
Biological Determinants and Influences Affecting Adolescent Pregnancy

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Starting from a Biological Perspective

The basic assumptions employed by professionals to define *adolescent pregnancy* give direction to research and authority to policy and interventions that form the services provided by the medical and helping professionals. Accordingly, because adolescent pregnancy is first of all a biological process, logically professional assumptions would start from a *biological* perspective. The biological reality is that adolescent girls and boys need sexual and reproductive health care and education designed to meet their needs given their physical and emotional development. The risk they face from genetic vulnerabilities and environmental exposures is too great to keep them ignorant about their sexual and reproductive development. One example of a service that is obvious from a biological perspective is based on data that show almost 1 in 8 girls reaches menarche while still in primary school. When designing programs to

provide sexual and reproductive health information and health services for adolescent girls, if we first consider the biological variations in sexual development among primary school girls, we would provide them with the education and services girls need when reaching menarche. Yet, in many countries, adolescent sexuality and pregnancy are seen as a moral problem not as a biological process. Thus, in many countries and cultural groups, primary school girls are viewed as too young to receive sexual and reproductive services and, too often as a result, suffer from long-term adverse consequences.

Adolescent pregnancy is a natural phenomenon that is biologically available to virtually all adolescent girls. This biological imperative means that essentially all adolescent girls have the potential to become pregnant. Because of this reality, there is a bona fide need to provide maternal health education and care in the most comprehensive way possible.

From a biological perspective, the answer is to intervene medically and psychosocially to prevent a pregnancy from doing harm to the adolescent mother and her child. This includes providing services to address specific physical and psychosocial issues that are common among adolescent girls. Novelist Hilary Mantel

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described the phenomenon of adolescent pregnancy as: “Having sex and having babies is what young women are about. And their instincts are suppressed in the interests of society’s timetable” (Davies 2010).

Far from being harmless, however, there is agreement among medical professionals that adolescent pregnancy and motherhood at a very young age are correlated with elevated health risks for young adolescent mothers and their children. For the most part, the harm is a result of the immaturity of the girl’s body. Conversely, there is substantial disagreement that delaying childbearing until adulthood results in better outcomes. This conclusion is based on the preponderance of research that describes adolescent pregnancy as a problem. However, from a biological perspective, reproductive maturity and adequate resources result in the best pregnancy outcomes.

Many who question the research that describes adolescent pregnancy as a problem point out that starting one’s research based on the assumption that adolescent pregnancy is a *problem* is likely to produce research describing adolescent pregnancy in terms of different *problems*. Given the basic assumption that adolescent pregnancy is a problem, it is no wonder that policies and interventions are designed to prevent adolescent pregnancy while neglecting sexual and reproductive health education and services.

When examining the medical, social, political, and public response to adolescent pregnancy in different countries around the world, it becomes apparent that the biological perspective (a culmination of physiological and anatomic processes) is not the dominant perspective and most often takes a backseat to political, cultural, religious, and vague moral interests (Furstenberg 2007). Next in this chapter, the biological evidence that can inform our understanding of adolescent sexuality and pregnancy is presented.

Biological Determinants

Maturation is the process of developing biological imperatives needed by living organisms to perpetuate their existence. The imperatives that

must emerge during maturation have been summarized as survival, territorialism, competition, reproduction, and quality of life-seeking. The reproduction imperative, as a focus of adolescent pregnancy in modern society, has little to do with nature and almost all to do with culture. Which begs the question, those who do not fulfill an imperative are by definition described as maladaptive, while those that do fulfill an imperative are described as adaptive? By definition, adolescent pregnancy satisfies the reproductive imperative.

Adolescent pregnancy in modern society, however, is maladaptive not because of some endemic organic force but because of the pre-eminence of individual economic security over procreation. There is no support in a modern society for dependent, pregnant, and parenting adolescents. Nevertheless, research, since the 1990s, has clearly demonstrated that there are both evolutionary and genetic influences that affect a girl’s early fertility and resultant sexual behaviors. Even behaviors that we had assumed were exclusively the result of environmental experiences that have been shown to be influenced by individual genetic makeup. The assumption is that there are genetic underpinnings of behavioral phenotypes. Studies using *behavior genetic designs* (i.e., identical twin studies and studies of the children of identical twins) in order to control for genetic influences have been conducted to rule out genetic influences. This line of research has not ruled out a genetic influence on adolescent sexual behavior. Instead, this research found considerable evidence that while the environment affects and influences a girl’s sexual behavior, a girl’s *genes* also affect and influence her sexual behavior. Including knowledge of this *gene–environment interplay* (D’Onofrio 2003; Jaffee and Price 2007) when designing health and education services could result in more adolescent-friendly and effective sexual and reproductive health services.

To come to the point, as specific genes are identified, we can begin to explore important and pressing questions about behavior. How do these genetic influences interact with environmental

factors to shape development and behavior? How do we interpret these findings? How do we ask new questions about these findings? How do we celebrate the knowledge? And how could we use or misuse this knowledge? These issues are pervasive in all areas of human research, and they are especially salient in human behavioral genetics.

Investigating Early Fertility

Behaviors related to menarche and fertility and particularly early fertility are prime candidates for investigating the importance of evolutionary and biological predisposition on adolescent sexual and reproductive behavior. Important to our understanding of adolescent pregnancy is this concept that the physiological and anatomic processes involved in puberty are affected by environmental exposure. In biological terms, puberty is a series of physiological and anatomic processes that occur during adolescence. Puberty is also the state of physiological development after which the adolescent is physically able to sexually reproduce. Grumbach and Styne (1998) defined puberty as an individual process of development driven by a gonadotropin-releasing hormone (often referred to as the growth hormone) in the hypothalamus. In addition to gonadotropin secretion, the gonadal steroids (often referred to as the sex steroid) combine and result in puberty.

Yet, the timing of normal puberty varies around the world and by some measures has changed over time. In the past, precocious puberty was defined as sexual development before the age of 8 in girls and age of 10 in boys. In 1999, these limits were revised to 7 years of age for Caucasian girls and 6 years of age for African-American girls. Precocious puberty is four to eight times more prevalent in girls than in boys.

Precocious puberty means having the premature signs of puberty such as the development of breasts, testes, pubic and underarm hair, body odor, menstrual bleeding, and increased growth.

Among girls, the first signs of precocious puberty are the appearance of pubic hair and budding breasts. Menarche is highly correlated with the appearance of breast buds and is therefore considered to be an indicator of early onset of puberty.

In previous studies, differences in the timing of puberty have been explained in terms of variations in ethnicity, geographical, and socioeconomic conditions. These models, however, are not a good explanation for an increased incidence of sexual precocity observed in the United States since the 1980s. While ethnicity, geographical, and socioeconomic conditions cannot adequately explain the drop in the age of puberty in the United States, the onset of puberty as a possible sensitive and early marker of the interactions between environmental conditions (such as industrial and household chemicals) and genetic susceptibility is hypothesized as a possible explanation (Parent et al. 2003).

Sociosexuality: Genes and Environmental Interaction

Theories about sociosexual development tend to focus on the environmental influences (for the most part the parental effects) that shape individual sexual behavior. The causal connections between parental influence and child outcomes using typical family samples are limited, however, by the inability of this approach to account for all of the malleable conditions both environmental and genetic that could influence behavior, particularly sexual behavior.

So far, we know that social learning and environmental influences explain a great deal about individual behavior and preferences. What we have learned since the 1980s is that environment and learning explain a lot less about behavior than previously thought. What we do know, in reference to sexual behavior, which is of importance to our understanding of adolescent pregnancy, is that substantial variation in human sexual and reproductive behaviors is inherited. Explained by evolutionary theory,

genetics is predicted to be a major influence on sexual behavior because sexual behavior is the most proximal determinant of fertility, the evolutionary process by which genic reproduction is modified or maintained. Moreover, in some developmental processes, it is reasonable to expect that some genetic influences will be stronger in older children. As children mature and are free to express their genetic preferences in selecting their environment and associates, they will be more influenced by their genetic influences.

To test genetic theory, twin studies have been conducted and show that monozygotic (MZ) twin pairs (fertility-related phenotypes) can vary in early onset or late onset of maturation. Among the twin pairs, however, whether development is early or later—the age of onset of menarche and the age of first sex, the desired age of marriage, and the desired age to have children are virtually the same for each twin in the pair. These findings strongly support the hypothesis that genetic differences between individual girls account for their variation in sexual timing (Aragona 2006; Bailey et al. 2000; Dunne et al. 1997; Lyons et al. 2004; Martin et al. 1977; Rowe 2002; Waldron 2004). Additionally, among males, functional polymorphisms for dopamine receptor genes (*DRD4 48 bp VNTR*) are associated with earlier age at first sex, migratory behavior, and a greater frequency of multiracial ancestries (Miller et al. 1999). What the twin studies have demonstrated is that variation in social behavior partly reflects individual genetic differences and environmental influences. In modern society, however, the individual's genetic predisposition is also influencing the shape and form of the individual's environment. When individuals are free to select their social environment (friends, schools, occupations, organizations, and sexual partners) they are more incline to select social environments based on their genetic predisposition than individuals with little control over selecting their social environment (Scarr and McCartney 1983). This self-selection phenomenon in humans is obvious in numerous situations. The self-

selection into a compatible profession, for instance, is virtually essential for success.

Children-of-twins and family comparison studies have added to our confidence in the *gene–environment interplay* explanation of adolescent pregnancy. This methodological design provides an additional rigorous test of the degree of genetic influence on a child's life trajectory. The children-of-twins design has been used to examine the influences of marital conflict (Harden et al. 2008), stepfathering (Mendle et al. 2006), harsh punishment (Lynch et al. 2006), smoking during pregnancy (D'Onofrio et al. 2003), marital dissolution (D'Onofrio et al. 2005, 2006), parental schizophrenia (Gottesman and Bertelsen 1989), and parental alcohol/drug problems (Jacob et al. 2003) on child adjustment.

As opposed to the *gene–environment interplay* construct, modeling theory explains that children acquire their mating strategy after observing their parents' relationship, which is an example of a specific behavior that would appear to be a case of social learning. Thus, if policy were based on modeling theory, a prevention strategy would be needed to shape the relationships of parents or at least to persuade a child that there are specific acceptable sociosexual behaviors.

If there is empirical support for this social learning hypothesis, researchers would find a strong shared environmental component among children from specific environments. The environment would have a statistically significant influence on the children's sexual behavior. This was not found to be the case.

In a number of studies, similar to the work by Bailey et al. (2000), a large, representative sample of volunteer twins showed that familial resemblance in sexual tendency appeared primarily due to genetic rather than similar environmental factors. This evidence is substantial. It makes the case for concluding that genetics has a profound influence on sexual behavior, and thus, these genetic influences must be incorporated into the design and development of reproductive and sexual policy and programming.

Adolescent Pregnancy and the Nature Versus Nurture Conundrum

The gaps in our knowledge about the different levels of influence from nature and nurture continue to create dissidence. Most reasonable people who have studied the issue agree that both nature and nurture shape our sexuality, however, to what degree is still in question.

Studies that support the gene–environment interplay have investigated how it affects menarche, a physical event thought to be purely biological. To make the point about the influence of the contributions of gene–environment interplay, body mass index (BMI) (body weight) and its role in causing variations in the age of the onset of menarche have been instructive. A study referred to as the FinnTwin16 study recruited twins (1,283 twin pairs) from consecutive birth cohorts from the national population registry, which included 100 % of all living twins in Finland. There were 468 MZ girls, 378 girls from like-sex dizygotic pairs, 434 girls from opposite-sex pairs, and 141 older female siblings of the twins.

Girls from opposite-sex dizygotic twin pairs had a significantly higher mean age at menarche (13.33 years) than like-sex dizygotic twin pairs (13.13 years). The MZ correlation for age at menarche was $r = 0.75$, the like-sex dizygotic correlation was $r = 0.31$, and for the opposite-sex twin pairs, the correlation was $r = 0.32$. A bivariate twin analysis of age at menarche and BMI indicated that 37 % of the variance in age at menarche can be attributed to additive genetic effects, 37 % to dominance effects, and 26 % to unique environmental effects. The correlation between additive genetic effects on age at menarche and BMI was $r = 0.57$, suggesting a sizable percentage of genetic effects on menarche and puberty (Kaprio 1995).

Subsequently, what is most striking about the genetic influence on early fertility is that it accounts for over 50 % of the variation in early fertility. This finding is especially important to providing sexual and reproductive services to young girls. It is especially troubling, however,

because of the social and emotional cost borne by adolescent girls who become pregnant. Costs that where far too many girls and adolescents end in a negative life cycle for them and their children (Kohler et al. 2002).

Where sufficient BMI is needed for menarche to begin, obesity is strongly associated with when girls reached menarche at a significantly earlier age than girls within a normal weight range. The report by Bau et al. (2009) is representative of this line of research. In the Bau study, girls who were overweight started menarche at 12.5 years of age, while girls within a normal weight range started at age 12.9. Underweight girls were much later at 13.7 years of age. The body weight for all girls was similar irrespective of age and height (Bau et al. 2009).

Puberty, Age of Menarche, and the Genetic Influences

The worldwide median age of menarche is estimated to be 14 years of age. About 50 % of girls began menarche before age 14 and 50 % start after age 14. Most often in developed countries and modern urban areas, the age of onset of menarche is under 14 years of age. Among girls living in developing countries, the age of onset of menarche is over 14 years of age. There are also significant differences by geographical region, race, and ethnicity. The average age of menarche in the United States is about 12.5 years of age. In China, the age of menarche onset is 12.8 years. In Nigeria, the average age is 13.7 years (Ikaraoha 2005). In Sudan, it is 13.85 (Attallah et al. 1983). In Morocco, it is 13.66 (Montero 1999), and in Mozambique, it is 13.9 years of age (Padez 2003). Table 1 provides age of menarcheal t from a sample of counties from around the world.

In addition to the average age of menarche varying from country to country, the average age of menarche also has varied significantly over decades. To illustrate this variation over time, the average age of menarche among girls in the

Table 1 Global variation in mean estimates of age at menarche

Argentina	12.6	Philippines	13.6
Australia	13	Portugal	12.5
Canada	12.7	Russia	13
Chile	13	South Africa	12.5
Columbia	12.8	South Korea	13.9
Germany	12.8	Spain	12.30
India	14.3	Sweden	13.1
Indonesia	13	Switzerland	13
Ireland	13.5	Turkey	13.3
Japan	12.5	USA	12.5
Netherlands	13.2	Uganda	13.4
Nicaragua	14	United Kingdom	12.9
Nigeria	13.7	Vietnam	12.7

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United States was 13.5 years in 1900. The age slowly dropped but was very similar between the 1930s and the 1960s (mean age at menarche, 12.72–12.99 years). Then, in the 1970s, the mean age at menarche began to drop again. By the 1980s, the average age of menarche was significantly younger ($p < 0.001$) than in the previous 50 years (mean age at menarche, 12.34 years). Then again, if the data are correct and they seem to be, the historical data on age at menarche were about the same during the first millennium as it is today. In ancient Rome, the menarcheal age of onset was 12–14 years of age. In medieval Europe, it was 12–14 years of age, and in medieval Middle East, it was 12–13 years of age. Which begs the question, what was going on between medieval times and modern times that so radically decreased and then increased the menarcheal age in the twentieth century?

The variation in the age of onset of menarche is important to our understanding of adolescent pregnancy. Based on the previous research, we can say with confidence that the correlation between the age of menarche dropping in the 1970s and the increase in US adolescent pregnancy beginning in the 1970s was no accident. As the twin studies reveal, the younger the girl, when she starts menarche, the more fertile she will be. This is because sexual behavior is not

just driven by modeling and decision making. Individual sexual behavior is also driven by instincts resulting from evolutionary development. *Why*, because sexual behavior is the most proximal determinant of genic reproduction and thus continued existence as an organism.

Urban Versus Rural Age at Menarche

Globally, the major differences in age of onset of menarche are not by country but by the distribution of wealth, most notably between urban and rural areas of the world. While the average age of onset is between 12 and 13 years of age, in rural areas, the age of onset is 14 years of age or slightly older. Starting menarche at age 17 is not unusual in many rural areas of poor countries. Studies of menarcheal age in urban and rural girls report that while there is a difference in age, the differences are the greatest in the less developed countries. Since 1940s, menarcheal age has decreased at a rate of 0.34 years per decade for rural girls, 0.73 years per decade for urban girls, and 0.46 years per decade for combined groups of both rural and urban. The decline in menarcheal age since the 1940s worldwide is attributed to the improving socioeconomic conditions worldwide (Cameron et al. 1991).

The most obvious explanation for the discernibly later average age of onset of menarche among girls living in rural areas (especially in developing countries) is poor nutrition. In most countries, a lack of resources available to rural communities predicts rural poverty and thus poor nutrition among the children. Other compounding factors, however, also contribute to the difference. In many rural cultures, boys are valued over girls. Boys are nurtured; girls receive less attention. Child marriage and girls marrying at a very young age is still a part of the culture. Formal female education is lacking or limited, especially in terms of sexual and reproductive health. Poorer nutrition and health among rural girls, when compared to their urban counterpart, slow their maturation process. Consequently, while the menarcheal age for both urban and rural girls declined over the decades because of improved nutrition, more resources were available to urban girls and that made all the difference. Among all girls, however, the timing of menarche is a combination of female biology, inherited genes, and environmental exposure.

In addition to the menarcheal age, there are other biological factors related to adolescent pregnancy risk such as hormone levels, genetic timing, and age of first sexual intercourse. These are important biological developments that can increase or decrease adolescent pregnancy risk. Yet, time and again, these biological factors been ignored in public policy and programming with wide-ranging consequences for adolescent girls. Not only is the social, emotional, and financial cost of this public policy failure borne by adolescent girls—it is a cost that too often results in a negative life trajectory for the girls and their children (Kohler et al. 2002).

The Belsky–Draper Hypothesis of Menarche

The global variation in menarcheal age between girls growing up in different economic environments (i.e., rural vs. urban) supports a hypothesis that, in general, girls who start menarche at a

younger age have adequate resources or economic advantage over girls who start menarche later in their adolescents. This broad-spectrum theory, however, does not always explain the difference between girls in the same region where menarcheal age can vary by racial and ethnic group.

This was precisely the case in the United States in the 1970s and 1980s when a controversy erupted over how much younger African-American girls were compared to Caucasian girls who were becoming pregnant. This phenomenon seems to be contrary to global trends, which would predict that girls' with fewer resources would have a later menarcheal age (Chumlea 2003). Although a number of explanations were pushed forward, most agreed to some extent with Conservatism (although often with fewer resources) that African-American girls were younger when they became pregnant because of a breakdown in traditional Christian sexual morals among African Americans in the United States. Given the perceived sexualization of US society, and the claims-makers who framed it as a moral issue, there were few other explanations advanced enough to explain the differences.

In one exception to the preponderance of professional opinion, even though arguably it was also incorrect, Belsky et al. in (1991) reasoned that early maturity among African-American girls was a form of *conditional adaptation*. They hypothesized that there are also psychological resources, which impact the start of menarche. The Belsky–Draper hypothesis states that variations in menarche (which is a reproductive-strategy-oriented event) can be predicted by traditional and nonevolutionary events in the environment. Belsky and colleagues proposed that the early family environment (communicated nonverbally through the infant–parent attachment relationship) conveys to children the risks and uncertainties they are likely to encounter in their lifetimes. They conclude that the girl's sense of security during the period when the infant–parent attachment relationship develops would impact pubertal onset and thus predict an earlier or later menarche.

The hypothesis is based on the rationale that a lack of secure relationships in early life, typically associated with unsupportive family relationships, would speed up pubertal development among girls in similar family circumstances. Furthermore, these girls would develop earlier than girls living in a secure and supportive family. This construct of early pubertal development is based on the assumption that early sexual development gives the species an evolutionary advantage for their survival in a dangerous environment. This evolutionary strategy gives girls in these hazardous circumstances a biological advantage to offset the risk in their purpose to reproduce to sustain the species.

Because of the contradictory explanation for early menarcheal age, there have been a number of studies that have tried to test the Belsky–Draper hypothesis that there are psychological resources that affect the onset of menarcheal age. Moreover, because of the extraordinarily high likelihood of selection bias in sampling to test this type of complex hypothesis, quantitative findings have been mixed. Some supports the Belsky–Draper hypothesis, while others seem to disprove the hypothesis. Two studies specially carried out to test this hypothesis are representative of the issues related to menarcheal age and selection bias.

In 2002, Rowe reported a study of data from female twins collected by the *National Longitudinal Study of Adolescent Health* (Add Health) in an attempt to test the Belsky–Draper hypothesis. (See: http://www.cpc.unc.edu/projects/lifecourse/research_projects/add_health). The data were used to determine differences and similarities between age of menarche and age of first sexual intercourse among a national sample of female twins. In this sample, the average age at menarche was 17 years. While, the menarcheal age of these twin pairs was somewhat older than the average menarcheal age in the United States at the time, which could have influenced the findings; Rowe reported that the results indicated that age of menarche and age of first sexual intercourse were significantly influenced by genetic variation. Moreover, age of menarche was slightly more influenced by genetic heritability

than age of first sexual intercourse. Nevertheless, the correlation between age of menarche and age of first sexual intercourse among the sets of MZ twins (identical twins) was $r = 0.72$. The level of correlation suggests that heredity more than environment accounts for individual differences in the variation in timing of the menarche. Or, does it? In this and other studies of identical twins, the sample sizes tended to be small. This precludes the use of covariance matrices in these biometric models to determine precise genetic effects. As a result, the possibility of selection bias even in identical twin studies continues to exist.

Sexualizing the Child

Child sexual abuse has long been known to result in a multitude of adverse effects for the victims. Among these are mental health problems, physical health problems, and risky sexual behaviors (Arata 2002; Breitenbecher 2001). In part, the harm to the child's life trajectory results from a phenomenon known as "revictimization." Among women in the United States who report being sexually abused as a child, they also report 2–3 times more sexual assaults in adolescence and adulthood than females without a history of child sexual abuse (Barnes et al. 2009). Even though the cause seems intuitive, the mechanism that explicates the link between childhood sexual abuse and later victimization is still enigmatic (Noll and Grych 2011).

In one example of many similar studies intended to investigate this observation, Vigil et al. (2005) used a life history theory to examine the relationship between child sexual abuse, childhood adversity, and patterns of reproductive development and behavior. They selected a sample of 623 women with an average age of 27 years. These women were a demographically diverse sample of American women from two US regions (rural and suburban), Missouri ($n = 418$) and the Albuquerque, New Mexico area ($n = 205$). Using a community survey approach, this sample of 623 women was

assessed for child sexual abuse, age of menarche, and social and family background.

The results of this study (given its obvious methodological flaws) showed that social and family environment had significantly less to do with the individual's age of menarche, first sexual intercourse, the desire to have children, first childbirth, and low self-valuation of physical attractiveness than having a history of child sexual abuse. These correlates within this group of abused women can be interpreted as meaning that childhood sexual abuse "in combination with other childhood circumstances" may "modify biological and behavioral patterns of individual maturation." While some of the findings related to behavior have been supported by similar research, the impact on the age of menarche is still left unanswered. Because of selection bias, there is still a question as to whether it is a combination of sexual abuse and other circumstances or whether the age of menarche is affected by other circumstances with a great influence on our the biology? For instance, we know that early biological maturation is significantly influenced by genetic inheritance. We also know that *early sexual maturation has been associated with child sexual abuse*. This study does not address these issues.

In a more recent study, Belsky and colleagues (2010), in an effort to support their original hypothesis, reported on a study where they used life history theory to test their hypothesis. Their study tested the evolutionary strategy of *early programming of human reproductive development*. This construct proposes a corollary of "early rearing experience, including that reflected in infant-parent attachment security, regulates psychological, behavioral, and reproductive development" (p. 1195).

In this study, Belsky and associates examined the annual physical examinations from 373 white females when they were between 9½ and 15½ years of age. They were enrolled in the National Institute of Child Health and Human Development Study of Early Child Care and Youth Development (2005). The test of the hypothesis was to determine whether self-

reported age of menarche was different among girls who experienced infant attachment *insecurity* at 15 months of age as compared to girls who experienced infant attachment *security at the same age*. Belsky and colleagues report results that support their conditional adaptation hypothesis. Girls who experienced infant attachment *insecurity* reported an earlier age of menarche.

Again, however, a good dose of skepticism is called for in relation to these findings. There is obvious selection bias in the sample of women they examined. Although selection bias does not on its own disprove a hypothesis, it leaves open the question about the study being evidenced that the hypothesis has a great deal of utility. Given that earlier biological maturation is inherited and that a younger age at puberty tends to predict a younger age at first sexual intercourse, it is not unexpected that it tends to predict earlier parenthood. What the findings do not rule out is the real possibility that girls who experienced *attachment insecurity* also inherited a predisposition to earlier sexual maturation, which in part given social norms could have contributed to the girl's *attachment insecurity*.

From another perspective, the Belsky-Draper hypothesis does not explain professional observations over 50 years in the field of child protection, especially among children who have experienced a dramatic loss (typically of their mothering figure). In child protection cases, it is fairly common to see a delay in pubertal development. Although there have been no studies of this phenomenon, the delay in development can be quite profound and has been observed by most professional caring for abused and neglected child. It is common among severely abuse children to find a 15-year-old that has the maturity level of a 10- or 11-year-old. As antidotal evidence, it is a similar reaction to the human body delaying maturity because of a lack of adequate nutrition.

There are several serious flaws in the Belsky-Draper hypothesis. The most obvious is that the primary worldwide threat to adolescent fertility is inadequate nutrition not family-transmitted child insecurity. In such cases, the evolutionary

response to a lack of adequate nutrition is to fit the organism to the environment in ways that delay puberty and reduce reproductive success until adequate nutrition is available.

A similar evolutionary response might explain precocious puberty among African-American girls? A major evolutionary strategy for survival is to increase one's individual value, which increases the organism's chances of reproduction. For African-American girls, when the opportunity to increase social capital is limited and reproductive strategies are the only option available, teen pregnancy rates will be high; much like they were between in the 1950s and the 1970s in the United States.

Nevertheless, the Belsky–Draper hypothesis to predict the timing of menarche was a major breakthrough in the thinking about sexual development. It added to the biological and hormone hypotheses a psychological component. Studies of conditional adaption continue to reveal the malleability of the biology of life. In the case of menarche, however, the research shows that while menarche might be influenced by conditional adaptation and psychological influences, it has been shown to influence the age of menarcheal onset within a window of time, a range of age during childhood between 7 and 17 years of age.

Children of Twins Approach

There are many other examples of the environments acting as a trigger to initiate early or delayed maturation, but not enough. More studies using socially and contextually informed analyses of behaviors once thought to be shaped by moral standards are sorely needed. Without a body of genetic studies that point to behaviors as being formed by the differential forces of *nature and nurture*, the focus of reproductive and sexual health policy and services to prevent adolescent pregnancy will be based on the dominant philosophy rather than the best scientifically based knowledge.

There are also other examples where evolutionary theory did not explain early puberty. One such study examined the environment to see if stepfathering could trigger early maturation. These researchers thought that evolutionary theory predicted early maturation among girls growing up in households or in close proximity to unrelated adult males. In other words, they theorized that girls would start menarche earlier than their peers who were not around unrelated adult males. This was presented as an evolutionary strategy for families under stress. While earlier studies supported the relationship, Mendle et al. (2006) tested the possibility that the finding was a result of a nonrandom selection bias. They reasoned that the girl and stepfather shared a similar environment, and combined with genetic predisposition, these circumstances created a spurious relation between stepfathering and early menarche among nonrelated girls in the family.

To control for genetic differences and shared environmental experiences, the researchers use a children-of-twins design to examine the relationship between stepfathers and early menarcheal onset. The researchers found that cousins with or without stepfathers did not differ in age when menarche started. Furthermore, when the mother's age was controlled for, the onset of menarche associated with stepfathering in unrelated girls was eliminated. These findings strongly suggest that selection of the sample, not the stepfathering environment, accounts for the finding that stepfathering caused early menarche in unrelated girls (Mendle et al. 2006).

Menarche is an event experienced individually by each girl. In general, it is laden with personal, biological, and social significance for the girl, her family, and society. Menarcheal onset is also important for studying puberty in girls because of the wide variation in pubertal development across individuals. Menarcheal age is also an easily identifiable marker that can be used to compare the developmental status and relationship among same-age peers. Using menarche as a developmental marker, in

addition to the studies mentioned earlier, researchers have examined a number of different behaviors that could be related or affected by early puberty. In general, early physical development among girls rather than their moral beliefs or religious controls is correlated with less-than-desirable outcomes when compared to girls who begin puberty later in their adolescence. Body dissatisfaction and dissatisfaction with weight were linked to early menarche (Petroski et al. 2006), so was early menarche and risk of depression (Stice et al. 2001; Joinson et al. 2009) and eating disorders (Gaudineaul et al. 2010). Early menarche, however, was not related to externalizing behavior (Carter 2011), mental health disorders (Rutter 2005), and alcohol and drug use (Al-Sahab et al. 2012).

Given the mixed findings related to early menarche and behavior, it is evident that a great deal of work is yet to be done. The gene–environment interplay, however, is unmistakable. Girls who mature earlier than their peers will experience some degree of pressure to engage in behaviors more appropriate based on their appearance rather than on their life experiences, coping skills, or cognitive abilities (Graber et al. 1997). All things considered, these research findings make it imperative that sexual and reproductive services, starting in elementary school, that are required by girls who mature early, be provided in a timely manner to prevent or modulate undesirable outcomes that result from early maturity.

Behavioral Genetic Analysis

There are few topics that can stimulate the level of acrimonious discourse that are found in discussions about adolescent sexuality. The debate and discussion was brought on by an increase in teenage pregnancies, the trend toward earlier puberty, and earlier initiation of sexual intercourse. These “disturbing” trends related to adolescent sexual activity were initially blamed on the outdated and nonfunctional sexual customs (Reiss 1990), and another sign of a catastrophic moral decline (Popenoe 1998). None of

these issues have less real or concrete substance than the debate over the initiation of sexual intercourse among young and unmarried adolescents. Emotions around the circumstances that result in the initiation of sexual intercourse, particularly among young girls, can derail even the most pragmatic discussion. Nonetheless, as the evidence grows about the biological basis for many of our “moral” behaviors, there is substantial evidence that the initiation of sexual intercourse is a subtle, interaction between our genes and our environment (Dunne 1997; Rowe 2002).

Research on hormone levels and genes has shown that biological influences are intricate in the initiation of sexual intercourse. These investigators have found that androgen hormone levels (assessed by blood serum assays) and dopamine receptor genes are related to adolescent sexual arousal, sexual behavior, and age of first sexual intercourse (Miller et al. 1999). Furthermore, these biological influences explain more of the variance in the age of first sexual intercourse than psychosocial variables alone (Rodgers et al. 1999). Despite the emotional reaction and the moral outcry from the public over adolescent sexual behavior, professionals must accept the reality that biological influences play a more important role in adolescent sexual behavior than has been generally acknowledged even in the professional literature.

First Sexual Intercourse: Genes and Environment

A substantial body of research has studied the impact of early first sexual initiation on behavior. The epidemiological surveys consistently identify a number of detrimental outcomes among adolescents who report early first sexual debut. Among problems that have been reported are mental health issues (Harden et al. 2008) and delinquency (Arsenault et al. 2003). Both are suspect because in other studies, mental health disorders and antisocial behavior/delinquency have been identified as being related to genetic influences.

In this type of case, genetically informed studies can be used to separate genetic influences from environmental influences; for example, environmental influences that affect age at first sexual encounter can be separated from genetic influences. Starting with the theory that timing of the first sexual experience is related to both nature and nurture, twin studies were used to control for genetic influences that “pull apart” the genetic and environmental effects. In one type of design, MZ twins who differ in their age at first sexual encounter were compared. Using this design, differences in behavior between the twins cannot be caused by genetic influence. Consequently, differences in adjustment between twins who differ in their age at first sex also cannot be attributed to aspects of the familial environment that the twins share such as sociodemographic status of family, family structure, or family relationships (Dick et al. 2000).

Twin studies not only control for genetic selection, but also control for shared environmental influences that cannot or were not measured. Comparing identical twins provides a rigorous test of whether the relation between timing of first sex and delinquency is causal. If Identical Twin A has sex earlier than her Identical Twin B, and Twin A also shows high levels of delinquency than her identical twin sister, this association cannot be due to any genetic or environmental third variables that are shared by the identical twin sisters.

This is important because the number of previous studies using nongenetically informed samples has suggested that there was a relationship between timing of first sex and later delinquency. In one such study, which typifies a common problem in the research on adolescent sexuality, Armour and Haynie’s (2007) using data on adolescents participating in three waves of the National Longitudinal Study of Adolescent Health ($N = 7,297$) conclude a causal relationship based on nonexperimental data. Based on their interpretation of their findings, adolescents who experience an early sexual debut are statistically more likely to participate in delinquent behavior a year later than peers

who debut on time and adolescents who experience late sexual initiation.

In contrast, when Harden et al. (2008) examined the association using the same data set (the National Longitudinal Study of Adolescent Health) using a genetically informed samples of 534 same-sex twin pairs, their findings were quite different in terms of delinquency. After controlling for genetic and environmental confounding variables and using a quasi-experimental design, Harden and associates found that an earlier age at first sex debut predicted lower levels of delinquency in early adulthood not higher levels of delinquency.

What we can say with confidence is that identical twins have similar characteristics that are influenced by genes. For instance, the age of maturation is influenced by genetic predisposition. We also know that age of maturation increases the risk of early sexual initiation. Added to this condition is the knowledge that a large number of twins differ a great deal in their age at first intercourse; this difference allows for the control of genetic influences when studying the effect of early adolescent sexual initiation on their life trajectory.

What we can also say with confidence is that early adolescent sexual activity has been repeatedly linked to other detrimental outcomes that are the result of nonshared environmental factors, conditions among twins that are not shared. Most notably inconsistent and ineffective is contraceptive and condom use, which results in pregnancy and disease. Nonshared environmental factors were found in traditional religious cultures such as in the United States where adolescents are provided limited sexual health and reproductive education. Other Western industrialized countries report similar rates and patterns of teenage sexual activity but have drastically lower rates of teenage pregnancy. Based on genetically informed research and international comparison studies, it is clear that a more nuanced view of adolescent sexuality is needed when we assume that adolescent sexuality is neither inherently wrong nor globally damaging.

Discussion

There is little disagreement among scientists that our genes influence individual sexual development and behavior; the disagreement, however, is related to how much influence our genes actually have on our sexual behavior. We now know that genetic predisposition plays an important and prominent role in many areas of adolescent sexual development, knowledge that is providing useful information for policy makers and providers. Although the sociological models explain a great deal of the variation in sexual behavior, integrated models (biopsychosocial models) are more accurate and give a richer picture of the determinants of adolescent sexuality. At its most basic level, genetics determines the timing of puberty and sexual arousal. Early maturation is predictive of early sexual arousal and early sexual initiation.

Neither phenomena (the timing of puberty and early sexual arousal) are given due respect in policy or programming related to providing adolescent sexual and reproductive health. Given this basic reconceptualization of adolescent sexuality, which is informed by our understanding of genetics, fundamental change in the way adolescent sexuality is viewed and responded to, is in order.

There is no question that a great deal of work remains to be done before we will understand the mechanism and function of the genetic transfer of behavior. We know of its existence, but we do not know the process. What we can determine, that may be even more important, is what proportion of behaviors are shaped by genetic influences and what behaviors are shaped by environmental influences.

Understanding the role of genetics will allow us to focus on environmental influences that can be modified. For example, using this knowledge, we could (but would not want to) use poor nutrition to delay puberty. This would have what some perceive as a positive effect because it also delays sexual arousal and sexual initiation. We also do not want to treat an adolescent involved in an early sexual experience as a predelinquent.

This response has been shown to be a self-fulfilling prophecy. Instead, a reconceptualized view of adolescent sexuality informed by our understanding of genetics will allow us to focus scarce resources in the areas of sexual and reproductive health where we can improve outcomes and trajectory for the adolescent and the children of adolescent mothers.

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