

Hepatitis B and Sex Ratios at Birth: Fathers or Mothers?

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Abstract

A number of papers have argued that, at the population level, parental Hepatitis B carrier status is associated with a higher offspring sex ratio (more boys) (Hesser, Economidou and Blumberg, 1975; Drew, London, Blumberg and Serjeanston, 1982; Drew, Blumberg and Robert-Lamblin, 1986; Chahnazarian, Blumberg and London, 1988; Cazal, Lemiare and Robinet-Levy, 1976; Livadas et al, 1979; Oster, 2005). These papers suggest that parents who are carriers of Hepatitis B have roughly 1.5 boys for every girls. Recent large-scale, micro-level evidence from Taiwan (Lin and Luoh, 2006) presents evidence that hepatitis B carrier status among mothers is associated with only a very tiny increase in the probability of a male birth. Both arguments could be correct if it was paternal, not maternal, hepatitis carrier status that drives higher offspring sex ratios. 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 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.

1 Introduction

In the 1970s, researchers studying the Hepatitis B virus (HBV) noticed significant elevation in the offspring sex ratio (boys divided by girls) among children born to parents who were carriers of HBV (Hesser, Economidou and Blumberg, 1975; Drew, London, Blumberg and Serjeanston, 1982; Drew, Blumberg and Robert-Lamblin, 1986; Chahnazarian, Blumberg and London, 1988; Cazal, Lemiare and Robinet-Levy, 1976; Livadas et al, 1979). In total, six studies found a significant relationship between adult HBV infection and child sex ratios (see Table 1). More recently, Oster (2005) explored this relationship using population-level data

on hepatitis infection rates. She demonstrates that sex ratios are significantly higher in countries with higher HBV infection rates. Further, sex ratios appear to fall in response to HBV vaccination campaigns in Alaska and Taiwan. In general, both the original evidence and the new evidence here suggest large effects of HBV infection on sex ratios: parents who are carriers of the virus have roughly 1.5 boys for each girl, versus around 1.05 in the general population.

Lin and Luoh (2006) present new evidence from three million births in Taiwan, which show that maternal HBV infection has only a very small effect on child sex ratios. In their data, women who are infected with HBV have roughly 1.052 boys for every girl. This effect is statistically significant, not surprising given the large sample, but it is *much* smaller than the effect suggested by the original micro-level evidence and data analyzed in Oster (2005). Lin and Luoh (2006) conclude that HBV infection plays only an extremely limited role in determining offspring sex ratios across populations. However, both strands of the literature could be consistent with the argument that paternal, not maternal, infection is correlated with higher offspring sex ratios.

In this paper, we present three pieces of data and argue that there does appear to be evidence that paternal infection is driving the HBV effect. First, we return to two of the original datasets – from Plati, Greece and the Philippines. Both of these datasets have information on child gender and on paternal and maternal HBV infection. In regressions with controls for mother’s and father’s age, child age and birth order, as well as measures of socioeconomic status, we find that paternal HBV infection significantly increases offspring sex ratios, but maternal infection does not.

In addition to this individual-level analysis, we also present evidence from Taiwanese population-level data. In this case, we have data on sex ratio of births by maternal and paternal age cohort. By matching these data to HBV infection levels by age cohort over time, we are able to estimate the effect of paternal and maternal HBV infection rates separately. Again, we find that higher levels of infection among fathers increase sex ratios, but higher levels among mothers do not.

Finally, we use the IPUMS Census data from the United States to explore whether high sex ratios among children born to Chinese immigrants are driven by male or female

immigrants. We find evidence that children born to fathers who immigrate from China are more likely to be male, but this is not true for children born to mothers who immigrate. This also suggests that paternal infection is the driver of the hepatitis B effect.

2 Plati, Greece and Philippines Data

Two of the original studies about the effect of HBV on sex ratio were run in Plati, Greece and in the Philippines. Other authors (Chahnazarian et al, 1988) have argued that these studies are the two highest-quality from the set shown in Table 1. Both studies are organized around families. For each individual in the family, data were recorded on their position in the family (father, mother, child), their gender and their age. For children, birth order was also recorded. And, importantly, adult HBV infection status is included in the data for both men and women.

In both studies, data were collected that provides some information on socioeconomic status. This is useful as a control, since it is possible that HBV infection is correlated with socioeconomic status and if socioeconomic status is also correlated with child gender, this could drive the apparent connection between HBV and offspring sex ratio. In Plati, the individual town of residence is recorded. In the Philippines, parental occupation is given. Although neither of these is a perfect measure of status, it is likely that both measures are correlated with income.

To analyze the effect of parental HBV status on child gender in a regression format, we run the four regressions below.

$$\text{Male} = \beta(\text{Either Parent HBV } +) + \delta_1 \text{Mom Age} + \delta_2 \text{Dad Age} + \delta_3 \text{birth order} + \Phi + \Theta + \epsilon$$

$$\text{Male} = \beta(\text{Dad HBV } +) + \delta_1 \text{Mom Age} + \delta_2 \text{Dad Age} + \delta_3 \text{birth order} + \Phi + \Theta + \epsilon$$

$$\text{Male} = \beta(\text{Mom HBV } +) + \delta_1 \text{Mom Age} + \delta_2 \text{Dad Age} + \delta_3 \text{birth order} + \Phi + \Theta + \epsilon$$

$$\text{Male} = \beta_1(\text{Dad HBV } +) + \beta_2(\text{Mom HBV } +) + \delta_1 \text{Mom Age} + \delta_2 \text{Dad Age} + \delta_3 \text{birth order} + \Phi + \Theta + \epsilon$$

All four regressions include controls for maternal age, paternal age and birth order. In addition, all include a full set of fixed effects for child age (Φ) and socioeconomic status

group (Θ). In all cases, we limit the regressions to children under 20. There are two reasons for this. First, although we are interested in estimating the relationship between parental HBV status at the child's birth and child gender, we only observe parental HBV status during the survey. For children, parental HBV status at the survey will likely be highly correlated with parental HBV status at the child's birth. However, for older individuals, current parental HBV status may not reflect the parent's HBV status at the child's birth. Limiting to children under 20 also avoids issues of out-migration, which may well be correlated with gender.

The results from the four regressions detailed above are shown in Table 2. Column 1 shows the effect of either parent being infected, which is positive and significant. This result is consistent with the initial published results from these data – the chance of a child being male is higher for families in which either parent tests positive for HBV. Comparing Columns 2 and 3, however, indicates that this effect seems to be driven by paternal HBV infection. Children born to fathers with HBV infection are much more likely to be male (Column 2) but the effect of maternal infection is much smaller and not significant (Column 3). Further, Column 4 suggests that any effect for maternal HBV status seems to be driven by the correlation between maternal and paternal infection. When both variables are included, the effect of paternal infection remains strong and significant while the effect of maternal infection is extremely close to zero.

3 Population-Level Data from Taiwan

The above analysis suggests that paternal infection seems to be driving the effect of HBV in at least two of the original studies of the effect of the virus on offspring sex ratios. However, the new evidence on the lack of a maternal effect comes from Taiwan. This leaves open the possibility that the HBV effect operates differently in different places, for example due to differences in viral clade. It is therefore valuable to test directly for these effects in Taiwanese data.

Oster (2005) takes advantage of a widespread vaccination campaign in Taiwan beginning in 1984 to look for an effect of changes in levels of HBV infection on sex ratios by birth cohort. We will be able to use the same methodology here to separate the effect of

maternal and paternal HBV prevalence.

More specifically, we use yearly data from the Taiwanese Demographic Factbook (Republic of China, 1979-2002) on the sex ratio of births by maternal and paternal age cohort (15-20, 20-24, 25-29 and 30-34) and year (1979-2002). Using data on HBV prevalence by age from testing done in primary school (Hsu et al, 1999; Ni et al, 2001; Lin et al, 2003) we construct expected HBV prevalence by age cohort. For example, HBV prevalence for 15-year-olds in 2000 is the infection rate among primary school children tested in the early 1990s who were born in 1985. We use these data to construct a dataset of births and HBV prevalence by parental age cohort. The unit of observation is the year-maternal age cohort-paternal age cohort (for example, an observation would be births to fathers aged 20-24 who had children with mothers aged 15-20 in 2000). In cases where the births are to mothers and fathers in the same age cohort, maternal and paternal prevalence will be the same. In order to separately identify effects of mothers and fathers infection, we are relying on births to parents in different age cohorts, so their probability of infection differs.

We run three regressions. The first considers only paternal infection rates, the second only maternal infection rates and the third includes both together. In all cases, regressions include fixed effects for parental age cohort and for year. All regressions are frequency weighted by the number of births in the cell since some age combinations have very few births. Regressions are clustered by year.

Columns 1 and 2 of Table 3 show these regressions for paternal and maternal infection, respectively. For fathers, the coefficient on cohort HBV infection is positive and strongly significant. For mothers the coefficient is much smaller and not significant. Column 3 of Table 3 considers the effect of maternal and paternal infection jointly. The regression here confirms the results from Columns 1 and 2: the coefficient on paternal prevalence is significant, and of a similar magnitude to Column 1, but the coefficient on maternal prevalence is extremely close to zero. The evidence here is consistent with the evidence from the individual level data discussed above. Further, it is largely consistent with the findings from Taiwan presented in Lin and Luoh (2006).

4 IPUMS Data on Births to Chinese Immigrants

Oster (2005) shows evidence, based on the IPUMS dataset, that the sex ratio of children born in the United States to parents who immigrated from China is high, similar to the sex ratio seen in China. She argues that since these births take place in the United States they are less subject to mis-reporting and sex-selective abortion. However, since the parents were born in China, their hepatitis infection rates should reflect the average levels there. Oster (2005) focuses on births to families in which both parents are immigrants. However, it is also possible to separate male and female infection by looking at births to Chinese-born men and women separately.

As in Oster (2005), this analysis relies on the IPUMS census data (the 1980, 1990 and 2000 censuses), and focuses on sex ratios of children under 20 who were born in the United States. Table 4 shows the sex ratios of children in this data. The first row shows the sex ratio of children born in the United States to Chinese-born men with any woman; this sex ratio is high, around 1.090 boys for each girl. The second row shows the sex ratio of children born to Chinese-born women, with any man. This ratio is also high, around 1.088. However, since Chinese immigrants are likely to be married to each other, this does not effectively separate the effect of maternal and paternal infection.

The third and fourth rows of Table 4 therefore focus on Chinese-born men married to American-born women, and Chinese-born women married to American-born men. In this case, although the sample sizes are smaller, we do see strong evidence that the effect is being driven by Chinese men. The sex ratio in the third row – where the father is Chinese – is around 1.10. However, in the fourth row, where only the mother is Chinese, it is close to normal, around 1.04.

5 Conclusion

The results presented in this paper suggest a possible reconciliation between two strands of literature on the relationship between carrier rates of hepatitis B and offspring sex ratios. The first strand uses population-level data, and argues that sex ratios are larger in areas with

more HBV carriers. The second strand uses a large dataset on births in Taiwan and presents evidence that maternal HBV infection is associated only with a very small increase in the offspring sex ratio.

The analysis here uses both the original individual-level data and population-level data from Taiwan and argues that there are higher sex ratios among the offspring of *men* with HBV infection, but not among women with the infection. Further, there is evidence of higher offspring sex ratios among men born in China living in the United States, but not among women born in China living in the United States.

In principle, this new evidence could reconcile both parts of the literature. The one remaining issue is whether the magnitude of the effect in the Lin and Luoh (2006) work is consistent with the correlation between male and female infection rates. In particular, the effect of maternal carrier status that Lin and Luoh (2006) estimate is extremely small. If there was a high correlation in HBV carrier status between spouses, we would expect a large effect of maternal carrier status even if the driver was paternal status.

In fact, correlation in HBV carrier state between spouses is positive, but relatively low. This is not surprising. Hepatitis B is a sexually transmitted infection, but *carrier* status is relatively unlikely to be sexually transmitted. Since the chance of becoming a carrier is relatively unlikely (only about 2%) for those infected in adulthood (World Health Organization, 2001), even if one spouse transmits the virus to the other, they are unlikely to both become carriers. Empirically, the correlations vary across studies. In the data from Plati and the Philippines used here, the correlations are reasonably high (around .1), perhaps reflecting clusters of the virus within areas. In contrast, in a sample of Afghan refugees, Quddus et al (2006) find no significant correlation in HBV carrier status between husband and wife. If paternal infection increases the share of boys born in the family by about 0.09, as suggested by much of the original individual-level data, and the correlation in spouse status is between 0 and 0.1, we would expect estimates of the effect for maternal infection to vary between 0 and 0.009. The estimates presented in Lin and Luoh (2006) are 0.0025, certainly within this window.

It is clear that more needs to be done to establish the connection discussed here. A large scale analysis of men with HBV infection, similar to that done with women in Taiwan,

would provide additional insight into whether paternal HBV infection is linked with child gender. Further, some understanding of the mechanism driving this result is important. Nevertheless, these results, along with the Lin and Luoh (2006) analysis, may provide important insight into exactly *why* we see the sex ratio-HBV infection connection.

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Table 1. *Offspring Sex Ratio by Parental HBV Infection, Original Data*

Location	HBV Status	Sons	Daughters	Sex Ratio
Greenland	Positive	64	60	1.07
Greenland	Negative	174	194	0.90
Kar Kar Island	Positive	63	54	1.17
Kar Kar Island	Negative	163	206	0.79
Greece 1	Positive	90	51	1.77
Greece 1	Negative	287	255	1.13
Philippines	Positive	66	41	1.61
Philippines	Negative	304	301	1.01
Greece 2	Positive	52	30	1.73
Greece 2	Negative	1006	955	1.05
France	Positive	20	12	1.66
France	Negative	149	122	1.22

Notes: This table shows sex ratios among the children of carrier and non-carrier parents in four regions. Data were collected by testing married women and, in all cases except for Greenland, their husbands for HBV. Detailed reproductive histories were also collected. The table represents all births to women in these samples, with generally more than one birth to each woman. The last two studies (Greece 2 and France) were designed specifically to test the hypothesis that HBV affects offspring sex ratio, and were run after the original theory was published. The citations for each study are as follows: Greenland – Drew, Blumberg and Robert-Lamblin (1986); Kar Kar Island – Drew et al (1982); Greece 1 – Hesser, Economidou and Blumberg (1975); Philippines – Chahnazarian et al (1988); Greece 2 – Lividas et al (1979); France – Cazal, Lemaire and Robinet-Levy (1976).

Table 2. Parental HBV Infection and Child Gender in Plati, Greece and the Philippines

	<i>Dependent Variable: Child is Male</i>			
	(1)	(2)	(3)	(4)
Explanatory Variables:				
Either Parent HBV +	.145*** (2.90)			
Father HBV +		.1562*** (2.87)		.1541*** (2.69)
Mother HBV +			.0474 (.70)	.0078 (.10)
CONTROLS				
Maternal Age	YES	YES	YES	YES
Paternal Age	YES	YES	YES	YES
Birth Order	YES	YES	YES	YES
Child age Dummies	YES	YES	YES	YES
Town or Occupation Group	YES	YES	YES	YES
Number of Observations	767	767	764	764
R ²	.08	.07	.07	.08

^a t-statistics in parenthesis

* significant at 10%; ** significant at 5%; *** significant at 1%

Notes: This table shows the relationship between parental HBV infection and child gender, using data from two family-level studies in Plati, Greece and the Philippines. “Either parent HBV +” means that either the mother or father in the family recorded being HBV +, regardless of the status of the other parent. For the Plati observations, fixed effects for town are included as the measure of socioeconomic status. For the data from the Philippines, occupation codes are included as the measure of status.

Table 3. Taiwanese Birth Data: Paternal Versus Maternal Infection

<i>Dependent Variable: Percent Male in Birth Cohort</i>			
	(1)	(2)	(3)
Explanatory Variables:			
Paternal Cohort HBV Prev (0-1)	.091*** (2.58)		.087** (2.09)
Maternal Cohort HBV Prev (0-1)		.022 (1.01)	-.001 (-.05)
Paternal Age Cohort FE	YES	NO	YES
Maternal Age Cohort FE	NO	YES	YES
Year FE	YES	YES	YES
Number of Observations	5,789,851	5,789,851	5,789,851
R ²	.63	.63	.63

^a t-statistics in parenthesis

* significant at 10%; ** significant at 5%; *** significant at 1%

Notes: This table shows the relationship between sex ratio by parental age cohort and HBV infection in that age cohort. The unit of observation is a paternal age cohort-maternal age cohort-year. The dependent variable is the share of boys born to that paternal age-maternal age-year group (for example, share boys born to fathers aged 20-24 and mothers aged 15-20 in 1980). Prevalence by parental age cohort is constructed based on HBV testing for school-age children in the early 1990s. These data provide estimated prevalence by birth cohort, which is used to construct the dataset. All regressions are frequency weighted by the number of births in the cell, and clustered by year.

Table 4. Offspring Sex Ratios to Chinese Immigrants: IPUMS Data

Parental Group	# Boys	# Girls	Sex Ratio
Chinese-born men, any woman	9,362	8,588	1.090
Chinese-born women, any man	9,068	8,335	1.088
Chinese-born men, American-born women	768	689	1.114
Chinese-born women, American-born men	818	783	1.044

Notes: This table shows offspring sex ratios among children under 20 in the 1980, 1990 and 2000 Census IPUMS. The parental group is shown in Column 1. “Chinese-born men, American-born women”, for example, implies that these are children born to men who immigrated from China who are having children with women born in the United States.