

## CHAPTER 5

### *Puberty*

#### **Its Role In Development**

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What is this mystical, dreaded, and shrouded period of development, labeled *puberty*, that is accompanied by rapid morphological body changes, including physical growth and hormonal changes as well as a myriad of psychological and social contextual changes? Puberty has intrigued scholars, artists, parents, and adolescents alike for centuries, and cultures have ritualized puberty to varying degrees. In some instances rituals feast the reproductive transition of puberty, whereas in other instances religious and social aspects of puberty are celebrated. At its core, puberty is the period of development through which its passage endows an adolescent with reproductive competence. At an even more basic analysis, puberty is a brain–neuroendocrine process that provides a stimulus for all the physical changes and putative psychological changes that accompany this period of development. How, then, does a biologically based process come to be so compacted with social and psychological significance?

Puberty's significance likely derives to some extent from its mistaken identity and overlap with the adolescent passage. Adolescence is a wider concept that encompasses the time and terrain that includes both puberty and the social, emotional, and wider psychological changes that characterize the chronological transition from childhood to adulthood. The primary functions of puberty and adolescence are principally nonoverlapping; puberty entails brain development consisting of neuroendocrine changes, characterized by activation of

gonadotropin-releasing hormone (GnRH) and elevated secretion of gonadotropins and sex steroids, resulting in sexual maturation and related physical growth changes (Rockett, Lynch, & Buck, 2004; Sisk & Zehr, 2005). Adolescence entails the acquisition of adult cognition, emotions, and social roles that are possible through maturation of brain functions and dynamic interactions with varying family, educational, and social contexts. How the biological and psychological processes interact to influence one another has been the challenge to scientists for decades. The goal of this chapter is to present a perspective on the neuroendocrinology of puberty and the implications of these brain changes for psychological development. The chapter begins with a historical and theoretical perspective on the role of puberty in development followed by a review of the major neuroendocrine changes that occur at puberty and how these changes affect physical morphological characteristics. Then, the literature is condensed to present an overview of the relations between pubertal status and pubertal timing and psychological development. Finally, we conclude with research and intervention recommendations for the future.

#### **HISTORICAL PERSPECTIVE**

A classic perspective on adolescence is that it is a period characterized by “storm and stress” (Hall, 1904). This view portrays adolescents as oppositional, emotionally labile, and in need of constant monitoring so as to mold the adolescent's developing character to prevent adult

psychopathology. The biological changes of puberty were considered a major influence on the storm and stress of adolescence (Freud, 1958). This viewpoint has been significantly reconceptualized and now adolescents are presented in a more balanced and positive perspective. Adolescence now is viewed as a transitional period that is characterized by storm and stress for only a fraction of boys and girls (Offer & Offer, 1975; Offer & Schonert-Reichl, 1992). If it even does exist, the storm and stress of adolescence currently can be conceptualized as neither a universal phenomenon nor an exclusively biologically based aspect of development.

Changes in this perspective on adolescence evolved primarily from two distinct lines of evidence. First, parents, teachers, and others endowed with the responsibility to care for youth were aware that all adolescents are not the stormy and stressed-out persons who were portrayed in the literature and past popular press reports. Offer and Offer (1975) published a seminal study showing that about 80% of adolescent boys remain well adjusted during this transitional period. Adolescents do have problems, and multiple studies of mental health problems have shown prevalence rates of about 20% for mental health problems (Costello et al., 1996; Kashani, Orvaschel, Burk, & Reid, 1985; Offord et al., 1987). Collectively, adolescence now seems to be viewed as a more positive period of development, and newer research focuses on positive youth development (Catalano, Haggerty, Oesterle, Fleming, & Hawkins, 2004; Larson, 2000; Lerner, Brentano, Dowling, & Anderson, 2002) rather than the negative perspective in past eras. In brief, adolescents are not consistently characterized by emotional distress and behavioral disruption and they are known to be competent to interact seamlessly with family, friends and others in their wider ecology. The socially valuable movement toward positive youth development is designed to foster growth potential along favorable directions.

### Theories of Puberty and Psychological Development

Puberty as a biological process that deterministically influenced psychological functioning and social behavior was a view derived from evolutionary (Parker, 2000) and psychodynamic theories (Blos, 1962; Freud, 1958; Freud, 1998; Hall, 1904) that dominated the early twentieth century. Sigmund and Anna Freud were instrumental in advancing a perspective that merged the biological-sexual and psychological aspects of pubertal development. For Sigmund Freud, the arrival of puberty signaled the end of infantile sexual life and the beginning of normal adult sexual life (Freud, 1998). In infancy, the oral phase, the sexual instinct was predominantly autoerotic; at puberty, the genital phase, the search becomes for a sexual object. The autoerotic zones of infancy become subordinated to the genital zone. The two sexes are considered to diverge as the basic anatomy of sexuality is different between males and females. Freud considered male sexuality as straightforward and understandable, whereas sexuality in females became a combination of an affectional and a sensual current. The sexual aim of males becomes discharge of the sexual products, and the sexual aim is subordinate to the reproductive function.

Anna Freud (1958) carried on and expanded the traditional psychoanalytic perspective of her father with regard to puberty and adolescence. She developed the notion that defense mechanisms are critical to understanding adolescent adjustment. Defense mechanisms ward off pregenital urges that are reawakened or new urges arising from endogenous biological changes. Unconsciously, adolescents call on defense mechanisms to protect the ego and reduce anxiety.

After the analytically oriented era waned, behavioral and contextually oriented models of development became predominant in the mid to late twentieth century. This period might aptly be described as the dark ages of empiricism with regard to puberty and psychological development. Skinnerian behaviorism (Skinner, 1953),

social learning theory (e.g., Bandura, 1978), ecological theories (Bronfenbrenner, 1979), and life-course perspectives gave virtually no attention to puberty or biology more broadly. The rich inner psychological life and sexual fantasies and urges of adolescents became as repressed by late twentieth century scientists as by the Victorian adolescent. However, a minority perspective erupted in the 1980s that had been brewing among developmental psychologists. A foundation for the rise of a new era of interdisciplinary models of the biological and psychological aspects of puberty sprung out of the now classic Petersen and Taylor (1980) chapter on pubertal development in Adelson's (1980) *Handbook of Adolescent Development*. Petersen and Taylor suggested that the biological changes during puberty play a major role in psychological development. Furthermore, they suggested that being off time in pubertal development was a risk for adjustment problems.

The publication was followed in the early 1980s, parallel to the rise of the Society of Research in Adolescence, by a movement originating from a small group of scholars (Jeanne Brooks-Gunn, Richard M. Lerner, David Magnusson, Anne Petersen, Laurence Steinberg, Elizabeth Susman, and others) who put forth a paradigm that had parallels with a phoenix rising from the ashes. Like the risen phoenix, puberty and its psychological correlates took on a new form as a period of development characterized by *integrated* biological, psychological, and contextual features. The role of puberty in development then began to receive moderate attention given its potential as a mechanism involved in the etiology of adolescent adjustment and psychopathological problems. Several of these scholars began or continued the first longitudinal studies that focused on the integration of biopsychosocial processes inherent in adolescent development. The more integrated perspective became the norm for considering puberty and psychological development. This perspective is now viewed as essential for understanding the

fragmented findings regarding the role of puberty in psychological development.

It follows that the current chapter presents a perspective on puberty as a biopsychosocial transition that is initiated by major neuroendocrine changes and is accompanied by psychological and behavior changes that simultaneously initiate changes in the social contexts in which adolescents find themselves. This theoretical approach is referred to as *dynamic integration* and refers to the essential and changing fusion of processes across psychological, biological, and contextual levels of analysis (Susman & Rogol, 2004). The neuroendocrine changes that initiate and control the progression of puberty now will be reviewed.

#### **PUBERTY: A BRAIN-NEUROENDOCRINE EVENT**

Puberty consists of a coordinated series of hormone and physical growth changes that form the core of the transition from childhood to adolescence. An increasing sophistication of brain and neuroendocrine changes has led scientists to consider the primacy of the neurobiological basis of puberty. The brain undergoes extensive remodeling at puberty and these changes form the basis for reproductive maturation (Sisk & Foster, 2004). These neurobiological changes are responsible for both the biological hormone and physical morphological changes and likely contribute to the social, cognitive, and emotional changes that occur during puberty. The structural and functional changes of the brain first will be considered, followed by neuroendocrine changes responsible for the onset and progression of puberty.

#### **Structure and Function of Brain Development**

Coming to know the structure of human brain during adolescence is the result largely of the implementation of new imaging technologies. Much of what we know about adolescent brain development is derived from studies using magnetic resonance imaging (MRI) or functional MRI

(fMRI). The yield from imaging changes in the brain is revolutionary as these studies show both structural and functional changes during puberty. Much of the richest work has come from the laboratories of Giedd (Giedd et al., 1999; Giedd, 2008), Casey (Casey, Jones, & Hare, 2008) and Paus (Paus, 2005). Importantly, some changes in the brain occur prior to puberty, some during puberty and some after puberty (Dahl, 2004).

Major changes in gray matter volume develop in a U-shaped function (Giedd, 2004; Gogtay et al., 2004). Motor and sensory systems mature earlier, in general, compared to the higher order functions that are responsible for associative areas of the cortex (Galvin et al., 2006; Gogtay et al.; Sowell, Thompson, & Toga, 2004). Specifically, the limbic system is proposed to develop earlier than the prefrontal control areas of the brain. The prefrontal cortex (PFC) is one of the last changes to occur in brain development (Lewis, 1997). Based on this model, the adolescent is considered to be biased more toward functional limbic activity relative to prefrontal control activity. With advancing development there is a functional connectivity between the limbic and prefrontal regions. Nonetheless, Casey and colleagues (Casey et al., 2008) propose a model indicating that bottom-up limbic and prefrontal top-down control regions should be considered together, even though they have different developmental trajectories. In brief, findings to date are interpreted to suggest differential development of bottom-up limbic systems, which are implicated in incentive and emotional processing, and top-down control systems during adolescence as compared to childhood and adulthood. Adolescents are assumed to have a heightened responsiveness to incentives and socioemotional contexts simultaneous with the period when impulse control is still relatively immature. This perspective has direct implications for social and emotional development and behavior. The relatively late development of the prefrontal cortex may be an underlying process related to risk-taking behavior in

adolescents (Steinberg, in press). In addition, Casey et al. (2008) note that the sequence of developmental events may be exacerbated in adolescents prone to emotional reactivity, increasing the likelihood of poor mental health outcomes. Finally, given the major brain changes at puberty, the adolescent brain may be more vulnerable to the effects of substance use and abuse or other environmental insults that predispose youth to a trajectory of cognitive, emotional, and behavioral problems.

#### *Neuroendocrine Changes and Reproduction*

In spite of the new knowledge about the brain presented earlier regarding the puberty-related structural and functional aspects of brain development, there is virtually no overlap in the literature between this newly derived knowledge and neuroendocrine, puberty-related brain changes. The neuroendocrine changes responsible for the onset and progression of puberty consist of reactivation of the hypothalamic-pituitary-gonadal (HPG) axis and are referred to as gonadarche. Reactivation of GnRH via the GnRH pulse generator is the primary component of the neurobiology of puberty. We refer to "reactivation" because the initial activation of GnRH occurs in the fetal and neonatal period.

GnRH is a decapeptide secreted in a pulsatile fashion by specialized neurons in the median eminence of the hypothalamus. GnRH stimulates the pituitary to secrete gonadotropins, luteinizing hormone (LH) and follicle-stimulating hormone (FSH). LH and FSH travel via peripheral circulating blood to affect target cells in the testes in males and ovaries in females. Vital to reproduction, gonadal hormones are stimulated by gonadotropins and regulate ovulation and spermatogenesis in females and males, respectively. Sisk and Foster (2004) suggest that in the brain gonadal steroids control GnRH secretion by way of neuroendocrine feedback loops and facilitate sexual behavior.

Much of what we know about the GnRH pulse generator and the onset of puberty derives from animal models, primarily from the rhesus monkey (see Plant, 2000; Knobil, 1988). The regulation of GnRH pulse frequency is responsible for producing a pattern of gonadotropin and steroid hormone secretion that is necessary for gonadal function and reproductive competence. The specific neural mechanism regulating GnRH secretion is not known in spite of decades of research. What is known is that activation of the HPG axis has a defined developmental history from the prenatal period to midadolescence. In prenatal and early postnatal life there is an increase in gonadal steroids that is responsible for sexual differentiation and organizing of the nervous system. In early infancy, GnRH pulse frequency declines and remains quiescent until puberty. At that time, GnRH secretion gradually increases and remains high, stimulating gonadotropin and gonadal hormone secretion during the reproductive years. In approximately the third decade of life, testosterone and estrogen begin to decline, reaching low but detectable levels within the older adult. The multiple species-specific signals for puberty are integrated in such a way as to allow for the increase in GnRH at puberty. Sisk and Zehr (2005) go on to suggest that permissive signals cannot fully explain the onset of reproductive competence, as such signals are not unique to puberty. For instance, GnRH pulse frequency goes from low to high in the resumption of fertility after lactation. It was suggested that an innate developmental clock senses the unfolding of primary genetic programs that produce signals that in turn determine responses to endogenous and exogenous signals (e.g., the environment) that bring about high-frequency GnRH pulses. There exists the notion that a master regulatory gene allows for programming of the multiple permissive genes, but to date, this master gene remains elusive.

The trigger that determines the timing of pubertal onset and the resurgence of GnRH pulses is not known. The logical explanation

that a specific gene triggers the onset of puberty has led to a search for a gene specific to the initiation of puberty. The identification of kisspeptin and its receptor, G protein coupled receptor 54 (GPR54), held promise as the gene responsible for the initiation of puberty (Seminara, 2003). GPR54 is a promising candidate, as the absence of this gene is associated with the absence of GnRH activity at puberty. But, to date, no single gene has been positively identified as a precipitant for the initiation of the complex pubertal process. Sisk and Foster (2004) suggest that there are multiple permissive signals rather than a singular cause of the onset of puberty: melatonin, leptin, ghrelin, body fat, and a complex of genes. Rather than a single trigger, it is likely the case that multiple triggers work in unison to bring about a resurgence of GnRH secretion.

#### *Sex Differences in Pubertal Triggers*

Males and females differ in permissive signals of puberty, and most of the triggers in women are related to energy balance. Sex differences likely stem from the different demands involved in reproduction. The beginning of the reproductive period is energy expensive for both males and females, given the rapid physical growth and metabolic changes that occur. In subhuman primates, males and females must defend the territory in which they will rear their young. However, females bear the added energy-intense period of pregnancy and childbirth and then must lactate to feed and rear offspring. Sisk and Foster (2004) suggest that organisms must determine whether growth is sufficient to reproduce (via metabolic cues), relationships with possible mates are possible (through social cues) and whether the conditions are conducive to reproducing (via environmental cues). In females, for successful reproduction, there must be, for example, insulin, glucose and leptin present in quantities sufficient to support pregnancy and lactation. Consider the case of leptin, an adipocyte-derived hormone that has been demonstrated to be a pivotal regulator for the

integration of energy homeostasis and reproduction (Fernandez-Fernandez et al., 2006). Leptin is necessary, but not sufficient to trigger the onset of puberty. When the permissive signals collectively bring about metabolic and growth conditions, reproduction becomes possible and is partially observable in secondary sexual characteristics.

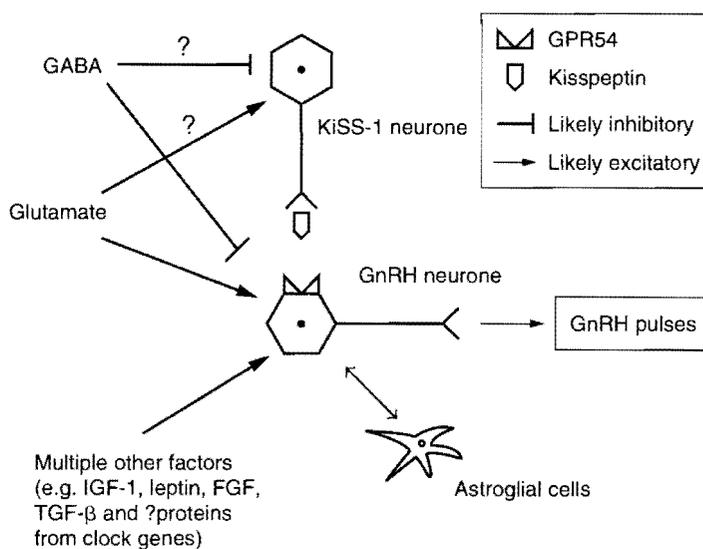
**Other Regulators of the Timing of Puberty**

Neurotransmitters also have an impact on GnRH. For example, gamma-aminobutyric acid (GABA) has primarily inhibitory control on GnRH (McCarthy, Davis, & Mong, 1997) and glutamate has excitatory control over GnRH (Grumbach, 2002). The system and its feedback loops are complex and are beyond the scope of this chapter. The reader is referred to Styne and Grumbach (2007). Figure 5.1 (Banerjee & Clayton, 2008) shows genetic and neurobiological pathways and other factors influencing puberty.

**Adrenarche**

The neurobiological and peripheral processes of gonadarche and the reproductive axis were described above. A second component of puberty, adrenarche, begins earlier than gonadarche at around age 6–8 years of age. Adrenarche reflects maturation of the adrenal glands, although the trigger for its initiation is unknown (Parker, 1991). Adrenal androgens such as dehydroepiandrosterone (DHEA), its sulfate (DHEAS), and androstenedione are products of the adrenal glands whose concentrations begin to rise at adrenarche and continue on an upward trajectory through the third decade of life (Saenger & Dimartino-Nardi, 2001). Adrenarche and gonadarche are two independent, yet overlapping, components of puberty.

Concentrations of adrenal androgens primarily have an impact on the development of axillary and pubic hair as well as acne and body odor. However, these concentrations generally are not high enough to act on peripheral target tissues until later, often after gonadarche. Pubic



**FIGURE 5.1** Neurobiological pathways and other factors influencing the activity of gonadotropin-releasing hormone neurons: Genetic variation in the components of these pathways and the other factors are likely to influence the time of onset and tempo of human puberty. GnRH, gonadotropin-releasing hormone; IGF-I, insulin-like growth factor-I; FGF, fibroblast growth factor; TGF, transforming growth factor. (Reprinted from Banerjee, I., & Clayton, P. (2007). The genetic basis for the timing of human puberty. *Journal of Neuroendocrinology*, 19(11), 831–838, with permission from Wiley-Blackwell Publishing Ltd.)

hair generally is not evident at age 6 when adrenarche alone has occurred. DHEA also serves as a precursor for the development of other androgens such as testosterone. In girls, much of the exposure of androgens comes from DHEA or its conversion. Concentrations of these hormones also have been associated with behavior problems and mood disturbances (Goodyer, Herbert, Tamplin, & Altham, 2000a, 2000b; Goodyer, Herbert, & Altham, 1998; Goodyer et al., 1996; Dorn, Hitt, & Rotentstein, 1999; Dorn et al., 2008). It is yet to be identified whether adrenal androgens have effects similar to testosterone on aggressive or other behaviors.

A third axis, the growth axis, also is involved in puberty. Growth, primarily linear growth, also involves neuroendocrine changes. Growth hormone (GH) increases, but there are also other growth factors that are key contributors to growth. Linear growth has been the focus of the psychology of youth as changes in linear growth, along with secondary sex characteristics, constitute the signal for adolescents themselves and others around them that the adolescent years have arrived.

#### Physical Changes at Puberty

External changes in height and weight and secondary sexual characteristics provide visible ways of marking the progress of pubertal development. These growth changes include increases in linear growth and body composition and development of primary sexual characteristics (ovaries, testes), as well as secondary sexual characteristics (pubic hair, genital and breast development). Collectively, all the changes are a result of the neuroendocrine development described earlier. Early descriptions of growth and pubertal changes in girls and boys were first described by Tanner (Tanner, 1962; Marshall & Tanner, 1969, 1970). Generally, the height spurt begins earlier in girls, at approximately age 12, than in boys, at about age 14 (Marshall & Tanner 1969, 1970). In girls the earliest external change evident at puberty is an increase in breast tissue (Tanner & Whitehouse, 1976; Parent et al.,

2003). Girls usually begin the development of secondary sex characteristics with breast development followed by pubic hair development, whereas boys generally begin genital growth with an increase in testicular volume followed by appearance of pubic hair. Girls tend to mature physically earlier than boys by approximately 18–24 months (Patton & Viner, 2007). *Menarche is a relatively late event in the pubertal process, occurring after the growth spurt and after breast development and pubic hair growth has begun.* The average age of menarche for White, African American, and Latino girls is: 12.6–12.9, 12.1–12.2, and 12.2–12.3 years, respectively (Wu, Mendola, & Buck, 2002; Chumlea et al., 2003). The rising gonadal and adrenal hormone (DHEA, DHEAS, and androstenedione) concentrations as a result of GnRH secretion result in the changes in primary and secondary sexual characteristics previously described. These hormones increase across puberty, but there are large individual differences resulting in overlap of hormones across adjacent stages of puberty (Styne & Grumbach, 2008; Nottelmann et al., 1987). The result of wide individual variations in hormone levels is that hormone levels cannot be matched to a specific pubertal stage. It follows that hormone levels to determine sexual maturity stage is not an option in research (See Dorn, Dahl, Woodward, & Biro, 2006, for further discussion).

#### Assessing Pubertal Maturation

Tanner and Marshall described methods of quantifying breast and pubic hair development for girls and genital and pubic hair for boys (Tanner, 1962; Marshall & Tanner, 1969, 1970). A five-stage rating system (1 = prepubertal to 5 = full maturation) was developed for each secondary sex characteristic, and the characteristics were originally adapted from Reynolds and Wines (1948). Photographs are available of these stages in endocrinology (e.g., Kronenberg, Melmed, Polonsky, & Larsen, 2008) or adolescent medicine textbooks (Neinstein, 2008), as well as psychology-related textbooks focusing

on adolescent development (Steinberg, 2007). Additional photographs showing these same stages were published later (van Wieringen, Roede, & Wit, 1985) and most recently appear in Biro and Dorn (2005). It is important to note that Tanner criteria for assessing pubertal maturation were developed from a relatively small sample of boys and girls (less than 400) housed in an institution in England. Thus, the sample is representative neither of the United Kingdom nor of other world populations. Further, the sample included only Caucasians youth. To our knowledge there are no photographs describing the stages in boys and girls of different ethnic groups.

#### *Components of Change*

Three characteristics of changes in puberty may have important implications for health and development in adolescence and beyond. These include timing, sequence, and tempo. Timing refers to being earlier or later compared to a normative age group. For example, breast development at age 3 signals the need to conduct an appropriate evaluation for precocious puberty. Alternatively, a 14- or 15-year-old boy with short stature (e.g., low percentile in height compared to norms) signals the need for an evaluation to determine a diagnosis of delayed puberty. A critical question is whether a developmental clock that determines the timing of GnRH maturation also determines gonadal maturation (Sisk & Foster, 2004). The majority of psychological research on puberty has focused on the importance of timing of puberty. This literature is briefly reviewed later in this chapter.

The sequence of pubertal components, that is, the order in which growth or development of secondary sex characteristic occurs, has received little empirical attention. Generalities of the sequence have been described by Tanner (1962) and others showing that in girls, breast development is often the first change, followed by the height spurt and pubic hair growth. Menarche is a relatively late event during the pubertal process. If this sequence deviates

substantially from the norm, it should set into motion further concerns by a parent or health care provider. For instance, early pubic hair development in the absence of breast development may reflect early adrenarche. In a recent paper, Biro and colleagues (2003) reported that most White girls with asynchronous maturation were more likely to begin puberty with breast development (thelarche) compared to initiation with pubic hair (adrenarche). Of interest is that the reported age of onset of puberty in both groups was 10.7 years. Specifically, girls in the thelarche pathway had a greater sum of skin folds, percent body fat, waist-to-hip ratio, and body mass index (BMI) at menarche, and many of these differences held across a 10-year longitudinal study. In this pathways paper, Biro et al. proposed that the sequence in which pubertal changes are experienced in girls may have long-term ramifications for health. For example, girls with breast development first, stimulated by gonadal axis activation, may be more at risk for obesity and breast cancer than girls with pubic hair development first. To our knowledge there are no studies that examine the psychological import of the sequence of pubertal development.

Finally, the tempo of puberty, that is, the rate of progression through puberty, also has received little empirical attention from either a medical or psychological perspective. Nonetheless, there have been citations indicating that the tempo of puberty has not really changed in the last several decades (Reiter & Lee, 2001). The tempo of puberty varies across adolescents, and differences in tempo will determine the later progression through puberty. For instance, at one time of measurement, one adolescent may be more advanced in physical maturation than another adolescent who began puberty at the same time, but one adolescent's maturational progression may slow so that measurement at a second time point finds the same adolescent as average in terms of tempo. In African American children aged 10-12 years old, Ge and colleagues (2003)

found that males who experienced accelerated pubertal maturation from the first to second assessment showed the highest increase in depressive symptoms. Does it follow then that a faster tempo constitutes a risk for depressive symptoms in males? The answer may be no. In a study of 327 male and female adolescents (aged 10–12 years at first assessment and 12–14 years at second assessment), boys with accelerated pubertal maturation had a *lower* risk of depression (Laitinen-Krispijn, van der Ende, & Verhulst, 1999). An unanswered question is whether age of onset of puberty is correlated with tempo of puberty or age of menarche. It seems appropriate to suggest, given the opposing findings above, the need to consider pubertal tempo when examining pubertal influences and depressive affect.

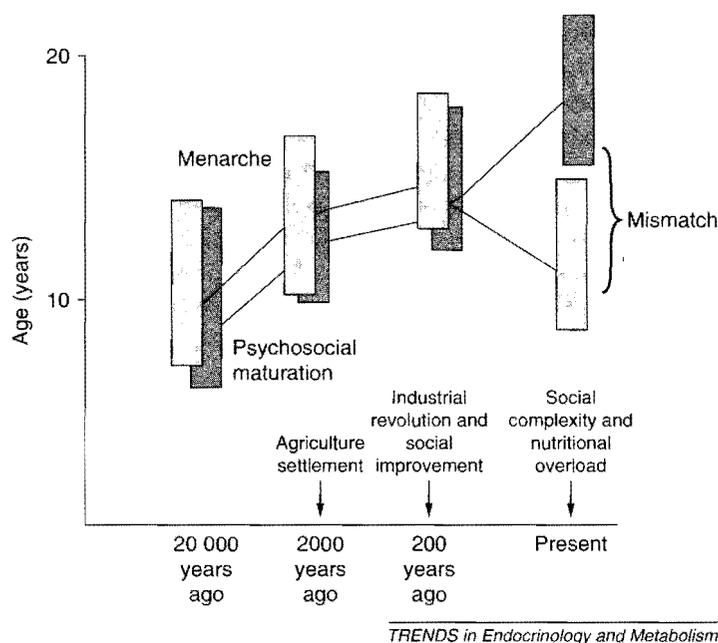
#### **Timing of Puberty: Its Psychological Significance**

The biological changes of puberty are universal, but the timing and social significance of these changes to adolescents themselves, societies, and scientific inquiry vary across historical time, individuals and families, and cultures. The psychological significance of the timing of puberty is one of the most popular topics examined by scholars of adolescent development.

#### ***Secular Trends in the Timing of Puberty***

The last decade evidenced a renewed interest in secular changes in the timing of puberty (Buck Louis et al., 2008; Euling et al., 2008; Golub et al., 2008; Kaplowitz, 2008). The increase in interest was sparked partially by a controversial paper by Herman-Giddens et al. (1997) showing that girls were entering puberty at an earlier age than was the norm in U.S. girls. Specifically, signs of puberty were evident in African American girls, on average, at age 7 and in Caucasian girls, on average, at age 9. The study was conducted on more than 17,000 girls, yet the report has been controversial based primarily on methodological issues (see Emans & Biro, 1998; Reiter & Lee, 2001; Rosenfield

et al., 2000). In another sample, Sun and colleagues reported older ages of onset of puberty based on breast development than did Herman-Giddens and colleagues (Sun et al., 2002). Other studies of girls showing earlier maturation (particularly by age at menarche) include findings from the National Health Examination Survey (Anderson, Dallal, & Must, 2003), the National Health and Nutrition Examination Survey (Chumlea et al., 2003), the Bogalusa Heart Study (Freedman et al., 2002), and the Fels Longitudinal Study (Demerath et al., 2004). Importantly, age at menarche was not reported to be earlier than in the past. Maturation in boys also is reported to be earlier than in previously described cohorts, as reported in a secondary data analysis from a cross-sectional survey of the National Health and Nutrition Examination Survey (Herman-Giddens, Wang, & Koch, 2001). The analysis included 2,114 boys who were identified as White, African American, or Mexican American. In all racial groups, boys matured earlier than reported in previous studies based on Tanner criteria for genital and pubic hair development. Further, African American boys had the earliest age of onset of puberty (age 11.2 years; Tanner 2) based on pubic hair and age 9.5 for genital development. The African American sample also reached full maturity of genital development (Tanner 5) at an earlier age than the other groups. Importantly, age at menarche, compared to age of onset of puberty, has not varied in recent history. Nonetheless, secular trends have shown dramatic differences in timing of puberty across time (Gluckman & Hanson, 2006) and may be changing too slowly to show an evolutionary change in the time period assessed (see Figure 5.2). In conclusion, Biro and colleagues (Biro, Huang, et al., 2006) note that the correlation is low between age of menarche and age of onset of puberty, and this correlation has gotten progressively lower across the decades. The divergence in the correlation between age of onset of puberty and age of menarche may result from the increase in weight in both boys and girls.



TRENDS in Endocrinology and Metabolism

**FIGURE 5.2** The relationship between the likely range of ages of menarche (light gray) and achievement of psychosocial maturation (dark gray) from 20,000 years ago to the present day. The mismatch in timing between these two processes is a novel phenomenon. (Reprinted from Gluckman, P. D., & Hanson, M. A. (2008). Evolution, development and timing of puberty. *Trends in Endocrinology & Metabolism*, 17(1), 7-12, with permission from Elsevier.)

Not all recent findings support the divergence of age of onset of puberty and age of menarche. Current speculation is that secular trends in the timing of puberty now may be characterized by stability rather than change. In Europe, Parent et al. (2003) reported that the downward trend in age of maturation is decreasing or is even slightly increasing in some instances. Similarly, Sun et al. (2005) noted no convincing evidence for earlier age of pubertal maturation from 1966 to 1994. Evidence to the contrary is that menarche, as opposed to pubertal maturation, also is decreasing in two longitudinal studies (Anderson & Must, 2005). A recent panel concluded that the findings for girls are sufficient to suggest a secular trend toward an earlier age of onset of breast development and age of menarche (Euling et al., 2008). In contrast, the panelists concluded that the data for boys were insufficient to make a conclusion about a secular trend in boys' development.

Recently, Biro (2006) raised the important question, "Whither goest puberty?" And what are the causes of the changes in timing of puberty? Although the exact cause of early timing of puberty is unknown, there are several plausible explanations. Several environmental factors beyond heritability could influence the earlier onset of puberty: nutritional status, chronic diseases, migration to a healthy environment, infectious diseases, pollution and exposure to environmental toxins (Delemarre-van de Waal, 2005). It is of note that we, and most of the literature on timing of puberty, are referring to a small variation in timing that is well within the normal range of timing of puberty or menarche from a health care perspective. Pediatric endocrinologists would likely not see these smaller differences as abnormal. Nonetheless, even minor variations in the timing of puberty may have implications for health and behavior. Following is a discussion regarding endogenous and exogenous influences on the timing of puberty.

### *Obesity and Timing of Puberty*

Obesity as an initiator of early timing of puberty is now a major concern given the epidemic of obesity that is rampant in North America and is spreading to other nations. The concern is that the obesity observed during adolescence will persist into adulthood. This concern was warranted in a prospective study of boys and girls (Bratberg, Nilsen, Holmen, & Vatten, 2007). The study examined the influence of early sexual maturation on subsequent overweight in late adolescence and found that timing did relate to later obesity, but this relation was modified by central adiposity in early adolescence.

A question in the past was whether early puberty initiates weight gain or whether heavier weight is an initiator of earlier puberty. The latter explanation has received recent support. Girls who were heavier at age 5 had an earlier timing of puberty based on both estradiol levels and Tanner stage (Davison, Susman, & Birch, 2003). In a similar vein, higher weight status even in young children was related to earlier timing of puberty (Lee et al., 2007). Surprisingly, infants' weight at 36 months and higher rate of change in BMI between 36 months and grade 1, earlier age of mothers' menarche, and non-White race each consistently and positively was associated with an earlier onset of puberty (Lee et al.). In a recent review, Biro and colleagues reported relatively consistent evidence for the relations of timing of puberty and obesity (Biro, Khoury, & Morrison, 2006) in older children and adolescents. Kaplowitz (2008) suggests that obesity may be causally related to earlier puberty in girls rather than earlier puberty causing an increase in body fat. Much of the research on timing of puberty and obesity is in girls and also shows that these relations are more evident in Black compared to White girls (Biro et al., 2006). These relations do not hold in boys for unknown reasons.

### *Endocrine Disruptors*

One of the proposed mechanisms of earlier timing of puberty focuses on endocrine

disruptors. Endocrine disruptors include a variety of substances that could be natural or synthetic environmental chemicals that disrupt or change normal endocrine functions (Nebesio & Pescovitz, 2007). Examples include such things as harsh pesticides or chemicals that come from industry processing or waste (e.g., polychlorinated biphenyls [PCBs], phthalates) that are converted to natural estrogens or phytoestrogens. Effects of these disruptors may be on the HPG axis concurrently or may result from accumulation over time, in turn contributing to later reproduction problems. Examination of the mechanisms involved in endocrine disruptors and timing of puberty has been carried out in both lower animal and human models.

There are extensive animal model studies showing the impact of environmental chemicals on pubertal timing, yet there is less empirical evidence in human models (see review by Buck Louis et al., 2008). Some of these studies report earlier timing of puberty as a result of exposure to disruptors, whereas others report later timing. It also seems that males may not be as affected by endocrine disruptors as females. Most of these studies using animals report on development following a known exposure to a chemical. Few studies of healthy children have included measures of endocrine disruptors. In studies that have found putative disruptor effects on timing of puberty, speculation is that endocrine disruptors may have played a role. For example, one of the proposed mechanisms involved in earlier puberty in African Americans was the use of hair products containing natural estrogens (Herman-Giddens et al., 1997). Exposure to specific chemicals in hair products, however, was not measured. In earlier reports, early puberty (precocious puberty, premature thelarche, premature adrenarche) in children in Puerto Rico was thought to be related to significant amounts of estrogen from the food supply (Bongiovanni, 1983; Comas, 1982; Saenz de Rodriguez, Bongiovanni, & Conde de Borrego, 1985). The amount of estrogen in the food supply was not determined.

Caution is warranted regarding the potential effect of these endocrine disruptors on health as well as on timing of puberty. Progress in the development of household and industrial products in our modern society, where there is also an increased use of household chemicals, pesticides, and plastics, may have a higher risk-benefit ratio than in the past.

#### *Measurement of Puberty: Pubertal Status and Pubertal Timing*

Pubertal status and pubertal timing are key variables in both biological and behavioral studies. Whether puberty is considered a cause or a consequence of the outcome at hand or whether it must be controlled for in a statistical analysis, both are variables deserving of considerable attention. Assessment of puberty is often missing in studies in which it is relevant or puberty may be measured in an inappropriate method with respect to the research question. Currently, the "gold standard" for measuring pubertal stage is by Tanner criteria (Marshall & Tanner, 1969, 1970) as assessed by a physical examination by a trained examiner. Frequently, psychosocial studies of adolescents measure pubertal maturation using self- or parent report of adolescents' maturation. The most widely used self-report measure is the Petersen Pubertal Development Scale (PDS; Petersen, Crockett, Richards, & Boxer, 1988). Adolescents report on specific puberty-related physical or auditory changes (e.g., voice, skin changes, body hair, linear growth). Other studies have used photographs or line drawings (Morris & Udry, 1980). Self-ratings are not perfectly related to assessments by physical examinations (i.e., by kappa coefficients) but in some cases can be strongly correlated with actual physical examinations. Although not perfectly correlated, there are some instances where self-report is an appropriate measure of puberty. The measure of puberty should match the research question. The reader is referred to a recent and more in-depth publication outlining methods of measuring puberty and their respective strengths and limitations

(Dorn et al., 2006). In that report, the more frequently used measures of pubertal status and pubertal timing are evaluated for strengths and weaknesses.

Age at menarche is another index frequently used to reflect pubertal development. This measure is most often obtained by self-report but also has been obtained via parent report using either a questionnaire or an interview. As mentioned previously, menarche is a relatively late event in the pubertal process. Thus, it can be equated neither with "pre- versus postpubertal" nor can it be used to determine the exact stage of pubertal development. Menarche does occur when the majority of girls are Tanner 4 (nearly two-thirds) but about 25% are Tanner 3, 5% Tanner 2, and 10% Tanner 5 when becoming menarcheal (Neinstein, 2008). Further, the consistency of reporting age at menarche across many years is not perfect. Correlations range from 0.60 to 0.81 (Bergsten-Brucefors, 1976; Casey et al., 1991; Damon & Bajema, 1974; Livson & McNeill, 1962; Must et al., 2002). In brief, age of menarche is an index of just that—age of menarche; it reveals little about pubertal maturation other than that gonadotropins and sex steroids are at levels essential for the beginning of the menstrual cycle.

The methods for estimating timing of puberty are varied. In some instances, indices of puberty are used to reflect adolescents as early, on time, or late with respect to a same-age cohort. Comparisons can be made to sex-specific national norms or the peer group to answer the question of whether a particular adolescent is on time, early, or late. Frequently, age at menarche or Tanner stage for age is categorized in this manner. For example, distributions of pubertal stage have been trichotomized or cut points have been determined by a specified number of standard deviations to estimate timing of puberty. Importantly, one needs to articulate the rationale for defining the method of timing of puberty in each instance.

Other studies have used a pubertal status (stage) measure to reflect pubertal timing as a continuous measure. For example, pubertal

stage has been regressed on chronological age and the residuals then used in analyses as an index of timing (Dorn, Ponirakis, & Susman, 2003; Ellis & Garber, 2000; Ellis et al., 1999). This methodology of considering the continuum of timing of puberty tends to be useful for smaller samples that are not population based. The use of stage residual scores allows for considering the relative age of pubertal stage or "earlier timing" rather than "early timing" as defined by a categorical variable.

***Does Pubertal Status or Timing Make a Difference in Psychological Development?***

This section presents a brief review of major findings on the relation between pubertal status and pubertal timing and psychological development. It first considers differences in psychological parameters between adolescents at different pubertal stages. Second, the section discusses the relation between psychological parameters and timing of puberty (earlier, later, or on time).

**Pubertal Status** The relationship between pubertal status and psychological functioning has not been as thoroughly examined as timing of puberty and psychological dimensions. Noteworthy are three reports from the Great Smokey Mountain longitudinal study that are relevant to pubertal status and psychological development (Costello et al., 1996). First, after the transition to midpuberty (Tanner Stage III and above), girls were more likely than boys to be depressed (Angold, Costello, & Worthman, 1998). Second, Angold, Costello, Erkanli, & Worthman (1999) examined whether the morphological changes associated with Tanner stage or the hormonal changes were more strongly associated with increased rates of depression in adolescent girls. Models that included testosterone and estradiol eliminated the seemingly strong Tanner stage differences and depression. The effect of testosterone on depression was nonlinear. These findings were interpreted as arguing against theories that

explain the emergence of the female excess of depression in terms of changes in body morphology and resultant psychosocial effects on social interactions and self-perception. The authors conclude that causal explanations of the increase in depression should focus on factors associated with changes in androgen and estrogen levels rather than the morphological changes of puberty.

Third, a later report from the Great Smokey Mountain study showed that after controlling for age, Tanner stage predicted alcohol use and alcohol use disorder (AUD) in both boys and girls (Costello, Sung, Worthman, & Angold, 2007). The effect of morphological development was strongest in early-maturing girls. Early pubertal maturation predicted alcohol use in boys and girls and AUD in girls. The highest level of excess risk for alcohol use was in early-maturing youth with conduct disorder and deviant peers. Lax supervision predicted alcohol use in early-maturing girls. In contrast, poverty and family problems predicted alcohol use in early-maturing boys. The dearth of recent studies on pubertal status differences in either internalizing or externalizing behavior problems prevents drawing definitive conclusions about pubertal status differences and psychological development. The need for assessment of stage differences is especially acute in studies that use sexual maturity ratings by a trained health care provider.

**Pubertal Timing** Pubertal timing and its connection to psychological adjustment has received significantly more attention than pubertal status. The literature on the psychological significance of timing of puberty has been extensively (See Alsaker, 1995) or partially reviewed in various publications (Mendle, Turkheimer, & Emery, 2007; Susman & Rogol, 2004). The next section presents representative studies showing the association between timing of puberty and internalizing and externalizing behavior problems.

When scientists began to consider the timing of the impact of physical maturation on mental

health and adjustment, primarily two hypotheses were articulated. First, the maturational deviance hypothesis, also coined the "off-time" hypothesis, suggested that those either early or late pubertal changes compared to same age peer group will likely experience more stress and adjustment difficulties than on-time developers (Brooks-Gunn, Petersen, & Eichorn, 1985; Caspi & Moffitt, 1991; Petersen & Taylor, 1980; Tschann et al., 1994). Off-time pubertal development may be more difficult in the absence of the necessary social support or resources to cope with earlier or later maturation. That is, if maturation is early, emotional and social resources may not yet be in place for the younger age adolescent. Alternatively, if maturation is late, social and family resources might be available but not at the time when they were most needed by the adolescent. In turn, adjustment problems may be more likely for either the earlier or the later maturer. Few studies focus on positive effects of either earlier or later timing of puberty.

The second hypothesis is the early-maturational or early timing hypothesis (Brooks-Gunn et al., 1985; Caspi & Moffitt, 1991; Petersen & Taylor, 1980; Tschann et al., 1994). This hypothesis suggests that early maturation, particularly in girls, is a disadvantage because girls bypassed the opportunity to complete the normative tasks of development that facilitate coping with the issues and problems associated with puberty. Adding to the stress of puberty, there are more advanced social role expectations for early maturers since their appearance is more physically mature, and in turn they are expected to act more maturely. Early-maturing girls also face more social pressures at a time when they are cognitively and emotionally unprepared to deal with abstractions and complex emotional issues. In brief, there is a mismatch between physical development and cognitive and emotional development. The findings from studies of timing of puberty and adjustment are not consistent in terms of whether earlier or later timing of puberty is good or bad for boys or girls.

### *Early Maturation and Internalizing Problems*

Early maturation for girls fairly consistently is associated with negative psychological and behavioral consequences (Ge, Brody, Conger, Simons, & Murry, 2002; Ge, Conger, & Elder, 2001a, 2001b; Graber, Lewinsohn, Seeley, & Brooks-Gunn, 1997; Hayward, Gotlib, Schraedley, & Litt, 1999). Representative findings are that early-maturing girls report more negative emotions than on-time or later maturing peers. For instance, Ge and colleagues (2001a) report findings showing that seventh-grade girls who experienced menarche at younger ages subsequently experienced a higher level of depressive symptoms than on-time and later maturing peers. The gender differences disappeared when pubertal transition indices (pubertal status and menarche) were included in the model. The finding is important as it indicates that pubertal maturation may explain the often reported gender difference in depressive symptoms. The effects of early maturation are true for boys as well as for girls. Early-maturing boys reported more hostile feelings and internalized distress symptoms compared to on-time and later maturing boys (Ge et al., 2001b). The negative effects of early maturation for boys are inconsistent with the earlier findings reporting positive effects of early timing of puberty on boys.

### *Early Maturation and Externalizing Problems*

The early-maturational hypothesis suggests that earlier maturing girls engage in more acting-out behavior than on-time or later maturing peers (Ge et al., 2001a). Studies such as those of Ge and colleagues do report that earlier maturing girls tend to exhibit more behavior problems and adjustment difficulties than their on-time or later maturing peers. But the relationship between earlier timing and externalizing behavior problems can be moderated by a history of vulnerability (Caspi & Moffitt, 1991). Early maturers with a history of behavior problems later reported more behavior

problems than on-time or later maturers without a history of behavior problems. In addition, earlier maturing girls with no history of behavior problems experienced fewer behavior problems than girls with a history of behavior problems who matured on time.

A consistent finding related to pubertal timing is the link between earlier maturation and substance use. Substance use is more prevalent in both earlier maturing boys and girls (Dick, Rose, Kaprio, & Viken, 2000; Orr & Ingersoll, 1995; van Jaarsveld, Fidler, Simon, & Wardle, 2007; Wilson, Killen, & Hayward, 1994). Specifically, early timing of maturation is linked to a higher incidence of smoking (Martin et al., 2001). Early timing of maturation and substance use also occurs across cultures. In East and West German youth, earlier timing was associated with more frequent cigarette and alcohol use (Wiesner & Ittel, 2002). On-time and later maturation was not associated with substance use. Similarly, in Norwegian and Swedish youth, earlier puberty was associated with alcohol use, alcohol intoxication, drunkenness onset, and number of units consumed (Andersson & Magnusson, 1990; Wichstrøm, 2001). The mechanisms relating early timing of puberty and substance use have received a moderate amount of attention. Substance use may lower resistance to peer pressure to use various substances increasing both substance use and other health-compromising behaviors, such as risky sexual behavior. In brief, the growing body of literature indicates that early pubertal maturation in girls is generally associated with negative psychological and health outcomes in Western societies. The findings are strong enough at this date to suggest to policy makers that programs to reduce externalizing behavior, principally substance use and risky behavior, should be initiated earlier than in the past for adolescent girls who exhibit early signs of puberty. The findings to date are deemed insufficient to make a similar recommendation for boys.

#### ***Timing of Puberty and Health***

A new set of findings shows that timing of puberty has potentially serious implications

for health problems, either during adolescence or later in the life span. Adolescents with earlier puberty are at risk for accelerated skeletal maturity and short adult height, earlier sexual debut, and a higher risk for sexual abuse (Golub et al., 2008). An even more serious implication of earlier timing of puberty is its association with reproductive tract cancers later in life. Earlier age of menarche is a risk for breast cancer and lower age of puberty in boys is associated with a higher incidence of testicular cancer (Golub et al., 2008). One explanation for the link between earlier timing of puberty and reproductive cancer is a longer duration of exposure to estrogen in girls and testosterone in boys. Estrogen and testosterone are growth factors that putatively contribute to reproductive cancer.

In the earlier discussion, the focus was on timing of gonadarche and health risks. Early adrenarche also carries a risk for health problems as well: metabolic syndrome or ovarian hyperandrogenism/polycystic ovarian syndrome (PCOS) in adulthood (Ibáñez, Dimartino-Nardi, Potau, & Saenger, 2000). PCOS is a disorder characterized by infertility, hirsutism, obesity, menstrual disorders, and ovarian atretic follicles. These problems in turn put females at risk for obesity, type 2 diabetes, cardiovascular disease, and infertility (Golub et al., 2008). Obesity and reproductive problems additionally are associated with mood and affect problems, and there is speculation that depression and mood problems may play a role in the onset of these disorders. Premature adrenarche also is associated with mood and behavioral problems (Dorn, Hitt, & Rotenstein, 1999; Dorn, 2007). Longitudinal studies to establish the connection between timing of gonadarche and adrenarche and metabolic and psychiatric disorders are not yet available.

Earlier timing of puberty has negative implications for lifestyle and health behaviors. In addition to the well-known relationship between earlier timing of puberty and smoking, earlier maturers reported more sedentary behavior, but earlier maturation was offset by

physical activity (van Jaarsveld et al., 2007). The same study also reported lower rates of breakfast eating and higher reported stress, especially among early-maturing girls. To date, no policy recommendations are available regarding education of adolescents and their families regarding timing of puberty and health risks.

#### *Spermarche as an Index of Timing of Puberty*

Spermarche (oigarche) is rarely used as an index of timing in boys compared to age of menarche as an index of timing in girls. Reaching spermarche at 11 years or younger was associated with higher levels of depression (Kaltiala-Heino, Marttunen, Rantanen, & Rimpela, 2003). But later timing of spermarche also was associated with depression. A second paper from the same sample yielded different results as the adolescents were grouped based on age at menarche or spermarche (10 or younger, 11 years, 12 years, 13 years, 14 years, and 15 years or older) (Kaltiala-Heino, Kosunen, & Rimpela, 2003). In boys, only early maturation increased the risk of depression. It is not surprising that spermarche is an infrequent index of timing of puberty given that the presence of sperm in urine is a preferred way to assess spermarche. But urine is difficult to accurately collect as it is an embarrassing and sensitive procedure for some youth. Nonetheless, indices of the timing of spermarche provide a potentially valid and needed index of pubertal maturation.

#### *Family Influences on Timing of Puberty and Menarche*

The evolution of puberty occurred so as to maximize the probability for successful procreation. Puberty-related mutations in successive generations have favored biological qualities fostering survival in particular geographic and cultural settings. Draper & Harpending (1982) proposed that individuals have evolved to be sensitive to features of their early childhood environment that may affect later reproductive competence. Belsky, Steinberg, & Draper

(1991) extended this perspective to consider family disruption/father absence and early timing of puberty or menarche. Psychosocial acceleration theory and life history theory have been used to explain the unusual connection between family disruption/father absence and timing of puberty. The psychosocial acceleration theory was proposed first by Belsky et al. to explain the role of family disruption/father absence and early timing of menarche. Specifically, Belsky et al. proposed an evolutionary function of family ecology and psychosocial influences to explain earlier timing of puberty. From an early age, children are socialized to develop an understanding of the availability and predictability of resources, the trustworthiness of meaningful others, and the enduring quality of close relationships. In brief, individuals are presumed to become sensitive to qualities of the environment that enhance or suppress reproductive tendencies. For instance, family stressors create conditions that undermine parental functioning, such as parental conflict, and lower parental investment in girls. Girls from families characterized by father absence or discordant male-female relationships perceive males as less salient to family relationships and lack parental investment in offspring. Given the perceived instability of relationships with males, girls accelerate the timing of menarche and subsequently engage in early sexual activity and unstable pair bonding. Mendle et al. (2006) later similarly proposed that unstable parental relationships subsequently lead girls to believe that resources are limited, people are untrustworthy, and relationships opportunistic. Further, parents' mating reproductive behavior characterized by multiple sexual partners and erratic relationships contribute to a girl's sense of a transient family situation.

In a somewhat similar perspective to the psychosocial acceleration theory, Ellis and Essex (2007) proposed a life history theory linking family structure and processes and timing of puberty. They emphasize the importance of life history traits, that is, the constellation of maturational and reproductive characteristics

that influence speed of reproduction and population turnover. A key assumption of the life history approach is that individuals make trade-offs in distribution of metabolic resources to sustain vital functions: growth, reproduction, and maintenance, given that resources are finite and unpredictable. Individuals choose evolutionarily relevant features of the environment as a basis for altering the timing and tempo of pubertal maturation (Ellis, 2004). A life history perspective asks questions about when individuals should stop converting surplus energy related to growth and begin to shift resources to reproduction.

Published studies based on acceleration and the life history perspectives show support for the hypotheses regarding timing of puberty or menarche and family structure or processes (see also Ellis et al., 2004). One notable and consistent finding is that menarche occurs at an earlier age among girls raised in stressful family circumstances (Doughty & Rodgers, 2000; Jones, Lenton, McLeod, & Wood, 1972; Kim & Smith, 1998a, 1998b; Kim, Smith, & Palermiti, 1997; Moffitt, Caspi, Belsky, & Silva, 1992; Surbey, 1990). The majority of the studies use father absence as a marker of family disruption in relation to timing of menarche. Other studies assess father absence, family dysfunction, or qualities of parenting (Belsky et al., 2007; Ellis & Essex, 2007; Surbey, 1998) or family composition (e.g., stepbrothers) (Matchock & Susman, 2006). Thus, it is difficult to separate the effects of family disruption as indexed by father absence and family disruption. Table 5.1 shows representative findings from father absence and family disruption studies.

#### *Father Absence*

The absence of a biological figure and its effects on timing of puberty or menarche was the original focus of studies of the sociobiology of puberty. The general conception was that father absence or some permutation of father absence (i.e., stepparent) was related to earlier menarche in father-absent homes. For instance, Surbey (1990) reported that girls

achieved earlier menarche in father-absent homes than in father-present homes. The girls in the survey who also experienced high levels of stress also achieved earlier menarche. Since the early Surbey (1990) study, more and more family and contextual measures have been added to the analyses of family structure and function and timing of puberty or menarche (See also Bogaert, 2008). The inclusion of family functioning as in the case of parental investment and early predictors of timing of puberty (e.g., Belsky et al., 2007) has enriched the wealth of findings on the sociobiology of puberty. Nonetheless, specific mechanism and pathways linking timing of puberty and menarche to contextual and individual level variables have not been identified.

Family stress or disruption refers to events that are considered stressors in families and include mental health problems of parents, family conflict, and financial and employment problems. The psychosocial acceleration and life history evolution-based perspectives propose that family disruption and father absence play a causal role in early timing of puberty (Belsky et al., 1991). Girls in these families engage in early sexual behavior and early reproduction (Draper & Harpending, 1982; Ellis, 2004). In one of the first tests of the psychosocial acceleration theory, Moffitt, Caspi, Belsky, and Silva (1992) showed that family conflict, divorce and father absence in childhood predicted earlier age of menarche. Weight also contributed to the influence on earlier menarche. Similarly, in a retrospective study of college age girls, earlier menarche was related to family stress in later childhood (age 7–11), conflict with mother and anxiousness and internalizing symptoms (anxiousness/depression) in early childhood (birth to age 6), and earlier age at dating boys and more boy-friends (Kim & Smith, 1998a). Other studies report that greater marital and family conflict is associated with primarily early timing of puberty in girls (Ellis & Garber, 2000; Graber, Brooks-Gunn, & Warren, 1995). Longer durations of father absence also correlate with an

**Table 5.1 Studies Examining Family Contextual Correlates of Pubertal Timing.**

Authors	Sample	Puberty Measures	Family/adolescent Characteristic	Findings	
				Males	Females
Belsky, Steinberg, Houts, Friedman, DeHart, Cauffman, Roisman, Halpern-Felsher, Susman, NICHD Early Child Care Research Network (2007).	N = 756 Boys and Girls	Tanner Stage	Rearing experiences Mother's age of menarche Infant negative emotionality Rearing by emotionally interaction	Pubertal onset was not significantly predicted by maternal age of menarche, any rearing variables, or by infant negativity.	Mothers with earlier menarche had daughters with earlier puberty. Insensitive parenting earlier in childhood, predicted earlier pubertal development. Maternal harsh control predicted earlier menarche
Bogaert (2008).	N = 5913 Girls	Menarche	Father absence Siblings		Earlier age of puberty was predicted by father absence
Ellis & Essex (2007).	N = 756 Girls	Pubertal Timing Menarche	Rearing experiences.	Pubertal onset was not predicted by maternal age of menarche, rearing variables or infant negativity.	Mothers who reported an earlier age of menarche had daughters who reported earlier age of menarche and initiated pubertal development earlier than did girls whose mothers reported later age of menarche. Infant negative emotionality did not significantly predict pubertal outcome.
Ellis, Bates, Dodge, Fergusson, Horwood, Pettit, & Woodward (2003).	N = 281 Girls	Pubertal Timing	Father absence Early sexual activity Teenage pregnancy		Greater exposure to father absence was strongly associated with elevated risk for early sexual activity and adolescent pregnancy. Elevated risk was either not explained or only partly explained by familial, ecological, and personal disadvantages associated with father absence. After controlling for covariates, there was stronger and more consistent evidence of effects of father absence on early sexual activity and teenage pregnancy than on other behavioral or mental health problems or academic achievement.

Ellis, Bates, Dodge, Fergusson, Horwood, Pettit, & Woodward (2003).	N = 630 Girls	Petersen Development Scale	Father absence	Father absence was associated with earlier sexual activity and teenage pregnancy Father absence earlier in the girl's life was associated with sexual activity and teenage pregnancy
Ellis & Garber (2000)	N = 87 Girls	Menarche	Maternal psychopathology Discordant family relationships and father/stepfather absence. Stress in mothers' romantic relationships	History of mood disorders in mothers predicted earlier pubertal timing in daughters. Relation was mediated by dyadic stress and biological father absence; step father presence was best accounted for earlier pubertal maturation in girls
Ellis, McFadyen-Ketchum, Dodge, Pettit, & Bates (1999).	N = 173 Girls	Pubertal timing	Negative-coercive parenting	Negative-coercive (or less positive-harmonious) family relationships in early childhood provoked earlier puberty. Fathers' presence in the home, more time spent by fathers in child care, greater supportiveness in the parental dyad, more father-daughter affection, and more mother-daughter affection, prior to kindergarten, each predicted later pubertal timing by daughters
Graber, Brooks-Gunn, & Warren (1995)	N = 75 Girls	Pubertal Timing Menarche	Heredity Transmission Weight and weight for height Stressful life events Family relationships Absence or present of an adult male	Breast development, weight, family relations, and depressive affect were predictive of age at menarche; family relations predicted age of menarche above the influence of breast development or weight.
Kim & Smith (1998a)	N = 28 Daughters N = 21 Mothers	Menarche	Childhood stress Conflict in family environment	Earlier menarche correlated with family stress at late childhood; more conflict with mother in early childhood; more rejection from, and less closeness to, mother throughout childhood; more anxious and internalizing symptoms in late childhood; earlier age at dating boys and more boyfriends.

(Continued)

**Table 5.1 (Continued)**

Authors	Sample	Puberty Measures	Family/adolescent Characteristic	Findings	
				Males	Females
Kim & Smith (1998b).	N = 357 Boys and girls	Age at Menarche and Spermarche		Earlier spermarche was associated with father absence; more stress in quality of family life, parental marital unhappiness and parental marital conflict in early childhood; more independence from mother and father in late childhood; earlier age at dating women, more girlfriends, and earlier age at sexual intercourse.	Earlier menarche was associated with parental marital conflict in early childhood; more parental marital unhappiness throughout childhood; more independence from mother and father in late childhood; less anxiousness or internalizing symptoms (anxiousness/depression) in late childhood; earlier age at dating men and more boyfriends.
Kim, Smith, & Palermi (1997).	N = 380 Boys and Girls	Menarche and spermarche	Early childhood stress or conflict in the family environment	Earlier spermarche was associated with more parental marital conflict, less emotional closeness to father; more aggressiveness, unruliness, and externalizing symptoms in early or late childhood. Earlier spermarche was associated with earlier age at dating women, having more girlfriends, having had intercourse and having more total frequency of intercourse partners.	Earlier menarche was associated with stress in quality of family life; unhappiness in parental marital relations during childhood; more conflict with mother; less emotional closeness to mother; and behavioral independence from mother or father in late childhood. Earlier menarche was associated with earlier age at dating men and older age of first sexual intercourse partner relative to own age at first intercourse.
Matchock & Susman (2006).	N = 1938 Women	Menarche	Absence of Biological Father Presence of half or step-brothers Living in an urban environment Body weight Race Siblings		Absence of the biological father, presence of half or step-brothers and living in an urban environment were all predictors of earlier menarche. Body weight and race were also related to menarche timing. The number of siblings and the birth order had no effect on menarche. Although presence of a sister showed delay in menarche, especially if they were older.

Mendle, Turkheimer, D'Onofrio, Lynch, Emery, Slustke, & Martin (2006).	N = 1284 Girls	Menarche	Childhood stress		Within the discordant MZ twin pairs, children raised by the twin providing a stepfather had an age of menarche slightly earlier than children raised by the co twin without a stepfather. The opposite pattern occurred in children of the discordant DZ pairs.
Moffitt, Caspi & Silva (1992).	N = 1037 Girls	Menarche	Family Stress Behavioral and Psychological Problems		Family conflict and father absence in childhood predicted an earlier age of menarche and these factors combined with weight showed some evidence of an additive influence on menarche.
Mustanski, Viken, Kaprio, Pulkkinen, & Rose (2004).	N = 1891 Boys and Girls	Pubertal Development Scale	Father absence Environmental influences	Genetic influences made the largest contribution to variance common to PDS items. Genetic and non shared environmental factors accounted for variation specific to PDS items in boys. Genetic effects were partially sex specific. Subsidiary analyses found accelerated maturation in boys who at age 14 were reared in father-absent homes.	Genetic influences made the largest contribution to variance common to PDS items. In girls, common environmental influences were added for growth spurt and menarcheal status. For both common and item-specific variation, genetic effects were partially sex specific. Subsidiary analyses found accelerated maturation in for girls who at age 14 were reared in father-absent homes.
Romans, Martin, Gendall, Herbison (2003).	N = 2225 Girls	Menarche	Father absence Family conflict Genetic determination of early puberty		Early menarche was predicted by low family socio-economic status, absence of father, family conflict, poor relationships between the girl and either/both parents, a self-rated childhood personality style as a loner, and childhood physical and sexual abuse.

(Continued)

Table 5.1 (Continued)

Authors	Sample	Puberty Measures	Family/adolescent Characteristic	Findings	
				Males	Females
Surbey (1998).	N = 1200 Girls	Pubertal Timing Menarche	Father absence Heightened levels of childhood stress		Menarche was earlier in girls from father-absent households and those reporting high levels of stress during childhood. The mothers of the father-absent girls also were early maturers, began dating early, tended to have their first child at an earlier age and have more children, and had more negative attitudes toward males and the family than mothers of father present girls.
Wierson, Long, & Forehand (1993).	N = 71 Girls	Menarche	Divorced Families Interparental Conflict		Earlier menarche in girls from divorced families. Earlier menarche in families with maternal reported interparental conflict.
Wierson, Long, & Forehand (1993).	N = 71 Girls	Menarche	Environmental Stress Divorce Interparental conflict		Compared to girls with intact families, those from divorced families had earlier onset of menarche. Higher maternal reports of interparental conflict were significantly related to earlier menarche in the total sample.

earlier menarcheal onset (Moffitt et al., 1992; Romans, Martin, Gendall, & Herbison, 2003; Surbey, 1990; Wierson, Long, & Forehand, 1993). Studies of family stress and timing of puberty in males are more rare than studies of girls.

Others have argued that the posited causal relationship between family structure and functioning and timing of puberty can be explained by uncontrolled genetic and environmental influences (Mendle et al., 2006). Girls who experience early menarche are more likely to be from lower socioeconomic status (SES) families, experience psychological difficulties, and transmit the likelihood of earlier development to children (e.g., Mustanski, Viken, Kaprio, Pulkkinen, & Rose, 2004; Stattin & Magnusson, 1990; Udry, 1979). These are potential confounds that cannot be controlled in research that compares age of menarche among unrelated individuals. Mendle et al. (2006) did a comparison of the stepparenting effects in the children of monozygotic (MZ) and dizygotic (DZ) twins and determined that this design can potentially discriminate whether confounds in the association between stepfathering and menarche are mediated via genetics or shared environment. A within-pair difference between the daughters of discordant DZ pairs but not between daughters of discordant MZ pairs suggests that the confound is primarily genetic. This is because DZ twins differ in genetic endowment and shared environment, whereas MZ twins differ only in their shared environment. A comparison of the stepparenting effects in the children of MZ and DZ twins can potentially discriminate whether confounds in the association between stepfathering and menarche are mediated via genetics or shared environment.

Variables indicative of stress were included by Mendle and colleagues (2006): divorce, being raised by both biological parents in the household, presence of a stepfather in the household, and absence of a biological father in child rearing. Results show that girls reared

in families with stepfathers exhibit a significantly earlier age of menarche than girls raised without stepfathers. Of note is that the presence of a step-uncle was as predictive of early menarche as the presence of a stepfather. That is, it was not necessary for a child to experience the direct environmental influence of a stepfather to experience an accelerated age of menarche, if she is genetically related to someone who does have a stepfather. In brief, in a pair of twin mothers, of which only one raises her children with a stepfather, the offspring of both twins are equally likely to exhibit early age of menarche. Mendle et al. conclude that some genetic or shared environmental confound accounts for the earlier menarche found in female children living with stepfathers. However, it is also possible that earlier menarche results from some shared environmental quality.

#### *Parental Investment*

Parental investment refers to the quantity and quality of parenting of offspring. For example, father investment as indexed by qualities of parenting and timing of puberty was considered (Moffitt et al., 1992). Negative aspects of family environment (family conflict, maternal harsh control) and earlier timing of menarche were related to timing of puberty. Recently, Ellis and Essex (2007) used a life history approach and proposed that humans have evolved to be sensitive to specific features of early childhood environments and that exposure to different parental environments bias children toward development of alternative reproductive strategies, including differential pubertal timing. Ellis (2004) extended the Belsky et al. (1991) perspective to parental investment by positing a unique and central role for fathers in the regulation of female offspring sexual development, separate from the effects of other dimensions of psychosocial stress and family support in the child's ecology. Higher quality parental investment in the preschool years and greater parental supportiveness

predicted lower rates of evidence of adrenarche, the early aspect of puberty, in boys and girls in the first grade and less development of secondary sexual characteristics in girls in the fifth grade (Ellis & Essex, 2007). Higher quality mother and father parental investment and less father-reported maternal conflict or depression predicted later adrenarche, the early phase of puberty. Older age at menarche in mothers, higher SES, greater mother-based supportiveness, and lower third-grade body mass index also uniquely and significantly predicted later sexual maturity in daughters. Ellis and Essex (2007) concluded that, consistent with a life history perspective, quality of parental investment emerged as a central dimension of the proximal family environment and pubertal timing.

These findings are consistent with the previous Ellis et al. (1999) findings. Parental warmth, positive family relationships, and paternal involvement in child rearing were related to a later age of menarche. The quality of the father's investment in the family emerged as the most important feature of the proximal family environment as a correlate of daughters' pubertal timing. Parenting qualities consisted of greater supportiveness in the parental dyad, more time spent by fathers in child care, more father-daughter affection, and more mother-daughter affection. Positive parenting characteristics, according to the child development theory (CDT), reap a longer childhood (later sexual maturation and onset of reproduction) and that high-investing family contexts foster development of sociocompetitive competencies. In contrast, the costs of truncating childhood are reduced in low-investing family contexts that do not meaningfully facilitate competitive competencies.

The argument proposed by Ellis and Essex (2007) regarding early age at adrenarche and family processes and the ecology of development has good internal consistency. Nonetheless, the logic is not consistent with the biology of reproduction. Adrenarche is independent of gonadarche; children can experience gonadarche without adrenarche and children

enter adrenarche in the absence of activation of the GnRH system. Specifically, adrenarche is not accompanied by increases in gonadal steroids, testosterone and estradiol, hormones that are essential for ovulation, spermatogenesis, and reproduction. Thus, the evolutionary significance of the timing of adrenarche is irrelevant to reproduction. In brief, family environment appears to affect a puberty-related event, adrenarche, that is not related to reproduction suggesting a reconsideration of the role of family environment and early timing of puberty as a reproductive strategy.

A limitation of many of the studies on family and contextual influences on age of onset of puberty or menarche is that they are cross-sectional or short-term longitudinal. Belsky et al. (2007) overcame this limitation by capitalizing on a 12-year longitudinal design to assess the effects of family influences on timing of puberty. Other major strengths of the study were that it included boys *and* girls and mothers and fathers, the adolescents had annual repeated assessments of pubertal development by trained nurses and physicians during puberty, and both family structure and process were considered. The results were both consistent and inconsistent with earlier findings. Rearing experiences predicted pubertal timing among girls but not boys. There are too few studies that included boys to know how to evaluate the consistency of this finding. Maternal age at menarche was a stronger predictor of girls' pubertal development than were early rearing conditions suggesting the strength of the genetic influence on timing of menarche. A unique finding was that early maternal harsh control at 54 months and at first and third grade predicted earlier menarche. An additional novel finding was that negative experiences with fathers and mothers predicted earlier age of menarche. In summary, the study yielded multiple novel findings supporting the influence of parenting characteristics on timing of puberty and menarche. A prominent limitation of the study is the small effect size and the limited number of significant findings.

Nonetheless, it is noteworthy that any effects emerged given the extended time frame of the study and the major genetic influences on timing of puberty.

Explanations are varied regarding the mechanisms involved in father absence, family disruption and timing of puberty. The studies based on the evolutionary model imply a causal relation between family disruption/father absence and timing of age at menarche or timing of puberty. One explanation is that a stressful family environment characterized by family conflict, absence of a biological father, poverty, and accompanying low parental investment creates an internalizing disorder that lowers metabolism, thereby inciting a weight gain that in turn accelerates menarche (Belsky, et al., 1991; Ellis & Essex, 2007). Recent evidence raises questions regarding mechanisms involved in weight and earlier puberty. Heavier weight status even during the preschool- and school-age period predicted earlier timing of puberty (Davison et al., 2003; Lee et al., 2007). The question, then, is: How does this early weight gain affect timing of puberty? To test the hypothesis that family environment and weight status in combination are mechanisms involved in early timing of puberty, future studies need to be longitudinal from infancy to the onset of puberty.

Family influences on acceleration of timing of puberty and menarche are based on the assumption that the family environment is stressful, likely arising from the unpredictability of parental absence, psychopathology, and other forms of family disruption. However, objectively measured stress is not part of the design of the studies, for the most part. Stress is merely implied by family conflict, poverty, and father absence or stepparenting. What is needed are good measures of subjective aspects of stress as well as the neuroendocrine indices of stress in family members to establish the mechanisms involved in stress and timing of puberty.

Noteworthy is that the acceleration and life history perspectives on stress and reproduction are at odds with the neuroendocrinology of

reproduction. Cortisol exerts inhibitory effects on reproduction at the levels of the GnRH neuron, the pituitary gonadotroph (responsible for secreting LH and FSH), and the gonad itself, thereby suppressing sex steroids (T and estrogen) and delaying maturation of reproductive function and physical growth (See Susman, Reiter, Ford, & Dorn, 2002). That is, CRH, ACTH, and cortisol suppress the reproductive axis. In brief, theories regarding putative family stress and timing of puberty or menarche run counter to the well-known physiology of stress and reproduction. The two sets of evidence can be reconciled nonetheless. Although acute stress may suppress reproductive functioning, chronic stress may release gonadotropins and sex steroids from the downregulating effects of CRH, ACTH, and cortisol. Family stress is likely to be of long duration, thus attenuating the classic acute response to stress. To advance the field, future studies would benefit from including: (1) objective indices of family stress, family structure and interactions, and reliable and valid measures of timing of puberty and menarche; and (2) longitudinal assessment of early family adversity and the neurobiology of stress and reproduction for long periods prior to the onset of puberty.

Genetics can be an alternative explanation to the evolutionary model of family relations and timing of puberty (See Mendle et al., 2006). Girls who mature early tend to have earlier sexual activity, and earlier age of first marriage and first birth, which increases the probability of divorce and lower quality paternal investment. Puberty, sexual, and reproductive timing may be genetically programmed (Ellis et al., 1999). Mothers who are early maturers tend to have daughters who are early maturers (Brooks-Gunn & Warren, 1989), suggesting a genetic linkage. In addition, the androgen receptor (AR) gene has been used as an explanation of the effect of father absence on age of menarche (Comings, Muhleman, Johnson, & MacMurray, 2002). Shorter alleles of the X-linked AR gene are associated with aggression, greater numbers of sexual partners, impulsivity, and divorce

in males, and with early age of menarche in females (Comings et al.). The same genes may predispose children to early timing and early sexual activity. Comings et al. also reminded the field that there are paternal as well as maternal influences on age of menarche. The same genetic factors that influence a man's likelihood to abandon marriages may also contribute to an earlier age of menarche in female offspring.

Mendle et al. (2006) tested an alternative hypothesis that nonrandom selection into stepfathering families related to shared environmental and/or genetic predispositions leads to a spurious relation between stepfathering and earlier menarche (also discussed earlier in this chapter). Using the controls for genetic and shared environmental experiences offered by the children-of-twins design, cousins discordant for stepfathering were found not to differ in age of menarche. Moreover, controlling for mother's age of menarche eliminated differences in menarcheal age associated with stepfathering in unrelated girls. These findings strongly suggest that selection and not causation accounts for the relationship between stepfathering and early menarche.

As an argument counter to genetic influences, it could be proposed that the effect is still there between family environmental factors and timing of puberty when mothers' age of menarche is controlled. Mothers' age of menarche could be considered a poor control for genetic influences. Age of menarche is a broad proxy for a genetic influence, and there is significant error of measurement in retrospective recall of age of menarche. Molecular genetics approaches yielding new genetic markers will likely be illustrative of the complexity of genetic influences on timing of puberty in functional and disruptive families.

### **Mechanisms Involved in Timing of Puberty and Adjustment**

The mechanisms relating timing of puberty and psychosocial adjustment, as discussed here and elsewhere, remain an empirical largely unknown. The findings are based on not

well-articulated theories and few mechanisms are proposed to explain the authenticated connection between timing of puberty and a diverse array of behaviors, emotions, and disorders and family structure and functioning. What then are the possible explanations for the long-standing connection between timing of puberty and psychological development? The explanation can be separated into two broad categories: social/contextual and brain development. Social/contextual explanations include peers, family, school, and the broader social ecology of youth. A fairly consistent explanation of earlier maturation and deviant behavior is that early maturers tend to associate with older and deviant peers. Older deviant peers may perceive the early-maturing adolescent as older than his/her stated age and include the early-maturing adolescent in adult-like deviant behaviors that include substance use and risky sexual activity. In the case of sexually risky behaviors, early-maturing girls also may be preyed upon by older boys and men, as these males perceive the girls as inexperienced and vulnerable to sexual coercion. Later maturing boys and girls may be protected by their childish appearance and are excluded by older, deviant peers.

Family members and teachers exert influences on an off-time maturing adolescent via expectations and social roles. The juvenile appearance of a late-maturing adolescent can lead to assigning less responsibility and independence to the childlike-appearing adolescent with the consequence to the adolescent of not being allowed to participate in peer activities that may lead to deviance or result in fewer opportunities to develop competencies. Similarly, institutions in the wider social environment, such as church groups and clubs, may assign less independent and challenging tasks to the immature-appearing adolescents.

The relationship between brain development, timing of puberty, and adjustment is now only beginning to be considered. Briefly, the question is whether there is asynchrony between higher order functions like decision making and impulse control and emotional areas of the

brain and hypothalamic functioning and the reactivation of the GnRH axis. Steinberg (in press) suggests an explanation for the association of brain development and risk taking that can be applied to timing of puberty and risk taking as well. As previously discussed, early-maturing adolescents may not have the social decision-making skills and impulse control required to avoid risky situations.

### **Puberty and Obesity**

The higher weight status of early timing adolescents, as previously discussed, has led to the suggestion that the increase in obesity is responsible for the earlier timing of puberty. Adolescents worldwide are now considered overweight or obese, and there is virtually no evidence that the epidemic is subsiding, which is an alarming trend, given the association of obesity with cardiovascular disease and diabetes and other chronic diseases (Dietz, 1998; Katzmarzyk, Tremblay, Pérusse, Després, & Bouchard, 2003). The increase in BMI in the United States in particular may be related to various eating behaviors characterized by high-energy dense foods. For example, in a review by Biro, Khoury, and Morrison (2006), calorie consumption has increased across the last three or so decades with high increases in soft drinks and snacks that are salty and lower intake of milk. Further, overweight adolescents seem to be particularly vulnerable to consuming more in a fast-food setting. With respect to activity, the Biro, Khoury et al. review also provides evidence that physical activity decreases across adolescence, and there is some evidence that the amount of television viewing and computer-related games may have an impact on weight status.

Puberty is a sensitive period for the development of overweight and obesity, since body fat increases secondary to pubertal development (Dietz, 1997). Early sexual maturation is related to obesity and overweight in adolescence and is carried over to the young adult years (Bratberg et al., 2007) and appears to have a heritable intergenerational component (Ong et al., 2007). Environmental factors may

also play a role in weight status during puberty. For example, Belsky et al. (1991) proposed that a stressful family environment, characterized by family conflict, absence of a biological father, poverty, and resultant insufficient parenting, predisposes girls to develop an internalizing disorder that lowers metabolism, thereby inciting weight gain that accelerates the timing of menarche. Overall, the psychological consequences of weight status are only beginning to be addressed, but it is increasingly clear that another negative consequence of early puberty is overweight and obesity.

### **Puberty and Bone Health**

In this section we provide an example of the importance that puberty can have on accrual of bone mineral density (BMD) or bone mineral content (BMC) and, in turn, how depression may play a role in bone health. Over 40% of BMD is accrued in adolescence, with the majority of accrual occurring in the two years surrounding menarche (Glastre et al., 1990). During these two years, as much bone is laid down as is lost in the last four decades of life (Bailey, Mirwald, McKay, & Faulkner, 2000). Thus, building an adequate store of bone mineral is crucial during this period to decrease the chance of becoming osteoporotic in the post-menopausal years. Optimum accrual depends on one's genetic and familial background, but lifestyle and behavioral factors such as exercise, nutrition, and smoking also influence accrual. Timing of puberty has been shown to have an impact on bone accrual. Later pubertal timing (later menarche) girls had lower BMD (Galuska & Sowers, 1999). The explanation is that later maturing girls have less estrogen exposure, and estrogen produces strong bones. In brief, a later maturer has fewer ovulations and menstrual cycles and in turn, less lifetime estrogen exposure.

Where does depression fit into the puberty-bone density model? In the adult literature, there is generally a strong association between depression and BMD. Individuals with depression or who report depressive symptoms are

more likely to have osteoporosis or lower BMD (Eskandari et al., 2007; Jacka et al., 2005; Schweiger, Weber, Deuschle, & Heuser, 2000; Yazici, Akinci, Sütçü, & Ozçakar, 2003; Michelson et al., 1996). It is not known if this relationship holds true in adolescents. If one considers that rates of depression become higher in girls during puberty (Costello, Mustillo, Erkanli, Keeler, & Angold, 2003) and that early maturers are more vulnerable to depression (Hayward et al., 1997; Kaltiala-Heino, Marttunen, Rantanen, & Rimpela, 2003), early timing of puberty may also contribute to bone density. Recognized is that other factors may influence both bone health and depression during puberty, and these include smoking and change in exercise and eating habits.

### Puberty and Sleep

The media frequently describes findings on the sequelae of lack of sleep in adolescents. Factors responsible for this lack of sleep are lack of parental monitoring, peer interactions and influences, pervasive use of television and communication media (cell phones and text messaging), music devices, and Internet surfing. Sleep characteristics of adolescence began to receive systematic attention in the last two decades. Carskadon, Acebo, & Jenni (2004) have produced seminal work showing the systematic changes that occur in the sleep patterns of adolescents. This research showed that there is a major shift in sleep characteristics during the pubertal period. The total amount of sleep decreases, and there is an increase in daytime sleepiness (Carskadon & Acebo, 2002; Dahl & Lewin, 2002). There is also a major shift in time of onset of sleep and time of awakening, referred to as phase delay. Phase delay is related to stage of puberty independent of age, suggesting that the biological changes of puberty have links to changes in sleep patterns (Carskadon, Vieira, & Acebo, 1993; see also Carskadon et al., 1997; Carskadon, Wolfson, Acebo, Tzischinsky, & Seifer, 1998; Carskadon & Acebo, 2002). The preference

for later onset and offset of sleep and societal demands for early school attendance results in an asynchrony in the biological and social needs of adolescents.

Changes in sleep quality and duration during puberty can be justified based on biological and social changes that occur at puberty. First, sleep is associated with the same hormones involved with endocrine changes of puberty (Knutson, 2005). For example, growth hormone-releasing hormone that is produced in abundance during puberty appears to be important in the regulation of sleep (Steiger & Holsboer, 1997). Ghrelin improves slow-wave sleep, which is the deeper stages of non-rapid eye movement (REM) sleep (Steiger, 2003). Sleep deprivation resulted in a stronger increase of slow-wave activity in Tanner 5 (39% above baseline) than in Tanner 1-2 adolescents (18% above baseline). Additional findings show that the buildup of homeostatic sleep pressure during wakefulness was slower in Tanner 5 adolescents compared with Tanner Stage 1-2 children (8.9 +/- 1.2 hours) (Jenni, Achermann, & Carskadon, 2005). The social changes of adolescence, such as increasing interaction with peers, have not been examined in relation to the changes in sleep quality and duration. An unfortunate side effect of lower duration of sleep is its association with obesity. Shorter sleep duration in third and sixth grade was independently associated with obesity at sixth grade (Lumeng et al., 2007).

A contributing factor to sleep problems during adolescence is morningness/eveningness (M/E) preference. M/E refers to a preference for activities in the morning or evening. Support was found for the hypothesis that trait circadian preference mediates mood, daytime functioning, and academic grades through its effect on sleep variables at school time. It was concluded that whereas the imposition of school schedules negatively impacted mood and daytime functioning for the sample as a whole, evening-oriented adolescents were the most vulnerable to poorer outcomes (Warner, Murray, & Meyer, in press). Eveningness students obtained poorer quality and less sleep

than morning-oriented students and were more likely to exhibit antisocial behavior (Warner et al., in press; Susman et al., 2007). The evening-preference adolescent gets less sleep and, as a result, has difficulty concentrating and controlling impulses.

### **What Do We Know for Sure About Puberty and Development?**

It is a given that puberty is an essential stage of reproductive development that has challenged scholars and scientists, communities and teachers, as well as parents and adolescents themselves. The degree to which adolescents experience mood and behavior changes during puberty varies considerably. Across time, much has become known about both the biological and psychological changes that occur at puberty. First, in the past several decades, discoveries have documented the neuroendocrine processes of puberty due primarily to technological advances in molecular biology and other life sciences, hormone assay development, and strong empirical studies in lower animal models, as well as in studies of humans. Second, in the psychosocial literature, studies are relatively consistent in pointing to early pubertal development as being behaviorally and psychologically problematic, primarily for girls. Such studies have generally focused on negative outcomes related to mood disturbances and risky behaviors and have used timing of puberty variables in associative or predictive models. Third, the measurement of pubertal progression has been examined in detail so that investigators now have access to assessments of the appropriateness of different measures of pubertal development, depending on the research question. Fourth, research on the psychological effects of pubertal development has become based on more complex theoretical and statistical models than in the past. These complex models are characterized by multiple levels of analysis that combine "protein to population" levels. Recent work on identification of genes and proteins that are responsible for the onset of puberty represent

the most basic level of analysis. At the population level, the ADD Health study has produced an abundance of information on sexual, psychological, and social influences on the U.S. population of adolescents. In addition, contextual variables are now being examined as mediators or moderators that may impact associations of biological pubertal processes on psychological outcomes. These multiple-level studies offer a richness of detail that has never before been available to scientists of adolescent development.

### **What We Would Like to Know in the Future and Some Conclusions**

Although the knowledge regarding puberty and its role in psychological development has certainly expanded, much is left to be uncovered. Puberty's "effects on health and wellbeing are considered profound and paradoxical" (Patton & Viner, 2007, p. 1130). Thus, the topic warrants further attention as a way of improving psychological and physical health and developmental outcomes in youth. Some of the gaps in the literature are as follows:

1. There is an asynchrony between medical and psychological research with regard to biological and psychological issues inherent in the consequences of pubertal status and pubertal timing. However, the medical research community has identified the significant impact of altered timing of puberty (e.g., precocious puberty and premature adrenarche) on current health or later adult health problems (Golub et al., 2008; see review by Dorn, 2007). For instance, there is now a growing recognition of the association between timing of puberty and adult disease, particularly reproductive cancers. There is also growing recognition in the medical community that the downward secular trend in timing of puberty may precipitate psychosocial and health problems by compromising growth, increasing the risk of early and

risky sexual activity, potential sexual abuse, and inappropriate expectation by others (Golub et al.). What we would like to know is whether these early psychosocial problems have adult psychological sequelae. As a beginning, basic knowledge on the mechanisms involved in early or late timing of puberty and psychosocial functioning is needed. As an additional issue, there is a continuing need for health policy that continues surveillance of human adolescents to define and refine the mechanisms of altered timing of puberty. For instance, screening for exposure to endocrine disruptors is a promising trend for the future. There is also asynchrony between the psychological and medical community in the precision of measuring puberty; its measurement has often lacked rigor in psychological studies. Whether puberty is a cause, correlate, or confound of the outcomes of interest, it can play a leading role in study outcomes and therefore deserves scrutiny in both the medical and psychological community.

2. Health and developmental problems of adolescence are complex and require an interdisciplinary focus. The roots of psychological and behavioral problems stem from, or contribute to, brain-behavior interactions, family dysfunction, reproduction and physical morphological changes, and peers. These inherent complex features of puberty mandate that research scientists are obligated to collaborate across disciplinary lines. The richness and expertise of each discipline in terms of methods, measures, and theoretical and empirical-statistical models could ultimately lead to an empirically based body of research to enhance prevention and intervention efforts. An area of inquiry with rich interdisciplinary attention is that of brain development. Cognitive functioning research from a neurobiological perspective appears to be an area already beginning to develop. Such functioning as

assessed by neuropsychologists and functional and structural brain imaging neuroscientists and neurologists, in combination with behavioral scientists, holds much promise for understanding adolescent behavior. A challenge for the scientific community is to garner funding support for such valuable interdisciplinary efforts.

3. Longitudinal studies with rigorous methodology for measuring puberty as well as other biological and psychological parameters are an additional necessary step in solving complex research problems encountered in pubertal-age adolescents. Several sources have commented on appropriate measures and methodologies for studies of pubertal development (Dorn et al., 2006; Euling et al., 2008). Future longitudinal studies will yield the most payoff by beginning to assess the biological and behavioral roots of pubertal processes by beginning at an early age so as to capture pubertal onset and the range of pubertal changes through adrenarche and gonadarche. Needed in these longitudinal assessments are better biomarkers of onset and progression of puberty, such as genomic and hormone assay studies. A collateral consideration is that to understand the role of puberty on psychological development, longer term perspectives are essential. We do not know for certain whether the links between internalizing and externalizing behavior are a result of puberty per se or whether they represent continuity from earlier phases of development.
4. Fourth, mechanisms impacting puberty and, in turn, its effect on psychological development need further examination. For example, we know little about why pubertal timing is early or late and whether timing is a consequence of genetics, environmental factors, other influences, or a combination of these processes. In turn, we do not know why early pubertal timing is generally negative for girls but positive or neutral for boys. Nor do we know why timing of

puberty seems to have less of an impact on boys than on girls, at least in the psychosocial arena. Importantly, there is a very small amount of knowledge on the positive effects of puberty on development. The majority of studies to date have focused on negative outcomes of puberty with a minority focused on positive youth development. For some adolescents, puberty is a positive sign of emerging adolescence, adulthood, independence, and respect. For others, puberty and its rapid physical growth may enhance self-esteem and vigor and be a protective influence for depression and anxiety.

5. Much more research needs to be carried out regarding structure and function of brain development across puberty. Since most studies have measured change only across chronological age rather than pubertal stage, appropriately little can be said about neuroendocrine changes and brain development as they relate to the external manifestations of puberty. Briefly, we need to know if pubertal status and pubertal timing are related to brain structure and function and, in turn, how those changes may or may not be related to emotions and cognitions as well as behavioral outcomes.

A concluding note is not complete without mentioning the current state of prevention and intervention to reduce problems of youth and increase positive youth development. The current state of knowledge regarding puberty and timing of puberty offers insights into prevention and intervention efforts. One issue is that most parents and school systems have not caught up with the importance of earlier timing of puberty in the health and development of adolescents. As previously indicated, earlier timing, especially for girls, is associated with and predictive of internalizing and externalizing problems, interactions with older and perhaps deviant peers, and early and risky sexual behavior. Thus, earlier maturing adolescents require earlier risk education efforts than their on-time peers on issues of

normal sexual development, risky behaviors, relationships, and Internet safety. Parents, the educational system, and health care providers also are encouraged to undertake earlier health promotion and disease prevention efforts with the aim of assisting adolescents to cope with peer expectations, early sexual development, and expectations for mature behavior.

It also may be advantageous to approach adolescents in a more creative manner than in educational programs that currently are available. For instance, adolescents are likely to be interested in new information about brain development during puberty and adolescence, especially if it is presented via the Internet. Specifically, adolescents should be assured that decision making will be difficult in some instances as brain structures and functions are still developing and adult decisions may be beyond their grasp. Adolescents need to be provided with the assurance that asking others for help is healthy and expected. Within this neurobiological approach, an aim could be to familiarize the adolescent with the awareness that since brains are still developing, they may be highly vulnerable to insults (e.g., negative effect of substance use) that have the potential for affecting academic performance and emotions later in life. In brief, an adolescent-friendly approach that emphasizes the developing brain and decision making is warranted. The effort to mount such an approach is daunting to consider but offers a novel approach to adolescent health promotion.

Finally, scientists, clinicians, and policy makers have not had an established venue for sharing new scientific information that is relevant to pubertal neurobiological processes and the psychological, health, and societal implications of timing of puberty and its wide-ranging impact on development. Similarly, funding agencies tend not to adopt an interdisciplinary approach to basic science, health promotion, or social policy regarding puberty and adolescence more broadly. Conversations and formal organizational structures to guarantee cross-discipline and interagency collaboration is most

assuredly guaranteed to improve the health and development of adolescents and their families in the next decade.

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