Can Biological Factors Like Hepatitis B Explain the Bulk of Gender Imbalance in China? A Review of the Evidence

Monica Das Gupta

A recent study challenges the assumption that the large deficit of girls in East and South Asia reflects the preference for sons, suggesting that much of the deficit—as much as 75 percent in China—is attributable to hepatitis B (HBV). The claim is inconsistent with the results of a study based on a large medical data set from Taiwan (China), which indicates that HBV infection raises a woman’s probability of having a son by only 0.25 percent. In addition, demographic data from China show that the only group of women who have elevated probabilities of bearing sons are those who have already borne daughters. This pattern makes it difficult to see how any biological factor can explain a large part of the imbalance in China’s sex ratios at birth, unless it can be shown that it somehow selectively affects those who have borne girls or causes them to first bear girls and then boys. The Taiwanese example suggests that this is not the case with HBV, the impact of which is unaffected by the sex composition of previous births. The data thus support the cultural rather than the biological explanation for gender imbalance.

JEL codes: J11, J13, J16

East and South Asia have large deficits of girls (Sen 1990; Coale 1991; Klasen 2002). This is not a new phenomenon. Sex ratios (the number of males divided by the number of females) in China were high throughout the twentieth century, especially during certain periods (figure 1). In India large shortfalls of girls have been evident since national censuses began in the late nineteenth century (Hutton 1931; Visaria 1969).

The size of the female deficit is striking, as Sen (1990) notes. In the 2000/01 censuses, the excess of boys over girls among children ages 0–4 was 17 percent in China, 10 percent in the Republic of Korea, and 8 percent in India. The gap
was even larger in certain subregions: in two states in northwest India and several provinces in south and east China, boys outnumbered girls by 24 percent or more.

Several mechanisms underlie this shortfall of girls. These include female infanticide and neglect of young girls and, more recently, prenatal sex selection. The Chinese data show rising sex ratios at birth by birth order even before the advent of technology for detecting the sex of unborn children (Zeng and others 1993);
the same phenomenon has been recorded in India since the first censuses (Panigrahi 1972; Miller 1981). Research on the mechanisms of neglect of female children indicates mild discrimination in food and significant discrimination in medical care, especially in South Asia (Hutton 1933; Wyon and Gordon 1971; Chen, Huq, and d’Souza 1981; Das Gupta 1987; Basu 1989). Since the advent of sex-selection technology, there has been a shift from postnatal to prenatal sex selection among those who can access and afford this new technology (Goodkind 1996).

The problem is not one of generalized discrimination against girls, practiced perhaps unconsciously by parents who have internalized certain norms favoring boys over girls. As Das Gupta (1987) shows, excess female child mortality is concentrated among girls born to parents who already have a daughter. There is also a (far smaller) rise in mortality among boys born into families that already have a son. This indicates a conscious parental strategy to manipulate the sex composition of their children, especially to avoid raising several girls.

These results have been replicated in a large number of studies across South and East Asia (Choe 1987; Muhuri and Preston 1991; Zeng and others 1993; Park and Cho 1995; Arnold, Kishor, and Roy 2002; Bhat and Zavier 2007; Lin and Luoh 2007). These studies find that when parents have borne a daughter, subsequent girls are at sharply elevated risk of dying either before or after birth. High sex ratios at birth are caused either by sex-selective abortion or by cases of infanticide that are not recorded as births. Since sex-selection technology became widely available, its use has been found to be strongly correlated with the sex composition of children: the sex ratio at birth rises sharply if the parents have borne a daughter and have not yet borne a son. These studies also show discrimination against nonfirstborn sons, although it is very small compared with discrimination against nonfirstborn girls.

Across South and East Asia, it is widely believed that discrimination against girls reflects cultural factors that make girls much less valuable to their parents than boys. The roles of cultural and economic factors become closely intertwined because the culture—in the form of a strongly patrilineal family system—creates a situation in which girls are excluded from contributing to their parents’ households after marriage (Das Gupta 1987, 1995; Park and Cho 1995; Croll 2001). A woman can contribute only to her husband’s household and to care for his parents. Thus, raising a girl is viewed as “watering another person’s garden” (Attane and Guilmoto 2007). In societies with bilateral kinship systems, such as those in Southeast Asia, where both sons and daughters can support their parents (Casterline, Chang, and Domingo 1993), child sex ratios are normal (Das Gupta et al. 2003, map 1).

On the basis of this understanding of the cultural and economic reasons underlying son preference, these countries have designed public policies to reduce
discrimination against girls. For example, both the Chinese and Indian governments have sought to increase gender equity through a wide range of interventions aimed at changing people’s perception that girls are less desirable than boys and at bringing women into public life. These interventions include vigorous media campaigns to change ideas about gender roles and equity, legislation to enhance gender equity (Croll 2000; Das Gupta and others 2004), and financial incentives to parents to raise daughters (George 2000; China Daily 2006).

Oster (2005) has strongly challenged the assumption underlying these policies, arguing that up to 75 percent of the female deficit in China (and 20 percent in South Asia) can be accounted for by the high prevalence of hepatitis B (HBV). Her claim would suggest that instead of focusing primarily on the difficult task of altering parental preference for sons, the first plank of policy interventions should be the much simpler task of immunizing people against HBV.

There is much evidence that biological factors can cause gender differentials in health outcomes (Waldron 1983; Vlassoff and Bonilla 1994; Hansen, Moller, and Olsen 1999). Studies also show that the sex ratio at birth in Sweden rose by several percentage points over several centuries as women’s nutritional status improved (Johansson and Nygren 1991). Other factors can also play a role. Norberg (2004) finds that sex ratios at birth are higher for women who are living with a spouse or partner before the child’s conception or birth.

This article examines Oster’s hypothesis, reviewing the medical and demographic evidence for and against it. Since her strongest conclusions are for China, the focus is on evidence from China. In contrast to her findings, the analysis here suggests that HBV infection has a very small effect on the female deficit in China. In fact, the patterns of sex ratio at birth in China (as in other Asian countries) are difficult to reconcile with a large role of biological factors of any kind.

Studies of the Relation between Hepatitis B Status and Sex Ratios at Birth

This section reviews the evidence from medical studies of the relation between HBV and sex ratios at birth and raises several questions about Oster’s hypothesis.

Evidence from Micro-studies

Six micro-studies carried out by medical scientists find a relation between HBV and the sex ratio of births. Blumberg (2006) summarizes these results:

In a study in Greece, families in which either parent was a carrier of HBV had a higher ratio of boys to girls than families where the parents
(particularly the mother) had anti-HBs... Similar studies were done in five other populations; they were consistent with the initial results. [Authors’s note: antibodies against the surface antigen of the Hepatitis B virus, indicating immunity to the virus.].

Chahnazarian, Blumberg, and London (1988) note that the populations studied were very small and that the sex ratios therefore fluctuate across studies. The magnitude of the effect is thus unclear, although the pattern is apparent. Summarizing the results from the three studies with the best data quality, they find that if either parent is an HBV carrier, the probability of a male birth is 1.47 times higher than in families in which both parents are uninfected. Given the small numbers of births, however, the confidence interval is so large that they can conclude only that the sex ratio at birth for children born to carrier parents is significantly different from the normal sex ratio at birth (of 1.06), and even this finding holds in only two of the three studies. Chahnazarian, Blumberg, and London tried hard to dissect the data from these three studies to see whether the carrier status of the father or the mother had a larger effect, but were unable to identify differential effects.

Such questions have potential relevance for medical science that goes far beyond sex ratios at birth. As Blumberg (1976, 288) notes, “This connection of HBV with sex selection may also explain why there is a greater likelihood of rejection of male kidneys by renal patients with anti-HBs and indicate how kidneys can be better selected for transplantation.”

Oster’s 2005 Study

Struck by these findings, Oster (2005) set out to test for them on a population level in a large number of countries. She uses a variety of data and analytical approaches, including cross-country analyses, time-series evidence using a natural experiment based on campaigns in Alaska and Taiwan (China) to vaccinate women against HBV, and an estimation of the proportion of the female deficit in China and India that is attributable to HBV.

Identifying a relation between sex ratios and HBV is not easy, for several reasons. First, the estimates of the prevalence of HBV have to be derived from scattered studies, which often represent a minuscule fraction of a country’s population. As Oster (2005, table 10 footnote) notes, “HBV prevalence is calculated...by aggregating published studies.... Only countries with more than 2,500 people tested for HBV [combining all studies] are used in the analysis.” Second, only an indirect estimate of the effect of HBV status on the sex ratio at birth can be made, because the direct estimates from the micro-studies are based on very small numbers.
In her cross-country analysis, Oster finds a clear correlation between the sex ratio at birth and the prevalence of HBV. However, she notes that because of issues of data quality, these regressions omit data from Sub-Saharan Africa, where the sex ratio at birth is low and the prevalence of HBV high. And, as discussed below, similar issues of quality affect the data for many other developing countries that were not dropped from her analysis, including India, which she analyzes extensively.

Oster finds that vaccination campaigns clearly reduced the sex ratio at birth in Alaska. In Taiwan (China), the effect is less clear. Taiwanese vital statistics show that the sex ratio at birth hovered around 109 between 1996 and 2002, rising slightly to 110 in 2003–04. This pattern does not suggest that the vaccination campaign had any significant effect on the sex ratio at birth, as girls who were vaccinated in 1984 (the year the campaign began) would have been 15 in 1999.

Oster (2005, 1166) then calculates the proportion of the female deficit in China and India that can be explained by the prevalence of HBV, combining her estimates of “the prevalence of hepatitis B and estimates of the effect of hepatitis on the sex ratio at birth to estimate and adjust the number of missing women.” These calculations appear to be estimates of the impact only of the mother’s (not the father’s) HBV status, as the prevalence data pertain only to pregnant women (2005, table 10).

Using these estimates of HBV prevalence and impact on the sex ratio at birth, Oster estimates that up to 75 percent of the “missing women” in China in the early 1990s and 20 percent of the “missing women” in South Asia are attributable to parental infection with HBV. Her conclusions are striking and have received much attention (Barro 2005; Dubner and Levitt 2005). Given their strong implications for policy, they warrant deeper exploration with more robust data.

**Lin and Luoh’s 2007 Study**

A very large national longitudinal data set from Taiwan (China) permits robust estimation of the impact of the mother’s HBV status on sex ratios at birth. These data were collected for a national HBV immunization program. Women were vaccinated against HBV, and babies born to women who tested positive were given a dose of HBV immune globulin within 24 hours of birth. These stringent requirements made for high-quality data collection for a very large sample covering up to 90 percent of births.

Using the data collected between 1988 and 1999, Lin and Luoh (2007) track the sex ratio of births to women whose HBV status was known. They find that women who were infected had only a 0.25 percent higher probability of having a son than other women. With a prevalence of HBV infection as high as 15 percent in the population, this translates into an increase of only 0.165 percent in the sex
ratio at birth, raising it from a baseline of 105.000 to 105.165. The sex ratio at birth for all births in the data set was 109, so HBV can account for only a tiny fraction of the elevation.

Oster (2005) estimates that the prevalence of HBV among pregnant women in China is 11.24 percent, which should result in a sex ratio at birth of 1.10. This is the basis for her estimate that HBV accounts for the bulk of excess male births in China. The findings from the detailed medical data set from Taiwan (China) suggest that she massively overestimates the impact of maternal HBV infection.

**Blumberg and Oster’s 2007 Hypothesis**

In response to Lin and Luoh’s (2007) study, Oster revised the premise of her argument. In a study with Blumberg, she now concludes that the effect of HBV on the sex ratio at birth is driven largely by the father’s HBV status and that the mother’s HBV status plays at most a much weaker role (Blumberg and Oster 2007). As in her earlier article, this study uses multiple approaches to arrive at its conclusions:

We present three pieces of evidence that this may be the case. First, using two of the original datasets on this topic we find that father’s infection is more strongly correlated with sex ratio than mother’s infection. Second, in population-level data from Taiwan we find that paternal cohort infection rates are more important than maternal cohort infection rates. Finally, we show using the IPUMS [Integrated Public Use Microdata Series] dataset that children born in the United States to men born in China are more likely to be boys, but this finding does not hold for children born to women from China. (Blumberg and Oster 2007, abstract)

Each of these analytical approaches raises some questions. First, Blumberg and Oster revisit the three individual-level data sets, on the basis of which Chahnazarian, Blumberg, and London (1988) concluded that sex ratios at birth were elevated if either parent was an HBV carrier and that the data did not permit them to conclude that one parent’s status was more important than the other’s. Blumberg and Oster run regressions on two of these three data sets to show that only the father’s HBV status is significantly associated with a distorted sex ratio at birth. This conclusion is at odds not only with the previous analyses, but also with the evidence from a larger study on Greece that Blumberg and Oster cite in their paper (reproduced here in table 1). This larger study (Greece 2) documents an increase in the sex ratio at birth associated with HBV status that is similar to that in the two data sets Blumberg and Oster reanalyze (Greece 1 and the Philippines). However, unlike these two studies the Greece 2 data set includes information only on the mother’s HBV status (Livadas and others 1979), so the observed differentials in the sex ratio at birth are associated with the mother’s
HBV status. Unless there was a near-perfect correlation between the mother’s and the father’s HBV status, these data would be inconsistent with Blumberg’s and Oster’s reformulated hypothesis.

Second, using population-level data from Taiwan (China) on HBV prevalence by age from testing done in primary school, Blumberg and Oster construct expected HBV prevalence by five-year age cohorts. They then examine the sex ratio of children born to parents who belong to different age cohorts, who have different probabilities of infection. Once again they find that only the expected HBV of the father’s cohort affects the sex ratio at birth. It is difficult to assess this information, because Blumberg and Oster do not present basic information on the data, such as the number of age cohorts available for this analysis.  

Third, the authors use U.S. census data to look for patterns in the sex ratios of children born to Chinese immigrants, who are assumed to have levels of HBV prevalence similar to those in their place of origin. They find that the offspring of Chinese couples have an elevated sex ratio at birth, as do those of Chinese men who marry non-Chinese women. However, Chinese women who marry non-Chinese men do not show an elevated sex ratio at birth among their offspring. Blumberg and Oster interpret these findings as showing that only paternal HBV status drives the elevated sex ratio at birth.

<table>
<thead>
<tr>
<th>Location</th>
<th>HBV status</th>
<th>Sons</th>
<th>Daughters</th>
<th>Sex ratio</th>
</tr>
</thead>
<tbody>
<tr>
<td>Greenland</td>
<td>Positive</td>
<td>64</td>
<td>60</td>
<td>1.07</td>
</tr>
<tr>
<td></td>
<td>Negative</td>
<td>174</td>
<td>194</td>
<td>0.90</td>
</tr>
<tr>
<td>Kar Kar Island</td>
<td>Positive</td>
<td>63</td>
<td>54</td>
<td>1.17</td>
</tr>
<tr>
<td></td>
<td>Negative</td>
<td>163</td>
<td>206</td>
<td>0.79</td>
</tr>
<tr>
<td>Greece 1</td>
<td>Positive</td>
<td>90</td>
<td>51</td>
<td>1.77</td>
</tr>
<tr>
<td></td>
<td>Negative</td>
<td>287</td>
<td>255</td>
<td>1.13</td>
</tr>
<tr>
<td>Philippines</td>
<td>Positive</td>
<td>66</td>
<td>41</td>
<td>1.61</td>
</tr>
<tr>
<td></td>
<td>Negative</td>
<td>304</td>
<td>301</td>
<td>1.01</td>
</tr>
<tr>
<td>Greece 2</td>
<td>Positive</td>
<td>52</td>
<td>30</td>
<td>1.73</td>
</tr>
<tr>
<td></td>
<td>Negative</td>
<td>1,006</td>
<td>955</td>
<td>1.05</td>
</tr>
<tr>
<td>France</td>
<td>Positive</td>
<td>20</td>
<td>12</td>
<td>1.66</td>
</tr>
<tr>
<td></td>
<td>Negative</td>
<td>149</td>
<td>122</td>
<td>1.22</td>
</tr>
</tbody>
</table>

Notes: This table shows sex ratios among the children of carrier and noncarrier parents in four regions. Data were collected by testing married women and, in all cases except Greenland, their husbands for HBV. The citations for each study are as follows: Greenland—Drew, Blumberg, and Robert-Lamblin 1986; Kar Kar Island—Drew and others 1982; Greece 1—Hesser, Economidou, and Blumberg 1975; the Philippines—Chanazarian, Blumberg, and London 1988; Greece 2—Livadas and others 1979; France—Cazal, Lemaire and Robinet-Levy 1976.

Source: Blumberg and Oster 2007.

Author’s Note: The “Greece 2” study tested only women (not men) for HBV (Livadas et al., 1979). The italicized sentence in the footnotes to the table above is incorrect.
However, there could be a completely different explanation for this phenomenon. The literature on son preference in China highlights the desire for sons to continue the husband’s lineage, but not the wife’s, since a lineage cannot be continued through a woman. Even if a man born in China marries a woman born in the United States, he would still be subject to pressure to continue his lineage; this would not apply to a woman born in China married to a man born in the United States.

Several questions thus arise about Blumberg’s and Oster’s claim that it is the father’s and not the mother’s HBV status that elevates sex ratios at birth.

The Demographic Evidence

The demographic data, like the medical data, are very difficult to reconcile with the hypothesis that HBV status, whether of the father or the mother, accounts for the bulk of the female deficit in China and India.

Only Women Who Have Borne a Daughter Show an Elevated Probability of Bearing Sons

A very clear pattern emerges from the more than 300,000 births in the 1 percent sample of the 1990 Chinese census. These data cover births during 1989–90, which is close in time to the period for which Oster (2005) calculates the proportions of the Chinese female deficit attributable to HBV. In this enormous database of births, the only group of women with elevated probabilities of bearing sons is women who have already borne daughters (figure 2). Those who have borne only sons show a mildly elevated probability of the next child being a girl—indicative of a mild preference for having a daughter if a son is already safely in place. A similar pattern is found in India (figure 3), although discrimination against girls is less pronounced there than in China.

Across East and South Asia, all the indicators—sex ratios at birth, sex ratios of aborted fetuses, and sex ratios of child mortality—show the same pattern of manipulation of family composition by parents, a pattern that is consistent with strong son preference. Whether females “go missing” is determined by the existing sex composition of the family into which they are conceived (Choe 1987; Muhuri and Preston 1991; Zeng and others 1993; Park and Cho 1995; Arnold, Kishor, and Roy 2002; Das Gupta 2005, Bhat and Xavier 2007; Lin and Luoh 2007). This suggests that cultural factors still provide the overwhelming explanation for the female deficit.

For Oster’s hypotheses to be consistent with the demographic data, women would have to be especially prone to contracting HBV if they had borne a daughter or the disease would have to somehow lead to women first bearing daughters and
Figure 2. Probability of Bearing a Son, by Sex Composition of Woman’s Existing Children, China, 1990

Note: The normal sex ratio at birth is 105–106 boys to 100 girls. The outer limits of the normal range are 104–107 (Chahnazarian 1988).

Source: Das Gupta 2006 based on data from Zeng and others (1993).

Figure 3. Probability of Bearing a Son, by Sex Composition of Woman’s Existing Children, India, 1998–99

Source: Author’s analysis based on data from Bhat and Zavier (2007).
then bearing an excess of sons. (Or men would have to be especially prone to contracting HBV if their wives had borne a daughter or the disease would have to somehow lead to their wives first bearing daughters and then bearing an excess of sons.) Either scenario would require a much more complex set of biological factors to be at work than is indicated by Oster or the micro-studies she cites.

Is it possible that somehow HBV works in these very complex ways? Lin’s and Luoh’s (2007) large medical data set makes it possible to examine this issue. They show that the impact of maternal HBV status is fairly constant across birth orders (figure 4). HBV+ and HBV− women show a similar sharp rise in the sex ratio at birth by birth order. This finding is consistent with that of studies across Asia that show that parents who have not yet borne a son have a higher probability of progressing to higher-order births and become increasingly desperate to ensure that they have a son (Choe and others 1992; Arnold, Choe, and Roy 1998; Larsen, Chung, and Das Gupta 1998). The fact that the HBV impact is fairly constant across birth orders suggests that it is unaffected by the sex composition of previous births.

Lin and Luoh (2007) also test the possibility that some complex biological factor makes women who are HBV+ more prone to bearing a son if they had previously borne girls. Their findings do not support this hypothesis. For women who have a third birth, the sex ratios at birth of HBV+ women remain

Figure 4. Sex Ratio at Birth by Mother’s Hepatitis B Status and Birth Order of Child, Taiwan (China), 1988–99

![Sex Ratio at Birth](image)

marginally higher than those of HBV− women, but there is no significant additional impact of HBV status on the sex ratio at birth of the third child if the previous children were all girls (table 2).

**Population-Level Evidence from Africa**

Oster (2005, 1196) notes that Sub-Saharan Africa appears to offer evidence that is inconsistent with her hypothesis: “To the extent that it is possible to tell, it seems that Africa has relatively low sex ratios and relatively high HBV prevalence.” She notes that the absence of high-quality birth registration data in Africa make it difficult to use data on sex ratios at birth. She therefore compiles data from Demographic and Health Surveys carried out in 18 Sub-Saharan African countries, cautioning that these survey data suffer from defects such as recall bias. After conducting some regressions on these data, she concludes that “these results provide some comfort that Africa is not a particular challenge to the robustness of the HBV–sex ratio connection” (Oster 2005, 1198).

But the limitations of African birth registration data apply to many developing economies, including India, a country Oster analyzes extensively; if anything, the recall bias toward male births would be expected to be higher in India than in Sub-Saharan Africa considering the stronger son preference in India. Also odd is the fact that Oster does not use Garenne’s (2002) compilation and analysis of data on sex ratios at birth from 56 Demographic and Health Surveys conducted in 29 Sub-Saharan African countries.

Neither Garenne’s compilation of the data nor UN estimates show high sex ratios at birth in the African countries Oster lists as having especially high prevalences of HBV (table 3). This is another piece of evidence suggesting that neither parent’s HBV status has a significant effect on the sex ratio at birth.

**Conclusions**

The Chinese data strongly support the cultural explanation for the female deficit that high sex ratios at birth result largely from parental preference for sons. These

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**Table 2.** Sex Ratio at Birth of the Third Child by Sex Composition of Previous Births, Taiwan (China) 1988–99

<table>
<thead>
<tr>
<th>Sex of previous two children</th>
<th>HBV+ mothers</th>
<th>HBV− mothers</th>
</tr>
</thead>
<tbody>
<tr>
<td>Both girls</td>
<td>122.4</td>
<td>121.4</td>
</tr>
<tr>
<td>At least one boy</td>
<td>110.5</td>
<td>110.9</td>
</tr>
</tbody>
</table>

*Source: Lin and Luoh 2007, table 6, and personal communication specifying actual sex ratios underlying the regressions.*
data show that whether females “go missing” depends heavily on the sex composition of the family into which they are conceived. Girls with no older sisters have similar chances of survival as boys. In contrast, girls conceived in families that already have a daughter experience steeply higher probabilities of being aborted or of dying in early childhood.

The demographic data are difficult to reconcile with Oster’s hypothesis that most of the distortion in the sex ratios at birth in China is attributable to the prevalence of HBV. Her hypothesis is difficult to reconcile with the fact that the only group of women who have elevated probabilities of bearing a son are those who have already borne a daughter. Given the strong relation between the sex of children already born and the probability of the next child being a boy, it is difficult to see how any biological hypothesis can explain a significant part of the distortion in China’s sex ratios at birth unless it can be shown that the biological factor works in very complex ways, selectively affecting women (or men) who have borne girls or causing them to first bear girls and then boys.

The medical data from Taiwan (China) show that HBV infection raises a woman’s probability of bearing a son by only 0.25 percent. These data indicate that the effect of HBV status is fairly constant across birth orders, even though the sex ratio at birth rises sharply by birth order, strongly suggesting that the impact of HBV status is unaffected by the sex composition of previous births. The data show that HBV+ status does not selectively affect women who have borne girls. That there is no significant additional impact of HBV status on the sex ratio at birth of the third child if the previous children were all girls seems to rule out the possibility that biological factors can explain the complex patterns found in the demographic data.

Table 3. Sex Ratios at Birth in Sub-Saharan African Countries with High Hepatitis B Infection Rates, 1995–2000

<table>
<thead>
<tr>
<th>Country</th>
<th>Demographic and Health Survey</th>
<th>United Nations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Botswana</td>
<td>0.98</td>
<td>1.03</td>
</tr>
<tr>
<td>Liberia</td>
<td>1.05</td>
<td>1.03</td>
</tr>
<tr>
<td>Malawi</td>
<td>1.01</td>
<td>1.03</td>
</tr>
<tr>
<td>Mali</td>
<td>1.02</td>
<td>1.05</td>
</tr>
<tr>
<td>Togo</td>
<td>1.02</td>
<td>1.02</td>
</tr>
<tr>
<td>Zimbabwe</td>
<td>1.02</td>
<td>1.02</td>
</tr>
<tr>
<td>Sub-Saharan Africa</td>
<td>—</td>
<td>1.04</td>
</tr>
</tbody>
</table>

*Note: Countries are those listed in Oster 2005 as those with high prevalence of Hepatitis B.*

*Source: Demographic and Health Survey data are from Garenne 2002, who cites data from national samples. Where there was more than one survey, data from the most recent year are shown. UN data are from United Nations Population Division 2003 and 2004.*
Oster’s hypothesis is interesting, but it cannot explain more than a very small part of the gender imbalance in China or other Asian countries with large numbers of “missing women.” It appears that the governments of these countries have been correct to focus their policies on changing the cultural roots of son preference.

Notes

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1. For India the age group is 0–6.

2. The figures are from the *Taiwan Demographic Fact Book* (various issues). The author is grateful to Ming-Jen Lin for sharing these figures, which were compiled from the *Taiwan Demographic Fact Book* for his own research.

3. Blumberg and Oster (2007, table 4) mention that they use data collected from school testing conducted in the early 1990s, that the data on births are through 2002, that the parents must belong to different age cohorts, and that both parents’ age cohorts must have been in school in the early 1990s when the testing was done. As women are typically younger than their husbands, the number of children born to the wives of men who were still in school in the early 1990s may not be large.

References


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