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Timing of Pubertal Maturation in Girls: An Integrated Life History Approach

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Life history theory provides a metatheoretical framework for the study of pubertal timing from an evolutionary-developmental perspective. The current article reviews 5 middle-level theories—energetics theory, stress-suppression theory, psychosocial acceleration theory, paternal investment theory, and child development theory—each of which applies the basic assumptions of life history theory to the question of environmental influences on timing of puberty in girls. These theories converge in their conceptualization of pubertal timing as responsive to ecological conditions but diverge in their conceptualization of (a) the nature, extent, and direction of environmental influences and (b) the effects of pubertal timing on other reproductive variables. Competing hypotheses derived from the 5 perspectives are evaluated. An extension of W. T. Boyce and B. J. Ellis's (in press) theory of stress reactivity is proposed to account for both inhibiting and accelerating effects of psychosocial stress on timing of pubertal development. This review highlights the multiplicity of (often unrecognized) perspectives guiding research, raises challenges to virtually all of these, and presents an alternative framework in an effort to move research forward in this arena of multidisciplinary inquiry.

Pubertal maturation is a dynamic biological process—punctuated by visible changes in stature, body composition, and secondary sexual characteristics—that culminates in the transition from the pre-reproductive to the reproductive phase of the human life cycle. The timing of this transition is variable and has substantial social and biological implications. An extensive body of research in Western societies now indicates that early pubertal maturation in girls is associated with a variety of negative health and psychosocial outcomes. In particular, early-maturing girls are at greater risk later in life for unhealthy weight gain (e.g., Adair & Gordon-Larsen, 2001; Wellens et al., 1992), breast cancer (e.g., Kelsey, Gammon, & John, 1993; Sellers et al., 1992), and a variety of other cancers of the reproductive system (e.g., Marshall et al., 1998; McPherson, Sellers, Potter, Bostick, & Folsom, 1996; Wu et al., 1988); have higher rates of teenage pregnancy, spontaneous abortion and stillbirths, and low-birth weight babies (reviewed below); and tend to show more disturbances in body image, to report more emotional problems such as depression and anxiety, and to engage in more problem behaviors such as aggression and substance abuse (e.g., Caspi & Moffitt, 1991; Dick, Rose, Viken, & Kaprio, 2000; Ge, Conger, & Elder, 1996; Graber, Lewinsohn, Seeley, & Brooks-Gunn, 1997). Given this sobering array of outcomes, it is critical to understand the life experiences and pathways that place girls at increased risk for early pubertal maturation.

Life history theory (Charnov, 1993; Roff, 1992; Stearns, 1992) provides a metatheoretical framework for the study of timing of pubertal maturation from an evolutionary-developmental perspective. It attempts to explain the timing of reproductive development and events across the life span in terms of evolved strategies for distributing metabolic resources between the competing demands of growth, maintenance, and reproduction. Life history theory constitutes a set of widely held basic assumptions that have shaped how evolutionary scientists generate and test middle-level theories of pubertal timing. In the current article, I review five middle-level theories—energetics theory, stress-suppression theory, psychosocial acceleration theory, paternal investment theory, and child development theory—each of which applies the basic assumptions of life history theory to the question of environmental influences on timing of pubertal maturation in girls. These middle-level theories are consistent with and subsumed by life history theory but in most cases have not been directly deduced from it (i.e., the middle-level theories are mostly inductions rather than deductions from the metatheory). Each middle-level theory reviewed in this article provides a different translation of the higher-order principles of life history theory into specific hypotheses and predictions that are tested in research. The current review demonstrates how these theories compete to achieve the best operationalization of the core logic of life history theory as it applies to variation in pubertal timing (see Ketelaar & Ellis, 2000, for further discussion of metatheoretical research programs).

The first section below discusses neurophysiological processes underlying pubertal development, defines pubertal timing, and reviews how it is measured. The second section discusses sources of variation in pubertal timing and critically reviews behavior genetic work in this area. Both genotypic and environmental sources of variation in pubertal timing are important and in need of explanation. The third section provides an overview of life history theory and its application to pubertal timing. The fourth and fifth sections review energetics theory (e.g., Ellison, 2001) and stress-suppression theory (e.g., Cameron, 1997; MacDonald, 1999), which posit that adverse physical or social conditions, whether

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experienced as chronically low energy availability or psychosocial stress, cause animals in *K*-selected species to delay pubertal development and reproduction until predictably better times. These theories have proved useful in explaining the effects of physical stress on pubertal timing but have had limited success in generalizing to the psychosocial domain. Energetics theory and stress-suppression theory are then contrasted with psychosocial acceleration theory (e.g., Belsky, Steinberg, & Draper, 1991; Chisholm, 1999) and paternal investment theory (e.g., Draper & Harpending, 1982; B. J. Ellis, McFadyen-Ketchum, Dodge, Pettit, & Bates, 1999) in the sixth and seventh sections. Psychosocial acceleration theory posits that girls whose experiences in and around their families of origin are characterized by relatively high levels of socioemotional stress will develop in a manner that speeds rates of pubertal maturation. Paternal investment theory parallels this logic but posits a special role for fathers and other men in regulation of girls' sexual development. These theories have been reasonably successful in accounting for psychosocial influences on pubertal timing. Mechanistic explanations for the observed stress–puberty relations are reviewed. An extension of Boyce and Ellis's (in press) theory of stress reactivity is proposed to account for both inhibiting and accelerating effects of psychosocial stress on timing of pubertal development.

Despite their predictive utility, psychosocial acceleration theory and paternal investment theory have faced a number of criticisms. In the final section I propose a revision of these theories—child development theory—that addresses most, but not all, of these criticisms. This new framework suggests that psychosocial acceleration and paternal investment theories have reached too far in conceptualizing pubertal timing as a link in the causal chain connecting childhood experiences, not only to age at onset of sex and reproduction, but also to qualitative differences in reproductive strategies such as pairbond stability and parental investment. Child development theory reconceptualizes pubertal timing as the endpoint of a developmental strategy that conditionally alters the length of childhood in response to the composition and quality of family environments (capitalizing on the benefits of high-quality family environments and mitigating the costs of low-quality ones).

For both theoretical and empirical reasons, the focus of this article is on girls' rather than boys' sexual development. First, at a theoretical level, the life history approach to pubertal timing pivots around the trade-off between allocation of resources to physical growth versus production of offspring. Because this trade-off is particularly relevant to females (given their direct somatic investment in production and nurturing of offspring), life history theory has been applied more broadly and successfully to the question of female rather than male pubertal timing. Second, at an empirical level, there is a clear and easily assessed marker of female but not male pubertal timing: age at menarche. Consequently, vastly more research has been conducted on timing of pubertal development in females than in males. A review of antecedents of male pubertal timing is not feasible at this time, given the current state of theory and data.

Pubertal Development and Its Measurement

Timing and tempo of pubertal development are regulated by the functional maturation of the adrenal glands (adrenarche) and the hypothalamic–pituitary–gonadal (HPG) axis (gonadarche). Adrenarche and gonadarche, which are largely independent processes, are responsible for increased secretion of sex steroids during the peripubertal and pubertal periods. Adrenarche has been described as the awakening of the adrenal glands, and it occurs at approximately 6 to 8 years of age in both boys and girls (Dorn & Chrousos, 1997; Grumbach & Styne, 2003). Adrenarche represents a distinct time in adrenal development when levels of the adrenal androgen dehydroepiandrosterone (DHEA) and its sulfate (DHEAS) begin to rise (Dorn & Chrousos, 1997; Grumbach & Styne, 2003). DHEA and DHEAS are produced by the zona reticularis in the adrenal cortex. Between the ages of approximately 5 and 7 years, children experience a sharp drop in levels of 3β-hydroxysteriod dehydrogenase (3βHSD) in the inner reticularis zone (Gell et al., 1998). Although specific control of adrenal androgens is not fully understood, this 3βHSD-deficiency contributes to the increased production of DHEA and DHEAS that occurs during adrenarche (Gell et al., 1998). The development of pubic hair, increased skeletal maturation, increased oil on the skin, changes in external genitalia in males, and body odor are all thought to represent physiological manifestations of increased concentrations of adrenal androgens (Dorn & Chrousos, 1997; McClintock & Herdt, 1996). Adrenarche is the starting point of an upward trajectory in adrenal androgens that plateaus at about age 20; thus, adrenarche and gonadarche are temporally overlapping processes.

Gonadarche occurs at approximately 9 or 10 years of age in girls and soon thereafter in boys (Dorn, Hitt, & Rotenstein, 1999; Grumbach & Styne, 2003), although actual ages vary widely across and within populations, and there is substantial controversy in the pediatric literature over age cut-offs for determining precocious puberty (e.g., Midyett, Moore, & Jacobson, 2003). Gonadarche begins with the reactivation of pulsatile secretion of gonadotropin-releasing hormone (GnRH) after a period of relative quiescence during childhood. GnRH is produced by neurons in the hypothalamus and causes the anterior pituitary to synthesize and secrete biologically potent gonadotropins: luteinizing hormone (LH) and follicle-stimulating hormone (FSH). At gonadarche, pulsatile secretion of LH and FSH markedly increases, causing a cascade of events—ovarian follicular development, increased production of ovarian steroid hormones, development of secondary sexual characteristics, peak height velocity, menarche, subcutaneous fat deposition, widening of the pelvis, and ultimately establishment of cyclic ovarian function—that culminate in maturity of the female reproductive system (see Cameron, 1990; Grumbach & Styne, 2003; and Plant & Barker-Gibb, 2004, for overviews of the neurophysiology of puberty).

Pubertal timing is an individual-differences variable that refers to levels of physical and sexual development of adolescents compared with their same-age peers. The large majority of studies reviewed in this article used a single indicator of pubertal timing: age at menarche. Menarche occurs late in the maturation of the HPG axis (in the United States, the mean age at menarche is 12.9 years [$SD = 1.2$] in Whites and 12.2 years [$SD = 1.2$] in African Americans; Herman-Giddens et al., 1997). Because many of the physical and hormonal changes associated with adrenarche and gonadarche occur prior to menarche, attainment of menarcheal status indicates that a girl has achieved an advanced level of pubertal development. Both adolescent girls and adult women are generally willing and able to report accurately on their ages at menarche, although inaccurate reports are sometimes obtained from young adolescent girls, and retrospective reports may be

more reliable than those obtained during puberty (reviewed in Graber, Petersen, & Brooks-Gunn, 1996; see also Dorn, Nottelmann, et al., 1999). Test–retest reliability has been established in several long-term prospective studies in which self-reported age at menarche was first obtained in adolescence and then again 17 to 37 years later. Correlations across these two measurement periods have been consistently high, ranging from .67 to .79 (Casey et al., 1991; Damon, Damon, Reed, & Valadian, 1969; Livson & McNeill, 1962; Must et al., 2002).

In addition to age at menarche, age-adjusted development of secondary sexual characteristics provides an index of pubertal timing. Development of secondary sexual characteristics is influenced by both adrenal and gonadal processes. Whereas adrenal androgens cause the appearance of sexual hair (pubarche), the effects of ovarian estrogens on dormant breast tissue causes breast budding (thelarche). In Western populations, both pubarche and thelarche typically occur around 10 to 11 years of age (Grumbach & Styne, 2003), although about two thirds of girls experience thelarche before pubarche (Biro et al., 2003). Given the different neuroendocrine pathways through puberty, it is not surprising that the timing of different pubertal indicators are only moderately correlated (*r*s range from .49 to .67; Qamra, Mehta, & Deodhar, 1990). Nonetheless, these indicators are often composited to form overall measures of pubertal development. The Pubertal Development Scale (Petersen, Crockett, Richards, & Boxer, 1988), for example, combines self-ratings of body hair development, growth spurt, skin changes, breast development, and menarcheal status. To assess pubertal timing, a small number of studies reviewed in the current article used either age-adjusted pediatrician ratings of Tanner stages (e.g., Galler, Ramsey, & Solimano, 1985; Qamra et al., 1990) or age-adjusted scores on the Pubertal Development Scale (B. J. Ellis & Garber, 2000; B. J. Ellis et al., 1999). Although physician ratings are generally considered the gold standard for assessment of secondary sexual characteristics and have been found to have better predictive validity than self-ratings (Dorn, Susman, & Ponirakis, 2003), correlations between Pubertal Development Scale scores and ratings by health care professionals have been moderate to high, ranging in value from .61 to .91 (Brooks-Gunn, Warren, Rosso, & Gargiulo, 1987; Dorn, Susman, Nottelmann, Inoff-Germain, & Chrousos, 1990). In sum, like self-reports of age at menarche, self-ratings of pubertal development have demonstrated acceptable validity.

Sources of Variation in Pubertal Timing

Individual differences in the timing of pubertal development are influenced by both genes and environment. Behavior genetic modeling has been used to partition sources of variance in pubertal timing into genetic and environmental components. Large behavior genetic studies using twin designs in Australia, Great Britain, Finland, Norway, and the United States have converged on the conclusion that genotypic effects account for 50%–80% of the variation in menarcheal timing and that the remaining variance is attributable to nonshared environmental effects and measurement error (Golden, 1981; Kaprio et al., 1995; Rowe, 2002; S. A. Treloar & Martin, 1990; van den Akker, Stein, Neale, & Murray, 1987).[1] Complementing these behavior genetic analyses are recent molecular genetic investigations that have begun to identify allelic variation associated with timing of development of secondary sexual characteristics (Kadlubar et al., 2003) and age at menarche (e.g., Comings, Muhleman, Johnson, & MacMurray, 2002; Stavrou, Zois, Ioannidis, & Tsatsoulis, 2002), although specific genetic determinants are still largely unknown. Some researchers have interpreted the absence of shared environmental effects in behavior genetic studies as evidence that the shared experiences of siblings does not increase similarity in pubertal timing (see Bailey, Kirk, Zhu, Dunne, & Martin, 2000; Comings et al., 2002; Rowe, 2000a, 2000b). Given the apparent absence of shared environmental effects, one might ask whether evolutionary models specifying psychosocial influences on pubertal timing are necessarily wrong.

I contend that the answer to this question is "no," for several reasons. First, heritability is a population statistic that indexes the degree to which individual differences in genes account for individual differences in an observed trait in a given environmental context. This definition must be kept in mind when using data from modern postindustrial societies to evaluate evolutionary theories, such as that of Belsky et al. (1991), concerning causes of individual differences in timing of puberty. From the perspective of evolutionary biology, the physiological mechanisms that control pubertal timing were designed by natural selection to take as input the range of physical and social conditions that were recurrently present in ancestral environments. Evolutionarily novel environments may provide inputs that are outside of this range, altering the normal operation of these mechanisms. In discussing sources of variation in pubertal timing, the authors of the Finnish Twin Cohort Study acknowledged that there may have been substantial environmental effects on timing of puberty a generation ago, but not today: "Finnish children born in the 1970s have lived their whole lifetime in a prosperous welfare state, and we can expect that in these cohorts environmental effects are minimized and genetic effects are large" (Kaprio et al., 1995, p. 740). Contemporary Western environments, in which some of the most relevant sources of environmental variation are often squeezed out, provide incomplete contexts for testing evolutionary models of pubertal timing.[2] The absence of shared environmental effects in this context does not imply that humans lack evolved psychobiological mechanisms that detect and encode information from the environment as a basis for adaptively calibrating timing of pubertal development.

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[1] It is important to note that only the U.S. (Rowe, 2000a) and Finnish (Kaprio et al., 1995) studies assessed menarcheal age during adolescence. In contrast to the heritability data on age at menarche, subsequent analyses on Finnish twin cohorts yielded approximately equal heritability (.40) and shared environmentality (.45) estimates for overall levels of pubertal development in 12-year-old girls (as indexed by the Pubertal Development Scale). By age 14, however, estimated heritability increased to .70 and shared environmentality decreased to .02 (Dick, Rose, Pulkkinen, & Kaprio, 2001).

[2] Indeed, all correlations with pubertal timing in contemporary Western societies are likely to be attenuated because of the reduction in variance in pubertal timing caused by the secular trend toward earlier pubertal development. Wellens, Malina, Beunen, and Lefevre (1990) provided data on the secular trend in age at menarche in Flemish girls in the 20th century. For girls born between 1915 and 1929, the average age of menarche was 14.41 years, and the average interval between the time when 10% and 90% of girls attained menarche was 4.12 years. Those numbers dropped to 13.09 years and 2.91 years, respectively, for girls born between 1960 and 1971. Hwang, Shin, Frongillo, Shin, and Jo (2003) reported similar data for South Korean girls.

Second, both Kaprio et al. (1995) and S. A. Treloar and Martin (1990) found that at least half of the genetic variance in age of menarche was nonadditive (i.e., genetic variance that does not cause parents and offspring to be more similar). Nonadditive genetic variance (whether detected or undetected) inflates heritability and deflates shared environmentality estimates in standard twin designs because shared environments and nonadditive genes have opposite effects on twin correlations (see Grayson, 1989). Because nonadditivity tends to obscure any possible shared environmental variance, J. M. Meyer, Eaves, Heath, and Martin (1991) suggested that alternatives to traditional twin designs are needed to detect effects of shared environment on menarcheal timing.

Third, alternative methods have produced clear evidence of shared environmental influence on age of menarche. Farber (1981) reported that monozygotic twins reared together were most similar in menarcheal age (average difference = 2.8 months), followed by monozygotic twins reared apart (average difference = 9.3 months), followed by dizygotic twins reared together (average difference = 12.0 months). That monozygotic twins reared apart were most similar in menarcheal timing to dizygotic twins reared together suggests that individual differences in age of menarche are influenced by the degree to which girls share common environments (as well as common genes). It should be noted, however, that Farber's study was very small and thus may have produced unreliable estimates. Further evidence of shared environmental influence is provided by comparisons of mother–daughter dyads with sister–sister dyads, of which both members share about 50% more of their genes in common than do two randomly selected members of a population. From a genetic perspective, intrapair correlations in age at menarche should be equivalent for mother–daughter and sister–sister dyads. Sister–sister correlations, however, are consistently higher than mother–daughter correlations (reviewed in Malina, Ryan, & Bonci, 1994, Tables 3 and 4; sister–sister correlations: $M = .39$, range = .25 to .61; mother–daughter correlations: $M = .27$, range = .15 to .40), which suggests that sharing the same home during ontogeny increases similarity in menarcheal timing.

Fourth, the types of environmental influences posited by psychosocial models of pubertal timing are likely to have a nonshared component because their effects are not equivalent across siblings in the same home. It is important to distinguish in this context between *objective* and *effective* environments (Goldsmith, 1993; Turkheimer & Waldron, 2000). "Objective environments refer to environmental events as they might be observed by a researcher, as opposed to how they affect family members" (Turkheimer & Waldron, 2000, p. 79). Environmental variables that extend across more than one sibling, such as socioeconomic status (SES) or marital quality, are objectively shared, regardless of whether these variables operate to make siblings more or less alike. Environmental variables that are unique to each sibling, such as birth order or peer relationships, are objectively nonshared. By contrast, "effective environments are defined by the outcomes they produce" (Turkheimer & Waldron, 2000, p. 79). Behavior genetic models incorporate only effective environmental influences. Thus, to the extent that objectively shared environmental variables have different effects on different siblings, these effects are defined as nonshared and allocated to the nonshared component of environmental variance in behavior genetic models. Objectively shared experiences may have nonshared effects because of genetic differences between siblings (e.g., the strength of relations between childhood abuse and the frequency of antisocial behavior in young adulthood differs significantly depending on the form of a genotypic marker of monoamine oxidase; Caspi et al., 2002; see also Caspi et al., 2003). Objectively shared experiences may also be effectively nonshared because of age differences between siblings (e.g., father absence has different effects on daughters' sexual behavior depending on the daughter's age when the father leaves the home; B. J. Ellis et al., 2003). Finally, to the extent that objectively nonshared environmental variables influence development, these influences are also nonshared. For example, parent–child processes vary substantially across siblings (e.g., Geary & Flinn, 2001; Sulloway, 1996) and thus contribute to the nonshared component of variance in children's developmental outcomes. In sum, consistent with behavior genetic models, major environmental influences posited by psychosocial models of timing of pubertal development (e.g., Belsky et al., 1991; B. J. Ellis & Garber, 2000; Surbey, 1990) are likely to have substantial nonshared effects on siblings.

Fifth, heritability estimates are context specific and can change dramatically when social or physical environments change (e.g., Dunne et al., 1997; Rowe, Jacobson, & Van den Oord, 1999; Turkheimer, Haley, Waldron, D'Onofrio, & Gottesman, 2003). Comparison of correlations across multiple levels of kinship pairs—cousins, half-siblings, full siblings, mother–daughter pairs, identical twins—is a common method for estimating genetic influences on menarcheal age and typically yields heritabilities in the range of .45 to .55 (Chern, Gatewood, & Anderson, 1980; Doughty & Rodgers, 2000; Rowe, 2000a). These heritability estimates, however, may be inflated by environmental continuity between members of kinship pairs. Consider Chasiotis, Scheffer, Restmeier, and Keller's (1998) investigation of mother–daughter correlations in age of menarche in comparable urban areas in East and West Germany. This study spanned the time period of reunification (which resulted in much greater social disruption and sociopolitical change for East Germans than for West Germans). In the East German sample, there was no significant correlation between mothers and daughters in either resource availability (e.g., SES) in childhood ($r = -.04$) or age at menarche ($r = -.07$). By contrast, in the West German sample, there were substantial correlations between mothers and daughters in both resource availability in childhood ($r = .51$) and age at menarche ($r = .60$). Consistent with these data, low mother–daughter correlations for age at menarche (rs $\leq .20$) were also recorded in a Czech Republic study in which mothers and daughters differed in having grown up in rural versus urban environments (Hajn & Komenda, 1985). These findings provide fuel for critics of twin research, who have argued that the range of environmental variation between members of twin pairs (whether raised in the same home or adopted into comparable ones; see Stoolmiller, 1999) consistently underestimates the range of environmental variation in the larger society. As Segalowitz (1999) suggested,

> The thought experiment of separating twins at birth to widely different settings—for example, one to urban New York, the other to rural Sahara; one to an affluent home in London, the other to a poor family in the third world—illustrates how heritability is artificially raised by restrictions of environmental variance. (p. 905)

Finally, as reviewed in this article, it has been well-documented that the timing of pubertal maturation in girls is sensitive to a variety of external factors, such as exercise, nutrition, and socio-

emotional stress. Indeed, the median menarcheal age varies across human populations from about 12.0 years in some urban postindustrial societies to 18.5 years in rural highland Papua New Guinea or high altitude Nepali groups (Worthman, 1999). This enormous variation underscores the evolved capacity of humans to adjust timing of sexual maturation to local physical and social conditions.

In conclusion, although it is beyond dispute that genotypic effects on timing of pubertal development are substantial, twin designs do not allow one to confidently estimate levels of heritability or environmentality in age at menarche. Evolutionary and behavior genetic models converge on the importance of nonshared environmental influences on pubertal timing. Nonetheless, considerable caution must be exercised when evaluating evolutionary models of pubertal timing solely on the basis of data from modern, postindustrial societies with a restricted range of relevant environmental variance. Finally, the theory and data reviewed above suggest that genotypic effects on timing of pubertal development are probabilistic and are best conceptualized as coding for a "reaction norm." That is, genotypes are capable of producing a range of phenotypic expressions, and actual timing of puberty is an emergent property of the genotype and the environment in which it occurs. This reaction norm perspective (see especially Stearns & Koella, 1986) potentially reconciles behavior genetic and psychosocial models of variation in pubertal timing.

The Life History Approach to Timing of Pubertal Development

The key units of analysis in life history theory (Charnov, 1993; Roff, 1992; Stearns, 1992) are life history traits: the suite of maturational and reproductive characteristics that define the life course (e.g., age at weaning, age at sexual maturity, adult body size, time to first reproduction, interbirth interval, litter size). Life history theory attempts to explain variation in life history traits in terms of evolved trade-offs in distribution of metabolic resources to competing life functions: growth, maintenance, and reproduction. These trade-offs are inevitable because metabolic resources are finite, and time and energy used for one purpose cannot be used for another. For example, resources spent on growth and development (e.g., later age at sexual maturity, larger adult body size, increased social quality and competitiveness) cannot be spent on current production of offspring; thus, the benefits of a prolonged childhood are traded off against the costs of delayed reproduction. Life history theory posits the existence of phenotypic mechanisms that actually make these trade-offs by selecting between or "making decisions" about alternative ways of distributing resources (Chisholm, 1999). Natural selection favors mechanisms that, in response to ecological conditions, trade off resources between growth, maintenance, and reproduction in ways that recurrently enhanced inclusive fitness during a species' evolutionary history.

There are two fundamental trade-offs that are central to life history theory: the trade-off between current and future reproduction and the trade-off between number and fitness of offspring. The fitness costs and benefits associated with variations in timing of reproductive development illustrate these trade-offs. These variations are indexed by such integral life history traits as timing of sexual maturation and time to first reproduction.

All else being equal, natural selection favors earlier reproductive development over later reproductive development for three reasons. First, because the probability of mortality is always greater than zero over any given time period, earlier onset of reproduction is associated with lower probability of mortality prior to reproduction. Fitness benefits of early reproductive development should be especially relevant under conditions in which life expectancies are low or highly variable (Chisholm, 1999). Second, early reproductive development increases the total reproductive output of lineages through shorter generation times. Third, because age at menarche and age at menopause are largely uncorrelated in humans (e.g., Borgerhoff Mulder, 1989b; Peccei, 2000; Snieder, MacGregor, & Spector, 1998; A. E. Treloar, 1974), earlier menarcheal age results in longer reproductive life spans. These selection pressures favoring early reproductive development are opposed by competing selection pressures favoring later reproductive development. Animals with longer periods of growth and development attain larger adult body size, which generally translates into lower adult mortality rates, greater energy production and stores to devote to reproduction over the life course, and increased success in intrasexual competition (Charnov, 1993; Hill & Kaplan, 1999).

Although these relations apply across species, they are also relevant to understanding variation in expression of life history traits within species (e.g., Hill & Kaplan, 1999; Stearns, 1992; Stearns & Koella, 1986). For example, a fitness cost of early reproductive development in humans is that it may divert resources away from growth before skeletal maturation has been completed and constrain metabolic resources available for production and nurturing of offspring (see especially Allal, Sear, Prentice, & Mace, 2004). Specifically, adolescent mothers ordinarily lack adult pelvic capacity (Moerman, 1982); tend to be smaller and convert less of their weight gain during pregnancy to fetal weight gain than do adult mothers (Garn, Pesick, & Petzold, 1986); experience higher rates of antenatal complications and mortality than do adult mothers; and their offspring are at increased risk of stillbirths, congenital abnormalities, prematurity, low birth weight, and retardation (Black & DeBlassie, 1985; Furstenberg, Brooks-Gunn, & Chase-Lansdale, 1989; Luster & Mittelstaedt, 1993).[3]

Conversely, a benefit of longer reproductive development is that older mothers have more time to acquire cognitive, survival, mate selection, and parenting skills prior to becoming parents (Bogin, 1999; Lancaster, 1986; Surbey, 1998). This is evidenced by lower rates of single motherhood, lower rates of divorce, higher educational and economic outcomes, and more competent parenting among adult mothers than adolescent mothers (Black & DeBlassie, 1985; Furstenberg et al., 1989; Luster & Mittelstaedt, 1993). Most relevant, the children of adult mothers tend to have better cogni-

[3] Timing of reproductive maturation varies across different racial groups in the United States. Because African Americans tend to experience earlier pubertal development than Whites do (Herman-Giddens et al., 1997) and are thus more gynecologically mature as teenagers, African American teenage mothers may not experience the same adverse health outcomes as do White teenage mothers. Geronimus, Korenman, and Hillemeier (1994) found that White teenage mothers, on average, experienced the highest levels of low birth weight babies and infant deaths, whereas African American teenage mothers, 15- to 19-years-old, experienced lower rates of these adverse outcomes than did African American mothers in their twenties. Konner and Shostak (1986) suggested that the special medical risks of adolescent childbearing are due more to improper prenatal nutrition and postnatal care than to reproductive immaturity, especially if adolescent mothers are at least 17 years old.

tive, behavioral, social developmental, and survival outcomes than do the children of adolescent mothers (Black & DeBlassie, 1985; Brooks-Gunn & Furstenberg, 1986; Konner & Shostak, 1986; Overpeck, Brenner, Trumble, Trifiletti, & Berendes, 1998). The greater competence and reproductive efficiency of older mothers has been documented in a wide range of mammalian species (Promislow & Harvey, 1990). In total, early reproductive development tends to bias individuals toward short-term (current) reproduction and greater number of offspring, whereas later reproductive development tends to bias individuals toward long-term (future) reproduction and greater fitness of offspring.

Given the mix of fitness costs and benefits associated with different timing of reproductive development in humans, selection should *not* favor phenotypic mechanisms that systematically bias intraspecific variation toward either current or future reproduction or greater number or fitness of offspring. Rather, consistent with the reaction norm perspective discussed above, selection can be expected to favor adaptive developmental plasticity of mechanisms (within genetic capacities and constraints) in response to particular ecological conditions (Belsky et al., 1991; Boyce & Ellis, in press; Chisholm, 1996; Ellison, 2001).[4] Indeed, many, if not most, organisms are capable of altering their life histories in response to their environment (H. S. Kaplan & Lancaster, 2003). Thus, from a life history perspective, phenotypic mechanisms should be engineered to monitor evolutionarily relevant features of one's environment as a basis for contingently allocating resources to survival, growth, development, and reproduction. These resources should be allocated in nonrandom ways that, during a species' evolutionary history, recurrently optimized trade-offs between current and future reproduction and number and fitness of offspring (see Chisholm, 1996, 1999).

A central question in life history theory is, When should individuals reach sexual maturity? That is, when should individuals stop converting surplus energy into growth and begin converting it into reproduction? And most critically, What are the relevant developmental experiences and environmental cues that bias individuals toward relatively early versus late reproductive development? Competing answers to this question have been proposed by the different middle-level life history theories reviewed in this article, as discussed below.

All of these middle-level theories (a) link variation in pubertal timing to individual differences in experiences of stress and (b) emphasize childhood exposure to recurrent, ongoing stressors. Consistent with this theorizing, the term *stress* is used herein to denote an ongoing condition that requires coping and that, over time, undermines efficient functioning by draining internal (physiological) or external resources; the term *stressor* is used, as it so commonly is in investigations cited throughout this review, to denote ongoing circumstances or events that cause stress. Accordingly, as highlighted by the various middle-level theories, a broad range of ongoing circumstances and events are referred to as stressors (e.g., nutritional deprivation, intensive physical exercise, poverty, low social rank, warfare, parental psychopathology, parental absence, residence in a stepfamily, marital discord, harsh parental discipline, absence of familial warmth, stressful life events). When stressors are primarily psychosocial, the resulting condition is referred to as *psychosocial stress* or *socioemotional stress*. Conversely, when stressors are primarily physical, the resulting condition is referred to as *physical stress* or *energetic stress*.

The Energetics Theory of Timing of Pubertal Development

Drawing on life history theory, various evolutionary biologists and psychologists (e.g., MacDonald, 1999; E. M. Miller, 1994; Surbey, 1998) have argued that in *K*-selected species (those characterized by high-investment/low-fertility reproductive strategies, such as humans) there should be a negative correlation between resource scarcity and speed of sexual maturation. These theorists posit that members of the human species, under conditions of chronically low energy availability, are primed to delay maturation and reproductive viability until predictably better times (see also Wasser & Barash, 1983). The core argument is that natural selection has favored physiological mechanisms that track variation in resource availability and adjust physical development to match that variation. Consistently good conditions in early and middle childhood signal to the individual that accelerated development and early reproduction are sustainable. Conversely, conditions of resource scarcity cause the individual to reserve energy for maintenance and survival (rather than growth or reproduction). As Ellison (2001) suggested,

> The adjustment of growth trajectories to chronic ecological conditions is an example of developmental plasticity that is itself assumed to be adaptive. An individual growing up under conditions of chronically low energy availability may be better off growing slowly and being smaller as an adult. Slower growth will divert less energy from maintenance functions. Smaller adult size will also result in lower average metabolic rate and lower maintenance costs. (pp. 133–134)

This theory, linking chronic resource availability to timing of pubertal development, is henceforth referred to as *energetics theory*.

Energetics theory yields the core hypothesis (Hypothesis 1) that children who experience chronically poor nutritional environments will grow slowly, experience late pubertal development, and achieve relatively small adult size, whereas children who experience chronically rich nutritional environments will grow quickly, experience early pubertal development (relative to their genetic potential), and achieve relatively large adult size. Food availability is critical because surplus metabolic energy—the extent to which energy production exceeds maintenance costs—can be harvested by animals and converted into growth and reproduction. The greater the surplus, the greater the capacity for both growth and reproduction. According to energetics theory, earlier maturing girls have more surplus energy. Indeed, Ellison (1990) posited that timing of pubertal maturation serves as a kind of bioassay of the chronic qualities of the environment, particularly energy availability, encountered during childhood. According to Ellison (1990, 1996, 2001), girls use this bioassay to establish a lifetime set point for baseline levels of adult ovarian function and reproductive effort, as evidenced by substantial integrity in ovarian function across the reproductive life span. In total, girls who experience earlier sexual development are in better physiological condition

[4] Although selection can be expected to favor adaptive developmental plasticity, this does not imply that all individuals are equally plastic. As reviewed by Belsky (2004) and Boyce and Ellis (in press), some individuals are more susceptible to rearing influences than others. This issue is addressed in greater detail below (see Criticisms of Psychosocial Acceleration and Paternal Investment Theories).

and have more metabolic resources to devote to reproduction. A second hypothesis (Hypothesis 2) derived from energetics theory, therefore, is that girls who experience relatively early sexual maturation have greater reproductive capacity than their later maturing peers (see also Udry, 1979; Voland, 1998). That is, they have greater biological capacity to produce viable offspring.

Evaluation of Energetics Theory: Hypothesis 1

SES. The hypothesis that greater energy availability will be associated with earlier timing of puberty has been indirectly tested in the great number of studies examining relations between SES and pubertal timing. The large majority of these investigations have used age at menarche as their index of pubertal timing, but some have also assessed development of secondary sexual characteristics. In societies in which there are substantial differences between social classes in nutritional and health status, girls from higher social classes experience earlier pubertal development than do girls from lower social classes (e.g., Ghana: Adadevoh, Agble, Hobbs, & Elkins, 1989; Sudan: Abioye-Kuteyi et al., 1997; Nigeria: Oduntan, Ayeni, & Kale, 1976; Mozambique: Padez, 2003; Iran: Ayatollahi, Dowlatabadi, & Ayatollahi, 2002; Egypt: Attallah, 1978; Israel: Belmaker, 1982; Morocco: Montero, Bernis, Loukid, Hilali, & Baali, 1999; Bangladesh: Foster, Menken, Chowdhury, & Trussell, 1986; Philippines: Adair, 2001; India: Chakravartti & Renuka, 1970; China: Wang & Murphy, 2002; Haiti: Allman, 1982; Brazil: Linhares, Round, & Jones, 1986; Venezuela: Lopez Contreras, Tovar Escobar, Farid Coupal, Landaeta Jimenez, & Mendez Castellano, 1981). These data are consistent with the secular trend (beginning at least 170 years ago in England) toward earlier onset of pubertal development, as well as faster tempo of pubertal development (de Muinck Keizer-Schrama & Mul, 2001; Worthman, 1999), in association with general improvements in health and nutrition accompanying modernization (Tanner, 1990). Specifically, age of menarche in Europe dropped from approximately 17 to 13 years of age between 1830 and 1960 (Eveleth & Tanner, 1990). The secular trend has been most intense within lower SES groups (Abioye-Kuteyi et al., 1997; Brudevoll, Liestol, & Walloe, 1979; Prado, 1984; Singh & Malhotra, 1988; Veronesi & Gueresi, 1994), where living conditions have improved most dramatically over time. Effects of SES on girls' pubertal timing are generally absent, however, in countries where lower SES groups do not suffer from systematic malnutrition and disease (e.g., Britain: Douglas & Simpson, 1964; Canada: Surbey, 1990; Denmark: Helm & Lidegaard, 1989; Germany: Merzenich, Boeing, & Wahrendorf, 1993; Greece: Petridou et al., 1996; Italy: Veronesi & Geuresi, 1994; New Zealand: Moffitt, Caspi, Belsky, & Silva, 1992; Portugal: Padez, 2003; Spain: Sanchez-Andres, 1997; Sweden: Lindgren, 1976; United States: B. J. Ellis et al., 1999; Wales: Roberts & Dann, 1975). Because many factors covary with SES—health care, hygiene, caloric intake, dietary composition, energy expenditure, exposure to artificial lighting, family functioning, frequency of divorce and remarriage, and so on—it is difficult to isolate the specific factors responsible for the observed relations between SES and pubertal timing. Consequently, the foregoing data are consistent with, but do not confirm, the hypothesized causal relation between nutritional status and timing of puberty.

Nutritional status. The hypothesis that greater food availability and concomitant surplus metabolic energy accelerates pubertal maturation has been tested in many studies. Rather than measuring surplus metabolic energy directly, these investigations have assessed energy intake and other indicators of nutritional status and examined their relations with pubertal timing. This method is imperfect for evaluating the current hypothesis, however, because it does not control for physical activity, which at increasing levels is associated with later puberty (e.g., Merzenich et al., 1993; Petridou et al., 1996; Warren, 1983). Nonetheless, human and animal research has produced a fairly coherent picture of the relations between nutrition and pubertal timing.

Experimental studies of the effects of nutrition on the speed or timing of pubertal development in animals have generally manipulated energy intake, protein intake, or both. In a review of the animal literature (based on rats, pigs, and cattle), Kirkwood, Cumming, and Aherne (1987) concluded that undernutrition can cause delays in pubertal development, but only under conditions of severe dietary restriction. As reviewed below, the human literature is largely consistent with this conclusion: Nutritional deprivation causes delays in onset of puberty, but variations in the quality and quantity of diets within adequately nourished populations have little effect.

A number of long-term prospective studies in developing countries have assessed caloric intake and other indicators of nutritional status in early or middle childhood and examined their subsequent relations with pubertal timing. Adair (2001) conducted multiple 24-hr dietary recalls on a cohort of 966 premenarcheal 8-year-old Filipino girls. Khan and colleagues (Khan, Schroeder, Martorell, Haas, & Rivera, 1996; Khan, Schroeder, Martorell, & Rivera, 1995) conducted approximately ten 24-hr dietary recalls on a sample of 250 Guatemalan girls whose home diet was repeatedly assessed between the ages of 15 and 84 months. Qamra, Mehta, and Deodhar (1990, 1991) conducted multiple 24-hr dietary recalls on a sample of 791 Indian girls aged 5 to 16 years. Galler et al. (1985) examined 216 Barbadian children, half of whom had histories of moderate to severe protein-energy malnutrition in their first year of life. Satyanarayana and Naidu (1979) studied a sample of 739 rural Hyderabad girls, 27% of whom were classified as suffering from severe chronic undernutrition during pre-school life, based on height and weight measurements at age 5. Finally, Frisch (1972) analyzed extensive childhood medical and nutritional data, beginning from ages 4–5 years, on 30 undernourished and 30 well-nourished girls from Alabama (see also Dreizen, Spirakis, & Stone, 1967). All of these investigations included girls with a broad range of dietary histories, ranging from sustained nutritional deprivation to fully adequate nutrition. In each of these studies, girls who were either malnourished or consumed fewer calories during childhood than their well-nourished peers experienced later puberty.[5] All of these research projects included timing of menarche as a downstream dependent variable. The Barbadian and Indian studies also included pediatricians' ratings of the de-

[5] Adair (2001) reported that higher total energy intake was not related to age at menarche. However, this analysis controlled for both SES and body mass index. If either SES or body mass index were left out of the equation, then the diet variables significantly predicted age at menarche in the expected direction (L. S. Adair, personal communication, September 17, 2003). Galler et al. (1985) also reported that the association between nutritional history and timing of pubertal development was substantially reduced by controlling for weight and height, suggesting that decreased growth may mediate relations between nutritional deprivation and delayed puberty (see also Moisan et al., 1990b).

velopment of secondary sexual characteristics. Taken together, these investigations provide convincing evidence that nutritional deprivation delays pubertal development.

The relations between nutritional status and pubertal timing has led some researchers to search for underlying endocrine mechanisms. A small number of studies have examined relations between nutritional status and plasma gonadotropin levels in preadolescent or adolescent girls. In an investigation of prepubertal Indian girls aged 6 to 10 years, Sreedhar, Ghosh, and Chakravarty (1983) found relatively low circulating levels of LH and FSH in individuals with severe histories of protein-energy malnutrition. Similarly, in a comparison between privileged Nairobi girls and rural Kenyan girls who experienced moderate malnutrition during childhood, Kulin, Bwibo, Mutie, and Santner (1984) found reduced levels of LH and FSH in the rural sample across the age range from 9 to 12 years. Finally, in a Dutch sample, de Ridder et al. (1991) found that 12-year-old girls with high intakes of dietary grain fiber had significantly lower plasma concentrations of LH and FSH. These data suggest that delayed puberty in nutritionally deprived girls may result from low circulating levels of pituitary gonadotropins.

A number of longitudinal investigations in North American and Western European countries have also examined relations between nutritional status and subsequent timing of puberty. These studies have assessed overall caloric intake as well as calorie-adjusted levels of specific dietary nutrients (e.g., fat, protein, carbohydrates, fiber). In these well-nourished populations, neither variations in overall caloric intake nor calorie-adjusted consumption of specific dietary nutrients consistently predicts timing of pubertal development (Berkey, Gardner, Frazier, & Colditz, 2000; de Ridder et al., 1991; Koo, Rohan, Jain, McLaughlin, & Corey, 2002; Koprowski, Ross, Mack, Henderson, & Bernstein, 1999; Maclure, Travis, Willett, & MacMahon, 1991; Merzenich et al., 1993; F. Meyer, Moisan, Marcoux, & Bouchard, 1990; Moisan, Meyer, & Gingras, 1990a, 1990b). The one exception was a reliable association between diets high in calorie-adjusted dietary fiber or foods high in fiber content (e.g., vegetarian diet) and later age at menarche (de Ridder et al., 1991; Kissinger & Sanchez, 1987; Koo et al., 2002; Soriguer et al., 1995). And in a remarkable international comparison of 46 countries and communities, R. E. Hughes and Jones (1985) found a very strong positive correlation ($r = .84$) between per capita intake of dietary fiber (g/1,000 kcal) and later age at menarche. They suggested an evolutionary explanation for this relation:

> It is possible that the fibre-fertility link is in fact an evolutionary adaptation and represents a protective mechanism to delay reproduction on non-optimal diets until the mother has attained an acceptable stage of physical development. Young mothers, if still growing and developing, could well compete with the foetus in certain critical areas for essential nutrients such as protein. Diets that are low in protein are frequently high in fibre; this is particularly true when the protein is of a low-quality vegetable type. A high intake of dietary fibre would, in such circumstances, delay the menarche and so reduce the possibility of foetal-maternal competition for the inadequate amount of available dietary protein. (R. E. Hughes & Jones, 1985, pp. 330–331)

In sum, the data support Hypothesis 1: Children who experience chronically poor nutritional environments, whether assessed indirectly through SES or directly in dietary studies, tend to experience relatively late pubertal development. A relevant intervening endocrine mechanism may be low levels of pituitary gonadotropins. The necessary conditions for delayed puberty, however, appear to be serious or sustained nutritional deprivation; the level of dietary variation found in modern Western societies does not appear to meet these conditions (with the exception of high-fiber diets).

Evaluation of Energetics Theory: Hypothesis 2

The second hypothesis derived from energetics theory is that earlier maturing girls have greater reproductive capacity. To evaluate this reproductive capacity hypothesis, it is useful to decompose *reproductive capacity* into more specific, measurable indicators: ovarian function (e.g., growth and maturation of follicles, production of ovarian steroid hormones); fecundity (the probability of becoming pregnant when reproductively cycling and exposed to sexual intercourse); fertility (number of offspring); lactational capacity; rates of spontaneous abortions, stillbirths, congenital abnormalities, prematurity, low birth weight, and retardation in offspring; and health and well-being of children. According to the reproductive capacity hypothesis, earlier maturing girls should have higher ovarian functioning, higher fecundity, higher fertility, greater lactational capacity, better pregnancy outcomes (i.e., lower rates of spontaneous abortions, stillbirths, congenital abnormalities, prematurity, low birth weight, and retardation in offspring), and greater fitness of offspring. As reviewed below, there are reasonably well-developed literatures on the relations between age of menarche and ovarian functioning, fetal wastage (spontaneous abortions and stillbirths), fetal growth, and fecundity and fertility.

Ovarian functioning. Ellison (1990, 1996) has proposed that adult levels of ovarian hormonal functioning—the endocrine function of the ovaries in producing steroid hormones—are related to the timing of childhood and adolescent growth and reproductive maturation. Ellison (1990, 1996) specifically hypothesized that earlier reproductive maturation is associated with a faster rise in indices of ovarian function with age and higher levels of ovarian steroid secretion in adulthood. As reviewed by Ellison (1996), individual differences in ovarian hormonal functioning influence variation in female fecundity through such intervening mechanisms as follicular development, endometrial proliferation, production of progesterone receptors, fertilizability of the oocyte, success of implantation, and maintenance of ongoing pregnancies.

An 18-year longitudinal investigation by Apter and Vihko (1983; Vihko & Apter, 1984; Apter, Reinila, & Vihko, 1989; Apter, 1996), which began with 200 Finnish schoolgirls, 7–17 years of age, and followed a subsample of them into their twenties and thirties, has provided the primary base of support for the ovarian function hypothesis. Among the main research results are three important findings. First, early in pubertal development prior to menarche, those girls who would subsequently experience menarche before age 13 had earlier and greater increases in FSH and estradiol concentrations than their peers who would experience menarche at age 13 or later.

Second, early menarche was associated with early onset of ovulatory menstrual cycles;[6] for example, the time from menarche

[6] First menstrual cycles are often infertile (anovulatory), and the time between first menstruation and attainment of fertile (ovulatory) menstrual cycles varies across individuals.

until 50% of cycles were ovulatory was approximately 1.0 year if menarche occurred before age 12, 3.0 years if menarche occurred during age 12, and 4.5 years if menarcheal age was 13 or older. This finding replicated work by MacMahon et al. (1982), who studied the probability of ovulation in relation to age at menarche in 15 to 19 year old girls in several countries. MacMahon et al. found that girls whose menarche occurred at under 12, 12, 13, and 14+ years of age had cycles that were 21%, 30%, 36%, and 44% anovular, respectively.

Third, the higher levels of serum estradiol and lower sex hormone binding globulin concentrations found in earlier maturing girls, compared with their later maturing peers, remained at 20–30 years of age (see also Kirchengast & Hartmann, 1994, who reported similar results in a study of adult Austrian women). In sum, these endocrine studies suggest that variation in menarcheal age is associated with meaningful individual-differences timing of ovarian maturation and in set points for regulation of the hypothalamic–pituitary–ovarian axis in the prime reproductive years. Ellison (1996) suggested that lower baseline levels of ovarian steroids in individuals with later menarche could result from either stably lower levels of pituitary gonadotropin stimulation or stably higher sensitivity of the hypothalamic–pituitary axes to the negative feedback of ovarian steroids.

In addition to this endocrine research, a number of investigators have examined relations between age at menarche and self-reported menstrual cycle characteristics. According to the reproductive capacity hypothesis, earlier age at menarche should be associated with earlier onset of regular menstrual cycles. Only Gardner (1983), in a longitudinal study of 54 American women, has provided support for this hypothesis, reporting that later age of menarche was associated with less regularity of menstrual functioning at age 18 ($r = -.31$). Given the age of the sample, however, the correlation could be an artifact of immaturity of the hypothalamic–pituitary–ovarian axis in the later developing girls. Another American longitudinal study found little difference in the length or variance of menstrual cycles for women whose menarche ranged from ages 10 to 14 but did find that women whose menarche occurred at ages 15 to 16 had relatively long and variable cycles (Wallace, Sherman, Bean, Leeper, & Treloar, 1978). Similarly, in a large retrospective investigation of a French cohort, the percentage of individuals with an interval of 5 years or more between age at menarche and age at onset of regular menses did not differ for women whose menarche ranged from ages 11 to 15, but it was almost twice as high for women whose menarche occurred at age 17 or later (Clavel-Chapelon & the E3N-EPIC Group, 2002). Finally, several retrospective American studies have found no substantive relations between age at menarche and time until regular cycling (Butler et al., 2000; Garland et al., 1998; Rockhill, Moorman, & Newman, 1998). In sum, although positive associations have been found between earlier age of menarche and higher levels of ovarian hormonal functioning, this enhanced functioning does not appear to translate into shorter latencies to regular menstrual cycling.

Fetal wastage. A second prediction derived from the reproductive capacity hypothesis is that earlier maturing females should be more likely than later maturing females to have successful pregnancies that culminate in live birth. In evaluating this prediction, it is important to control for levels of biological maturity because earlier maturing females may be at increased risk of spontaneous abortion and stillbirth because they are more likely to become pregnant as adolescents. An extensive literature exists on the relation between age at menarche and fetal wastage. Several different research methodologies have been used to test the prediction that earlier maturing females have less fetal wastage. These include case-control methodologies, prospective pregnancy-based studies, and cross-sectional studies. Case-control methodologies (al-Ansary, Oni, & Babay, 1995; Parazzini et al., 1991, 1997; Prado, 1990) generally have compared patients admitted to a hospital for spontaneous abortion with controls at the same hospital having normal deliveries, controlling for chronological age. Prospective pregnancy-based studies have followed samples of women longitudinally until they became pregnant and the outcome of their pregnancy was determined (i.e., spontaneous abortion, stillbirth, or live birth; Mayaux, Spira, & Schwartz, 1983; Sandler, Wilcox, & Horney, 1984). These investigations have either used samples of adult (postadolescent) females (Mayaux et al., 1983) or have controlled for age at conception in the analyses (Sandler et al., 1984). In cross-sectional studies, large samples of women of varying ages have provided retrospective information on age at menarche and the outcome of specific pregnancies. Several of these projects examined the effect of menarcheal age on risk of spontaneous abortion in first pregnancy, controlling for age at first pregnancy (Bracken, Bryce-Buchanan, Stilten, & Holford, 1985; Casagrande, Pike, & Henderson, 1982; Martin, Brinton, & Hoover, 1983). Wyshak (1983) examined the relation between age at menarche and number of unsuccessful pregnancy outcomes, controlling for total number of pregnancies and age at first birth. Finally, a number of other cross-sectional investigations tested for associations between menarcheal age and miscarriage rates without controlling for biological maturity (Helm & Lidegaard, 1989; Helm, Munster, & Schmidt, 1995; Liestol, 1980; Madrigal, 1991; Varea, Bernis, & Elizondo, 1993), making their results difficult to interpret. All of this research excluded cases in which pregnancies were terminated by induced abortion.

Across the 16 empirical studies reported in 15 research articles cited above, only al-Ansary et al.'s (1995) research on Saudi women provided support for the prediction that earlier maturing women have more successful pregnancy outcomes. The other investigations either directly contradicted the prediction by showing that early age at menarche was associated with increased (rather then decreased) risk of miscarriage (Casagrande et al., 1982; Helm et al., 1995; Liestol, 1980; Madrigal, 1991; Martin et al., 1983; Parazzini et al., 1991; Prado, 1990; Varea et al., 1993; Wyshak, 1983) or found no relation between age at menarche and pregnancy outcomes (Bracken et al., 1985; Helm & Lidegaard, 1989; Mayaux et al., 1983; Parazzini et al., 1997; Sandler et al., 1984). Nonetheless, 2 of the inquiries showing no relations found that early-maturing females were overrepresented in cases of recurrent spontaneous abortion (Bracken et al., 1985; Sandler et al., 1984). Finally, several studies found curvilinear relations between age at menarche and rates of miscarriage, with both early and late menarcheal age associated with elevated risk (Martin et al., 1983; Prado, 1990; Varea et al., 1993; Wyshak, 1983). In sum, a considerable body of research using diverse methodologies clearly rejects the prediction that early reproductive development increases the odds of having successful pregnancies that culminate in live birth.

Fetal growth. A third prediction of the reproductive capacity hypothesis is that earlier maturing mothers should be more successful than later maturing mothers in promoting fetal growth.

Specifically, earlier maturing females should be more likely to have term deliveries and to produce offspring that achieve normal birth weight (both of which are major predictors of infant health and survival; see, e.g., de Courcy-Wheeler et al., 1995; McCormick, 1985). Again, it is important in evaluating this prediction to control for levels of biological maturity in the mother. There have been several investigations of the relation between maternal age at menarche and indices of fetal growth. Hennesey and Alberman (1998), in their prospective research on first births among members of the British Birth Cohort, found support for the prediction that earlier maturation in mothers promotes greater fetal growth. Specifically, early age of menarche ($<$ 12 years) was associated with greater birth weight adjusted for gestational age (i.e., faster growth in utero). This effect remained after controlling for such confounding variables as mother's age at birth, height, weight for height, and smoking during pregnancy. The study may not have accurately gauged the relation between menarcheal age and fetal growth, however, because it excluded preterm deliveries, which have been found to occur disproportionately among early-maturing mothers (Berkowitz, 1981; Li & Zhou, 1990; Scholl, Miller, Salmon, Vasilenko, & Johnston, 1987). Nonetheless, some research has provided evidence that is not inconsistent with the prediction. Both DaVanzo, Habicht, and Butz (1984) and Strobino, Ensminger, Kim, and Nanda (1995) found that very late menarche was associated with low birth weight. In both studies, however, no relation was found between maternal age at menarche and birth weight of offspring across the early-to-normal range of menarcheal timing (9–18 years in DaVanzo et al.'s, 1984, study of Malaysian mothers; 9–15 years in Strobino et al.'s, 1995, study of young American mothers).

Similarly, in a population-based Chinese birth cohort, Xu et al. (1997) found that ages at menarche over the median ($\geq$ 15 years) were associated with small-for-gestational-age births, but only among thin mothers (body mass index $\leq$ 21). Because thin girls tend to experience relatively late menarche (e.g., Kaplowitz, Slora, Wasserman, Pedlow, & Herman-Giddens, 2001), it seems likely that the relation between menarche and fetal growth was driven by a small percentage of mothers who, consistent with DaVanzo et al. (1984) and Strobino et al. (1995), had both very late ages at menarche and produced low birth weight offspring. Both Strobino et al. and Xu et al. controlled for mother's age at birth in the analyses.

Other published research on the relation between maternal age at menarche and fetal growth directly contradicts the prediction that early maturation is associated with more fetal growth. Both Berkowitz (1981) and Li and Zhou (1990) conducted hospital-based case-control studies in which mothers delivering preterm infants were compared with mothers delivering term infants. Both investigations found that the mothers delivering preterm infants had significantly earlier age at menarche, although neither investigation controlled for maternal age at birth. Scholl et al. (1987, 1989) examined the effects of age at menarche on preterm delivery, low birth weight, and small-for-gestational-age births in two cohorts of adolescent mothers. Both studies found that earlier age at menarche was associated with intrauterine growth retardation. Scholl et al. (1989) is especially informative in this context because they studied a narrow chronological age band, 17–18 years, and thus earlier maturing mothers would have had greater gynecological age (i.e., longer intervals between menarche and first birth). Despite being more biologically mature, the adolescent mothers with younger menarcheal ages were more likely to deliver growth-retarded infants (Scholl et al., 1989). Finally, in research on nearly 5,000 births at a hospital in Austria, Kirchengast and Hartmann (2000) found that earlier maturing mothers tended to give birth to lighter and smaller babies. The inquiry was limited to term births, included only adult mothers (ages 19–43), and adjusted for maternal age.

In sum, it is difficult to draw strong conclusions about the relations between age at menarche and fetal growth because of methodological limitations of many investigations and because of contradictory results. Nonetheless, there is very little support for the prediction that earlier maturing mothers will be more successful than later maturing mothers in promoting fetal growth.

The growing body of evidence linking early age of menarche to either fetal wastage or fetal growth retardation has led some researchers to attempt to explain these links at a mechanistic level. As discussed above, early maturers tend to have higher circulating levels of estrogen through adulthood than do late maturers. These increased endogenous estrogen levels may increase uterine cramping and bleeding and induce tonic uterine contractions, which could both predispose early maturers to spontaneous abortions and reduce uterine blood flow, resulting in fetal growth retardation and decreased newborn size (Kirchengast & Hartmann, 2000; Scholl et al., 1989).

Fecundity and fertility. A central assumption of the reproductive capacity hypothesis is that earlier maturing females have healthier and more efficient reproductive systems. A derivative prediction is that earlier maturing females should be more fecund than later maturing females. This prediction can be tested only in natural fertility (noncontracepting) populations or in noncontracepting groups within populations. As an index of fecundity in natural fertility populations, several researchers have measured the protogenesic interval—the time elapsed between marriage and first birth—and then examined the association between this interval and age at menarche. Two studies of the protogenesic interval, one among Sudanese women (Otor & Pandey, 1998) and the other among Malaysian women not using contraception (Udry & Cliquet, 1982), have provided measured support for the reproductive capacity hypothesis. In both investigations, early-maturing females (age at menarche $\leq$ 11 in the Malay sample and $\leq$ 12 in the Sudanese sample) had shorter intervals than did later maturing females (age at menarche 15 or above). Neither of these studies, however, reported differences in protogenesic intervals between early maturing and normatively maturing girls.

Other published research on the protogenesic interval directly contradicts the prediction that earlier pubertal maturation will be associated with greater fecundity. In their investigation of Moroccan women, Varea et al. (1993) found no relations between the protogenesic interval and either age at menarche or age at marriage and concluded that fecundability and menarcheal timing were independent. Other studies conducted in rural Bangladesh (Foster et al., 1986; Riley, 1994), among the Ladiya of India (Adak, Gharami, Singhai, & Jain, 2001), among the Gond of India (Sharma & Chowdhury, 1995), among the Kipsigis of Kenya (Borgerhoff Mulder, 1989b), and in a largely noncontracepting population in Romania (Cristescu, 1975) have documented shorter protogenesic intervals among women with later ages at menarche. Although in each of these investigations women who matured earlier also tended to marry earlier than did their later maturing peers, this difference was at least partially recovered by the shorter

protogenesic intervals (i.e., catch-up fecundity) among the later maturing women. It is possible, however, that this catch-up fecundity could have been caused by either longer menarche-to-marriage intervals (resulting in greater gynecological age at marriage) or greater chronological age (i.e., greater biological maturity) among later maturing women. Only the Bangladesh research specifically addressed these alternative explanations: No relations were found between age at marriage and menarche-to-marriage intervals (Riley, 1994, Figure 4). When age of marriage was controlled for, age at menarche was still associated with shorter protogenesic intervals (Riley, 1994).

In addition to protogenesic intervals, a reliable negative indicator of fecundity is infertility problems: either difficulty becoming pregnant or complete infertility. Relations between pubertal timing and infertility can be usefully studied in contracepting populations because women who are trying to become pregnant do not use contraception. Komura, Miyake, Chen, Tanizawa, and Yoshikawa (1992) found no relation between variation in menarcheal timing across the age range of 11 to 17 years and rates of infertility among married Japanese women (all of whom had fertile husbands and had tried to become pregnant). However, rates of infertility increased significantly among women who attained menarche at age 18 or later. Two studies of American women, by contrast, found that very early menarche ($\leq$ 11 years of age) was associated with difficulties in becoming pregnant (Wyshak, 1983) or no live births (Sandler et al., 1984). Sandler et al. also found that late menarche ($\geq$ 15 years of age) increased risk for no live births. Finally, in a large random sample of Dutch women, Helm et al. (1995) found that age of menarche was unrelated to whether women ever became pregnant, ever gave birth, or had difficulties becoming pregnant when desiring to have a child. In sum, most research examining relations between age at menarche and infertility has found that women who experienced pubertal maturation either early or late have more infertility problems than do women whose pubertal maturation occurred within the normative range.

A final method of assessing fecundity is reproductive success. Studies of the relations between timing of pubertal maturation and reproductive success can be legitimately conducted only in natural fertility populations. Because greater number of offspring tends to be associated with higher child mortality rates (Cristescu, 1975; Crognier, 1998; Kunstadter et al., 1992; Strassmann & Gillespie, 2002; Syamala, 2001), the relevant dependent variable in such investigations should be number of surviving offspring, rather than number of live births. Accordingly, research on the links between menarche and fitness needs to assess completed family size in postmenopausal women in traditional societies. This presents formidable measurement problems because retrospective questioning of female elders about age at menarche in nonliterate populations is vulnerable to profound bias and memory lapse (Borgerhoff Mulder, 1989b). One study circumvented this problem by estimating age of menarche in a small sample of postmenopausal Kipsigis women ($N = 33$) through reference to clitoridectomy ceremonies that could be easily dated (Borgerhoff Mulder, 1989b). A meaningful negative correlation was found between estimated menarcheal age and number of currently surviving children ($r = -.53$, $p < .001$), although the relation was not linear. As suggested by the scatterplot shown in Figure 3 of Borgerhoff Mulder (1989b), there was no relation between variation in menarcheal timing across the age range of 12 to 16 years and completed family size. However, women who attained menarche at age 17 or later had notably fewer surviving children. Borgerhoff Mulder (1989a) also reported the correlation between age of menarche and number of live births per year of marriage in a sample of premenopausal Kipsigis women ($N = 80$; $r = -.22$, $p < .05$). As suggested by the scatterplot in Figure 4 of Borgerhoff Mulder (1989a), there was again no relation between variation in menarcheal timing across the age range of 12 to 16 years and number of live births per year; but the data did suggest a small decrease in live births among women who attained menarche at age 17 or later.

In sum, there is no evidence in any of the research reviewed above that women who experience early menarche are more fecund than women who experience menarche in the normative range for their population. Although several studies found that women whose pubertal development occurred in the early-to-normative range were more fecund than women who experienced delayed puberty, a number of other investigations found that later maturing women had shorter protogenesic intervals. Finally, some work suggests that early-maturing women are at elevated risk for infertility. Taken together, these data present a severe challenge to the reproductive capacity hypothesis (Hypothesis 2): There appears to be no reproductive advantage to maturing early over maturing on time (i.e., maturing at a rate that is average for the population).

Summary and Conclusion

Energetics theory suggests that energy availability during childhood influences timing of pubertal maturation, that in fact pubertal timing operates as a bioassay of chronic childhood conditions, and that females use this bioassay to establish lifetime set points for reproductive functioning. Extant data support the first part of this theory (Hypothesis 1) but not the second part (Hypothesis 2). Although girls who experience chronically rich nutritional environments tend to grow more quickly and experience earlier pubertal development than do girls in chronically poor nutritional environments, and although there is evidence that earlier age at menarche is associated with higher levels of ovarian hormonal functioning, there is no consistent evidence that earlier pubertal maturation translates into higher reproductive functioning. Compared with girls whose age at menarche is in the average range for their population, early-maturing girls do not have shorter latencies between menarche and regular menstrual cycling, are not more successful at maintaining pregnancies that culminate in live birth, are not more successful at promoting fetal growth, and are not more fecund or reproductively successful.

The effects of nutrition and SES on pubertal timing provide strong support for a basic assumption of energetics theory: that natural selection has favored physiological mechanisms that track variation in resource availability and adjust timing of physical development to match that variation. Nonetheless, there could be a simpler absence-of-impairment explanation for the data. The absence-of-impairment hypothesis posits that, given sufficiency of resources and absence of biological insults, organisms will achieve their full developmental potential (e.g., fast growth, large body size; Worthman, 1999). The completeness of the absence of impairment hypothesis has been strongly challenged by Worthman (1999), however, who presented several lines of evidence demonstrating that absence of impairment does not influence pubertal timing in a simple linear or unidirectional manner. For example, poor children adopted from developing countries into affluent

Western families experience significantly earlier puberty than do children from either their countries of origin or their host countries, despite histories of infection and malnutrition prior to adoption (reviewed in Mul, Oostdijk, & Drop, 2002). Moreover, Indian and Bangladeshi girls adopted into Swedish families experience earlier menarche if they are adopted at *later* ages (mean age at menarche is 11.1 years and for girls adopted at $\geq$ 3 years of age and 11.9 years for girls adopted at $<$ 3 years of age; Proos, Hofvander, & Tuvemo, 1991), even though the older adoptees experienced more sustained deprivation prior to adoption. Consistent with the adaptationist framework positing physiological mechanisms that track energy availability, Worthman (1999) stated that "the life history model would predict that girls experiencing persistent deprivation would react to a dramatic improvement in environmental quality by hastening reproduction in order to exploit a narrow window of resource availability" (p. 141).

As suggested in the preceding discussion of life history theory, timing of pubertal maturation (whether early or late) represents a trade-off in distribution of metabolic resources toward different potential reproductive strategies. Earlier reproductive development tends to bias individuals toward short-term (current) reproduction and greater number of offspring, whereas later reproductive development tends to bias individuals toward long-term (future) reproduction and greater fitness of offspring. Although earlier pubertal development in girls predicts earlier age at first sexual experience and reproduction (reviewed below, see Psychosocial Models of Pubertal Timing: IV. Child Development Theory), girls whose pubertal development is in the normative range for their population are no less fertile or fecund than their earlier maturing peers. Most important, earlier developing girls may be sacrificing offspring quality, as suggested by the literatures on fetal wastage and fetal growth. The other side of the coin is that later developing girls have more time to build physical and social capital prior to maturity (see especially below, Psychosocial Models of Pubertal Timing: IV. Child Development Theory). In sum, early pubertal development is not a reliable indicator of high reproductive capacity. Rather, consistent with life history theory, it can be conceptualized as an important component of a reproductive strategy that is biased toward current reproduction and offspring number.

Psychosocial Models of Pubertal Timing: I. Stress-Suppression Theory

The energetics theory of pubertal timing, positing that resource scarcity delays pubertal development, has been applied more broadly to encompass psychosocial stressors. According to this expanded version of the theory, adverse physical or social conditions, whether experienced as chronically low energy availability or psychosocial stress, should cause animals in *K*-selected species to delay pubertal development and reproduction until predictably better times (MacDonald, 1999; E. M. Miller, 1994). This theory, linking both physical and social stressors to timing of pubertal development, is henceforth referred to as *stress-suppression theory*.

Stress-suppression theory has been supported by neurophysiological research linking stress to suppression of the HPG axis. Environmental events signaling threats to survival or well-being produce a set of complex, highly orchestrated responses within the neural circuitry of the brain and peripheral neuroendocrine pathways regulating metabolic, immunologic, and other physiological functions. As comprehensively detailed in the writings of neuroscientists such as Chrousos (1998), Meaney (2001), and McEwen (1998), the neural substrates for the organism's stress response comprise two anatomically distinct but functionally integrated circuits: the corticotropin-releasing hormone (CRH) and locus coeruleus-norepinephrine (LC-NE) systems and their peripheral effectors, the pituitary–adrenal axis and the limbs of the autonomic nervous system. The coactivation of the these two systems, along with their linkages to limbic structures, such as the amygdala and anterior cingulate, as well as the mesolimbic dopaminergic system and the medial prefrontal cortex, produce the coordinated biobehavioral changes associated with the stress response in mammalian species. When activation of these stress-response systems is of sufficient duration and magnitude, the functioning of the HPG axis can be suppressed at several levels, including decreased GnRH pulsatility, disrupted GnRH surge secretion, decrease in pituitary responsiveness to GnRH, and alteration of stimulatory effects of gonadotropins on sex steroid production (Cameron, 1997; Dobson, Ghuman, Prabhakar, & Smith, 2003; Johnson, Kamilaris, Chrousos, & Gold, 1992; Rivier & Rivest, 1991; cf. Ferin, 1999, who reviewed primate research indicating a paradoxical increase in gonadotropin in response to stressors during the mid-to-late follicular phase of the menstrual cycle). Linkages between the stress-response systems and the HPG axis thus provide a clearly articulated mechanism through which psychosocial stress could delay pubertal development. In humans, these linkages are supported by a substantial body of research indicating that energetic stress—and some research suggesting that psychosocial stress—can induce reproductive dysfunction in women (e.g., Ellison, 2001; Ferin, 1999; Marcus, Loucks, & Berga, 2001; Nappi & Facchinetti, 2003).

Human and nonhuman primate research investigating the stress-suppression hypothesis, however, has examined the effects of stress on ovarian functioning in mature females. No published experimental work has manipulated psychosocial stress in immature female primates and then followed those animals prospectively to determine downstream effects on timing of pubertal development. To my knowledge, relevant experimental research in large mammals has been conducted only on pigs. This applied agricultural research has assessed the impact of management stressors (i.e., mixing with unfamiliar conspecifics, relocation to new pens, truck transport) on attainment of puberty in gilts. Contrary to the stress-suppression hypothesis, management stressors, either on their own or in combination with boar contact, generally stimulate earlier pubertal development in gilts (see P. E. Hughes, Philip, & Siswadi, 1997, and references therein). Gilts raised in total confinement systems, however, tend to experience delayed puberty (Thompson & Savage, 1978).

Potential effects of psychosocial stress on pubertal timing have also been indirectly investigated in nonhuman primates. A number of researchers have studied primate social groupings, measured the social rank of different group members, and then correlated social rank with timing of pubertal development. The underlying assumption is that low social rank is emotionally and physiologically stressful, and thus low social rank should delay pubertal maturation and impair ovarian hormonal functioning (e.g., Blanchard, McKittrick, & Blanchard, 2001; Cameron, 1997). This theorizing has been supported by a number of primate studies showing a negative correlation between female social rank and age at puberty. As reviewed by French (1997), in a variety of *Callitrichid*

primates, including cotton-top tamarins, saddleback tamarins, red-bellied tamarins, and common marmosets, puberty is delayed or does not occur in subordinate daughters that remain in their natal groups. Further, in an investigation of outdoor-housed rhesus monkeys, higher ranking females experienced earlier age at first ovulation than did lower ranking females, even though lower ranking females spent significantly more time feeding (Schwartz, Wilson, Walker, & Collins, 1985). Finally, in research on free-ranging savanna baboons, Bercovitch and Strum (1993) found that the daughters of high-ranking females had earlier onset of reproductive maturation than did the daughters of low-ranking females, but only when resource availability was taken into account.

These observed relations between social rank and pubertal timing have sometimes been interpreted as supporting a causal role for stress, both social and nutritional, in suppressing reproductive development (e.g., Cameron, 1997; Dunbar, 1988; Hacklander, Mostl, & Arnold, 2003; Schwartz et al., 1985). This interpretation has been strongly challenged by Creel (2001), however, in his review of the literature on the relations between social dominance and glucocorticoid (GC) levels. One of the primary mammalian responses to environmental stressors is enhanced activation of the hypothalamic–pituitary–adrenal (HPA) axis, causing an increase in levels of plasma GCs. Agonistic encounters can cause large and persistent increases in GC levels in both winners and losers, but especially in losers (Creel, 2001). Although agonistic encounters play a role in establishment of dominance hierarchies, it is generally thought that social dominance relations evolve to avoid the costs of escalated conflicts under conditions in which winning and losing can be reliably predicted (Enquist & Leimar, 1990). Once a stable dominance hierarchy has been established, there is no a priori reason to expect that lower social rank will be associated with higher basal GC levels. Creel found that relations between basal GC levels and social rank were highly variable across species, with dominant animals displaying elevated basal GC levels as often as subordinates. Further, in species that lived in permanent social groups, it was uncommon for subordinates to experience chronically elevated GCs. Creel suggested that research is needed to identify the non-GC-mediated mechanisms through which social rank affects sexual development and behavior. One possibility, pheromonal regulation of pubertal timing, is discussed below (see Psychosocial Models of Pubertal Timing: III. Paternal Investment Theory).

The mechanisms question remains controversial. Cameron (1997) has argued that at least part of the social rank effect on reproductive endocrine function results from social interactions that put subordinate animals in more stressful situations. Although Cameron (1997) acknowledged that subordinates do not always show endocrine markers of stress (such as increased basal GC levels), they may still display greater adrenocortical reactivity than more dominant animals (e.g., J. R. Kaplan, Adams, Koritnik, Rose, & Manuck, 1986). Cameron (1997) also reviewed primate evidence indicating that behavioral intimidation by dominant animals plays an important role in reproductive suppression of subordinates. This evidence is largely based on captive populations, however, which differ from wild populations in ways that are directly relevant to experiences of stress (e.g., capture and handling stress, placement of unfamiliar individuals in small enclosures, limited ability of subordinates to avoid dominants or move away from the group). It is important to note that endocrine research on wild populations suggests that it may be as stressful to be dominant as it is to be subordinate (Creel, 2001). In any case, the relations between dominance status and stress are complex and modified by a number of social conditions (e.g., Bercovitch & Ziegler, 2002; Boyce, O'Neill-Wagner, Price, Haines, & Suomi, 1998).

Controlled human research, of course, does not exist. But some relevant information has been obtained by analyzing timing of pubertal development under conditions of war. A number of studies have been conducted in relation to World War II. These investigations examined median ages at menarche in given regions before, during, and after the war. In Europe, the Soviet Union, and Japan, the secular trend toward earlier pubertal development was already well under way by the time the Second World War began. In Belgium (Wellens et al., 1990), Finland (Kantero & Widholm, 1971), France (Olivier & Devigne, 1983), Germany (Tanner, 1962), Japan (Hoel, Wakabayashi, & Pike, 1983), the Netherlands (van Noord & Kaaks, 1991), and Russia (Bielicki, 1986), this trend was reversed during the period of World War II. There can be little doubt that adverse conditions associated with the war delayed pubertal development. This research does not enable determination of the specific conditions that caused this delay, however. There were many confounding stressors—food rationing, changes in dietary composition, increased physical activity, suffering from cold, prevalence of disease, physical injury, as well as psychological trauma—any of which could have plausibly contributed to the temporary reversal in the secular trend.

A more recent investigation of menarcheal timing during the recent war in Yugoslavia has attempted to deconfound some of these factors. Prebeg and Bralic (2000; see also Tahirovic, 1998) studied changes in mean age at menarche of girls in the Croatian town of Sibenik from the mid-1980s to the mid-1990s. Sibenik was exposed to hard war conditions in 1991–1995. Although no measurements were taken of caloric intake (or of energy expenditure), Prebeg and Bralic claimed that there were not notable food shortages during the war and that rates of infectious disease did not increase. Nonetheless, mean menarcheal age increased significantly from 12.87 years in 1985 ($N = 1{,}270$) to 13.13 years in 1996 ($N = 1{,}680$). Among girls whose homes were damaged during the war ($n = 278$), mean menarcheal age was 13.53 years. And among the group of girls who lost a family member ($n = 76$), menarche occurred at an average age of 13.76 years. Prebeg and Bralic (2000) concluded that "the reversal of menarcheal age in Sibenik girls is probably related to the prolonged psychological stress associated with war" (p. 507). These data are consistent with clinical observations of delayed puberty in children who have suffered severe socioemotional stress (i.e., psychosocial dwarfism; reviewed in Hopwood et al., 1990). Nonetheless, most human research on the effects of familial environments on pubertal timing suggests that family adversity is associated with earlier, rather than later, pubertal development (see Psychosocial Models of Pubertal Timing: II. Psychosocial Acceleration Theory below).

In sum, although connections between the stress-response systems and the HPG axis provide a plausible mechanism through which psychosocial stress could delay pubertal development, there is only limited evidence that psychosocial stress actually does delay puberty. The primate data on social rank and pubertal timing provide only weak support at best for the stress-suppression hypothesis. The data on changes in pubertal timing during periods of war are interesting but confounded, though the Yugoslav data (as well as the data on psychosocial dwarfism) suggest that extreme

psychosocial stress can delay puberty. Most critical, as reviewed below, the hypothesis that psychosocial stress delays human pubertal development runs counter to the results of most longitudinal research on this topic. In total, the current empirical literature does not support expanding energetics theory into a more general stress-suppression theory of pubertal timing that encompasses psychosocial stressors. Admittedly, relevant research is scant, often indirect, and mostly nonexperimental. The point is not that these limited investigations disconfirm the hypothesis that psychosocial stress inhibits pubertal development but rather that little research has supported it. Nonetheless, the possibility that moderate psychosocial stress accelerates pubertal development whereas extreme psychosocial stress delays it is explored further in the next section.

Psychosocial Models of Pubertal Timing: II. Psychosocial Acceleration Theory

As discussed above, life history theory comprises a broad set of theoretical principles which can be used to derive a number of more specific theoretical models. In some cases, these derivative models provide competing perspectives on a common question. In contrast to the stress-suppression theory presented above, an alternative set of life history models focuses on the role of familial and ecological stressors in provoking early onset of pubertal development and reproduction (Belsky et al., 1991; Chisholm, 1993, 1996, 1999; Wilson & Daly, 1997).

Belsky et al. (1991) were the first to propose a life history model of the role of psychosocial stressors in accelerating timing of puberty in girls. Indeed, they regarded the proposition of a linkage between psychosocial experiences early in life and pubertal timing as a unique and uncanny prediction distinguishing their evolutionary theory of socialization from more traditional theories of socialization as well as from mainstream thinking about determinants of pubertal timing. Belsky et al. (1991) posited that

> a principal evolutionary function of early experience—the first 5–7 years of life—is to induce in the child an understanding of the availability and predictability of resources (broadly defined) in the environment, of the trustworthiness of others, and of the enduringness of close interpersonal relationships, all of which will affect how the developing person apportions reproductive effort. (p. 650)

Drawing on the concept of sensitive-period learning of reproductive strategies, Belsky et al. (1991) theorized that humans have evolved to be sensitive to specific features of their early childhood environments and that exposure to different environments biases children toward the development of different reproductive strategies. Children whose experiences in and around their families of origin are characterized by relatively high levels of stress (e.g., scarcity or instability of resources, father absence, negative and coercive family relationships, lack of positive and supportive family relationships) are hypothesized to develop in a manner that speeds rates of pubertal maturation, accelerates sexual activity, and orients the individual toward relatively unstable pairbonds and lower levels of parental investment. In contrast, children whose experiences in and around their families are characterized by relatively high levels of support and stability are hypothesized to develop in the opposite manner (Belsky et al., 1991).

In essence, Belsky et al. (1991) proposed that the context of early rearing "sets" the person's reproductive strategy in a way that was likely to have functioned adaptively in that context in the environments in which humans evolved. Over the course of humans' natural selective history, ancestral females growing up in adverse family environments may have reliably increased their reproductive success by accelerating physical maturation and beginning sexual activity and reproduction at a relatively early age, without the expectation that paternal investment in child rearing would be forthcoming and without the precondition of a close, enduring romantic relationship (Belsky et al., 1991). A shortened reproductive timetable in this context may have increased the probability of having at least some offspring that survive and reproduce. As Chisholm (1996) suggested, "When young mammals encounter conditions that are not favorable for survival—i.e., the conditions of environmental risk and uncertainty indexed by emotional stress during development—it will generally be adaptive for them to reproduce early" (p. 21).

Although the stress-suppression theory posits that stress and uncertainty should result in later pubertal development and lower fertility, Chisholm (1996, 1999) proposed that this should be the case only when parents have the capacity to shape conditions in ways that significantly enhance the health, competitiveness, and eventual reproductive success of their offspring. When parents lack this capacity, allocation of resources should be biased toward reproducing early and often. One element of this accelerated reproductive strategy is to shorten the time before sexual maturity (i.e., accelerate pubertal development). As Chisholm (1999) has stated,

> From the perspective of life history theory (and contrary to a great deal of "common sense") when parents' resources are limited it is *not* necessarily adaptive or rational for them to have fewer offspring so as to be able to invest more in each one. In other words, even when mortality rates are not high the optimal strategy for parents who lack the material or social resources (e.g., power, prestige) to *make a difference* in their children's reproductive value (e.g., health, education, employment or marriage prospects, competence as parents. . .) may well be to *increase* fertility (to maximize current reproduction) while reducing investment in each child (which tends to decrease future reproduction). . . . The "non-intuitive message" here (as Monique Borgerhoff Mulder [1992:350] described this apparent paradox) is that when the flow of resources is chronically low or unpredictable—which is when we might otherwise expect parents to be most solicitous of their offspring—it may in fact be (or have been) evolutionarily adaptive for parents to "hedge their bets" against lineage extinction by *reducing* parental investment and allocating their limited resources not to parenting effort (or even, beyond some threshold, to their *own* health and longevity), but to *offspring production* instead. (pp. 57–58)

In sum, low-quality parental investment may signal an environment in which variations in parental care and resources are not closely linked to variation in reproductive success. Under these conditions the developing child should accelerate reproductive maturation. This theory, linking psychosocial stress to earlier puberty, is henceforth referred to as *psychosocial acceleration theory*.

Empirical Investigations of the Relations Between Psychosocial Stress and Timing of Pubertal Maturation in Girls

As reviewed by Blanchard et al. (2001) and Pacak and Palkovits (2001), there are stressor-specific neuroendocrine pathways and

circuits within the central nervous system, and different types of stressful events have qualitatively different effects on both physiology and behavior. For example, in research on rats, socioemotional stressors (e.g., repeated social defeat) and physical stressors (e.g., electric footshock) have been found to produce opposites effects on systolic blood pressure and mean arterial blood pressure (Adams, Lins, & Blizard, 1987). Following B. J. Ellis and Garber (2000), I distinguish between three general types of environmental stressors that have been found to covary with girls' pubertal timing: physical stressors (e.g., malnutrition, physical exercise stress), socioemotional stressors (e.g., harsh and neglecting family relationships, absence of parental warmth and support), and father absence. This distinction is important because it decomposes a multiplicity of experiences which Belsky et al. (1991) and Chisholm (1999) did not explicitly treat as distinctive in their developmental consequences when theorizing about early experience and reproductive strategies (including pubertal timing).

Physical stressors. There is general agreement in the literature that physical stressors tend to delay pubertal timing. The relation between low caloric intake and delayed pubertal timing was reviewed above (see The Energetics Theory of Timing of Pubertal Development). There is also substantial evidence that physical exercise stress, such as intensive running or dancing, delays pubertal maturation (e.g., Brooks-Gunn & Warren, 1988; Georgopoulos et al., 1999; Lounana, Bantsimba, Silou, Packa-Tchissambou, & Medelli, 2002; cf. Malina, 1998, who argued that sports-specific selective factors may channel late-maturing girls into many forms of athletics).

Studies of physical stress are conceptually distinct from studies of socioemotional stress, given that individuals in a physically rich environments can still have substantial exposure to socioemotional stressors and vice versa. Hulanicka and colleagues (Hulanicka, 1999; Hulanicka, Gronkiewicz, & Koniarek, 2001) have specifically compared the effects of physical and socioemotional stressors on timing of pubertal development in Polish school girls. Consistent with energetics theory, the Polish data show a strong main effect of poverty on pubertal timing: Poorer girls mature later. At the same time, however, girls growing up in dysfunctional families in which they were exposed to prolonged distress (e.g., father absence, parental alcohol abuse, prolonged illness of a parent) had significantly earlier ages at menarche than did girls who lived in families that were free of strong traumatic events—despite the lower SES and nutritional status of girls from dysfunctional families (Hulanicka, 1999; Hulanicka et al., 2001). These data suggest that physical and socioemotional stressors may have independent, and perhaps countervailing, effects on timing of pubertal development. In light of such evidence, it is not surprising that the alternative theories under consideration in this article have been advanced.

Socioemotional stressors. There is considerable controversy regarding the role of socioemotional stressors and father absence in regulation of pubertal timing. Extant research on this topic has assessed overall quality of family relationships during childhood as well as the father's role in the family (e.g., father absence, father involvement) more specifically. The possibility that fathers play a special role in regulation of daughters' pubertal timing is reviewed in the next section (Psychosocial Models of Pubertal Timing: III. Paternal Investment Theory).

When evaluating the possible impact of family relationships on pubertal timing, it is important to note that causation may be bidirectional (see especially Steinberg, 1988). Both adrenarche and gonadarche result in changes in sex steroids that may influence family relationships. As stated above, in girls, adrenarche occurs from 6 to 9 years of age and gonadarche occurs at approximately 9 or 10 years of age. Although adrenal androgens are weaker than gonadal steroids and are thus thought to have less influence on behavior (Dorn, Hitt, & Rotenstein, 1999), a pilot project comparing individuals who experienced premature adrenarche with controls who experienced on-time adrenarche suggests that premature adrenarche is associated with poor behavioral adjustment (Dorn, Hitt, & Rotenstein, 1999). To date, however, no empirical research has examined relations between normal variation in adrenal androgens prior to maturation of the HPG axis and either behavioral adjustment or family relationships. By contrast, many studies have implicated changes in gonadal steroid hormones at puberty as causal influences on mood and behavior in adolescence (reviewed in Dorn & Chrousos, 1997).

The potential influence of adrenarche and gonadarche on family dynamics presents special methodological difficulties for researchers attempting to investigate the effects of family relationships on timing of pubertal development. Several strategies can be used, however, to control for or rule out puberty effects. One method is to assess quality of family relationships prior to onset of puberty, at least before age 9 and ideally before age 6. Another method is to use indices of family environment that are not likely to be influenced by the child's pubertal development (e.g., parental psychopathology). A third method is to assess pubertal development at two time points and then assess the relation between family environment at Time 1 and pubertal development at Time 2, controlling for pubertal development at Time 1. As reviewed below, various studies have used each of these methodologies.

Another methodological issue concerns the dimensional conceptualization of family environments. Recent research suggests that family relationships have fairly independent positive and negative dimensions (e.g., B. J. Ellis & Malamuth, 2000; Hetherington & Clingempeel, 1992; Pettit, Bates, & Dodge, 1997) and that each of these dimensions often accounts for unique variance in child outcomes (e.g., Belsky, Hsieh, & Crnic, 1998; B. J. Ellis et al., 1999; Hetherington & Clingempeel, 1992). Indeed, B. J. Ellis et al. (1999) and W. B. Miller and Pasta (2000) recommended analyzing the positive–harmonious and negative–coercive dimensions of family relationships separately when testing for effects on pubertal timing. The current review adheres to this recommendation.

Familial warmth and positivity. A number of prospective longitudinal studies conducted in the United States have examined relations between familial warmth and positivity and subsequent timing of pubertal development in daughters. In a sample of 173 girls and their families, B. J. Ellis et al. (1999) assessed positivity in family relationships on the basis of both behavioral observations in the home and interviews with the mothers. Daughters were 5 years old at the time of these assessments and would not yet have experienced any hormonal changes of puberty. Levels of pubertal development were assessed 7 years later on the basis of daughters' self-report on the Pubertal Development Scale. B. J. Ellis et al. (1999) found that greater warmth and positivity in early family relationships, whether gauged through interviews, $r(N = 157) = -.31$, or home observations, $r(n = 40) = -.45$, predicted lower levels of age-adjusted pubertal maturation in adolescence. Similar results were obtained by Graber, Brooks-Gunn, and Warren (1995) in their longitudinal research on 75 initially premenarcheal girls.

These girls were between the ages of 10 and 14 at Time 1, when they completed measures of parental approval and warmth. Physician ratings of Tanner stages for breast development were also collected at this time. Girls were then followed prospectively to determine subsequent age at menarche. After controlling for age, maternal age at menarche, and pubertal maturation (breast development) at Time 1, greater parental approval and warmth predicted later age at menarche, $\beta(N = 75) = .22$, suggesting that parent–child closeness may decelerate pubertal maturation.

Finally, in a study of 78 girls and their families, Steinberg (1988) assessed levels of pubertal development at two time points during adolescence and then examined effects of parent–child relationships at Time 1 on pubertal development at Time 2, controlling for pubertal development at Time 1.[7] Consistent with Graber et al. (1995), mother–daughter closeness and cohesion had a decelerating effect on daughters' pubertal development: daughter report, $\beta(N = 59) = -.18$; mother report, $\beta(N = 59) = -.20$. Contrary to these findings, however, Steinberg found that frequency of arguments, both mother–daughter, $\beta(N = 59) = -.17$, and father–daughter, $\beta(N = 47) = -.23$, also decelerated pubertal development. In total, Steinberg's data suggest that it may not be parent–child closeness per se but frequency of parent–child interactions—whether positive or negative—that slows pubertal development.[8] B. J. Ellis et al. (1999, Table 5) also reported data consistent with this viewpoint, wherein the frequency of both positive and negative father–daughter interactions predicted later pubertal development in daughters.

Several other researchers have also examined relations between family warmth and positivity and pubertal timing in daughters, but they have either collected family relationship and pubertal timing data concurrently in adolescence (Rowe, 2000a) or retrospectively in adulthood (Jorm, Christensen, Rodgers, Jacomb, & Easteal, 2004; Kim & Smith, 1998a, 1998b; Kim, Smith, & Palermiti, 1997; W. B. Miller & Pasta, 2000; Romans, Martin, Gendall, & Herbison, 2003). These methods do not allow plausible inferences to be made regarding the direction of causation. Nonetheless, most of these studies have reported significant associations between greater family warmth and positivity and later pubertal development (Kim & Smith, 1998a; Kim et al., 1997; W. B. Miller & Pasta, 2000; Romans et al., 2003; Rowe, 2000a).

Familial conflict and coercion. A small number of prospective longitudinal studies have also examined relations between familial conflict and coercion and subsequent timing of pubertal development in daughters. In an investigation of a birth cohort of New Zealand girls and their families, Moffitt et al. (1992) found that mothers' reports of conflictual family interactions, obtained when daughters were age 7, forecast earlier age at menarche, as reported by the daughters at age 15, $r(N = 379) = -.13$.[9] B. J. Ellis et al. (1999), however, failed to replicate this finding: No significant relations were found between either observation-based or interview-based measures of family conflict and coercion, obtained at age 5, and subsequent timing of puberty. Furthermore, as described above, Steinberg (1988) found that both mother–daughter and father–daughter conflict decelerated, rather than accelerated, daughters' pubertal development. Finally, in research that followed 87 adolescent girls and their families, B. J. Ellis and Garber (2000) found that a history of mood disorders in mothers was associated with more advanced pubertal development by daughters in the seventh grade, $r(N = 87) = .30$ (adjusted for age). B. J. Ellis and Garber were able to date the onset of psychopathology in the mothers and rule out the possibility that early pubertal timing in daughters was causing maternal mood disorders. They made the important observation that the effect of maternal psychopathology on daughters' pubertal timing was mediated by quality of family relationships. However, the indices of family relationships (the Dyadic Adjustment Scale, the Family Relationships Index, the Family Assessment Device) all combined familial warmth and positivity and familial coercion and conflict into single measures; thus, it is not possible to determine whether low levels of warmth and positivity, high levels of coercion and conflict, or a combination of the two accounted for the effects of maternal psychopathology on timing of pubertal development. In total, longitudinal research on the relations between family coercion and conflict and timing of pubertal development in daughters has produced inconsistent results.

Other researchers have also addressed this question, but have either collected family coercion–conflict and pubertal timing data in adolescence without controlling for initial levels of pubertal development (Mezzich et al., 1997; Wierson, Long, & Forehand, 1993) or retrospectively in adulthood (Jorm et al., 2004; Kim & Smith, 1998a, 1998b; Kim et al., 1997; W. B. Miller & Pasta, 2000; Romans et al., 2003). Again, the direction of causation cannot be plausibly determined. Nonetheless, with the exception of W. B. Miller and Pasta (2000), all of these investigations found significant associations between higher levels of family coercion and conflict and earlier pubertal development.

Finally, some research has been conducted on the relation between a particular form of family coercion—sexual abuse—and timing of puberty. Although this research is relevant to psychosocial acceleration theory, it is especially pertinent to evaluating pheromonal explanations of pubertal timing and is discussed below in that context (see Psychosocial Models of Pubertal Timing: III. Paternal Investment Theory).

Summary. Psychosocial acceleration theory posits that warm, cohesive family environments slow down pubertal development, whereas dangerous or conflictual family environments accelerate it. Empirical research to date has provided reasonable, though

[7] Assessment of pubertal development was based on ratings by "trained observers" concerning facial characteristics, body proportion and shape, and coordination. Although reliability across raters was good, the validity of this method is unknown.

[8] N. B. Ellis (1991), however, failed to replicate Steinberg's findings. This failure may have been due to methodological weaknesses of the study. N. B. Ellis did not assess frequency of parent–child interactions and used only minimalistic measures of parent–child closeness with unknown validity.

[9] Commenting on this finding, Graber et al. (1995) suggested that, at age 7, some of the daughters in this study would have already begun the hormonal changes of puberty, which could plausibly increase family conflict. This criticism is probably unjustified. Although some girls are likely to have experienced adrenarche by age 7, they would not yet have experienced gonadarche. Moffitt et al.'s (1992) dependent variable—menarche—is part of the cascade of events triggered by gonadarche and maturation of the HPG axis. Adrenarche and gonadarche are largely uncorrelated; that is, girls who experience premature adrenarche do not tend to experience earlier menarche than their peers who experience on-time adrenarche (Apter & Vihko, 1985; Ibanez et al., 1992). Thus, hormonal changes at age 7, though possibly influencing family conflict, are unlikely to influence menarcheal timing.

incomplete, support for the theory. On the one hand, there is converging evidence from a number of methodologically sound studies that greater parent–child warmth and cohesion is associated with later pubertal development. This research also suggests that greater frequency of parent–child interactions predicts later puberty. On the other hand, the proposed accelerating effect of parent–child conflict and coercion on pubertal development is yet to be clearly established.

Although the size of the correlations between family environment and timing of puberty are generally small, these effects may nonetheless have important ramifications. As discussed earlier, the time from menarche until 50% of cycles are ovulatory is approximately 1 year if menarche occurs before age 12 and 4.5 years if menarcheal age is 13 or older. Thus, even small effects of family environment on timing of puberty may have substantial effects on timing of onset of reproductive status.

Possible Mechanisms

The research reviewed so far presents a paradox. On the one hand, activation of the stress-response systems has been found to suppress *activity* of the HPG axis in mature females. Indeed, there is even evidence that psychosocial stress impairs ovarian functioning in women, as suggested by the literature on functional hypothalamic amenorrhea (e.g., Marcus et al., 2001; Nappi & Facchinetti, 2003), though experimental data are lacking. On the other hand, descriptive longitudinal research on humans, as well as experimental research on pigs, suggests that psychosocial stress, at least under some circumstances, can stimulate *maturation* of the HPG axis in prepubertal females. This paradox raises very important questions for future research: Why does psychosocial stress appear to stimulate the reproductive axis in prepubertal girls when most research suggests that stress suppresses the reproductive axis in adults? Is there some difference in the way that psychosocial stressors affect the brain before and after puberty?

Stress reactivity and pubertal development. A recent evolutionary-developmental theory of the origins and functions of stress reactivity, proposed by Boyce and Ellis (in press), may provide the groundwork for resolving this paradox. Paralleling psychosocial acceleration theory, Boyce and Ellis's theory posits that natural selection has favored mechanisms that detect and internally encode information about levels of social resources and support versus stress and adversity in early childhood environments as a basis for adaptively calibrating development. According to the theory, an important function of childhood experience is to entrain development of the stress-response systems, in terms of activation thresholds and magnitudes of response, to match the physical and social world of the child. The theory conceptualizes stress reactivity as openness or permeability to environmental influence, both positive and negative. From this perspective, heightened reactivity within the stress-response systems not only increases awareness of and readiness for danger but also enables children to experience and incorporate more fully the beneficial, protective features of their environments. Accordingly, Boyce and Ellis have hypothesized that there is a curvilinear, U-shaped relation between early exposures to adversity and the development of stress-reactive profiles, with high-reactivity phenotypes disproportionately emerging within both highly stressful and highly protected early social environments (see Figure 1).

B. J. Ellis, Essex, and Boyce (in press) reported data that are not inconsistent with this hypothesis. Specifically, in two studies of 249 children and their families, B. J. Ellis et al. (in press) found that supportive, stable childhood environments were consistently associated with the emergence of high autonomic reactivity. In addition, in one of the two studies, a relatively high proportion of

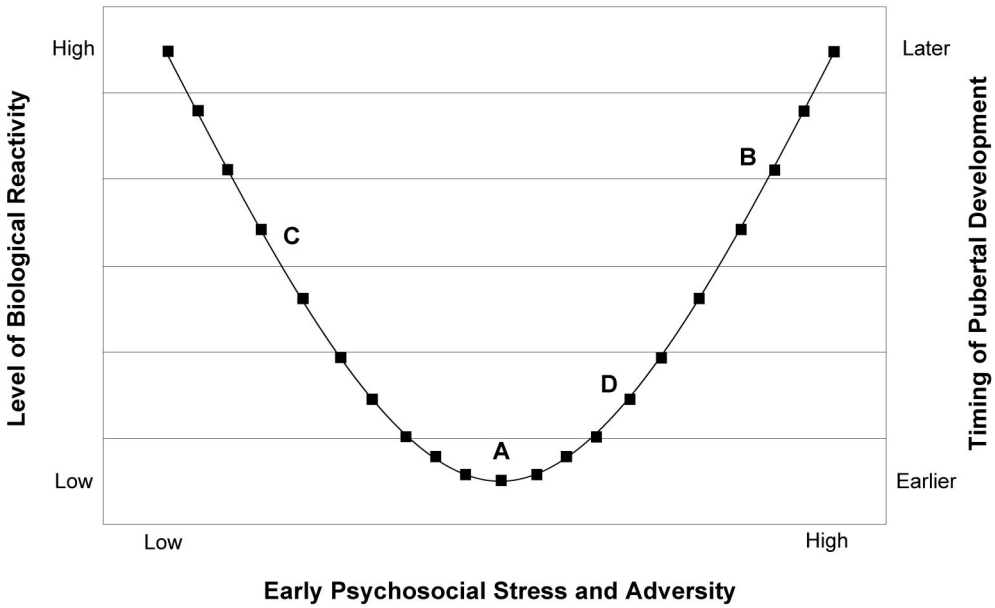

Figure 1. Hypothesized curvilinear relations of early psychosocial stress and adversity to biologic reactivity and pubertal timing. Comparisons of subjects at Points A and B would result in a conclusion that early adversity is associated with greater stress reactivity and later puberty. Comparisons at Points C and D, on the other hand, would generate the inference that early adversity produces diminished reactivity and earlier puberty.

children in very stressful environments showed evidence of heightened sympathetic and adrenocortical reactivity. In both studies, children from moderately stressful environments displayed the lowest reactivity levels.

In light of this theory and data, consider the following extension of Boyce and Ellis's (in press) theory of stress reactivity, proposed here, to explain observed relations between psychosocial stress in childhood and pubertal timing: There is pronounced early plasticity in the neurobiological mechanisms that underpin the development of the CRH and LC-NE systems, and aspects of early experience, particularly parent–child experiences, appear to play a central role in the calibration of stress responses (Hofer, 1994; Meaney, 2001). Growing up in highly protective environments—high levels of social support and stability, low levels of conflict and adversity—up-regulates (i.e., increases) reactivity of the LC-NE system and its effector limbs in the autonomic nervous system. Likewise, developmental exposures to acutely stressful environments up-regulate reactivity of both the LC-NE and CRH systems (e.g., de Bellis et al., 1999; Yehuda, 2002).

As reviewed by Dobson et al. (2003), stressors increase the firing rate of noradrenaline-neuropeptide Y neurons in the regions of the brain stem that control the LC-NE system. These neurons project either indirectly through the medial preoptic area of the hypothalamus or directly through the paraventricular nucleus of the hypothalamus to release CRH and arginine vasopressin (AVP). It is likely that stress stimulates CRH and AVP neurons through other neurocircuits as well. Although the mechanisms that control the GnRH pulse generator are not fully understood, GnRH neurons synapse with CRH and AVP axons in the medial preoptic area (Dobson et al., 2003). CRH and AVP are centrally involved in all stress reactions, have inhibitory effects on secretion of gonadotropins, and appear to be important intervening mechanisms through which activation of the stress-response systems suppresses activity of the HPG axis (Dobson et al., 2003; Ferin, 1999; see also above, Psychosocial Models of Pubertal Timing: I. Stress-Suppression Theory).

If both highly protective and acutely stressful childhood environments cause up-regulation of stress-reactivity systems (Boyce & Ellis, in press; B. J. Ellis et al., in press), and if this up-regulation inhibits maturation of the HPG axis, then there should be U-shaped curvilinear relations between levels of social resources and support versus stress and adversity in early childhood environments and not only stress reactivity but also timing of puberty (see Figure 1). The right side of Figure 1 (Point B) depicts expected reactivity levels and pubertal timing for individuals who experience very high levels of psychosocial stress in early childhood. These individuals are hypothesized to develop heightened reactivity profiles and, consequently, to experience relatively late pubertal development. It is not expected, however, that reactivity levels and pubertal timing will change in a linear fashion with decreasing childhood stress. The left side of Figure 1 (Point C) shows predicted reactivity levels and pubertal timing for individuals whose early childhoods are characterized by intensive, stable caregiving and family support. These individuals are also hypothesized to develop exaggerated reactivity profiles and, consequently, to experience relatively late puberty. Finally, the middle of Figure 1 (Points A and D) reflects the anticipated, relatively muted reactivity profiles and early pubertal development of individuals whose early childhood experiences are characterized by moderately high levels of ongoing psychosocial stress and threat. In most studies conducted in modern Western societies, such as those reviewed in the preceding section, *Empirical Investigations of the Relations Between Psychosocial Stress and Timing of Pubertal Maturation in Girls*, early environments regarded as high in stress and adversity would actually fit into this middle area (as compared, for example, with the severe stress experienced in cases of psychosocial dwarfism or by war victims in Yugoslavia).

Such an account would reconcile important contradictions, reviewed above, in the existing literature on psychosocial determinants of pubertal timing in girls. Investigators comparing individuals from Points A and B in Figure 1, for example, would conclude, as have Hopwood et al. (1990) and Prebeg and Bralic (2000), that psychosocial stress inhibits pubertal development. On the other hand, studies comparing individuals from Points C and D would find, as have those reviewed immediately above, that psychosocial stress accelerates pubertal development. The current theorizing, which posits two oppositionally distinctive ontogenies for late pubertal timing, generating the proposed U-shaped curve, explains both of these inhibiting and accelerating effects. These distinctive ontogenies share the common underlying mechanism of high reactivity of the stress-response systems.

The proposed U-shaped curvilinearity hypothesis potentially reconciles the countervailing evolutionary arguments advanced by psychosocial acceleration theory and stress-suppression theory. Consistent with psychosocial acceleration theory, the model posits that natural selection has favored phenotypic mechanisms that bias allocation of resources toward relatively early sexual development under conditions of moderately high psychosocial stress and uncertainty. Under such conditions, it is generally adaptive to mature and reproduce early. Conversely, consistent with stress-suppression theory, the model posits that natural selection has favored phenotypic mechanisms that bias allocation of resources toward relatively late sexual development under conditions of very high psychosocial stress. That is, under very bad conditions in which current reproduction is unsustainable, it is generally adaptive to mature slowly and delay reproduction until predictably better times.

GCs and pubertal development. Observed relations between childhood stress and pubertal timing are likely to be subserved by multiple mechanisms. CRH and AVP interact synergistically to control secretion of adrenocorticotropin hormone from the anterior pituitary, which in turn regulates secretion of GCs, principally cortisol, from the adrenal cortex. Family adversity, disruptions in early attachment relationships, and other traumatic childhood experiences have been linked to abnormal cortisol profiles (e.g., elevated cortisol responses, elevated 24-hr urinary cortisol excretion, increased density of lymphocyte GC receptors, a flattening in the circadian pattern of cortisol secretion; reviewed in Boyce & Ellis, in press). Though speculative, these altered cortisol profiles may in turn affect the timing or tempo of adrenarche or gonadarche.

One possibility is that activation of the HPA axis increases secretion of adrenal androgens (i.e., accelerates adrenarche). Research by Dorn and colleagues has shown that girls with premature adrenarche have more than a twofold elevation of serum and salivary cortisol levels relative to controls (Cizza et al., 2001; Dorn, Hitt, & Rotenstein, 1999). Cizza et al. (2001) hypothesized that girls with premature adrenarche are characterized by exaggerated reactivity of the HPA axis. This hypothesis is consistent with research documenting high levels of behavioral and mental health

problems in children with premature adrenarche (Dorn, Hitt, & Rotenstein, 1999).

The effects of GCs on gonadarche are poorly understood. Although GCs are often presumed to suppress activity of the HPG axis (e.g., Dorn & Chrousos, 1997), the suppressive effects of CRH and AVP on the GnRH pulse generator are not mediated by GCs and are readily observed in adrenalectomized primates (Ferin, 1999). Relations between GCs and activity of the HPG axis in mature animals is an active and unresolved area of research. By contrast, the influence of GCs on maturation of the HPG axis in immature animals has yet to be studied. Again, the most relevant experimental research has been conducted on pigs. As discussed below (see Psychosocial Models of Pubertal Timing: III. Paternal Investment Theory), prepubertal gilts tend to accelerate puberty in response to contact with boars. Contrary to the stress-suppression hypothesis, the efficacy of the boar effect appears to depend on adrenocortical activity (reviewed in Booth & Signoret, 1992). Specifically, plasma cortisol levels in prepubertal gilts have been found to increase in response to contact with boars. This increased cortisol in turn appears to increase basal LH secretion just before the onset of puberty in gilts. Conversely, adrenalectomy or inhibition of adrenocortical function by dexamethazone tends to delay puberty in response to boar contact.

Little is known about the effects of GCs on development of the HPG axis in humans. Nonetheless, correlational studies have shown that concentrations of salivary cortisol (Keiss et al., 1995; Netherton, Goodyer, Tamplin, & Herbert, 2004), serum cortisol (Elmlinger, Kuhnel, & Ranke, 2002), and urinary free-cortisol excretion (Legro, Lin, Demers, & Lloyd, 2003) all increase with pubertal maturation, as indexed by Tanner stage. In any case, the causal role of GCs in regulation of girls' pubertal timing, and particularly the hypothesis that cortisol mediates observed relations between quality of family environments and timing of pubertal maturation, remains to be investigated. These investigations will require solid theoretical and methodological grounding, given the anomalous research literature linking childhood trauma to, not only hypercortisolism, but hypocortisolism as well (reviewed in Boyce & Ellis, in press).

Psychosocial Models of Pubertal Timing: III. Paternal Investment Theory

The paternal investment theory of the timing of pubertal development is a variant of psychosocial acceleration theory and is based, fundamentally, on the theorizing of Draper and Harpending (1982, 1988). These authors hypothesized that the developmental pathways underlying variation in daughters' reproductive strategies are especially sensitive to the father's role in the family and mother's sexual attitudes and behavior in early childhood. Both psychosocial acceleration theory and paternal investment theory specify relevant developmental experiences and psychosocial cues that bias individuals toward earlier versus later sexual development. But in specifying those experiences and cues, psychosocial acceleration theory focuses on a multiplicity of qualities and features of the family ecology (including quality of father–daughter relationships and father absence) as they relate to the child's overall experiences of stress versus support. By contrast, paternal investment theory focuses specifically on the father's role in the family and the mother's sexual attitudes and behavior toward men. In other words, paternal investment theory, as formulated by me and my colleagues (B. J. Ellis & Garber, 2000; B. J. Ellis et al., 1999, 2003), posits a unique and central role for quality of paternal investment in regulation of daughters' sexual development, separate from the effects of other dimensions of psychosocial stress and support in the child's environment. Paternal investment theory is not inconsistent with psychosocial acceleration theory, given that the narrow set of predictions generated by paternal investment theory are almost fully subsumed by the broader set of predictions generated by psychosocial acceleration theory. Rather, paternal investment theory narrows the focus of psychosocial acceleration theory and moves it closer to its roots in the theorizing of Draper and Harpending (1982).

Humans are the only great ape in which males engage in provisioning or care of offspring. Human paternal investment, therefore, is almost certainly a recent evolutionary development (i.e., less than 5 million years). Indeed, mothers (and sometimes their female kin) form the primary foundation of parental care in all societies (Geary, 2000), and the contribution of fathers to the family is—and presumably always has been—widely variable. In his review of the evolution and proximate expression of human paternal investment, Geary (2000) proposed (a) that over human evolutionary history, fathers' investment in families tended to improve, but was not essential to, the survival and fitness of children and (b) that selection consequently favored a range of paternal strategies, with different men varying in the extent to which they allocated resources to care and provisioning of children. Because fitness is always relative, and because variation in paternal investment influences variation in female fitness, selection can be expected to have favored psychological mechanisms in women that are especially attuned to variation in the willingness and ability of men to invest in families. Consistent with this logic and drawing on the concept of sensitive-period learning of reproductive strategies, paternal investment theory posits that girls detect and internally encode information specifically about the quality of paternal investment during approximately the first 5 years of life as a basis for calibrating the development of (a) neurophysiologic systems involved in timing of pubertal maturation and (b) related motivational systems, which make certain types of sexual behavior more or less likely in adolescence.

Relevant cues to paternal investment are provided by both fathers and mothers. Perhaps the most important cue is father presence versus absence (i.e., the extent to which women rear their children with or without consistent help from a man who is father to the children). Other important cues may include frequency of father–daughter interactions, levels of cohesion and conflict in father–daughter relationships, quality and stability of the father–mother relationship, the mother's attitudes toward men, the mother's sexual and repartnering behavior, and the daughter's exposure to her mother's boyfriends and stepfathers.

An underlying assumption of paternal investment theory is that human paternal investment is facultatively expressed in accordance with varying proximal conditions. As reviewed by Geary (in press), paternal investment generally varies as a function of the degree to which it enhances the fitness of offspring, the extent to which it must be traded off against mating opportunities, and the level of paternity uncertainty (see also Marlowe, 2000, 2003). For example, across foraging societies in the Standard Cross-Cultural Sample, societies with higher levels of paternal provisioning are more monogamous ($r = .44$, $p = .01$, $n = 30$; Marlowe, 2003). This covariation between paternal investment and important re-

productive parameters implies that quality of paternal investment conveys reproductively relevant information to children. Over human evolution, quality of paternal investment afforded reliable cues to the mating systems into which children were born and the reproductive opportunities and constraints that they were likely to encounter at adolescence and beyond.

Paternal investment theory posits that early experiences associated with low-quality paternal investment function to entrain the development of reproductive strategies that, during human evolution, were statistically linked to increased reproductive success in that social milieu—a milieu in which male parental investment is relatively unreliable and/or not closely linked to variation in reproductive success. Girls in this context are predicted to develop in a manner that accelerates pubertal maturation and onset of sexual activity and orients the individual toward relatively unstable pairbonds. As Belsky et al. (1991) suggested, in environments in which paternal investment is not forthcoming,

> a young woman who waits for the right man to help rear her children may lose valuable reproductive opportunities at a time when her health and physical capability are at their peak and when her mother and senior female kin are young enough to be effective surrogates. (p. 653)

This theorizing has been supported by cross-cultural analyses demonstrating that young women are more likely to have adolescent pregnancies and become single mothers when they have diminished prospects of obtaining paternal investment (Barber, 2001, 2003).

Conversely, early experiences associated with high-quality paternal investment are hypothesized to entrain development of reproductive strategies that, during human evolution, were statistically linked to increased reproductive success in that social milieu—a milieu in which male parental investment is reliable and forthcoming and in which variations in offspring quality are sensitive to provision of paternal care and resources. Girls in this context are predicted to develop in a manner that slows pubertal maturation, delays onset of sexual activity and reproduction, and increases reticence in forming sexual relationships. Under these conditions, a longer pre-reproductive developmental period enables daughters to practice and refine sociocompetitive competencies (Geary & Flinn, 2001) and facilitates formation of relatively long-term pairbonds with reliable and nurturant mates.

Paternal investment theory links timing of pubertal maturation to variation in levels of intrasexual competition associated with different mating systems. Monogamy tends to produce a shortage of high-quality prospective husbands and thus increases female–female competition for mates, whereas polygyny tends to have the opposite effect (see Hoier, 2003; Kanazawa, 2001). Among women, therefore, successful long-term mating requires greater accumulation of resources and competitive skills in more monogamous societies. Furthermore, father presence and high paternal investment experienced by girls in the home function as microlevel indicators of the degree of monogamy in society at the macrolevel (Kanazawa, 2001; see also Marlowe, 2003). Kanazawa (2001) defined monogamy broadly to include both low levels of divorce and remarriage in legally monogamous societies and bias toward monogamous marriage in legally polygynous societies; for these reasons, a further prediction of paternal investment theory is that higher levels of monogamy at the societal level will be associated with later pubertal development.

It is important to note that paternal investment theory does not equate father absence with stress, even though girls in father-absent homes in Western societies tend to be economically disadvantaged. In cross-cultural perspective, father-absent societies are characterized by aloof husband–wife relationships, little or inconsistent direct paternal investment in children, polygyny, relatively high levels of childcare by female kin, and high levels of male violence and intrasexual competition (Broude, 1990; Draper & Harpending, 1988). Among hunter–gatherers, father-absent social systems are generally found in rich, stable environments in which women can often provide adequate parental care and resources without the direct contribution of the father. By contrast, father-present social systems are more likely to be found among hunter–gatherers in harsher or unstable environments in which biparental care is important for offspring survival and reproductive success (Draper & Harpending, 1988; Geary, 2000; Marlowe, 2003). If father-absent social systems, on average, were statistically associated with resource-rich ecologies during human evolutionary history, then it is unlikely that our evolved psychological mechanisms would be engineered to read father absence as an indicator of stress or uncertainty. Consequently, a premise of paternal investment theory is that variation in quality of paternal investment, on the one hand, and more general variation in familial and ecological stressors, on the other, constitute separate and largely independent paths to timing of sexual development (B. J. Ellis & Garber, 2000; B. J. Ellis et al., 1999, 2003). Quality of paternal investment should provide unique information about future mating conditions, separate from the information provided by familial and ecological stressors more generally.

Because the effects of stress and father absence on sexual development appear to be largely independent, these effects can potentially either reinforce or counteract each other. For example, Waynforth (2002) has studied the effects of father absence on the reproductive strategies of both a hunter–gatherer group (the Ache of Eastern Paraguay) and a subsistence-level horticulturalist population (the Maya of Belize). In both societies, paternal investment is important to offspring quality and survival. Waynforth found that men and women who were raised in father-absent home environments tended to have later, rather than earlier, ages at first reproduction. Consistent with stress-suppression theory, Waynforth attributed this delayed reproduction to nutritional and social stress and insufficient resources to secure a long-term mate. Consistent with paternal investment theory (as well as more traditional social learning theories of development), however, father-absent Mayan men were less oriented than father-present Mayan men toward maintaining long-term mating relationships and investing in their children. (Equivalent data were not collected for Mayan women or the Ache.) Thus, in environments where paternal investment is important, its absence may have paradoxical (i.e., bidirectional) effects on development of reproductive strategies.

As stated above, paternal investment theory conceptualizes stepfathers and other unrelated men in the home as indicators of low-quality paternal investment. Repeated repartnering by the mother provides an especially strong cue to the child that paternal investment is relatively unreliable and/or unimportant. Furthermore, the presence of stepfathers in the home dramatically increases risk of child abuse and neglect (Daly & Wilson, 1988) and generally degrades the quality of parental investment (e.g., Lancaster & Kaplan, 2000). Waynforth (2002) noted that the presence of stepparents, stepsiblings, and half-siblings in the home all

reduce the fitness benefits of cooperating with other household members. Thus, the genetic benefits of performing and receiving nepotistic acts, such as sharing of resources and cooperating in childcare, cannot be fully realized in blended families (see Jankowiak & Diderich, 2000). As discussed earlier (see Psychosocial Models of Pubertal Timing: II. Psychosocial Acceleration Theory), pubertal development is characterized by distancing of parent–child relationships and increased orientation of children toward peers and mating relationships (see also Surbey, 1998). From a life history perspective, it is to the child's advantage to make the pubertal transition earlier in adverse home environments. Thus, a further prediction of paternal investment theory is that the effects of father absence on daughters' sexual development will be partially mediated by the presence of stepfathers and mother's boyfriends in the home environment (B. J. Ellis & Garber, 2000).

Empirical Investigations of the Relations Between Paternal Investment and Daughters' Pubertal Timing

Father absence versus father presence. The large majority of research on the relation between paternal investment and daughters' pubertal timing has examined father absence effects. Father absence has been operationalized as the absence of the biological father from the home, usually prior to the onset of daughters' puberty. Several father absence studies have assessed pubertal timing prospectively as it occurred in adolescence (Campbell & Udry, 1995; B. J. Ellis & Garber, 2000; B. J. Ellis et al., 1999; Hetherington & Kelly, 2002; Moffitt et al., 1992; Rowe, 2000a; Wierson et al., 1993). The most common dependent variable has been age at menarche, but a few prospective investigations have also assessed development of secondary sexual characteristics (B. J. Ellis & Garber, 2000; B. J. Ellis et al., 1999; Rowe, 2000a). Other father absence research has used adult samples and assessed age at menarche retrospectively (Doughty & Rodgers, 2000; Hoier, 2003; Jones, Leeton, McLeod, & Wood, 1972; Jorm et al., 2004; Kiernan & Hobcraft, 1997; Quinlan, 2003; Romans et al., 2003; Surbey, 1990). Finally, some studies have relied on convenience samples (Hoier, 2003; Surbey, 1990; Wierson et al., 1993), some have compared community-based psychopathology samples with carefully selected controls (B. J. Ellis & Garber, 2000; Romans et al., 2003), and others have obtained broad, representative community or national samples (e.g., Doughty & Rodgers, 2000; B. J. Ellis et al., 1999; Jorm et al., 2004; Moffitt et al., 1992; Quinlan, 2003). The research has been conducted in a variety of Western countries, including Australia, Canada, Germany, New Zealand, the United Kingdom, and the United States. Despite this diversity of methods and samples, the results have been remarkably consistent: Girls from father-absent homes tend to experience earlier pubertal development than girls from biologically intact families.

The most direct test of the paternal investment hypothesis involves comparing girls whose biological fathers were absent at or before age 5 (early father absence) with girls who grew up in biologically intact families (father presence). Jones et al. (1972, Table 2) found that early father-absent girls were almost 3 times more likely than father-present girls to have experienced menarche before age 12 (37% vs. 13%, $n = 371$; odds ratio = 3.83). Likewise, in my reanalysis of data reported in B. J. Ellis et al. (1999), early father-absent girls were found to be almost twice as likely as father-present girls to have completed pubertal development by the seventh grade (45% vs. 24%, $n = 139$; odds ratio = 2.62).[10] Similarly, Quinlan (2003) reported that early father-absent girls had almost twice the risk of experiencing early menarche than did father-present girls ($N = 10{,}135$; hazard ratio = 1.80). Kiernan and Hobcraft (1997), however, did not find a significant difference in menarcheal age between girls whose parents divorced before age 9 and girls from intact families.

Similar analyses have also been reported by Moffitt et al. (1992) and Romans et al. (2003). These researchers, however, did not demarcate early father-absent girls in their analyses and instead operationalized biological father absence as having occurred either by age 11 (Moffitt et al., 1992) or anytime during childhood (Romans et al., 2003). This clustering of early father-absent and late father-absent girls makes their results more difficult to interpret in terms of paternal investment theory because it both precludes testing of the sensitive period hypothesis and leaves open the possibility that girls' pubertal timing influenced father absence rather than the reverse. Nonetheless, both studies found that girls from father-absent homes had more than twice the odds of experiencing menarche before age 12 than did girls from father-present homes (Moffitt et al., 1992 [$N = 416$]: 27% vs. 15%, odds ratio = 2.17; Romans et al., 2003 [$N = 475$]: odds ratio = 2.62).

A number of other researchers have examined differences between father-absent and father-present girls in mean age at menarche (Campbell & Udry, 1995; Doughty & Rodgers, 2000; Hetherington & Kelly, 2002; Hoier, 2003; Jorm et al., 2004; Rowe, 2000a; Surbey, 1990; Wierson et al., 1993). Only Campbell and Udry (1995), however, specifically compared early father-absent girls with father-present girls. Nonetheless, with the exception of Rowe (2000a), all of these researchers found that father-absent girls tended to experience earlier menarche than did father-present girls (Campbell & Udry, 1995: 2 months earlier; Doughty & Rodgers, 2000: 1.3 months earlier; Hetherington & Kelly, 2002: 4 months earlier in single-mother families, 9 months earlier in stepfather families; Hoier, 2003: 4 months earlier; Jorm et al., 2004: 2.9 months earlier; Surbey, 1990: 4 to 5 months earlier; Wierson et al., 1993: 5 months earlier). The father-absent effect has not emerged in African American samples, however (Campbell & Udry, 1995; Rowe, 2000a).[11]

Finally, three studies have examined relations between father absence–presence and composite measures of pubertal timing (operationalized as levels of pubertal maturation in adolescence, controlling for age). Rowe (2000a) concurrently assessed family composition and pubertal status (breast and body curve development) when girls were approximately 16 years old ($SD = 1.7$). Father-absent girls were found to be significantly more sexually developed than father-present girls (with an effect size of about two 10ths of a standard deviation), but only among Caucasians.

[10] Girls were 12 to 13 years old at the seventh grade data collection. Girls were categorized as having completed pubertal development (status = "postpubertal") on the basis of their scores on the Pubertal Development Scale. After adjusting for age and race, the odds ratio increased to 2.97.

[11] One possible explanation for this null finding is that the extraordinary secular trend among African Americans, who to my knowledge experience earlier pubertal development than any other population (see Herman-Giddens et al., 1997), has effectively squeezed the variance out of pubertal timing and thus attenuated its relations with other variables.

Rowe's (2000a) results must be interpreted with caution, however, given the late assessment of father absence and the postpubertal assessment of pubertal timing. In addition, B. J. Ellis et al. (1999) assessed father absence status at age 5 and then correlated it with levels of pubertal development in the seventh grade. Similarly, B. J. Ellis and Garber (2000) assessed father absence status in the sixth grade and correlated it with seventh grade pubertal development. The dependent variable in both investigations was a composite of breast development, body hair growth, and menarcheal status, controlling for age. Father absence consistently predicted greater pubertal development, B. J. Ellis & Garber, 2000: $r(N = 87) = .30$; B. J. Ellis et al., 1999: $r(N = 163) = .17$. In B. J. Ellis et al. (1999), this correlation increased to .23 ($n = 134$) when African Americans were excluded from the analysis. In sum, although the effect sizes are small, there is widely converging evidence that father absence predicts earlier timing of sexual development. More research is needed, however, to determine the extent of this effect across different racial groups and in non-Western populations.

Timing of father absence. Based on the concept of a sensitive period for acquisition of reproductive strategies, a prediction of paternal investment theory is that earlier onset of father absence (particularly in the first 5 years of life) will be associated with earlier pubertal development. Surbey (1990) examined years of exposure to the biological father before age 10. The analysis included the full sample, including father-present girls who would have all received maximum scores. More years of exposure to the biological father was associated with later age at menarche, $r(N = 1115) = .13$. Analogously, Moffitt et al. (1992) and B. J. Ellis and Garber (2000) examined years of biological father absence prior to puberty. Their analyses included only the subsets of girls who had been exposed to father absence. More years of father absence was associated with earlier age at menarche, Moffitt et al.: $r(n = 143) = -.12$, and more pubertal development at seventh grade, B. J. Ellis and Garber: $r(n = 47) = .13$. Further, Quinlan (2003; $N = 10{,}135$) compared hazard functions for early menarche for girls who experienced parental separation at either 0–5 years of age, 6–11 years of age, or 12–17 years of age. Using father-present girls as the reference group, the hazard ratio monotonically decreased, from 1.80 to 1.49 to 1.18, with later age at parental separation. In sum, consistent with the theory, the available evidence suggests that girls who experience father absence from an earlier age tend to experience earlier pubertal development.

Father involvement in the family. Another prediction of paternal investment theory is that close father–daughter relationships (e.g., frequent father–daughter interactions, father–daughter cohesion) and father–mother relationships in early childhood forecast later pubertal timing in daughters. To test the father–daughter prediction, B. J. Ellis et al. (1999) collected mother-reported data on the amount of time that fathers spent taking care of their daughters during the first 5 years of life and conducted home observations of father–daughter and mother–daughter interactions at age 5. Consistent with the theory, results showed, first, that more time spent by fathers in childcare was associated with later pubertal timing in daughters; that is, less pubertal development by daughters in the seventh grade, controlling for age, $r(N = 173) = -.23$. This relation held even in the subset of families in which the fathers had been present in the home throughout their daughters' entire childhood, $r(n = 107) = -.24$. Second, greater father–daughter affectionate–positivity during the home observations was associated with later pubertal timing in daughters, $r(n = 41) = -.43$. (All of the father–daughter observations were conducted in father-present homes.) Third, although both mother–daughter and father–daughter affectionate–positivity were associated with later pubertal timing in daughters, only father–daughter affectionate–positivity made a unique contribution to the prediction of daughters' puberty (after controlling for the quality of mother–daughter relationships), $\beta(n = 40) = -.36$. Fourth, when father–daughter affectionate–positivity and father–daughter coercive control were entered into the regression equation together, both variables uniquely, significantly, and additively predicted later pubertal timing in daughters. Because the affectionate–positivity and coercive control measures were both sensitive to frequency of father–daughter interactions, it may be that more father–daughter interaction or involvement per se, whether positive or negative, delays pubertal maturation in daughters (as also suggested by Steinberg, 1988; see earlier discussion). In total, consistent with the theory, these data suggest that amount of paternal care and father–daughter interactions beginning early in life are associated with later pubertal development in daughters.

Several studies have also examined links between parents' relationship quality and daughters' pubertal timing. B. J. Ellis et al. (1999) assessed both levels of supportiveness and severity of conflict in the parental dyad (on the basis of interviews with the mothers) when daughters were 5 years of age. Only the supportiveness variable significantly correlated with pubertal timing: More supportive mother–father relationships forecast less pubertal development by daughters in the seventh grade, controlling for age, $r(N = 162) = -.25$. Similar results were reported by B. J. Ellis and Garber (2000), who found that better dyadic adjustment, as reported by mothers when daughters were in the sixth grade, predicted less age-adjusted pubertal development by daughters in the seventh grade, $r(N = 74) = -.37$. In addition, a number of studies have examined relations between marital quality and age at menarche in daughters, but these have either collected marital quality and menarche data concurrently in adolescence (Wierson et al., 1993) or retrospectively in adulthood (Kim & Smith, 1998a, 1998b; Kim et al., 1997; Romans et al., 2003). These methods do not allow plausible inferences to be drawn regarding the direction of causation. Nonetheless, most of these studies have reported significant associations between higher marital quality (e.g., more happy marital relations, less marital conflict) and later ages at menarche (Kim & Smith, 1998b; Kim et al., 1997; Romans et al., 2003). In sum, converging evidence from both prospective and retrospective studies indicates that better quality relationships between mothers and their male partners correlates with later pubertal timing in daughters.

Stepfather presence. The evolutionary logic underlying potential relations between exposure to unrelated men and girls' pubertal timing is not well developed (but see B. J. Ellis, 2002; B. J. Ellis & Garber, 2000; Surbey, 1990). There are several ways of approaching this question. One is to conceptualize stepfathers and other mating partners of the mother as indicators of parental reproductive strategies; that is, as indicators that male–female relationships are unstable and paternal investment is unreliable and unimportant. From this perspective, the important variable should be number of different male partners in the home and the mother's sexual behavior and attitudes toward men more generally. Accordingly, the presence of a single, long-term stepfather could be a protective factor against early maturation, whereas a succession of

different male partners would be expected to increment risk. Unfortunately, no one has studied the effects of mothers' sexual and repartnering behavior, as experienced by father-absent girls in early and middle childhood, on timing of pubertal development.

Another approach is to conceptualize "father figures" (stepfathers and other cohabitating partners of the mother) as indicators of a degraded family environment. A simple prediction from this perspective is that earlier exposure to father figures (i.e., longer exposure to the degraded environment) should be associated with earlier pubertal maturation. This prediction is almost certainly too simplistic, however, because it does not take into account the quality of the father figures' investment in the family. A more complex prediction is that the relation between duration of exposure to father figures and timing of pubertal development in daughters will be moderated by the quality of the father figure's investment in and relationships with family members. Along these lines, B. J. Ellis and Garber (2000, Figure 3) found that girls in families with father figures tended to experience early pubertal development only when the relationship between the mother and the father figure was quite stressful. B. J. Ellis and Garber also reported a significant correlation between the age of the daughter when the father figure came into her life and timing of pubertal development, $r(n = 31) = -.37$. Quinlan (2003), however, failed to replicate this relation. Viewed in light of the current moderational hypothesis, this failure is not surprising.

Finally, exposure to father figures may operate as a trigger among prepubertal girls to accelerate pubertal development in the presence of a genetically appropriate (i.e., unrelated) adult male. This is a widespread phenomenon among mammalian species (see *Possible Mechanisms: The Male Effect*) and has been given various names such as *the ram effect* and *the boar effect*. For example, over a 13-year period in a stable colony of hamadryas and hybrid baboons at the Madrid zoo, the average age of menarche (first signs of sexual swelling) was 173 weeks (Colmenares & Gomendio, 1988). As is typical of captive primates, this age at menarche was considerably lower than has been reported for baboons in the wild (Sigg, Stolba, Abegglen, & Dasser, 1982). Nonetheless, with the introduction of 3 unfamiliar and genetically unrelated adult males into the colony, the average age of menarche dropped by a full year, or 30%, to 121 weeks. Colmenares and Gomendio (1988) reported that immature females responded within 2–3 months of the novel males' entry into the group and tended to synchronize their first estrus.

A carefully designed study by Mekos, Hetherington, and Clingempeel (1992) suggests that a similar effect may operate among humans. The research consisted of 71 families with a daughter between the ages of 9 and 13 at Time 1. Twenty-eight girls lived in biologically intact families; 22 in divorced, single-mother families; and 21 in remarried, stepfather families. Remarriage had occurred within 5 months of the beginning of the project. Dummy variables were created that contrasted (a) girls in single-mother families with all others (male absence) and (b) girls in stepfather families with all others (stepfather presence). Pubertal status was assessed both at Time 1 and Time 2 (2 years later) on the basis of menarcheal status, breast development, and body hair. After controlling for pubertal development at Time 1, stepfather presence, but not male absence, predicted significantly greater pubertal development at Time 2. Analogous to the findings with hamadryas baboons, these data raise the possibility that prepubertal girls respond to an unrelated adult male in the home by increasing tempo of pubertal maturation.

In addition, various researchers have reported comparisons between girls living in biologically intact, single-mother, and stepfather families in average ages at menarche (Hetherington & Kelly, 2002; Hoier, 2003; Rowe, 2000a; Surbey, 1990). None of these studies, however, either controlled for initial levels of pubertal development or considered timing of stepfather exposure, duration of stepfather exposure, number of different father figures, or quality of the father figures' investment in the family. It is not surprising that these investigations have produced mixed results. The potential effects of father figures on girls' pubertal development are complex, and theory and research are needed that embrace this complexity.

Are father effects distinct from stress? An assumption of psychosocial acceleration theory is that it is not father absence per se but a variety of other stressors associated with father absence (e. g., divorce, poverty, conflictual family relationships) that provoke early sexual maturation in daughters (Belsky et al., 1991, p. 658; Chisholm, 1999, p. 162). This raises an important question: Are the effects of paternal investment on daughters' sexual development distinct from the effects of stress, including family relationship stress more generally? Surbey (1990) was the first to address this question by collecting retrospective data both on years of father presence and number of stressful life events in the first 10 years of life. These two measures were negatively correlated, $r(N = 1127) = -.31$. As predicted by psychosocial acceleration theory, more stressful life events were associated with earlier age at menarche, $r(N = 1104) = -.14$. Nonetheless, the correlation between years of father presence and age at menarche—though slightly reduced, $r(N = 1115) = .09$—remained statistically significant after partialing out the life events measure.

Surbey's (1990) data are consistent with the notion of multiple unique influences on pubertal timing, including independent effects of father presence. This conclusion has been supported by subsequent prospective research showing that father absence and stressful family relationships each uniquely and significantly predict earlier timing of puberty in daughters (B. J. Ellis & Garber, 2000; Moffitt et al., 1992). Finally, B. J. Ellis et al. (1999) collected a variety of measures of familial and ecological stress and paternal investment in early childhood and examined their pattern of relations with pubertal timing. A more specific father-effects model fits the data better than a more general stress model. As B. J. Ellis et al. (1999) concluded, "In total, *the present data suggest that the quality of fathers' investment in the family is the most important feature of the proximal family environment relative to daughters' pubertal timing*" (p. 398). Taken together, these studies are consistent with the hypothesis that quality of paternal investment constitutes a unique path to timing of pubertal development in daughters.

Summary. Paternal investment theory provides the foundation for a series of predictions about the role for fathers and other men in regulation of girls' pubertal timing. Although the theory began with a focus on father absence versus presence, it has since been elaborated to include multiple dimensions of paternal investment (e.g., the dimensional quality of paternal involvement in father-present homes, quality of father–mother relationships, the effects of father figures) and specifically conceptualizes father effects as distinct from the more general effects of familial and ecological stressors. Paternal investment theory has now been tested in a

number of investigations and has received provisional empirical support. In well-nourished populations, girls from father-absent homes tend to experience earlier pubertal development than do girls from father-present homes, and the earlier father absence occurs, the greater the effect. There is also initial longitudinal evidence that within father-present homes higher levels of paternal caretaking and involvement are associated with later pubertal development in daughters. In addition, converging results from both prospective and retrospective studies indicate that better quality relationships between mothers and their male partners predict later pubertal timing in daughters. The possibility that father figures accelerate girls' pubertal timing is intriguing but in need of further theoretical development and empirical testing. Finally, there is consistent evidence that quality of paternal investment uniquely predicts timing of pubertal development in daughters independently of other aspects of the family ecology.

Possible Mechanisms: The Male Effect

Reproductive development in mammals is often regulated by social cues. In a variety of species—mice, rats, gerbils, lemmings, musk shrews, prairie voles, prairie dogs, pigs, goats, red deer, cows, marmosets, tamarins, baboons—contact with members of one's natal group inhibits female pubertal development, whereas exposure to unfamiliar adult males accelerates it (Amoah & Bryant, 1984; Clark & Galef, 2002; Fisher, Meikle, & Johnstone, 1995; Hoogland, 1982; Sanders & Reinisch, 1990; Sigg et al., 1982; Vandenbergh, 1983). Interactions with conspecifics presumably influence pubertal timing through neural and chemosensory mechanisms that affect the production and secretion of GnRH and related gonadal processes. One class of external cues that acts both to inhibit and stimulate maturation of the HPG axis is *pheromones* (Bronson, 1989; Vandenbergh, 1983): "airborne chemical signals that are released by an individual into the environment and which affect the physiology or behavior of other members of the same species" (Stern & McClintock, 1998, p. 177). Pheromones are encoded through olfactory channels and can impact reproductive endocrinology either alone or in combination with visual, auditory, and tactile stimuli from conspecifics (Bronson, 1989; Solomon, Vandenbergh, Wekesa, & Barghusen, 1996; Vandenbergh, 1983; Widowski, Ziegler, Elowson, & Snowdon, 1990).

Social inhibition of pubertal development by adult females has been widely reported in the literature. For example, in a number of *Callitrichid* primates, only the dominant female becomes pregnant, and subordinate females either do not experience onset of ovulation or have impaired ovulatory function (Saltzman, Schultz-Darken, & Abbott, 1997; Vandenbergh, 1983; Ziegler, Snowdon, & Uno, 1990; see also above, Psychosocial Models of Pubertal Timing: I. Stress-Suppression Theory). Although the focus of most research in this area has been on the suppressive effects of the breeding female or other female group members, an emerging literature now suggests that the presence of any familiar members of the natal group, male or female, including siblings, can retard female pubertal development (Clark & Galef, 2002; Hoogland, 1982; Schadler, 1983; Widowski et al., 1990).

The accelerating effect of unfamiliar adult males on female pubertal maturation—"the male effect"—has been extensively studied. The nature of the male effect appears to be contingent on several physical and social factors. First, immature male stimuli are ineffective. Experimental research in which gilts were either exposed to juvenile or adult boars (Kirkwood & Hughes, 1981) and in which female mice were either exposed to juvenile or adult male urine (Drickamer & Murphy, 1978) indicates that only exposure to adult males and their pheromones accelerates female puberty. This conclusion is consistent with human research showing no differences in pubertal timing between girls who attend same-sex versus mixed-sex schools (Douglas, 1966). Second, acceleration of puberty following exposure to adult males is only partly attributable to pheromones; chemical signals are most effective in combination with visual, auditory, and tactile cues (Dellovade, Hunter, & Rissman, 1995; Solomon et al., 1996; Vandenbergh, 1983; Widowski et al., 1990; Widowski, Porter, Ziegler, & Snowdon, 1992). Direct physical contact appears to be especially important. Third, not all adult males are of equal stimulus value. In mice, acceleration of female puberty occurs only in response to the urine of dominant males (Lombardi & Vandenbergh, 1977). In pigs, boars with high libido are more effective at stimulating female puberty than are boars with low libido (P. E. Hughes, 1994). Fourth, females at different stages of physical development respond differently to exposure to adult males. In gilts, prepubertal exposure to boars is most effective at stimulating puberty, whereas boar exposure beginning at very young ages tends to delay puberty (Izard, 1983). Similarly, only heifers above a certain weight accelerate puberty in response to bull urine, presumably because of immaturity in lighter heifers (Izard, 1983). Prepubertal female mice also accelerate puberty in response to adult male urine; however, this effect is enhanced by previous, preweaning exposure to the urine of other adult males (Caretta, Caretta, & Cavaggioni, 1995). Finally, there may be a synergism between exposure to adult males and experiences of stress. In gilts, a combination of frequent boar exposure and trailer-transport stress was found to be more effective at stimulating puberty than frequent boar contact alone (transport stress alone did not alter pubertal timing; P. E. Hughes et al., 1997). Perhaps the initial increase in gonadotropin that occurs in response to acute stress (Rivier & Rivest, 1991) increases the female's susceptibility to the male effect.

What is the relevance to humans? There is now clear evidence of regulation of women's reproductive functioning by pheromones (Monti-Bloch, Jennings-White, & Berliner, 1998; Sanders & Reinisch, 1990; Stern & McClintock, 1998). For example, controlled experimental studies have shown that exposure to pheromones produced by men's axillary sweat glands reduces variability in women's ovarian cycles (Cutler et al., 1986) and that exposure to pheromones produced by other women's axillary sweat glands alters the timing and length of ovarian cycles (Preti et al., 1986; Stern & McClintock, 1998). These data raise the question: Are there pheromones that accelerate or inhibit pubertal development in human females?

Although no experimental research has directly investigated this question, the possibilities are intriguing. Two lines of inquiry have provided indirect support for the hypothesis that contact with members of one's natal group inhibits girls' pubertal development. First, as reviewed above (see Psychosocial Models of Pubertal Timing: II. Psychosocial Acceleration Theory and Psychosocial Models of Pubertal Timing: III. Paternal Investment Theory), cohesive family relationships and frequency of contact with biological parents are associated with later pubertal timing in daughters. Along these lines, Burger and Gochfeld (1985) have hypothesized that menarche will occur later in girls whose mothers are at

home throughout the day than in girls whose mothers work outside of the home.[12] Second, girls from larger families generally attain menarche later than girls from smaller families (reviewed in Malina, Katzmarzyk, Bonci, Ryan, & Wellens, 1997, see especially Table 6). Although this effect is routinely attributed to SES, later menarche in large families has been documented in a number of well-nourished populations in which SES and age at menarche are uncorrelated (Malina et al., 1997). In total, consistent with the animal literature, family crowding, cohesiveness, and physical interaction may inhibit pubertal development.

The phenomenon of accelerated female pubertal development in response to contact with unfamiliar adult males may also have relevance to humans. Animal studies provide the basis for a set of hypotheses. Girls should be most likely to accelerate pubertal development when (a) an unrelated adult male moves into the home at or around the age of gonadarche, (b) there is substantial direct interaction and physical contact between the girl and the adult male, (c) the male's physiological traits embody high stimulus value (perhaps high testosterone; Vandenbergh, 1983), and (d) there is substantial stress in the family environment. The proposed synergism between exposure to adult males and familial stress is consistent with the work of B. J. Ellis and Garber (2000), as described above (see *Stepfather presence*). Another relevant issue, though unresolved in the animal literature, is whether repeated exposure to the same unrelated adult male or exposure to a series of different unrelated adult males is more effective at stimulating puberty.

It is likely that all four of the preceding conditions are often met in the case of sexual abuse of stepdaughters. Four studies have investigated relations between sexual abuse and timing of pubertal development (Herman-Giddens, Sandler, & Friedman, 1988; Jorm et al., 2004; Romans et al., 2003; Turner, Runtz, & Galambos, 1999), and in each investigation a history of sexual abuse was associated with earlier puberty. Jorm et al. (2004) and Romans et al. (2003) both used large, random community samples, which are useful for establishing effect sizes. Jorm et al. found that girls who were sexually abused in childhood (up to age 16) experienced menarche an average of 6.4 months earlier than girls who were not sexually abused. Romans et al. found that girls who were sexually abused prior to menarche had odds 1.6 times higher of experiencing menarche before age 12 than did girls who were not abused (32% vs. 20%). Of the girls who were sexually abused ($n = 97$), girls whose abuse lasted for more than 1 year had odds 3.5 times higher of experiencing menarche before age 12 than did girls whose abuse lasted for less than 1 year (63% vs. 18%). The odds ratio increased to 5.35 after adjusting for family structure and parent–child relationships. It is important to note, however, that none of these studies was able to establish that sexual abuse occurred prior to puberty. Both Jorm et al. and Turner et al. (1999) used measurement procedures that encompassed some postmenarcheal experiences of sexual abuse. Although Herman-Giddens et al. (1988) and Romans et al. both assessed sexual abuse prior to menarche, early pubertal changes would have already been under way in many of the girls they studied.

In sum, the direction of causation remains an open question. Herman-Giddens et al. (1988) speculated that early development of secondary sexual characteristics may increase the probability of a child becoming a victim of sexual abuse. In Romans et al. (2003), most cases of sexual abuse occurred prior to menarche but during the early stages of the pubertal transition. Consistent with the animal literature reviewed above, it may be that prolonged intimate contact between men and prepubertal or peripubertal girls, particularly contact lasting more than a year, accelerates maturation of the HPG axis. The data of Mekos et al. (1992), as described above (see *Stepfather presence*), are not inconsistent with this hypothesis. Further research is needed.

Criticisms of Psychosocial Acceleration and Paternal Investment Theories

Psychosocial acceleration and paternal investment theories have been challenged in the literature on a number of grounds. The most compelling empirical critique has been offered by behavior geneticists. Theoretical critiques have come from within the fields of evolutionary psychology and biology.

The Behavior Genetic Critique

An important limitation of all of the human research on antecedents of pubertal timing reviewed in this article is that it is not genetically informative. The psychosocial models of pubertal timing presented herein rest on the concept of conditional reproductive strategies; that is, they emphasize environmentally triggered processes that shunt individuals toward given reproductive strategies. An alternative explanation, however, is that individual differences in pubertal timing and associated characteristics represent heritable reproductive strategies, which result from genetic differences. Consider the following two related possibilities.

First, as reviewed below (see Psychosocial Models of Pubertal Timing: IV. Child Development Theory), girls who mature earlier tend to exhibit earlier onset of sexual activity and earlier age of first marriage and first birth. This covariation may occur because early pubertal timing results in precocious sexual and reproductive behavior or because pubertal, sexual, and reproductive timing are genetically correlated traits (Rowe, 2002). Early reproduction in turn is associated with increased probability of divorce and lower quality paternal investment (e.g., Amato, 1996; Bennett, Bloom, & Miller, 1995). Because mothers who are early maturers tend to have daughters who are early maturers (see Sources of Variation in Pubertal Timing), the correlation between family environments and timing of pubertal maturation in girls may be spurious; that is, it may simply be due to genetic transmission of pubertal timing and associated characteristics (e.g., Belsky et al., 1991; Kim & Smith, 1998a; Rowe, 2000a; Surbey, 1990).

The correlational methods used by researchers to examine relations between social environments and pubertal timing cannot rule out this alternative explanation; indeed, Moffitt et al. (1992) embraced just this interpretation upon reporting linkages between early experience and pubertal timing. However, many researchers have incorporated control variables into their analyses to account, at least in part, for possible genetic influences. These controls have included child's initial level of pubertal development (N. B. Ellis, 1991; Graber et al., 1995; Steinberg, 1988), mother's age at menarche (Campbell & Udry, 1995; Graber et al., 1995; Kim & Smith, 1998a; Surbey, 1990), mother's sexual and reproductive

[12] In testing this hypothesis, it would be important to control for father–daughter contact, given that maternal employment may be negatively correlated with paternal involvement.

history (B. J. Ellis & Garber, 2000; Kim & Smith, 1998a; Quinlan, 2003), and daughters' physical characteristics such as weight, percentage of body fat, and biliac diameter (e.g., Campbell & Udry, 1995; Graber et al., 1995; Moffitt et al., 1992). In most cases the observed relations between family environment and pubertal timing have not been meaningfully altered by inclusion of these control variables. Nonetheless, genetically controlled research designs that incorporate environmental measures are greatly needed, as researchers cannot be certain by any means that the controls implemented to date fully take into account biological inheritance.

Second, Comings et al. (2002) have proposed a more specific version of the genetic transmission theory based on a variant of the X-linked androgen receptor gene. According to Comings et al., fathers carry X-linked genes that are associated with aggression and impulsivity, sexual promiscuity, and associated patterns of marital conflict and dissolution. These genes are transmitted to daughters, in whom they are associated with paternal absence, earlier age at menarche, and precocious sexual activity. Comings et al. found support for this theory in molecular genetic research with two clinical samples (males hospitalized for substance abuse, female outpatient volunteers for a weight control program). Jorm et al. (2004), however, found no support for the theory in two epidemiological molecular genetic studies using general population samples. Further research is needed to reconcile these contradictory results, as the current balance of evidence does not yet permit evaluation of the X-linked genetic transmission theory.

Finally, Belsky (2000) has proposed that the environmental and genetic transmission models could both be right but that each applies only to a subset of the population. Specifically, covariation between childhood experiences and timing of puberty may be primarily genetic for some individuals but not for others. In an extensive review of the literature, Belsky (2004) has documented wide variation between children in the extent to which they are affected by particular styles of parenting or other aspects of childrearing (see also Boyce & Ellis, in press). Such variation appears to have a substantial genetic basis (see Caspi et al., 2002, 2003). Indeed, Caspi et al. (2002, 2003) have argued that the very reason why molecular genetic studies, such as those reported above by Comings et al. (2002) and Jorm et al. (2004), so often prove inconsistent in their findings is because gene–environment interactions are likely to be widespread, and sampling from different populations may well lead to different proportions of individuals who are and are not susceptible to a particular environmental experience. In sum, the psychosocial acceleration and paternal investment theories of pubertal timing may apply only to those subsets of the population who are genetically susceptible to rearing influences.

An Evolutionary Theoretical Critique

The psychosocial acceleration and paternal investment theories of pubertal timing have also been challenged on conceptual grounds (Bailey et al., 2000; Rowe, 2000a, 2000b). This challenge concerns the evolutionary logic underlying early experiential calibration of reproductive strategies. Bailey et al. (2000) suggested that paternal investment theory necessitates several rather strong assumptions about ancestral social environments:

> First, in ancestral environments, frequent shifts must have occurred between high and low paternal investment mating systems (respectively, "Dads" and "Cads" [Wilson, 1994]). Such shifts would be necessary for the evolution of such a complex, contingent adaptation. Second, although frequent shifts must have occurred within populations over time, in general, fathers' behavior must have been a reliable indicator of paternal investment at daughter's age of reproduction; cross-generational changes in mating system would disrupt father–daughter signaling. Third, within ancestral breeding populations, men would have needed to be rather homogenous in their sexual strategies (nearly all "Dads" or all "Cads"). Otherwise, there would be little benefit to a daughter drawing inferences about the likelihood of paternal investment from her father's behavior. (p. 538)

This critique has been further articulated by Kanazawa (2001):

> Assume that 50% of men in a society comprises "cads" and the other 50% "dads" (Draper and Harpending, 1982, 1988). Further assume that there is no inherited tendency for girls to mate with one kind or the other; daughters of women who mated with cads are no more likely to mate with cads than those of women who mated with dads. . . . In this situation, if girls from father-absent homes experience early puberty and adopt a more promiscuous reproductive strategy (mating without long-term commitment), then their strategy will be just as likely to be maladaptive as to be adaptive because they will be just as likely to mate with a dad as with a cad. The same is true of girls from father-present homes. If they delay their puberty and avoid sexual promiscuity, their strategy will be just as likely to be maladaptive as to be adaptive because they will be just as likely to mate with a cad as with a dad. Under such circumstances, any evolved tendency to take cues from the mating situations of their mothers, as is posited by the model, will not be selected. (p. 330)

These critiques contend that it would be maladaptive for girls to use childhood exposures to fathers' and mothers' reproductive strategies as a basis for calibrating development of their own reproductive strategies unless there is homogeneity within populations.

Developmental plasticity is necessarily a constrained process. Although it would seem advantageous for individuals to respond to environmental changes quickly, appropriately, and with maximal flexibility throughout their lives, high levels of responsiveness are not always either possible or desirable. Instead, for many phenotypic characteristics, individuals have been selected to register particular features of their childhood environments as a basis for entraining relevant developmental pathways (e.g., Boyce & Ellis, in press; Chisholm, 1999; Shonkoff & Phillips, 2000; West-Eberhard, 2003). As discussed in Boyce and Ellis (in press), there are several reasons to expect early entrainment. I reiterate only one of those reasons here: Many complex adaptations are built during development and cannot be easily rebuilt when environments fluctuate. For example, age at menarche is influenced by programmed patterns of gonadotropin release that are established in utero, when androgen concentrations imprint the fetal HPG axis, and are subsequently modified by fat accumulation during childhood (Cooper, Kuh, Egger, Wadsworth, & Barker, 1996; Koziel & Jankowska, 2002).

The core issue raised by the preceding critiques, however, is whether fathers' and mothers' reproductive strategies provide children with reliable information about the reproductive opportunities and constraints that they are likely to encounter in adulthood. As extensively reviewed by Chisholm (1999), the answer to this question is almost certainly "yes." Familial and ecological conditions in childhood prepare individuals for the sociosexual niche that they are likely to inhabit in adulthood (Belsky et al., 1991).

This preparation occurs internally through personality development and externally through intergenerational transmission of social and economic resources. These transmissions affect the reproductive opportunities and constraints in nonrandom ways, moving children into greater alignment with their parents.

Kanazawa (2001) has studied empirically the nature of information transmitted by parental reproductive strategies. His starting assumption was that father presence versus absence and quality of paternal investment experienced by girls in the home function as microlevel indicators of the degree of monogamy versus polygyny in the society at large. Kanazawa conducted cross-cultural analyses in which he coded for either simultaneous polygyny (pervasiveness of polygyny in legally polygynous societies) or serial polygyny (annual divorce rates in legally monogamous societies). These indices were then correlated with mean age at menarche in each society, after controlling for race, year of study, and population measures of health and welfare (per capita GDP and female literacy rates). Consistent with paternal investment theory, Kanazawa found that menarche occurred earlier in societies characterized by higher levels of simultaneous or serial polygyny. These data are consistent with the proposition that female pubertal timing is responsive to parental reproductive strategies and that these strategies (contrary to Kanazawa's, 2001, own criticism quoted above) provide reliable cues to the macrolevel mating systems that children mature into. Moreover, quality of parental resources and investment prepare children more specifically for their likely position in those mating systems.

Psychosocial Models of Pubertal Timing: IV. Child Development Theory

In the preceding section I argued, in line with Draper and Harpending (1982), Belsky et al. (1991), Chisholm (1999), and others, that children's experiences in and around the family (and particularly childhood exposures to parental reproductive strategies) provide them with reliable information about the reproductive opportunities and constraints that they are likely to encounter at adolescence and beyond. Although this information is almost certainly reliable in a statistical sense, it is far from perfect. Many factors introduce noise into the system: Weather cycles change, periods of feast and famine occur, rapid social changes occur, wars are won and lost, parents and children differ in their sociocompetitive competencies, and so forth. The criticism thus remains that it may be a poor evolutionary choice to calibrate adolescent and young adult reproductive strategies on the basis of childhood experiences that are many years out of date (Rowe, 2000a, 2000b).

A possible resolution to this problem involves a reconceptualization of the function of pubertal timing. Both psychosocial acceleration theory and paternal investment theory conceptualize timing of puberty as part of an integrated reproductive strategy that (a) is responsive to social and ecological conditions in childhood and (b) feeds forward to sociosexual and parental behavior in adulthood. This feed-forward function probabilistically links earlier pubertal timing not only to earlier onset of sexual activity and reproduction but also to a more unrestricted sociosexual orientation characterized by relatively unstable pairbonds, greater number of sexual partners, and less parental investment (Belsky et al., 1991; Chisholm, 1999). Conversely, later pubertal timing is linked probabilistically to later onset of sex and reproduction, a more restricted sociosexual orientation, more pairbond stability, fewer sexual partners, and greater investment in parenting.

By contrast, *child development theory*, as proposed here, conceptualizes timing of puberty as part of an integrated developmental strategy that conditionally alters the length of childhood in response to the composition and quality of family environments. These alterations function to adaptively extend childhood (delay puberty) in high-quality social developmental environments and to shorten childhood (accelerate puberty) in adverse social developmental environments. Child development theory converges with psychosocial acceleration theory and paternal investment theory in its conceptualization of (a) how childhood experiences affect pubertal timing and (b) how pubertal timing affects timing of onset of sexual activity and reproduction, but it diverges from these other theories in its conceptualization of the relation between pubertal timing and qualitative differences in mating and parenting strategies. The key criterion in child development theory is the timing of the pubertal transition from a pre-reproductive state to a reproductive state, that is, the timing of the change in allocation of resources from physical growth to mating and parenting. The theory links quality of family environments to timing of pubertal development and onset of sexual activity and reproduction but does not in turn link these reproductive timing variables to qualitative differences in mating and parenting strategies (e.g., unrestricted vs. restricted sociosexual orientation). According to child development theory, an important function of childhood experience is to adaptively coordinate the length of the pre-reproductive period (pubertal timing) with the value of the child's social developmental environment.

Changes in the length of the pre-reproductive period translate into changes in the timing of the transition to the reproductive period (i.e., puberty). This transition has important implications for social as well as physical development. Adolescence is characterized by a distancing of parent–child relationships and declining parental investment, increased resistance to parental control and information, increased orientation toward peer relationships, and cognitive and emotional reorganization away from the behavioral modes of childhood toward participation in adult social, sexual, and economic activities (Bogin, 1999; Schlegel & Barry, 1991; Steinberg, 1988; Surbey, 1998). Earlier pubertal development, therefore, means an earlier transition away from reliance on parental investment and toward immersion in and dependence on peer and sexual relationships; later pubertal development means the opposite.

The human life history is characterized by lengthy infancy and juvenile periods prior to sexual maturation. Many authors (e.g., Bjorklund & Pellegrini, 2002; Bogin, 1999; Geary, 2002; H. Kaplan, Hill, Lancaster, & Hurtado, 2000) have argued that this prolonged childhood allows an extended period for brain development; increased flexibility of learning; and the time to acquire physical, behavioral, and cognitive competencies (e.g., large body size, child care skills, hunting and food processing skills, socioemotional skills). This accrued reproductive potential presumably translates into increased survival, productivity, and reproductive success in adulthood. The implicit assumption is that the benefits of large body size and accumulated skills and knowledge compensate for the reproductive opportunities lost through prolonged growth. The costs and benefits associated with earlier versus later timing of reproductive maturation were described earlier (see The Life History Approach to Timing of Pubertal Development). Child

development theory posits that the weighting of these costs and benefits varies as a function of the quality of the child's social developmental environment. Specifically, the costs of delayed maturation—increased probability of mortality prior to reproduction, longer generation times, shorter reproductive life spans—are reduced in higher quality environments that facilitate greater development of sociocompetitive competencies. Children should be selected to capitalize on the benefits of high-quality parental investment, and to reduce the costs of low-quality parental investment, by contingently altering the period of growth and development prior to reproductive maturity.

Developmental mechanisms for adjusting timing of pubertal maturation in response to experiences in and around the family may have resulted from a long and recurrent evolutionary history in which (a) different children confronted substantially different rearing environments; (b) low-quality parental investment signaled an environment in which parental care and resources were relatively unreliable and/or not closely linked to variation in reproductive success; (c) earlier pubertal transitions in this context were, on average, associated with greater reproductive success; (d) high-quality parental investment signaled a competitive environment in which variations in offspring quality and success were sensitive to provision of parental care and resources; and (e) later pubertal transitions in this context were, on average, associated with greater reproductive success.

Relations Between Pubertal Timing and Sexual and Reproductive Behavior

As stated above, child development theory differs from psychosocial acceleration theory and paternal investment theory in its downstream predictions about mating and parenting strategies. There are areas of agreement and disagreement. First, all of the theories reviewed in this article converge on the prediction that earlier pubertal timing will be associated with earlier onset of sexual activity and reproduction. This uncontroversial prediction has been tested in dozens of studies and, not surprisingly, has received substantial support. Most investigations have found that earlier timing of pubertal development is associated with earlier ages at first dating, first kissing, and first genital petting (e.g., Flannery, Rowe, & Gulley, 1993; Lam, Shi, Ho, Stewart, & Fan, 2002; B. C. Miller, Norton, Fan, & Christopherson, 1998), earlier ages at first sexual intercourse (e.g., Bingham, Miller, & Adams, 1990; B. C. Miller et al., 1997; Phinney, Jensen, Olsen, & Cundick, 1990), and higher rates of adolescent pregnancy (e.g., Manlove, 1997; Romans et al., 2003; Udry, 1979). There is also extensive cross-cultural evidence, based on natural fertility populations, that earlier age at menarche is strongly associated with earlier age at first birth (e.g., Ann, Othman, Butz, & DaVanzo, 1983; Borgerhoff Mulder, 1989a; Udry & Cliquet, 1982). Along these lines, Rosenberg (1991) found a positive correlation between age of menarche and age at first birth in Norway over the period from 1830 to 1960, and the farther back in time, the stronger the correlation.

Second, psychosocial acceleration and paternal investment theory, but not child development theory, predict that variation in pubertal timing will be associated with variation in sociosexual orientation, pairbond stability, partner number, and orientation toward parental investment. Relatively little is known about the effects of pubertal timing on these dimensions of mating and parenting. A search of the literature revealed only six relevant investigations. Five of these studies explicitly analyzed the relation between age at menarche and number of sexual partners (Helm & Lidegaard, 1989; Hoier, 2003; Kim & Smith, 1998b; Kim et al., 1997; Mikach & Bailey, 1999); none found a significant association. Kim et al. (1997) reported that earlier age at menarche was associated with greater age differences between young women and their first sexual intercourse partner, but Kim and Smith (1998b) failed to replicate this relation. Likewise, Kim and Smith (1998b) reported that earlier age of menarche was associated with greater number of boyfriends, but Kim et al. (1997) failed to replicate this relation. Furthermore, Mezzich et al. (1997), analyzing a clinical sample of teenage girls diagnosed with substance use disorders, found that earlier age at menarche was associated with higher levels of risky sexual behavior. This finding is difficult to interpret, however, because the measure of risky sexual behavior composited timing variables (e.g., occurrence of first pregnancy) with promiscuity variables (e.g., multiple sexual partners).

Hoier (2003) has conducted the most extensive investigation of the relations between age at menarche and theoretically relevant mating and parenting variables. Her study included 3 measures of age at onset of sexual activity (age at first petting, age at first romantic relationship, and age at first sexual intercourse) and 13 other measures of mating and parenting (lifetime number of sexual partners, number of sexual partners per year, incidence of sexual infidelity, inclination toward choosing partners of poor match, sociosexual orientation, preference for a mate who displays indicators of good parenting, preference for a mate who displays indicators of good genes, attitudes toward sexual fidelity, idealization of romantic relationships, desired number of future sexual partners over the next year, desired number of future sexual partners over the next 10 years, desired number of future sexual partners over the rest of one's life, and attitudes toward investment in children). Consistent with the literature reviewed above, all 3 of the age at onset variables had statistically significant associations with age at menarche in the predicted direction. By contrast, only 1 of the 13 other indices was significantly associated with age at menarche. Thus, in the same sample, earlier age at menarche was associated with earlier onset of dating and sexual activity but was not associated with other theoretically relevant facets of mating and parenting.

In sum, although earlier timing of puberty clearly predicts earlier onset of major forms of sexual experience and reproduction, there is currently no empirical basis for the hypothesis that earlier timing of puberty leads to a more unrestricted sociosexual orientation, unstable pairbonds, greater number of sexual partners, or lower parental investment. Admittedly, more research is needed, given that the small number of studies reviewed above mostly relied on retrospective data and convenience samples. My point is not that these limited investigations falsify the hypothesis but rather that no extant research has supported it. Instead, the evidence to date concurs with the delimited focus of child development theory on the timing of sexual and reproductive milestones—age at first sexual intercourse, age at first pregnancy, age at first birth—as the reproductive sequelae of pubertal timing. Contrary to the psychosocial acceleration and paternal investment theories, the data do not currently support expanding these timing variables to include other qualitative aspects of mating and parenting strategies, independent of age at onset.

It is important to note that child development theory does not challenge the core idea, which is fundamental to attachment theory and social learning theory as well as psychosocial acceleration theory and paternal investment theory, that childhood experience influences the development of qualitative dimensions of sexual behavior and parental investment. Rather, child development theory posits that timing of pubertal development is not an intervening factor in these relations. That is, pubertal timing is not a generative causal mechanism through which experiences in and around the family influence sociosexual orientation, pairbond stability, partner number, parental investment strategies, and so forth.

Summary and Evaluation

Child development theory constitutes a revision of some of the logic and predictions of psychosocial acceleration theory and paternal investment theory. This revision potentially addresses three anomalies in these earlier theories. First is the problem of long-term inference. As Rowe (2000a, 2000b) has argued in his critique of the psychosocial acceleration and paternal investment theories, in an uncertain world where conditions can greatly improve or worsen over time, it is a risky proposition for young children to use parental behavior as a guide to the future some 10 to 15 years later when they will be of reproductive age. Child development theory avoids the problem of long-term inference by reconceptualizing the function of childhood experience in relation to timing of sexual development. According to the theory, an important function of childhood experience is to adaptively coordinate the duration of childhood (pubertal timing) with the value of the child's social developmental environment. Timing of sex and reproduction are linked to timing of puberty only because one follows the other (i.e., puberty marks the transition from the pre-reproductive to the reproductive phase of the human life cycle). The theory requires no long-term inferences about the future; the female child is adjusting the timing or tempo of maturation to capitalize on the benefits, or mitigate the costs, of extant qualities of parental investment and other social resources in and around her family of origin.

Second is the problem of shared environmental variance. As discussed earlier (see Sources of Variation in Pubertal Timing), behavior genetic research has converged on the conclusion that at least half of the variance in age at menarche is genetic and that the rest of the variance is attributable to nonshared environmental effects and measurement error. Although I have criticized these heritability estimates, argued that there are shared environmental effects, and have suggested that the independent variables posited by the current psychosocial models of pubertal timing should have both shared and nonshared environmental effects, the relative paucity of shared environmental variance remains an issue. Bailey et al. (2000) contended that if father absence and other facets of parental reproductive strategies provide reliable information to one sibling about future mating conditions, then they should also provide reliable information to other siblings; that is, there should be shared environmental effects. However, even if it turns out that shared environmental effects are weak, this does not challenge child development theory because children are not assumed to be inferring the macrolevel qualities of the mating system that they will encounter at reproductive age. Rather, children are inferring microlevel qualities of their own social developmental environments. Because "family environments" in fact constitute multiple microenvironments inhabited by different siblings (Sulloway, 1996), child development theory is consistent with the predominance of nonshared environmental influences on pubertal timing.

Third, the absence of relations between pubertal timing and qualitative aspects of mating and parenting strategies, independent of age at onset of sexual and reproductive events, poses an anomaly for psychosocial acceleration theory and paternal investment theory but not for child development theory.

Conclusion

What are the nature of environmental influences on timing of pubertal maturation in girls? If that question had been asked 15 years ago, before the application of life history theory to human sexual development, the answer would have been very different from the one presented herein. The answer provided in a 1988 review, for example, included weight and body mass, intensity and duration of exercise, nutrition, physical illness, number of children in the family, and altitude (Brooks-Gunn, 1988). The notion that social experiences influence something as biological and presumably genetic as pubertal timing was not taken seriously, especially among psychologically minded students of human development. That changed with the publication of psychosocial acceleration theory by Belsky et al. in 1991, which advanced uncanny predictions about relations between family processes and pubertal timing. That theorizing stimulated the major body of research and theory reviewed in this article. Life history theory provided the framework—missing from previous developmental theories—for conceptualizing psychosocial influences on timing of pubertal development.

From a life history perspective, there is no single answer to the question of when puberty should occur. Although genotypic effects on timing of pubertal development are substantial, these effects are probabilistic and are best conceptualized as coding for a reaction norm. Because different points along the spectrum of pubertal timing are characterized by different fitness costs and benefits and trade-offs between them, natural selection is unlikely to favor genetically canalized developmental mechanisms that systematically bias individuals toward either earlier or later pubertal maturation. Rather, selection can be expected to favor adaptive developmental plasticity in response to particular ecological conditions. The critical questions then become, When should individuals reach sexual maturity? and What are the relevant developmental experiences and environmental cues that bias individuals (or at least that subset of individuals who are susceptible to environmental influences) toward relatively early versus late pubertal development?

There can be little doubt that energetics play a key role in determining timing of pubertal maturation. Variation in the median menarcheal age across human populations, which ranges from about 12.0 years to 18.5 years, cannot possibly be explained without reference to energy availability. Children who experience chronically poor nutritional environments, whether assessed indirectly according to SES or directly in dietary studies, tend to experience relatively late pubertal development. An intervening endocrine mechanism may be low levels of pituitary gonadotropins. These data provide strong support for the theory that natural selection has favored physiological mechanisms that track variation in resource availability and adjust physical development to match that variation.

Energetics theory further conceptualizes pubertal timing as a bioassay of chronic childhood conditions. Ellison (1990, 1996, 2001) posited that females use this bioassay to establish lifetime set points for reproductive functioning. This leads to the hypothesis that girls who experience relatively early sexual maturation have greater reproductive capacity than their later maturing peers. This hypothesis has not been supported. Although earlier age at menarche has been found to predict higher levels of ovarian hormonal functioning and earlier reproductive onset, early puberty does not translate into higher reproductive functioning. Compared with girls whose ages at menarche are in the average range for their population, early-maturing girls do not have shorter latencies between menarche and regular menstrual cycling, are not more successful at maintaining pregnancies that culminate in live birth, are not more successful at promoting fetal growth, and are not more fecund or reproductively successful.

Life history theory conceptualizes timing of pubertal maturation as a trade-off in distribution of metabolic resources toward different strategies of reproduction. Early reproductive development biases allocation of resources toward short-term (current) reproduction and greater number of offspring, whereas later reproductive development biases resources toward long-term (future) reproduction and greater fitness of offspring. Earlier pubertal development in girls is associated with earlier age at first sexual activity and reproduction on the one hand but perhaps lower offspring quality on the other, as suggested by the literatures on fetal wastage and fetal growth. In and of itself, timing of pubertal development is not an indicator of reproductive capacity. Rather, consistent with life history theory, timing of puberty is an indicator of different trade-offs in reproductive strategies.

The basic logic of energetics theory has also been generalized to the psychosocial domain. Theorists such as E. M. Miller (1994) and MacDonald (1999) have hypothesized that adverse physical or social conditions, whether experienced as chronically low energy availability or psychosocial stress, should cause animals in *K*-selected species to delay pubertal development and reproduction until predictably better times. This stress-suppression theory has been supported by neurophysiological research linking activation of the stress-response systems to suppression of the HPG axis. Primate studies investigating the stress-suppression hypothesis, however, have examined only the effects of stress on ovarian functioning in mature animals and have not examined its effects on pubertal maturation in younger animals. Overall, the experimental research linking psychosocial stress to delays in pubertal development is scant and inconclusive. Nonetheless, human clinical data on psychosocial dwarfism as well as demographic studies tracking increases in age at menarche under war conditions are consistent with stress-suppression theory and suggest that severe psychological stress can inhibit pubertal development.

A key direction for future research involves untangling the effects of physical and socioemotional stressors on timing of puberty. Hulanicka's (1999; Hulanicka et al., 2001) research on Polish school girls is especially informative in this regard. Within the same samples, poverty was found to forecast later pubertal development, and family dysfunction predicted earlier development. These data suggest that physical and socioemotional stressors have independent (and perhaps countervailing) effects on pubertal timing. Coall and Chisholm (2003) have proposed that the effects of physical and socioemotional stressors on pubertal timing are hierarchically ordered, whereby pubertal timing is contingent firstly on health and nutrition and, when these are adequate, secondly on socioemotional conditions.

Psychosocial acceleration theory and paternal investment theory share the core assumption that humans have evolved to be sensitive to specific features of their early childhood environments and that exposure to different environments biases children toward acquisition of different reproductive strategies. Psychosocial acceleration theory posits that girls whose experiences in and around their families of origin are characterized by relatively high levels of interpersonal stress (e.g., negative and coercive family relationships, lack of positive and supportive family relationships) develop in a manner that speeds rates of pubertal maturation, accelerates sexual activity, and orients the individual toward relatively unstable pairbonds and lower levels of parental investment. Paternal investment theory predicts these same outcomes in response to family adversity, but it proposes a special role for fathers and other men in regulation of girls' sexual development. Both theories have received reasonable empirical support. Converging evidence from a number of methodologically sound studies has indicated that (a) girls from father-absent homes tend to experience earlier pubertal development than do girls from father-present homes, and the earlier father absence occurs, the greater the effect; (b) better marital quality is associated with later pubertal development in daughters; and (c) greater parent–child warmth and cohesion predicts later pubertal development. In addition, there is consistent evidence that quality of fathers' investment in the family uniquely predicts timing of pubertal development in daughters independently of other aspects of the family ecology. Not all tests of the theory have been favorable, however. The hypothesis that parent–child conflict and coercion accelerate pubertal development has received mixed support.

An extension of Boyce and Ellis's (in press) evolutionary-developmental theory of stress reactivity was proposed to account for both inhibiting and accelerating effects of psychosocial stress on timing of pubertal development. Boyce and Ellis posited that both highly protective and acutely stressful childhood environments cause up-regulation of stress reactivity systems. If this up-regulation inhibits maturation of the HPG axis, then there should be U-shaped curvilinear relations between levels of support and social resources versus stress and adversity in early childhood environments and timing of puberty (see Figure 1). This account, which concurs with neurophysiological research documenting suppressive effects of stress on the reproductive axis, potentially reconciles important contradictions in the literature by explaining why late pubertal development disproportionately occurs in both highly supportive and extremely stressful socioemotional environments.

There are likely to be multiple pathways through which family relationships or family composition affect pubertal timing. The possible role of cortisol was discussed. Another possibility, consistent with an extensive animal literature, is that contact with members of one's natal group inhibits pubertal development in girls, whereas exposure to unfamiliar men accelerates it. The intervening mechanism is hypothesized to be pheromones, which are encoded through olfactory channels and can impact reproductive endocrinology either alone or in combination with visual, auditory, and tactile stimuli from conspecifics. Direct physical contact appears to be especially important. The data on sexual abuse and pubertal maturation are not inconsistent with this proposed mechanism.

Psychosocial acceleration theory and paternal investment theory have been criticized on several grounds. The most severe criticism is methodological: Correlational research methods that are not genetically informative have been used to test these theories. The correlation between family environments and timing of pubertal maturation in girls could be spurious; that is, it could be due simply to genetic transmission of pubertal timing and associated characteristics (i.e., selection effects). Although many studies have incorporated appropriate control variables to account for possible genetic influences, correlational methods cannot rule out selection effects. Experimental research designs are needed to test for the causal influence of family environments on pubertal timing. This could be accomplished by incorporating pubertal development measures into the many randomized, longitudinally designed early intervention trials that have been implemented to promote more harmonious or stable family relationships. Other criticisms of psychosocial acceleration theory and paternal investment theory include the absence of shared environmental effects on pubertal timing in behavior genetic studies, the questionable (but not necessarily totally flawed) logic of basing adult reproductive strategies on early childhood experiences, and the lack of associations between pubertal timing and other aspects of reproductive strategies specified by the theories.

A proposed revision of these theories—child development theory—addresses these latter three criticisms. Child development theory conceptualizes timing of puberty as part of an integrated developmental strategy that conditionally alters the length of childhood in response to the composition and quality of family environments. These alterations function to adaptively extend childhood (delay puberty) in high-quality social developmental environments and to shorten childhood (accelerate puberty) in adverse social developmental environments. Child development theory is consistent with the predominance of nonshared environmental influences on pubertal timing, does not require children to use parental behavior as a guide to the future some 10 to 15 years later when they will be of reproductive age, and links timing of puberty to timing of sex and reproduction but not to other qualitative aspects of reproductive strategies (e.g., orientation toward long- vs. short-term mating or high- vs. low-investment parenting).

Much remains to be learned about the effects of family environments on pubertal timing. Most critical is the need for genetically controlled research designs that incorporate environmental measures. Neurophysiological studies that test for intervening mechanisms are also greatly needed. Finally, more careful attention must be paid to the nature of psychosocial effects on pubertal timing (e.g., sensitive period and other age effects, effects of chronic vs. acute exposure to stressors, curvilinear relations, interactions between socioemotional and physical stressors). Despite these complexities, it is my hope that the current review leads to new knowledge about the causes of pubertal timing in girls and that this knowledge is ultimately helpful in predicting and controlling the pubertal transition.

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