Cultural versus Biological Factors in Explaining Asia’s “Missing Women”: Response to Oster

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EMILY OSTER (2005, 2006) argues that 75 percent of the “missing women” in China in the period 1980–90 can be accounted for by infection with hepatitis B. This is a remarkable claim, based on some micro-studies indicating that women with hepatitis B have an elevated probability of having sons (Chahnazarian et al. 1988). If correct, it would imply a need to revise the widely held view that the phenomenon of China’s missing women is largely driven by a cultural preference for sons, which is reflected in parental intervention to manipulate the sex composition of their family. This would be especially good news because if hepatitis B vaccination campaigns could redress most of the problem of unbalanced sex ratios, they would greatly ease the task of Chinese policymakers who are currently engaged in the far harder task of reducing people’s preference for sons.

Unfortunately, this argument is hard to reconcile with the demographic data. The data from a huge sample of births for the period 1989–90 (Zeng et al. 1993) show that the only groups of women with elevated probabilities of bearing a son are those who have already borne daughters (see Figure 1 and Table 1).

These data are consistent with the view that son preference is the predominant explanation for the missing women. The normal sex ratio at birth is between 105 and 106 boys per 100 girls. The more daughters already borne, the more steeply elevated the probability of the next birth being a boy (Table 1). Among women who have borne only daughters, the probability of the next child being a son is elevated by 41 percent if the woman has one daughter, and by a staggering 111 percent if she has more than one daughter. This is consistent with a growing desperation to bear a son. Meanwhile, women who have borne only one or more sons show a mildly el-
FIGURE 1  Probability of bearing a son, by sex composition of a woman’s prior existing children

- **Woman’s first child** (53% of births)  
  normal probability of having a boy

- **Woman has only boy(s)**  
  (18% of births)  
  8% reduced probability of next birth being a boy

- **Woman has only girl(s)**  
  (21% of births)  
  56% excess probability of next birth being a boy

- **Woman has both boy(s) and girl(s)**  
  (8% of births)  
  12% excess probability of next birth being a boy

SOURCE: Table 1.

### TABLE 1  Sex ratio at birth by sex composition of prior existing children in the family, China 1989–90

<table>
<thead>
<tr>
<th>Prior existing children</th>
<th>Percent of births</th>
<th>Sex ratio at birth</th>
</tr>
</thead>
<tbody>
<tr>
<td>None</td>
<td>53.3</td>
<td>105.6</td>
</tr>
<tr>
<td>Sons only</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>15.1</td>
<td>101.4</td>
</tr>
<tr>
<td>2+</td>
<td>2.5</td>
<td>72.8</td>
</tr>
<tr>
<td>All</td>
<td>17.6</td>
<td>96.6</td>
</tr>
<tr>
<td>Daughters only</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>16.0</td>
<td>149.4</td>
</tr>
<tr>
<td>2+</td>
<td>5.5</td>
<td>223.5</td>
</tr>
<tr>
<td>All</td>
<td>21.5</td>
<td>164.8</td>
</tr>
<tr>
<td>Both sons and daughters</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1 son, 1 daughter</td>
<td>4.6</td>
<td>116.4</td>
</tr>
<tr>
<td>1+ sons, 1+ daughters</td>
<td>3.0</td>
<td>121.9</td>
</tr>
<tr>
<td>All</td>
<td>7.6</td>
<td>118.4</td>
</tr>
<tr>
<td>Total</td>
<td>100.0</td>
<td>115.3</td>
</tr>
</tbody>
</table>

evated probability of the next child being a girl—indicative of a mild preference for having a daughter if the son or sons are already safely in place.

For Oster’s hypothesis to be consistent with the data, women would have to be especially prone to contracting hepatitis B if they had borne a daughter. Or the disease would somehow have to lead to women first bearing daughters followed by an excess of sons. Either of these scenarios 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 hepatitis B affects the sex of children in these complex ways? Another piece of evidence is at odds with this possibility. A study of the sex ratio of aborted fetuses in China (see Table 2) shows a pattern entirely consistent with that of the sex ratio of births—namely, that women who have only daughters account for the bulk of the excess of female fetuses among the total abortions. This finding substantially dilutes the possibility that biological factors account for women who first have daughters subsequently bearing an excess of sons.

Many other kinds of evidence support the view that cultural preferences are the primary reason underlying the missing women of China. I summarized some of the large literature on this subject in my first response to Oster (Das Gupta 2005), so here I simply point out one issue about parental intervention that she seems to have misunderstood.

Oster argues that parental intervention cannot be the main driving factor behind the high sex ratios at birth in China. She adduces two kinds of evidence for this claim. One is that sex ratios at birth are high among women of Chinese birth living in the United States: she views this as indicative of their exposure to hepatitis B. But there is no reason why high

<table>
<thead>
<tr>
<th>Prior existing children</th>
<th>Percent of total abortions</th>
<th>Sex ratio of aborted fetuses</th>
</tr>
</thead>
<tbody>
<tr>
<td>None</td>
<td>41.9</td>
<td>107.9</td>
</tr>
<tr>
<td>Sons only</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>23.7</td>
<td>108.0</td>
</tr>
<tr>
<td>2+</td>
<td>0.8</td>
<td>97.6</td>
</tr>
<tr>
<td>Daughters only</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>29.0</td>
<td>51.0</td>
</tr>
<tr>
<td>2+</td>
<td>1.1</td>
<td>53.9</td>
</tr>
<tr>
<td>Both sons and daughters</td>
<td>3.5</td>
<td>106.5</td>
</tr>
<tr>
<td>All</td>
<td>100.0</td>
<td>86.7</td>
</tr>
</tbody>
</table>

sex ratios in this population could not result from choosing to abort a child if routine tests reveal that it is a girl.

The second piece of evidence she adduces is that sex ratios at birth have historically been high in China. Here she appears to believe that parental intervention is possible only when the technology of sex-selective abortion is available. But in fact parents can (and do) intervene through postnatal discrimination, including infanticide, abandonment, and neglect of young children. Cases of infanticide and abandonment are rarely reported as births, and these practices therefore raise the sex ratio of reported births. These practices have been common in China, as the Chinese government and Chinese and Western scholars have acknowledged. When sex-selective technology becomes available, people shift from postnatal to prenatal sex selection (Goodkind 1996).

Across East and South Asia, there is evidence of parental discrimination against daughters, whether prenatally, at birth, or during early childhood. Discrimination during early childhood is reflected in the excess female child mortality widely documented in these regions, which is also concentrated among girls born into families that already have a girl. It is striking that across East and South Asia all the relevant 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, consistent with strong son preference.

All the evidence, then, is consistent with the cultural explanation for the missing women—namely, that parental intervention is the predominant factor underlying the high sex ratios at birth in China. Biological factors such as hepatitis B infection may play a marginal role, but, as I indicated, they would have to work in convoluted ways if they are to explain more than a small part of the totality of missing women. Oster is right to conclude that the subject of missing women is important enough to warrant a complete understanding of causes. What is puzzling is that despite overwhelming evidence to the contrary, she argues that hepatitis B accounts for as much as 75 percent of the problem in China.

Notes

1 Johansson and Nygren (1991), using two centuries of data on births in Sweden, found that the sex ratio at birth rose from around 104.4 in the 1750s, to 105.8 in the 1980s. They attribute this to enhanced viability of male fetuses as mother’s nutritional status improved. Hansen et al. (1999) find that in Denmark in the 1990s the sex ratio is above 106. Chahnazarian (1988) reviews the literature and puts the normal range at 104–107.

2 See the official report of the China Population Information Centre (1984), where female infanticide and abandonment are viewed as responsible for the observed skewed sex ratios. For more recent evidence of these practices, see Johnson (1996).

3 For example, Jha et al. (2006) document in India the same pattern of variation in sex ratios of births and of abortions, according to the preexisting sex composition of children.


References


