

Puberty and Its Measurement: A Decade in Review

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Since the early 1980s, the focus on the importance of puberty to adolescent development has continued with variability in the methodology selected to measure puberty. To capture the relevant and important issues regarding the measurement of puberty in the last decade, this paper will address (1) the neuroendocrine aspects of puberty and its components, as well as the timing of puberty and its tempo; (2) why puberty is measured, including the relevance of puberty and its timing to health and development as well as the relevance of being off-time, that is, early or late with respect to a reference group; (3) the measurement of puberty and its methodology with respect to pubertal staging by physical examination, self-report measures, and their agreement with other methods and measures, hormones and their methods of measurement, and comparison of hormone concentrations to pubertal stage; and (4) recommendations for what is needed in the next decade regarding the measurement of puberty.

Puberty was recognized as early as ancient Greek history. In the scientific and lay literature of the past 60 years, there has been an awareness of the impact of puberty on multiple facets of adolescent development. The external physical changes of puberty were evident, and individuals in the adolescent's environment often surmised that pubertal development impacted emotional and behavioral issues as well as adjustment in the lives of adolescents. Importantly, however, empirical studies that objectively quantified puberty were not evident until the 1940s and 1950s, when Reynolds and Wines captured the physical changes of puberty in both girls and boys (Reynolds & Wines, 1948, 1951). This was followed by the classic works of Tanner and colleagues with the five-level graded categories of pubertal development for girls and boys (Marshall & Tanner, 1969, 1970; Tanner, 1962). "Tanner" staging remains the primary system used for pubertal staging.

Combining the measurement of puberty with key aspects of adolescent development (e.g., socioemotional, cognitive, behavioral) first became evident in the 1950s when Jones and colleagues (Jones & Bayley, 1950; Mussen & Jones, 1957) reported on the impact of timing of puberty on later development. In those reports, bone age was used as a measure of pubertal development. Studies then followed that examined linear growth and age at peak height velocity as a measure of puberty. It was not until the late 1970s that Petersen began her classic longitudinal study focusing on biopsychosocial changes in early ado-

lescents (Petersen, Tobin-Richards, & Boxer, 1983). With her investigations came the development of the Petersen Pubertal Development Scale (PDS; Petersen, Crockett, Richards, & Boxer, 1988), a self-report measure focusing on physical changes in growth and development that represents some of the early pubertal changes (e.g., breast development) but that is more heavily weighted toward those that become evident in mid- to late puberty for both boys (e.g., facial hair, voice change) and girls (e.g., menarche). During a similar time frame, studies examining puberty with psychosocial variables used self-report along with line drawings of maturational stages (Morris & Udry, 1980), whereas others conducted physical examinations for pubertal staging and collected blood for serum pubertal hormone concentrations (Brooks-Gunn & Warren, 1989; Nottelmann et al., 1987; Susman, Nottelmann, Inoff-Germain, & Dorn, 1987).

To capture relevant issues on the measurement of puberty in the last decade, this paper will address four primary areas. First, puberty will be defined and described in terms of the neuroendocrine aspects of puberty and its components as well as the relative timing of puberty and its tempo. Second, the rationale of why puberty is measured will be discussed, including the relevance of puberty and its timing in understanding health and development as well as its relevance to psychological and behavioral development. Third, the methodology of the measurement of puberty will follow. Specifically, pubertal staging by physical examination will be included along with self-report measures and their

agreement with other methods and measures, followed by methods of hormone measurement and comparison of hormone concentrations to pubertal stage. The paper will conclude with recommendations for what is needed in the next decade regarding the measurement of puberty.

WHAT IS PUBERTY?

Neuroendocrine Aspects of Puberty

Puberty is a process, not an event, that results from a complex series of coordinated neuroendocrine changes leading to internal and external physical changes in primary and secondary sexual characteristics and eventual reproductive competence. Puberty occurs between childhood and adulthood and is initiated in the brain after reactivation of the hypothalamic–pituitary–gonadal (HPG) axis. This sequence of events has been known for decades, first observed from lower animal models. Gonadotropin-releasing hormone (GnRH) neurons undergo reactivation from the previous fetal and neonatal periods via the GnRH pulse generator (Grumbach & Styne, 2003; Knobil, 1988; Plant, 2002). During the pre- and perinatal periods of development, an increase in gonadal steroids is responsible for sexual differentiation as well as organizing neural systems. In the first year of postnatal life, the GnRH pulse generator becomes quiescent until its reactivation before pubertal onset. As much as 1 year before the external changes of puberty are evident, reactivation of the secretion of GnRH occurs from the median eminence of the hypothalamus in a pulsatile fashion. In turn, GnRH stimulates the pituitary gland to release gonadotropins (luteinizing hormone [LH] and follicle-stimulating hormone [FSH]) into the circulation, first during sleep (Ojeda et al., 2006). The amplitudes of LH and FSH pulses increase. These increases eventually lead to production of estradiol and testosterone from the target tissues of the ovary and testes, respectively. With the stimulation of these gonadal steroid hormones, breast and uterine tissue, as well as the testes and phallus, increase in size and structure.

Regulation of the HPG axis occurs through an intricate feedback system that matures during puberty and, in healthy individuals, remains functional throughout the reproductive years. The mechanisms for reactivation of this system remain somewhat of a mystery, particularly in human adolescents. There are likely numerous permissive signals that trigger pubertal onset, including the hormones leptin and ghrelin as well as body composition (Sisk & Foster, 2004). New in this decade was the exciting discovery

of kisspeptins from the *KISS1* gene and its receptor, G protein-coupled receptor 54 (GPR54) or *KISS1r* receptor. Kisspeptin and *KISS1* are linked as regulators to reproduction and pubertal initiation (Banerjee & Clayton, 2007; de Roux et al., 2003; Seminara et al., 2003). In the brain, kisspeptin neurons signal and actually stimulate GnRH neurons; with respect to puberty, they are “triggering and guiding the tempo of sexual maturation” (Oakley, Clifton, & Steiner, 2009). Additional studies have examined adolescents with abnormal timing of puberty to determine a genetic component to timing of onset (Gajdos, Hirschhorn, & Palmert, 2009; Wehkalampi, Widen, Laine, Palotie, & Dunkel, 2008). Further knowledge of the triggers for onset and tempo of puberty is potentially relevant to understanding an association between psychological development and abnormal puberty. For example, males with constitutional delay and functional hypogonadotropic hypogonadism were noted to have attention deficit disorder (Sedlmeyer & Palmert, 2002).

Components of Puberty

There are two distinct yet overlapping components of puberty particularly relevant to its measurement (Grumbach, 2002): adrenarche and gonadarche. Adrenarche, or “awakening of the adrenal glands,” includes maturation of the adrenal gland and the ensuing rise of adrenal androgens (e.g., dehydroepiandrosterone [DHEA], its sulfate [DHEAS], and androstenedione; Grumbach & Styne, 1992). This rise occurs around ages 6 through 8 years in girls and about 1 year later in boys (Cutler et al., 1990; Parker, 1991). These androgens continue to rise during gonadarche and on into the third decade of life (Saenger & DiMartino-Nardi, 2001). Adrenal androgens are primarily responsible for axillary and pubic hair, but such development does not occur immediately when adrenarche begins. Adrenal androgens must reach concentrations high enough to meet the sensitivity of the target tissue at the hair follicles. Adrenal androgens also are considered neurosteroids (Maninger, Wolkowitz, Reus, Epel, & Mellon, 2009) and thus may contribute to behavior and psychological processes. Further, it is believed that onset of adrenarche is a necessary component for the subsequent occurrence of gonadarche.

Little is known about the mechanism for onset of adrenarche. Lack of knowledge may be due to the fact that adrenarche occurs only in higher primates, and thus lower animal models cannot be used to study its development. Belgorosky, Baquedano, Guercio, and Rivarola (2009) provided a recent

review on the development of adrenarche in which they indicated multiple factors are likely involved, including both local changes in the adrenal gland as well as peripheral metabolic changes. In particular, the review summarized newer information and focused on the growth hormone-insulin-like growth factor (GH-IGF) axis and insulin sensitivity as well as the estrogen receptor and GPR30 pathway. The field would benefit from further studies regarding adrenarche's trigger.

Gonadarche, the second component of puberty, occurs with reactivation of GnRH neurons (see the Neuroendocrine Aspects of Puberty section) and secretion of estradiol and testosterone. In gonadarche, the primary sex organs develop (ovaries, testes) and external signs of puberty (e.g., breast and genital development) begin, leading subsequently to reproductive competence. These external signs are used in the staging process of puberty as described by Tanner (Marshall & Tanner, 1969, 1970): Stage I, prepubertal; Stage II, breast and genital development indicating entry into puberty; and up to Stage V, full maturity. Pubic hair is also staged from I to V (see the Measurement of Puberty and Its Methodology section).

In considering the role that puberty may play in psychological and behavioral development, both adrenarche and gonadarche may be relevant. Each component represents a different endocrine axis as well as different external physical characteristics. Thus, it is important to consider the outcome variable when selecting a measure of puberty that represents one axis rather than the other. One should ask, does the outcome reflect the gonadal or adrenal axis? Rarely is it wise to combine the two stages by taking the average (e.g., breast and pubic hair).

Timing of Puberty

In the previous decade, the scientific community was stunned by a controversial publication proposing that the timing of puberty in girls occurred earlier than in the past (Herman-Giddens et al., 1997). This cross-sectional study, based on more than 17,000 girls in the United States, noted that puberty began as early as age 6 or 7 based on breast development, but age at menarche did not show a similar decline. The controversial methodological issues of the study have been articulated elsewhere (Emans & Biro, 1998; Reiter & Lee, 2001; Rosenfield et al., 2000), and some subsequent publications suggest minimal changes in age of onset of puberty (Sun et al., 2002). However, the majority of recent studies primarily conducted in the United States and Northern and Western Europe have begun to document an earlier

timing of puberty, particularly in girls. Ong, Ahmed, and Dunger (2006) described how the secular trend in age at menarche has slowed or stopped in many Western countries. However, they indicated that in subgroups with nutritional deprivation, age at menarche may continue to fall even as nutrition and social class improve in countries as they develop. In U.S. studies, the majority of a panel of experts agreed that puberty was occurring earlier in girls from 1940 to 1994 and was confirmed by Aksglaede et al. (2009), but insufficient evidence was available to note changes of pubertal onset in boys (Euling et al., 2008). Although the study methodologies may differ (e.g., pubertal measures), this secular trend is generally accepted in girls and is now thought to be relatively stable (e.g., little recent change) with respect to age at menarche in European girls (Parent et al., 2003). Specifically, pubertal onset in girls occurs earlier in African Americans compared with Caucasians, and age at menarche has not fallen at the same rate as onset of puberty.

The literature is more controversial surrounding the impact of timing of puberty on height and growth parameters. Most studies agree that later onset of puberty is associated with lower peak height velocity and pubertal height gain in girls (Vizmanos, Martí-Hennenberg, Cliville, Moreno, & Fernandez-Ballart, 2001). Age of pubertal onset affects the intensity and duration of the pubertal growth peak but not final height in girls (Biro et al., 2001; Vizmanos et al., 2001) and in boys (Vizmanos et al., 2001). The literature is mixed regarding timing of pubertal onset and adult height in traditional studies as well as in studies published in the last decade. For example, studies have reported no impact of timing of pubertal onset on adult height (Vizmanos et al., 2001), whereas others noted that early maturers are shorter as adults (Biro et al., 2001). Two recent publications were important in this decade and may shed some light on these apparent disparities. Bratberg, Nilsen, Holmen, and Vatten (2006) noted that early maturation led to shorter adult stature only in those with lower body mass index (BMI). Huang, Biro, and Dorn (2009) investigated relative timing through ordinal logistic regression and found that when menarche was used as the basis of timing, early-maturing girls were shorter as adults; however, when onset of breast development was used to determine timing, there was no impact of timing on adult height. As these publications demonstrate, the pubertal parameters selected by investigators may impact the conclusions. Although most investigators agree that earlier-maturing youth have a greater height velocity, BMI may interact with timing to impact final height

and may affect how investigators define timing (relative to which pubertal event is selected or which mathematical model is used to determine onset).

To make a cross-cultural comparison of timing of puberty, we will focus on some of the international studies pertaining to timing of puberty published in the last decade. Importantly, some of the studies were conducted in developing or transforming countries, whereas others represent more recent European studies that likely have not been reviewed elsewhere. Table 1 describes such studies for boys and girls, in which the majority shows an earlier pubertal onset.

Pathways of Puberty

In the vast majority of girls, thelarche (breast development) is thought to be the first visible secondary sexual characteristic followed by the appearance of pubic hair. In some cases, pubic hair may begin first or both breast and pubic hair may appear simultaneously. In boys, increase in testicular volume is generally first. Although most studies focus on thelarche as the first sign of puberty in girls, Biro and colleagues report two papers in this decade on the issue of thelarche and pubarche (pubic hair first) (Biro et al., 2006; Biro, Huang, Daniels, & Lucky, 2008). In the longitudinal National Growth and Health Study, Biro et al. (2003) examined two groups of White girls who had asynchronous development; that is, either thelarche or pubarche occurred first but not both simultaneously. They reported age of onset of maturation in the two groups was similar, but the thelarche group had greater adiposity and ponderosity at pubertal onset as well as throughout puberty. In a subsequent report of 9-year-old Black girls and White girls ($n = 478$) from the same data set, the authors argued that height velocities of the pubarche and thelarche groups did not differ and that both were in the pubertal range. Thus, both pathways may represent onset of puberty (Biro et al., 2008). Other investigators have examined the impact of pathway, confirming the relationship (Christensen et al., 2010; Schubert et al., 2005). It would be desirable for the National Children's Study to examine this issue with research-grade physical examinations because the study will be longitudinal and in a more recent cohort of both boys and girls.

Tempo of Puberty

Tempo of puberty (progression to established milestones after entry into puberty) is an understudied phenomenon in the psychosocial and medical liter-

ature. An inverse correlation exists between the onset of puberty and the interval between onset of puberty and menarche; that is, the tempo through puberty is longer with early maturers and shorter with late maturers. The correlation in girls is reported from $-.28$ (Biro et al., 2006) to $-.62$ (Martí-Hennenberg & Vizmanos, 1997). A similar association was noted by Pantisotou et al. (2008), although high attrition in the longitudinal study raises concern about a biased sample.

Few studies have examined the impact that tempo of puberty has on psychosocial development in adolescence. How quickly (or slowly) an adolescent progresses from one stage to another may have implications for his or her self-perception, perception by others, or even for alterations in mood or behavior considering that receptors for puberty-related hormones are evident in the brain. Ge et al. (2003) conducted an important study on African American boys and girls aged 10–12 years in which they reported boys with accelerated pubertal maturation across two times also showed the greatest increase in depressive symptoms. However, an alternate view was reported in which boys with accelerated pubertal maturation (aged 10–12 and 12–14 years) had a lower risk of depression (Laitinen-Krispijn, van der Ende, & Verhulst, 1999). Based on these disparities, it is important to further examine the impact of tempo on psychosocial development in the next decade.

WHY MEASURE PUBERTY?

Relevance of Puberty and Timing of Puberty to Health and Development

The importance of puberty and timing of puberty to adolescent health and development is not always recognized. Numerous studies examining biological phenomena or physical health outcomes have neglected to measure puberty and account for its contribution; thus, potentially erroneous conclusions in outcomes can be made. Several decades ago, Hein (1987) addressed the impact that puberty may have on metabolic and therapeutic effects of pharmacologic agents. She subsequently demonstrated how Tanner stage was associated with the half-life of an asthma medication and that chronological age alone was not the best way to determine dosage during puberty when rapid growth is occurring (Cary, Hein, & Dell, 1991). Still, only a few noteworthy studies are including pubertal stage by physical examination when testing a physiological hypothesis. With rare exception (see Feinberg, Higgins, Khaw, & Campbell,

TABLE 1
 Summary of International Studies Examining Onset of Puberty in Boys and/or Girls Published in the Last Decade (2000–2009)

Citation	Age at Enrollment (Years)	Country	Sample Size	Method	Age at Onset, Breast Stage	Age at Onset, Genital Stage	Age at Onset, Pubic Hair Stage	Age at Menarche
Zukauskaitė, Lasiene, Lasas, Urbonaite, and Hindmarsh (2005)	7.0–11.6	Lithuania	1,231	PE	10.2 ^a	<i>na</i>	11.2 ^a	<i>nr</i>
Hosny et al. (2005)	6.5–18.5	Egypt	1,550	PE	10.71	<i>na</i>	10.46	12.44, mean ^a
Garnier, Simondon, and Benfice (2005)	Mean 11.4 in 1995	Senegal (rural)	406	PE	12.6, median ^b	<i>na</i>	<i>nr</i>	15.9, median ^b
Juul et al. (2006)	6.0–19.9	Denmark	1,926	PE; testicular volume	10.88 ^{a,c}	11.83 ^{a,c}	11.29 girl; 11.88 boy	13.42 ^{a,c}
Mascaretti and Di Berardino (2008)	5.9–18.2	Italy	1,266	PE	9.2	11.39	9.10 girl; 11.74 boy	<i>nr</i>
Semiz, Kurt, Kurt, Zencir, and Sevinç (2008)	6.0–16.5	Turkey	3,311	PE; testicular volume	10.16	11.76	10.57 girl; 12.02 boy ^{a,c}	12.41, mean
Rabbani et al. (2008)	6–20	Iran	4,020	PE	10.15	<i>na</i>	10.48	14.54
Jones, Griffiths, Norris, Pettifor, and Cameron (2009)	Not stated	South Africa	607	PE at ages 9–10; self-report at ages 11 and up	10.1 Black; 10.2 White	10.4 Black; 9.8 White	Boys: 10.8 Black, 10.2 White; girls: 10.3 Black, 10.5 White	<i>nr</i>
Facchini et al. (2008)	7–18	Kazakhstan ^d	4,805	PE	<i>nr</i>	<i>nr</i>	<i>nr</i>	13.43 rural; 12.89 urban
Aksøglæde, Sorensen, Petersen, Skakkebaek, and Juul (2009)	5.6–20.0	Denmark	1,100 ^e 995 ^e	PE	10.88 ^c 9.86 ^e	<i>na</i>	11.29 ^c 11.09 ^e	13.42 ^c 13.13 ^e

Note. *nr* = not applicable; *nr* = not reported; PE = physical examination.

^aEstimated by probit analysis.

^bEstimated by log-normal parametric survival modeling.

^c1991–1993 cohort.

^dUrban versus rural sample.

^e2006–2008 cohort.

2006), the norm tends either toward neglecting measuring puberty or toward measurement by self-report. Understanding how the physiology of puberty may impact physical or mental health conditions, treatment regimens, or behaviors would enhance knowledge and improve outcomes.

In the psychosocial literature, puberty or timing of puberty has often been recognized as an important factor in various outcomes. Overall, earlier timing of puberty has been shown to be associated with or be a risk factor for negative psychological (e.g., depression) and behavioral (e.g., risky behaviors, acting out) outcomes, but there are exceptions. Puberty may be an important factor because it may impact the phenomenon under study either directly (e.g., brain changes occur at different times during puberty and in turn may impact neuropsychological function or behavioral changes) or indirectly (e.g., parents or peers notice external pubertal changes in an adolescent and in turn respond differently to the adolescent). Alternatively, it may be that underlying concentrations of sex hormones are influencing outcomes. Maturation can be studied directly through hormone concentrations or indirectly through pubertal staging as the hormones influence the degree of physical development. Thus, in all these cases, it is important to measure puberty accurately so as to understand whether puberty is contributing to the outcome under study. Many psychosocial studies have used self-report of pubertal status or timing, which sometimes has been appropriate. However, some psychosocial outcomes may have an underlying biological component (e.g., depression). We have argued for the necessity of selecting a measure of puberty that is congruent with the outcome under study (Dorn, Dahl, Woodward, & Biro, 2006). Unfortunately, many select the measure that is easier to collect rather than the measure that is more appropriate to the scientific and theoretical aspect of the study. In the following paragraphs, we provide examples of studies examining the relevance of "clinically early" onset of puberty along with those investigating the relevance of early puberty with respect to peers when examining physical as well as psychosocial outcomes.

Relevance of puberty that is clinically early. Two clinical examples that represent more extreme cases of early puberty are found in children manifesting either premature adrenarche (PA) or precocious puberty (PP). Although puberty is described in the most recent literature as occurring earlier than in past decades (e.g., breast development before age 6 in Black girls and before age 7 in White girls;

Kaplowitz & Oberfield, 1999), PA is still defined in the literature as occurring in girls 8 years of age or younger and in boys aged 9 years or younger (Siegel, Finegold, Urban, McVie, & Lee, 1992). PA is more common in girls than in boys. In PA, adrenal androgens are found in higher concentrations than in on-time adrenarche peers and, thus, pubic hair is evident. Additionally, body odor and acne may also be apparent as well as increased linear growth. It is generally believed that gonadarche and menarche do not present any earlier in girls with PA; notably, there may be some discrepancy in that contention.

PA is referred to as benign when no pathological source is determined (e.g., no pituitary or adrenal tumor). Although PA is usually considered benign, an expanding body of literature suggests otherwise (Ibáñez, DiMartino-Nardi, Potau, & Saenger, 2000). In brief, girls with PA are at higher risk for developing disorders in adulthood, including polycystic ovarian syndrome (PCOS) and/or metabolic syndrome, with a complex of other health issues and complications associated with both. More recently, girls with PA have been described as experiencing more mood disturbances and behavioral problems (Dorn, Hitt, & Rotenstein, 1999; Dorn et al., 2008), indicating that the disorder may have ramifications beyond physical health.

PP refers to early gonadarche, occurring as young as toddlerhood to age 6 years. As in PA, PP occurs more commonly in girls than in boys and can be the result of a tumor. However, the etiology of PP is often unknown, especially in girls. PP can be treated pharmacologically with agents that temporarily "turn off" the HPG axis. These medications include leuprolide acetate, given as an injection every 1 or 3 months, and histrelin acetate, a newer, long-acting implantable delivery system. Pharmacotherapy is stopped once bone age is concordant with chronological age and adult height prediction is significantly improved. If left untreated, children with PP are unlikely to reach their full height potential as adults. Psychological concerns also surface at the onset of PP and may continue into adolescence. Recent studies show that girls with PP have internalizing problems and poor self-image (Baumann et al., 2001; Officioso et al., 2000); see a recent review by Dorn (2007).

There is a lack of recent studies of PP and PA that examine psychosocial or cognitive issues. More in-depth studies with PP and PA children using bio-behavioral methodologies could enhance our understanding of normal puberty. Off-time PP and PA children may experience parallel changes as do youth who are older and experience on-time and normal development.

Relevance to Physical Health of Clinically Early Puberty That Is Not “Abnormal”

Puberty has a tremendous impact on health and well-being (Patton & Viner, 2007). Previous studies have noted that early onset of menarche is associated with risk of breast cancer; however, this association is weakened considerably when age at peak growth is included in the analysis. Menarche may serve as a proxy for age at peak growth, or both factors may reflect age at onset of puberty (Kindblom et al., 2006). Similarly, women who reached maximum height at a young age (under 12 years) had a much greater risk of breast cancer (Li, Littman, & White, 2007). Of note, younger age at peak growth is associated with greater growth velocity, as stated earlier. Additionally, tall as well as obese children have greater levels of IGF-1 in response to growth hormone, which may account for the increased risk of some cancers later in life (Bouhours-Nouet, Gatelais, Boux de Casson, Rouleau, & Coutant, 2007). Few studies report on the impact that puberty may express in physical health in males. An excellent review of the issues surrounding pubertal timing and physical health is available (Golub et al., 2007). Most of these studies use age at menarche and look at outcomes quite distal to puberty. Few look at the impact of puberty on more proximal health issues.

Relevance to Psychosocial Development of Early Puberty Not Clinically “Abnormal”

Extensive literature can be found regarding the impact of timing of puberty on psychosocial development. The majority of these studies continue to use a self-report measure of puberty regardless of the focus of the study. With rare exception (see Ellis & Essex, 2007), most literature focuses on timing of gonadarche rather than adrenarche. In the literature on girls, findings generally show that early timing of puberty is a risk for various affective states and negative behaviors (Mendle, Turkheimer, & Emery, 2007; Susman & Rogol, 2004). Over the past decade, such studies have continued their trend of more studies conducted in girls than boys, with few longitudinal studies evident (for exception, see studies by Angold and Costello [e.g., Costello, Sung, Worthman, & Angold, 2007]). We refer the reader to several excellent sources for reviews on the timing of puberty and its impact on psychosocial development (Angold & Costello, 2006; Negriff & Susman, in press; Reardon, Leen-Feldner, & Hayward, 2009). This past decade also brought with it more evidence on family influences of timing of puberty, including

the life history theory focusing on timing of puberty and the structure and processes in families (Ellis & Essex, 2007). For a review of family influences, see Susman and Dorn (2009).

MEASUREMENT OF PUBERTY AND ITS METHODOLOGY

Pubertal Staging

In the last decade, no new measures of puberty have emerged. Importantly, two reviews pertinent to measuring puberty appeared. First, Coleman and Coleman (2002) provided a review on the measurement of puberty. This was followed by a later paper that emphasized the necessity of measuring puberty, reviewed measures of pubertal status and timing, and characterized potential ways of determining the appropriate measure of puberty for a research study (Dorn et al., 2006). In the following paragraphs, we update the status of some of the issues pertaining to measuring puberty that have emerged or persisted in the last decade.

Dorn et al. (2006) addressed several issues that emerged in the literature regarding the actual measurement of puberty. First, the gold standard for measuring pubertal status continues to be physical examination by a trained clinician using the criteria attributed to Tanner (Marshall & Tanner, 1969, 1970), including staging of breast and pubic hair for girls and genital and pubic hair for boys. With respect to breast development, staging was originally done by visualization (Marshall & Tanner, 1970). The current consensus is that staging is best done by palpation and visualization so one can distinguish breast tissue from adipose tissue; as rates of obesity continue to rise, it becomes relevant to distinguish adipose tissue from breast tissue. In our review of the literature and in numerous discussions with investigators, continued reluctance lingers for measuring puberty by physical examination in situations that would recommend doing so. Numerous investigators state that parents and adolescents are not likely to consent to a physical examination or that other obstacles impede conducting them. This unfortunate lack of staging by physical examination hampers advancement in our understanding of puberty and its impact on many outcomes. Second, we and others have identified limitations in the photographs of breast, genital, and pubic hair used for pubertal staging. The Tanner photographs were taken decades ago and include only White youth. Although higher-quality photographs are available (van Wieringen, Roede, & Wit, 1985), to our knowledge, no studies are using them

in their research. Further, no photographs are available for pubertal stages of other races/ethnicities, limiting the examination of potential differences in characteristics. A third issue involves the methodological problems of measuring puberty that could enhance the reliability and reproducibility of studies. Frequently, methods sections contain scant information about training/certification and interrater agreement for physical examination or other information relevant to determining staging quality. When studies have used self-report, the specific methodology may also be missing, thus making it difficult to ascertain the quality of the article. By addressing all of these issues, the quality of pubertal research could be enhanced.

Self-Report Measures

Self-report continues to be a popular way to account for pubertal development in research studies, including reports by the adolescent using either photographs (Dorn, Susman, Nottelmann, Inoff-Germain, & Chrousos, 1990) or line drawings (Morris & Udry, 1980) of pubertal stages or using the PDS (Petersen et al., 1988), the most widely used measure of self-report over the years. Few studies in this decade have examined interrater agreement of self-report of puberty and assessment by physical examination by trained health care providers. In the paragraphs that follow, we review such studies but also cite studies that compare two types of self-report measures. Although the latter seems less relevant without a comparison with the gold standard of a physical examination, self-report does provide an estimate of maturation that often is biased.

Agreement of self-report and physical examination. Wu, Schreiber, Klementowicz, Biro, and Wright (2001) examined agreement of self-assessment of puberty (areolar and pubic hair) via line drawings with examiner assessments in 1,396 healthy girls aged 11–14 years. κ coefficients were relatively low and ranged from .32 to .55. In a second study of healthy boys and girls ($n = 240$), Desmangles, Lappe, Lipaczewski, and Haynatzki (2006) reported that κ coefficients were moderate for girls' ratings of breast and pubic hair ($\kappa = .49$ and $.68$, respectively) and boys' ratings of pubic hair ($\kappa = .49$). Genital staging was not reported in boys. Desmangles and colleagues suggest that self-report was not reliable and, therefore, not useful in studies of pubertal development when precise estimates are required.

Three international studies examined agreement of self-report and physical examinations for pubertal

stage. In the first study, reliability of the PDS was examined in a group of 10- to 18-year-old male and female Black, multiethnic South African youth (Norris & Richter, 2008). The investigators reported that the PDS was less reliable than physical examinations. Further, when comparing the PDS to sexual maturity ratings by examination, κ coefficients were very low in females and percent agreement was 26% in males. The investigators indicated that the features measured in the PDS may not always be relevant to this population or that progression of these indicators may vary in this population (Norris & Richter, 2008). To our knowledge, this is the first international study involving Black, multiethnic youth that examined agreement between physical exams and the PDS. Based on the study, investigators should be cautious in using the PDS without further testing in similar samples. The second international study examined agreement of physical examination with self-report in 354 Chinese children using line drawings and a brief explanation (Chan et al., 2008). κ coefficients were generally strong (.72 and .83 in girls and .58 and .80 in boys, respectively). The third study included a small sample of 47 male and female elite Canadian athletes who were 12–17 years of age (Leone & Comtois, 2007). Comparing physical examination to self-report by line drawings, κ coefficients were high for both boys ($\kappa = .79$, genital; $.67$, pubic hair) and girls ($\kappa = .85$, breast; $.75$, pubic hair). Agreement may have been influenced by the small size per cell and by the restricted range owing to large percentages in Stages I–II.

Three studies included adolescents with varied anthropomorphic characteristics. First, Bonat, Pathomvanich, Keil, Field, and Yanovski (2002) included overweight girls and boys aged 6–12 years. Of the 244 participants, 41% were defined as obese. Kendall rank correlations with physical examination and self-report for breast stage were $.37$ in obese and $.54$ in nonobese girls and for pubic hair were $.64$ in obese and $.66$ in nonobese girls. Boys' pubic hair stage had a correlation of $.45$ in obese and $.35$ in nonobese participants. The authors concluded that self-report was not very accurate for breast development, particularly in obese girls, and for pubic hair development in both groups of boys (Bonat et al., 2002). The second study included 100 children with Crohn's disease (aged 8–18 years). κ coefficients were high ($.74$ – $.85$) when comparing physician ratings to self-report by line drawings (Schall, Semeao, Stallings, & Zemel, 2002). Investigators emphasized that children and adolescents with Crohn's disease tend to be delayed in both linear and pubertal development and that even though they are off-time from their healthy peers,

their perceptions are on target. In another study, 87 boys and girls (aged 8–16 years) with type 1 or type 2 diabetes were recruited to examine the accuracy of self-report of puberty (Stephen, Bryant, & Wilson, 2008). A physical examination was conducted, and adolescents independently viewed drawings of the pubertal stages. κ coefficients were substantial ($> .61$) for both genders. Age, metabolic control, race, type of diabetes, and BMI had no impact on accuracy of assessment.

Based on the aforementioned studies, if one-to-one agreement between self-report and physical examination is required for research purposes, then self-reports may not be a valid and reliable measure. Many of these studies had very low κ coefficients, including the study with a sample size of nearly 1,400 (Wu et al., 2001). Also, in some subpopulations (i.e., obese), self-report of pubertal stage may not be a reasonable alternative. Investigators may want to consider these newer studies when weighing their proposed methodology for staging, considering that large samples were represented containing racial/ethnic diversity and that specific subgroups represented variability in weight and health issues. Two studies reported above appear to show that adolescents undergoing regular care for a chronic illness may possess more knowledge than others about how their disease affects growth and development; in turn, their agreement was higher with clinician ratings than self-ratings reported in other studies.

Agreement with different self-report methods. In 9- to 16-year-old boys and girls ($N = 2,864$), Bond et al. (2006) compared agreement between two methods of self-report: the PDS and sexual maturation using line drawings and written descriptions. κ coefficients for males ranged from .13 to .36 in grades 5, 7, and 9, but the κ was somewhat higher for the full sample ($\kappa = .42$). For girls, κ coefficients were lowest in grade 9 (.17 and .19 for pubic hair and breast, respectively); κ coefficients were .47 and .50 in the total sample. The investigators acknowledged that comparisons of the two self-report measures did not include a comparison with the “gold standard” of a physical examination by a health professional. Therefore, even with moderate κ coefficients, it is still not known whether the ratings were reflective of degree of physical maturation. Importantly, the study illustrates how two self-report measures can provide different ratings and, in turn, impact the conclusion of a study.

Self-report of age at menarche. We found no new information within the last decade regarding

methods to collect age at menarche, nor were studies reported in the literature regarding reliability of age at menarche across time or method. In the past, correlations ranged from .60 to .81 (Bergsten-Brucefors, 1976; Casey et al., 1991; Damon & Bajema, 1974; Livson & McNeill, 1962; Must et al., 2002; Susman & Ponirakis, 1997) and were obtained in females across the teen years to the seventh or eighth decade. Accuracy was hampered by longer time to recall (Koo & Rohan, 1997) or socioeconomic factors (Artaria & Henneberg, 2000). In the future, it would be helpful for studies to determine ways to enhance reliability as well as to examine “accuracy” or to report age at menarche across time in longitudinal studies. It is likely that age at menarche will continue to be the most frequently used measure to determine timing of puberty in girls, and any method to enhance its reliability would be beneficial to the field.

Hormones

Sex steroids and adrenal androgens are the underlying substrate of the external changes reflected by pubertal stage. Here we review two important issues regarding measurement of pubertal hormones over the last decade. First, we discuss some of the methodological advances pertaining to hormone assays and methods of collecting hormones. Second, we review the issue of comparing hormone concentrations to pubertal stage.

Methodology of hormone measurement. Over the last decade, some of the most important advances in hormone measurement have involved perfecting blood spot and saliva assays and conducting studies using these less invasive methodologies in children and adolescents.

Use of saliva samples for assaying hormone concentrations has gained more attention in recent literature, which may be due to several factors. For example, saliva samples are often easier and less expensive to collect than blood samples. However, the same issues exist for collection of both saliva and blood, depending on the specific hormone (e.g., time of day, number of samples, or day in menstrual cycle; see review in Dorn et al., 2006). Additionally, salivary assays have been perfected and are more readily available either by contract or by purchasing assay kits for use in one’s laboratory. Testing has played an important role in providing new assays via enzyme-linked immunosorbent assay (ELISA) (e.g., androstenedione), thereby improving the sensitivity of existing assays (e.g., estradiol),

conducting quality-control data and more normative data from different age and gender groups (Shirtcliff, Granger, & Likos, 2002), and examining the impact of medication on hormone concentrations (Granger, Hibel, Fortunato, & Kapelewski, 2009; Hibel, Granger, Cicchetti, & Rogosch, 2007). Others have examined the reliability of concentrations of salivary steroids based on the collection device used (Shirtcliff, Granger, Schwartz, & Curran, 2001).

Blood spot analyses using finger stick methodology appeared less frequently in the literature with biobehavioral studies in children and adolescents. (For an exception, see Angold & Costello, 2006.) The method is relatively simple. Following the finger stick, drops of blood are placed on special filter paper for later analysis. This methodology has advantages in that only a small amount of blood is needed and storage of papers is easier than processing and storage of tubes used in venipuncture. The most widely used blood spot tests in biobehavioral research include cortisol, progesterone, gonadotropin, and estradiol concentrations.

Some studies examined the reliability of blood spot analyses with serum and/or saliva samples. In a well-conducted study of adults, Shirtcliff, Reavis, Overman, and Granger (2001) examined reliability and sensitivity using blood spots for testosterone, estradiol, and progesterone. They reported high correlations between serum samples and blood spots but indicated limitations when measuring progesterone and estradiol in men, likely due to low variability in those hormones.

The reliability of serum, saliva, and/or blood spot methodology for hormones that are changing during puberty is also an important issue. Estradiol increases during puberty in girls and boys, but the magnitude is greater in girls. Estradiol measurement may be challenging because of diurnal as well as monthly changes in girls, for the regularity of the cycle may not be predictable until several years after menarche. Thus, measuring estradiol pre- or peripubertally may be problematic. In a small sample of boys ($n = 17$; aged 8–9 years) and nonmenstruating girls ($n = 18$; aged 10.78–12.27 years), correlations of serum and blood spot assays for estradiol were $r = .73$ and $.96$, respectively, whereas correlations between saliva and blood spot assays were nonsignificant for boys ($r = -.18$) and significant for girls ($r = .72$) (Shirtcliff et al., 2000). Investigators reported that the sensitivity of the salivary and blood spot analyses was adequate for most prepubertal girls and boys. However, as critiqued earlier, prepubertal was incorrectly equated with premenarcheal, and some of the nonmenstruating girls were likely in early or even mid-puberty (Dorn et

al., 2006). Thus, one cannot be sure that estradiol assays are sensitive enough for girls who are truly prepubertal or for boys who are pre- or peripubertal. Since the Shirtcliff publication, a more sensitive assay for estradiol has been developed by a saliva analysis company, but we remain unaware of any testing in pre- and peripubertal girls and boys in which puberty is documented by a research-grade physical examination.

Assay sensitivity is particularly important for pre- and early pubertal boys and girls primarily with regard to gonadal steroids due to lower concentrations of pubertal hormones in these groups (Grumbach & Styne, 2003). It is likely that prepubertal concentrations of estradiol are below the detection level of the assay, particularly when using radioimmunoassay and ELISA. Thus, in prepubertal girls, concentrations may be undetectable in the majority of the group. Recently, a more reliable and valid serum method has been proposed: liquid chromatography tandem mass spectrometry (LC/MS-MS; Albrecht & Styne, 2007; Herold & Fitzgerald, 2003). Caution should be exercised when interpreting studies that do not use LC/MS-MS as the assay methodology in girls or boys in pre- and early puberty (for testosterone and estradiol) and in girls and women (for testosterone). Biro and Emans (2008) illustrated this point in a recent editorial focusing on PCOS and the interpretation of steroid hormone concentrations. However, we encourage the reader to follow the literature closely because additional studies may be published soon that utilize this newer technology.

Comparing hormone concentrations to pubertal stage. Some studies have opted to include pubertal hormones in their study not only to reflect pubertal development but also because hormones may directly or indirectly influence the behavioral or affective outcomes under study. For some it has been tempting to consider measuring puberty-related hormones to indicate “stage of puberty” rather than determining pubertal stage by physical examination, especially because the former is thought to be less invasive. This is particularly true when measuring hormones in saliva. However, most endocrinologists would agree that, in general, a reproduction-related hormone cannot be matched to a specific Tanner stage. Puberty-related hormones show a wide range of concentration within stage and by gender. Further, there can be overlap across stages. Depending on the hormone (e.g., morning testosterone), one sample *may* be able to distinguish “prepubertal versus pubertal” boys with

relative certainty if that concentration is high enough and is measured in the morning (Wu, Brown, Butler, Stirling, & Kelnar, 1993).

A recent important study has shed light on the issue of “comparability” of pubertal stage and saliva hormone concentrations. Shirtcliff, Dahl, and Pollak (2009) conducted a unique study in 160 boys and girls aged 9–14 years in which they examined associations of pubertal ratings by physical examination, the PDS, and a picture-based interview about puberty. The PDS was converted to a 5-point scale using a gonadal score (e.g., breast, menarche, growth in height) and adrenal score to match Tanner criteria. They determined associations of these three measures with salivary DHEA, testosterone, and estradiol. In comparing the physical examination with the self-report using photographs and the PDS, κ coefficients were quite low and similar to other studies (e.g., $\kappa = .29-.43$).

The novel component of the study by Shirtcliff et al. (2009) is the comparison of hormone concentrations to the various pubertal stage ratings obtained by different methodologies. The two measures of self-report were sometimes stronger correlates with hormones than was the physical examination. However, the statistical model predicting hormones was quite weak for girls, citing the limitation of estradiol based on its cyclical nature (or lack thereof in some premenarcheal girls or those recently menarcheal). The model for boys was somewhat stronger. Without further methodological controls regarding the menstrual cycle or change in sensitivity of the assay, the usefulness of estradiol in this sample is questionable. Additionally, one must remember that salivary hormones were used in this study. Differences with serum hormone concentrations remain to be empirically tested.

THE NEXT DECADE: WHAT IS NEEDED?

In the past decade, puberty has made the news! For example, new and exciting research has emerged regarding kisspeptin and the kisspeptin receptor and their roles in initiation of puberty. Earlier timing of puberty continued to be documented, with some general indication that this secular trend may now be stable, particularly with respect to age at menarche. However, age at onset of puberty may continue to decrease in populations in emerging countries where resources are now more plentiful or with changes in social status. This past decade also introduced two reviews on the importance of puberty and of measuring puberty (Coleman & Coleman, 2002; Dorn et al., 2006), and several studies reported agreement

between self-reported measures of puberty and those obtained by physical examination. Overall, agreement between self-report and physical examination by κ coefficients remains poor, and studies with participants who are obese observed even lower agreement. However, at least two studies indicated higher agreement by adolescents experiencing some chronic disorders, leading to the hypothesis that they may be more knowledgeable than some of their peers about how their bodies are developing or changing. With respect to pubertal hormones, newer methodologies are in use that may improve our understanding, particularly regarding the early stages of puberty. This area is still evolving and therefore should be monitored to note progress.

Although the last decade has revealed important progress on the measurement of puberty and its application across various studies, much remains undone. Progress could be enhanced in various ways (see Susman & Dorn, 2009, for further elaboration). First, it would be beneficial to increase collaboration among developmental scientists and those in various subspecialties of medicine, neuroscience, and statistics as well as other areas of relevant technology (e.g., assay development, neuroimaging, computer programming and usage). Such collaboration would allow more complex biobehavioral questions to be addressed using appropriate and perhaps multiple measures of puberty. Timing and tempo of puberty should be included. In particular, data analytic strategies could enhance our understanding of puberty or timing of puberty by incorporating use of latent constructs of puberty, growth curve modeling with multiple samples at multiple time points, or other appropriate strategies as exemplified in the longitudinal study reported by Belsky et al. (2007). Second, studies of physiological or pathophysiological processes as well as studies of behavior could increase consideration and inclusion of the potential relevance of puberty or timing of puberty to the question at hand. For example, biobehavioral studies examining teenagers undergoing treatment for asthma may benefit by including measures of puberty. Such measures may explain a physiological reason for treatment success as well as a psychosocial reason for compliance with treatment. Third, studies are lacking on the structure and function of the brain in which puberty is determined by research-grade physical examinations for staging. Romeo (2003) wrote a provocative review supporting the notion that puberty is also a critical period “. . . of neural development that further organizes and shapes an organism’s brain and behavioural potential” (p. 1190). In many respects, this research

on puberty is in its infancy, and further development regarding changes across puberty could contribute to our understanding of emotion, cognition, behavior, and psychopathology. Fourth, diligence by reviewers and editors in maintaining high standards regarding measurement issues of pubertal status and pubertal timing is necessary. We and others have articulated this need and have offered specific suggestions (Dorn et al., 2006; Euling et al., 2008; Susman & Dorn, 2009). Additionally, the newer, more sensitive analytic techniques for assessment of sex hormone levels should be utilized in serum hormone measurement. Fifth, more longitudinal studies on timing and tempo of puberty are needed as well as more studies in boys. Rigorous methodologies pertaining to measurement of puberty are crucial.

Finally, with respect to timing of puberty, we have indicated that numerous studies in the literature look at varied outcomes in association with timing of puberty. Certainly, additional studies need to be conducted, keeping in mind the use of rigorous methodologies when measuring puberty that are appropriate to the question. Virtually all studies examining timing of puberty, regardless of methodology of measurement, are summarized into a global conclusion regarding puberty. We believe that some of the studies using self-report would have been better served by conducting physical examinations for pubertal staging. Conclusions in these studies regarding puberty may have been incorrect. Graber (2003) suggests that with respect to puberty, we need to "... move beyond demonstrating effects, to better understanding of why effects occur, for whom and for how long" (p. 320). Studies that examine timing of puberty and an outcome need to account for mechanisms (or mediators) as well as moderators that impact these associations and also to consider the context in which timing of puberty may be influential. Several examples of such examinations are beginning to appear in the literature (Conley & Rudolph, 2009; Ge, Brody, Conger, Simons, & Murry, 2002; Haynie, 2003; Obeidallah, Brennan, Brooks-Gunn, & Earls, 2004; Sontag, Graber, Brooks-Gunn, & Warren, 2008; Stice, Presnell, & Bearman, 2001). Last, few studies examine the antecedents of timing of puberty. One example that did consider antecedents is the longitudinal National Institute of Child Health and Human Development early child care study, which reported that early parenting behaviors predicted pubertal development, particularly in girls (Belsky et al., 2007). Altering the conceptual model to examine which variables may predict timing of

puberty can provide insight into the reasons for the observed earlier occurrence of puberty as well as the contexts involved in these observations.

REFERENCES

- Akslae, L., Sorensen, K., Petersen, J. H., Skakkebaek, N. E., & Juul, A. (2009). Recent decline in age at breast development: The Copenhagen Puberty Study. *Pediatrics*, *123*, e932–e939.
- Albrecht, L., & Styne, D. (2007). Laboratory testing of gonadal steroids in children. *Pediatric Endocrinology Reviews*, *5*(Suppl. 1), 599–607.
- Angold, A., & Costello, E. J. (2006). Puberty and depression. *Child and Adolescent Psychiatric Clinics of North America*, *15*, 919–937, ix.
- Artaria, M. D., & Henneberg, M. (2000). Why did they lie? Socio-economic bias in reporting menarcheal age. *Annals of Human Biology*, *27*, 561–569.
- Banerjee, I., & Clayton, P. (2007). The genetic basis for the timing of human puberty. *Journal of Neuroendocrinology*, *19*, 831–838.
- Baumann, D. A., Landolt, M. A., Wetterwald, R., Dubuis, J. M., Sizonenko, P. C., & Werder, E. A. (2001). Psychological evaluation of young women after medical treatment for central precocious puberty. *Hormone Research in Pediatrics*, *56*, 45–50.
- Belgorosky, A., Baquedano, M. S., Guercio, G., & Rivarola, M. A. (2009). Expression of the IGF and the aromatase/estrogen receptor systems in human adrenal tissues from early infancy to late puberty: Implications for the development of adrenarche. *Reviews in Endocrine and Metabolic Disorders*, *10*, 51–61.
- Belsky, J., Steinberg, L. D., Houts, R. M., Friedman, S. L., DeHart, G., Cauffman, E., et al. (2007). Family rearing antecedents of pubertal timing. *Child Development*, *78*, 1302–1321.
- Bergsten-Brucefors, A. (1976). A note on the accuracy of recalled age at menarche. *Annals of Human Biology*, *3*, 71–73.
- Biro, F. M., & Emans, S. J. (2008). Whither PCOS? The challenges of establishing hyperandrogenism in adolescent girls. *Journal of Adolescent Health*, *43*, 103–105.
- Biro, F. M., Huang, B., Crawford, P., Lucky, A., Stiegel-Moore, R., Barton, B. A., et al. (2006). Pubertal correlates in black and white girls. *The Journal of Pediatrics*, *148*, 234–240.
- Biro, F. M., Huang, B., Daniels, S. R., & Lucky, A. W. (2008). Pubarche as well as thelarche may be a marker for the onset of puberty. *Journal of Pediatric and Adolescent Gynecology*, *21*, 323–328.
- Biro, F. M., Lucky, A. W., Simbartl, L. A., Barton, B. A., Daniels, S. R., Striegel-Moore, R. H., et al. (2003). Pubertal maturation in girls and the relationship to anthropometric changes: Pathways through puberty. *The Journal of Pediatrics*, *142*, 643–646.
- Biro, F. M., McMahon, R. P., Striegel-Moore, R., Crawford, P. B., Obarzanek, E., Morrison, J., et al. (2001). Impact of

- timing of pubertal maturation on growth in black and white female adolescents: The National Heart, Lung, and Blood Institute Growth and Health Study. *The Journal of Pediatrics*, *138*, 636–643.
- Bonat, S., Pathomvanich, A., Keil, M., Field, A., & Yanovski, J. (2002). Self-assessment of pubertal stage in overweight children. *Pediatrics*, *110*, 743–747.
- Bond, L., Clements, J., Bertalli, N., Evans-Whipp, T., McMorris, B. J., Patton, G. C., et al. (2006). A comparison of self-reported puberty using the Pubertal Development Scale and the Sexual Maturation Scale in a school-based epidemiologic survey. *Journal of Adolescence*, *29*, 709–720.
- Bouhours-Nouet, N., Gatelais, F., Boux de Casson, F., Rouleau, S., & Coutant, R. (2007). The insulin-like growth factor-I response to growth hormone is increased in prepubertal children with obesity and tall stature. *The Journal of Clinical Endocrinology and Metabolism*, *92*, 629–635.
- Bratberg, G., Nilsen, T., Holmen, T., & Vatten, L. (2006). Combined influence of early sexual maturation and central adiposity on subsequent stature: A four-year follow-up of 1,605 Norwegian boys and girls: The Young-HUNT study. *European Journal of Pediatrics*, *165*, 787–793.
- Brooks-Gunn, J., & Warren, M. P. (1989). Biological and social contributions to negative affect in young adolescent girls. *Child Development*, *60*, 40–55.
- Cary, J., Hein, K., & Dell, R. (1991). Theophylline disposition in adolescents with asthma. *Therapeutic Drug Monitoring*, *13*, 309–313.
- Casey, V. A., Dwyer, J., Colman, K. A., Krall, E. A., Gardner, J., & Valadian, I. (1991). Accuracy of recall by middle-aged participants in a longitudinal study of their body size and indices of maturation earlier in life. *Annals of Human Biology*, *18*, 155–166.
- Chan, N., Sung, R. Y. T., Kong, A. P. S., Goggins, W. B., So, H. K., & Nelson, E. A. S. (2008). Reliability of pubertal self-assessment in Hong Kong Chinese children. *Journal of Paediatrics and Child Health*, *44*, 353–358.
- Christensen, K. Y., Maisonet, M., Rubin, C., Flanders, W. D., Drews-Botsch, C., Dominguez, C., et al. (2010). Characterization of the correlation between ages at entry into breast and pubic hair development. *Annals of Epidemiology*, *20*, 405–408.
- Coleman, L., & Coleman, J. (2002). The measurement of puberty: A review. *Journal of Adolescence*, *25*, 535–550.
- Conley, C. S., & Rudolph, K. D. (2009). The emerging sex difference in adolescent depression: Interacting contributions of puberty and peer stress. *Development and Psychopathology*, *21*, 593–620.
- Costello, E. J., Sung, M., Worthman, C., & Angold, A. (2007). Pubertal maturation and the development of alcohol use and abuse. *Drug and Alcohol Dependence*, *88*(suppl 1), S50–S59.
- Cutler, G. B. Jr., Schiebinger, R. J., Albertson, B. D., Cassorla, F. G., Chrousos, G. P., Comite, F., et al. (1990). Adrenarche (human and animal). In M. M. Grumbach, P. C. Sizonenko, & M. L. Aubert (Eds.), *Control of the onset of puberty* (pp. 506–533). Baltimore, MD: Williams & Wilkins.
- Damon, A., & Bajema, C. J. (1974). Age at menarche: Accuracy of recall after thirty-nine years. *Human Biology*, *46*, 381–384.
- de Roux, N., Genin, E., Carel, J. C., Matsuda, F., Chaussain, J. L., & Milgrom, E. (2003). Hypogonadotropic hypogonadism due to loss of function of the KiSS1-derived peptide receptor GPR54. *Proceedings of the National Academy of Sciences of the USA*, *100*, 10972–10976.
- Desmangles, J. C., Lappe, J. M., Lipaczewski, G., & Haynatzki, G. (2006). Accuracy of pubertal Tanner staging self-reporting. *Journal of Pediatric Endocrinology and Metabolism*, *19*, 213–221.
- Dorn, L. D. (2007). Psychological and social problems in children with premature adrenarche and precocious puberty. In O. H. Pescovitz & E. C. Walvoord (Eds.), *When puberty is precocious: Scientific and clinical aspects* (pp. 309–327). Totowa, NJ: Humana Press.
- Dorn, L. D., Dahl, R. E., Woodward, H. R., & Biro, F. (2006). Defining the boundaries of early adolescence: A user's guide to assessing pubertal status and pubertal timing in research with adolescents. *Applied Developmental Science*, *10*, 30–56.
- Dorn, L. D., Hitt, S. F., & Rotenstein, D. (1999). Biopsychological and cognitive differences in children with premature vs. on-time adrenarche. *Archives of Pediatric and Adolescent Medicine*, *153*, 137–146.
- Dorn, L. D., Rose, S. R., Rotenstein, D., Susman, E. J., Huang, B., Loucks, T. L., et al. (2008). Differences in endocrine parameters and psychopathology in girls with premature adrenarche versus on-time adrenarche. *Journal of Pediatric Endocrinology and Metabolism*, *21*, 439–448.
- Dorn, L. D., Susman, E. J., Nottelmann, E. D., Inoff-German, G., & Chrousos, G. P. (1990). Perceptions of puberty: Adolescent, parent, and health care personnel. *Developmental Psychology*, *26*, 322–329.
- Ellis, B. J., & Essex, M. J. (2007). Family environments, adrenarche, and sexual maturation: A longitudinal test of a life history model. *Child Development*, *78*, 1799–1817.
- Emans, S. J., & Biro, F. (1998). Secondary sexual characteristics and menses in young girls. *Pediatrics*, *101*, 949–950.
- Euling, S. Y., Herman-Giddens, M. E., Lee, P. A., Selevan, S. G., Juul, A., Sorensen, T. I. A., et al. (2008). Examination of US puberty-timing data from 1940 to 1994 for secular trends: Panel findings. *Pediatrics*, *121*, S172–S191.
- Facchini, F., Fiori, G., Bedogni, G., Galletti, L., Ismagulov, O., Ismagulova, A., et al. (2008). Puberty in modernizing Kazakhstan: A comparison of rural and urban children. *Annals of Human Biology*, *35*, 50–64.
- Feinberg, I., Higgins, L. M., Khaw, W. Y., & Campbell, I. G. (2006). The adolescent decline of NREM delta, an indicator of brain maturation, is linked to age and sex but not to pubertal stage. *American Journal of Physiology-Regulatory, Integrative and Comparative Physiology*, *291*, R1724–R1729.
- Gajdos, Z. K. Z., Hirschhorn, J. N., & Palmert, M. R. (2009). What controls the timing of puberty? An update on

- progress from genetic investigation. *Current Opinion in Endocrinology, Diabetes and Obesity*, 16, 16–24.
- Garnier, D., Simondon, K., & Benefice, E. (2005). Longitudinal estimates of puberty timing in Senegalese adolescent girls. *American Journal of Human Biology*, 17, 718–730.
- Ge, X., Brody, G. H., Conger, R. D., Simons, R. L., & Murry, V. M. (2002). Contextual amplification of pubertal transition effects on deviant peer affiliation and externalizing behavior among African American children. *Developmental Psychology*, 38, 42–54.
- Ge, X., Kim, I. J., Brody, G. H., Conger, R. D., Simons, R. L., Gibbons, F. X., et al. (2003). It's about timing and change: Pubertal transition effects on symptoms of major depression among African American youths. *Developmental Psychology*, 39, 430–439.
- Golub, M. S., Collman, G. W., Foster, P. M. D., Kimmel, C. A., Rajpert-De Meyts, E., Reiter, E. O., et al. (2007). Public health implications of altered puberty timing. *Pediatrics*, 121, S218–S230.
- Graber, J. A. (2003). Puberty in context. In C. Hayward (Ed.), *Gender differences at puberty* (pp. 307–325). New York, NY: Cambridge University Press.
- Granger, D. A., Hibel, L. C., Fortunato, C. K., & Kapelewski, C. H. (2009). Medication effects on salivary cortisol: Tactics and strategy to minimize impact in behavioral and developmental science. *Psychoneuroendocrinology*, 34, 1437–1448.
- Grumbach, M. M. (2002). The neuroendocrinology of human puberty revisited. *Hormone Research*, 57(Suppl. 2), 2–14.
- Grumbach, M. M., & Styne, D. M. (1992). Puberty: Ontogeny, neuroendocrinology, physiology, and disorders. In J. D. Wilson & P. W. Foster (Eds.), *Williams textbook of endocrinology* (8th ed., pp. 1139–1231). Philadelphia, PA: W. B. Saunders.
- Grumbach, M. M., & Styne, D. M. (2003). Puberty: Ontogeny, neuroendocrinology, physiology, and disorders. In P. R. Larsen, H. M. Kronenberg, S. Melmed, & K. S. Polonsky (Eds.), *Williams textbook of endocrinology* (10th ed., pp. 1115–1286). Philadelphia, PA: Elsevier Science.
- Haynie, D. L. (2003). Contexts of risk? Explaining the link between girls' pubertal development and their delinquency involvement. *Social Forces*, 82, 355–397.
- Hein, K. (1987). The use of therapeutics in adolescence. *Journal of Adolescent Health Care*, 8, 8–35.
- Herman-Giddens, M. E., Slora, E. J., Wasserman, R. C., Bourdony, C. J., Bhapkar, M. V., Koch, G. G., et al. (1997). Secondary sex characteristics and menses in young girls seen in office practice: A study from the pediatric research in office settings network. *Pediatrics*, 99, 505–512.
- Herold, D. A., & Fitzgerald, R. L. (2003). Immunoassays for testosterone in women: Better than a guess? *Clinical Chemistry*, 49, 1250–1251.
- Hibel, L. C., Granger, D. A., Cicchetti, D., & Rogosch, F. A. (2007). Salivary biomarker levels and diurnal variation: Association with medications prescribed to control children's problem behavior. *Child Development*, 78, 927–937.
- Hosny, L. A., El-Ruby, M. O., Zaki, M. E., Aglan, M. S., Zaki, M. S., El Gammal, M. A., et al. (2005). Assessment of pubertal development in Egyptian girls. *Journal of Pediatric Endocrinology and Metabolism*, 18, 577–584.
- Huang, B., Biro, F. M., & Dorn, L. D. (2009). Determination of relative timing of pubertal maturation through ordinal logistic modeling: Evaluation of growth and timing parameters. *Journal of Adolescent Health*, 45, 383–388.
- Ibáñez, L., DiMartino-Nardi, J., Potau, N., & Saenger, P. (2000). Premature adrenarche—Normal variant or forerunner of adult disease. *Endocrine Reviews*, 21, 671–696.
- Jones, L. L., Griffiths, P. L., Norris, S. A., Pettifor, J. M., & Cameron, N. (2009). Is puberty starting earlier in urban South Africa? *American Journal of Human Biology*, 21, 395–397.
- Jones, M. C., & Bayley, N. (1950). Physical maturing among boys as related to behavior. *Journal of Educational Psychology*, 41, 129–148.
- Juul, A., Teilmann, G., Scheike, T., Hertel, N., Holm, K., Laursen, E., et al. (2006). Pubertal development in Danish children: Comparison of recent European and US data. *International Journal of Andrology*, 29, 247–255.
- Kaplowitz, P., & Oberfield, S. (1999). Reexamination of the age limit for defining when puberty is precocious in girls in the United States: Implications for evaluation and treatment. *Pediatrics*, 104, 936–941.
- Kindblom, J. M., Lorentzon, M., Norjavaara, E., Hellqvist, A., Nilsson, S., Mellstrom, D., et al. (2006). Pubertal timing predicts previous fractures and BMD in young adult men: The GOOD study. *Journal of Bone and Mineral Research*, 21, 790–795.
- Knobil, E. (1988). The hypothalamic gonadotropin-releasing hormone (GnRH) pulse generator in the rhesus monkey and its neuroendocrine control. *Human Reproduction*, 3, 29–31.
- Koo, M. M., & Rohan, T. E. (1997). Accuracy of short-term recall of age at menarche. *Annals of Human Biology*, 24, 61–64.
- Laitinen-Krispijn, S., van der Ende, J., & Verhulst, F. C. (1999). The role of pubertal progress in the development of depression in early adolescence. *Journal of Affective Disorders*, 54, 211–215.
- Leone, M., & Comtois, A. (2007). Validity and reliability of self-assessment of sexual maturity in elite adolescent athletes. *Journal of Sports Medicine and Physical Fitness*, 47, 361–365.
- Li, C. I., Littman, A. J., & White, E. (2007). Relationship between age maximum height is attained, age at menarche, and age at first full-term birth and breast cancer risk. *Cancer Epidemiology, Biomarkers and Prevention*, 16, 2144–2149.
- Livson, N., & McNeill, D. (1962). The accuracy of recalled menarche. *Human Biology*, 34, 218–221.
- Maninger, N., Wolkowitz, O. M., Reus, V. I., Epel, E. S., & Mellon, S. H. (2009). Neurobiological and neuropsychiatric effects of dehydroepiandrosterone (DHEA) and

- DHEA sulfate (DHEAS). *Frontiers in Neuroendocrinology*, 30, 65–91.
- Marshall, W. A., & Tanner, J. M. (1969). Variations in pattern of pubertal changes in girls. *Archives of Disease in Childhood*, 44, 291–303.
- Marshall, W. A., & Tanner, J. (1970). Variations in the pattern of pubertal change in boys. *Archives of Disease in Childhood*, 45, 13–23.
- Martí-Hennenberg, C., & Vizmanos, B. (1997). The duration of puberty in girls is related to the timing of its onset. *The Journal of Pediatrics*, 131, 618–621.
- Mascaretti, G., & Di Berardino, C. (2008). Study of pubertal development in Abruzzo (Italy) and analysis of factors implicated in puberty variability. *Clinical and Experimental Obstetrics and Gynecology*, 35, 117–120.
- Mendle, J., Turkheimer, E., & Emery, R. E. (2007). Detrimental psychological outcomes associated with early pubertal timing in adolescent girls. *Developmental Review*, 27, 151–171.
- Morris, N. M., & Udry, J. R. (1980). Validation of a self-administered instrument to assess stage of adolescent development. *Journal of Youth and Adolescence*, 9, 271–280.
- Mussen, P. H., & Jones, M. C. (1957). Self-conceptions, motivations, and interpersonal attitudes of late- and early-maturing boys. *Child Development*, 28, 243–256.
- Must, A., Phillips, S. M., Naumova, E. N., Blum, M., Harris, S., Dawson-Hughes, B., et al. (2002). Recall of early menstrual history and menarcheal body size: After 30 years, how well do women remember? *American Journal of Epidemiology*, 155, 672–679.
- Negriff, S., & Susman, E. J. (in press). Pubertal timing, depression and externalizing problems: A framework, review, and examination of gender differences. *Journal of Research on Adolescence*.
- Norris, S. A., & Richter, L. M. (2008). Are there short cuts to pubertal assessments? Self-reported and assessed group differences in pubertal development in African adolescents. *Journal of Adolescent Health*, 42, 259–265.
- Nottelmann, E. D., Susman, E. J., Inoff-Germain, G. E., Cutler, G. B., Jr., Loriaux, D. L., & Chrousos, G. P. (1987). Developmental processes in early adolescence: Relations between adolescent adjustment problems and chronological age, pubertal stage, and puberty-related hormone levels. *The Journal of Pediatrics*, 110, 473–480.
- Oakley, A. E., Clifton, D. K., & Steiner, R. A. (2009). Kisspeptin signaling in the brain. *Endocrine Reviews*, 30, 713–743.
- Obeidallah, D., Brennan, R. T., Brooks-Gunn, J., & Earls, F. (2004). Links between pubertal timing and neighborhood contexts: Implications for girls' violent behavior. *Journal of American Academy of Child and Adolescent Psychiatry*, 43, 1460–1468.
- Officioso, A., Salerno, M., Lettiero, T., Micillo, M., Pisaturo, L., & Di Maio, S. (2000). Adolescent girls with idiopathic central precocious puberty: Typical character traits. *Journal of Pediatric Endocrinology and Metabolism*, 13(Suppl. 1), 835–839.
- Ojeda, S. R., Roth, C., Mungenast, A., Heger, S., Mastroianni, C., Parent, A. S., et al. (2006). Neuroendocrine mechanisms controlling female puberty: New approaches, new concepts. *International Journal of Andrology*, 29, 256–263.
- Ong, K. K., Ahmed, M. L., & Dunger, D. B. (2006). Lessons from large population studies on timing and tempo of puberty (secular trends and relation to body size): The European trend. *Molecular and Cellular Endocrinology*, 254–255, 8–12.
- Pantsiotou, S., Papadimitriou, A., Douros, K., Priftis, K., Nicolaidou, P., & Fretzayas, A. (2008). Maturational tempo differences in relation to the timing of the onset of puberty in girls. *Acta Paediatrica*, 97, 217–220.
- Parent, A. S., Teilmann, G., Juul, A., Skakkebaek, N. E., Toppari, J., & Bourguignon, J. P. (2003). The timing of normal puberty and the age limits of sexual precocity: Variations around the world, secular trends, and changes after migration. *Endocrine Reviews*, 24, 668–693.
- Parker, L. N. (1991). Adrenarche. *Endocrinology and Metabolism Clinics of North America*, 20, 71–83.
- Patton, G. C., & Viner, R. (2007). Pubertal transitions in health. *The Lancet*, 369, 1130–1139.
- Petersen, A. C., Crockett, L., Richards, M., & Boxer, A. (1988). A self-report of pubertal status: Reliability, validity, and initial norms. *Journal of Youth and Adolescence*, 17, 117–133.
- Petersen, A. C., Tobin-Richards, M., & Boxer, A. (1983). Puberty: Its measurement and its meaning. *Journal of Early Adolescence*, 3, 47–62.
- Plant, T. M. (2002). Neurophysiology of puberty. *Journal of Adolescent Health*, 31(Suppl. 6), 185–191.
- Rabbani, A., Khodai, S., Mohammad, K., Sotoudeh, A., Karbakhsh, M., Nouri, K., et al. (2008). Pubertal development in a random sample of 4,020 urban Iranian girls. *Journal of Pediatric Endocrinology and Metabolism*, 21, 681–687.
- Reardon, L. E., Leen-Feldner, E. W., & Hayward, C. (2009). A critical review of the empirical literature on the relation between anxiety and puberty. *Clinical Psychology Review*, 29, 1–23.
- Reiter, E. O., & Lee, P. A. (2001). Have the onset and tempo of puberty changed? *Archives of Pediatric and Adolescent Medicine*, 155, 988–989.
- Reynolds, E. L., & Wines, J. V. (1948). Individual differences in physical changes associated with adolescence in girls. *Archives of Pediatrics and Adolescent Medicine*, 75, 329–350.
- Reynolds, E. L., & Wines, J. V. (1951). Physical changes associated with adolescence in boys. *AMA: American Journal of Diseases in Children*, 82, 529–547.
- Romeo, R. D. (2003). Puberty: A period of both organizational and activational effects of steroid hormones on neurobehavioural development. *Journal of Neuroendocrinology*, 15, 1185–1192.
- Rosenfield, R. L., Bachrach, L. K., Chernausk, S. D., Gertner, J. M., Gottschalk, M., Hardin, D. S., et al. (2000). Current age of onset of puberty. *Pediatrics*, 106, 622.

- Saenger, P., & DiMartino-Nardi, J. (2001). Premature adrenarche. *Journal of Endocrinological Investigation*, *24*, 724–733.
- Schall, J. I., Semeao, E. J., Stallings, V. A., & Zemel, B. S. (2002). Self-assessment of sexual maturity status in children with Crohn's disease. *The Journal of Pediatrics*, *141*, 223–229.
- Schubert, C. M., Chumlea, W. C., Kulin, H. E., Lee, P. A., Himes, J. H., & Sun, S. S. (2005). Concordant and discordant sexual maturation among US children in relation to body weight and BMI. *Journal of Adolescent Health*, *37*, 356–362.
- Sedlmeyer, I. L., & Palmert, M. R. (2002). Delayed puberty: Analysis of a large case series from an academic center. *The Journal of Clinical Endocrinology and Metabolism*, *87*, 1613–1620.
- Seminara, S. B., Messenger, S., Chatzidaki, E. E., Thresher, R. R., Acierno, J. S., Jr., Shagoury, J. K., et al. (2003). The GPR54 gene as a regulator of puberty. *New England Journal of Medicine*, *349*, 1614–1627.
- Semiz, S., Kurt, F., Kurt, D., Zencir, M., & Sevinç, O. (2008). Pubertal development of Turkish children. *Journal of Pediatric Endocrinology and Metabolism*, *21*, 951–961.
- Shirtcliff, E. A., Dahl, R. E., & Pollak, S. D. (2009). Pubertal development: Correspondence between hormonal and physical development. *Child Development*, *80*, 327–337.
- Shirtcliff, E. A., Granger, D. A., & Likos, A. (2002). Gender differences in the validity of testosterone measured in saliva by immunoassay. *Hormones and Behavior*, *42*, 62–69.
- Shirtcliff, E. A., Granger, D. A., Schwartz, E., & Curran, M. J. (2001). Use of salivary biomarkers in biobehavioral research: Cotton-based sample collection methods can interfere with salivary immunoassay results. *Psychoneuroendocrinology*, *26*, 165–173.
- Shirtcliff, E. A., Granger, D. A., Schwartz, E. B., Curran, M. J., Booth, A., & Overman, W. H. (2000). Assessing estradiol in biobehavioral studies using saliva and blood spots: Simple radioimmunoassay protocols, reliability, and comparative validity. *Hormones and Behavior*, *38*, 137–147.
- Shirtcliff, E. A., Reavis, R., Overman, W. H., & Granger, D. A. (2001). Measurement of gonadal hormones in dried blood spots versus serum: Verification of menstrual cycle phase. *Hormones and Behavior*, *39*, 258–266.
- Siegel, S. F., Finegold, D. N., Urban, M. D., McVie, R., & Lee, P. A. (1992). Premature pubarche: Etiological heterogeneity. *Journal of Clinical Endocrinology and Metabolism*, *72*, 239–247.
- Sisk, C. L., & Foster, D. L. (2004). The neural basis of puberty and adolescence. *Nature Neuroscience*, *7*, 1040–1047.
- Sontag, L. M., Graber, J. A., Brooks-Gunn, J., & Warren, M. P. (2008). Coping with social stress: Implications for psychopathology in young adolescent girls. *Journal of Abnormal Child Psychology*, *36*, 1159–1174.
- Stephen, M., Bryant, W., & Wilson, D. (2008). Self-assessment of sexual maturation in children and adolescents with diabetes mellitus. *Endocrine Practice*, *14*, 840–845.
- Stice, E., Presnell, K., & Bearman, S. K. (2001). Relation of early menarche to depression, eating disorders, substance abuse, and comorbid psychopathology among adolescent girls. *Developmental Psychology*, *37*, 608–619.
- Sun, S. S., Schubert, C. M., Chumlea, W. C., Roche, A. F., Kulin, H. E., Lee, P. A., et al. (2002). National estimates of the timing of sexual maturation and racial differences among US children. *Pediatrics*, *110*, 911–919.
- Susman, E. J., & Dorn, L. D. (2009). Puberty: Its role in development. In I. Wiley (Ed.), *Handbook of adolescent psychology* (3rd ed., pp. 116–151). New York, NY: John Wiley & Sons Inc.
- Susman, E. J., Nottelmann, E. D., Inoff-Germain, G., & Dorn, L. D. (1987). Hormonal influences on aspects of psychological development during adolescence. *Journal of Adolescent Health*, *8*, 492–504.
- Susman, E. J., & Ponirakis, A. (1997). Hormone-context interactions and antisocial behavior in youth. In A. Raine, D. Farrington, P. Brennan, & S. A. Mednick (Eds.), *Unlocking crime: The biosocial key* (pp. 251–269). New York, NY: Plenum.
- Susman, E. J., & Rogol, A. D. (2004). Puberty and psychological development. In R. M. Lerner & L. Steinberg (Eds.), *Handbook of adolescent psychology* (2nd ed., pp. 15–44). New York, NY: John Wiley & Sons Inc.
- Tanner, J. M. (1962). *Growth at adolescence* (2nd ed.). Oxford, U.K.: Blackwell Scientific Publications.
- van Wieringen, J. C., Roede, M. J., & Wit, J. M. (1985). Growth diagrams for patient care. *Tijdschrift voor kindergeneeskunde*, *53*, 147–152.
- Vizmanos, B., Martí-Hennenberg, C., Cliville, R., Moreno, A., & Fernandez-Ballart, J. (2001). Age of pubertal onset affects the intensity and duration of pubertal growth peak but not final height. *American Journal of Human Biology*, *13*, 409–416.
- Wehkalampi, K., Widen, E., Laine, T., Palotie, A., & Dunkel, L. (2008). Association of the timing of puberty with a chromosome 2 locus. *The Journal of Clinical Endocrinology and Metabolism*, *93*, 4833–4839.
- Wu, F. C. W., Brown, D. C., Butler, G. E., Stirling, H. F., & Kelnar, C. J. H. (1993). Early morning plasma testosterone is an accurate predictor of imminent pubertal development in prepubertal boys. *The Journal of Clinical Endocrinology and Metabolism*, *76*, 26–31.
- Wu, Y., Schreiber, G. B., Klementowicz, V., Biro, F. M., & Wright, D. (2001). Racial differences in accuracy of self-assessment of sexual maturation among young black and white girls. *Journal of Adolescent Health*, *28*, 197–203.
- Zukauskaitė, S., Lasiene, D., Lasas, L., Urbonaitė, B., & Hindmarsh, P. (2005). Onset of breast and pubic hair development in 1231 preadolescent Lithuanian school-girls. *Archives of Disease in Childhood*, *90*, 932–936.

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