Missing Women

Some Recent Controversies on Levels and Trends in Gender Bias in Mortality†

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Abstract and Keywords

This chapter discusses two recent controversies surrounding levels and trends in the number of ‘missing women’ in the world. First, the impact of fertility decline on gender bias in mortality is examined. Contrary to the expectations of some, fertility decline has not generally led to an intensification of gender bias in mortality. Second, the chapter finds that the claim that a substantial portion of ‘missing women’ is due to higher sex ratios at birth linked to hepatitis B prevalence in the affected regions is on rather weak foundations, while there is substantial evidence countering this claim.

Keywords: missing women, fertility decline, hepatitis B, sex-selective abortions, gender bias, mortality

I. Introduction

ONE of Amartya Sen's important contributions in the field of development economics has been his work on gender bias in mortality in parts of the developing world. Although imbalances in aggregate sex ratios favoring males had been
known for some time (see e.g. Visaria 1961; Bardhan 1974), he added to this literature by providing new empirical evidence on gender discrimination in India (Sen and Sengupta 1983), by suggesting a modeling framework that has powerfully influenced the literature on intra-household resource allocation (Sen 1990a), by coining the term “missing women”, referring to the cumulative impact of past and present gender bias in mortality on the current sex structure of the population, and by (p.281) providing a first estimate of the number of missing women in the countries affected by gender bias in mortality (Sen 1990b). In well-known articles in 1989 and 1990, he suggested that more than 100 million women are “missing” in South Asia, East Asia, the Middle East and North Africa, and that they have fallen victim to gender bias in mortality (Sen 1989, 1990b). He arrived at this figure by comparing the sex ratios of these countries and regions with those prevailing in Sub-Saharan Africa and assuming that the difference was due to gender bias in mortality. A literature subsequently developed that has refined these estimates of missing women, extended them to more countries, and updated them using more recent census information (see e.g. Coale 1991; Klasen 1994; Klasen and Wink 2002, 2003; OECD 2006). A much larger literature has examined gender bias in mortality in South and East Asia in much greater detail to understand its demographic correlates as well as its socioeconomic causes (see e.g. Drèze and Sen 2001 for a summary of many of these contributions). Due to its obvious importance from a policy point of view, this literature has continued to grow.

One issue that has sparked a particular amount of debate has been the effect of fertility decline on gender bias in mortality. In particular, several scholars have suggested that fertility decline in countries with son preference leads to intensified gender bias in mortality (e.g. Das Gupta and Mari Bhat 1997; Basu 1999, 2000; Rajan et al. 2000). Others have suggested, however, that this link is empirically weak, at least in India, or appears actually to go in the opposite direction (e.g. Drèze and Murthi 2001; Mari Bhat and Zavier 2003). The first purpose of this chapter is to reflect on this debate using some recent data from countries affected by gender bias in mortality. In particular, I will suggest that there is no evidence of a solid relationship between fertility decline and intensification of gender bias; in certain circumstances, particularly those...
where fertility decline is partly a result of coercive family planning policies (as in China) or where son preference has remained strong but desired fertility is already very low (as in South Korea), fertility decline can lead to an intensification of gender bias, at least for some length of time; in most other circumstances, however, the evidence for such a link is weak, either across or within nations. In fact, often the opposite appears to be the case: fertility decline goes hand in hand with declines in gender bias in mortality.

The last two years have also seen a return of debates on the magnitude of the number of “missing women”. In particular, a paper by Emily Oster (2005) has claimed that existing estimates of “missing women” vastly overstate the phenomenon, as they neglect a link between the Hepatitis B virus (HVB) and the sex ratio at birth. She argues that there is a positive link between the proportion of parents who are carriers of HVB and the sex ratio at birth. Since many of the countries with gender bias in mortality also have (or at least have had) a fairly substantial prevalence of HVB carriers, about 50% of the “missing women” alleged to be victims of gender bias in mortality are missing, more innocuously, as a result of the HVB carrier status of their parents. This claim has also sparked a controversy, (p.282) with most of the evidence presented by Oster having come under critical scrutiny and other conflicting evidence has been put forward (see Abrevaya 2005; Das Gupta 2005, 2006; Lin and Luoh 2008; Ebenstein 2007). In fact, Oster herself has very recently produced evidence that goes against her original claims and she has therefore retracted the claim that HVB prevalence was responsible for bias sex ratios, at least in China (Chen and Oster 2008). The second aim of the paper is to comment on the original claims made by Oster as well as the literature that has developed since. I will argue that while the claim was rather intriguing and the empirical evidence presented by Oster substantial, closer inspection of that evidence generates serious questions and problems with all pieces of the evidence presented in support of this claim. Moreover, the evidence against a link between HVB carrier status and the sex ratio at birth, as well as the evidence on parental strategies of son preference, is so substantial that the claim that much of gender bias in mortality is mostly due to this biological linkage appears to be largely baseless. It appears instead that any possible link between HVB carrier status and the sex ratio at
birth has at most a tiny impact on the number of “missing women”. As a result, it remains the case that gender bias in mortality (including pre-birth strategies of sex selection) accounts for an overwhelming portion of the sex ratio imbalances observed in South and East Asia.

II. Fertility Decline and Gender Bias in Mortality
Most of the countries affected by gender bias in mortality have undergone a significant fertility decline in the past 10 to 20 years. In fact, as shown in Table 15.1, which presents total fertility rates (TFRs) matched to the two most recent censuses, which have been used for the missing women calculations in Klasen and Wink (2003), TFRs have fallen dramatically in many nations, including Bangladesh, India, Pakistan and most Middle Eastern countries. In China and Korea, much of the fertility decline occurred prior to the 1990s, and is now below replacement levels. Only in Afghanistan has fertility persisted at extremely high levels.

There are a number of reasons why one might expect such a fertility decline to lead to an intensification of gender bias in mortality. Given an unchanging desire of many parents in the regions affected by gender bias in mortality to have at least one surviving son, a lower TFR could mean that parents will be particularly concerned that the one surviving son be male. This is called the intensification effect by Das Gupta and Mari Bhat (1999). In addition, Basu (1999) argues that son preference (p.283)
<table>
<thead>
<tr>
<th>Country</th>
<th>Most recent data</th>
<th>Females “missing” (%)</th>
<th>TFR</th>
<th>Previous data</th>
<th>Females “missing” (%)</th>
<th>TFR</th>
<th>Decline in TFR</th>
<th>Change in percentage missing</th>
</tr>
</thead>
<tbody>
<tr>
<td>Afghanistan</td>
<td>2000</td>
<td>9.30</td>
<td>6.84</td>
<td>1979</td>
<td>9.70</td>
<td>6.9</td>
<td>0.06</td>
<td>0.40</td>
</tr>
<tr>
<td>Algeria</td>
<td>1998</td>
<td>1.24</td>
<td>3.05</td>
<td>1987</td>
<td>2.69</td>
<td>5.72</td>
<td>2.67</td>
<td>1.45</td>
</tr>
<tr>
<td>Bangladesh</td>
<td>2001</td>
<td>4.23</td>
<td>3.13</td>
<td>1981</td>
<td>8.91</td>
<td>6.0</td>
<td>2.87</td>
<td>4.68</td>
</tr>
<tr>
<td>China</td>
<td>2000</td>
<td>6.69</td>
<td>1.89</td>
<td>1990</td>
<td>6.31</td>
<td>2.1</td>
<td>0.21</td>
<td>−0.38</td>
</tr>
<tr>
<td>Egypt</td>
<td>1996</td>
<td>4.46</td>
<td>3.55</td>
<td>1986</td>
<td>5.14</td>
<td>4.3</td>
<td>0.75</td>
<td>0.68</td>
</tr>
<tr>
<td>India</td>
<td>2001</td>
<td>7.89</td>
<td>3.07</td>
<td>1991</td>
<td>9.44</td>
<td>3.81</td>
<td>0.74</td>
<td>1.55</td>
</tr>
<tr>
<td>Iran</td>
<td>1996</td>
<td>3.68</td>
<td>3.0</td>
<td>1986</td>
<td>4.54</td>
<td>5.9</td>
<td>2.9</td>
<td>0.86</td>
</tr>
<tr>
<td>Korea</td>
<td>1995</td>
<td>0.73</td>
<td>1.75</td>
<td>1985</td>
<td>−0.14</td>
<td>2.04</td>
<td>0.29</td>
<td>−0.87</td>
</tr>
<tr>
<td>Nepal</td>
<td>2001</td>
<td>0.50</td>
<td>4.27</td>
<td>1981</td>
<td>7.71</td>
<td>6.1</td>
<td>1.83</td>
<td>7.22</td>
</tr>
<tr>
<td>Pakistan</td>
<td>1998</td>
<td>7.76</td>
<td>4.77</td>
<td>1981</td>
<td>10.78</td>
<td>7.0</td>
<td>2.23</td>
<td>3.01</td>
</tr>
<tr>
<td>Sri Lanka</td>
<td>1991</td>
<td>−0.02</td>
<td>2.5</td>
<td>1981</td>
<td>3.44</td>
<td>3.25</td>
<td>0.75</td>
<td>3.47</td>
</tr>
<tr>
<td>Syria</td>
<td>1994</td>
<td>3.06</td>
<td>4.2</td>
<td>1981</td>
<td>4.98</td>
<td>7.4</td>
<td>3.2</td>
<td>1.92</td>
</tr>
<tr>
<td>Tunisia</td>
<td>1994</td>
<td>2.13</td>
<td>2.9</td>
<td>1984</td>
<td>4.45</td>
<td>4.32</td>
<td>1.42</td>
<td>2.33</td>
</tr>
<tr>
<td>Turkey</td>
<td>1990</td>
<td>2.40</td>
<td>3.0</td>
<td>1985</td>
<td>3.15</td>
<td>3.79</td>
<td>0.79</td>
<td>0.75</td>
</tr>
</tbody>
</table>

**Sources:** Klasen and Wink (2003); World Bank (2007).
might even increase as a result of fertility decline, further intensifying gender bias. As a result, the whole range of pre- and post-birth strategies could be applied to ensure that the far fewer children include at least one male. Among the strategies parents would have at their disposal are selective stopping rules, i.e. ending child-bearing as soon as the first son is born, using sex-selective abortions to increase the likelihood of sons, and favoring boys in the allocation of survival-related resources such as health and nutrition (see Klasen 2003). Of course, all three strategies could be applied at the same time. It is likely that the effects on sex ratios would be particularly severe in countries with substantial son preference where the fertility decline is a result of coercive family planning policies (such as China), or where fertility levels are so low that a large share of parents would not have a surviving son unless some of these strategies to manipulate the sex of the offspring were applied (such as Korea).

While these arguments are all plausible, they treat fertility decline as an exogenous variable affecting gender bias in mortality, assuming unchanging (or even increasing) son preference. Given the literature on the determinants of fertility levels, however, it is much more reasonable to treat fertility decline as an endogenous variable affected by a whole set of developments in a country, including rising prosperity and rising levels of female education, which in turn also have an impact. 

![Graph](image_url)  

**Fig. 15.1.** Fertility decline and changes in the share of missing females  
*Source:* Data from Table 15.1.
Missing Women

on son preference and thus on gender bias in mortality. Thus one might plausibly argue that fertility decline and gender bias in mortality are jointly determined by these developments. In that case, it could well be that endogenous fertility declines are associated with reductions in gender bias in mortality, in particular if the causal factors driving these developments are rising incomes, rising female education, and rising female employment levels, all of which are likely to be associated with reduced gender bias in mortality.

As a result, the relation between fertility decline and gender bias in mortality is a complex empirical question. In the following I want to present briefly some empirical evidence on this relation, both across countries and within selected countries, including China, Korea and India, where these issues have been particularly prominent.

Table 15.1 and Figure 15.1, which plots the two rightmost columns of Table 15.1, speak rather clearly on the relationship between fertility decline and gender bias in mortality in the countries with the most serious problem of “missing women”. Table 15.1 reports on the share of “missing females” in the last two censuses as an indicator of (pre- and post-birth) gender bias in mortality in the “missing women” countries, as calculated by Klasen and Wink (2003), and relates this to absolute fertility decline over the same period. If the fears about an intensification of gender bias as a result of fertility decline were correct, we would expect a positive relationship between fertility decline and change in gender bias in mortality, i.e. the larger the fertility decline, the higher the increase in gender bias in mortality. Figure 15.1 quite clearly shows nothing of the sort. In fact, the correlation goes the other way and is actually highly significant. In countries where fertility decline has been largest, the share of missing women has tended to fall the most.

China and (South) Korea are included in Table 15.1 and Figure 15.1 and therefore one might plausibly object that in these cases most of the fertility decline had taken place before 1990 (1985 in the case of Korea), so it could have been the previous, stronger fertility decline that is responsible for an intensification of gender bias. In the case of China, there clearly is some force to this argument. As demonstrated very clearly in Banister (1987), Banister and Coale (1994), Lai (2005), Attane (2007), and Ebenstein (2007), the fertility decline since the late 1970s has been associated with a sharp
rise in both pre- and post-birth discrimination against female children, leading to a sharp rise in the share of missing females in China since the early 1980s. In the 1990s, fertility decline slowed considerably and gender bias in mortality has not deteriorated much further from the already poor state of affairs it had reached in 1990. But it should be pointed out that the fertility decline in China is of a special sort, as it has been heavily affected by the one-child policy instituted in 1977, where parents have to contend with severe sanctions if they have more than one child. In contrast to other countries, fertility decline in the context of the coercive one-child policy can be treated as largely “exogenous”. In an environment of significant remaining son preference, it is therefore not surprising that some of the intensification effects predicted for India have been found in China, at least in the initial phases of the one-child policy.

It is unclear, however, whether this large increase in gender bias in mortality in China since the introduction of the one-child policy is entirely due to the fertility decline the latter generated. While there is no doubt that the relative deterioration in female survival is largely due to the effects of the one-child policy, the sharp rise in sex-selective abortions in China since the mid-1980s (increasing the male—female ratio at birth from about 1.06 in the early 1980s to 1.20 in 2000: see Klasen and Wink 2003 and Attane 2007) might be only partly related to the one-child policy. Instead, it is possible to view this rising incidence of sex-selective abortions as an exogenous technological change (brought about by the increasing availability of pre-natal sex determination through ultrasound screening) that has enabled parents to achieve their desired sex composition of their offspring more easily than had been possible.

South Korea is an interesting test for this argument. Here fertility decline took place without recourse to draconian family planning policies; in addition, pre-birth sex determination has been available since the 1980s. As shown by Chung and Das Gupta (2007), the male—female ratio at birth rose sharply from the early 1980s to the early 1990s (from about 1.07 to 1.15), coinciding with the increasing availability of pre-birth sex determination. This supports the claim that a change in technology alone can severely affect gender bias in
mortality in an environment of strong son preference and falling or low fertility.

The Korean story does not end there. In the midst of rising education, modernization, economic development and continued fertility decline, the sex ratio at birth has been falling since the early 1990s and at 2003 was close to its levels in the early 1980s. As shown by Chung and Das Gupta (2007), the main driver of this change is falling son preference associated with a secular change in attitudes brought about by economic and social development. Thus after a rise in the male—female ratio at birth brought about by technological change, Korea now fits the pattern described above, where economic and social development appears to reduce both fertility and son preference. Chung and Das Gupta expect a similar trend to happen in due time in China (and India).

Thus the Chinese experience suggests that fertility decline resulting from coercive family planning can powerfully intensify gender bias in mortality. At the same time, not all of the intensification of gender bias in mortality is due to this, as technological changes in China and Korea have helped to increase the ability of parents to determine the sex composition of their offspring.

Let me now turn to India. One way to examine the relationship between fertility decline and gender bias in mortality for India is to examine this relationship at the state level. Table 15.2 presents some simple regressions of the statewise sex ratio (males/females) in India’s largest states between 1961 and 2001. I show pooled-OLS, fixed-effects and random-effects results. The pooled-OLS results suggest that high fertility is associated with a higher sex ratio, i.e. a higher share of males, at the state level, contrary to the hypothesis examined here. It is unlikely, however, that high fertility is “causing” the high sex ratio; rather the fertility rate is an endogenous variable, with some underlying conditions affecting both fertility as well as gender bias at the state level. This suspicion is confirmed by the random effects and, in particular, the fixed-effects specification. With the latter, one would hope that all state-specific factors affecting the sex ratio at birth are captured by the fixed effects and only the impact of the change of fertility on the sex ratio is being examined. The results show that there is no longer a positive significant relationship, but neither is
there a negative relationship. So the results for India also demonstrate that there is no evidence of fertility decline leading to an intensification of gender bias in mortality.

(p.287)

**Table 15.2. Determinants of the sex ratio (males/females) of India’s largest states, 1961-2001**

<table>
<thead>
<tr>
<th></th>
<th>OLS</th>
<th>Random effects</th>
<th>Fixed effects</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Total fertility rate</strong></td>
<td>1.91 (0.86)</td>
<td>0.29 (0.62)</td>
<td>0.09 (0.16)</td>
</tr>
<tr>
<td>South</td>
<td>−6.15 (-3.09)**</td>
<td>−8.53 (-3.21)**</td>
<td></td>
</tr>
<tr>
<td>East</td>
<td>−3.68 (-2.42)**</td>
<td>-3.88 (-1.14)</td>
<td></td>
</tr>
<tr>
<td>West</td>
<td>−1.32 (-0.86)</td>
<td>−2.72 (-0.88)</td>
<td></td>
</tr>
<tr>
<td>1971</td>
<td>−4.17 (-1.86)**</td>
<td>−0.07 (-0.05)</td>
<td>0.39 (0.28)</td>
</tr>
<tr>
<td>1981</td>
<td>−2.77 (-1.62)*</td>
<td>−0.49 (-0.56)</td>
<td>−0.21 (-0.24)</td>
</tr>
<tr>
<td>1991</td>
<td>−0.32 (0.75)</td>
<td>0.39 (0.75)</td>
<td>0.49 (1.07)</td>
</tr>
<tr>
<td>Adjusted $R^2$</td>
<td>0.52</td>
<td>0.54</td>
<td>0.13</td>
</tr>
<tr>
<td>n</td>
<td>58</td>
<td>58</td>
<td>58</td>
</tr>
</tbody>
</table>

*Notes: t-ratios based on robust standard errors are reported in parentheses.*

(*) 90%,

(**) 95% and

(*** 99% significance level (one-tail test). For details of the states included, see Klasen and Wink (2003).

*Source: Own analysis based on data from Registrar General (2003).*
Other research also supports the contention that fertility decline has not led to an intensification of gender bias in India. In particular, Mari Bhat and Xavier (2003) examine changes in son preference and fertility decline in India using the Indian National Family Health Surveys. They find that son preference has declined in the course of fertility decline in every state but one. This is further evidence that one cannot treat fertility decline as an exogenous factor affecting gender bias in mortality, holding son preference fixed. It seems much more plausible that the factors driving fertility decline, including rising female education, employment and income, are precisely the factors that also reduce son preference.

In the Indian case, however, it is also likely that the increasing availability of sex-determination technologies is having a one-off impact on the level of sex-selective abortions and thus pre-birth gender bias in mortality. Thus the worrying increase of the sex ratio for 0- to 6-year-olds in the 2001 census is likely to be related mostly to this one-off technological shift, which has increased the ability of parents to affect the sex of their offspring, rather than a result of fertility decline. This can also be seen by examining the year dummies in the regressions in Table 15.2. Compared to 2001, the sex ratio in prior decades was always lower, so there appears to be a secular upward trend, particularly since the 1981 census. This would be perfectly consistent with the impact of such a technological shift. If this argument is correct, the prediction would be that the proportion of males in the younger age groups should slowly drift downwards together with declining son preference, as it recently has in South Korea.
III. Hepatitis Band the Number of Missing Women

Let me turn now to the second debate about levels and trends in gender bias in mortality. As noted earlier, Emily Oster (2005) suggested that a considerable share of the “missing women”, particularly in China, are not due to gender bias in mortality, but to the prevalence of hepatitis B virus (HVB) carriers among the adult population, which, she argued, has been found to increase the proportion of males at birth. Females have not died through pre- or post-birth sex-selection strategies, but were never born due to this epidemiological link; thus many “missing women” are due to “biology” rather than “discrimination”, a finding that has also been widely discussed in the popular press (see e.g. Barro 2005). As shown in Table 15.3, reproduction of a table from her paper, she believed that about half of the estimated 60 million missing women in Coale’s (1991) calculation (based on census data available at the time) can be explained this way. The impact on the estimates of missing women is largest in China (a reduction of −77%), where the problem of gender bias in mortality largely vanishes, followed by Egypt (−54%) and West Asia (−30%), and the impact is lowest in South Asia (−18% in India, −20% in Bangladesh, and no impact in Pakistan).

As shown in Table 15.3, Oster also compared her estimates of missing women with those of Sen (1992), whereupon the share of missing women supposedly explained by this biological link rises to 70%. This particular comparison makes little sense, however. In contrast to Coale, Sen’s estimates are not based on an explicit assumption about the sex ratio at birth. Instead, he generated his figures by simply
### Table 15.3. Missing women reported by Sen and Coale and “explained” by Oster

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Missing</td>
<td>Adjusted missing (m)</td>
</tr>
<tr>
<td>China</td>
<td>30.4</td>
<td>7.2</td>
</tr>
<tr>
<td>India</td>
<td>22.7</td>
<td>18.6</td>
</tr>
<tr>
<td>Pakistan</td>
<td>3.1</td>
<td>3.1</td>
</tr>
<tr>
<td>Bangladesh</td>
<td>1.6</td>
<td>1.3</td>
</tr>
<tr>
<td>Nepal</td>
<td>0.2</td>
<td>0.2</td>
</tr>
<tr>
<td>Western Asia</td>
<td>1.6</td>
<td>1.1</td>
</tr>
<tr>
<td>Egypt</td>
<td>0.6</td>
<td>0.3</td>
</tr>
<tr>
<td>Total</td>
<td>60.3</td>
<td>31.8</td>
</tr>
</tbody>
</table>

(p.289) comparing the sex ratios in the whole population (not the sex ratios at birth) in the “missing women” countries with those in Sub-Saharan Africa. In fact, if there is a link between HVB carrier status and sex ratio at birth, there is no need to adjust population sex ratios from Sub-Saharan Africa to account for this link, as its rate of HVB carriers is similar to that of China. Nonetheless, if Oster’s analysis of Coale’s figures is sound, she has just about defined away the problem in China and sharply reduced it in Egypt, West Asia and most of South Asia, so it is important to assess the empirical evidence for her analysis. Before doing this, it is useful, however, to comment briefly on one important implication of her results if they proved correct. Since the 1980s, most countries affected by gender bias in mortality, including China, Taiwan and parts of South Asia, have successfully undertaken massive immunization campaigns to reduce the prevalence of HVB, thereby reducing the carrier rate drastically among young couples, which should in turn increasingly affect the sex ratio at birth (and, more slowly, the population sex ratio). If there is a link between HVB carrier status and the sex ratio at birth, this should serve to lower the sex ratio at birth (and of the population as a whole) over time; these effects should have become strongly visible in the 1990s and have affected the sex ratios of the censuses around 2000. Consequently, in the most recent censuses, the gap between the actual sex ratio at birth and the HVB-free sex ratio at birth disappears or at least becomes much smaller; consequently, the number of missing women reported in these censuses increases. One way to gauge the quantitative impact is to examine the estimate of the number of missing women, using Coale’s methods and census data from the late 1990s and early 2000s (which I shall call “Coale updated”, calculated in Klasen and Wink 2003). Using these comparable methods but not accounting for the alleged link between HVB and the sex ratio at birth implies that the number of missing women went up only slightly by about 3 million and fell as a share of the female population (from 5.3% around 1990 to 4.7% around 2000). If, however, the figures in Coale (1991) were inflated due to the neglect of this link, as suggested by Oster, but are no longer inflated in 2000 due to the success of immunization campaigns, the number and share of missing females increased dramatically in the 1990s, from 32 million to 64 million (and from about 2.8% to 4.7% of the female population). This is a rather extreme assumption to illustrate the argument. Even if the immunization campaigns had been totally successful by the
early 1990s in eradicating HVB carrier status among mothers, the effect of HVB on the sex ratio at birth in older cohorts would still be present. But the main point, that the improvements found by Klasen and Wink (2002, 2003) would vanish and turn into a deterioration, remains. Thus (p.290) while Oster’s findings might be comforting in that the levels of gender bias in mortality were smaller in the early 1990s than commonly thought, her results imply that the problem has since got a lot worse than previously believed. This rather worrying implication was not mentioned in her paper.

There are two principal ways to assess the merits of her evidence. One is to examine the different pieces of evidence to see whether they make a convincing case when assembled. The other is to look for evidence on this linkage that has not been put forward by Oster. In the following, I will do both briefly.

Oster begins by presenting time series evidence on the sex ratio at birth and in young age groups in countries affected by gender bias in mortality, with particular emphasis on China. The data on sex ratios in young age groups are difficult to assess given the incentive of parents to under-report children (particularly female children) after the introduction of the one-child policy in 1977.10 The data on the sex ratio at birth are based on a careful historical reconstruction from Banister and Coale (1994) using data from fertility surveys.11 While on average they indeed show elevated (male—female) sex ratios at birth on average and for most time periods since 1930 (compared to the international norm of about 1.06), they oscillate considerably and there are periods in the 1960s where the sex ratio at birth is not much different from the norm in Western countries. As there is no evidence for similar swings in HVB prevalence, this already casts doubt on the presumed link. Also, as these data are based on retrospective surveys, one has to wonder whether in a country with strong son preference there is a sex-specific recall bias. Consistent with this view is the evidence that the sex ratios reported are larger the older the birth cohort.

More seriously, more disaggregated data show particular groups without elevated sex ratios at all. For example, data from the 1982 census on sex ratios at birth (reported in Lai 2005) show that in 11 provinces (out of 29 for which data are reported) the sex ratio at birth is close to 1.06 or below and
thus very similar to the international norm. Similarly, the same paper by Banister and Coale (1994) shows that among first-born children, the sex ratio was within the international norm of about 1.06 for the birth cohorts between 1960 and 1980. Das Gupta (2005) also reports that in 1982 and 1989 the sex ratio at birth in China was equal to the international norm for the first-born child. Both Banister and Coale and Das Gupta find a sharply increased sex ratio at birth only for later-born children, and the sex ratio among these later-born children has increased sharply since the early 1980s. Lastly, Ebenstein (2007), in the most comprehensive assessment of the issue, also presents evidence of the sex ratio at birth in China, India and Taiwan from 1980 to 2000. He also finds that sex ratios of first births are perfectly normal and only rise for higher parities, particularly since 1990, consistent with increasing recourse to sex-selective abortions. All this evidence casts doubt on the claim that sex ratios (p.291) at birth are in general higher in China due to HVB carrier status. The only way this evidence would be consistent with a link to HVB carrier status is if carrier status only affected higher-order parities, but not first births. It is hard to think of a way this could be possible, given that all higher-order births by definition imply lower-parity prior births and that HVB carrier status in high-prevalence countries is usually determined in childhood well before the beginning of childbearing. Also, the microevidence discussed below suggests precisely the opposite relationship between HVB, parity, and the sex ratio at birth.

A second piece of evidence on elevated sex ratios at birth related to HVB carrier status among Chinese populations comes from Chinese immigrants in the USA. According to census data, the average sex ratio among Chinese immigrants is 1.082 when birth registry data are used and 1.105 when smaller census samples are used, which Oster claims to be related to higher HVB prevalence among Chinese immigrants, whose carrier status is affected by conditions in China prior to immigration. Given the source, these data are likely to be much more reliable. But closer inspection also reveals two problems. First, as shown in Table 2 in Oster's paper, between 1940 and 1970 the sex ratio of children born to Chinese immigrants was perfectly normal at 1.047. Only much further
back and since 1980 has the sex ratio at birth been abnormally high.

Related to this is a second problem identified in a recent paper by Abrevaya (2005). Using the same data as Oster plus a richer subsample of all births from California, he first shows that among Chinese, Indian and South Korean immigrants, there is significant gender bias in fertility strategies. The likelihood of these immigrant groups to have a second or third child is significantly higher if the first child (or the first two) has been a girl than if it has been a boy (or two boys). Possibly as a result of fertility decline, this link has become somewhat stronger since 1980, thereby in fact connecting to the discussion above about the impact of fertility decline on gender bias. While this is an interesting finding in itself, such a selective stopping rule does not affect the sex ratio at birth in the absence of sex-selective abortions (see Klasen 2003). But Abrevaya’s paper also reports that the actual sex ratio at birth is affected by family size and the sex composition of the family. Table 15.4 reports some descriptive statistics from that paper on the sex of

Table 15.4. Sex ratio of second child by sex of first child, USA, 1980–1995

<table>
<thead>
<tr>
<th></th>
<th>Girl</th>
<th>Boy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chinese</td>
<td>1.110</td>
<td>1.033</td>
</tr>
<tr>
<td>Indian</td>
<td>1.123</td>
<td>1.016</td>
</tr>
<tr>
<td>Korean</td>
<td>1.070</td>
<td>1.033</td>
</tr>
</tbody>
</table>

Source: Abrevaya (2005).
the second child of Chinese, Indian and Korean immigrants if the first child was a girl or a boy. In each case, the sex ratio is significantly (in a statistical and economic sense) higher when the first child was a girl than when it was a boy. This relationship also holds up in regressions that control for other factors affecting the sex ratio at birth. This is rather strong evidence that the higher sex ratios observed in these Asian immigrant groups are also due to sex-selective abortions of parents trying to ensure that at least one of their children is male. Abrevaya (2005) also presents a range of circumstantial evidence supporting this contention. Conversely, these data, particularly the perfectly normal sex ratios of births when the first child was male, provide strong evidence against the hypothesis that HVB carrier status causes the abnormally high sex ratios among immigrants in the US. If such a link existed, one would expect a generally higher sex ratio at birth and not a link between the sex ratio at birth and parity. Oster then turns to individual and time series evidence for further support. The individual-level evidence reports on the results of several small microstudies that particularly examine this link, while the time series evidence reports on the impact of vaccination campaigns on populations heavily affected by HVB carrier status. The individual-level evidence finds a strong and significant relationship between HVB carrier status and the sex ratio at birth, while the time series evidence finds that for Alaskan Natives as well as Taiwanese, vaccination campaigns are associated with reduced sex ratios at birth.

Let me raise a few empirical issues regarding the microstudies. The sample sizes from the microstudies are very small and the results thus quite unreliable. More importantly, the results are actually not as clear as they have been made out to be, in two ways. First, there are considerable differences in results regarding the role of the mother's versus the father's carrier status on the sex ratio of the offspring. While in several studies, the elevated sex ratio is related to the mother's HVB carrier status (Chahnzarian et al. 1988), in one sample of Melanesian populations, the HVB carrier status of the mother is associated with a significantly lower sex ratio at birth (Hesser et al. 1976), and while in some studies the father's carrier status has a significant effect on raising the sex ratio at birth, in others it does not (Hesser et al. 1976, Chahnazarian et al. 1988). In general, the results seem to be weaker on the effect of father's HVB status than on that of mothers, an issue to which I will return below.
Second, the interaction with birth order is ignored. In her Ph.D. dissertation on the sex ratio at birth (the late) Anoush Chahnazarian found that the carrier status of parents appears to increase the sex ratio at birth in low birth orders but not in higher parities (Chahnazarian 1986). She concludes by saying that “the negative relationship observed here between birth order and the sex ratio at birth in children of carrier parents fails to provide an explanation of the unusually high sex ratios at birth observed at higher parities in China, a country of high hepatitis B prevalence” (135).

(p.293) Thus the empirical evidence from these microstudies is very much open to question. So are the conclusions we can draw from this evidence, such as it is, for the “missing women” countries. Oster reports that there are nine types of hepatitis B virus, with different transmission rates and effects. Given that the link between carrier status and the sex ratio at birth is a pure black-box relationship, it is unclear whether variation in type partly account for the plethora of results in the different microstudies. The type of virus differs by region and none of the microstudies come from the “missing women” countries, so it is unclear which studies reflect the relationship that might prevail there. As a result, it is totally unclear what the evidence from these microstudies suggests about the impact of HVB status on the sex ratio at birth in the “missing women” countries.

Not only is the evidence from these microstudies and its meaning for “missing women” countries inconclusive, but a very large data set from a “missing women” country demonstrates that the HVB carrier status of the mother exerts only a tiny effect on the sex ratio at birth. The study is by Lin and Luoh (2008), who analyze the sex ratio of over 3 million births between 1988 and 1999 to mothers who were included in the Hepatitis B Mass Immunization Program in Taiwan. The very robust result from this study is that there is a positive and significant effect from the HVB status of the mother, but it is extremely small. Using a range of specifications, with or without controlling for covariates, and using different subsamples, the authors find a rather robust effect of the mother’s HVB carrier status increasing the likelihood of a child being male by only 0.25 percentage points. Given the prevalence of HVB in China in the 1980s, this would only raise
the expected sex ratio at birth by 0.17 percentage points (from 1.06 to 1.0617), with a negligible impact on the calculation of “missing females”.

In response to this rather conclusive finding on the relationship between mother’s HVB carrier status and the sex ratio at birth, Blumberg and Oster (2007) have recently argued that it is the father’s HVB status that is affecting the sex ratio at birth. They take two of the microstudies that are consistent with this view, as well as some aggregate data on HVB prevalence rates of males and females in Taiwan and among Chinese immigrants. In each case, there is some evidence consistent with the view that the HVB status of the father raises the sex ratio at birth. While the Taiwanese data used by Li and Luoh cannot be used to assess this new claim (as the carrier status of the father is not included), this new paper takes a rather selective look at the evidence. The papers that earlier investigated this link using individual microstudies and pooling these studies (including the two used by Blumberg and Oster) found that the link between mother’s HVB status and the sex ratio at birth is stronger, while the evidence on father’s HVB status is more mixed (e.g. Chahnazarian et al. 1988). Also, no interaction with birth order is examined in the new study, so that this new claim has to be viewed with a great deal of skepticism. The need for skepticism is confirmed by the findings from Chen and Oster (2008) who examined the births by HVB status of mothers and fathers in a large sample (p. 294) in China and came to the conclusion that the HVB carrier status of either parent did not have a significant impact on the sex ratio at birth. As a result Oster now no longer claims that HVB carrier status is a cause of elevated sex ratios in China.

Turning to the time series evidence, Oster (2005) discusses the effect of immunization programs on the sex ratio at birth among populations with high HVB status and finds that immunization campaigns are associated with falling sex ratios at birth. As noted earlier, the case studies she examines are Alaskan Natives and Taiwanese. The discussion above already addresses the issue of the Taiwanese data. Regarding the Alaskan data, these effects are quite small. In the regression which should generate more precise estimates, as it focuses on Native Alaskans (who were the ones with high HVB prevalence), being a Native Alaskan in 1990 leads to a 0.6% increase in the chance of being male, which is rather small.
indeed and would affect the sex ratio at birth by about one percentage point. Second, being an Alaskan Native has a much larger effect on the sex ratio in whatever time period. This goes against the claim that the unusually high sex ratio at birth among Alaskan Natives is mostly due to their high HVB rates. Third, one should try interactions between Native Alaskans and birth order to see whether the effects are dependent on birth order as well. And as before, it is unclear what this tells us about China and India, given the different types of the virus.

Lastly, Oster's paper presents some cross-country evidence on the link between the sex ratio at birth and HVB prevalence. First, she presents evidence based on WHO classification and shows that the sex ratio at birth is higher in high-prevalence countries, even if one excludes China. The effect is also true within Europe. But the effects are very small. If China is excluded the effect is a little more than one percentage point and would not change the missing women calculation by much. Second, there are no controls for other factors that affect the sex ratio at birth (including overall fertility and mortality conditions). Third, the data on the sex ratio at birth come from the Demographic Yearbook and it is not clear whether she uses all of the data, or only those based on 100% registration of births. There are serious problems of under-reporting, including sex-selective under-reporting, and so one would have to be quite careful here.

Oster then collects 63 studies about HVB prevalence from the medical literature and generates prevalence rates by the weighted average of these studies by country. After dropping studies with fewer than 2,500 individuals, she ends up with a sample of 38 countries. A scatterplot is presented for all countries and also one including just OECD countries and then a regression is presented, with the sex ratio at birth on the left-hand side and HVB prevalence and GDP per capita and other controls on the right-hand side. She finds a significant positive influence of HVB prevalence on the sex ratio at birth in all countries (income has no effect, life expectancy a positive effect) and a subsample of OECD countries (16 countries) and in a sample where there is at least 90% complete birth registration.
The scatterplots and the regression have a number of very serious problems, and for several reasons the results cannot be seen as reliable. First, the sample is very small and it is not clear how representative it is. (In my own analysis with Wink of the influence of life expectancy on the sex ratio at birth, we used more than 200 observations: see Klasen and Wink 2002, 2003). It would be essential to report the HVB rates for all 63 countries (along with sample sizes) to reassure the reader that the sample is not a selective one. Second, the sample is entirely cross-sectional, which raises all sorts of questions about unobserved heterogeneity. The *Demographic Yearbook* does have time series information on sex ratios at birth and one should try to investigate this issue in a panel framework. Third, the reliability of reported sex ratios at birth in the *Demographic Yearbook* is open to question. We know about sex-selective under-reporting of births in China and India (e.g. Johannsson and Nygren 1991; Dyson 2001) and it is likely to be an issue in other countries as well. Fourth, in the figures and the regressions, it appears that the results are heavily influenced by China and South Korea, which ought to be excluded due to questions about the reliability of their reporting of the sex ratio at birth (as well as the impact of sex-selective abortions). Regarding the regressions, one should omit all the countries with evidence of under-reporting of female births and/or evidence of sex-selective abortions. Moreover, the reliability of all the data on the sex ratio at birth need to be checked carefully. Similarly, other factors influencing the sex ratio at birth (e.g. fertility and mortality conditions) should be included in the regressions.

Extending the cross-country analysis, Oster presents evidence on the sex ratios of various immigrant groups in the US, links them to HVB rates in their home countries, and again finds a positive relationship. But again, South Korea, India and China should be thrown out of the analysis for the reasons stated above, including the apparent prevalence of sex-selective abortions. Once this is done, the effect largely disappears. Second, the data concern children, not births, with all the problems stated above with such data. Third, it is bizarre to see that quite a few immigrant groups have sex ratios at birth below 1. Surely this indicates sample size or measurement problems. The regressions should also control for birth order and mortality conditions.
Lastly, Oster discusses a major empirical problem for a link between HVB and the sex ratio at birth, namely evidence from Africa which points to a lower (rather than a higher) sex ratio at birth despite high HVB carrier rates (see also Das Gupta 2005). The author claims that there is a positive relationship (results are not shown) and that the low sex ratios at birth in Africa are due to other reasons.

As shown in many studies (including Klasen and Wink 2003), it is indeed the case that Africans have a slightly lower sex ratio at birth. This is well documented in the US, where the difference is about 2 to 3 percentage points (i.e. 1.03 instead of 1.05-1.06); in the Caribbean, the effect is similar (in Africa the data are very sparse but point to similarly lower sex ratios at birth). Thus the race effect is small and, given the high prevalence of HVB in Africa (the rates are among the highest in the world), the presumed HVB effect should easily more than outweigh this race effect. (p.296) Thus we are left to wonder how the high prevalence of HVB in Africa does not lead to a higher sex ratio.

While Oster has presented a large amount of evidence, what my discussion has shown is that all of her pieces of evidence suffer from serious problems, while there is considerable evidence suggesting that the link between HVB carrier status and the sex ratio at birth in “missing women” countries is very small or even nonexistent. Thus at this stage there is little reason to adjust the estimates of missing women as proposed by Oster. If anything, the evidence seems to suggest that such an effect is very small, with negligible impact on the magnitude of the problem. Oster’s retraction of the claim based on her new work in China is further confirmation that the missing women problem cannot be explained away through this link to HVB.

Maybe one of the larger issues regarding this question is the sheer size of the estimates of missing women, which may strike some observers as simply too large to be the result of gender bias in mortality. But a back-of-the-envelope calculation might help to clarify how these numbers can come about. First, it has to be remembered that the number of missing women is a stock, not a flow, thus representing the impact of past and present (pre- and post-birth) gender bias in mortality on the sex composition of the population at the census date. How could India’s 38 million and China’s 34
million missing females in 1991 have come about? Assume that all gender bias in mortality took place among children below 5 and this gender bias ensured that under-5 mortality rates for females were some 50% higher than they would have been in the absence of gender bias in mortality. For the generations alive in 1991, the average female under-5 mortality rate might have been 150 per 1,000 instead of 100 per 1,000 in India (and 90 instead of 60 per 1,000 in China). Given about 20 million births on average among the cohorts alive today in both India and China, that would generate about 1 million missing females per year in India and 0.6 million in China. Aggregation over the cohorts (and allowing for lower life expectancy in India than in China among all the cohorts) could easily generate the numbers of missing females found for the early 1990s. The increasing spread of sex-selective abortions can then have contributed to the increase of the phenomenon, particularly in China. This rough and ready calculation can only give a rough impression of how these staggeringly large numbers might have come about.12

IV. Conclusion
This short essay has focused on recent discussions of the role of fertility decline and the Hepatitis B carrier status on levels and trends in gender bias in mortality. The (p.297) good news is that there is little reason to worry that a fertility decline occurring as a response to overall economic and social development will intensify gender bias in mortality. Only forced fertility decline through coercive family planning policies is likely to have such an effect. The bad news is that the evidence presented by Oster claiming to have found about 45% of the “missing females” is weak and contradicted by more powerful evidence to the contrary. Thus it still is the case that gender bias in mortality is as large as presumed by the calculations made by Sen, Coale, and Klasen and Wink. But even in the bad news there is some good news. While the implication of Oster’s claims had been that gender bias in mortality has been getting much worse recently, the fact that her claim appears to rest on weak foundations suggests that the earlier suggestions made by Klasen and Wink (2002, 2003) still seem to be valid, namely that in most parts of the “missing women” regions, there has been a slight to moderate improvement. The exception is China, where the one-child
policy, combined with the increasing availability of sex-selective abortions, has led to lower female survival.

Bibliography references:


Missing Women


Notes:
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(1) See Basu (1999) for a discussion of the possible reasons for increased son preference.

(2) See, for example, Murthi *et al.* (1995), Drèze and Murthi (2001) and Bhattacharya (2006) for a discussion in the Indian context.

(3) For a discussion of these links from both theoretical and empirical standpoints, see Klasen and Wink (2002).

(4) In Figure 15.1, Nepal is omitted from the scatterplot. The change in the share of missing females in Nepal between 1981 and 2001 was strongly affected by sex-specific out- and in-migration patterns, so the calculation of “missing females” for this country has to be treated with some caution. Even if it were included, however, there would remain a clear negative relationship between fertility decline and the change in the share of missing women (although the correlation would be somewhat weaker).
(5) It is, of course, debatable to what extent the fertility decline in China has actually been driven by the one-child policy. Clearly, significant fertility decline was well under way before the one-child policy was instituted. But it is also plausible that the pace and extent of fertility decline were significantly influenced by that policy. For discussion, see Sen (1998) and Drèze and Sen (2001).


(7) Specification tests suggest that the random-effects results are to be preferred.

(8) See also Klasen and Wink (2003) and Mari Bhat and Zavier (2003) for a similar argument.

(9) There are other reasons why the use of the sex ratio in Sub-Saharan Africa is unlikely to produce reliable estimates of the number of missing women. These are discussed in detail in Klasen (1994) and Klasen and Wink (2002, 2003). Nevertheless, the unusually low male—female ratios in Sub-Saharan Africa, despite very high HVB carrier prevalence, clearly works against the hypothesis proposed by Oster. See the discussion below.


(11) These data are not affected by the incentive to hide daughters associated with the one-child policy.

(12) A more careful assessment would have to consider birth and mortality rates over time by sex and cohort; given data limitations, this cannot easily be accomplished.