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Missing women: some recent controversies on levels and trends in gender bias in mortality

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**Missing Women: Some Recent Controversies on Levels
and Trends in Gender Bias in Mortality**

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Missing Women: Some Recent Controversies on Levels and Trends in Gender Bias in Mortality

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Abstract

This paper 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 authors, fertility decline has not generally led to an intensification of gender bias in mortality. Second, the paper 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 in mortality

JEL Codes: J16, J71m O15

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1. 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 favouring males had been known for some time (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 modelling framework that has powerfully influenced the literature on intrahousehold 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 providing a first estimate of the number of missing women in the countries affected by gender bias in mortality. 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 developed subsequently that refined these estimates of missing women, extended them to more countries, and updated them using more recent census information (e.g. Coale, 1991; Klasen, 1994; Klasen and Wink, 2002; 2003; OECD, 2006). A much larger literature 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 (e.g. see Drèze and Sen, 2001 for a summary of many contributions). Due to its obvious importance, also from a policy point of view, this literature has remained very active.

One issue that has sparked a particular amount of debate has been the role of fertility decline in affecting gender bias in mortality. In particular, several scholars suggested that fertility decline in countries with son preference would lead to intensified gender bias in mortality (e.g. Rajan et al., 2000; Basu, 1999, 2000; Das Gupta and Mari Bhat, 1997). Others suggested, however, that this linkage is empirically weak, at least in India, or appears to actually go in the opposite direction (e.g. Mari Bhat and Xavier, 2003; Drèze and Murthi, 2001). The first purpose of this paper is to comment on this debate and reflect on it 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 was partly a result of coercive family planning policies (as in China) or where son preference remained strong but desired fertility was already very low (as in South Korea), fertility decline can lead to an intensification of gender bias, at least for some period of time; in most other circumstances, however, the evidence for such a link is weak, either cross-nationally or within nations. In fact, often even the opposite appears to be the case where 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 (2006) has claimed that existing estimates of 'missing women' vastly overstate the phenomenon as they neglect a linkage between the carrier status of the Hepatitis B virus (HVB) and the sex ratio at birth. She argues that there is a positive link between the carrier status of the Hepatitis B Virus of the parents and the sex ratio at birth. Since many of the countries affected by gender bias in mortality also have (or at least have had) a fairly substantial prevalence of the HVB carrier status, more than 50% of the 'missing women' alleged to be victims of gender bias in mortality were more innocuously missing as a result of the HVB status of their parents. This claim has also sparked a controversy where most evidence Oster presented has come under

critical scrutiny and other evidence going against her claims have been presented (see Das Gupta, 2005, 2006; Ebenstein, 2007, Lin and Luoh, 2006, Abrevaya, 2005). The second aim of the paper is to comment on the claims made by Oster as well as the literature that has developed since. Here I will argue that while the claim is rather intriguing and the empirical evidence that presented by Oster substantial, closer inspection of the evidence presented generates serious questions and problems with *all* pieces of the evidence presented. Moreover, the evidence against such a link between HVB carrier status and the sex ratio at birth as well as the evidence on parental strategies of son preference is also so substantial that, at this stage, the claim that much of gender bias in mortality is mostly due to this biological linkage appears to be on weak foundation; instead it appears that a possible link between HVB carrier status and the sex ratio at birth has at most a minor impact on the number of 'missing women'. As a result, it remains the case that gender bias in mortality (including now pre-birth strategies of sex selection) accounts for the overwhelming part of the sex ratio imbalances observed in South and East Asia.

2. 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 10-20 years. In fact as shown in Table 1 which presents total fertility rates (TFR) matched to the two most recent censuses that has been used for the missing women calculations in Klasen and Wink (2003), the TFR has fallen dramatically in many of them, including Bangladesh, India, Pakistan, and most Middle Eastern countries. In China and Korea, much of the fertility decline has occurred prior to the 1990s, and now it is below replacement levels in both. Only in Afghanistan, fertility has persisted at extremely high levels.

There are a number of reasons why one might expect that such fertility decline could lead to an intensification of gender bias in mortality. Given the (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 is a male. This had been called the intensification effect by Das Gupta and Mari Bhat (1999). In addition, Basu (1999) argued that son preference might even increase as a result of fertility decline, further intensifying gender bias.¹ As a result, the whole range of pre- and post-birth strategies could be applied to ensure that the much fewer children include at least one male. Among the strategies parents would have at their disposal would be selective stopping rules, i.e. stopping child-bearing as soon as the first son is born, using sex-selective abortions to increase the likelihood to have sons, and favouring 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 to achieve the objective. It is likely that these effects 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 (such as Korea) that a large share of parents would not have a surviving son unless some of these strategies to manipulate the sex composition of the off-spring were applied.

[Table 1 about here]

While these are all plausible arguments, 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, it is much more reasonable,

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

however, 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 on son preference and thus on gender bias in mortality.² 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 this endogenous fertility decline is associated with *reductions* in gender bias in mortality. This would particularly be the case if the causal factors driving these developments were rising incomes, rising female education, and rising female employment levels, all of which are likely to be associated with reduced gender bias in mortality.³

As a result, this issue becomes an empirical question and in the following I want to briefly present some empirical evidence on the link between fertility decline and developments in gender bias in mortality, both in a cross-country context as well as in selected countries including China, Korea, and India, where these issues have been raised particularly.

Table 1 and Figure 1 speak a rather clear language on the relationship between fertility decline and gender bias in mortality in the countries with the most serious problem of ‘missing females.’ Table 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 fertility decline over the same time 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 the extent of fertility decline and the change in gender bias in mortality, i.e. the larger fertility decline, the higher the increase in gender bias in mortality. Figure 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 fallen the most.⁴

[Figure 1 about here]

China and (South) Korea are included in Table 1 and Figure 1 and one might plausibly object here that most of the fertility decline had taken place before 1990 (1985 in the case of Korea) so that it may still be the case that the stronger previous fertility decline was associated with an intensification of gender bias. In the case of China, there clearly is some relevance to this argument. As demonstrated very clearly in Banister (1987), Banister and Coale (1994), Attane (2007), Ebenstein (2007), and Lai (2005), the fertility decline that took place 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 this already poor state of affairs it had reached by 1990. But it should be pointed out that the fertility decline in China was of a special sort, as it was 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 the other countries, fertility decline in the context of the coercive one-child policy can be

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 a theoretical and empirical, see Klasen and Wink (2002).

⁴ Note that in Figure 1, Nepal is omitted from the scatterplot. The change in the share of missing females in Nepal between 1981 and 2001 is strongly affected by sex-specific out and in-migration patterns there so that the calculation of ‘missing females’ for this country has to be treated with some caution. Including it would, however, not change the conclusion of a negative relationship between fertility decline and the change in missing women (although the correlation would be somewhat weaker).

treated as largely ‘exogenous’.⁵ In an environment of significant remaining son preference, it is therefore not surprising some of the intensification effects that were predicted for India were 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 it generated. While there is no doubt that the relative deterioration in female survival post-birth 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 sex ratio at birth from about 1.06 in the early 1980s to 1.20 in 2000, see Attane 2007 and Klasen and Wink, 2003) might only partly be 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 off-spring more easily than was previously possible.⁶

South Korea is an interesting case study to test the relevance of this argument. Here fertility decline took place without recourse to draconian family planning policies; in addition, also there pre-birth sex determination became available since the 1980s. As shown by Chung and Das Gupta (2007), the sex 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 context of rising education, modernization, economic development and continued fertility decline, the sex ratio at birth has been falling since the early 1990s and is by 2003 close to the levels it had 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 in the past years. Thus after the level-shift of a rising sex ratio at birth brought about by technological change, Korea now fits into the pattern described above where economic and social development appears to both reduce fertility and reduce son preference. Chung and Das Gupta (2007) expect a similar trend to happen in due time in China (and India).

Thus the Chinese experience suggests that the fertility decline occurring as a result of coercive family planning can be a powerful force of intensification of 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 contributed to increasing the ability of parents to determine the sex composition of their off-spring.

Let me now turn to India to examine the relationship between fertility decline and gender bias in mortality. One way to do this is to examine this relationship in at the state level. In Table 2 below, I show some simple regressions of the state-wise sex ratio (females/males) in India’s largest states between 1961 and 2001. I show pooled OLS, fixed as well as random effects

⁵ It is, of course, debatable to what extent the fertility decline in China was actually 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 the one-child policy. For a discussion of the one-child policy, see Drèze and Sen (2001) and Sen (1998).

⁶ See Chung and Das Gupta (2007), Mari Bhat and Xavier (2003) and Klasen and Wink (2002, 2003) for a more detailed discussion and evidence supporting this argument.

results.⁷ The pooled OLS results suggest that high fertility is associated with a higher sex ratio at the state level, thus providing the opposite finding of the hypothesis examined here. It is unlikely, however, that the high fertility is ‘causing’ the high sex ratio, but that the fertility rate is indeed an endogenous variable, where some underlying conditions affect both fertility as well as gender bias at the state level. This suspicion is confirmed by the random and particularly the fixed effects specification. In 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 examined. The results show that there is no longer a positive significant relationship, but that there is not a negative relationship either. So the results for India also show that there is no evidence that fertility decline is leading to an intensification of gender bias in mortality.

Other research also supports this 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 further supports the contention that one cannot treat fertility decline as an exogenous factor affecting gender bias in mortality, holding son preference fixed. Instead it seems much more plausible that the factors that are driving fertility decline, including rising females education, employment, and incomes are precisely the factors also reducing son preference.

Also in the Indian case, however, it is likely that the increasing availability of sex determination technologies is having a one-time level impact on sex-selective abortions and thus pre-birth gender bias in mortality. Thus the worsening of the sex ratio 0-6 in the 2001

[Table 2 about here]

census is likely to be mostly related to this one-time technological shift that has increased the ability of parents to affect the sex of their off-spring, rather than be a result of fertility decline. This can also be seen by examining the year dummies in the regressions below. Compared to 2001, the sex ratios in prior decades were always lower, so that 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.⁸ The prediction would be that the sex ratio also in the younger age groups should slowly drift downwards together with declining son preference, as it has in South Korea recently.

3. Hepatitis B and the Number of Missing Women

Let me now turn to the second debate about levels and trends in gender bias in mortality. In her paper, Emily Oster (2006) suggests that a considerable share of the ‘missing women’, particularly in China, are not due to gender bias in mortality, but due to the prevalence of hepatitis B carriers (HVB) among the adult population which, she argues, has been found to increase the sex ratio at birth. Thus the females did not die through pre-or post-birth sex selection strategies, but were never born due to this epidemiological link; thus they are due to ‘biology’ rather than ‘discrimination’, a finding that was also widely discussed in the popular press (e.g. Barro, 2005). As shown in Table 3 reproduced from her paper, about half of the

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

⁸ See also Klasen and Wink (2003) and Mari Bath and Xavier (2003) for a similar argument.

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 (-77%), where the problem of gender bias in mortality largely vanishes, followed by Egypt (-54%) and West Asia (-30%), and lowest in South Asia (-18% in India, -20% in Bangladesh, and no impact in Pakistan).

As shown in the table, she also compares her estimates of missing women with those of Sen (1992), where the share of missing women explained rises to 70%. This particular comparison makes little sense, however. In contrast to Coale, Sen's estimates were not based on an explicit assumption about the sex ratio at birth. Instead, he generated his figures by simply comparing the *population* sex ratio (not the sex ratio at birth) in the 'missing women' countries with those in Sub-Saharan Africa. Thus it makes little sense to adjust these figures by changing assumptions about the presumed sex ratio at birth. In fact, if there were a link between hepatitis B carrier status and the sex ratio at birth, there would be no need to adjust population sex ratios from Sub-Saharan Africa to account for this link as it also has a similarly high rate of HVB as China.⁹ But even sticking to the figures of Coale just about defines away the problem in China and sharply reduces it in Egypt, West Asia, and most of South Asia so that it is important to assess the empirical evidence to support this claim.

Before doing this, it is useful, however, to briefly comment on one important implication of her results if they proved to be 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 increasingly affect the sex ratio at birth (and, more slowly, the population sex ratio). If there was a link between HVB carrier status and the sex ratio at birth, this should have served to lower the sex ratio at birth (and of the population as a whole) over time; these effects should become strongly visible in the 1990s and affect 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 (called "Coale updated" and 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 would imply 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 1991 by Coale were, as suggested by Oster, inflated due to the neglect of this link, but are no longer inflated in 2000 due to the success of immunization campaigns, the number and share of missing females has increased dramatically in the 1990s, from 32 million to 64 million (and from about 2.8 to 4.7% of the female population). Thus while Oster's findings might comfort us that the *levels* of gender bias in mortality were smaller in the early 1990s than commonly thought, her results imply that the problem has gotten a lot worse since then than previously found. This rather worrying implication is not mentioned in her paper.

[Table 3 about here].

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 sex ratios in Sub Saharan Africa despite very high HVB carrier prevalence clearly works against the hypothesis proposed by Oster. See discussion below.

There are principally two ways to assess the merits of her evidence. One is to examine the different pieces of evidence to see whether they together make a convincing case. The other is to look for other evidence on this linkage that have 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 show indeed elevated 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 the HVB prevalence, this already casts doubt on this presumed linkage. 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 not having 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 the increasing recourse to sex-selective abortions. Thus all this evidence puts into question whether sex ratios at birth are in general higher in China due to the HVB carrier status. The only way this evidence would be consistent with a link to HVB carrier status is if the effect of the carrier status only affected higher order parities, but not first births. It is hard to think of a way how this could be possible given that all higher-order births by definition imply lower parity prior births and that the HVB carrier status in high prevalence countries is usually determined in childhood far before the beginning of child-bearing. Also, the micro evidence to be 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 among Chinese populations related to HVB carrier status comes from Chinese immigrants in the USA. Based on 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 she claims to be related to higher HVB prevalence among the 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.

¹⁰ See Johansson and Nygren (1991) for a careful empirical assessment of the hiding of daughters in China.

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

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 when going back much further and when examining the period 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 increased somewhat since 1980, thereby in fact linking 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 the paper also reports that the actual sex ratio at birth is affected by family size and the sex composition of the family. Table 4 reports some descriptive statistics from the paper on the sex composition of the second child to 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 had been 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 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 a 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.

[Table 4 about here].

Apart from reporting unusually high sex ratios at birth in China and among Chinese immigrants, Oster then turns to individual and time series evidence to further support her contention. The individual-level evidence reports on the results of several small micro studies that particularly examined 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 micro studies. The sample sizes from the micro studies 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 versus the father's carrier status on the sex ratio of the off-spring. While in several studies, the elevated sex ratio is related to the mother's HVB carrier status (Chahnazarian et al. 1988), in one sample of Melanesian populations, the HCB carrier status of the mother is associated with a significantly *lower* sex ratio at birth (Hesser et al. 1976), and in some studies the father's carrier status has a significant effect on raising the sex ratio at birth, in others not (Chahnazarian et al. 1988, Hesser et al. 1976). In general, the results seem to be weaker on the effect of father's HVB status than those of mothers, an issue to which I will return below.

Second, the interaction with birth order is ignored. In his 1986 Ph.D. in Princeton on the sex ratio at birth the (late) 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). He 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 (p.135).”

Thus the empirical evidence from these micro studies is very much open to question. So are the conclusions we can take from this evidence, such as it is, for the ‘missing women’ countries. Oster reports that there are 9 types of the 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 different types partly account for the plethora of results in the different micro studies. The type of virus differs by region and none of the micro studies come from the ‘missing women’ countries so that it is unclear which studies reflect the relationship that might prevail there. As a result, it is totally unclear what the evidence from these micro studies 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 micro studies and its meaning for ‘missing women’ countries inconclusive, but a very large data set from a ‘missing women’ country demonstrates that there is only a tiny effect of the HVB carrier status of the mother on the sex ratio at birth. The study is by Lin and Luoh (2006) who analyze the sex ratio of over 3 million births between 1988 and 1999 born to mothers who were included in the Hepatitis B Mass Immunization Program in Taiwan. The very robust result from this study has been that there is a positive and significant effect of 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 micro studies 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 is raising 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 micro studies 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 done in the new study so that this new claim has to be viewed with a great deal of skepticism.

Turning to the time series evidence, Oster (2006) discusses the effect of immunization programs on the sex ratio at birth among populations with high HVB status and finds that

immunization campaigns were associated with falling sex ratios at birth. 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 HIB prevalence) being an Native Alaskan in 1990 leads to an 0.6% increase in the chance of being male which is rather small indeed and would affect the sex ratio at birth by about 1 percentage point. Second, there is a much larger effect of being an Alaskan Native on the sex ratio in whatever time period. This goes against the claim that the unusually high sex ratio at birth among Alaskan Native's was mostly due to their high HVB rates. Third, one should try interactions between natives 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, the paper presents some cross-country evidence on the linkage 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 data, or only those based on 100% registration of births. There are serious problems of underreporting, including sex-selective under-reporting and one would have to be quite careful here.

Then she collects many studies about HVB prevalence from the medical literature and generates prevalence rates by the weighted average of these studies by country. It yields a sample of 38 countries (where at least 1500 have been tested). A scatterplot is presented for all counties and one just including OECD countries and then a regression is presented with the sex ratio at birth on the left-hand side and HVB prevalence and GDP/capita and other controls on the right-hand side. She finds a significant positive influence on HVB prevalence on the sex ratio at birth (income has no effect, life expectancy a positive effect) in all countries and a sub-sample of OECD countries (which includes 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 the results cannot be seen as reliable for several reasons. First, the sample is very small and one does not know how representative it is. (In my own analysis 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 critical to report the HVB rates for all 63 countries (along with sample sizes) to reassure the reader that the sample is not a selected one. Second, the sample is entirely cross-section 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 birth in China and India (e.g. Dyson, 2001; and Johannsson and Nygren, 1991) 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 the countries I have just mentioned. Second, the reliability of the sex ratio at birth data

needs to be checked. Third, 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, she presents evidence on the sex ratio of various immigrant groups in the US and links them to HVB rates in their home countries and finds again 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 would largely disappear. Second, it includes 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 here are sample size or measurement problems. The regressions should also control for birth order and mortality conditions.

Lastly, she discusses a major empirical problem for the link between HVB and the sex ratio at birth which is 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 2-3 percentage points (i.e. 1.03 instead of 1.05-1.06); in the Caribbean, the effect is similar (in Africa the data is very sparse but points 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. Thus we are left to wonder how the high HVB in Africa does not lead to a higher sex ratio.

While Oster has presented a large amount of evidence, what the discussion has shown is that all pieces of evidence presented 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 non-existent. Thus at this stage there is little reason to adjust the estimates of missing women as had been proposed by Oster. If at all, the evidence seems to suggest that such an effect is very small with negligible impact on the magnitude of the problem.

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 in clarifying 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 five and this gender bias ensured that under-five mortality rates for females that were some 50% higher than it would have been in the absence of gender bias in mortality. For the generations alive in 1991, the average female under five mortality rate might have been 150/1000, instead of 100/1000 in India (and 90 instead of 60/1000 in China). Given about 20 million births on average among the cohorts alive today in both India and China, would generate about 1 million missing females per year in India and 0.6 million in China. Aggregated over the cohorts (and making allowance 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

4. Conclusion

This short paper has picked up recent discussions on the role of fertility decline and the Hepatitis B carrier status on levels and trends in gender bias in mortality. The good news is that there is little reason to worry that a fertility decline that occurs 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 ‘missing females’ is rather 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 would have been that gender bias in mortality has been getting much worse recently, the fact that her claim appears to be on weak foundations suggests that the earlier suggestions made by Klasen and Wink (2002, 2003) still seems to be valid. There is was argued that in most parts of the ‘missing women’ regions, there has been a slight to moderate improvement. The exception had been China where the one-child policy, combined with the increasing availability of sex-selective abortions has lead to a worsened situation for female survival.

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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.

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Table 1: Fertility decline and Changes in Gender Bias in Mortality

	Most Recent	Percent Females „Missing“	TFR	Previous	Percent Females „Missing“	TFR	Change TFR	Change Share Missing
Afghanistan	2000	9.30%	6.84	1979	9.70%	6.9	0.06	0.40%
Algeria	1998	1.24%	3.05	1987	2.69%	5.72	2.67	1.45%
Bangladesh	2001	4.23%	3.13	1981	8.91%	6	2.87	4.68%
China	2000	6.69%	1.89	1990	6.31%	2.1	0.21	-0.38%
Egypt	1996	4.46%	3.55	1986	5.14%	4.3	0.75	0.68%
India	2001	7.89%	3.07	1991	9.44%	3.81	0.74	1.55%
Iran	1996	3.68%	3	1986	4.54%	5.9	2.9	0.86%
Korea	1995	0.73%	1.75	1985	-0.14%	2.04	0.29	-0.87%
Nepal	2001	0.50%	4.27	1981	7.71%	6.1	1.83	7.22%
Pakistan	1998	7.76%	4.77	1981	10.78%	7	2.23	3.01%
Sri Lanka	1991	-0.02%	2.5	1981	3.44%	3.25	0.75	3.47%
Syria	1994	3.06%	4.2	1981	4.98%	7.4	3.2	1.92%
Tunisia	1994	2.13%	2.9	1984	4.45%	4.32	1.42	2.33%
Turkey	1990	2.40%	3	1985	3.15%	3.79	0.79	0.75%

Source: Klasen and Wink (2003) and World Development Indicators.

Table 2: Determinants of the sex ratio of India's largest states, 1961-2001

	OLS	Random Effects	Fixed Effects
Total Fertility Rate	1.91 (0.86)	0.29 (0.62)	0.09 (0.16)
South	-6.15 (-3.09)***	-8.53 (-3.21)***	
East	-3.68 (-2.42)***	-3.88 (-1.14)	
West	-1.32 (-0.86)	-2.72 (-0.88)	
1971	-4.17 (-1.86)**	-0.07 (-0.05)	0.39 (0.28)
1981	-2.77 (-1.62)*	-0.49 (-0.56)	-0.21 (-0.24)
1991	-0.32 (0.75)	0.39 (0.75)	0.49 (1.07)
Adj. R-Squared	0.52	0.54	0.13
N	58	58	58

Source: Registrar General (2003). T-ratios based on robust standard errors are reported in parentheses. * refers to 90%, ** to 95% and *** to 99% significance levels (one-tail test). For details of the states included, refer to Klasen and Wink (2003).

Table 3: Missing Women, Reported by Sen and Coale and ‘Explained’ by Oster

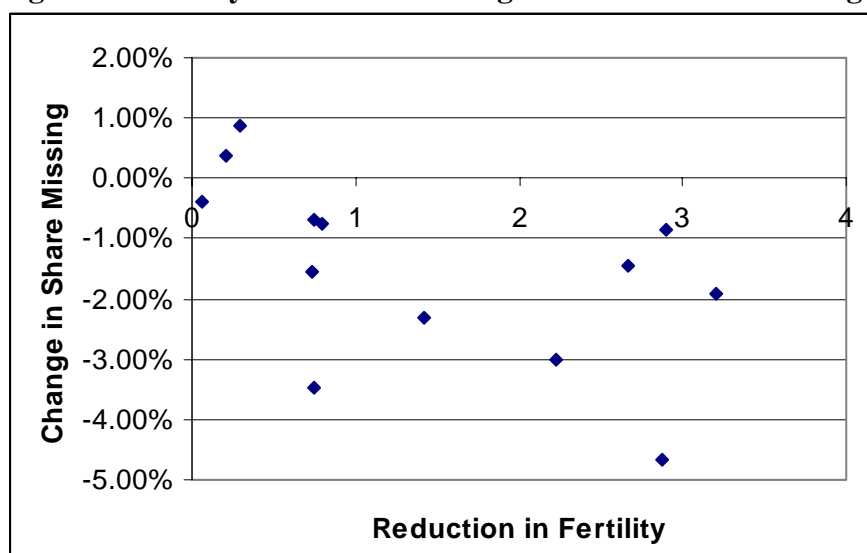
	Coale (1991)			“Coale updated”	Sen (1992)		
	Missing	Adjusted Missing (m.)	Share ‘Explained’ (%)	Missing	Missing	Adjusted Missing (.m)	Share ‘Explained’ (%)
China	30.4	7.2	76.4	32.3	50.0	7.2	85.6
India	22.7	18.6	18.2	24.6	41.6	18.6	55.3
Pakistan	3.1	3.1	0.3	3.4	5.2	3.1	40.6
Bangladesh	1.6	1.3	19.6	1.0	3.8	1.3	65.7
Nepal	0.2	0.2	0.0	-0.3	0.6	0.2	60.8
Western Asia	1.6	1.1	29.5	1.8	4.7	1.1	75.8
Egypt	0.6	0.3	53.7	0.7	1.7	0.3	82.9
Total	60.3	31.8	47.2	63.6	107.5	31.8	70.4

Source: Oster (2006). “Coale” (2003) is taken from Klasen and Wink (2003) using his method and applying it to census data of the late 1990s and early 2000s.

Table 4: Sex ratio of second child by sex of first child in 1980-1995 in the USA

	Girl	Boy
Chinese	1.110	1.033
Indian	1.123	1.016
Korean	1.070	1.033

Source: Abrevaya (2005).

Figure 1: Fertility Decline and Changes in the Share of Missing Females

Source: Table 1.