

The Development of Reproductive Strategy in Females: Early Maternal Harshness → Earlier Menarche → Increased Sexual Risk Taking

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To test a proposition central to J. Belsky, L. Steinberg, and P. Draper's (1991) evolutionary theory of socialization—that pubertal maturation plays a role in linking early rearing experience with adolescent sexual risk taking (i.e., frequency of sexual behavior) and, perhaps, other risk taking (e.g., alcohol, drugs, delinquency)—the authors subjected longitudinal data on 433 White, 62 Black, and 31 Hispanic females to path analysis. Results showed (a) that greater maternal harshness at 54 months predicted earlier age of menarche; (b) that earlier age of menarche predicted greater sexual (but not other) risk taking; and (c) that maternal harshness exerted a significant indirect effect, via earlier menarche, on sexual risk taking (i.e., greater harshness → earlier menarche → greater sexual risk taking) but only a direct effect on other risk taking. Results are discussed in terms of evolutionary perspectives on human development and reproductive strategy, and future directions for research are outlined.

Keywords: reproductive strategy, puberty, parenting, risk taking

It is well established that earlier onset of puberty among females is associated with greater sexual risk taking (Ellis, 2004; Steinberg, 2008). Many investigations have found that earlier age of menarche, or some other index of pubertal development, is associated with earlier age of first dating, first kissing, and first genital petting (e.g., Flannery, Rowe, & Gulley, 1993; Lam, Shi, Ho, Stewart, &

Fan, 2002; Miller, Norton, Fan, & Christopherson, 1998); earlier age at first sexual intercourse (e.g., Bingham, Miller, & Adams, 1990; Miller et al., 1997; Phinney, Jensen, Olsen, & Cundick, 1990); and higher rates of adolescent pregnancy (e.g., Manlove, 1997; Romans, Martin, Gendall, & Herbison, 2003; Udry, 1979). Cross-cultural studies of fertility further indicate that earlier age of

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menarche is strongly associated with earlier age of first pregnancy and birth (e.g., Ann, Othman, Butz, & DaVanzo, 1983; Borgerhoff Mulder, 1989; Udry & Cliquet, 1982).

The fact that virtually all research linking timing of puberty with sexual behavior derives from investigations that initiate data collection sometime late in childhood or adolescence, or even thereafter, raises a fundamental developmental question regarding the evidence just considered: Is the accelerating effect of early puberty on the onset and frequency of sexual behavior itself part of a developmental process set in motion well before sexual maturation? This report addresses this query by testing a unique prediction central to Belsky, Steinberg, and Draper's (BSD; 1991) evolutionary theory of socialization, which recast much of child and adolescent development and even functioning in young adulthood in reproductive-strategy perspective.

BSD Theory

BSD theory grew out of Draper and Harpending's (1982) anthropological effort to reinterpret, in evolutionary-biological terms, documented links between father absence in girls' childhood and promiscuous sexual behavior in girls' adolescence. It specifically sought to advance a hypothesis derived from an evolutionary analysis of development that could not be generated from prevailing perspectives and that would be consistent with Draper and Harpending's (1982) reproductive-strategy thinking; this was deemed important, given the fact that social-learning theory (Bandura, 1977), life-course theory (Elder, 1981), and attachment theory (Bowlby, 1969) could all account for effects of father absence without appealing to Darwinian processes. Thus, BSD theory uniquely and originally hypothesized that pubertal maturation played a previously unrecognized role in linking early rearing experiences with subsequent mating and parenting.

BSD theory sought to extend evolutionary-biological analysis beyond father absence to a broader array of early developmental experiences, especially those involving family factors and processes; indeed, the first 5–7 years of life were conceptualized as a sensitive period for the contextual regulation of reproductive strategy, including pubertal development. Thus, BSD regarded a variety of distal and proximate rearing conditions, including parenting and parent-child relationships and not just father absence, as developmentally influential.

Drawing on, but further developing, the same evolutionary-biological, life-history framework that Draper and Harpending (1982) advanced, BSD postulated that natural selection shaped individuals to mature earlier than would otherwise be the case under conditions of heightened contextual risk and uncertainty, thereby setting the stage for earlier sexual debut, more promiscuous mating, and the bearing of more offspring, along with more limited parental investment. The evolutionary argument was that natural selection shaped individuals to respond with accelerated development when experiences early in life conveyed to the developing child that the future was precarious, that others could not be trusted, and that intimate relations were not enduring—for two reasons. First, given evident risks, slower physical maturation would increase the likelihood of the individual dying before mating and bearing offspring. Second, given the same risks, developmental opportunities for offspring, including survival, could be reduced. This would make it more risky, in terms of passing on

genes to future generations (the fundamental evolutionary imperative), to mature later and bear few rather than many offspring. In sum, in the face of risks to both the child and its future progeny, maturing quickly and breeding promiscuously would enhance reproductive fitness more than would delaying development, mating cautiously, and investing heavily in parenting. The latter strategy, in contrast, would make biological sense, for virtually the same reproductive-fitness-enhancing reasons, under conditions of contextual support and nurturance.

Evidence

In direct response to BSD's prediction linking early family rearing experiences with biological maturation, a good deal of research has been conducted over the past 20 years in an effort to test this core proposition. In a comprehensive review of research on female pubertal development, Ellis (2004, pp. 935–936) concluded that “empirical research has provided reasonable, though incomplete” support for BSD theory. Although Ellis noted that “there is converging evidence . . . that greater parent-child warmth and cohesion is associated with later pubertal development,” he went on to observe that “the proposed accelerating effect of parent-child conflict and coercion on pubertal development is yet to be clearly established.”

In the time since his review appeared, Ellis has published additional findings consistent with BSD. One longitudinal study found that a composite index of family nonsupportiveness during the preschool years—which included measures of authoritarian parenting and negative family relationships—was associated with advanced adrenarcheal status at age 7 and more mature secondary sex characteristics in 5th grade (~10 years; Ellis & Essex, 2007). A second study, employing a within-family sibling design that elegantly controlled for genetic and environmental confounds, showed that family disruption and, especially, father psychological disturbance (hypothesized to index problematic father-daughter relationships) predicted earlier age of menarche (Tither & Ellis, 2008). Costello, Sung, Worthman, and Angold (2007) discovered in an analysis of data gathered from the Great Smoky Mountain Study that maltreated girls reached pubertal maturity 8 months earlier than did nonmaltreated girls. Of particular importance to the present effort are findings based on data from the NICHD Study of Early Child Care and Youth Development showing that maternal harshness before the start of school predicted earlier age of menarche (Belsky, Steinberg, Houts, Friedman, DeHart, et al., 2007).

Current Inquiry

Given the available human evidence linking early family experiences with female pubertal timing and linking pubertal timing with sexual behavior, we sought to determine—in this longitudinal extension of the aforementioned NICHD study research by Belsky et al. (2007)—whether a chain of causation can be modeled with nonexperimental data, whereby harsh maternal parenting measured in early childhood contributes to early menarche and early menarche contributes to sexual risk taking between the ages of 14 and 15. It is important to note that maternal harshness is the exclusive focus of this inquiry, not because it holds a privileged position in BSD theory (see above) or its derivatives (e.g., Ellis, 2004) but for empirical reasons. Although BSD theory highlights a variety of

environmental influences and not just ones restricted to maternal behavior or even harsh parenting, in the earlier investigation of rearing effects on pubertal development (Belsky et al., 2007), which the current inquiry seeks to extend, only maternal harshness predicted earlier menarche among factors measured in the first 5–7 years of life; recall that, according to BSD, this is the developmental period during which reproductive strategy is regulated by developmental experience, including family relations. Thus, because father absence, observed maternal sensitivity, and reported mother–child and father–child closeness and conflict did not successfully predict age of menarche in the NICHD sample, as they have in other studies (Ellis, 2004), they could not be used here to test the critical and unique prediction of BSD theory, namely, that the effect of early rearing on sexual behavior in adolescence will be, at least in part, indirect and will involve pubertal timing (i.e., greater maternal harshness → earlier age of menarche → greater sexual risk taking). Even though it was the case that father harshness measured in middle childhood also “predicted” pubertal timing in the prior study, this parenting measure is not included here because, as Belsky et al. (2007) noted, it remains very possible that such an apparent effect of fathering on puberty actually reflects the reverse, given the timing of measurements made in the NICHD study. In any event, readers should be cautioned that even though only the indirect effect of early maternal harshness on sexual risk taking is evaluated in this work specifically designed to test a critical prediction of BSD theory, this should not be taken to mean that maternal harshness is the only rearing experience or environmental factor that could influence pubertal timing and, thereby, sexual behavior, or that is hypothesized to do so, according to BSD theory.

In addition to focusing upon sexual risk taking, the present inquiry investigates other risk taking, namely, use of alcohol, tobacco, or other drugs and engagement in theft and violence. Even though BSD theory is a theory of reproductive strategy and therefore emphasizes sexual behavior, pair bonding, and parental investment, it also posits, like other psychological theories of development, that psychological dispositions are shaped by rearing experiences. In particular, the accelerated reproductive strategy fostered by conditions of risk and uncertainty was hypothesized to promote an opportunistic, advantage-taking orientation toward others, whereas the slower developing reproductive strategy induced by more supportive and harmonious rearing conditions was theorized to encourage a mutually beneficial, reciprocal orientation toward others. Thus, it seemed reasonable to speculate that maternal harshness, by promoting opportunistic advantage taking (which goes unmeasured in this inquiry), could, perhaps via early pubertal development, also contribute to other forms of risk taking. Consistent with this view are data showing that girls who mature earlier are more likely than their later maturing age-mates to smoke and to drink alcohol (e.g., Dick, Rose, Viken, & Kaprio, 2000; Stice, Presnell, & Bearman, 2001; Wichstrom, 2001) and to engage in delinquent activity (Piquero & Chung, 2001). A more theoretical reason to focus upon nonsexual risk taking is the opportunity it affords to evaluate the boundaries of BSD theory. Should it prove to be the case that the hypothesized indirect effect of early maternal harshness on risk taking, via pubertal timing, is restricted to sexual risk taking, this would be evidence for a narrow interpretation of the theory. Should such an indirect effect emerge

in the case of other risk taking, too, a broader reading would seem appropriate.

Method

Participants

Participants were a subset of the families in the NICHD Study of Early Child Care and Youth Development. Families in the study were recruited during the first 11 months of 1991 from 24 hospitals in the vicinity of 10 data collection sites (Charlottesville, VA; Irvine, CA; Lawrence, KS; Little Rock, AR; Madison, WI; Morganton, NC; Philadelphia, PA; Pittsburgh, PA; Seattle, WA; and Wellesley, MA). A total of 8,986 women who gave birth during selected 24-hr periods and their infants were screened in the hospital for participation in the study.

Mother–newborn dyads were excluded from the study if the mother was under 18 years of age, did not speak English, had acknowledged substance abuse, was too ill to participate, was placing her infant for adoption, or refused the hospital screening interview or a follow-up telephone call 2 weeks later; if the infant had serious medical complications or was part of a multiple birth; or if the family lived more than an hour’s drive from the lab site, planned to move from the area within 1 year, lived in a neighborhood deemed by police too unsafe for visitation, or was enrolled in another study. A total of 5,416 families met the eligibility criteria. Study participants were selected from among eligible families based on conditionally random sampling to ensure that the sample would include at least 10% single-parent households, 10% mothers with less than a high school education, and 10% ethnic minority mothers. Recruitment and selection procedures are described in detail in previous study publications (e.g., NICHD Early Child Care Research Network, 1997) and on the study website (<http://secc.rti.org>).

In total, 1,364 families with healthy newborns were ultimately enrolled in the study, with approximately equal numbers of families at each site. The study sample was demographically similar to the population of families with young infants in the communities from which it was recruited. The analysis sample for the current report included all White ($n = 433$, 82%), Black ($n = 62$, 12%), and Hispanic ($n = 31$, 6%) females who had data on at least one of the measurements used in the research presented herein. These 526 females differed from the 100 White ($n = 69$), Black ($n = 22$), and Hispanic ($n = 9$) females not included in the analysis in terms of maternal education and presence of a partner in the home at birth. Girls in the sample had mothers with more education ($M = 14.52$ years, $SD = 2.43$ vs. $M = 13.54$ years, $SD = 2.63$), $F(1, 624) = 13.36$, $p = .0003$, and their mothers were more likely to have a partner in the home when the girl was born (88.0% vs. 71.0%), $\chi^2(1, N = 626) = 19.42$, $p < .0001$. Females in the sample were more likely to be White and less likely to be Black or Hispanic than were females not in the sample, $\chi^2(1, N = 626) = 9.66$, $p < .008$. No differences were noted in income-to-needs when girls were 6 months old ($M = 3.85$, $SD = 3.15$ vs. $M = 3.38$, $SD = 3.82$), $F(1, 588) = 1.27$, $p = .26$.

Procedures and Measures

Three sets of measurements central to this report and pertaining to parenting, pubertal development, and risk taking were obtained

when children were, respectively, 4.5 years of age, between 9.5 and 15 years of age, and 15 years of age.

Maternal harshness was assessed when children were 4.5 years of age. Mothers completed a questionnaire assessing parenting strategies from which an internally consistent, 10-item measure of maternal harshness was derived (Cronbach's $\alpha = .67$; Shumow, Vandell, & Posner, 1998). Mothers who scored high on harsh control spanked their child for doing something wrong, expected their child to obey without asking questions, expected the child to be quiet and respectful when adults were around, regarded respect for authority as the most important thing for the child to learn, believed praise spoiled the child, and did not give lots of hugs and kisses.

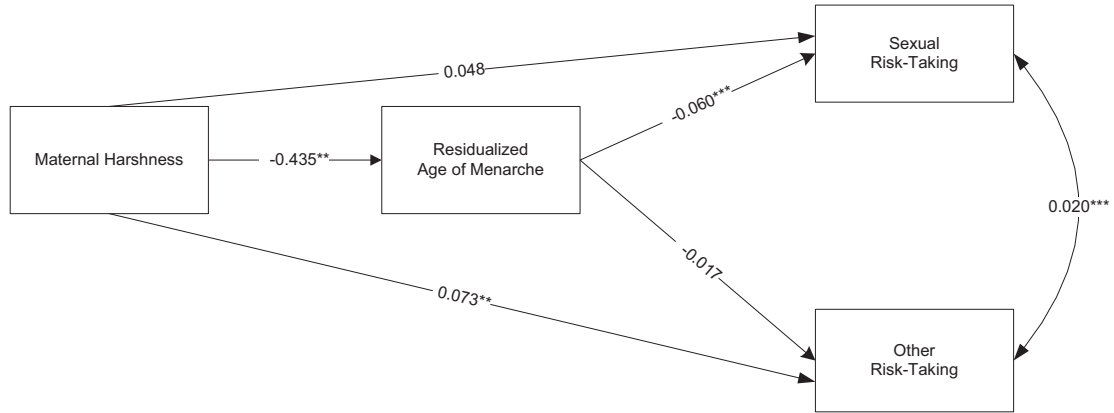
Age of menarche was determined during yearly physical exams between the ages of 9.5 and 15 years by asking girls whether they had begun to menstruate and, if so, their age at their first menstrual period (in years and months). Mothers were also queried about their daughter's first menstrual period, and these data were used if information from the girls was missing. (At age 15, only 3 girls reported not having begun menstruating. They were dropped from the current analysis.) Mothers also reported their own age of menarche in years and months. Following Belsky et al. (2007) and other state-of-the-art studies in the field (e.g., Ellis & Essex, 2007), daughter's age of menarche was adjusted (i.e., residualized) for maternal menarcheal age in the statistical analysis to be presented in an attempt to (partly) control for genetic effects on daughters' timing of puberty, given extensive evidence that timing of puberty is heritable (e.g., Rowe, 2002; Treloar & Martin, 1990). To the extent that mother's own age of menarche was influenced by the maternal harshness the mother received while growing up and that such rearing practices are intergenerationally transmitted, this produces a more conservative test of the hypothesis at hand.

Sexual and other risk taking were assessed at age 15 when adolescent participants completed a survey using audio computer-assisted self-interview. Risk-taking survey items were drawn from instruments used in prior studies of adolescents that clearly document the validity of self-reports by adolescents of their sexual behavior, alcohol and drug use, and delinquent behavior (Halpern-Felsher, Biehl, Kropp, & Rubinstein, 2004; Halpern-Felsher, Cornell, Kropp, & Tschann, 2005; King & Chassin, 2007). For sexual risk taking, the measure included four items assessing the number of times the adolescent had oral sex, had vaginal sex, had been diagnosed with an STD, or had been pregnant or gotten someone pregnant over the past year (e.g., Halpern-Felsher, et al., 2005), as these items on the risk-taking inventory were collectively considered to be markers of earlier onset of sexual behavior and had been linked with earlier pubertal maturation in past work. For other risk taking, adolescents responded to 36 items assessing the extent to which, over the past year, they had used alcohol, tobacco, or other drugs; behaved in ways that threatened their own safety (e.g., ridden in a vehicle without the use of seat belts); used or threatened to use a weapon; stolen something; or harmed property. Responses for both measures were made on a 3-point scale (*never*, *once or twice*, and *more than twice*). For each measure, ratings were summed across component items and then subjected to square root transformation to reduce skew and kurtosis. Internal consistency reliabilities (standardized alpha) for sexual and other risk taking in this sample were 0.59 and 0.88, respectively.

Data Analysis

A path analysis using MPlus (Version 5.1) was conducted to examine associations linking early parenting to daughter's age of menarche, and, subsequently, to sexual and other risk taking at age 15, as well as direct links between early parenting and risk taking (see Figure 1). The model was run using full information maximum likelihood (FIML) to account for missing data in one or more of the variables under consideration. FIML under the assumption of missing at random (MAR) is considered the "practical state of the art" (Schafer & Graham, 2002, p. 173) for dealing with missing data. Although the assumption of MAR is not testable, tests for the stronger assumption of missing completely at random (MCAR) are available (Little, 1988). The test of the MCAR assumption suggests that evidence against random missingness is weak for the sample as a whole, $\chi^2(15, N = 626) = 24.64, p = .06$; in the Black subsample, $\chi^2(11, N = 84) = 3.66, p = .98$; and in the Hispanic subsample, $\chi^2(7, N = 40) = 7.22, p = .41$. In the White subsample, the assumption of MCAR may not hold, $\chi^2(14, N = 502) = 25.75, p = .03$. Further investigation, however, found no differences in the means of the observed values as a function of data being missing on other variables: for example, means of those missing residualized menarche for maternal harshness ($M = 2.11, SD = 0.32$), sexual risk taking ($M = 0.09, SD = 0.25$), and other risk taking ($M = 0.33, SD = 0.20$) versus means of those not missing residualized menarche for maternal harshness ($M = 2.06, SD = 0.33$), sexual risk taking ($M = 0.09, SD = 0.25$), and other risk taking ($M = 0.33, SD = 0.19$); $F(1, 414) = 2.35, p = .13$; $F(1, 377) = 0.03, p = .86$; and $F(1, 378) = 0.04, p = .84$, respectively. This result suggests that the assumption of MAR is not unfounded.

Two nested models were tested to determine whether the processes being examined worked similarly for the three racial/ethnic groups (i.e., White, Black, Hispanic). The first model allowed all direct and indirect paths to be freely estimated in each racial/ethnic group, $\chi^2(2, N = 526) = 7.33, p = .03$, comparative fit index (CFI) = .95, Tucker-Lewis Index (TLI) = .57, root-mean-square error of approximation (RMSEA) = .12, 95% confidence interval (CI) = 0.04–0.22. The second model constrained the various paths to be equal across the three racial/ethnic groups, $\chi^2(12, N = 526) = 14.38, p = .28$, CFI = .98, TLI = .97, RMSEA = .03, 95% CI = 0.00–0.09. Adding the constraints did not significantly decrease model fit, $\Delta\chi^2(10, N = 526) = 7.05, p = .72$; thus, the constrained model was retained and is interpreted in the Results. It should be noted, however, that the lack of difference between the models should not be interpreted as evidence that the pathways are equivalent across the three racial/ethnic groups, because we had limited numbers of participants and reduced power in the Black ($n = 62$) and Hispanic ($n = 31$) subsamples. (It is for this reason that separate estimates for racial/ethnic subgroups are not included in Figure 1; see Table 2 for race/ethnic-specific estimates.) A model run with age of menarche rather than, as reported in Results, residualized age of menarche fit equally well, $\chi^2(12, N = 526) = 11.43, p = .49$, CFI = 1.00, TLI = 1.01, RMSEA = .00, 95% CI = 0.00–0.07.



Tests of Model Fit:

$\chi^2(12, N = 526) = 14.380; p = 0.2771$

Comparative Fit Index (CFI)	0.979
Tucker-Lewis Index (TLI)	0.968
Root Mean Square Error of Approximation (RMSEA)	0.034
95% Confidence Interval	(0.000, 0.088)

Figure 1. Unstandardized model estimates connecting maternal harshness with sexual and other risk taking via residualized age of menarche. ** $p < .01$. *** $p < .001$.

Results

Descriptive statistics and correlations between variables are presented in Table 1. Greater maternal harshness was associated with girls' earlier age of menarche (as reported in Belsky et al., 2007) and with both more sexual risk taking and more other risk taking. Earlier menarche was associated with greater sexual and other risk taking. These results replicate findings cited in the opening paragraph of the Introduction and thus support the validity of the risk-taking measures used in this inquiry. Finally, girls who engaged in greater sexual risk taking also engaged in more other risk taking.

The final hypothesized model fit the data well: $\chi^2(12, N = 526) = 14.38, p = .28, CFI = .98, TLI = .97, RMSEA = .03,$

95% CI = 0.00–0.09. Greater maternal harshness was associated with earlier menarche (controlling for maternal age of menarche). Earlier (residualized) menarche, in turn, was associated with more sexual risk taking but not more other risk taking. In contrast, early maternal harshness was directly associated with more other risk taking but not more sexual risk taking. As noted above, sexual risk taking and other risk taking were modestly correlated (see Table 1).

In order to determine whether the apparent influence of early rearing on risk taking was dependent on pubertal timing, we tested the significance of the indirect effects in the model. These tests revealed a significant indirect path from maternal harshness through menarche to sexual risk taking (estimate =

Table 1
Sample Descriptives and Correlations Between Variables

Variable	N	%	M	SD	1	2	3	4	5
Race/ethnicity									
White	433	82.3							
Black	62	11.8							
Hispanic	31	5.9							
1. Maternal harshness	525		2.13	0.35	—				
2. Age of menarche	377		12.31	1.14	-0.23 [†]	—			
3. Residualized age of menarche	362		0.00	1.07	-0.19***	0.93 [†]	—		
4. Sexual risk taking	478		0.10	0.26	0.11*	-0.25 [†]	-0.24 [†]	—	
5. Other risk taking	479		0.35	0.19	0.19 [†]	-0.18***	-0.14**	0.46 [†]	—

Note. Residualized age of menarche = residuals after controlling for maternal age of menarche. * $p < .05$. *** $p < .001$. [†] $p < .0001$.

0.026; 95% bias-corrected bootstrap CI = 0.009–0.052). The indirect effect from maternal harshness through menarche to other risk taking was not significant, however (estimate = 0.007; 95% bias-corrected bootstrap CI = 0.000–0.019).

When we constrained the paths from menarche to sexual risk taking and from menarche to other risk taking to be equal to determine if the effect of earlier maturation on the two forms of risk taking was equivalent, the model did not fit well, $\chi^2(13, N = 526) = 26.94, p = .01, CFI = 0.88, TLI = 0.83, RMSEA = 0.08, 95\% CI = 0.04–0.12$. Indeed, fit was significantly worse than it was for the unconstrained model, $\Delta\chi^2(1, N = 526) = 12.56, p = .0004$. The Wald chi-square test of the difference between the two parameters was marginally significant, $\chi^2(1, N = 526) = 3.36, p = .066$. Taken together, these results suggest that the path from menarche to sexual risk taking is stronger than the path from menarche to other risk taking.

When the direct paths from maternal harshness to sexual risk taking and from maternal harshness to other risk taking were constrained to be equal, the model fit well, $\chi^2(13, N = 526) = 14.87, p = .32, CFI = 0.98, TLI = 0.98, RMSEA = 0.03, 95\% CI = 0.00–0.08$, and was not significantly worse fitting than the unconstrained model, $\Delta\chi^2(1, N = 526) = 0.49, p = .48$. However, the Wald chi-square test indicated that the two parameters were significantly different, $\Delta\chi^2(1, N = 526) = 7.44, p = .006$. Although the chi-square difference test and the Wald chi-square test are asymptotically equivalent, the discrepancy noted above is likely due to the fact that we have three groups, one of which is much larger than the other two and two of which are very small. In this case, it is unclear which test provides the most accurate assessment of the difference between the parameters. As such, we can tentatively conclude only that the direct path from maternal harshness to other risk may be somewhat stronger than the direct path from maternal harshness to sexual risk taking.

In sum, the effect of maternal harshness on sexual risk taking was only indirect, via age of menarche, whereas its effect on other risk taking was only direct and apparently did not involve pubertal maturation.

Discussion

Given evidence from separate sets of studies cited in the Introduction showing that family rearing environment is predictive of pubertal timing and that pubertal timing is predictive of sexual and other types of risk taking, this study sought to determine whether (a) maternal harshness in early childhood, (b) earlier age of menarche, and (c) higher levels of sexual and other risk taking could be linked empirically in a sizable sample of females prospectively studied from birth through age 15. Path analysis provided support for the claims of BSD theory: Not only did maternal harshness predict pubertal timing but pubertal timing predicted sexual—but not other—risk taking in a manner consistent with theoretical expectations unique to BSD.

The fact that only sexual risk taking, the indisputably reproductively oriented outcome studied here, and not other risk taking appeared indirectly influenced by maternal harshness via pubertal timing is noteworthy. Had such indirect effects emerged for both outcomes, this would have suggested a broader interpretation of BSD theory than now seems appropriate. Although evidence link-

ing greater maternal harshness and greater other risk taking emerged, the effect detected was direct and did not involve pubertal timing; this result is consistent with more traditional developmental perspectives, such as attachment, social-learning, and life-course theories. Thus, unless and until future research proves otherwise, it appears that indirect effects of early experience on adolescent risk taking, via pubertal timing, are likely to be restricted to explicitly reproductive-strategy-related functioning, most notably sexual risk taking.

Of importance also is that estimated pathways of influence proved to be equivalent—insofar as could be determined—among Whites, Blacks, and Hispanics. The fact that this result emerged despite groups differing in mean levels of maternal harshness, age of menarche, and risk taking (see Table 2) is consistent with the claim that the developmental processes under consideration apply to all these groups, just as an evolutionary analysis would presuppose. Caution is warranted in embracing this conclusion, however, given our inability, due to the limited Black and Hispanic subsample sizes, to estimate with reasonable power effects for each group separately. The reader is thus reminded that embracing null findings—such as “developmental pathways of influence do not vary across race/ethnic groups”—is always precarious, because the absence of evidence (of nonequivalent pathways) is not evidence of absence.

Although attention should be called to the rather modest effect sizes and variance accounted for by the evolutionary model tested in this inquiry (see bottom of Table 2), disproportionate concern for such matters risks missing the core contribution of this inquiry: its empirical evaluation of a unique prediction that simply could not be derived from any prevailing psychological or sociological theory of human development in general or of adolescent risk taking in particular. Only an evolutionary theory with its concern for reproductive-fitness goals leads to Belsky et al.’s (1991) “uncanny” prediction that rearing experiences should influence the timing of biological maturation and thereby, and thus indirectly, reproductive behavior in adolescence. From the standpoint of classical philosophy of science, confirming highly specified and theoretically unique predictions is of as much, if not greater, scientific importance as the magnitude of effect sizes or variance explained in the analysis of data. This would seem especially so, given that the current sample is neither nationally nor internationally representative and thus does not include the full range of rearing environments, a fact that could very well attenuate the effects detected. Just as important is that the dramatic reduction in the age of pubertal maturation that the Western world has witnessed over the past 150 years may well have reduced much of the plasticity in pubertal timing that once characterized the human condition—and still could in many parts of the world. Finally, and perhaps most important, small changes in age of menarche, as Ellis (2004) has explained, can exert great influence on fertility over the life course, especially among noncontracepting individuals and populations.

Limitations and Future Directions

Even though the indirect effects of maternal harshness on sexual risk taking via age of menarche proved significant and consistent with BSD theorizing, it must be acknowledged that the nonexperimental nature of this research limits our ability to draw causal

Table 2
Unstandardized Model Estimates and 95% Bias-Corrected Bootstrap Confidence Intervals

Variable	White (<i>n</i> = 433)		Black (<i>n</i> = 62)		Hispanic (<i>n</i> = 31)	
	Estimate	95% CI	Estimate	95% CI	Estimate	95% CI
Means and intercepts						
Maternal harshness ^a	-0.054	-0.088, -0.024	0.356	0.257, 0.435	0.103	0.012, 0.192
Residualized age of menarche ^a	0.074	-0.050, 0.203	-0.276	-0.616, 0.046	-0.306	-0.922, 0.384
Sexual risk taking	0.101	0.072, 0.129	0.158	0.027, 0.280	0.065	0.002, 0.181
Other risk taking	0.335	0.317, 0.355	0.391	0.323, 0.454	0.384	0.282, 0.492
Paths						
Maternal harshness → residualized age of menarche	-0.466	-0.795, -0.170	-0.366	-1.350, 0.450	0.314	-2.008, 2.351
Maternal harshness → sexual risk taking	0.045	-0.010, 0.126	-0.065	-0.345, 0.257	0.384	0.057, 0.756
Maternal harshness → other risk taking	0.052	-0.012, 0.118	0.106	-0.019, 0.241	0.294	-0.085, 0.561
Residualized age of menarche → sexual risk taking	-0.063	-0.095, -0.034	-0.022	-0.105, 0.065	-0.098	-0.249, -0.021
Residualized age of menarche → other risk taking	-0.019	-0.040, 0.000	-0.010	-0.057, 0.029	-0.014	-0.133, 0.086
Covariances						
Sexual risk taking → other risk taking	0.020	0.014, 0.027	0.020	0.014, 0.027	0.020	0.014, 0.0278
Indirect effects						
Maternal harshness → age of menarche → sexual risk taking	0.029	0.010, 0.061	0.008	-0.023, 0.120	-0.031	-0.306, 0.189
Maternal harshness → age of menarche → other risk taking	0.009	0.001, 0.024	0.004	-0.023, 0.047	-0.004	-0.133, 0.136
R²						
Residualized age of menarche	0.021		0.013		0.005	
Sexual risk taking	0.080		0.009		0.269	
Other risk taking	0.023		0.068		0.131	

Note. Residualized age of menarche = residuals after controlling for maternal age of menarche.

^a Grand mean centered.

inferences from these data. Not only is it possible—and probably likely—that adjusting girl's age of menarche for maternal age of menarche did not fully account for heritability and, thereby, genetic influences that affect both timing of puberty and risk taking, but it remains possible that unmeasured third variables could be responsible for the findings of this inquiry. Note, however, that recent experimental work on female rats clearly documents causal effects of early rearing experience (i.e., maternal licking and grooming) on pubertal timing (Cameron et al., 2008) and sexual behavior (Cameron et al., 2008) that are in line with BSD theory (Champagne et al., 2006). And recall that Tither and Ellis's (2008) recent work using a within-family sibling design that controlled for genetic confounds yielded human evidence consistent with BSD theorizing regarding effects of rearing on pubertal development.

A further limitation of this study concerns the strategy of controlling for maternal age of menarche, as this strategy could have resulted in a conservative test of the hypothesis under consideration. This would be the case if the positive association between mothers' and daughters' menarcheal ages derives from the very process under study in this inquiry (i.e., that both mothers and daughters have similarly earlier or later ages of menarche as a result of similar early rearing experiences that, in fact, could be intergenerationally transmitted). Recall, however, that results remained virtually unchanged when the analyses reported were conducted without first adjusting daughters' age of menarche for that of mothers.

Perhaps the major limitation of the present work was that we were restricted to using a single measure of rearing experience to

predict risk taking via pubertal timing; recall that this was because early maternal harshness proved to be the only such measurement made before any children would have started puberty that Belsky et al. (2007) found to predict pubertal timing in the present sample. This means, of course, that future studies of other samples addressing the same issues investigated herein should not restrict their focus to maternal behavior or even just to maternal harshness. In view of the fact that other studies reviewed by Ellis (2004) or cited in the Introduction have found any number of rearing factors implicated by BSD theory to predict pubertal timing, future work should cast a wider net than we could to identify facets of the early rearing environment that might indirectly affect reproductive strategy via pubertal development.

Evidence linking social and psychological experience in the family early in life with pubertal maturation raises a core theoretical question that the current inquiry could not address but that merits special attention in future work: By what physiological mechanisms might rearing experience, including harsh control by mothers, come to regulate biological development and, thereby, reproductive strategy? Belsky et al. (1991) speculated that a neuroendocrine subsystem intertwined with other endocrine systems could provide a pathway linking experiences in the family with pubertal timing. More recently, Chisholm, Burbank, Coall, and Gemmiti (2005) theorized that the hypothalamic-pituitary-adrenal axis, which is directly involved in stress regulation, may play a critical role in the process. And recent elegant experimental research with rats by Cameron, Fish, and Meaney (2008) showed not only that maternal licking and grooming of the newborn pup

enhances stress regulation, delays the onset of puberty, and reduces sexual activity, with the reverse being true of its absence, but that such effects on rat reproductive strategy are mediated by maternal-care effects on gene expression, via methylation. What remains unclear to date, though, is whether the effects of gene expression on stress regulation are directly linked to the documented rearing effect on puberty and, thereby, sexual behavior in the rat and, of course, whether the same processes operate in the early regulation of reproductive strategy in humans.

It will also be important to determine in future work whether indirect effects of rearing, via puberty, extend to other aspects of reproductive strategy (most notably, number of sexual partners, pair-bond stability, and the quality of parenting beyond the incidence of early sexual activity outcomes) that are more appropriately measured at an older age than studied here. Although such aspects of reproductive strategy are hypothesized in BSD theory to be linked as part of a probabilistic chain of causation to early rearing, pubertal development, and onset of sexual activity, the intriguing alternative view of Ellis (2004) suggests otherwise. His “child development theory” stipulates that because one evolutionary function of early rearing is to regulate the duration of childhood—and not to shape reproductive strategy, as BSD theory contends—pubertal timing should not be linked with mating and parenting outcomes after adolescence beyond, perhaps, onset of sexual activity. Thus, Ellis would not expect puberty to play an indirect role in linking early rearing with mating and parenting, whereas BSD theory would. Prospective longitudinal studies that follow individuals beyond adolescence and into adulthood are necessary to adjudicate between these two evolutionary models.

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Call for Nominations: *Psychology of Men and Masculinity*

The Publications and Communications (P&C) Board of the American Psychological Association has opened nominations for the editorship of *Psychology of Men and Masculinity*. The editorial search is co-chaired by Glenn Good, PhD and Lillian Comas-Diaz, PhD.

Psychology of Men and Masculinity, official journal of APA Division 51 (Society for the Psychological Study of Men and Masculinity), is devoted to the dissemination of research, theory, and clinical scholarship that advances the psychology of men and masculinity. This discipline is defined broadly as the study of how boys' and men's psychology is influenced and shaped by both sex and gender, and encompasses both the study of biological sex differences and similarities as well as of the social construction of gender.

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