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Age at First Birth and Fathers' Subsequent Health: Evidence From Sibling and Twin Models

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Using a sample of 540 siblings and twins from the National Survey of Midlife Development in the United States, this study examines the relationship between the age at which men become biological fathers and their subsequent health. The analysis includes both between-family models that treat brothers as independent observations and within-family models that account for unobserved genetic and early-life environmental endowments shared by brothers within families. Findings indicate that age at first birth has a positive, linear effect on men's health, and this relationship is not explained by the confounding influences of unobserved early-life characteristics. However, the effect of age at first birth on fathers' health is explained by men's socioeconomic and family statuses. Whereas most research linking birth timing to specific diseases focuses narrowly on biological mechanisms among mothers, this study demonstrates the importance of reproductive decisions for men's health and well-being.

Keywords: age at first birth; fatherhood; fixed-effects models; health; midlife

Does age at first birth have long-lasting implications for parental health? This question has been traditionally addressed from a biomedical perspective focusing almost exclusively on women (see Mirowsky, 2002; Mirowsky & Ross, 2002, for exceptions). In biomedical epidemiology, women's age at first birth has been shown to either increase or lower the risk for certain diseases, such as breast cancer (Colditz, 1993), ovarian cancer (Whiteman, Siskind, Purdie, & Green, 2003), myocardial infarction (Palmer, Rosenberg, & Shapiro, 1992), and ischemic heart disease (Beard, Fuster, & Annegers, 1984). The neglect of men in this line of research suggests that because men do not experience pregnancy, parturition, and lactation, then birth timing is unrelated to fathers' health.

Yet, age at first birth and men's health may be connected by social and psychological mechanisms. Birth timing may affect men's well-being via psychosomatic channels. Therefore, the influence of age at first birth on fathers' health can be explored with generalized measures of physical well-being. Using several indicators of health, including perceived health, energy and fitness, and the sum of chronic conditions, Mirowsky (2002) showed a positive linear association between fathers' age at first birth and subsequent health. Similarly, Mirowsky and Ross (2002) documented that later age at first birth was associated with fewer depressive symptoms among fathers.

The present study uses a subsample of sibling and twin dyads from the National Survey of Midlife Development in the United States (MIDUS) to examine the impact of age at first birth on men's health. The analysis explores the extent to which men's adult socioeconomic resources and family roles account for the association between age at first birth and fathers' health. This study extends previous research by considering early-life factors that can potentially confound the association between age at first birth and health. The analysis includes a wide array of retrospective measures of family background, early-life health, and relationships with parents. In addition to these observed measures, the study uses fixed-effects sibling and twin models to account for unobserved genetic and environmental early-life endowments shared by brothers within families.

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Following Mirowsky (2002), this study's conceptual framework integrates social epidemiological and life-course approaches. A social epidemiological approach to examining health problems focuses on diffuse social conditions, such as social class, that are associated with a broad array of diseases and symptoms. A life-course approach emphasizes both temporally proximate and distal antecedents of an individual's health status (Pearlin & Skaff, 1996). Synthesizing these two approaches, age at first birth is conceptualized here as an important component in the life-course processes of cumulative advantage and disadvantage, and a pivotal element in the associations between biological and psychosocial factors (Mirowsky, 2002; Stein & Susser, 2000).

Psychosocial Mechanisms Linking Age at First Birth and Health

Age at first birth is strongly affected by one's early life resources and experiences, particularly parents' socioeconomic resources (Hofferth, Reid, & Mott, 2001; Williams, McGee, Olaman, & Knight, 1997). Birth timing, in turn, affects one's adult socioeconomic and family statuses. Early nonmarital fatherhood was shown to have negative economic consequences. Men who have children prior to marrying leave school earlier, have lower earnings, work fewer weeks per year, and are more likely to live in poverty than their peers who postpone fatherhood (Nock, 1998). Conversely, delayed parenthood can be an effective strategy for accumulating human capital and successfully balancing the demands of career and family (Blossfeld & Huinink, 1991). Highly educated individuals are more likely to enjoy financial stability and rewarding careers, which are conducive to superior health outcomes (Grzywacz & Dooley, 2003). Beneficial health consequences of education have been widely reported as well (Ross & Wu, 1995). Consistent with this, Mirowsky (2002) reported that men derive health benefits from delaying the transition to parenthood largely due to salutary socioeconomic consequences and career advantages of the delay.

Marital status and family relationships in adulthood also may account for the association between age at first birth and health. Early first birth tends to be associated with premarital parenthood, unstable marriage, and high subsequent fertility (Heaton, 2002; Quesnel-Vallée & Morgan, 2003). Such conditions may create enduring strains that adversely affect parental health (Evenson & Simon, 2005; McLanahan & Adams, 1987). Men who have children early in life are more likely to be unmarried and to have less contact with their children than men who become fathers later (Casper & Bianchi, 2002). Extensive research documents that marital status and family relationships are fundamental to men's health (Strohschein, McDonough, Monette, & Shao, 2005; Umberson, 1992; Umberson, Williams, Powers, Liu, & Needham, 2006). Therefore, age at first birth can be associated with men's health indirectly, via the nature and quality of one's family relationships.

Thus, it is hypothesized that age at first birth is related positively to men's subsequent health, and this relationship is at least partly due to men's socioeconomic achievement and family statuses. In addition, physical outcomes associated with birth timing may reflect early-life factors that affect both age at first birth and men's subsequent health. Family-level approaches are particularly useful for exploring the potentially confounding effects of early-life characteristics.

Family Influences on the Association Between Age at First Birth and Health

The association between age at first birth and health observed in previous studies may reflect genetic and early-life environmental endowments that affect both age at first birth and health and thus may create a spurious relationship between parenthood and well-being (Kohler, Behrman, & Skytthe, 2005). Research has identified early-life family influences that can potentially influence both the timing of first birth and health.

First, children of teenage parents tend to enter parenthood earlier themselves than children of older parents, and there appears to be a similar intergenerational continuity of later parenthood (Barber, 2001; Kahn & Anderson, 1992). Children of teenage parents may fare worse as adults than children of older parents with respect to health and healthy behaviors (Barber, 2001).

Second, individuals who experienced sensitive and responsive parenting in childhood tend to report higher levels of health and well-being in adulthood (Taylor, Repetti, & Seeman, 1997). Parenting behaviors also influence children's attachment styles—the ways children think about their relationships with primary caregivers (Bowlby, 1988). An insecure attachment style is related to offspring's early maturation, earlier sexual activity, and a greater number of births, whereas secure attachment is associated with later maturation and the ability to form lasting bonds with intimate partners (Belsky, Steinberg, & Draper, 1991; Hazan & Shaver, 1987).

Third, health in childhood and adolescence predicts both adult health (Haas, 2007) and the likelihood of marrying and remaining married (Pudrovska & Carr, 2005). Fourth, early-life adversities, including economic hardship and parental divorce, can lead to offspring's enduring health problems (McLeod, 1991; Pudrovska, Schieman, Pearlin, & Nguyen, 2005). In contrast, higher socioeconomic status (SES) in childhood has salutary long-term consequences for men's health and survival (Hayward & Gorman, 2004). Similarly, parental divorce and low SES are associated with an increased risk for offspring's teen parenthood (Kahn & Anderson, 1992). Conversely, men who were born to parents with higher levels of education and income are more likely to postpone parenthood to realize their own educational and professional aspirations (Sewell & Hauser, 1975).

Finally, the relationship between age at first birth and health can be influenced by genetically transmitted attributes and predispositions. Certain genetic dispositions are associated both with a higher level of psychological well-being and a greater probability of being in a partnership and having a larger number of children (Kohler et al., 2005). Recent research suggests that at least two genetic variants may be related both to well-being and to reproductive behaviors: a functional polymorphism in the promoter region of the serotonin transporter gene (5-HTTLPR) and the dopamine D4 receptor exon III repeat (DRD4). 5-HTTLPR and DRD4 are associated with the frequency of sexual activity among middle-aged men (Hamer, 2002) and age at first sexual intercourse (Guo & Tong, 2006). These two polymorphisms also are related to psychological traits and well-being (Lesch et al., 1996; Tochigi et al., 2006).

A Family-Level Approach to Exploring Adult Health

An ideal examination of how heredity and early-life environmental factors influence outcomes in adulthood would require both direct and prospective measures of genetic and environmental early-life endowments. Because few data sources have such measures, researchers have relied on alternative approaches. The use of retrospective reports of early-life characteristics is a common practice in survey research. The strength of the MIDUS data set is that it contains detailed retrospective information about early-life factors, including sociodemographic characteristics, parental affection, and adolescent health. Thus, it is possible to assess directly the extent to which these childhood factors account for the association between age at first birth and adult health.

Early-life endowments also may be taken into account indirectly. One approach is to use fixed-effects sibling models; these models compare siblings from the same family to eliminate the influences of unobserved environmental and genetic factors shared by individuals within families. The fixed-effects model can be viewed as a within-family model because it compares members of the same family to each other.

The limitation of the fixed-effects approach is that it cannot account for genetic and environmental influences that are not shared by family members. Yet, this limitation can be overcome to some extent with the MIDUS data that have retrospective measures of nonshared early-life characteristics. In addition, the sample contains brother dyads of different degrees of genetic similarity: full nontwin siblings, monozygotic (MZ) twins, and dizygotic (DZ) twins. If the association between age at first birth and health is partly due to 50% of genes not shared by full nontwin siblings and DZ twins, then the effect of age at first birth should be weaker among identical twins who share 100% of their genes than among nontwin siblings and fraternal twins.

In sum, this study examines the association between age at first birth and fathers' subsequent health. Furthermore, the analysis explores how the health impact of birth timing changes when men's adult socioeconomic and family statuses, retrospectively reported early-life factors, and unobserved genetic and environmental influences are taken into account.

Methods

Data

The Midlife Development in the United States (MIDUS) is a study of noninstitutionalized, English-speaking adults in the coterminous United States, aged 25 to 74 years. The data were collected in 1995 to 1996. The MIDUS sample comprises several subsamples, including a national random-digit dialing

Table 1. Descriptive Statistics for the Study Variables: Male Siblings and Twins, Midlife Development in the United States

	Sibling Dyad (n = 150)		MZ Twin Dyad (n = 218)		DZ Twin Dyad (n = 172)	
Variable	*M*	*SD*	*M*	*SD*	*M*	*SD*
Number of chronic illnesses	2.35	2.47	1.65	1.86	1.95	1.90
Age at first birth	26.04	4.14	25.25	4.81	25.48	4.88
Age at the time of the interview	52.37	11.68	46.83	10.56	46.79	11.88
Race						
White (Yes = 1)	0.94		0.86		0.89	
Black (Yes = 1)	0.02		0.10		0.09	
Other race (Yes = 1)	0.04		0.04		0.02	
Early-life factors						
Two-parent family (Yes = 1)	0.80		0.78		0.80	
Father's education[a]	5.05	3.08	4.57	2.53	4.34	2.36
Mother's education[a]	5.11	2.50	4.50	2.17	4.58	2.31
Family was on welfare (Yes = 1)	0.02		0.06		0.10	
Self-rated physical health at age 16	4.50	0.79	4.60	0.63	4.49	0.75
Self-rated mental health at age 16	4.39	0.89	4.38	0.81	4.27	0.87
Maternal affection scale	3.25	0.53	3.30	0.57	3.23	0.62
Paternal affection scale	2.86	0.76	2.78	0.69	2.82	0.69
Socioeconomic status						
Net worth (natural log)	6.36	2.71	5.53	3.41	4.92	3.60
Currently employed (Yes = 1)	0.65		0.72		0.76	
Education						
Less than high school (Yes = 1)	0.05		0.10		0.19	
High school diploma (Yes = 1)	0.21		0.29		0.28	
Some college (Yes = 1)	0.30		0.34		0.31	
College or postgraduate (Yes = 1)	0.44		0.27		0.22	
Family statuses						
Number of children	2.70	1.36	2.65	1.36	2.44	1.34
Currently married (Yes = 1)	0.89		0.85		0.89	

NOTES: MZ = monozygotic; DZ = dizygotic; *M* = mean; *SD* = standard deviation. *N* = 540.
a. Range: 0 = *no formal education*; 12 = *graduate degree*.

(RDD) sample (n = 3,487), siblings of individuals from the RDD sample (n = 950), and a national RDD sample of twin pairs (n = 1,914). The main respondents were selected from working telephone banks. For each household contacted, a list was generated of all people between 25 and 74 years old, and a respondent was randomly selected. Older people and men were oversampled.

Of the main respondents who reported having one or more siblings, the survey team randomly selected 529 people. Complete telephone interviews were obtained for 950 full nontwin biological siblings of the selected main respondents. Respondents in the twin-pair sample are unrelated to main respondents and their siblings. Twin dyads were recruited in a two-part sampling design. The first part of the twin sample design involved screening a representative national sample of approximately 50,000 households for the presence of twins. The 14.8% of respondents who reported the presence of twins in the family were asked whether it would be possible for the research team to contact the twins to invite their participation in the survey. The 60% of the respondents who gave such permission were then referred to the MIDUS recruitment process. Respondents in all three subsamples participated in a 30-min telephone interview and completed a self-administered questionnaire. In addition, the twin subsample was administered a short screener to assess zygosity and other twin-specific information.

The analysis in this study was restricted to sibling and twin dyads, both members of whom were male and had at least one biological child. The final sample comprises 75 nontwin full sibling dyads, 109 MZ twin dyads, and 86 DZ twin dyads. Summary statistics characterizing the sample are presented in Table 1.

Measures

Dependent Variable

Health was measured as a count of chronic illnesses that a respondent experienced or was treated for in the 12 months prior to the interview, including asthma, arthritis, thyroid disease, diabetes, hypertension, autoimmune disorders, heart conditions, and ulcers. Out of 29 possible conditions, the count of reported illnesses in this study ranges from 0 to 12. Factor analysis reveals that indicators of specific chronic conditions load on a single factor with an eigenvalue of 1.22 after orthogonal varimax rotation. The physical health scale has a relatively high internal consistency ($\alpha = .72$). As shown in Table 1, siblings had, on average, 2.35 chronic illnesses in the 12 months prior to the interview, MZ twins had 1.65 illnesses, and DZ twins reported an average of 1.95 illnesses.

Independent Variable

The key focal predictor variable in this analysis is age at first birth, measured as a man's age (in years) when his first biological child was born. Table 1 indicates that an average age at first birth is 26 years among nontwin siblings, 25.3 years among MZ twins, and 25.5 years among DZ twins. The oldest age at first birth in this sample is 53 years old, and the youngest is 16 years old.

Sociodemographic Characteristics

All models control for the respondent's age at the time of the interview. Because the MIDUS survey oversampled older and middle-aged adults, individuals aged 40 years or older constitute roughly 70% of the MIDUS subsample used in our study. Siblings tend to be somewhat older than twins: The average age is 52 years among nontwin siblings and 46.8 years among twins.

Three mutually exclusive dummy variables reflecting race were included in the between-family models: "White" (the reference category), "Black," and "Other race." As indicated in Table 1, the majority of men in this sample are White: specifically, 94% of nontwin siblings, 86% of MZ twins, and 89% of DZ twins.

Adult Socioeconomic and Family Statuses

Adult statuses and roles are a possible pathway that could account for an observed association between age at first birth and health; thus this analysis includes indicators of socioeconomic status and family roles. Education is represented with four mutually exclusive categories: "less than high school," "GED or high school diploma" (the reference category), "some college," and "college degree or postgraduate education." Net worth reflects the respondent's total household assets. Employment status was coded 1 if a respondent was working for pay at the time of the interview.

Marital status is represented with a dummy variable coded 1 if a respondent was married at the time of the interview and 0 if unmarried. Because married men constitute nearly 90% of our subsample, the numbers of widowed, divorced, and never-married respondents were not sufficient to assess heterogeneity among the unmarried. The number of children reflects the number of all biological and nonbiological children that a respondent had by the time of the interview, which is, on average, between two and three children.

Early-Life Factors

To assess whether the relationship between age at first birth and adult health is spurious this analysis includes measures of early-life characteristics. A dichotomous measure of family structure was coded 1 if a respondent reported living with both biological parents in childhood and 0 if only one or no biological parents were present. Indicators of father's and mother's education reflect the highest grade of school the father or the mother completed, ranging from 0 = *no school/some grade school* to 12 = *doctoral or professional degrees*. A binary measure of welfare is coded 1 if there was at least one period during a respondent's childhood and adolescence when his family was on welfare. Early-life physical and mental health was assessed with two separate questions: "Now, think about when you were 16 years old. Was your [physical/mental] health at that time: (1) poor; (2) fair; (3) good; (4) very good; (5) excellent?" Maternal and paternal affection scales ($\alpha = .91$ and .92, respectively) are based on seven questions that were asked separately about each of the respondent's parents, including the quality of relationships with this parent and the amount of understanding, love, affection, time, and attention this parent provided when the respondent was a child. Each scale was constructed by calculating the mean of the respective seven items.

Analytic Strategy

The analysis includes two types of models. First, simple Poisson regression models are used to evaluate variation in the association between birth timing and health across families. These models are between-family models because they compare individuals across families and do not account for common characteristics shared by members of the same family. The between-family model can be represented by the following equation:

$$\mu_i = \exp\,[(\beta_0 + \beta_1 X_{i1} + \beta_2 X_{i2} + \beta_3 X_{i3} + \ldots + \beta_n X_{in})],$$

where μ_i is the expected count of illnesses, or the incidence rate (Long & Freese, 2003). Robust standard errors are used to account for the fact that observations drawn from the same family are not independent within clusters.

The relative explanatory power of three blocks of variables is assessed by entering each block separately into respective equations: early-life factors (Model 2), SES characteristics (Model 3), and marital and parental statuses (Model 4). Model 5 adjusts for all observed explanatory variables simultaneously. The effect of age at first birth in Models 2 to 5 is compared to baseline Model 1 that adjusts only for age, race, and genetic relatedness.

Next, to factor out potentially confounding effects of unobserved endowments shared by siblings or twins within families, fixed-effects Poisson models are estimated. These are within-family models because they compare siblings from the same family:

$$\mu_{ij} - \mu_j = \exp\,(\beta_0 - \beta_0) + \beta_1\,(X_{ij1} - X_{j1}) + \beta_2\,(X_{ij2} - X_{j2}) + \ldots + \beta_n\,(X_{ijn} - X_{jn}),$$

where μ_{ij} is the expected count of illnesses for brother i from family j; β_0 is a fixed term capturing the influence of unobserved factors related to family j and shared by both brothers from family j; X_{ijn} denotes the values of independent variables for each sibling; and μ_j and X_{jn} are overall family means that are subtracted from individual values.

Because fixed-effects models eliminate the effects of characteristics that are invariant between siblings, these variables cannot be included as predictors in the equation. Therefore, race, genetic similarity, retrospectively measured shared aspects of family background, and twins' ages were not included as predictors in fixed-effects models because their main effects cannot be estimated. Yet it is still possible to estimate the interactive effects between sibling-invariant and sibling-varying measures (Johnson, 1995). Thus, to examine whether the effect of age at first birth on health depends on the degree of brothers' genetic relatedness, the type of a brother dyad is added to fixed-effects models as a component of the respective interaction terms.

If the analysis reveals that the relationship between age at first birth and health is explained by men's socioeconomic and family statuses, it will support the hypothesis that men's age at first birth is linked to their health via psychosocial processes. If the effect of age at first birth on men's health declines in the within-family models compared to the between-family models, it will provide evidence that the association between age at first birth and health is partly due to unobserved genetic and environmental early-life influences shared by brothers within families. Conversely, if the effect of age at first birth is the same in the between- and within-family models, it will suggest that unobserved shared early-life endowments do not affect the relationship between first birth timing and men's health.

Results

Findings from the between-family models comparing individuals across families are presented first, followed by results from the within-family models that compare brothers from the same family. Both between- and within-family models include retrospective measures of early-life factors and interaction terms between age at first birth and the type of a brother dyad.

Between-Family Models

Model 1 in Table 2 reveals that age at first birth is associated negatively with the number of chronic conditions. Men who became fathers later report fewer illnesses than men who made the transition to fatherhood earlier in life. The relationship is linear as indicated by fitting and comparing both quadratic and spline models (not shown). The health impact of age at first birth is significant net of age, race, and genetic relatedness. Nonsignificant interactions between age at first birth and the type of a sibling dyad (not shown) indicate that the effect of birth timing on health is similar regardless of brothers' genetic similarity.

Model 2 of Table 2 adjusts for retrospectively reported early-life factors. The effect of age at first birth on health declines by roughly 15% but remains statistically significant. As shown in Model 3 of Table 2, after adjusting for men's socioeconomic status and resources, the coefficient for age at first birth declines by 31% compared to baseline Model 1

Table 2. Coefficients From Poisson Regression Models Predicting the Count of Chronic Illnesses Among Male Siblings and Twins: Midlife Development in the United States

Variable	Model 1	Model 2	Model 3	Model 4	Model 5
Age at first birth	–.026*	–.022*	–.018	–.024*	–.019
	(.011)	(.011)	(.011)	(.011)	(.011)
Age at interview	.012**	.014***	.013*	.012*	.013*
	(.004)	(.004)	(.005)	(.005)	(.005)
Race					
White (omitted category)					
Black (Yes = 1)	–.700***	–.751***	–.769***	–.708***	–.888***
	(.127)	(.146)	(.128)	(.128)	(.142)
Other race (Yes = 1)	–.115	–.237	–.280	–.126	–.360
	(.253)	(.259)	(.219)	(.245)	(.213)
Genetic relatedness					
Monozygotic twins (omitted category)					
Full siblings (Yes = 1)	.258*	.273*	.301**	.261*	.317**
	(.117)	(.113)	(.112)	(.117)	(.110)
Dizygotic twins (Yes = 1)	.158	.148	.082	.164	.113
	(.108)	(.104)	(.107)	(.109)	(.106)
Early-life factors					
Two-parent family (Yes = 1)	—	–.182	—	—	–.118
		(.117)			(.121)
Father's education	—	.014	—	—	.021
		(.021)			(.020)
Mother's education	—	–.036	—	—	–.038
		(.025)			(.026)
Family was on welfare (Yes = 1)	—	–.038	—	—	–.127
		(.215)			(.203)
Self-rated physical health at age 16	—	.097	—	—	.119
		(.076)			(.075)
Self-rated mental health at age 16	—	–.169*	—	—	–.159*
		(.069)			(.067)
Maternal affection scale	—	–.143	—	—	–.152*
		(.087)			(.076)
Paternal affection scale	—	–.085	—	—	–.069
		(.074)			(.069)
Socioeconomic status					
Net worth (natural log)	—	—	–.019	—	–.014
			(.013)		(.013)
Currently working for pay (Yes = 1)	—	—	.011	—	.037
			(.148)		(.150)
Education					
High school diploma (omitted category)					
Less than high school education (Yes = 1)	—	—	.554***	—	.461***
			(.148)		(.145)
Some college education (Yes = 1)	—	—	.038	—	.042
			(.118)		(.114)
College or postgraduate education (Yes = 1)	—	—	–.020	—	.040
			(.132)		(.136)
Family statuses					
Married (Yes = 1)	—	—	—	–.092	.036
				(.130)	(.132)
Number of children	—	—	—	.005	–.001
				(.038)	(.040)
Constant	–.028	1.112	–.027	.045	1.032

NOTES: Each cell contains unstandardized regression coefficients with standard errors (in parentheses) robust to clustering of individuals within families. $N = 540$.
*$p < .05$. **$p < .01$. ***$p < .001$ (two-tailed tests).

Table 3. Coefficients From Fixed-Effects Poisson Regression Models Predicting the Count of Chronic Illnesses Among Male Siblings and Twins: Midlife Development in the United States

Variable	Model 1	Model 2	Model 3	Model 4	Model 5
Age at first birth	–.025*	–.021*	–.022*	–.019	–.009
	(.011)	(.011)	(.011)	(.012)	(.012)
Nonshared early-life factors					
Self-rated physical health at age 16	—	.075	—	—	.177*
		(.073)			(.080)
Self-rated mental health at age 16	—	–.147*	—	—	–.202**
		(.060)			(.066)
Maternal affection scale	—	–.169	—	—	–.208
		(.115)			(.125)
Paternal affection scale	—	–.019	—	—	.029
		(.096)			(.106)
Socioeconomic status					
Net worth (natural log)	—	—	–.008	—	.007
			(.016)		(.017)
Currently working for pay (Yes = 1)	—	—	–.022	—	–.007
			(.076)		(.078)
Education					
High school diploma (omitted category)					
Less than high school education (Yes = 1)	—	—	.624***	—	.610***
			(.178)		(.188)
Some college education (Yes = 1)	—	—	.110	—	.197
			(.137)		(.144)
College or postgraduate education (Yes = 1)	—	—	.021	—	–.009
			(.173)		(.188)
Family statuses					
Married (Yes = 1)	—	—	—	–.047	.044
				(.141)	(.157)
Number of children	—	—	—	.055	.065
				(.038)	(.042)

NOTES: Each cell contains unstandardized regression coefficients with standard errors (in parentheses). *N* of observations = 540; *N* of groups = 270.
$^{*}p < .05$. $^{**}p < .01$. $^{***}p < .001$ (two-tailed tests).

and becomes nonsignificant, thus indicating that the health advantage of men who delay fatherhood is explained by adult socioeconomic status. In contrast, when men's marital status and the number of children are controlled in Model 4, the effect of age at first birth remains basically unchanged compared to the baseline model, which suggests that health benefits of delayed parenthood are not explained by men's family status in the between-family models.

Within-Family Models

Table 3 presents the results from fixed-effects models that take into account unobserved genetic and environmental early-life endowments shared by brothers within families. Model 1 of Table 3 shows that when brothers from the same family are compared to each other, the effect of age at first birth does not change compared to Model 1 in Table 2 that treats brothers as if they were unrelated individuals. This suggests that the association between age at first birth and health is not a spurious reflection of unobserved genetic and environmental early-life factors shared by brothers.

Moreover, a model including interaction terms (not shown) reveals that the association between age at first birth and health is similar across three types of family dyads (i.e., full nontwin siblings, identical and fraternal twins). This implies that genetic endowments not shared by brothers are unlikely to influence the association between age at first birth and health. If nonshared genetic endowments were partly responsible for this association, the effect of age at first birth would have been significantly weaker among identical twins who share 100% of their genes than among full siblings and fraternal twins who share on average 50% of their genes.

Model 2 of Table 3 adjusts for early-life factors that are not shared by brothers: respondents' own health in adolescence and perceived parental affection. Shared environmental influences, such as parental education and family structure, cannot be included in the fixed-effects models because these models are unable to estimate the effects of covariates that do not vary between brothers. After adjustment for nonshared early-life factors, the effect of age at first birth on men's health declines by 16% (from –.025 in Model 1 to –.021 in Model 2), yet it retains statistical significance. Thus, even a simultaneous adjustment for unobserved shared endowments and observed nonshared early-life factors does not account for the effect of age at first birth on men's health.

As indicated in Model 3 of Table 3, men's adult socioeconomic status does not explain the health impact of age at first birth in the within-family model. In contrast, the association between age at first birth and health was explained by socioeconomic factors in the respective between-family model (Model 2 of Table 2). This suggests that men's socioeconomic status is more important in explaining variation among men from different families than in explaining differences between brothers within the same family.

After adjustment for marital status and the number of children in Model 4 of Table 3, the coefficient for age at first birth declined by roughly 25% and became nonsignificant, although the effect size was still comparable to Models 2 and 3 of Table 3. Thus, in the within-family analysis, the association between age at first birth and men's health is partly explained by family statuses. Specifically, men who had their first child after age 25 and, especially, age 30 were more likely to be married at the time of the interview and had fewer children than men who became fathers earlier. In turn, marriage and moderate family size are related positively to men's health, in our data. Compared to the respective between-family model in Table 2, family statuses explain a greater proportion of the association between age at first birth and fathers' health. This suggests that when two brothers from one family are compared to each other, family statuses are more important than in comparisons of unrelated individuals across families. Finally, Model 5 of Table 3 shows that all explanatory variables combined reduce the effect of age at first birth from –.025 in the baseline model to –.009 in the full model (or by 64%).

Discussion

This study used sibling and twin data to examine the relationship between age at first birth and health among fathers. The analysis reveals that later age at first birth is associated with fewer subsequent chronic illnesses. This relationship is linear, indicating that the longer men wait to become fathers, the better health they can expect in the long term. In addition, the analysis with the MIDUS sample reported here was replicated using sibling data from the Wisconsin Longitudinal Study (available upon request), and the findings were remarkably consistent in the two data sets. Moreover, Mirowsky (2002) documented a similar monotonic increase in fathers' health with advancing age at first birth, with no noticeable upper limit to the beneficial postponement of parenthood. Yet, it should be noted that fatherhood is viewed in these studies as the birth of the first biological child rather than active father involvement.

The effect of age at first birth is similar in the between- and within-family models, suggesting that this association is unlikely to be driven by unobserved early-life genetic and environmental endowments shared by brothers. Similarly, the association between age at first birth and fathers' health is not accounted for by nonshared genetic influences: The effect of age at first birth on health is virtually the same among brothers who share 100% of their genes and among brothers who share only 50% of their genetic makeup.

Research on men's well-being has typically focused on their work and socioeconomic roles rather than family roles, and this is a fairly limited view of men's lives (Eggebeen & Knoester, 2001). This study underscores the importance of reproductive decisions for men's health. In the between-family models that compare men across families, the health premium of delayed fatherhood is explained by men's SES. Men benefit from delaying their transition to parenthood largely due to salutary socioeconomic consequences and career advantages of the delay (Mirowsky, 2002). Men who delay the transition to fatherhood also tend to accumulate education, wealth, and other socioeconomic resources. In turn, higher SES is associated with better health (Preston & Taubman, 1994). Being an older father thus may carry long-term health advantages, given the beneficial educational, financial, and professional outcomes associated with later age at first birth. Therefore, studies of men's health should examine the interplay of family and work trajectories over the life course because these domains are closely intertwined in men's lives—just as they are in women's.

Yet work and family decisions in adulthood are made against the backdrop of social constraints. In particular, adult socioeconomic conditions are partly shaped by early-life factors and resources of the family of origin (Hayward & Gorman, 2004). This analysis shows that socioeconomic status is more important in explaining the effect of age at first birth among men from different families than in accounting for differences between brothers within the same family. When unobserved family background characteristics are taken into account in the within-family models, men's adult SES and resources have no independent effect on the relationship between age at first birth and health. This finding can be explained by the fact that men's adult SES is strongly influenced by characteristics of the family of origin, largely because men with advantaged social background have high educational aspirations (Kiernan & Diamond, 1983; Sewell & Hauser, 1975; Stevens-Simon & Lowy, 1995). High educational and occupational aspirations cultivated in the family of origin may influence both delayed childbearing and socioeconomic achievement in adulthood.

Even though the MIDUS sample used in this study includes relatively few low-income and minority fathers, these findings may be relevant for disadvantaged populations and have important implications for policy and practice. Early childbearing and its negative socioeconomic consequences may contribute to the poorer health of men in low-income populations. The negative socioeconomic antecedents and consequences of early fatherhood reinforce each other. Young men with lower earnings and weaker labor force commitment are more likely to have children before marriage (Nock, 1998) and then suffer the adverse socioeconomic consequences of early and nonmarital fatherhood (Heath, McKenry, & Leigh, 1995; Nock, 1998). Low-income nonresidential fathers face multiple stressors, in particular, economic hardship, that elevate men's depressive symptoms and make it difficult to balance work and family responsibilities (Anderson, Kohler, & Letiecq, 2005). Thus, public health campaigns promoting education among men from economically disadvantaged background and encouraging them to achieve economic self-sufficiency prior to family formation may have long-range implications for men's health. Such programs may not only increase fathers' economic security and reduce their children's poverty (Anderson et al., 2005) but also improve men's health in the long term.

Finally, this study suggests that men's health researchers can benefit from considering both within- and between-family differences. Sociological and demographic research on health disparities underscores the importance of early-life influences on adult health (Elo & Preston, 1992; Hayward & Gorman, 2004). Yet, although the importance of family influences is well documented, most research on health and well-being continues to rely on individual-level data that treat individuals as independent observations rather than family members. This study shows the importance of using family-level models. Specifically, men's adult family statuses contribute more to the explanation of the health impact of birth timing when brothers are compared to each other than when unrelated individuals are compared across families. In other words, the importance of men's family statuses becomes evident only in the within-family model that accounts for unobserved genetic and environmental characteristics shared by brothers. This suggests that between-family models that do not take into account heterogeneity across families may underestimate the role of men's marital and parental statuses as important psychosocial pathways linking age at first birth and subsequent health.

Limitations and Future Directions

To take into account early-life factors, this study relied on the fixed-effects approach and on retrospective measures that may be subject to cognitive biases (Schaefer & Presser, 2003). Ideally, prospective measures in the context of longitudinal panel designs are necessary to account for the effects of early-life variables; yet, such studies are very difficult to implement. Moreover, recent research shows that retrospective measures of childhood health (Haas, 2007) and early-life hardships (Pudrovska et al., 2005) tend to have good reliability.

All men in this study identified themselves as heterosexual, and most respondents were married. An important direction for future research is to examine how the timing of the transition to fatherhood affects the health of gay men and other men who enter fatherhood via nonbiological pathways, such as adoption or marriage to a woman with children from a prior relationship. Furthermore, most respondents in this sample are White and of relatively advantaged social background. Yet the implications of age at first birth for fathers' health are likely to differ by race and class.

Future research should examine how the association between age at first birth and fathers' subsequent health is shaped by the intersection of men's race and SES. Finally, this study did not consider the nature or quality of father-child relationships or fathers' financial and social responsibilities to their children. It will be interesting to explore in future studies whether the effect of age at first birth is explained by fathers' actual involvement with children.

Conclusion

Age at first birth is related positively and linearly to men's health, and this relationship is explained by men's adult socioeconomic and family statuses. Thus, work and family roles are inextricably intertwined in men's lives, and reproductive decisions are important for men's health—just as they are for women's. Yet work and family decisions in adulthood may be constrained or facilitated by social background. Men's adult SES has no independent effect on the relationship between age at first birth and health once unobserved family background characteristics are taken into account. It is possible that early childbearing and its negative socioeconomic consequences may explain poorer health of men in low-income population subgroups. Therefore, policies and programs that promote education and encourage men from economically disadvantaged background to achieve economic self-sufficiency prior to family formation may improve men's health in the long term. Finally, this study shows that researchers of men's health can benefit from using family-level models rather than traditional individual-level approaches.

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