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Trivers–Willard at birth and one year: evidence from US natality data 1983–2001

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Trivers & Willard (TW) hypothesized that evolution would favour deviations from the population sex ratio in response to parental condition: parents in good condition would have more sons and parents in poor condition would have more daughters. We analyse the universe of US linked births and infant deaths to white mothers 1983–2001, covering 48 million births and 310 000 deaths. We find that (i) married, better educated and younger mothers bore more sons and (ii) infant deaths were more male if the mother was unmarried and young. Our findings highlight the potential role of offspring sex ratio as an indicator of maternal status, and the role of infant mortality in shaping a TW pattern in the breeding population.

Keywords: Trivers–Willard; human population; early life mortality; sex allocation

1. INTRODUCTION AND MOTIVATION

Trivers & Willard (1973; TW) hypothesized that evolution would favour systematic deviations from the population sex ratio—mothers in good condition would have more sons and mothers in poor condition more daughters. The argument was based on the observation that while the average number of offspring to males and females equalize,[1] the reproductive success of a male offspring tends to be more variable and resource sensitive. For instance, if each female bears two offspring, a daughter yields two grandchildren, while a son yields two times his number of female partners. Assuming that the number of partners depends on the condition of the son, and that this condition, in turn, is related to maternal condition, mothers in good condition obtain more grandchildren through sons. Conversely, for mothers in poor condition, daughters out-reproduce sons.[2]

We test the TW hypothesis using US natality data covering the period 1983–2001. This extends previous research in two ways. First, we compile the largest micro-dataset of birth records yet analysed in evaluating the TW hypothesis. Our analysis confirms the presence of a TW effect at birth with respect to maternal marital status, education and age, assuming for the latter that younger mothers are in better condition. Second, we investigate the sex differences in 310 000 infant deaths, and how these relate to maternal characteristics. We find that infant deaths reinforced the relationship between sex and socio-economic background found at birth.

TW conjectured that parental control over offspring mortality would be one mechanism. Obviously, the closer to conception, the lower the replacement cost of terminated offspring. Mortality *in utero* would for this reason be more advantageous than mortality after birth and most studies have focused on the sex ratio at birth.[3] However, barring pre-natal sex determination and elected abortions, the effects are small (see James 1987). Greater parental discretion can be exercised once the child is born, and here the empirical literature has mainly focused on developing countries where a combination of high infant mortality and pronounced son preference can make for dramatic departures from what could be considered biologically normal. For instance, a recent large-scale Indian study found sex ratios to be substantially more male when mothers were better educated (Jha *et al.* 2006).[4] Whether infant mortality varies in a way predicted by TW in societies with relatively neutral preferences for offspring sex is less well established.[5]

The TW hypothesis has generated a substantial empirical literature outside the social sciences (see Cameron 2004). Its relevance for human populations is, however, controversial (e.g. Freese & Powell 2001). *A priori*, there are several reasons for the TW prediction to be weaker or not hold. We are a species with not only high parental investment, but also unusually high *paternal* investment. A high level of paternal investment reduces the scope for TW, because once the father provides more than gametes, the provision for one offspring reduces the ability to provide for another.[6] To further understand this, consider the case where male parental investment is high, for instance because men provide parenting in the form of own time. In this case, men without previous children are more desirable than men who are already fathers, and monogamy may characterize equilibrium mating. If so, sons and daughters would provide the same number of grandchildren. At the opposite extreme, if men only provide gametes, as in the case of sperm donations, only bureaucratic rationing precludes desirable donors from fathering a large number of children. Women whose son could be that donor would do much better in terms of grandchildren with a son than a daughter. Material resources fall somewhere in between. Clearly, the wealth distribution is heavily skewed. Still, the extent to which reproductive success increases with socio-economic status (SES) is modest.[7] It may be that a preference for time involvement in the rearing of children and a desire to pass on wealth reduces the extent to which wealthy men convert wealth into offspring. On a related note, the low

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fertility and mortality of contemporary Western societies call into question the extent to which behaviour is governed by fitness maximization.

The social sciences have mainly focused on a converse but related question: how a child's sex impacts parental investments. In addition to the vast literature documenting son-biased parental investments ranging from asset devolution to food allocation, there has recently been an interest in the possibility that sons prompt greater paternal commitment of resources in the contemporary United States (for marital status, see Dahl & Moretti (2004) and Lundberg & Rose (2003); for labour supply, see Lundberg & Rose (2002)). Studies that explicitly engage the TW hypothesis include Gaulin & Robbins (1991; the USA) and Koziel & Ulijaszek (2001; Poland), both these studied the length of breast-feeding.

Studies by Abernethy & Yip (1990) and Norberg (2004) are the ones most closely related to ours. Abernethy & Yip (1990), using linked birth–death records 1976–1983 for the state of Tennessee, found the pattern for post-neonatal infant deaths to be supportive of the TW hypothesis. Norberg (2004) focused on maternal partnership status at the time of conception as a determinant of the sex ratio, relying on survey data during the period 1959–1998. While she considered other markers of SES such as education and household income (in a multivariate analysis), they were not consistently significant.

2. DATA

We analyse National Center for Health Statistics (NCHS) Vital Statistics Birth Cohort Linked Birth/Infant Death Data, available for the years 1983–1991 and 1995–2001. These data contain information extracted from the universe of birth certificates and in the case of an infant death, information from the corresponding death certificate. We have information on the sex of the child, maternal characteristics, whether the child survived its first birthday and, if not, age at death. We restrict the analysis of singleton births to white mothers—approximately 80% of births.

Details about variable construction are in appendix A.

3. MATERIAL AND METHODS

Whereas a person's sex is immutable, we adopt a perspective germane to the TW hypothesis: sex of offspring being endogenous to the socio-economic and biological characteristics of the mother. As mentioned, TW hypothesized that the mechanism involved sex-biased mortality modulated by the condition of the parent(s), or mother for the intrauterine environment. We have individual-level data and we therefore formulate the regression analysis in terms of sex of the individual as the dependent variable. While transitions are impossible at the individual level, our formulation is in keeping with the spirit of the TW hypothesis which invites us to consider whether the sex ratio of *survivors* may be influenced by parental characteristics.

Assuming no systematic variation in the sex ratio at conception, mortality *in utero* shapes the sex ratio at birth. We do not observe foetal deaths, but can study how the sex ratio at birth varies with condition. The linked birth–death records allow us to repeat this analysis for those who survived their first birthday. Moreover, since these data register infant deaths, we can explicitly study the extent to which mortality in the first year may be considered a proximate mechanism for a TW effect. In both the foetal and infant periods, heightened mortality among males borne by mothers in poor condition would lead to a positive association between maternal condition and male sex among survivors.

We start by estimating the following regression model:

$$\text{male}_i = \alpha_0 + \alpha_1 x_i + \alpha_2 h_i + \gamma_t + \epsilon_i, \tag{3.1}$$

where male_i is an indicator variable that takes on the value 1 if child i is male; x_i is a vector of socio-economic and biological characteristics capturing the condition of the mother (marital status, education and age); h_i is a vector of controls for the infant's health status at birth (indicator variables for gestation length and birth weight); and γ_t is a vector of birth-year indicator variables accounting for annual differences in the share male. The h vector seeks to control for the direct measures of the infant's health status at birth. Whether to include such variables or view them as a result of maternal condition (possibly captured by the x variables) is debatable. The compromise we strike is to include gestation length and birth weight (our results are not sensitive to their inclusion). These variables may be viewed merely as controls. For instance, boys are heavier than girls and may at a given birth weight suffer different mortality risk than girls. Alternatively, these variables may be picking up information about the condition of the mother not accounted for by the x variables. For instance, mothers in poor condition may be more likely to deliver pre-term or low-birth-weight babies.

Under the TW hypothesis, for positive characteristics, we would expect the corresponding elements of α_1 to be positive (and vice versa). For instance, assuming that married mothers are in better condition than unmarried mothers, we would expect married mothers to be more likely to have a son, and, consequently, the coefficient on the married indicator variable to be positive.

We estimate equation (3.1) on three samples: all live births; all children who died in their first year; and all children who survived their first year. If mortality is a mechanism through which a TW effect is obtained, then we would expect the relationship between the probability that a child is male and, for instance, mother's marital status to be stronger in the population at age 1 than at age 0 (birth). Moreover, we would expect the relationship between sex and marital status to be the weakest, or negative, among infant decedents.

Whereas comparisons of the α_1 estimates obtained from the different populations can be informative, the statistical significance of such differences cannot be readily ascertained. To formally test the hypothesis that infant mortality operates as a determinant of the sex composition by SES, we estimate the following regression model:

$$\text{male}_i = \beta_0 + \beta_1 x_i + \beta_2 h_i + \gamma_t + \beta_3 \text{d}_i + \beta_4 \text{d}_i x_i + \beta_5 \text{d}_i h_i + \text{d}_i \gamma_t + \epsilon_i, \tag{3.2}$$

where d_i indicates whether infant i died. β_1 has the interpretation of the effect of x_i on offspring sex among infants who survived until age 1. Our focus is on β_4, the vector of coefficients on the interaction terms $\text{d}_i\, x_i$. Loosely speaking, specification (3.2) allows infant deaths to change the relationship between maternal characteristics x_i and offspring sex, and β_4 assesses whether this flexibility is important. Formally, the vector of point estimates $\hat{\beta}_4 = \hat{\alpha}_1 - \tilde{\alpha}_1$, where $\hat{\alpha}_1$ is the vector of coefficients obtained from estimating equation (3.1) on the subset of infant

decedents, and $\tilde{\alpha}_1$ is that obtained from estimating (3.1) on the subset of infant survivors. (Furthermore, $\tilde{\alpha}_1 = \hat{\beta}_1$.)

If infant mortality is consistent with TW, we would expect elements of β_4 to be negative if the variable signals good condition (and positive if the opposite). In other words, under the TW hypothesis, we expect mortality to reinforce a positive relationship between good condition and maleness among *survivors*.

The tendency for more males at birth will be reflected in positive estimates of parameters α_0 and β_0. Since males suffer higher infant mortality rates than females, we expect the coefficient on d, (β_3) to be positive. These three parameters—α_0, β_0 and β_3—fully capture average differences in births and infant deaths by sex. We turn now to the empirical results.

4. RESULTS

Results are generally supportive of the TW hypothesis. For the sex ratio at birth, married, better educated and younger mothers are more likely to bear sons.

Columns (1)–(3) of table 1 report results from estimating (1) by ordinary least squares. Column (1) shows the results from estimating (1) on the sample of all live births. Consistent with TW, married mothers were more likely to give birth to male offspring. The coefficient indicates that married mothers were 0.2% (1.022/(0.513×1000)) more likely to give birth to a son than unmarried mothers. The other direct measure of the economic circumstances of the mother is her education level. We find that lower education was associated with a more female sex ratio. For instance, relative to a mother with some college, a mother without a high school degree was approximately 0.6% (3.064/(0.513×1000)) less likely to bear a boy. We find that mothers in the age group of 15–19 were more likely to give birth to sons and mothers older than 35 were more likely to give birth to daughters (compared with mothers in the age group of 20–34). Biologically, younger women may be in better condition, an observation that would bring this finding in line with the TW hypothesis. The negative gradient is also consistent with the observation (noted by TW) that sons are a more risky parental investment.

If mortality operates in a manner consistent with TW, we would expect the patterns found among live births to be reinforced in the sample of 1-year-olds. The results largely confirm this hypothesis (column (3)). The coefficient on the married indicator variable is now 1.132 instead of 1.022, and, similarly, the absolute values of the coefficients on the education variables are larger. However, the coefficients on the age variables are attenuated.

Clearly, the difference between the coefficient estimates for equation (3.1) estimated on the sample of live births and the sample of 1-year-olds are driven by infant deaths. To investigate this further, column (2) reports the results from estimating the same model on the sample of infant deaths. For infant deaths to reinforce a TW pattern among survivors, it must be that the condition has a weaker or opposite effect on sex. This is also largely what we find. The mother's being married *lowered* the probability that a decedent was male by 0.94% (5.383/(0.573×1000)). While education failed to come in with the expected sign or significance, age now shows an interesting pattern: more advanced maternal age reduced the probability that a decedent was male.

Assuming that young mothers are biologically in good condition (and recall that they were more likely to give birth to sons) suggests that higher male infant mortality reflects young mothers being negatively selected with respect to social and economic characteristics. Conversely, despite a tendency for older mothers to bear female offspring, daughters were relatively more at risk in their first year if the mother was older than 35. The greater survival chances of boys suggest that the biological disadvantage from being borne by an older mother is countered by her more favourable socio-economic circumstances (see Royer (2004) and discussion thereof in §4).[8] Thus, maternal age provides an interesting example of biological and socio-economic conditions being negatively correlated, and it appears that with respect to foetal deaths (only observed indirectly) the biological advantage conferred by young age dominated the socio-economic disadvantage, whereas for infant deaths, socio-economic factors dominated.

While the coefficient estimates for infant decedents and survivors (columns (2) and (3), respectively) are different, whether the difference is statistically significant cannot be directly ascertained. Therefore, we now turn to estimating specification (2) on the whole sample of live births (infant survivors plus infant decedents), column (4). In this case, β_1 captures the effect of maternal condition, x_i, on survivors and β_4 (the vector of coefficients on the interaction terms $d_i\, x_i$) captures the additional effect from infant deaths. For instance, the marital status effect is almost five times as large for infant deaths as for survivors (5.383/1.132), and the difference is statistically significant. That is, married mothers were not only more likely to bear sons, but marriage was also associated with reduced risk for male infants. The age effects are notable for both their direction and magnitude. Mothers older than 35 years were 4% less likely to lose a son in the first year. As for education, whereas mothers for whom education information was missing were less likely to lose a son (column (2)), they were also less likely to bear a son in the first place, and the difference is not statistically significant (column (4)).

To further investigate whether the reinforcement of the TW pattern found at birth was driven by neonatal or post-neonatal deaths, we repeat the analysis for the sample of infant survivors and neonatal decedents, and infant survivors and post-neonatal decedents, respectively. The results are reported in columns (5) and (6). Since the coefficients on the uninteracted terms estimate the effect from infant survivors, they do not change (to make this clear, they are italicized in the table). Instead, our focus is on the interaction terms. These terms give the additional effect of an independent variable if the infant died. For instance, since the coefficient on married is −5.383 among infant decedents (column (2)), but 1.132 among infant survivors (column (3)), the difference in the coefficient estimates is −6.515 (column (4)).

Focusing on post-neonatal mortality (column (6)), marriage reduced the probability that a decedent was male, as did age. A married mother was 2.5% ((15.503−1.132)/(0.582×1000)) less likely to have lost a son rather than a daughter, and compared with a married mother

Table 1. High-status mothers bear more sons—pattern reinforced by post-neonatal mortality, US linked birth–infant death data 1983–1991 and 1995–2001. (All explanatory variables are indicator variables and scaled by 1/1000 (reported means pertain to the full sample of live births and are unscaled). For age, 20–34 years is the excluded category. For education, some college or more is the excluded category. All regressions include a constant and indicator variables for gestation length, birth weight and year-of-birth dummies. In regressions (4)–(6), these indicator variables are interacted with the indicator variable for whether the infant died. *t*-statistics in square brackets are computed using robust standard errors. ***, significance at (or below) the 1% level; **, significance at or below the 5% level (but above the 1% level); *, significance at or below the 10% level (but above the 5% level.).)

		(1)	(2)	(3)	(4)	(5)	(6)
		dependent variable: infant is male					
		sample					
					alive age 1 and decedents		
		alive age 0[b]	infant deaths[c]	alive age 1[d]	infant[c]	neonatal[e]	post-neonatal[f]
	mean[a]	0.513	0.573			0.568[g]	0.582[h]
married	0.786	1.022***	−5.383**	1.132***	*1.132****	*1.132****	*1.132****
		[5.25]	[2.57]	[5.79]	*[5.79]*	*[5.79]*	*[5.79]*
sub-15 years	1.50×10^{-3}	4.929***	32.132**	4.244**	*4.244***	*4.244***	*4.244***
		[2.64]	[2.29]	[2.25]	*[2.25]*	*[2.25]*	*[2.25]*
15–17 years	0.037	2.965***	24.301***	2.679***	*2.679****	*2.679****	*2.679****
		[7.14]	[6.12]	[6.42]	*[6.42]*	*[6.42]*	*[6.42]*
18–19 years	0.072	1.282***	17.059***	1.076***	*1.076****	*1.076****	*1.076****
		[4.37]	[5.61]	[3.65]	*[3.65]*	*[3.65]*	*[3.65]*
35 years+	0.1	−1.613***	−22.368***	−1.485***	*−1.485****	*−1.485****	*−1.485****
		[6.60]	[7.18]	[6.06]	*[6.06]*	*[6.06]*	*[6.06]*
EdMis	0.104	−2.086***	−7.001**	−2.133***	*−2.133****	*−2.133****	*−2.133****
		[7.71]	[2.31]	[7.85]	*[7.85]*	*[7.85]*	*[7.85]*
EdLow	0.18	−3.064***	0.974	−3.195***	*−3.195****	*−3.195****	*−3.195****
		[13.44]	[0.36]	[13.97]	*[13.97]*	*[13.97]*	*[13.97]*
EdHS	0.324	−1.074***	−0.435	−1.106***	*−1.106****	*−1.106****	*−1.106****
		[6.07]	[0.18]	[6.24]	*[6.24]*	*[6.24]*	*[6.24]*
dead×							
married					−6.515***	0.919	−15.503***
					[3.09]	[0.34]	[4.68]
sub-15 years					27.888**	32.017*	21.991
					[1.97]	[1.81]	[0.93]
15–17 years					21.622***	24.984***	16.658***
					[5.41]	[4.77]	[2.71]
18–19 years					15.983***	14.558***	16.531***
					[5.23]	[3.58]	[3.58]
35 years+					−20.883***	−17.174***	−27.536***
					[6.68]	[4.58]	[4.88]
EdMis					−4.869	−5.045	−5.526
					[1.60]	[1.35]	[1.05]
EdLow					4.169	3.158	2.567
					[1.52]	[0.89]	[0.59]
EdHS					0.67	−2.816	5.412
					[0.28]	[0.96]	[1.35]
dead	6.48×10^{-3}				55.638***	39.337***	68.157***
					[11.76]	[6.45]	[8.91]
N		48 023 459	310 974	47 712 485	48 023 459	47 905 746	47 830 195

[a] Column values pertain to the mean of the independent variable in the same row. Row values pertain to the fraction males in the respective sample, unless otherwise indicated.
[b] All live births.
[c] Infant deaths refer to deaths in the first year after birth.
[d] Live births minus infant deaths.
[e] Neonatal deaths refer to death that took place 0–28 days after birth.
[f] Post-neonatal deaths refer to death that took place 29–365 days after birth.
[g] Pertains to the fraction males among neonatal decedents.
[h] Pertains to the fraction males among post-neonatal decedents.

over 35, this figure was 10% ((15.503−1.132+16.531+1.076+27.536+1.485)/(0.582×1000)) for an unmarried teen mother.

As a robustness check, we also estimated regression models that included paternal information (age, race and education), but these neither altered the findings nor

yielded results of independent interest. Obviously, this does not necessarily imply that paternal characteristics are unimportant, but only that they were sufficiently captured by information on the mother.

5. SUMMARY AND DISCUSSION

Sex is not random. We have documented a TW pattern in the USA for the period 1983–2001. Whether evaluated at age 1 or birth, our findings are strongly supportive of the TW hypothesis. Mothers in 'good condition' (married, better educated and younger) bore more sons than mothers in 'poor condition', confirming a pattern noted by Darwin.[9] Moreover, these effects were generally stronger in the infant decedent population, suggesting a potentially quantitatively important role of infant mortality in shaping a TW pattern in the breeding population.

In the case of marital status, the results are unambiguous. Married mothers bore more sons and infant mortality reinforced the pattern observed at birth. For maternal age, the post-natal effect is the opposite of the pre-natal effect. As noted by Royer (2004), the effect of maternal age on infant health subsumes a tension between competing forces. While younger mothers tend to be healthier than older mothers, their socio-economic characteristics were worse. The reversal of the effect of maternal age may be interpreted in this light: as the opportunity for more nuanced proximate risks (e.g. those operating through parental discretion) is greater post-natally, the opportunity for the SES component of maternal age to be registered may also be greater.

One way to assess magnitudes is to consider the implied mortality. A married mother who had some college education was approximately 0.80% more likely to bear a son (or have a son survive to age 1) than an unmarried mother who did not complete high school. Assuming that this difference is achieved through higher mortality (foetal and infant) for low SES mothers—'TW mortality'—a lower bound on the heightened offspring mortality would be obtained by assuming that the TW mortality is 100% male.[10] If so, the 0.8% reduction would correspond to an additional 0.8% mortality.[11] The Centers for Disease Control estimated that 16% of pregnancies ended in foetal loss in 1997 (NCHS 2001). Taken at face value, this 16% suggests that a lower bound on TW mortality would be 1/20th (0.8/16) of foetal loss. Alternatively, a comparison with infant mortality rates—0.65% among white mothers in the USA in our sample—would imply that low SES mothers suffer an excess (pre-natal and infant) mortality that is higher than the current rate of US infant mortality.

The found association between maternal characteristics and offspring sex is substantially larger among infant decedents, suggesting an important role of post-natal mortality in shaping a TW pattern. For instance, for post-neonatal deaths, the mother being married and over 35 reduced the probability that the decedent was male by some 10% compared with the mother being a teenager and unmarried. By virtue of the exceedingly low infant mortality rates, these patterns were much muted in the surviving population. However, historically, infant mortality has been substantially higher and, possibly, quantitatively more important for shaping a TW pattern in the breeding population.

Lastly, while the found effect sizes are generally small, they may nevertheless signal important differences in underlying condition. For instance, we find that the mother being married was associated with a 0.1%-age point higher probability of the infant being male. Black–white differences in the offer one benchmark. Black mothers have long born fewer boys than white mothers in the United States. In 2001, a white mother bore a son with probability 0.5115 (1047/2047), while the same figure for a black mother was 0.5079 (1032/2032) (NCHS 2005), yielding a 0.36%-age point difference. In other words, the difference associated with the mother being married was approximately one-third of the raw difference in the sex ratio between black and white mothers. Another benchmark is the 0.4%-age point increase in the US sex ratio during the 1960s (temporarily reversing the downward post-war trend). This increase coincided with major public health programmes aimed at improving maternal health in the United States (Davis & Schoen, 1978). Thus, seemingly minor differences in the sex ratio of the offspring may signal large differences in parental condition.

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ENDNOTES

[1]At balanced breeding sex ratios.

[2]Greater variance in male reproductive success implies polygyny among successful males (and possibly polyandry (or abstinence) among less successful males, at balanced sex ratios). Polygyny of successful males, in turn, implies hypergamy, the systematic marrying up of females (and down of males who marry). While hypergamy may be another rationale for a TW effect, greater variance in male reproductive success may be the more fundamental asymmetry between the sexes (Trivers 1972).

[3]Male sex ratios have been associated with, for instance, dominant (Grant 1994) or aggressive (Kanazawa 2005) personality, stature (Kanazawa 2006) and nutritional status (Gibson 2003).

[4]Generalized daughter preference is more rare (e.g. Cronk 1989). However, for an endogamous population, male-biased sex ratios among the higher socio-economic strata are likely to prevail for TW reasons (Edlund 1999).

[5]Arguably, the case in the United States, although Gallup polls show a consistent bias towards sons in the United States among those with a preference (see, for instance, *The New York Times*, Sugar and Spice, and Sour Dads, November 16, 2003).

[6]Compare with the definition of parental investment in Trivers (1972).

[7]Essock-Vitae (1984) studied the number of children among Forbes list of the 400 wealthiest Americans. While she found that they had more children than the population at large, the women on the list actually had more children than the men.

[8]Teen mothers were less likely to be married and had less education than older mothers in the 1983–2001 NCHS data. The 1988 US Maternal and Infant Health Survey, which also records income, indicates that teen mothers had significantly lower household income and were more likely to receive welfare than older mothers (results available from authors).

[9]Darwin (1871, p. 281) noted as a 'mysterious fact that…the excess of male to female births is less when they are illegitimate than legitimate' and hypothesized that males suffer disproportionately from adverse conditions.

[10]If the excess mortality was not 100% male, then the mortality rate would have to be adjusted up accordingly.

[11]To see this, consider an initial offspring population of 50 sons and 50 daughters. If one son were to die, then this would correspond to mortality rate of 1%, and a reduction in the probability of a son of approximately 1% ((49/99-50/100)/0.5).

APPENDIX A. DATA

We constructed the following indicator variables using information on mother and child.

male	indicates whether the infant was male
dead	indicates whether infant died
married	indicates whether the mother was married at the time of giving birth
sub-15 years	indicates whether the mother was 14 years or younger at the time of giving birth
15–17 years	indicates whether the mother was 15–17 years old at the time of giving birth
18–19 years	indicates whether the mother was 18 or 19 years old at the time of giving birth
35 years+	indicates whether the mother was 35 years or older at the time of giving birth
EdMis	indicates whether the mother's education was missing
EdLow	indicates whether the mother had 11 or fewer years of education
EdHS	indicates whether the mother had 12 years of education, but not more

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