; it would have been particularly interesting to examine anxiety *symptoms* rather than the relatively stable characteristic of trait anxiety.

In the first of 11 studies using mixed sex samples, Canals, Marti-Henneberg, Fernandez-Ballart, Cliville, and Domenech, (1992) annually evaluated both state and trait anxiety in relation to pubertal status during a large four-year longitudinal study of Spanish youth. Tanner stage was unrelated to trait anxiety for either sex at every assessment. Mean scores of state anxiety tended to decrease with advancing Tanner stage among girls; indeed, compared to Tanner stages II–V, state anxiety scores were highest at Tanner stage I at every assessment. Among boys, the pattern was more mixed across assessments with the only significant pattern (observed at the third assessment) suggesting elevated state anxiety at the beginning and end of puberty. This study is especially noteworthy for its large sample size and longitudinal design, although it relies exclusively on self-report.

In the United States, the National Institute of Mental Health (NIMH) sponsored one of the most comprehensive studies of hormones, physical changes, and psychological development in early adolescence conducted to date. A number of relevant manuscripts discussed in this and subsequent sections are based on data from this study (referred to as the NIMH study); the general methodology is briefly discussed here. Pubertal status and anxious and depressed symptomatology were examined at three time points (six months apart) over a one-year period. At each assessment, physicians rated genital development among boys and breast development among girls, youth and parents rated pubertal status, a panel of hormones was obtained via venipuncture, a combined index of *emotional tone* (i.e., ratings of sad and anxious affect) was indexed via self-report, and mothers provided ratings of adolescents’ anxious and depressive symptoms. At the final assessment, anxious and depressive symptoms were assessed in a diagnostic interview. In one of the early reports, Susman, Dorn, and Chrousos (1991) examined pubertal stage in relation to anxious and depressive symptoms. Age and pubertal stage were entered simultaneously into regression equations. Concurrent analyses indicated a positive association between pubertal stage and anxious ($R^2 = .08$; $\beta = .29$) as well as depressive (R^2

= .09; β = .30) symptoms among boys at Time Three only. The magnitude of these associations was small. Further, despite a number of concurrent and prospective analyses, there were no other significant findings. This study provided a comprehensive, multimodal examination of clinical symptoms during puberty; however, as noted by the authors, their findings were limited by a relatively small sample size.

Extending upon evaluations of pubertal status in relation to *general* anxiety symptoms, two studies are notable in their focus on *specific* anxiety-relevant outcomes. First, Carrion, Weems, Ray, and Reiss (2002) examined pubertal status as it related to post-traumatic symptoms in a small sample of ethnically diverse, trauma-exposed children. Findings suggested the nature of PTSD symptom clusters evidenced “increased aggregation” (p. 168) as a function of pubertal stage; regardless of sex, participants in Tanner stage III, IV, or V were more likely to experience symptoms in more than one of the PTSD cluster groups; this was not true for the younger group (Tanner stage I and II). Second, using a multi-informant approach, Ge, Brody, Conger, and Simons (2006) studied the relation between pubertal status and symptoms of social anxiety, generalized anxiety disorder, and major depressive disorder in a large sample of African American youth. Pubertal status correlated positively with self-reported anxiety (e.g., generalized anxiety disorder) and depressive symptoms for both boys and girls in this sample; associations were more robust for boys (r 's ranged from .22–.28) compared to girls (r 's ranged from .12–.17). The same pattern was observed for parent report of social anxiety symptoms among boys only. Together, these studies are important in that they speak to linkages between pubertal development and DSM-defined anxiety outcomes; further, the study by Ge and colleagues is noteworthy in its focus on African American youth. Both studies are limited by the restricted age range of the participants, a methodological challenge that constrains conclusions that can be drawn about developmental differences in anxiety symptomatology across the entire course of puberty.

The remaining studies consider conceptually-relevant mediators and moderators of the pubertal status-anxiety association. The first two consider the roles of weight and body dissatisfaction which are common sources of distress for pubescent youth (Stice, 2003). First, O'Dea and Abraham (1999) examined pubertal status in relation to state and trait anxiety in a large study of disordered eating attitudes among Australian youth. There was no main effect of puberty on state or trait anxiety, although pre-pubertal girls had fewer depressive symptoms compared to post-pubertal girls. There also was a significant three-way interaction of sex, weight, and pubertal status in predicting trait anxiety (i.e., more developed, heavier girls reported elevated trait anxiety) and a two-way interaction between sex and pubertal status such that more developed girls had the highest depressive symptoms. In another study focused on eating pathology and puberty, McCabe, Ricciardelli, and Banfield (2001) examined body image, body change, pubertal stage, and anxious and depressive symptoms among a large sample of adolescents. Zero-order correlations (not adjusted for age) indicated no association between pubertal status and anxiety for boys or girls and a modest positive correlation with depression among girls. However, a more complex picture emerged using structural equation modeling. For instance, among girls, one model suggested pubertal status related negatively to body satisfaction and positively to strategies aimed at decreasing weight, each of which, in turn, predicted anxiety.

Another important potential moderator of the pubertal status-anxiety association relates to social support; three studies evaluated this variable. In a set of studies conducted in England, Goodyer and colleagues (Goodyer, Wright, & Altham, 1989, 1990) compared youth with anxiety or depressive disorders with matched community non-clinical controls. They sought to evaluate the independent and combined effects of several risk factors in the development of anxiety versus depression. In the earlier manuscript, the authors examined friendship quality (i.e. “good” versus “moderate/poor”) in relation to diagnostic status and found that while

clinical youth were more likely to have lower quality friendships, pubertal status did not affect the likelihood of being a clinical versus control case. Among the clinical youth, the link between diagnostic status and friendship quality was influenced by pubertal status such that pre-pubertal youth were just as likely to be anxious (32.5%) or depressed (50%) in the presence of lower quality friendships while post-pubertal youth were more likely to be anxious (70%), but not depressed (39%), in the presence of lower quality friendships. There were no effects of sex. The authors suggest puberty marks a change in the influence of friendships on anxiety among youth. However, in the follow-up study, the authors evaluated the triple interaction of friendship quality, undesirable life events, and pubertal status in relation to anxiety and depression. Here, the significant effects of life stress and friendship quality on diagnostic status were *not* influenced by pubertal status. Finally, Papini, Roggman, and Anderson (1991) studied the relations among early adolescents' perceptions of their attachment to their mother and father and whether such perceptions interacted with pubertal status in relation to depression, anxiety, and family cohesion variables. With the exception of one finding in which more developed youth reported elevated depressive symptoms, there were no other main effects of pubertal status, nor did this variable interact with maternal/paternal attachment to predict anxious or depressed symptoms.

Finally, Leen-Feldner and colleagues (2006) recently examined the moderating role of anxiety sensitivity (AS), a cognitive vulnerability variable for anxiety and panic development that reflects a tendency to fear the consequences of anxiety (Reiss & McNally, 1985), on the association between pubertal status and anxious responding to a three-minute hyperventilation challenge. After controlling for baseline anxiety, age, and sex, there was no main effect of Tanner stage. However, a significant interaction between pubertal status and AS in predicting anxiety ratings emerged such that adolescents reporting more advanced pubertal status and higher levels of AS reported the greatest post-challenge self-reported anxiety, whereas pubertal status had relatively less of an association with anxiety ratings among low AS adolescents. Further, the AS by Tanner stage interaction term was not related to self-reported negative affectivity, suggesting the interaction between AS and pubertal status is specifically related to anxious responding to bodily arousal, as opposed to generalized negative affectivity. The unique effect size of the interaction term was small in overall magnitude (i.e., $sr^2 = .03$), although it is worth noting that more than 25% of variance had already been accounted for at prior levels of the model. In a follow-up analysis conducted with the same dataset, Leen-Feldner, Reardon, and Zvolensky (2007) investigated whether challenge response (conceptualized to index panic vulnerability) would interact with pubertal status to predict self- and parent-reported panic-type symptoms. After controlling for sex, age, and pre-experimental anxiety, findings indicated a significant interaction between pubertal stage and challenge response in the prediction of multi-informant ratings of panic symptoms. Specifically, the highest elevations in panic symptoms were observed among participants who reported more advanced pubertal status and who also responded anxiously to the challenge. Collectively, the foregoing studies examining the roles of weight/body dissatisfaction, social support, and cognitive vulnerability to panic make the important contribution of beginning to disentangle potential mechanisms that may underlie the puberty-anxiety association. However, with two exceptions (Leen-Feldner et al., 2006, 2007), all of these investigations relied exclusively on self-report, an approach that may inflate shared method variance. Furthermore all were cross-sectional in design, and as such can only speak to developmental *differences* (cf., *changes* in puberty-anxiety linkages across the course of puberty).

Indirect Evidence

In an all-male, four-year longitudinal study, Ge, Conger, and Elder (2001) assessed pubertal status annually in a cross-sectional sample of youth, thereby methodologically controlling for the effects of age. Findings indicated pubertal status indexed at grade 7 related positively to

internalized distress at grades 8 and 10, although pubertal status assessed at all other time points did not predict internalized distress. Although the magnitude of the associations was modest (e.g., r 's ranged from .20 to .23), these findings suggest pubertal status is more strongly related to subsequent distress when measured relative to peers of the same age and sex and when measured in early adolescence when variability in pubertal status is relatively large. This impressive, prospective study evidenced some limitations in terms of generalizability, including sampling based on geography (all participants were from rural Iowa), ethnicity (participants were largely European American), and family structure (the study utilized two-parent families with a close-age sibling).

The remaining four studies utilized mixed sex samples to examine indirect links between pubertal status and anxiety. Patton and colleagues (1996) investigated pubertal status in relation to psychiatric symptoms in a large representative sample of Australian students. For girls, pubertal status was operationalized in terms of menarche. Specifically, the authors developed an index of menarcheal recency, comprised of five levels ranging from premenarche to more than 36 months since menarche. Age adjusted findings indicated an effect of menarcheal recency on psychiatric morbidity (including anxiety) such that, starting at three months post-menarche, the further girls were from menarche, the more likely they were to evidence symptoms, with girls who were more than 36 months past menarche evidencing a 60% increase in symptoms relative to pre-menarcheal girls. These effects remained significant after examining the mediating effect of six domains of stress (e.g., peer relations; money). Psychiatric "caseness" (symptom scores above a pre-determined threshold) similarly related positively to time since menarche, with girls more than 36 months past menarche exhibiting more than a twofold increase in morbidity ($OR = 2.2$). Among boys, there was a trend for increased psychiatric morbidity with advancing Tanner stage, although this relation was non-significant after controlling for age. These authors used sophisticated sampling techniques to ensure the representativeness of the sample in terms of youth attending secondary school, although the potential impact of certain sampling biases (e.g., absenteeism) on findings cannot be ruled out.

Extending Patton and colleagues' (1996) cross-sectional work, Laitinen-Krispijn, van der Ende, and Verhulst (1999) examined the relation between pubertal stage and changes in anxious/depressed symptoms in a large, two-year prospective study. The authors employed a multi-informant strategy, asking both parents and youth to report on anxiety/depressive symptoms at the beginning and end of the study. Only one significant relation was observed; parents reported that boys who had experienced relatively greater pubertal progress (i.e., change of two to four stages as compared to zero or one stage) were about half as likely to evidence increases in anxious/depressed symptomatology ($OR = .41$). This study represents one of the few longitudinal, multi-informant assessments focused on the puberty-anxiety relation. Potential limitations of this study include the fact that parent report data were not available for all subjects, and the authors required a change of at least one standard deviation for inclusion in final analyses, leaving open the question of the effects of advancing pubertal status on more nuanced increases/decreases in anxious symptomatology. Granger and colleagues (2003) also used a multi-informant, multi-method approach to examine the association between pubertal status and internalizing and externalizing problems among adolescents. In age-adjusted analyses, there were no significant associations between pubertal status and anxious-depressed symptoms. Despite the use of a non-specific anxiety measure, this study is noteworthy for its comprehensive use of assessments and informants.

Finally, in one of the only puberty-focused studies to consider biological vulnerability in the prediction of anxiety, Twitchell, Hanna, Cook, Fitzgerald, and Zucker (2000) investigated the relation between serotonergic functioning (5-HT), anxious/depressed symptoms, and pubertal stage in 62 children of alcoholics. Results indicated that puberty moderated the relation between

whole blood 5-HT and anxious/depressed symptoms such that pubescent youth with serotonergic dysfunction (i.e., low whole blood 5-HT levels) were the mostly likely to evidence elevations in anxious/depressed symptoms. More specifically, pubescent adolescents with serotonergic dysfunction evidenced elevations in anxious/depressed symptomatology ($n = 14$; $r = -.57$), whereas this relation was non-significant among prepubescent children ($n = 48$; $r = -.15$). While important in its evaluation of biological vulnerability in terms of the anxiety-puberty association, the authors note that whole blood 5-HT may relate to certain psychosocial factors (i.e., social competence, dominance, parental abuse or alcoholism); additional work is needed to clarify relevant mechanisms here.

Summary

A number of high quality studies of the association between pubertal status and anxiety have been conducted to date. Nonetheless, there are significant methodological limitations that characterize much of the early work in this area including small sample sizes, cross-sectional designs, assessment of only one aspect of puberty, analyses with boys and girls combined, and utilization of non-specific measures of anxiety. Accordingly, any conclusions drawn from the current research base should necessarily place greater weight on those studies where some of these limitations are addressed (e.g., Ge et al., 2001; Hayward et al., 1992). With this in mind, the following conclusions can be made at this stage of research development. Among girls, advancing pubertal status generally is associated with an increased likelihood of anxiety symptomatology, including panic attacks. This association does not appear to be accounted for by increasing chronological age. The findings among boys are more mixed, although Ge and colleagues provide some evidence of a similar trend as that observed for girls. Finally, there is evidence that social (Goodyer et al., 1989), somatically-oriented (McCabe et al., 2001), panic-relevant (Leen-Feldner et al., 2006, 2007), and biological (Twitchell et al., 2000) factors influence the association between pubertal status and anxiety among youth. Collectively, these findings support the idea that puberty may mark an important window of vulnerability to anxiety and related problems, and that this “sensitive period” may be particularly problematic for youth at risk for psychopathology by virtue of specific individual-difference or contextual characteristics.

Anxiety and Pubertal Timing

Direct Evidence

Key methodological characteristics of each pubertal timing-anxiety study are presented in Table 2. Pubertal timing is objectively measured unless otherwise stated. Three studies utilized female samples to explore the direct relation between pubertal timing and anxiety. In the earliest study in this domain, Sonis and colleagues (1985) compared girls who experienced precocious puberty to matched controls in terms of parent-reported anxiety and other internalizing-type problems. Findings indicated early maturers were elevated on internalizing-type symptoms, including somatic complaints and obsessive-type problems. The use of a matched control group is a noteworthy feature of this study; particular study specific biases (e.g., parents were treatment seeking) may have affected findings. Building on early pubertal timing work, Graber, Brooks-Gunn, and Warren (2006) examined multiple pathways by which pubertal timing is associated with adjustment (i.e., depressive affect and aggression). Anxious arousal was examined as a potential mediator. A descriptive analysis indicated early maturation, compared to late and on-time development, was linked to anxious arousal and depressive symptoms (not adjusted for age). In addition, path analyses controlling for age suggested anxious arousal mediated the association between early timing and depressive symptoms. This study is important in that it seeks to evaluate specific pathways (mediators) that may account for associations between pubertal timing and clinical symptoms; a key limitation of both of the foregoing studies was the use of a cross-sectional design.

In a six-year longitudinal study, Hayward and colleagues (1997) examined the relation between pubertal timing and internalizing problems in a large sample of girls. Pubertal timing was adjusted for age (Wilson et al., 1994). Early puberty was associated with 1) the development of internalizing symptoms (Hazard Ratio [HR]: 1.8), 2) ever having had a panic attack (HR: 3.1), and 3) the development of any internalizing disorder (HR: 1.7). Notably, the relation between early puberty and panic attack symptoms was more robust than that observed for eating disorders and depressive symptoms. Finally, a significant interaction was observed such that the combination of early puberty and a positive history of internalizing symptoms resulted in the highest overall risk of developing an internalizing disorder (38% of early maturers with previous internalizing symptoms developed a disorder during the follow-up period, compared to 16% of girls with neither early puberty nor a history of internalizing; OR: 3.3). There are several significant strengths of this study, including its prospective design and the use of gold standard assessment instruments. A limitation relates to the fact that a smaller subsample of the larger sample was followed into high school (although this group did not differ on several key characteristics, including ethnicity).

Two studies investigated the direct relation between pubertal timing and anxiety in all-male samples. Peskin (1967) examined anxious behavior among early-maturing and late-maturing boys. Specifically, trained interviewers provided ratings of participants' manifest anxiety on a yearly basis between the ages of 5 and 16 years; ratings were based on direct observation and interviews with family members and teachers. Comparisons were made at 10 equivalent points of maturational age, thereby methodologically controlling for age effects. Findings indicated early maturing boys were significantly more anxious compared to late-maturing participants at two years post-pubertal onset (but not at any other time during the study). In a similar study of an independent sample, Jones and Bayley (1971) found conflicting results. Specifically, late-maturing boys observed in naturalistic settings were found to be more socially anxious than early-maturing boys. Although this pattern was consistent at each assessment point across the 4.5 year study, it was particularly prominent at chronological age 16. The conflicting data from these two seminal, prospective studies are difficult to reconcile but may be attributed to small sample sizes.

The remaining seven studies examined the pubertal timing-anxiety relation among mixed-sex samples. In a groundbreaking set of studies, the first reported here to evaluate *perceptions* of pubertal timing, Graber and colleagues (1997) examined the relation between pubertal timing and psychopathology in an epidemiologically-defined public school sample. All analyses were adjusted for age. There were no significant effects for the anxiety disorders among boys or girls. Among girls, early maturers were more likely to meet criteria for any psychiatric disorder, as well as substance use, disruptive behavior, eating, and major depressive disorders. Late maturing boys were *less* likely to meet criteria for a substance use problem compared to on-time developers. However, these authors conducted follow-up interviews with a subsample of participants who originally took part in the 1997 study (Graber, Seeley, Brooks-Gunn, & Lewinsohn, 2004). Analyses were adjusted for age (at Time One). Findings indicated that early maturing women, compared to on-time developers, were nearly twice as likely (OR: 1.7) to meet criteria for an anxiety disorder at the follow-up assessment. Early-maturing girls also evidenced higher lifetime prevalence rates for depression (OR: 2.0), disruptive behavior, anti-social personality, and any Axis I disorder, as compared to on-time developers. There were no effects of late maturation among women. In contrast, men who had been late-maturers during adolescence as compared to on-time developers demonstrated higher lifetime prevalence rates of disruptive behavior disorder and current substance use. Pubertal timing was unrelated to the anxiety disorders and other Axis I psychopathology among men. These studies are noteworthy in terms of assessment approach, prospective design, and sample size; one potential limitation was the over-sampling of individuals with psychological disorders in the follow-up interviews, a methodological design that may limit generalizability.

Silbereisen and Kracke (1997) compared the effects of “perceived” timing (i.e., relative to peers) and “actual” timing (i.e., self-reports of when specific pubertal events occurred) on anxiety and depressive symptoms in a representative sample of German youth divided into two age groups (i.e., “young:” 13–14 years; “old:” 15–16 years). Interestingly, there were virtually no relations between perceived timing and either anxiety or depression across sex and age groups. In contrast, when “actual” timing was examined, older early maturing boys and younger early maturing girls evidenced elevated anxiety symptoms. Older early maturing boys also reported increased levels of depressive symptoms. The comparison of perceived and actual timing is an intriguing and important contribution, although the cross-sectional nature of the study limits conclusions related to change over time.

Using a multi-informant approach to measure (objective) pubertal timing Dorn, Susman, and Ponirakis (2003) examined self-reported, parent-reported, and physician evaluated pubertal timing (based on Tanner staging) in relation to anxious and depressed symptoms among participants from the NIMH study. Despite numerous analyses (i.e., concurrent analyses at each of the three assessment points; prospective models), there were relatively few associations observed, although late maturing boys (self-reported) and girls (parent-reported) evidenced elevated anxious symptoms in one concurrent analysis, with pubertal timing accounting for approximately 10% of variance in anxiety outcomes. This study is the first to evaluate the relation of pubertal timing and behavior problems using different raters of pubertal timing. However, the results of this study may have been limited by the relatively small sample size and the fact that parent ratings were not available for all participants.

Kaltiala-Heino et al. (2003) explored the relation between pubertal timing and emotional and behavioral problems in a large Finnish sample of youth. All analyses were adjusted for age. Youth were divided into five groups based on age at menarche/oigarche (ranging from < 10 years to 14 years) and compared to a reference group of late developers (i.e., age at oigarche/menarche $\geq$ 15 years). Findings indicated that the earlier menarche occurred, the more likely girls were to experience internalizing symptoms (i.e., separate indices of anxiety, psychosomatic, and depressive symptoms), odds ratios became smaller with increasing age at menarche (OR range: 1.4–3.4; see Table 2). Among boys, the earliest maturers evidenced more than a four-fold increase in the likelihood of anxiety and psychosomatic symptoms, although on-time developing boys (e.g., experienced oigarche between the ages of 12 and 14 years) were less likely than late maturers to endorse internalizing symptoms. This study highlights relatively large effect sizes in terms of anxiety for the earliest maturing boys and girls. A similar pattern, though less robust, was observed for depression. Interestingly, this effect was attenuated among both sexes when youth with externalizing symptoms were excluded, suggesting the presence of externalizing symptoms may account for some variance in the observed pubertal timing-anxiety association. The sample was representative of Finnish youth, supporting the generalizability of findings, although prototypical selection biases (e.g., absenteeism on assessment day) may be at play.

In the study of African American adolescents described in the previous section, Ge and colleagues (2006) also examined pubertal timing. Findings indicated early maturing girls evidenced significantly higher generalized anxiety and major depressive symptoms compared to late and on-time developers; boys evidenced a comparable pattern with early maturing boys also evidencing significantly higher social anxiety. The magnitude of the associations were small, with pubertal timing evidencing smaller correlations with internalizing outcomes among girls (r 's range from .11–.15) as compared to boys (r 's range from .21–.28).

Finally, Dorn and colleagues (1999) studied a small sample of children experiencing either on-time or premature adrenarche. Children and parents reported anxiety and depressive symptoms and a number of hormonal indices were collected. Although the mean differences were

generally small, early maturers evidenced greater somatic complaints and depressive symptoms compared to their on-time counterparts, and there were similar trends for anxious symptoms and diagnostic status. The association between hormones and anxiety symptoms was not examined. While this was the first study to examine hormones, psychological changes, and cognitive factors in a premature adrenarche and comparison group, this difficult-to-recruit sample was small.

Indirect Evidence

Drawing from the Iowa Youth and Families Project (IYFP), a four-year longitudinal study, Ge, Conger, and Elder (1996) reported linkages between pubertal timing and psychological distress among participating girls. Concurrent and predictive analyses suggested early maturing girls were more anxious and distressed than on-time or late maturing girls. Furthermore, the association between early maturation and distress appeared to be influenced by a number of variables. Specifically, peer group composition moderated the timing-distress association such that, compared to on-time and late developers, early maturing girls who also were associated with mixed (as opposed to same-sex) peer groups in the 7th through 9th grades were more likely to evidence psychological distress in the 10th grade. Next, using multigroup analyses comparing early maturers to late and on-time maturers, the authors examined an integrative model of vulnerability, where early psychological symptoms, paternal hostile feelings and behavior, and association with deviant peers were considered in relation to subsequent psychological distress. A number of interesting pathways were significant in the model; among early maturing girls only (cf. on-time and late developers), early psychological distress related positively to later psychological distress. In addition, early maturing girls were more likely to associate with deviant peers, which in turn predicted later psychological distress. A similar pattern was observed for paternal feelings of hostility. The total model explained 57% of variance among early maturing girls, compared to 9% among on-time/late maturing girls.

In a follow-up study, Ge and colleagues (2001) examined pubertal timing and internalized distress for the boys from the IYFP in grades 8, 9, and 10. Significant findings were observed at grades 8 and 10 such that early maturing boys evidenced higher levels of internalized distress compared to both on-time and late maturers; groups were indistinct at grade 9. In addition, the interaction between pubertal timing in grade seven and stressful life events in the past 12 months was examined in terms of subsequent distress (i.e., at grades 8, 9, and 10). Findings indicated that early maturing boys who also had a high frequency of recent stressful life events (relative to all other variable combinations), had the highest distress in grades 8 and 10, accounting for an additional 8% of unique variance. A similar test of the interaction between pubertal timing and distress in grade 9 was non-significant. The IYFP studies highlight the complexity of processes at play during puberty that may enhance psychological distress. Further, they are noteworthy for their multi-wave, multimodal, and multi-informant assessment, thus allowing for analysis of change across time. Its limitations are few, although participants had to be willing to allow investigators to conduct two-hour, in-home data collection at each wave.

Three studies utilized mixed-sex samples to study pubertal timing and psychological distress. First, using data from the NIMH study, Susman and colleagues (1985) examined the effects of maturational timing as defined by chronological age standardized within each pubertal stage. Boys and girls were analyzed separately; two subscales from the OSIQ are relevant (i.e., emotional tone and psychopathology). Findings showed that later maturing males evidenced elevated emotional tone – this effect was medium in terms of magnitude. There were no effects for girls. Additional hormone findings are reported in the next section.

Second, Petersen and Crockett (1985) studied the effects of pubertal timing on adjustment in a cohort-sequential study of youth. Early maturers, compared to late maturers, reported the

highest symptom levels on a general index of psychopathology, including anxiety. However, when the general psychopathology scale was reduced to its subscales in subsequent analyses, pubertal timing was unrelated to anxiety symptoms (i.e., as reported on the Emotional Tone subscale) and it did not interact with sex to predict this outcome. The developmental design of this study is notable, as it allows for evaluation of developmental change while controlling for cohort effects (Baltes, 1968). A limitation of this study is the use of an author-designed index of anxiety, which may have lacked adequate psychometric properties.

Finally, Sonis and colleagues (1987) examined a sample of youth who experienced precocious puberty to determine whether specific physical characteristics (i.e., height-age to chronological age ratio; breast and pubic hair stage) were related to internalizing-type problems among these early developers. After controlling for age, the authors found participants' height-age (age when child's height was in the 50th percentile of national norms) to chronological age ratio and pubic hair stage were *negatively* associated with internalizing problems, accounting for 16–22% of variance. In other words, older girls who looked most similar to their peers in terms of height and had less pubic hair evidenced the highest symptom levels. There was no association between breast stage and internalizing problems. The authors noted that their findings may have been limited by measurement error and restricted range of variability within their pubertal assessment.

Directional Effects: Does Anxiety Affect the Timing of Puberty?

The five studies in this section focus on whether pre-pubertal anxiety affects the onset of puberty (e.g., does childhood anxiety delay/potentiate pubertal onset?). Two studies utilized all-female samples to examine these directional effects, while none examined this research question exclusively among males. First, Kim and Smith (1998a) examined relations among pubertal timing, childhood stress, and clinical symptoms in a small sample of English mothers and daughters. Girls retrospectively reported age of menarche as well as anxiety and depressive symptoms occurring between the ages of 7 and 11 years. While a moderate association between elevated childhood anxiety and early menarche was observed in initial analyses, path modeling in which other variables were considered (e.g., maternal age at menarche), found no effects for childhood anxiety/depression on age at menarche. Reliance on retrospective report is a limitation of this study, an issue that was not problematic for Graber, Brooks-Gunn, and Warren (1995), who prospectively examined psychosocial predictors of age at menarche among pre-menarcheal girls (at baseline). After controlling for age, there was a negative correlation between self-reported internalizing symptoms and age at menarche; girls reporting fewer internalizing symptoms evidenced a delay in the onset of menses ($r = -.25$). This association was not significant after controlling for maternal age at menarche, nor did it predict above and beyond variance accounted for by physical indicators of puberty (e.g., breast development; body fat). Indeed, the most robust predictor of age at menarche in this study was generalized familial stress; youth who reported more stress in their rearing environments (e.g., conflictual relationship with mother) were the most likely to experience earlier menarche. Generalizability of this study may have been limited as their sample consisted of white, educated, middle- to upper-class girls from private schools in one geographic area of the country.

The three remaining studies utilized mixed sex samples. Kim and Smith (1998b) investigated a similar research question as in their 1998a study in a larger mixed-sex sample of Canadian college students. Findings indicated no effects for boys and a very small *negative* association between childhood anxiety and early menarche. In an independent sample of English college students, Kim and Smith (1999) utilized simultaneous regression to consider a number of conceptually relevant predictors of menarche/spermarche (e.g., quality of family life). They found no linkage between childhood internalizing symptoms and early menarche, although early spermarche evidenced a small, negative association with childhood internalizing

symptoms. The Kim and Smith studies were primarily limited by their cross-sectional nature and reliance on retrospective self-reports of pubertal events and psychological symptoms.

In a separate study, Meschke, Johnson, Barber, and Eccles (2003) examined self-reported math, school, and economic anxiety in relation to subsequent pubertal timing among 896 adolescents using data from the seven year Michigan Study of Adolescent Life Transitions. There were no significant relations among girls, although there were some small effects for boys, with elevated school anxiety predicting delayed puberty, and economic anxieties predicting earlier puberty. The authors note that potential mediators and moderators of levels of stress should be included in future analyses to better understand how stress relates to pubertal timing.

Summary

Several exceptional studies on the pubertal timing-anxiety association support the following conclusions. First, the most robust and consistent finding relates to *early* maturation among girls, which is associated with increased likelihood of anxiety symptoms and disorders. These findings do not appear to be accounted for by chronological age. One particularly well-done study provided evidence for sleeper effects; early maturation predicted the development of anxiety disorders at a 10 year follow-up after no associations were observed in the baseline analysis (Graber et al., 2004). In concert with findings from other prospective research (Ge et al., 1996; Hayward et al., 1997), there is evidence that the effects of early maturation on anxiety among girls are persistent and clinically important. *Late* maturation was generally unrelated to anxiety among girls. Second, there is less compelling evidence for the effects of early or late maturation on anxiety among boys, although the work conducted by Ge and colleagues (2001; 2006) highlights potential iatrogenic effects of early maturation. This literature is relatively small and characterized by mixed findings; additional research is needed to clarify the association between off-time maturation and anxious symptomatology among boys. Third, when examined, individual difference and/or contextual factors typically affected the pubertal timing-anxiety association. For instance, early maturation interacted with pre-pubertal internalizing symptoms, sex make-up of peer groups, life stress, and paternal feelings of hostility in the prediction of anxiety across sexes (Ge et al., 1996, 2001; Hayward et al., 1997). Taken together, available work supports the conceptualization of pubertal timing as a *fixed marker* for anxiety, particularly among girls.

Finally, in regard to the effects of pre-pubertal anxiety on the timing of pubertal onset, the literature is too mixed to draw integrative conclusions. Indeed, the temporal relation between puberty and anxiety has been significantly understudied and deserves further attention. Empirical work supports a significant effect of contextual stress (e.g., mother-daughter conflict; Kim, Smith, & Palermi, 1997) on the timing of puberty, so it makes conceptual sense that childhood anxiety would affect pubertal onset. Much of the extant literature relies on retrospective self-report, highlighting the utility of prospective research with youth. One theoretically interesting and empirically unexplored question relates to the potential role of childhood traumatic event exposure on pubertal onset and subsequent anxiety-related problems (see Hayward & Sanborn, 2002). In any case, continued work on the likely complex (bi-directional) interplay between anxiety and pubertal timing is indicated.

Anxiety and Hormone Levels

Direct Evidence

Key methodological characteristics of each pubertal hormone-anxiety study are presented in Table 3. It should be noted here that we identified a few hormone-anxiety studies in which pubertal status was measured, but not analytically considered (i.e., Carrion et al., 2002; Susman, Dorn, Inoff-Germain, Nottelmann, & Chrousos, 1997), likely because it did not relate to the

authors' primary study objectives. Similarly, some studies employed samples that lacked pubertal variation (e.g., all participants in Tanner Stage I; King, Mandansky, King, Fletcher, & Brewer, 2001; see also De Bellis et al., 1999; Feder et al., 2004). Although interesting and important in their own right, the design of these studies precludes conclusions about hormonal effects on anxiety in terms of the effects of puberty, the primary theme of the current review. In the interest of comprehensiveness and generating future research, the details of these studies are presented in Table 3, but they are not reviewed in the narrative or considered in the summary section.

In the all-female study described in the *Pubertal Timing* section, Graber and colleagues (2006) examined DHEAS and estradiol in relation to anxious and depressive symptoms. With the exception of a positive association between DHEAS and depressive symptoms (age/pubertal status not adjusted), there were no zero-order correlations between these hormonal indices and anxious/depressive affect. However, after controlling for age, the authors reported a near-significant interaction between DHEAS and pubertal timing predicting anxious symptoms such that early maturing girls with elevated DHEAS, relative to all other variable combinations, evidenced the highest levels of anxious symptomatology.

In the only all-male study focused on the anxiety-hormone relation, Olweus, Mattsson, Schalling, and Low (1980) examined the relations among testosterone (T) levels and several personality variables including anxiety in a small sample of Swedish male adolescents in Tanner stages III, IV, and V. Testosterone was unrelated to self-reported general anxiety (e.g., somatic anxiety), participant ratings of worry in relation to the venipuncture, or nurse ratings of participant state anxiety during the blood draw. The venipuncture as the anxiety-relevant stimulus was an elegant feature of this study, which may also have been limited by the constrained Tanner stage range.

The remaining four studies in this section utilized mixed-sex samples. The first two reports are drawn from the NIMH study, the strengths and limitations of which have been previously discussed. First, Nottelmann and colleagues (1987) examined several pubertal hormones in relation to psychosocial adjustment. Findings are reported after controlling for chronological age (CA) and pubertal status (PS). Among boys, the T/E₂ ratio related negatively to emotional tone and Δ 4-A related positively to obsessive-compulsive symptoms. Testosterone also related negatively to obsessive compulsive symptoms, but this effect disappeared when covariates (i.e., CA; PS) were included in the model. There were no hormone-anxiety associations observed among girls.

Second, Susman and colleagues (1991) examined concurrent and prospective associations between pubertal hormones and negative affectivity. After controlling for pubertal stage and chronological age, T and E₂ related negatively, and Δ 4-A positively, to emotional tone in concurrent analyses with boys. Testosterone also was related to depressive symptoms among boys. Among girls, T related positively to emotional tone, DHEAS related negatively to maternal-reported internalizing symptoms, and cortisol related positively to anxious symptoms. Across a number of analyses, no other concurrent associations with hormone levels were observed. In terms of prospective analyses (i.e., time one hormone levels predicting time three symptoms), after controlling for time one symptoms, chronological age, and pubertal status, LH and cortisol related negatively to maternal-reported internalizing symptoms, DHEA related positively to both anxious and depressive symptoms, and Δ 4-A related positively to anxious symptoms among boys. There were no findings for emotional tone. Among girls, the only significant effects were for anxious symptoms, which were predicted by LH (positive association) and DHEAS (negative association). The authors also examined a number of interactive effects, but there were no significant findings. The magnitude of associations was

small to medium, with hormones accounting for between 8 and 20% of variance in symptom outcomes (see Table 3).

In a particularly elegant study, Forbes and colleagues (2006) compared children (i.e., Tanner I/II) and adolescents (i.e., Tanner III, IV, V) in terms of peri-sleep-onset cortisol levels (i.e., cortisol levels in the 2 hours before and after sleep onset). Several demographic variables were evaluated and were unrelated to cortisol levels, including age and sex. Findings suggested children with anxiety disorders had higher mean peri-sleep-cortisol levels compared to healthy and depressed children. Further, cortisol levels were highest in the anxious group in the two hours preceding sleep. No significant group differences were observed among adolescents, although mean cortisol levels were higher among depressed adolescents, as compared to depressed children. These controlled, laboratory-based studies employed state-of-the-art assessment techniques, although the cross-sectional design limits our understanding of potential changes in cortisol levels across the course of puberty.

The final study focuses on the relation between cortisol and panic-relevant responding among youth. Coplan and colleagues (2002) examined salivary cortisol concentrations in a sample of psychiatrically ill and non-ill youth before and after a 5% carbon-dioxide inhalation procedure designed to elicit a panic-relevant state. The patient group was sorted according to whether the participant had panicked in response to the challenge. Age and sex were examined as covariates. Findings indicated that patients who panicked in response to the CO₂ inhalation had significantly elevated salivary cortisol immediately prior to the challenge (relative to patients who had not panicked as well as controls). Pre-challenge CO₂ cortisol levels were unrelated to both Tanner stage and subjective anxious responding to the challenge. Across groups, there were no significant increases in salivary cortisol concentrations related to the provocation procedure. A key strength of this study is the use of a biological challenge to elicit anxious and fearful responding, an approach that limits reliance on self-report. The representation of Tanner stages in the sample was unclear in the report; an interesting next step would be to compare pre- and post-pubertal youth in terms of pre- and post- challenge cortisol reactivity.

Indirect Evidence

Both studies in this section utilized mixed-sex samples in their studies. Using data from the NIMH study, Susman and colleagues (1985) explored the relations among hormone levels, pubertal status, peer relations, and adjustment problems. In a novel approach, these authors conceptualized pubertal timing in terms of hormone level-for-age (e.g., compared to same-aged peers within the sample, youth with higher pubertal hormone levels were considered early maturers). Analyses were standardized by age and sex. A greater number of findings were observed for boys, with early maturation, defined by higher-for-age levels of E₂ and T/E₂ ratios, related to *decreased* emotional tone/psychopathology scores, and higher-for-age levels of Δ4-A associated with *increased* emotional tone/psychopathology scores. Girls with higher-for-age FSH levels evidenced higher emotional tone/psychopathology scores. After adjusting for age, hormones accounted for between 6 and 21% of variance in anxiety-relevant outcomes, with larger effects for girls. The direct effect of hormones (after statistically controlling, rather than methodologically matching, for age) also were examined; findings were similar in pattern and magnitude (see Table 3).

In the same multi-informant, multi-method study discussed in the *Pubertal Status* section, Granger and colleagues (2003) examined salivary testosterone in relation to internalizing and externalizing problems in a mixed sex sample of youth. There were generally no significant zero-order correlations between testosterone and self/parent-reported anxious-depressed symptoms. Using structural equation modeling in which age and pubertal status were co-varied, boys with lower levels of testosterone and whose testosterone decreased less across the course

of a day had the most anxious-depressed symptoms. These symptoms were not associated with either level or diurnal slope of testosterone among girls.

Summary

The reader is directed to the “*Definition and Measurement of Key Constructs and Study Selection Criteria*” section for a brief review of the major hormones that drive the growth spurt, adrenarche, and gonadarche during puberty. The current section summarizes how these pubertal hormones may relate to anxious symptomatology. In general, the hormone-anxiety literature is in its infancy, making definitive conclusions impossible to draw. However, interesting trends are apparent, and appear to be consistent with the notion of pubertal hormones as contributing to a period of enhanced vulnerability to anxiety. Among girls, there were relatively few significant associations, although this literature is very small. More consistent findings were observed among boys, with several studies reporting links between T, E₂, Δ 4-A, and anxiety, suggesting both gonadal and adrenal hormones may affect anxiety symptomatology. However, the majority of data were drawn from the NIMH study, highlighting the need for independent replication and extension. A handful of studies examined cortisol and anxiety, with mixed results, including positive associations among girls but not boys (Susman et al., 1991), sleep-related elevations among anxiety-disordered children in early, but not later puberty (Forbes et al., 2006), and no associations with pre- or post- biological challenge cortisol as a function of pubertal status (Coplan et al., 2006). The major methodological differences across these studies likely accounts for the inconsistent findings, although future work also should attend to potential confounding variables (e.g., smoking; Rasmussen, Picciotto, & Krishnan-Sarin, 2006; see also Miller, Chen, & Zhou, 2007). Given the relative ease with which cortisol samples can be obtained (e.g., Dorn et al., 2006), as well as its established linkages with some types of anxiety (e.g., PTSD; Yehuda, 2002), addressing cortisol-anxiety associations in samples with significant pubertal variation as well as state-of-the-art assessments of puberty represents a key next step in this line of work. Indeed, the primary limitation of the hormone-anxiety literature pertains to its size and the absence of pubertal measures in samples of youth in which hormones are measured; it will be critical to systematically replicate and extend findings from existing hormone-focused studies that suggest a main effect of specific hormones on anxious symptoms.

A Critical Analysis of the Literature: Strengths, Challenges, and Future Directions

As noted in the introduction, the morphologic and hormonal changes of puberty may mark a period of enhanced vulnerability to anxiety-type problems, particularly among youth at-risk for other reasons. In addition, individual differences in pubertal timing, a fixed marker, may further enhance susceptibility to anxiety. At a general level, available work is supportive of this conceptualization. Indeed, there are a number of common strengths that characterize the existing research base, including multiple large/representative, longitudinal, multi-informant, and cross-cultural studies. However, the significant challenges inherent to this literature qualify these strengths; there is a pressing need to resolve existing inconsistencies and promote a more sophisticated and comprehensive understanding of the link between anxiety and puberty. Accordingly, the objective of this section is to delineate the overarching methodological challenges of the current research base and, in this context, provide specific recommendations for future research.

Assessment of Anxiety

A primary challenge of the extant literature pertains to the of assessment anxiety. While some studies employed well-standardized indices of anxiety (e.g., SCL-90; Ge et al., 2001) or

structured clinical interviews to index participants' diagnostic status (Hayward et al., 1992, 1997; Patton et al., 1996), many relied on single-item reports of current anxiety or examined anxiety as part of a larger internalizing construct. This heterogeneity complicates the matter of delineating *specific* relations among various types of anxiety and puberty. That is, given the objective of elucidating the role of puberty in developmental trajectories of anxiety psychopathology, this literature would benefit from increased precision and sophistication in the measurement of anxiety. We have three specific recommendations aimed at addressing this challenge.

First, it will be useful to isolate anxiety processes. Specifically, instead of the commonly used tactic of examining puberty in relation to broad-based internalizing-type problems, investigators could explore pubertal processes in the context of specific anxiety outcomes, including symptom checklists that correspond to specific anxiety disorders and diagnostic status as indexed via structured clinical interview. These data will be useful in clarifying the nature and magnitude of a putative puberty-anxiety association; in addition, researchers will be well positioned to examine whether and how pubertal processes affect anxious symptomatology across disorders (e.g., is early puberty more likely to influence social anxiety as compared to panic disorder?). Second, multi-informant strategies are particularly useful in anxiety assessment, as they reduce mono-method bias and delimit the impact of social desirability, which may be particularly likely among anxious youth (Kazdin, 1986; Kendall & Flannery-Schroeder, 1998). As in the Ge et al. (1996) study, the use of teacher, mother, father, and self-report indices to create a latent variable of anxious symptom levels will increase confidence in observed relations among puberty and anxiety. Finally, there may be significant merit in continued integration of laboratory-based assessments of anxiety processes, as such approaches allow researchers to examine anxious reactivity among healthy youth in the absence of potentially confounding psychopathology (e.g., anxiety and co-morbid problems; Olatunji et al., in press). For instance, are there systematic differences in response to a social evaluation task among psychologically healthy youth at each of the pubertal stages? Here, any observed associations between puberty and laboratory-based anxious responding cannot be explained by the presence of a disorder, and inferences related to the role of puberty in anxiety etiology/vulnerability are more readily made.

Assessment of Puberty

There are similar challenges related to the assessment of puberty. The reader is directed to an excellent guide for the puberty researcher, recently published by Lorna Dorn and colleagues (2006), for a comprehensive set of guidelines for conducting puberty research, which include 1) assessing puberty using multiple methods across several assessment points, 2) the need for longitudinal research that enrolls participants prior to starting puberty, 3) recommendations for standardizing indices of pubertal timing, including appropriate use of perceived versus objective assessments, and 4) conducting repeated assessments aimed at understanding the unique and interactive effects of pubertal hormones. Systematic incorporation of these guidelines will help to standardize puberty research, facilitating cross-study comparisons and supporting integrated conclusions.

Effect sizes

Effect sizes are a key characteristic to consider when seeking to draw integrative conclusions about any literature (Cohen, 1990). Solid conclusions about the strength of the relationship between the various aspects of puberty in relation to anxiety cannot currently be drawn because formal indices of effect size were not reported in a number of studies. This is a notable challenge of this literature that investigators are tasked with addressing in future work. Nonetheless, some preliminary statements can be made based on available data. Specifically, overall effect sizes were generally small to medium for pubertal stage (e.g., Ge et al., 2001; 2006; Susman et al.,

1991), timing (e.g., Dorn et al., 2003; Ge et al., 2006; Sonis et al., 1987; Susman et al., 1985), and hormones (Forbes et al., 2006; Susman et al., 1991; 1985); two-fold or greater increases in the likelihood of anxiety-relevant outcomes were observed as a function of advancing pubertal stage (e.g., Hayward et al., 1992; Patton et al., 1996) and between three and four-fold increases in anxiety were reported in relation to pubertal timing (Graber et al., 2004; Hayward et al., 1997; Kaltiala-Heino et al., 2003). This overall pattern of relatively modest effect sizes is not particularly surprising; Cohen (1988) suggests that effects observed in clinical psychology research are expected to be small due to a number of factors, including difficulty obtaining reliable and valid measurement as well as the nuanced nature of the phenomena studied. Further, as noted throughout the paper, a sophisticated understanding of changes in anxiety during the pubertal period will likely only be obtained when the features of puberty (e.g., timing, advancing stage) are considered within the larger context of individual-difference and environmental variables that also contribute to associations with clinically-relevant outcomes during this period (Graber, 2003). Indeed, as evidenced by some of the work reviewed (e.g., Ge et al., 1996), multi-variable models that include pubertal indices along with other theoretically-relevant factors will enable researchers to obtain larger effects in terms of predicting anxiety outcomes. We turn to this issue next.

Moderators and Mediators

There was consistent evidence across the literature suggesting other theoretically-relevant variables affect the puberty-anxiety association. There are at least three key reasons to examine mediators and moderators of the puberty-anxiety linkage. First, there are several variables that typify adolescence generally that may explain or enhance the association. Indeed, puberty is associated with profound changes across a number of dimensions, including sleep deprivation (Carskadon et al., 2002), conflict in parent-child relationships (Paikoff & Brooks-Gunn, 1991), enhanced emotional lability (Buchanan, Eccles, & Becker, 1992; Spear, 2003), alcohol, cigarette, and marijuana use (Johnston, O'Malley, Bachman, & Schulenberg, 2007), enhanced body dissatisfaction (Stice, 2003), and the onset of dating and sexual experimentation (Compian, Gowen, & Hayward, 2004). At this stage of research development, it is impossible to draw conclusions about the role of such pubertal correlates in terms of the puberty-anxiety association. One possibility is that puberty simply demarcates a window during which these developmental changes occur and increase the likelihood of the threat-relevant learning central to anxiety development (Craske, 2003). In this scenario, the normative changes of puberty (advancing status; hormonal shifts) as well as individual differences in pubertal timing play little or no role in anxiety development. The lack of systematic controls for such third variables that could potentially explain the puberty-anxiety relation in the studies reviewed leaves open this possibility and represents a critical area for future research. For example, in studies where advancing pubertal status is linked with elevated anxiety, it will be important to statistically or methodologically control for variables such as substance use and sexual experience. On the other hand, particularly from a developmental psychopathology perspective that highlights the likely complex and dynamic linkages across biological, psychological, and social domains in the onset of outcomes such as anxiety, there is theoretical reason to believe that the distinct changes associated with puberty have direct, additive, and/or interactive effects on anxiety. Such a speculation is consistent with a relatively large body of work that points to individual differences in pubertal timing in terms of its theorized and empirically supported effects on clinically-relevant outcomes (e.g., Graber et al., 2004; Hayward et al., 1997; Hyde, Mezulis, & Abramson, 2008). As an illustrative example, the hallmark characteristic of panic is fearful reactivity to bodily arousal (Barlow, 2002); panic is conceptualized to emerge following a series of conditioning trials in which fear/anxiety is paired with somatic perturbation (Bouton, Mineka, & Barlow, 2001). It stands to reason that specific characteristics of puberty could contribute directly to such interoceptive conditioning; for instance, hormonal changes during puberty appear to enhance stress responsiveness (e.g., Higley, Suomi, & Linnoila, 1990) and

may increase reactivity to threat-relevant cues, including bodily arousal.). Taken together, what is needed in order to draw firm conclusions about the role of puberty in anxiety etiology and maintenance is the systematic study of pubertal correlates that may affect the puberty-anxiety association.

Also germane to the issue of mediators and moderators is evidence in the studies reviewed that the vulnerability associated with puberty is not specific to the clinical phenomenon of anxiety. Indeed, although the literature is not uniform, it appears that the relations among puberty and anxiety, as well as puberty and depression, are similar in direction and magnitude. This is perhaps not surprising, given the relatively high rates of co-morbidity between the disorders (i.e., lifetime comorbidity estimated at 59.2% in the National Comorbidity Survey Replication; Kessler et al., 2003). These and other data suggest that puberty frames a period of enhanced risk for clinically-relevant outcomes generally, including anxiety (Ge et al., 2006), depression (Angold et al., 1998), eating disorders (Killen et al., 1992), and substance use problems (Patton et al., 2004). In the service of better understanding the puberty-anxiety linkage, it will be beneficial to identify variables that may help increase model specificity. For instance, the identification of outcome-specific moderators (e.g., dysfunctional attitudes may interact with puberty to predict depression [Lewinsohn, Joiner, & Rohde, 2001], whereas behavioral inhibition may interact with puberty to predict anxiety [Hirshfeld et al., 1992]) will help researchers to determine which specific clinical outcomes are most probable for particular subpopulations of youth (e.g., by virtue of risk factor constellations; Kazdin et al., 1997). Other conceptually relevant moderators include trait-like anxiety-relevant individual characteristics such as generalized negative affectivity, as well as environmental events such as traumatic event exposure. In this same vein, very few studies examined other psychiatric conditions, although there were linkages between the various aspects of puberty and, for instance, externalizing-type behaviors (Graber et al., 2004, 2006). Future work that includes, for example, tests of incremental validity and comparison of samples with specific clinical problems is needed to clarify the specificity of the puberty-anxiety association, relative to other commonly co-morbid psychiatric conditions.

Finally, a better understanding of the roles of specific cultural and demographic characteristics is needed. One important research endeavor will be to clarify the relative influence of interpersonal (e.g., autonomy-seeking; dating; Paikoff & Brooks-Gunn, 1991) and cultural features (e.g., differential timing and effects of puberty among African American girls; Herman-Giddens et al., 1997; Michael & Eccles, 2003) as compared to the distinct puberty-relevant biological and hormonal changes (e.g., breast development) in terms of anxiety. Indeed, with one exception (Ge et al., 2006), a study that found early maturation was linked to elevated anxious symptoms among African American boys and girls, no work has evaluated the role of ethnicity. Because the meaning and consequences of puberty may differ across racial/ethnic groups (e.g., Hayward, Gotlib, Schreadley, & Litt, 1999; Piran & Ross, 2006) it will be important to ascertain the effects of puberty on anxiety in terms of this characteristic. Similarly, although the role of culture is receiving some consideration, with studies from Spain, Mexico, England, Finland, Sweden, Australia, Canada, and the Netherlands (see Tables for details), the evidence base is relatively small and fairly mixed, precluding integrated conclusions about the generalizability of puberty-anxiety linkages that have been observed within the United States. Finally, as noted in the introduction, biological sex can be usefully distinguished from gender (IOM, 2001). Given both constructs likely are relevant to the experience of and potential vulnerability related to puberty, efforts to disentangle the effects of each on anxiety development among pubertal youth represents an important area for future research. Overall, it will be critical to build upon the extant work primarily focused on *whether* puberty and anxiety evidence a reliable association, and move toward addressing questions related to the *nature* of the association (Graber et al., 2006).

Applied/Clinical Implications of this Domain of Study

There is a tremendous amount of work yet to be done if we are to gain a sophisticated understanding of the puberty-anxiety association. Although daunting, advancing this field is important from both theoretical and public health perspectives. Indeed, it is clear from the foregoing review, as well as findings from other literatures (e.g., Alsaker, 1996), that, while not a target of change in and of itself, puberty marks a window of increased risk for anxiety psychopathology, making it an important period for screening and intervention efforts. Pubertal timing, in particular, is a *fixed marker* (Kraemer et al., 2001), and as such can aid in the identification of youth at enhanced risk for anxiety and related problems. Specifically, malleable individual-difference and contextual factors could be targeted among early/late maturing youth to prevent the development or exacerbation of anxiety symptomatology during this sensitive period (Craske & Zucker, 2002; Leen-Feldner, Reardon, Hayward, & Smith, 2008). As an illustrative example, it appears that early maturation among girls is linked with increased risk for panic-type problems (e.g., Hayward et al., 1997). Given evidence that the panic vulnerability factor of anxiety sensitivity (AS) further enhances risk for these girls (Hayward, Killen, Kraemer, & Taylor, 2000), investigators in a primary care setting could identify early-maturing, high AS girls and design a brief intervention aimed at reducing AS (e.g., exposure trials to reduce fear of anxiety-related sensations; Schmidt et al., 2007).

Taken together, puberty, the most significant milestone of adolescence (Hayward, 2003), relates in a meaningful way to anxiety psychopathology, particularly among female adolescents. Continued research aimed at clarifying the complex interplay between puberty and anxiety will be critical for articulating the specific role of puberty and its parameters in conceptual models of the development and maintenance of anxiety psychopathology among youth.

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Table 1

Studies of Anxiety and Pubertal Status

Authors	Participants/Study Type	Measures of Puberty and Anxiety	Findings
Canals, Marti-Henneberg, Fernandez, Ballart, Cliville, & Domenech (1992)	336 Spanish youth (161 girls) 11–15 years (assessed annually starting at age 11 [girls]; 12 [boys]) <i>Four-year longitudinal study</i>	Pubertal status: Tanner stage (practitioner evaluated) Anxiety: STAI-C *Depression (CDI; no findings reported for depression)	Trait anxiety: no significant differences as a function of pubertal status. State anxiety (girls): tendency for anxiety to be elevated at Tanner I and decrease across puberty; pattern significant only at 2 nd assessment (when girls were 12 years old; Tanner I-V; $M = 52.7$; 27.9; 28.6; 27.2; 26.3, respectively). State anxiety (boys): mixed findings. Significant effect at 3 rd assessment (when boys were 14 years old); boys in Tanner stages I ($M = 52$) and V ($M = 28.8$) reported higher state anxiety than boys in stages II – IV ($M = 27.8$; 27.2; 28.2, respectively).
Carroll, Weems, Ray, & Reiss (2002)	59 children (25 girls; $M_{age} = 10.6$; range: 7–14 years) <i>Cross-sectional</i>	Pubertal status: self-reported Tanner stage Anxiety: CAPS-CA K-SADS-PL CBCL (parent report)	Children in the “older” pubertal status group (Tanner stage of III, IV, V) more likely to experience aggregation of PTSD clusters; children of more advanced pubertal status more likely to meet symptoms in more than one cluster group [Cluster B (Reexperience) and C (Avoidance and Numbness); $\chi^2_1 = 3.83$ ($p = .05$); Cluster B and D (Hyperarousal); $\chi^2_1 = 8.97$. Children in the younger pubertal status group (Tanner I and II) did not evidence such aggregation.
Ge, Conger, & Elder (2001)	202 boys; 12–14 years at Time One ($M_{age} = 12.68$; $SD = .55$) <i>Four-year longitudinal study; assessed annually</i>	Pubertal status: PDS Anxiety: “internalized distress” (i.e., depression, anxiety, somatization, and eight other items on the SCL-90-R)	Pubertal status at grade 7 predicted internalized distress at grades 8 ($r = .23$, $p < .05$) and 10 ($r = .20$, $p < .05$), but was unrelated to internalized distress at grade 9. Pubertal status at grades 8, 9, and 10 was unrelated to internalized distress at any other assessment point.
Ge, Brody, Conger, & Simons (2006)	867 African American youth (467 girls; $M_{age} = 10.5$; range: 10–12 years) <i>Cross-sectional</i>	Pubertal Status: PDS Anxiety: parent and child-reported internalizing symptoms (i.e., social anxiety, generalized anxiety, and major depressive disorder [DISC-4.0])	Boys: pubertal status correlated positively with self-reported symptoms of social anxiety (SA; $r = .22$), generalized anxiety disorder (GAD; $r = .26$), and major depression (MDD; $r = .28$), as well as parent-reported symptoms of SA ($r = .10$) Girls: status correlated positively w/self-reported GAD ($r = .12$) and MDD ($r = .17$) symptoms, but not parent-reported symptoms.
Goodyer, Wright, & Altham (1989)	100 (51 girls) English youth with anxiety or depressive disorders ($M_{age} = 12.7$; $SD = 2.6$; range: 7 – 16 years) 100 non-clinical controls matched for age, sex, and social class ($M_{age} = 13.1$; $SD = 2.5$; range: 7 – 16 years) <i>Cross-sectional</i>	Pubertal Status: semi-structured interview based on Tanner’s criteria Anxiety: clinical interview; anxiety-dominant features during past two weeks (e.g., fearfulness, phobias, somatic symptoms) and absence of sadness *Depression: clinical interview; depressive-dominant features	Pubertal status did not moderate the relation between friendship status and diagnostic status (i.e., case versus control) Among clinical youth, friendship quality associated with equal likelihood of being anxious or depressed among pre-pubertal children; 32.5% of those with moderate to poor friendships reported anxiety while 50% reported depressive symptoms Post-pubertal children more likely to be anxious (70%) but not depressed (39%) in the presence of poor friendships
Goodyer, Wright, & Altham (1990)	Same as above	Same as above	Pubertal status did not affect the significant associations between friendship quality and life stress on the likelihood of being diagnosed with anxiety or depression.
Granger et al. (2003)	213 adolescents (107 girls; 11–17 years; $M_{age} = 13.66$; $SD = 1.53$) <i>Short-term (two-day) Longitudinal</i>	Pubertal status: PDS (boys: genital/pubertal hair; girls: breast/pubertal hair) Anxiety-Depression: (single latent variable) -Parent and youth reported: anxiety/depression and social withdrawal (YSR/CBCL) and diagnostic Status (DISC-4.0)	No association between anxious-depressed symptoms (single latent variable) and pubertal status for boys or girls

Authors	Participants/ <i>Study Type</i>	Measures of Puberty and Anxiety	Findings
Hayward et al. (1992)	754 girls 10–15 years ($M_{age} = 12.3$; $SD = .8$); <i>Cross-sectional</i>	Pubertal status: Tanner stage [†] Anxiety: Panic attack history (SCID)	Two-fold (OR: 2.3) increase in the likelihood of having had a panic attack for each one-point change in Tanner Stage.
Huerta & Britzuela-Gamino (2002)	971 Mexican girls 8–16 years ($M_{age} = 12.5$; $SD = 2.4$); <i>Cross-sectional</i>	Pubertal Status: Tanner stage (breast development) Anxiety: STAI-trait *Depressive symptoms (author-developed measure)	Compared to girls in Tanner stages I – IV ($M = 22.76$; 20.6; 22.9; 23.1, respectively), girls in Tanner stage V ($M = 27.4$) reported higher trait anxiety scores. Nearly identical pattern for depressive symptoms.
Laitinen-Krispijn, van der Ende, & Verhulst (1999)	1837 Dutch adolescents *Only youth with a ≥ 1 SD symptom change included (parent reports: $n = 207$ [98 girls]; youth reports: $n = 476$ [250 girls]) Time one $M_{age} = 10.9$; $SD = .4$ [range 10–12]; Time two $M_{age} = 13.1$; $SD = .4$ [range 12–14] <i>Two-year longitudinal study</i>	Pubertal Status: Physician assessed Tanner stage Anxiety: Anxious/Depressed subscale of CBCL and YSR	Compared to boys evidencing little pubertal progress during the study (i.e., change of 0 to 1 stage), boys who progressed 2–4 stages were <i>half as likely</i> to show an increase in parent-reported anxious/depressive symptoms (OR = .41). No relation between pubertal status and symptoms for male or female self-report or female parent report.
Leen-Feldner, Reardon et al. (2006)	124 adolescents (57 girls) 12–17 years ($M_{age} = 15.04$; $SD = 1.49$) <i>Cross-sectional; laboratory-based</i>	Pubertal status: Tanner stage Anxiety: -Anxiety sensitivity (CASI) -Anxiety ratings following a hyperventilation challenge (SUDS) *Negative affectivity (PANAS-C)	No main effect of Tanner stage on ratings following a hyperventilation challenge. Pubertal status interacted with anxiety sensitivity (AS) to predict anxiety ratings ($sr^2 = .03$, $p < .05$); high AS youth adolescents who also reported more advanced pubertal development evidenced the greatest anxiety. Finding specific to anxiety; the interaction between negative affectivity and pubertal status was not significant.
Leen-Feldner, Reardon, & Zvolensky (2007)	Re-analysis of sample described above	Pubertal status: Tanner stage Anxiety: -Panic symptoms (SCARED-P) -Somatic complaints (CBCL & YSR) -Anxiety ratings following a hyperventilation challenge (SUDS)	No main effect of Tanner stage on panic symptoms or somatic complaints Pubertal stage interacted with emotional reactivity to bodily sensations to predict adolescent-reported panic symptoms ($sr^2 = .03$, $p < .05$) and somatic complaints ($sr^2 = .09$, $p < .05$), as well as parent-reported somatic complaints ($sr^2 = .03$, $p < .05$); adolescents who reported an advanced pubertal status and more anxious responding to a hyperventilation challenge evidenced the most panic-relevant symptoms.
McCabe, Ricciardelli, & Banfield (2001)	1185 adolescents (598 girls) 12 to 16 years (boys: $M_{age} = 13.2$; $SD = 1.10$; girls: $M_{age} = 13.21$; $SD = 1.01$) <i>Cross-sectional</i>	Pubertal status: PDS items (boys: body hair/voice change; girls: body hair/breast development) Anxiety: DASS *Depression (DASS)	Correlations: pubertal development unrelated to anxious symptomatology for both sexes; depressive symptoms pubertal status among girls only ($r = .10$, $p < .05$).
O'Dea & Abraham (1999)	470 Australian adolescents Grades 7 and 8 173 boys ($M_{age} = 12.8$; $SD = 0.6$); 297 girls ($M_{age} = 12.7$; $SD = 0.6$) <i>Cross-Sectional</i>	Pubertal status (dichotomized): -menarcheal status -males self-report of having “reached puberty” (no other information provided) Anxiety: STAI *Depression: BDI	Main effects: No differences in state anxiety for pre, as compared to post, boys ($M = 30.7$; 30.4, respectively) or girls ($M = 30.4$; 31.4), same pattern for trait anxiety. Pre-pubertal girls ($M = 46.2$) had significantly lower depressive symptoms than post-pubertal girls ($M = 50.1$); no differences for boys ($M = 47.1$; 45.6). Interaction: three-way between sex, puberty, and weight – more developed, heavier girls reported elevated trait anxiety ($F = 5.1$) Puberty and sex interacted to predict depressive symptoms ($F = 4.8$); more advanced girls had elevated depression.
Papini, Rogman, & Anderson (1991)	231 7 th graders (133 girls) $M_{age} = 12.8$ (range 12–13 yrs) <i>Cross-sectional</i>	Pubertal status: PDS (trichotomized w/sample-specific means) Anxiety: Social anxiety (CMAS) *Depression (CDI)	Four regression models examining main and interactive effects of pubertal status and maternal/paternal attachment on anxious/depressed symptoms: Main effect of pubertal status on depression ($\Delta R^2 = .04$) in maternal attachment model, no other main or interactive effects.

Authors	Participants/ <i>Study Type</i>	Measures of Puberty and Anxiety	Findings
Patton et al. (1996)	2,525 Australian adolescents (~half girls) selected in ~equal numbers in Grades 7, 9, and 11 (12–17 years); <i>Cross-sectional</i>	Pubertal status: -Tanner stage -Menarcheal status Anxiety: -Psychiatric morbidity (combined index 14 CIS-R subscales, including anxiety) -Psychiatric "caseness" (CIS scores $\geq$ 12)	Girls: Starting at three months post-menarche, psychiatric symptoms positively associated with increasing time since menarche: 3–12 months post-menarche ($\beta = .28$), 12–36 months post-menarche ($\beta = .29$), and > 36 months post-menarche ($\beta = .47$). -Logistic Regressions predicting "caseness" (CIS scores ≥ 12) yielded similar findings; 3–12 months post-menarche (OR: 1.3); 12–36 months post-menarche (OR: 1.6) and > 36 months post-menarche (OR: 2.2). No findings reported for Tanner stage. Boys: No significant findings.
Stone & Barker (1939)	564 post-menarcheal and 387 pre-menarcheal girls (11–15.5 years); <i>Cross-sectional</i>	Pubertal status: -Menarcheal status Anxiety: -Author-developed measure of fear and worry	Compared to post-menarcheal girls, premenarcheal girls evidenced slightly elevated fear and worry: of 50 possible responses reflecting <i>elevated</i> fear/worry, 30 were more likely to be endorsed by pre- menarcheal girls; of 61 items reflecting <i>relatively less</i> fear/worry, 39 were more likely to be endorsed by post-menarcheal girls.
Susman, Dorn, & Chrousos (1991)	NIMH Study: method detailed in narrative 56 boys (10–15 years) 52 girls (9–14 years) <i>Cross-sectional and prospective comparisons at 0 (T1), 6 (T2), and 12 (T3) months</i>	Pubertal status: -practitioner evaluation of Tanner Stage Anxiety: -Depressive and anxiety symptoms (DISC; T3 only) -Self-reported emotional tone (ratings of sadness and anxiety; OSIQ; T1, T2, T3) -Mother-reported internalizing problems (CBCL; T1, T2, T3)	Concurrent findings: advanced pubertal stage correlated with elevated anxious ($R^2 = .08$; $\beta = .29$) and depressive ($R^2 = .09$; $\beta = .30$) symptoms among boys at the third assessment. -No significant findings at any time point for girls -No significant findings at any time point for either sex in terms of maternal report or self-report of emotional tone. Prospective findings: none.
Twitchell, Hanna, Cook, Fitzgerald, & Zucker (2000)	62 Caucasian children (17 females) of alcoholics 7–16 years ($M_{age} = 10.88$; $SD = 2.03$) <i>Cross-sectional</i>	Pubertal status: -Tanner stage -pre-pubescent $\geq$ Tanner III: pubescent Anxiety: Anxious/Depressed subscale of CBCL	Puberty moderates the relation between whole blood 5-HT and anxious/ depressive symptomatology; pubescent adolescents with serotonergic dysfunction evidenced elevations in anxious/depressed symptomatology ($n = 14$; $r = -.57$), whereas this relation was non-significant among prepubescent children ($n = 48$; $r = -.15$, <i>ns</i>).

Note. Unless otherwise noted, findings are statistically significant at conventional levels. AS: Anxiety Sensitivity; BDI: Beck Depression Inventory (Beck, Steer, & Garbin, 1988); CAPS-CA: Clinical Administered PTSD Scale-Child and Adolescent Version (Nader et al., 1996); CASI: Childhood Anxiety Sensitivity Index (Silverman, Fleisig, Rabian, & Peterson, 1991); CBCL: Child Behavior Checklist (Achenbach, 1991); Achenbach & Rescorla, 2001); CDI: Children's Depression Inventory (Kovacs, 1992); CMAS: Child Manifest Anxiety Scale (Reynolds & Richmond, 1978); CIS-R: Clinical Interview Schedule—Revised, computer form (Lewis et al., 1988); DISC: Diagnostic Interview Schedule for Children (Costello, Edelbrock, & Costello, 1985); Diagnostic Interview Schedule for Children, Version 4 (Schaffer et al., 1993); DASS: Depression, Anxiety, and Stress Scale (Lovibond & Lovibond, 1995); GAD: Generalized Anxiety Disorder; K-SADS-PL: Schedule for Affective Disorders and Schizophrenia for School-Age Children—Present and Lifetime Version (Kaufman et al., 1997); MDD: Major Depressive Disorder; OR: Odds Ratio; OSIQ: Offer Self-Image Questionnaire (Offer, Ostrov, & Howard, 1977); NEO-PI: NEO Personality Inventory (Costa & McCrae, 1985); PANAS-C: Positive Affect and Negative Affect Scale for Children (Wilson, Gullone, & Moss, 1998); PDS: Pubertal Development Scale (Petersen et al., 1988); SA: Social Anxiety; SCID: Structured Clinical Interview for DSM-III-R Non-Patient Version (Spitzer, Williams, & Gibbon, 1987); SCARED-P: Screen for Child Anxiety Related Emotional Disorders—Panic Subscale (Birmaher et al., 1997); SCL-90-R: Symptom Checklist 90—Revised (Derogatis, 1983); STAI-C: State-Trait Anxiety Inventory for Children (Spielberger, 1973); SUDS: Subjective Units of Distress Scale (Wolpe, 1958); Tanner stage: Tanner stages of pubertal development (Tanner, 1962); TRF: Teacher Report Form (Achenbach, 1991); YSR: Youth Self Report (Achenbach, 1995).

*Unless otherwise noted, Tanner stage is self-reported by participants.

Table 2

Studies of Anxiety and Pubertal Timing

Authors	Participants/Study Type	Measures of Puberty and Anxiety	Findings
Dom, Hitt, & Rothenstein (1999)	29 children (6–9 years); 9 Children with premature adrenarche (8 girls; $M_{age} = 7.8$; $SD = 1.3$) 20 Children with on-time adrenarche (16 girls; $M_{age} = 8.0$; $SD = 1.2$) <i>Cross-sectional</i>	Pubertal Timing: -Adrenarche (practitioner evaluated) -Adrenal and gonadal hormones Anxiety Diagnostic Status (DISC) Anxious/Depressed symptoms. (CBCL) Anxiety (STAI) *Depression (CDI)	Psychiatric Diagnoses: trend -early maturers (44%; $n = 4$ [three simple phobia, one enuresis]) > on-time maturers (5%; $n = 1$ [enuresis]); ns. Symptoms: early maturers > on-time developers for parent-reported somatic complaints ($M = 56.8$; 51.4) and self-reported depressive symptoms ($M = 10.2$; 5.4) -Trends: early maturers > on-time developers for self-reported state ($M = 46.6$; 44.4) and trait ($M = 56.2$; 48.7) anxiety, as well as parent-reported anxious-depressed symptoms ($M = 55.4$; 52.2). **Association between hormones and anxiety not examined.
Dom, Susman, & Pomirakis (2003)	NIMH Study described above	Pubertal Timing: <i>Tanner Stage Adjusted for age:</i> Self-reported (SR) Parent-reported (PR) Practitioner-evaluated (PE) Anxiety @ each assessment: -Emotional Tone (OSIQ) -CBCL (Girls: anxious-obsessive; somatic; depressed-withdrawn; total internalizing symptoms; Boys: obsessive-compulsive; somatic; and total internalizing symptoms)	Concurrent analyses -Boys: no significant findings at T1 or T2. Compared to early-maturers, late maturing boys (SR) evidenced higher internalizing problems ($R^2 = .09$; $\beta = -.30$) at T3. -Girls: no significant findings at T1 or T3. Compared to early-maturers, late maturing girls (PR) evidenced poorer emotional tone ($R^2 = .10$; $\beta = -.32$) at T2. Prospective analyses (controlling for T1 symptoms) -pubertal timing unrelated to subsequent anxious or depressed symptomatology
Ge, Brody, Conger, & Simons (2006)	867 African American youth (467 girls) ($M_{age} = 10.5$; range: 10–12 years) <i>Cross-sectional</i>	Pubertal Timing: pubertal status (PDS) adjusted for age; trichotomized using sample-specific means Anxiety: parent and child-reported symptoms of social anxiety, generalized anxiety, and major depressive disorder [DISC-4.0]	(**Pattern of zero-order correlations between timing and self and parent-reported symptoms nearly identical to that reported for pubertal status in Table 1) Boys: early maturers > on time and late maturers for self-reported symptoms of SA ($M = 4.55$; 3.59; 3.28, respectively), GAD ($M = 4.27$; 3.43; 2.76) and MDD (7.53; 5.84; 4.97) Girls: early maturers > on time maturers for self-reported symptoms of GAD ($M = 4.20$; 3.64); late maturers < early and on time for MDD (5.04; 6.16; 7.19)
Ge, Conger, & Elder (1996)	216 girls grades 7–10 <i>Four-year longitudinal study assessed annually</i>	Pubertal timing: retrospective self-report of age at menarche (at 4 th wave) -early-maturers: menses before age 12.5 years; -on-time: menses 12.5–13.5; -late-maturers: menses > 13.5 Anxiety: -Internalized Distress (described above) at all 4 waves -Teacher ratings of adolescent distress (anxiety/depression subscale; TRF) at 4 th wave -Parent report of adolescent anxiety/depression (3 items from the NEO-PI); at 4 th wave *Self-report of deviant peer association (DPS) at 3 rd wave*Self-report of peer group sex composition (all waves) *Paternal self-report of hostility (SCL-90-R; first 3 waves); observation of hostile behavior at 3 rd wave	Main Effects: At 2 nd and 3 rd waves (8 th and 9 th grade), early-maturing girls ($M = 3.25$; 3.35) reported more psychological distress than both on-time ($M = 2.5$; 2.7) and late maturers ($M = 2.2$; 2.2). At final wave, early maturers ($M = 3.4$) more distressed than late maturers ($M = 2.2$). Interaction Effects: Composition of peer groups and timing (four groups) compared to on-time and late maturers (combined) with all-girl (peer groups), early maturers with all-girl, and on-time/late with mixed sex, early maturing girls with mixed-sex peer groups (composite across waves 1–3) evidenced highest father-reported ($M = 6.2$; 5.5; 5.7; 6.5, respectively) and mother-reported ($M = 5.8$; 5.2; 5.6; 6.0), but not teacher-reported ($M = 2.5$; 3.2; 2.6; 2.7) distress (adolescent self-report of internalized distress not examined). Prospective Findings -Predictors of Wave 4 Distress (composite of all informant ratings): -T1 distress related to T4 distress for early maturing girls ($\beta = .37$) but not on-time/late youth ($\beta = .10$). -T1 distress related to deviant peers among early maturing ($\beta = .31$), but not on-time/late maturing ($\beta = .15$) girls. Association with deviant peers, in turn, predicted T4 distress among early maturing ($\beta = .20$) but not on-time/late maturing ($\beta = -.06$) girls.

Authors	Participants/Study Type	Measures of Puberty and Anxiety	Findings
Ge, Conger, & Elder (2001)	202 boys 12–14 years at Time One ($M_{age}=12.68$; $SD=.55$) <i>Four-year longitudinal study</i>	Pubertal Timing: pubertal status (PDS) adjusted for age; trichotomized using sample-specific means Anxiety: Internalized distress (described above) *Concurrent life events in past 12 mos. (JHLES)	-Father's hostile feelings predicted T4 distress among early-maturing ($\beta=.32$) but not late/on-time youth ($\beta=.08$) Group Comparisons: Early-maturing boys > on-time, late developers at grades 8 ($M=63$; $M=57.42$; $M=51.28$, respectively) and 10 ($M=63.63$; $M=53.06$; $M=53.33$) Regression Analyses: after controlling for distress at grade 7 and concurrent life events, the interaction between life events and pubertal timing predicted distress in grades 8 ($\beta=2.22$; $\Delta R^2=.08$; [total model $R^2=.41$] and 10 ($\beta=2.19$; $\Delta R^2=.08$ [total model $R^2=.16$]) – early maturing boys with greater life stress reported highest distress.
Graber, Brooks-Gunn, & Warren (2006)	100 girls 10–14 years ($M_{age}=12.1$, $SD=.8$) <i>Cross-sectional</i>	Pubertal timing: -Tanner stages (practitioner evaluated); dichotomized using national norms Anxiety: Anxious-obsessed symptoms (YSR) *Depressive-withdrawal symptoms (YSR)	Descriptive: Early maturers (girls) > non-early maturers for anxious ($M=5.42$; 3.74) and depressive ($M=8.67$; 6.14) symptoms Path Models: Early maturation linked with anxious symptoms ($\beta=.22$), which in turn predicted depressive symptoms ($\beta=.55$ [anxious symptoms mediated link between timing and depression, reducing β from .26 to .14, ns])
Graber, Brooks-Gunn, & Warren (1995)	75 girls 10–14 years ($M_{age}=11.9$; $SD=.8$) <i>Longitudinal (sub-sample of pre-menarcheal girls drawn from a larger study)</i>	Pubertal timing: -Age at menarche Anxiety: -Internalizing symptoms (YSR)	Internalizing symptoms among pre-menarcheal girls at Time one predicted later age at menarche ($r=-.25$; $p<.05$).
Graber, Lewinsohn, Seeley, & Brooks-Gunn (1997)	1,709 adolescents (890 girls) 14–18 years ($M_{age}=16.6$; $SD=1.2$) <i>Cross-sectional</i>	Pubertal timing: Perceived pubertal timing (PDS); Anxiety Structured Clinical Interview (adapted K-SADS) *Internalizing Symptoms (self-reported state anxiety, worry, hypomanic behavior, sleep problems, and hypochondriasis) *Depressive symptoms (CES-D)	-Internalizing symptoms: no differences across groups for girls; late-maturing boys ($M_{score}=28$) higher than on-time developers ($M=-.09$), -Depressive symptoms higher among early maturing boys ($M=.14$) and girls ($M=.18$), compared to on-time boys ($M=-.07$) and girls ($M=-.05$). Late-maturing boys ($M=.18$) also higher than on-time maturers. -Psychiatric disorders *Anxiety Disorders: no significant effects *Major depression: early maturing > on-time girls (30.2% vs. 22.1%; Wald test = 5.01); late maturing > on-time girls (33.8% vs. 22.1%; Wald test = 4.71) *Any (lifetime): early maturing > on-time girls (52.9% vs. 37.8%; Wald test = 12.69).
Graber, Seeley, Brooks-Gunn, & Lewinsohn (2004)	931 young adults (539 women) ($M_{age}=24.4$; $SD=.6$) <i>Follow-up study: 6–10 years after Time one</i>	Pubertal timing: Perceived pubertal timing (PDS); Anxiety Structured Clinical Interview (Longitudinal Interval Follow-up Evaluation)	- Early maturing young women , compared to on-time developers, higher prevalence of lifetime anxiety (30.3% vs. 20.0%; OR: 1.7), depression (66.5% vs. 49.4%; OR: 2.0), disruptive behavior (11.3% vs. 3.2%; OR: 3.9), any Axis I (86.0% vs. 70.6%; OR: 2.6), and anti-social personality (6.5% vs. 2.7% OR: 2.5) disorders. -Late maturing young men, compared to on-time developers higher lifetime prevalence rates of disruptive behavior disorder (19.1% vs. 10.0%; OR: 2.1) and current substance use (19.0% vs. 8.5%; OR: 2.5).
Hayward et al. (1997)	758 girls Grades 6–8; (Time one $M_{age}=12.8$; $SD=.9$) <i>Six-year Longitudinal</i>	Pubertal Timing: Age at which Tanner III was reached Anxiety Panic attack history (SCID) – assessed yearly *Internalizing symptoms: above cutoff for Eating disorder symptoms (EDI), depressive symptoms (CES-DC), or having had a panic attack (assessed yearly)	-Subjects with onset of internalizing symptoms during study reached midpoint of puberty ~5 mos earlier than those without (log-rank $\chi^2=14.4$) - Early maturers (girls) were more likely to develop internalizing symptoms (hazard ratio [HR]: 1.8) than non-early maturers - Early maturers more likely to have had a panic attack (HR: 3.1), as well as eating disorder (HR: 2.7) and depressive (HR: 1.5) symptoms. -Trend for early maturers to develop an internalizing disorder ($\chi^2=2.8$, $p=.099$; OR = 1.7) -Interaction: early maturers with a positive history of internalizing symptoms evidenced highest overall risk of developing an internalizing disorder (OR: 3.3; [Percentages for Panic Disorder specifically: non-early puberty + no history of symptoms (0%); early puberty + no history (2.4%);

Authors	Participants/Study Type	Measures of Puberty and Anxiety	Findings
Jones & Bayley (1971)	16 late-maturing 16 early-maturing boys (ages 12–17 years) 4.5 year longitudinal study	*Internalizing disorders: bulimia nervosa, social phobia, depression, agoraphobia (SCID); final assessment Pubertal timing -skeletal age assessments made twice yearly Anxiety -Social anxiety (naturalistic observation of participants in free-play context with same-sex peers over nine semesters)	puberty + history of symptoms (3.6%); early puberty + history of symptoms (6.2%)) Compared to early-maturers, late maturing boys were, on average, significantly more tense in the social context ($M_{overall} = 40.6$ and 61.1 respectively on mean standardized [T] scores of “relaxation”; age 12 ($T = 55$ [early]; 45[late]), age 12.5 ($T = 55$; 46), age 13 ($T = 56$; 48), age 13.5 ($T = 53$; 47), age 14 ($T = 53$; 48), age 14.5 ($T = 54$; 52), age 15 ($T = 56$; 51), age 16 ($T = 60$; 40), age 17 ($T = 58$; 45)
Kaltiala-Heino, Marttunen, & Rimpela (2003)	36,549 Finnish adolescents (19,196 girls) (14–16 years) $M_{age} = 15.3$; $SD = .59$; <i>Cross-sectional school-based survey</i>	Pubertal timing: self-report of age at menarche/oigarche Anxiety: -single author-developed question about general anxiety -Psychosomatic symptoms in past 6 months (e.g., nervousness; headache) *Depression (R-BDI)	Girls: compared to a late reference group (menarche ≥ 15 years), earlier menarche associated with anxiety; the effect decreased with age (< 10 years onset, OR: 3.4; age 11 onset, OR: 2.3; age 12 onset, OR: 1.6; age 13 onset, OR: 1.6; age 14 onset, OR: 1.4). Boys: compared to a late reference group (oigarche ≥ 15 years) earlier oigarche associated with increased anxiety, decreased w/age (< 10 years onset, OR: 4.2; age 11 onset, OR: 1.4; age 12 onset, OR: .8; age 13 onset, OR: .7; age 14 onset, OR: .7) Nearly identical pattern for psychosomatic and depressive symptoms.
Kim & Smith (1998a)	28 English girls (12–15 years) $M_{age} = 14$, 3 and their mothers <i>Cross-sectional</i>	Pubertal timing: self-reported age at menarche Anxiety: retrospective recall of anxiety (between 7–11 years): single Likert-type question (author-developed) *Depression: same format	Linear Regression Correlations: Earlier menarche associated with increased anxious ($\beta = .49$; adj. $r^2 = .21$), but not depressive symptoms. Path Model: childhood anxiety and depression not related to age at menarche. Three-way interaction between the maternal variables of age at menarche, age at first intercourse, and number of intercourse partners interacted to predict daughter's age at menarche
Kim & Smith (1998b)	357 Canadian students (18–24 years) 268 girls ($M_{age} = 20.5$) 89 boys ($M_{age} = 20.1$) <i>Cross-sectional, retrospective</i>	Pubertal timing: self-report of age at menarche/spermarche Anxiety: retrospective recall of anxiety (between 8–11 years): single Likert-type question (author-developed) *Depression: same format *Internalizing symptoms (composite anxiety/depression)	Linear Regression Correlations: less childhood anxiety ($\beta = .13$; adj. $r^2 = .01$) and fewer internalizing symptoms ($\beta = .13$; adj. $r^2 = .01$) predicted earlier menarche . No effects for depression for girls. No effects for boys.
Kim & Smith (1999)	509 English students (16–23 years) 341 girls ($M_{age} = 18.6$; $SD = 1.38$) 168 boys ($M_{age} = 18.7$, $SD = 1.47$) <i>Cross-sectional; retrospective</i>	Pubertal timing: self-report of age at menarche/spermarche Anxiety: internalizing symptoms as assessed in Kim & Smith (1998)	No association between childhood internalizing symptoms and earlier menarche. Fewer childhood internalizing symptoms predicted earlier spermarche ($\beta = -.16$)
Meschke, Johnson, Barber & Eccles (2003)	412 boys 484 girls Time One age 9.7 – 12.9 years <i>Seven-year longitudinal study</i>	Pubertal Timing: retrospective report of grade and season during which participant experienced growth spurt (at Wave 5) Anxiety: self-reported math, social, and economic anxiety (e.g., “do you worry that you will not be able to get a good job when you are an adult?” author-developed; Wave 1)	Boys: School anxiety in late childhood predicted delayed puberty (risk ratio = .78); economic anxiety predicted earlier puberty (risk ratio = 1.14). Girls: No significant findings.
Peskin (1967)	40 boys (ages 5 – 16) -22 early maturing boys (M_{age} at <i>pubertal onset</i> = 11 years) -18 late-maturing boys (M_{age} at <i>pubertal onset</i> = 12.4 years)	Pubertal timing -skeletal age at age 17 -Pubertal onset: one year prior to detectable secondary sexual characteristics (yearly photographs taken) Anxiety	Comparisons using t-tests (means not provided) made at 10 equivalent points of maturational age (i.e., 5 years before and after pubertal onset): - Early maturing boys significantly more anxious compared to late-maturing boys at two years post pubertal onset -No differences at any of the other comparisons -Nearly identical pattern for “sombeness”

Authors	Participants/Study Type	Measures of Puberty and Anxiety	Findings
Petersen & Crockett (1985)	12-year longitudinal study (annual assessments) 143 girls (grade 6) 118 boys (grade 6) Age at study onset not reported 3-year cohort-sequential study	-Manifest anxiety (e.g., worry about being liked, getting good grades) and somberness (e.g., low sociability, expressiveness) based on interview ratings Pubertal timing: -Age at peak height velocity Anxiety: OSIQ Emotional Tone subscale	No relation between pubertal timing and emotional tone; sex did not interact with pubertal timing to predict emotional tone.
Silbereisen & Kracke (1997)	Four groups (by age): 224 boys (age 13–14) 286 girls (age 13–14) 222 boys (age 15–16) 218 girls (age 15–16) <i>Cross-sectional (German Shell Youth Study)</i>	Pubertal timing: -Perceived maturational timing (i.e., faster/slower than peers) among young (13–14 years) and old (15–16 years) youth -“Actual” maturational timing: -girls’ age at menarche -boys’ age at peak height velocity Anxiety & Depression -3 items from GGHQ (e.g., “I feel nervous ...”) for anxiety and depression	Boys: “perceived timing” -compared to all other groups, 13–14 year old “off-time” boys had highest anxiety symptoms (data not reported) no differences for depression; “Actual” timing - Early maturing boys in the 15–16 age group > on-time maturers for anxiety ($M = 1.63$; 1.32) and depressive ($M = 1.43$; 1.19) symptoms. No differences for the younger group. Girls: “perceived timing” - no differences for depression or anxiety. “Actual” timing - Early maturing girls in the 13–14 age group > late maturers ($M = 1.62$, 1.37 respectively) for anxiety symptoms. No differences for anxiety in the older group or for depressive symptoms across groups.
Sonis et al. (1985)	33 girls with TPP (6–11 years) $M_{age} = 8.1$ years; 33 matched (age, sex, race) controls from CBCL standardization sample <i>Cross-sectional</i>	Pubertal Timing: practitioner diagnosis of TPP Anxiety: Parent-reported internalizing problems; somatic complaints, social-withdrawal, depressive, and schizoid-obsessive symptoms (CBCL)	Compared to girls without TPP, females with TPP (early maturers) evidenced significantly greater internalizing symptoms ($M_{standardized\ T\ score} = 63.4$; $M = 50.2$, respectively), including depressed ($M = 62.7$; $M = 57.5$), social withdrawal ($M = 67.3$; $M = 57.3$), somatic complaint ($M = 64.2$; $M = 58.6$), and schizoid obsessive ($M = 61.4$; $M = 56.8$) symptoms.
Sonis et al., (1987)	77 youth (55 girls) with TPP (4–11 years; $M_{age} = 6.69$, $SD = 1.81$ years) <i>Cross-sectional</i>	Pubertal Timing: practitioner diagnosis of TPP; evaluation of pubic hair and breast stage Anxiety: Parent-reported internalizing problems (CBCL)	Internalizing symptoms negatively related to Height/Chronological Age Ratio ($r = -.51$) and pubic hair stage ($r = -.40$); no association with breast stage.
Susman et al. (1985)	NIMH sample described above <i>Cross-sectional analysis</i>	Pubertal Timing: Tanner stage adj., for age Anxiety: Emotional Tone and Psychopathology subscales (OSIQ)	Boys: emotional tone was related to late maturation ($\beta = .24$; $R^2 = .11$) Girls: no effects for girls.

Note. Unless otherwise noted, findings are statistically significant at conventional levels. Bolded phrases reflect significant associations with anxiety-relevant outcomes. CBCL: Child Behavior Checklist (Achenbach, 1981; 1991); CDI: Children’s Depression Inventory (Kovacs, 1992); CES-D: Center for Epidemiologic Studies Depression Scale (Radloff, 1977); CES-DC: Center for Epidemiologic Studies Depression Scale-Child Version (Fendrich, Weismann, & Warner, 1991); DISC: Diagnostic Interview Schedule for Children (Costello, Edelbrock, & Costello, 1985); Diagnostic Interview Schedule for Children, Version 4 (Schaffer et al., 1993); DPS: Deviant Peers Scale (Elliott, Huizinga, & Ageton, 1985); EDI: Eating Disorders Inventory (Garner, Olmstead, & Polivy, 1983); GGHQ: Goldberg’s General Health Questionnaire (Goldberg, 1972); JHLES: Junior High Life Experiences Survey (Swearingen & Cohen, 1985); K-SADS: Schedule for Affective Disorders and Schizophrenia for School-Age Children (Orvaschel, Puig-Antich, Chambers, Tabrizi, & Johnson, 1982); Longitudinal Interval Follow-up Evaluation (Keller, Lavori, Friedman, Nielsen, Endicott, et al., 1987); NEO-PI: Neuroticism, Extroversion, and Openness-Personality Inventory (Costa & McCrae, 1985); OSIQ: Offer Self-Image Questionnaire (Offer, Ostrov, & Howard, 1977); PDS: Pubertal Development Scale (Petersen et al., 1988); R-BDI: Beck Depression Inventory, short form, Finnish version (Beck & Beck, 1972); Kaltiana-Heino, Rimpela, Rantanen, & Laippala, 1999); SCID: Structured Clinical Interview for DSM-III-R Non-Patient Version (Spitzer et al., 1987); SCL-90-R: Symptom Checklist 90—Revised (Derogatis, 1983); STAI: State-Trait Anxiety Inventory (Spielberger, Gorsuch, & Lushene, 1970); Tanner stage: Tanner stages of pubertal development (Tanner, 1962); TPP: True Precocious Puberty; TRF: Teacher Report Form (Achenbach, 1991); YSR: Youth Self Report (Achenbach, 1995).

Table 3
Studies of Anxiety and Hormone Levels within the Context of Puberty

Authors	Participants/Study Type	Hormone Measures	Findings
Carrion et al. (2002)	51 trauma-exposed children (21 girls; 7–14 years; $M_{age} = 10.7$; $SD = 1.6$) 31 control participants (13 girls; 8 to 14 years $M_{age} = 10.9$; $SD = 1.9$) <i>Short-term (three-day) longitudinal</i>	Hormones: Salivary cortisol levels collected prebreakfast, pre-lunch, pre-dinner, and prebed over 3 days Pubertal status: Tanner stage [†] Anxiety: self- and interview assessed PTSD symptoms (PRI; CAPS-CA)	Trauma-exposed youth evidenced higher levels of salivary cortisol throughout the day; significant at pre-dinner ($M_{control} = 1.4$ $\mu\text{g/dL}$; $M_{trauma} = .17$) and prebed ($M_{control} = .04$; $M_{trauma} = .07$) No differences between children with and without co-morbid depression or, within the trauma-exposed group, with or without PTSD.
Coplan et al. (2002)	98 youth (47 girls) 9–17 years ($M_{age} = 13.6$; $SD = 2.5$) 62 participants had an anxiety/mood disorder; 36 were healthy controls <i>Cross-sectional</i>	Hormones: salivary cortisol levels at 4 points before and after CO ₂ inhalation Pubertal status: Tanner stage Anxiety: status determined via semi-structured interview Panic symptoms before and after a 5% CO ₂ inhalation (API)	Psychiatrically ill (PI) participants who panicked to the challenge had significantly elevated pre-CO ₂ salivary cortisol levels ($M = 2.69\text{ng/mL}$; $SD = 2.86$), compared to PI participants who did not panic ($M = 1.40$; $SD = 1.16$) and controls ($M = 1.41$; $SD = 1.23$). Pre-challenge cortisol unrelated to Tanner stage and subjective response to the challenge. Salivary cortisol concentrations did not increase in response to the challenge, regardless of group.
De Bellis et al. (1999)	18 youth with PTSD (8 girls ages 8 to 13 years; $M_{age} = 10.4$; $SD = 1.4$) 10 youth with OAD (6 girls ages 9 to 13 years; $M_{age} = 10.9$; $SD = 1.3$) 24 controls (9 girls ages 8 to 12 years; $M_{age} = 10.5$; $SD = 1.1$) <i>Cross-Sectional</i>	Hormones: 24-hour urinary free cortisol Pubertal Status: practitioner evaluated Tanner stage Anxiety: status determined via structured interview with parent and child (KSADS) CBCL *Depression: CDI	Cortisol levels were higher in youth with PTSD ($M = 57.24\mu\text{g/day}$) compared to controls ($M = 43.62$), but not different from youth with OAD ($M = 55.99$). Cortisol levels related to duration of abuse ($r = .28$), PTSD intrusive ($r = .35$) and hyperarousal ($r = .33$) symptoms, CBCL anxious-depressed T score ($r = .27$), and history of suicide attempts ($r = .32$).
Feder et al. (2004) ^{††}	124 participants (51 girls; 6–12 years) Depressed: $N = 75$ ($M_{age} = 9.3$; $SD = 1.5$), Anxiety disorders: $N = 31$ ($M_{age} = 8.7$; $SD = 1.7$) Controls $N = 17$ ($M_{age} = 9.2$; $SD = 1.1$)	Hormones: plasma cortisol sample every 60min for 24 hours starting at 9pm Pubertal Status: practitioner evaluated Tanner stage Anxiety: status determined via structured interview with parent and child (KSADS)	Anxiety disorders group had lower mean nighttime cortisol ($M = 4.2$ $\mu\text{g/dL}$) as well as 9pm to 8am AUC ($M = 2687.7$), and higher daytime maximum cortisol (13.2 $\mu\text{g/dL}$) compared to depressed ($M = 5.0$; 7540–5; 11.3) and control groups ($M = 5.6$; 7478.2; 10.8; latter two not different from one another). No other differences in 24-hour maximum, nighttime maximum, daytime mean, or other AUC analyses
Forbes et al. (2006)	Controls: 44 children (17 girls; $M_{age} = 10.36$; $SD = 1.43$); 32 adolescents (14 girls; $M_{age} = 13.44$; $SD = 1.61$) Depressed: 76 children (24 girls; $M_{age} = 10.36$; $SD = 1.42$); 40 adolescents (25 girls; $M_{age} = 13.98$; $SD = 1.36$) Anxious: 18 children (6 girls; $M_{age} = 10.49$; $SD = 1.38$); 14 adolescents (5 girls; $M_{age} = 13.38$; $SD = 1.99$) <i>Cross Sectional</i>	Hormones: plasma cortisol sample every 20min 2 hours before and 2 hours after sleep onset on second night of a three-night sleep evaluation Pubertal Status: practitioner evaluated Tanner stage Anxiety: status determined via structured interview with parent and child (KSADS)	Children with anxiety disorders ($M = 2.5$ $\mu\text{g/dL}$) had higher peri-sleep-onset mean cortisol than children with depression ($M = 1.0$ $\mu\text{g/dL}$; $\eta^2 = .20$) or controls ($M = 1.5$ $\mu\text{g/dL}$; $\eta^2 = .12$). Adolescents with depression ($M = 2.0$ $\mu\text{g/dL}$) had higher peri-sleep-onset cortisol than and children with depression ($\eta^2 = .12$), but not compared to anxious ($M = 1.8$ $\mu\text{g/dL}$) or control ($M = 1.5$ $\mu\text{g/dL}$) adolescents Repeated measures: children with anxiety had significantly elevated cortisol in the two hours preceding sleep than children with depression ($\eta^2 = .20$). No differences for the two hours after sleep onset or among adolescents
Graber, Brooks-Gunn, & Warren (2006)	100 girls 10–14 years ($M_{age} = 12.1$, $SD = .8$) <i>Cross-sectional</i>	Hormones: DHEAS (blood serum) E ₂ (blood serum)	Correlations: Anxious symptoms not associated with E ₂ ($r = .08$, <i>ns</i>) or DHEAS ($r = -.01$, <i>ns</i>); Depressive symptoms linked with E ₂ ($r = .27$) but not DHEAS ($r = -.03$, <i>ns</i>)

Authors	Participants/Study Type	Hormone Measures	Findings
Granger et al. (2003)	213 adolescents (107 girls; 11–17 years; $M_{age} = 13.66$; $SD = 1.53$) <i>Short-term (two-day)</i> <i>Longitudinal</i>	-Tanner stages (practitioner evaluated); dichotomized using national norms Anxiety: -Anxious-Obsessive YSR scale -Depressive-Withdrawal YSR scale Hormones: Testosterone (saliva samples collected at 7:30, 11:30, & 4:30 on two separate days) -Pubertal status: PDS Anxiety-Depression: (single latent variable) - Parent and youth reported: Anxiety/ Depression and Social Withdrawal (YSR/ CBCL) Diagnostic Status (DISC-4.0)	Interaction: (Trend: anxiety) -early maturing girls with high DHEAS had highest anxious ($M = 6.4$) and depressed ($M = 10.0$), compared to all other girls ($M_{range} = 3.5 - 3.9$; $p < .07$; $5.40-6.80$; $p < .05$). No significant findings for E_2 . Boys: higher anxious/depressive symptoms related to T level ($\beta = -.58$) and diurnal slope ($\beta = .85$) Girls: no association between T, T slope, and anxious-depressed symptoms.
King, Mandansky, King, Fletcher, & Brewer (2001)	10 sexually abused girls ($M_{age} = 6.4$; SD $= .9$) 10 control girls ($M_{age} = 5.8$; $SD = 1.3$)	Hormones: pre-exam salivary cortisol between 9–11am Pubertal Status: Physician evaluated Tanner Stage Anxiety: PTSD symptoms via parent and child interview (PSSC)	Morning salivary cortisol levels were significantly lower in abused ($M = .5$ nmol/L) versus non-abused ($M = 1.1$ nmol/L)
Nottelmann et al. (1987)	NIMH Study described above <i>Cross-sectional analysis</i>	Hormones: LH; FSH; T; E_2 ; E, DHEA, DHEAS; Δ 4-A, T/ E_2 ratio; three blood serum levels taken 20 min apart between 8am and 10am -Practitioner assessed genital stage for boys; breast stage for girls Anxiety -Maternal report (CBCL: scales described in Dom et al., 2003 except total internalizing) -Emotional tone (OSIQ)	Boys: T/ E_2 ratio related to emotional tone ($\beta = -.54$); Δ 4-A related to obsessive compulsive symptoms ($\beta = .61$). No associations for somatic complaints. Girls: no significant findings.
Olweus, Mattsson, Schalling, & Low (1980)	58 Swedish boys (15–17 years; $M_{age} = 16$) <i>Three to six week longitudinal</i>	Hormones -Plasma T assays; two morning draws 3 – 6 weeks apart -Practitioner evaluated Tanner Stage Anxiety: -MCA (1 month before blood draw) -adolescent worry about venipuncture (author-developed) -nurse ratings of anxiety during venipuncture	No relation between T and trait-like somatic anxiety ($r = .03$, ns); psychic anxiety ($r = .10$, ns), muscular tension ($r = .07$, ns), or total anxiety ($r = .07$, ns) No relation between T and self-reported anticipatory worry ($r = -.03$, ns), current worry ($r = .06$, ns), unpleasantness ($r = .11$, ns) or nurse ratings of participant anxiety ($r = -.21$, ns) during initial venipuncture Tanner stage not examined in relation to anxiety variables.
Susman, Dorn, & Chrousos (1991)	NIMH sample described above <i>Cross sectional and prospective analyses</i>	Hormones: same as Nottelmann et al. (1987) with the addition of cortisol and CBG -Practitioner assessed genital stage for boys; breast stage for girls Anxiety: indices described in Susman et al., 1991 (Table 1)	Concurrent Findings: -Boys: emotional tone related to T ($R^2 = .09$; $\beta = -.53$) and Δ 4-A (R^2 $= .10$; $\beta = .39$) at T1 and E_2 ($R^2 = .20$; $\beta = -.35$) at T3. T ($R^2 = .10$; $\beta =$ $-.48$) also related to depressive symptoms (assessed at T3 only). No other findings; no associations between maternal reported or interview- assessed anxious symptoms and hormones. -Girls: emotional tone related to T ($R^2 = .09$; $\beta = .36$) at T3 Internalizing symptoms related to DHEAS ($R^2 = .11$; $\beta = -.33$) at T1. Cortisol related to anxious symptoms ($R^2 = .12$; $\beta = .36$; assessed at T3 only) Prospective Findings (T1 hormone levels $\rightarrow$ T3 symptoms): -Boys: internalizing problems related to LH ($\Delta R^2 = .05$; $\beta = -.23$) and cortisol ($\Delta R^2 = .05$; $\beta = -.22$); depressive symptoms related to DHEA

Authors	Participants/Study Type	Hormone Measures	Findings
Susman, Dorn, Inoff-Germain, Nottelmann, & Chrousos (1997)	36 youth from NIMH sample who completed longitudinal assessments (15 girls; 9–14 years; $M_{age} = 11.35$; $SD = 1.52$; 21 boys; 10–15 years; $M_{age} = 12.64$; $SD = 1.45$) <i>Prospective analysis</i> ; 0, 6, and 12 months	Hormones: cortisol drawn at 0, 20, and 40 minutes beginning at 8am Pubertal Stage; practitioner evaluation, parent and child Tanner Stage ratings Anxiety: observer-rated indices of behavioral distress (including fear) during blood draw (author-developed) Emotional Tone (OSIQ) Anxiety and depressive symptoms (DISC); T3 only	($\Delta R^2 = .08$; $\beta = .29$); anxious symptoms related to DHEA ($\Delta R^2 = .12$; $\beta = .35$) and $\Delta 4-A$ ($\Delta R^2 = .17$; $\beta = .41$) -Girls: anxious symptoms related to LH ($\Delta R^2 = .07$; $\beta = .27$) and DHEAS ($\Delta R^2 = .08$; $\beta = -.29$). No effect of cortisol reactivity (i.e., stable increasing, or decreasing across draws at Time One) on behavioral distress elicited by blood draws at subsequent assessments (6 month and 12 month) No effect of cortisol reactivity on emotional tone or anxious symptoms assessed by the DISC. Youth in the “increase” group ($M_{total} = 14$) evidenced higher depressive symptoms compared to those in the “decrease” ($M_{total} = 7$) or “stable” ($M_{total} = 7$) groups
Susman et al. (1985)	NIMH sample described above <i>Cross-sectional analysis</i>	Hormones: identical to Nottelmann et al (1987) -Timing: hormone level for age and Tanner stage adj. for age Anxiety: Emotional Tone and Psychopathology subscales (OSIQ)	Boys: emotional tone was related to T/E ₂ ($\beta = -.34$; $R^2 = .14$) and $\Delta 4-A$ ($\beta = .21$; $R^2 = .06$). Psychopathology was related to FSH ($\beta = .24$; $R^2 = .08$), E ₂ ($\beta = -.25$; $R^2 = .09$), T/E ₂ ($\beta = -.30$; $R^2 = .12$), and $\Delta 4-A$ ($\beta = .24$; $R^2 = .10$) Emotional tone also related to early maturation defined by higher-for-age E ₂ ($\beta = -.19$; $R^2 = .06$), T/E ₂ ($\beta = -.26$; $R^2 = .11$), and $\Delta 4-A$ ($\beta = .28$; $R^2 = .12$). Psychopathology related to early maturation defined by higher-for-age E ₂ ($\beta = -.27$; $R^2 = .12$), T/E ₂ ($\beta = -.26$; $R^2 = .11$), and $\Delta 4-A$ ($\beta = .30$; $R^2 = .16$) Girls: psychopathology related to DHEAS ($\beta = -.20$; $R^2 = .08$) Emotional tone related to early maturation defined by higher-for-age FSH ($\beta = .37$; $R^2 = .20$). Psychopathology related to early maturation defined by higher-for-age FSH ($\beta = .29$; $R^2 = .21$) and DHEAS ($\beta = -.17$; $R^2 = .07$).

Note. Unless otherwise noted, findings are statistically significant at conventional levels. $\Delta 4-A$: androstenedione; API: Acute Panic Inventory (Dillon, Gorman, Liebowitz, Fyer, & Klein, 1987); CAPS-CA: Clinical Administered PTSD Scale-Child and Adolescent Version (Nader et al., 1996); CBCL: Child Behavior Checklist (Achenbach, 1981; 1991); CBG: cortisol binding globulin; CDI: Children's Depression Inventory (Kovacs, 1992); DHEA: dehydroepiandrosterone; DHEAS: dehydroepiandrosterone sulfate; DISC: Diagnostic Interview Schedule for Children (Costello, Edelbrock, & Costello, 1985); Diagnostic Interview Schedule for Children, Version 4 (Schaffer et al., 1993); E: Estrogen; E₂: Estradiol; FSH: Follicle Stimulating Hormone; K-SADS: Schedule for Affective Disorders and Schizophrenia for School Age Children – Present and Lifetime versions (Kaufman et al., 1997); MCA: Multi-Component Anxiety Inventory (Schalling, Cronholm, & Asberg, 1975); LH: Luteinizing Hormone; OSIQ: Offer Self-Image Questionnaire (Offer, Ostrov, & Howard, 1977); OAD: overanxious disorder; PDS: Pubertal Development Scale (Petersen et al., 1988); PRI: Post-Traumatic Stress Disorder (PTSD) Reaction Index (Nader et al., 1996); PSSC: Psychosocial Schedule for School Aged Children (Lukens et al., 1983) T: Testosterone; Tanner stage: Tanner stages of pubertal development (Tanner, 1962); YSR: Youth Self Report (Achenbach, 1995).

* Unless otherwise noted, Tanner stage is self-reported by participants.

** Feder and colleagues (2004) combined datasets from two earlier studies of pre-pubertal youth (i.e., Birmaher et al., 1992; Puig-Antich et al., 1989), both of which failed to find differences in 24-hour cortisol secretion patterns between groups of healthy controls, anxious, and depressed participants. To limit redundancy, only the combined analysis is presented here.