Online Resource 1

A Mathematical Details

A.1 Selective Live Birth

Here, we carry through the formal discussion on potential empirical implications of selective live birth, as a complement to the discussion in section 2.

Eq. (2) explicates the fact that differences in marriage fitness between parents of firstborn sons and parents of firstborn daughters is only observed *conditional* on live birth. Recall that equation:

$$\mathrm{E}\left[\Delta m^{\star}\left|\mathbf{x},\sum_{p=1}^{P}\omega_p<P\right.\right]=\Big(\delta_2-\delta_1\Big)+\underbrace{\left\{\mathrm{E}\left[\eta\left|\mathbf{x},b=1,\sum_{p=1}^{P}\omega_p<P\right.\right]-\mathrm{E}\left[\eta\left|\mathbf{x},g=1,\sum_{p=1}^{P}\omega_p<P\right.\right]\right\}}_{\boldsymbol{\Delta}_s}$$

And recall that interpreting $\left(\hat{\delta}_2-\hat{\delta}_1\right)$ in Eq. (2) as reflecting *nothing but* the parental divorce risk that daughters *would have faced* if they had been male requires assuming that $\boldsymbol{\Delta}_s=0$.

To examine this critical assumption, we will clean up notational clutter by suppressing the $\mathbf{x}$ in conditional expressions, and introduce a few new terms. Denote the probability that male pregnancy survives to live birth with π^b, and for female pregnancies, π^g; the weighted average of these will be Π. We will denote the probability density function of η with $f(\eta)$, and note that the assumption that sex of pregnancy is assigned as if by coin flip also implies that $f(\eta\,|b=1\,)=f(\eta\,|g=1\,)$. With all that notation, and applying Bayes' rule and the definition of the expected value function, we arrive at this expression for $\boldsymbol{\Delta}_s$:

$$\begin{aligned}\boldsymbol{\Delta}_s &= \int \eta\tfrac{f(\eta|b=1\,)}{(1-\Pi)^{P-1}\pi^b}\,d\eta-\int \eta\tfrac{f(\eta|g=1\,)}{(1-\Pi)^{P-1}\pi^g}\,d\eta\\ &= \int \eta\tfrac{f(\eta)}{(1-\Pi)^{P-1}}\left[\tfrac{1}{\pi^b}-\tfrac{1}{\pi^g}\right]\,d\eta\end{aligned}$$

Now, suppose $\pi^b\approx 1$ and $\pi_g\approx 1$ (that is, almost all pregnancies end in live birth). It follows then that for any couple $P=b+g$, so that all denominators above would be about equal to 1. As long all the covariates *other* than b and g (if any) are properly chosen, then $\int \eta f(\eta)d\eta=0$, so Δ_s would reduce to zero. In that

sense, belief that $\pi^b \approx 1$ and $\pi^g \approx 1$ could implicitly underlie an inclination to assign a counterfactual interpretation to $\left(\hat{\delta}_2 - \hat{\delta}_1\right)$. However, in the text and in online supplement B.1, we discuss evidence that survival probabilities may be considerably less than 1.

Alternatively, suppose that $\pi^g \approx \pi^b$, but both are less than 1. In that case, the term in square brackets would reduce to zero, and again the assumption that $\Delta_s = 0$ would be reasonable. In that sense, a counterfactual interpretation of $\left(\hat{\delta}_2 - \hat{\delta}_1\right)$ may be justified based on a belief that $\pi^g \approx \pi^b$. However, in the text and in online supplement B.2 we have discussed evidence that there may be a substantial prenatal female survival advantage.

Finally, suppose that $\pi^b < \pi^g$, but both survival probabilities and P are constant with respect to η. In that case, the denominator and the term in square brackets can be factored out of the integral. As long as covariates *other* than b and g (if any) are properly chosen, then a counterfactual interpretation is justified. However, in section 3 we have discussed evidence that at least one factor that is left in η in almost any marital "fitness" regression– namely, stress– may be negatively associated with π^b and π^g.

Most generally, a *purely* counterfactual interpretation of $\left(\hat{\delta}_2 - \hat{\delta}_1\right)$ effectively rests on this **assumption**:

$$\frac{\partial}{\partial \eta}\left(\frac{1}{(1-\Pi)^{P-1}}\left[\frac{1}{\pi^b} - \frac{1}{\pi^g}\right]\right) = 0 \tag{A.1}$$

Our study investigates what could *realistically* be the consequences of a failure of that assumption.

We conclude this discussion by emphasizing that assumption (A.1) is **not** equivalent to assuming that the impact of η on female pregnancies is equal to its impact on male pregnancies. In fact, assumption (A.1) probably cannot hold unless exactly the *opposite* is true. **In order for assumption** (A.1) **to hold in the presence of underlying female survival advantage, unmeasured characteristics in η (including biological stress) must be *more* pernicious (in an absolute sense) for female pregnancies than male ones.**

The same dynamics could also be observed using the principle of the force of mortality (Keyfitz and Caswell, 2005). Consider a cohort aging from fertilized egg (at time 0) to live birth (at time 1). Represent female survivors as of time $s \leq 1$:

$$l_F(s) = l_F(0) \exp\left\{-\int_0^s \mu(t)\, dt\right\}$$

and male survivors:

$$l_M(s) = l_M(0) \exp\left\{-\int_0^s a(t)\mu(t)\, dt\right\}$$

Where $\mu(\cdot)$ represents the force of mortality and $a \geq 1$ indicates the size of any prenatal female survival advantage. It follows that the sex ratio (males per female) in the cohort at time s will be

$$r(s) = r(0) \exp\left\{\int_0^s (1 - a(t))\, \mu(t)\, dt\right\}$$

Since $\mu(t) \geq 0$, it follows that this ratio will decline with an upward shift in $\mu(\cdot)$ as long as the shift does not have an offsetting effect on $a(\cdot)$, and as long as $a(t) > 1$ for at least some t. Therefore if there is heterogeneity in the population with respect to $\mu(\cdot)$, then (by Bayes' rule) at time 1, average fecundity will be lower among parents of daughters than among parents of sons.

It remains an empirical question whether assumption (A.1) holds. We find it unsettling.

In our simulation tool, we require that the effect of biological stress on pregnancy survival is independent of the sex of the pregnancy.

A.2 Simulation Tool

Here, we describe the mathematical details underlying the simulation tool, as a complement to the discussion in section 4.2.

At the outset of each simulation, every couple is randomly assigned four characteristics:

- An underlying **fertility behavior/fecundability** (ν). This parameter establishes a distribution across the couples in terms of likelihood of establishing a pregnancy in any given period.
- An underlying **fecundity** (μ). This parameter establishes a distribution across the couples in terms of the likelihood that a pregnancy, once established, will survive to live birth.
- An underlying **marital stability** (λ). This parameter establishes a distribution across the couples in terms of the likelihood that they will divorce in any given period.
- A level of biological **stress** (θ). Specifically, the cohort is divided into 1001 categories, from the "least stressed" 0.1% of the population ($\theta = -500$), to the "most stressed" 0.1% ($\theta = 500$).

Here is how the simulation proceeds.

New Pregnancies

The simulation tool follows these steps to assign pregnancies to still intact couples who are not already pregnant:

1. It computes for each intact couple who is not pregnant, a period-and-parity specific latent fecundability, using the following equation:

$$P^{\star}_{i,t} = \nu_i + K^P_{t,c} - \eta_{1,i,t}$$

   $K^P_{t,c}$ is a period-specific constant chosen to ensure that the fraction of couples with c children already born who get pregnant will match the pre-specified overall parity-specific pregnancy hazard for that period, and η_1 is an independent random draw from a standard normal distribution.

2. It assigns a pregnancy to all couples for whom the latent fecundability score is greater than or equal to zero, or equivalently:

$$P_{i,t} = \begin{cases} 1 & \text{if} \quad \nu_i + K^P_{t,c} \geq \eta_{1,i,t} \\ 0 & \text{if} \quad \nu_i + K^P_{t,c} < \eta_{1,i,t} \end{cases}$$

3. It assigns a sex to each new pregnancy, as follows:

$$M_{i,t} = \begin{cases} 1 & \text{if} \quad \Phi^{-1}\left(\frac{r_0}{1+r_0}\right) \geq \eta_{2,i,t} \\ 0 & \text{if} \quad \Phi^{-1}\left(\frac{r_0}{1+r_0}\right) < \eta_{2,i,t} \end{cases}$$

   $\Phi^{-1}(\cdot)$ represents the inverse cumulative normal distribution, and η_2 is an independent random draw from a standard normal distribution.

New Births

The simulation tool follows these steps to determine the outcome of pregnancies established in the previous period:

1. It computes for each pregnancy a latent "fitness" score, using the following equation:

$$L^{\star}_{i,t} = \mu_i + \alpha_1(\theta) + \gamma M_{i,t-1} + K^L_t - \eta_{3,i,t}$$

K_t^L is a constant chosen to ensure that the fraction of overall pregnancies matches the pre-specified overall mortality risk, and γ reflects the female survival advantage which is necessary to drive a decline in the sex ratio from r_0 at fertilization to r_1 at live birth. $\alpha_1(\theta)$ is a function that reflects the relationship between biological stress and pregnancy "fitness." In most of our simulation runs, this function is linear in form– $\alpha_1 \times \theta$, but we also experiment with more flexible forms. η_3 is an independent random draw from a standard normal distribution.

2. It assigns a first birth to all couples who have a pregnancy with nonnegative "fitness" score. Or, equivalently:

$$L_{i,t} = \begin{cases} 1 & \text{if} \quad \mu_i + \alpha_1(\theta) + \gamma M_{i,t-1} + K_t^L \geq \eta_{3,i,t} \\ 0 & \text{if} \quad \mu_i + \alpha_1(\theta) + \gamma M_{i,t-1} + K_t^L < \eta_{3,i,t} \end{cases}$$

3. If a couple has three live births, its family size is topcoded and it is removed from eligibility for future pregnancies (since birth hazards beyond the third are generally low enough that they cannot be reliably identified in data).

New Divorces

The simulation follows these steps to assign divorces to still intact couples:

1. It computes for each intact couple a period specific latent divorce "vulnerability" score, using the following equation:

$$d_{i,t}^{\star} = \lambda_i + \alpha_2(\theta) + K_t^d - \eta_{4,i,t}$$

K_t^d is a period-specific constant chosen to ensure that the fraction of couples who divorce in each period will match the pre-specified period-specific divorce hazard. η_4 is an independent random draw from a standard normal distribution.

2. It assigns a new divorce to all couples with nonnegative latent divorce "vulnerability." Equivalently:

$$d_{i,t} = \begin{cases} 1 & \text{if} \quad \lambda_i + \alpha_2(\theta) + K_t^d \geq \eta_{4,i,t} \\ 0 & \text{if} \quad \lambda_i + \alpha_2(\theta) + K_t^d < \eta_{4,i,t} \end{cases}$$

3. For every couple experiencing a divorce in time t, the simulation tool assigns to them a permanent indicator $D_{i,\tau} = 1$ for every $\tau \geq t$, and excludes them from any further followup.

B Survival Bias and Offspring Sex

Within a cohort of pregnancies, those reaching live birth may comprise a minority of the original cohort. Furthermore, the cohort of pregnancies may become increasingly female as it ages– reflecting the fact that male pregnancies more likely to drop out of the cohort.

B.1 The Extent of Selection into Live Birth

Human reproduction is strikingly inefficient when compared against that of other species. Even after an egg cell is fertilized, the probability that it will develop to live birth is smaller than may be widely appreciated. Epidemiologists have estimated this probability from as high as 70% to as low as 30% (Benagiano et al., 2010; Grudzinskas and Nysenbaum, 1985; Macklon et al., 2002). This range is wide, because measurement is difficult; even using state of the art techniques, a fertilized egg– or "zygote"– cannot be reliably detected until it is about 10 days old.

In four important longitudinal studies conducted in the United States (Wilcox et al., 1988), China (Wang et al., 2003), Bolivia (Nepomnaschy et al., 2004), and Guatemala (Vitzthum et al., 2006), epidemiologists recruited women volunteers who were in sexual unions, were not contracepting, and had no known fertility problems. Biological samples from these volunteers were then tested regularly for the signature hormone of early pregnancy, in an attempt to detect surviving zygotes and thereby to map out a survival curve for normal early pregnancies.

In these studies, 30-40% of observed pregnancies terminated without a live birth. Although these studies aimed to represent the broader population by specifically excluding couples who had demonstrated difficulty establishing a pregnancy, it is important to note that they did not involve probability-based, population representative sampling designs. To our knowledge, no study has combined this measurement approach with standard population survey sampling techniques in order to identify the prevalence of early pregnancy loss among non-contracepting humans.

These studies can only speak to the fraction of zygotes that make it to live birth, among those that survive the first 10 days. Even less is known about so-called "pre-implantation loss"– that is, the risk that a zygote will pass from a woman without ever implanting in the placenta. Distinguishing that event from a normal menstrual period is prohibitively expensive, even in small samples. Given these technological limits to measurement, physicians and epidemiologists have used clever approaches to assess the likely scale of this type

of loss in humans, including demographically comparing the expected and observed number of clinical pregnancies in populations (Roberts and Lowe, 1975).

Taken together, epidemiological evidence collected over the past 50 years indicates that live births may represent something on the order of a 50%-30% subsample of zygotes (Benagiano et al., 2010). When one accounts for the fact that about half of the zygotes lost contain major, fatal defects resulting from random errors in cell reproduction (Macklon et al., 2002), this still leaves only about 40-50% of normal, viable zygotes surviving to live birth.

B.2 Prenatal Female Survival Advantage

What determines which half of normal, viable zygotes become live births? Another body of evidence indicates that sex may be one factor; specifically, female zygotes appear overrepresented among those that survive to live birth.

Because sex does not change over the course of pregnancy, a prenatal female survival advantage will lead to a decrease in the number of males per female as a cohort of pregnancies progresses. Since the sex ratio at *live birth* is readily observed wherever vital registration is complete, pinning down the extent of prenatal female survival advantage "only" requires identifying the sex composition of normal zygotes as early as possible in gestation. If the sex ratio declines over the course of pregnancy– just as it does throughout the life course after birth– then this implies that the female survival advantage begins before birth.

Owing to high costs of measurement, studies have not been conducted that directly quantify sex ratios in very early pregnancy. Instead, studies have tried to shed light on this question by examining the products of induced abortions. A typical approach involves collecting biological material from embryos and fetuses that were medically aborted as early as 5 weeks into pregnancy. Many such studies were conducted decades ago, before technology existed that would allow parents or their physicians to know the sex when the pregnancy was aborted. In that case, it may be plausible that the sex ratio among the *aborted* embryos and fetuses represents the sex ratio among *all* embryos and fetuses at a similar stage of development. One of the earliest studies to do this (Kellokumpu-Lehtinen and Pelliniemi, 1984) found sex ratios of 164 males per 100 females among Finnish women in the first 8 weeks of pregnancy, and 111 males per 100 females for the 8-24 week stage. Comparable studies in different populations and at different times, have found similar patterns– the earlier a fetus or embryo is recovered in the course of a pregnancy, the greater is the probability that it is male (McMillen, 1979; Pergament et al., 2002).

Other research draws inferentially from in vitro fertilization (IVF) records. Depending on the strategy favored by parents and their physicians, an embryo produced by IVF can be introduced into a woman very early in its development, or it can be allowed to mature through several rounds of cell division first. The earlier in its development that an embryo is introduced into the woman, the less viable it is (Gardner et al., 1998). In populations around the globe, live births resulting from pregnancies involving further developed (and, thus, more stable) embryos are more likely to be male than those resulting from pregnancies involving the less stable embryos (Chang et al., 2009; Dean et al., 2010).

Based largely on evidence from studies like these, many epidemiologists have speculated that the sex ratio of newly formed viable zygotes may be substantially higher than the sex ratio of live births (Vatten and Skjaerven, 2004); this ratio may vary from as low as 110 male zygotes per 100 female to as many as 170 male per 100 female (Chahnazarian, 1988; Kellokumpu-Lehtinen and Pelliniemi, 1984; McMillen, 1979; Pergament et al., 2002).

B.3 Is Sex of Offspring Like a Coin Flip?

We have discussed evidence that survival bias may generate associations between fecundity and sex of offspring– even if the sex of *pregnancies* is randomly assigned. This would imply that the sex of live births would *not* be indpendent and identically distributed across parents– specifically, within a population, less fecund parents would be more likely to have daughters.

The sex composition of birth cohorts is remarkably stable over time and across place. In the absence of conscious manipulation, ratios typically fall within the range of 102-107 boys per 100 girls (Wilcox and Baird, 2011). The mechanisms generating variation within this range are still poorly understood, although the nature and extent variation of systematic in these ratios has been a topic of interest for over a quarter of a millenium (Laplace, 1781; Trivers and Willard, 1973; Éric Brian and Jaisson, 2007). Detecting patterns rests largely on observational studies, which risk discovering false positive associations driven by measurement error or failure to account for the multiplicity of tests of the same hypothesis (Gelman and Weakliem, 2008; Wilcox and Baird, 2011; Simpson, 2012; Maconochie and Roman, 1997).

On the other hand, *within* a population in the same time and place, the sex of live births has been observed to covary with some demographic characteristics. Perhaps the most salient example is the well-documented racial patterning in sex ratios at birth. Black women giving birth in the United States are more likely to have a girl than white women. This pattern is robust, stable over years, and has been widely noted in the

epidemiology literature (Pergament et al., 2002; Chahnazarian, 1988; Davis et al., 2007; Marcus et al., 1998). In this sense, the sex of live births is *not* like a coin flip– flipped many times, a coin will come up heads about the same fraction of times, whether a black woman or white woman is doing the flipping.

Other characteristics and behaviors that have been observed to statistically predict offspring sex relate directly with fecundity– they include nutritional status (Cagnacci et al., 2004; Song, 2012; Rosenfeld and Roberts, 2010), parental age (Nicolich et al., 2000; Jacobsen et al., 1999), when in the ovulation cycle the relevant insemination occurs (James, 2012), and coital frequency (Wadley and Martin, 1997). Sex ratios at birth have also been observed to covary with the occurrence of disruptive events (Hansen et al., 1999; Fukuda et al., 1998; Bruckner et al., 2010; Torche and Kleinhaus, 2012; Song, 2012).

All of these characteristics and behaviors remain controversial as candidate predictors of offspring sex. Many hypotheses have been proposed and tested to account for both the observed variability *within* a place and time of sex ratios at birth, and their consistency *across* place and time; consensus remains elusive. Factors affecting the viability of pregnancies may play a role (James, 2012; Chahnazarian, 1988; Pergament et al., 2002).

In light of this controversy, our approach does not directly specify any characteristics as determinants of sex ratio at birth. Instead, we draw two lessons from this literature– first, a fecundity-related mechanism is plausible, by which the sex of a couple's offspring could provide information about that couple's circumstances. Such a mechanism amounts simply to survival bias; variation in fecundity interacts with prenatal female survival advantage to generate small but systematic differences in the probability that a couple's offspring will be female. The second lesson we draw from the ongoing empirical controversy regarding determinants of sex ratio at birth is that the assumption that offspring sex is independent and identically distributed across all parents, in the way that the outcome of a coin flip would be, is not self-evident (Wilcox and Baird, 2011).

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