Are fertility estimates from retrospective data biased by maternal mortality? An assessment based on parametric models of family size distribution*

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Introduction

Conclusions based on data from retrospective maternity histories from women are cautiously accepted because they are subject to several sources of error. Older women tend to report lower fertility than younger women leading many demographers to believe that the data are biased by factors such as memory lapse or omission of deceased offspring (Brass, 1968). Some studies comparing fertility recorded from birth registries and retrospective fertility histories of women have confirmed the underreporting problem (Blacker and Brass, 1979) while others have not (Brittain, 1991). Recovery from African infertility (Caldwell and Caldwell, 1983; Frank, 1983; Pennington and Harpending, 1991; Pennington and Harpending, 1992) may also be a reason older women on that continent report lower fertility than younger women.

Even if women accurately report their births, there is concern that estimates based on these kinds of data may be biased because women who have survived to report their fertility may have maternity histories that do not reflect the true experience of their cohort. If maternity is an important

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cause of death, the group of women who survive to report their fertility will be a less fertile subset of their original cohort. In much of the developing world, the majority of deaths to women during pregnancies are due to maternal causes (Royston and Lopez, 1987). For this reason, maternal mortality may lead to significantly underestimated fertility rates.

In this paper, we measure the magnitude of bias that may result from selective maternal mortality using probability distributions that have been used to approximate the distribution of family sizes in human populations. Our analysis indicates that the selectivity bias is small and for even moderately large sample sizes is less important than other sources of error.

Today, many demographers are less concerned with bias caused by maternal mortality than in the past because they believe that improving health care has rendered the incidence of maternal mortality negligibly low. However, areas where demographers must rely on retrospective fertility histories to infer trends in vital rates tend to be those where most women are still far removed from health care services. Assessing the level of potential bias from maternal mortality may also improve the reliability of older data sets from which historical trends in demographic rates are inferred.

Bias from maternal mortality

We assume that in the absence of any maternal mortality the distribution of completed family size (CFS) among post-menopausal women would conform to one of several parametric distributions. We will discuss the cases of the Poisson, the negative binomial, and the modified geometric distributions. We choose the Poisson and negative binomial distributions because they have properties suitable for models of completed fertility that have been proposed by other researchers (Brass, 1958; Cavalli-Sforza and Bodmer, 1971; Howell, 1979; Golbeck, 1981). The geometric distribution is appropriate for modeling family size distributions in which the parity progression ratios are constant. We develop a modified geometric distribution with parameters related to the primary and secondary sterility rates in the population.

We also assume that the rate of maternal mortality is a constant μ per confinement. That is, a fraction $1-\mu$ of women survive the birth of each child while a fraction μ die. Since the rate is constant per confinement

¹Women are considered to have primary sterility if they never have live birth.

²Women are considered to have secondary sterility if they have had at least one live birth but have ceased reproduction because they have become infertile

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the fraction of a cohort who survive the birth of the k'th child is $(1-\mu)^k$. If, for example, a number n_5 of a cohort of women entering childbearing would have exactly five births if there were no maternal mortality, in the presence of maternal mortality we would only observe $n_5(1-\mu)^5$ at the end of childbearing. If maternal mortality were as high as five per cent per confinement ($\mu = .05$) and if 100 women would have no children at the end of childbearing while another 100 would have five births in the absence of death associated with childbirth, we would in fact observe 100 childless women when we interviewed in the population but only 77 women $(100 \times .95^5)$ who had had five births. At the end of childbearing, the proportion of surviving women with low fertility is higher than it would be in the absence of maternal mortality, while the proportion of women with high fertility is underestimated. This process is illustrated by Figure 1. The No maternal mortality curve shows the frequencies of family sizes that one would observe among postreproductive women if fertility conformed to a (hypothetical) negative binomial distribution. The Maternal mortality curves shows how this distribution would be modified by a high maternal mortality rate.

Empirical evidence suggests that the true pattern of maternal mortality is J-shaped, being highest at the first and also at the higher ordered parities (Heady and Daly, 1955; Yerushalmy et al., 1956; Nortman, 1974). Women with high parity may also have reduced life spans beyond the reproductive years, though nulliparous women in developing countries typically have reduced life spans (United Nations, 1983). Although these factors imply an accelerating rate of maternal mortality, only a proportion of women who reproduce have subsequent births so that the additional bias introduced may not be large. Our analysis below also shows that the effect of even very high rates of maternal mortality are very small so that our assumption of a constant maternal mortality rate is probably not a limitation of our model.

Poisson distribution

A simple model of the distribution of CFS is the Poisson distribution, which would arise if the hazard of giving birth were constant during the childbearing years. In this case the distribution of CFS p_x would follow

$$p_x = \frac{e^{-\lambda}\lambda^x}{x!} \qquad x = 0, 1, \dots$$

in the absence of maternal mortality. The mean and the variance of this distribution are both equal to λ .

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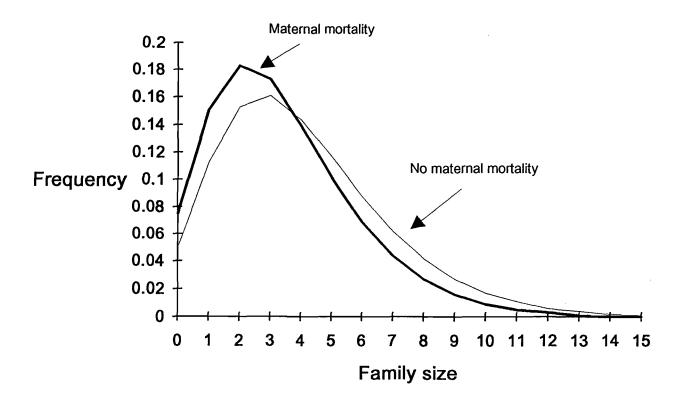


Figure 1: Effect of maternal mortality on birth distributions

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With maternal deaths occurring at a rate of μ per confinement, the frequency of post-menopausal women who had had x births would be proportional to

$$f_x = \frac{e^{-\lambda} \lambda^x}{x!} (1 - \mu)^x \qquad x = 0, 1, \dots$$

In order to derive the probability distribution of observed CFS each f_x must be divided by the sum of the all the f's. Since

$$\sum_{x=0}^{\infty} \frac{e^{-\lambda} \lambda^x}{x!} (1 - \mu)^x = e^{-\lambda \mu}$$

the probability distribution of CFS as modified by the occurrence of maternal mortality is

$$p_x = \frac{e^{-\lambda(1-\mu)}[\lambda(1-\mu)]^x}{x!}$$
 $x = 0, 1, \dots$

This is again a Poisson distribution with parameter $\lambda(1-\mu)$. The CFS observed at the end of childbearing would be reduced by maternal mortality by $1-\mu$, that is by one less the proportion of women who die per confinement. A high rate of maternal mortality would be about 1% per confinement (Royston and Lopez, 1987), leading to a change in the estimate of CFS of 1%. This is such a small change that it is of a lower order of magnitude than sampling errors.

Pennington and Harpending (1991) found that postreproductive Herero women in southern Africa had an average of 3.47 births per woman (n = 239, $\hat{s}^2 = 10.8$). If the maternal mortality rate of their cohort was $\mu = 0.01$, then under this Poisson model the true CFS was 3.51. This change is much smaller than their sampling error and within rounding error.

Negative binomial distribution

Several processes might lead to a negative binomial distribution of CFS. For example if births occurred to each woman according to a Poisson process but if the intensity of the process, i.e. the intrinsic fecundity of each woman, varied according to a gamma distribution, the population distribution would be negative binomial. The distribution is

$$p_x = {x+r-1 \choose x} p^r q^x \qquad x = 0, 1, \dots, \ r > 0, \ 0 \le p \le 1, \ q = 1-p$$

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The mean of this distribution is rq/p and the variance is rq/p^2 . If maternal mortality occurs at rate μ per confinement, the distribution of completed family size observed at the end of reproduction becomes

$$p_x = {x+r-1 \choose x} (p+q\mu)^r (q-q\mu)^x \qquad x=0,1,\ldots$$

This is again a negative binomial distribution with the same parameter r as the original but with the second parameter changed to $p + q\mu$. The mean is now $r(q - q\mu)/(p + q\mu)$. The effect of maternal mortality is to change the completed family size observed at the end of reproduction by the rate of maternal mortality, just as in the case of the Poisson.

Geometric and modified geometric

A geometric distribution of family size would result if a cohort of women experienced a constant parity progression ratio (PPR). Each PPR is the proportion of women who, having had an n'th birth, have an n+1'th birth, n=0... The completed family size distribution of Herero women past menopause described by Pennington and Harpending (1991) is fit remarkably well by a model that specifies that the parity progression ratio from 0 to 1 births is 0.858 and that each later parity progression ratio is .753. This model leads to a modified geometric distribution of completed family size since the range of x includes 0, which we specify separately. If π_0 is the primary sterility rate (in our case, .142) and if π is the probability of terminating reproduction after any order birth, the resulting distribution of completed family size is

$$p_0 = \pi_0$$

 $p_x = (1 - \pi_0)(1 - \pi)^{i-1}\pi$ $x = 1, 2, ...$

The mean of this distribution is $(1 - \pi_0)/\pi$ which, using our simple representation of Herero parity progression ratios, is .858/.247 = 3.47. The variance of the distribution is $(1 - \pi_0)(1 - \pi + \pi_0)/\pi^2$.

If this distribution is modified by allowing a constant probability μ of maternal mortality per birth the new distribution is again a modified geometric

$$p_0 = \pi'_0$$

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$$p_x = (1 - \pi'_0)(1 - \pi')^{i-1}\pi' \qquad x = 1, 2, \dots$$
 with
$$\pi'_0 = \frac{\pi_0(\pi + (1 - \pi)\mu)}{\mu\pi_0 + \pi(1 - \mu)}$$

$$\pi' = \pi + (1 - \pi)\mu$$

Using our estimates $\pi_0 = .142$ and $\pi = .247$ that fit the Herero data, we can compute that a maternal mortality rate of 1% per birth would reduce the observed CFS from 3.47 to 3.35, i.e. a change of about 3.5%. Again, this is much smaller than the sampling error.

Discussion

Our examination of the effects of maternal mortality on parametric models of family size distributions indicate that maternal mortality is probably not an important source of bias in retrospective fertility histories. The negative binomial and the Poisson distributions are often used to model family size distributions. Our analysis shows those birth distributions that follow these models underestimate the true CFSs by a constant rate that is equal to the maternal mortality rate. This means that populations that differ in their levels of fertility but that have equal maternal mortality rates will be equally affected by maternal mortality. That is, estimates of CFSs in populations with high fertility are not more biased by maternal mortality than estimates of CFSs in populations with low fertility. The change caused by even a high rate of maternal mortality (i.e. 1 per 100 confinements) is smaller than sampling errors of even moderately large studies and is within rounding error.

We also examined how maternal mortality would reduce the CFSs of populations whose birth distributions approximated a geometric distribution and found that they were more greatly affected by maternal mortality. However, the effect of maternal mortality is still small. The magnitude of the bias depends on the secondary sterility rate of a population. Holding the maternal mortality rate and the primary sterility rate constant, populations with high secondary sterility rates are less biased by maternal mortality than populations with low secondary sterility rates as fewer and fewer women are at risk of maternal mortality following every birth. All else being equal, varying the primary sterility rate has very little influence on the magnitude of bias caused by maternal mortality. This means that

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populations marked by pathologically low fertility (i.e. the Herero and other groups in the African infertility belt) are less affected by maternal mortality than populations with normal levels of fertility.

We are not aware of previous studies in which the geometric distribution has been used to model family size distributions. However, our modified geometric distribution provides an excellent fit to the Herero data in Figure 2 $(X_{11}^2 = 6.6, p > .10)$. The data can also be adequately fit to a negative binomial $(X_{11}^2 = 9.8, p > .10)$. The fit to the Poisson was not at all satisfactory $(X_{12}^2 = 237.6, p < .0001)$. Although we have yet to test the geometric on other fertility data, it will probably be a useful method for modeling fertility in other populations where desired fertility is high but dampened by high secondary sterility rates. The Herero, in common with a number of peoples of central Africa, suffered from pathological infertility earlier in this century⁴ (Pennington and Harpending, 1991). As a result, many women ceased childbearing early in their reproductive spans.

Conclusion

These models show that the effect of even a high rate of maternal mortality does not lead to a serious bias when we estimate CFS from retrospective maternity histories of women past childbearing age. We have only solved several special cases here, and it probably possible to construct feasible models where the bias is larger. However, our choice of parametric models was guided by empirical evidence suggesting that they approximate the distribution of births in human populations.

³We estimated r and p using the method of moments so that r = 1.64 and p = .321

⁴Herero fertility probably increased when antibiotics became available in northwestern Botswana in the 1950s. In other parts of Africa, fertility increased as a secondary effect of widespread anti-Yaws campaigns in which millions of people were injected with large doses of long-acting penicillin (Guthe, 1962; Guthe et al., 1972).

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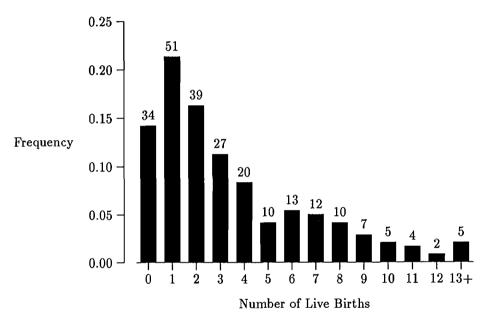


Figure 2: The distribution of family sizes among postreproductive Herero women

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