The social science literature on Asia's "missing women" (Sen, 1992; Sen, 2003) uses population sex ratios to draw conclusions about women's welfare. Since as a biological matter women tend to be hardier and more resistant to disease or malnutrition than men, the fact that women are actually outnumbered in many Asian countries has been taken as evidence of substantial neglect and discrimination. In recent work, Emily Oster (2005), however, presented evidence that this demographic indicator may be confounded by another factor which is arguably unrelated to women's welfare. Specifically, she draws on an empirical pattern reported in the medical and biological literatures, that parents who are chronic carriers of Hepatitis B Virus (HBV) are especially likely to give birth to sons rather than daughters, and presents evidence that this relationship is causal. Chronic carriers of HBV are especially prevalent in Asia. Therefore, Oster's results suggest that this epidemiological characteristic—and not discrimination and neglect—accounts for nearly half of Asia's estimated 100 million "missing women."

Understanding the causal nature of any HBV-sex of offspring association is critical in this context. On the one hand, suppose the observed association were driven by third factors associated with both chronic HBV infection and sex ratios at birth. Then, identifying these factors would be critical in determining the extent to which sex ratios reflect women's welfare (for example, what if one of them were related to the quality of antenatal care?). On the other hand, suppose the association were shown to be causal, and to be strong enough to affect sex ratios at birth. Then the fact that age cohorts in Asia tend to have particularly large fractions male would ultimately be driven by the factors determining chronicity of HBV infection, which are arguably unrelated to women's welfare (Pope and Gage, 2004; Chang, 2007). Using the "missing women" as an indicator of the level of discrimination and neglect would therefore overestimate the extent of the problem.

Central to the evidence of a causal relationship between HBV chronicity and sex ratios at birth, Oster presents findings from a natural experiment represented by a mass vaccination campaign in Alaska. Alaskan natives have historically been at elevated risk (relative to whites) of being asymptomatic carriers of HBV. Shortly after an effective vaccine was first developed in teh 1980s, state agencies undertook a broad public vaccination campaign, leading to sharp reductions in the prevalence of HBV carriers in the native population. Oster's analysis draws on that historical event, and compares the changes in sex ratios at birth among Alaskan natives and whites before and after the vaccination campaign. The results indicate that before the vaccine was available, when

prevalence of chronic infection with HBV was especially high among Alaskan natives, they were especially likely to give birth to sons. After the vaccine was made available, however, the proportion male among Alaskan native births declined, and eventually came to match that among whites. Unless there was some other change around the same time that affected only the Alaskan native community, and not whites, the vaccine appears to have caused this change in sex ratios.

This paper begins by drawing on the same historical event, and present findings that call into question both the strength and the causal nature of the HBV-sex of offspring relationship. I find that repeating the analysis using data from a different source—one which is better suited to the analysis—produces strikingly different results. The findings I report call into question both the strength and causal nature of the HBV-sex of offspring association. Whereas the original analysis was conducted using data from three censuses, I use data from the vital registration system. Both provide information on the sex of the child and ethnic group of the mother for a large number of Alaskan children before and after the mass vaccination campaign. However, I present evidence that calls into question the suitability of the census data for this purpose. In the census data a child's mother can only be identified if she lives with her child, and in fact the identity of the mother can only be established by inference. The 10% of children who do not appear to be living with their mothers, therefore, cannot be included in the analysis. By contrast, the vital registry explicitly links all newborns to their mothers—by comparing the samples, I show evidence of sex- and ethnicity-specific sample selection dynamics which would bias the original estimated effects of the vaccination campaign. Using the vital registration data, which are not subject to these problems of endogenous sample selection, I find no evidence of a systematic relationship between sex ratios and HBV vaccination. Furthermore, I find that the statistical significance of both the original results, and the results of the replication, depend on very strong assumptions about unobserved factors, and about how these factors correlate over time and between the two ethnic groups.

Taken together, these results call into question both the strength and the causal nature of the HBV-sex of offspring relationship. They indicate that there is at best mixed evidence that population sex ratios at birth are affected by the prevalence of HBV among parents. Therefore, they may ameliorate the concern that using estimates of "missing women" as a measure of women's relative welfare in Asia might be confounded by this factor. Given the current evidence on this matter, it is still too early to conclude that any of the missing women can be attributable to their parents' chronic HBV infection.