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ESTIMATING THE EFFECT OF CHILD MORTALITY ON THE NUMBER OF BIRTHS

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ABSTRACT

This article rigorously derives the properties of the regression of births on child deaths. It is shown how the raw regression coefficient may be corrected for the effects of fertility on mortality so that the rate at which dead children are replaced may be estimated. The method is applied to data from Colombia. It is found that the mortality rate differs across individuals and is correlated with fertility. Such conditions vitiate the use of birth intervals and parity progression ratios yet can be dealt with using the new method. On average each death produces 0.2 new births as a direct result of the death. Fertility hoarding may raise the total fertility response to roughly one-half birth per death.

I. Introduction

When economists examine the subject of human fertility they bring with them the notion that couples are rational. This notion of rationality leads to the conclusion that the number of children borne by women reflects a decision which has been made regarding the desired number of births. Child mortality is quite common in less developed countries. Because it seems more reasonable to suppose that parents' desires are formulated in terms of live offspring, if we wish to detect the effects of rationality on fertility we should focus our attention upon the number of surviving children. While rationality is not the only force at work in man, when we examine the behavior of many individuals it becomes more easily detected. In the words of William James:

> . . . weak as reason is, it has the unique advantage over its antagonists that its activity never lets up and that it presses always in one direction, while men's prejudices vary, their passions ebb and flow, and their excitements are intermittent.

If couples try to attain some number of surviving children, then we expect the death of a child to lead its parents to try to offset this disturbance to their plans. This conjecture is known as the replacement hypothesis. In its strongest form this hypothesis predicts that among otherwise identical couples, those suffering one more child death will tend to have one more birth.

The extent of replacement is an important issue. If there is no replacement, measures taken to reduce child mortality will increase population growth. If replacement is complete, such measures will not affect population. Clearly it is important to know the rate at which child deaths (or prevention of child deaths) produce more (fewer) births when measures taken to influence development of a country have an effect upon mortality.

The occurrence of a death or the anticipation that such a death may occur may also enter into the determination of the desired number of surviving children. A dynamic strategy with respect to fertility has been considered by Ben-Porath and Welch (1972). The occurrence of a child death may lead the couple to revise its subjective belief as to the likelihood of future child deaths. It is possible for the death of a child to reduce subsequent fertility if desired fertility falls substantially as the subjective probability of death in infancy rises, and if the occurrence of a child death greatly increases the estimated infant mortality rate which a couple believes it faces.[1]

If parents choose to act to offset the effects of higher child mortality, more than one aspect of their behavior may be modified. Parents in a high child mortality environment will require more births to achieve the same number of survivors. This may lead to earlier marriage in order to allow the couple more time to achieve its desired fertility. Schultz [ ] has observed a tendency in Taiwan for higher child mortality rates to be associated with earlier marriage which may be taken as indirect evidence of replacement type behavior. Similarly, high rates of child mortality may result in deaths when the couple is older and less able to adjust fertility subsequent to deaths. In response to a high rate of mortality, the couple may produce additional children in anticipation of some deaths. If such hoarding is the only response to higher mortality rates, there will be no direct connection between an additional child death in the family and additional fertility even though replacement-type behavior exists. While pure hoarding may be a possible response to mortality, even very modest direct replacement behavior can substantially improve a couple's ability

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[1]This presumes mortality rates differ across individuals, and that these differences cannot be explained solely by differences in observable factors such as age, education, income and the like. Below we will see that after controlling for regional and urban versus rural (but not other factors) mortality rates differ quite substantially across individuals. Based upon this variation a couple almost certainly does not know the mortality rate it faces.

to approach or achieve its desired fertility. For example, suppose the mortality rate is 15% with all deaths occurring in infancy. If a couple desires six surviving children, this will require on average seven births. If the couple follows a hoarding strategy of producing seven children and hoping for the best, it will have six surviving children 30% of the time. If the couple is able to have an eighth contingency birth depending upon realized mortality, it will achieve its goal 47% of the time and will be one child closer to its goal an additional 15% of the time. Since the expected number of deaths is one, even this limited capability for replacement can offset much of the uncertainty of child mortality. If replacement behavior exists, we should expect to detect some direct effect of deaths on fertility beyond indirect effects such as child hoarding or earlier marriage.

Unfortunately, it is easy to confuse the general binomial association of more deaths with more births with a behavioral tendency of couples to have more children in order to replace children who may have died. This complication is widely recognized; it is rare to see a published regression of children ever born on child deaths. The shortcomings of such an approach have been noted by Williams (1977) and Brass and Barrett (1978), although recognition of the problem predates these articles. The methodological response to this dilemma has been to use a variety of statistical specifications using mortality rates, interval analysis and parity progression ratios as well as simulation experiments. Aside from the work done by Williams which cast doubt on the use of mortality rate regressions, there has been little analytical study of the statistical properties of these methods. In this paper we conduct a rigorous statistical analysis of the properties of the regression of children ever born (n) on child deaths (d). By determining the bias in such ordinary least squares (OLS) regressions, it is possible to separate the behavioral signal from the statistical noise. The advantage of this approach is that it involves the direct estimation of the relation under study, namely the effect of mortality on fertility rather than attempting to estimate the relation indirectly via mortality rates or birth intervals. These other techniques may still be useful tools provided certain conditions are met but the method described here will be shown to be more flexible. We will develop this method using a variety of assumptions about the true nature of child mortality and its relation to fertility. The basic question to be answered is what is the effect of an additional child death on fertility?

Our starting point in section II will be the simple case where p, the probability of a death, is constant. We will solve for the bias in the OLS estimate of r, and then consider the bias in the estimated replacement rate when the mortality rate d_i/n_i is substituted for d_i in (1.1). Next, we will consider the case where p is itself a random variable and will examine how this affects the bias of OLS.

In section III we will apply our formulae to Colombian fertility data to show how the various corrections which may be applied to OLS alter our inferences about replacement. Section IV summarizes the paper.

II. Measurement Error and Bias in the Replacement Rate

If the mortality rate were 10% and each woman had many children, each additional child death for a woman would be associated with roughly ten additional children ever born. Thus the number of child deaths may be viewed as actual fertility measured with error where the measurement error arises because deaths arise from a Bernoulli process with $p=0.10$ where p is the probability a child will die. As is well known, the use of a regressor subject to measurement error biases least squares coefficients towards zero. This means that because fractional deaths cannot occur, when we regress children ever born on deaths in our simple example we obtain a coefficient which is less than ten. In fact, we shall see that under conditions which exist for a variety of societies, this measurement error alone will produce a coefficient on deaths which is approximately one, leading to a fallacious conclusion that replacement is complete.

We will assume throughout this paper that the true relationship connecting fertility and mortality is:

$$n_i = \bar{n} + r(d_i - \bar{d}) + u_i \tag{1.1}$$

where n_i is children ever born, d_i is the number of child deaths and u_i is a random error, $\bar{n}$ and $\bar{d}$ are the sample means of n_i and d_i respectively. Cross-sectional data will be used so the $\underline{i}$ indexes couples.

A more general model would allow for the presence of fertility hoarding as well as direct replacement of child deaths, that is

$$n_i = \bar{n} + r(d_i - \bar{d}) + \gamma(p_i - \bar{p}) + v_i$$

where p_i is the probability of child death for the i^{th} couple and $\bar{p}$ is the mean of mortality ratio in the sample. If hoarding is present we should observe $h>0$, that is, if each couple knows p_i those couples with larger values of p_i may plan on a higher number of births from the outset in anticipation of more child mortality. There may also be hoarding in response to the general level of mortality ($\bar{p}$), however we will not be able to detect such behavior using cross sectional data. The use of data from different populations may indicate hoarding which varies according to the mean mortality rate, but as we look across populations (either across space or time) there may be factors which influence both the mortality rate and desired fertility. We will content ourselves with the estimation of (1.1) in this section. Later in section III we will discuss the estimation of the hoarding response.

In this section we derive the bias in OLS for successively more complex assumptions about the structure of mortality. In section A we assume the probability of a death is constant for all women and derive the limiting value or probability limit (plim) of the least squares coefficient. It is assumed that the number of child deaths is the only regressor. In section B we investigate whether the use of the observed mortality rate d_i/n_i offers a solution to the least squares bias. It will be shown that while replacing d_i with d_i/n_i as a regressor does not provide much help, d_i/n_i can be used as an excellent instrumental variable which avoids the least squares bias provided certain conditions hold. Section C drops the assumption that p is fixed and shows how random p affects the bias and describes how the correlation between p and n can be determined. The case of p random and correlated with n is the most general stochastic specification considered. The effect of additional regressors on the analysis is briefly described in section D.

A. Least Squares with a Fixed Mortality Rate

We will start by assuming p_i has the same value for all women. As a result, d_i is a random variable which, for given n_i , follows a binomial distribution. We may write

$$d_i = pn_i + \varepsilon_i$$

where pn_i is the mean of d_i and, given n_i , ε_i follows a binomial distribution but with mean zero and variance $n_i p(1-p)$.

$$d_i = p\bar{n} + \frac{pu_i}{1-pr} + \frac{\varepsilon_i}{1-pr}$$

Since d_i is a stochastic regressor which is correlated with u_i , the least squares estimate of r converges in probability to

$$\text{plim}\,(\hat{r}) = r + \text{cov}\,(d_i, u_i)/\ \text{var}\,(d_i) \quad .$$

For given n_i, d_i is a simple binomial variable, but n_i is itself random, so from lemma 1 in the appendix

$$\mathrm{var}(d_i) = \bar{n}p(1-p) + p^2\mathrm{var}(n) \tag{2.1}$$

so that

$$\mathrm{plim}\ (\hat{r}) = r + \frac{1}{(1-pr)\left[p+(1-p)\frac{\bar{n}}{\mathrm{var}(n)}\right]} \tag{2.2}$$

Var(n) represents the variance in children ever born, so if $r=0$ and $\bar{n} = \mathrm{var}(n)$ then plim $(\hat{r}) = 1$. Table 1 gives scattered findings of $\bar{n}$ and var(n) and shows plim $(\hat{r})$ under the assumption that $r=0$ and $p=0.10$. As p falls, plim $(\hat{r})$ moves slightly away from one. Note we have used var(n) = var(u). This is approximately correct since $\grave{r}$ is likely less than one and $\mathrm{var}(d_i)$ is small compared to var(n).

If the probability of a child death is in fact a constant, then (2.2) provides a method for estimating the replacement rate which takes the bias into account. Wallace (1979) has independently derived a correction similar to this one. The chief difference is that he uses the probability density function on n_i whereas we simply use the mean and variance. If n_i followed a distribution for which the mean and variance were sufficient statistics the two methods would be using equivalent information and should produce very similar results. We will see this is the case in section III.

Table 1

Comparison of Bias in Estimating Replacement with Least Squares

Country[1]	$\bar{n}$	var (n)	plim ($\hat{r}$)
Israel[2]	2.55	4.71	1.70
USA[3]	2.74	2.83	.897
Colombia[4]	7.36	10.2	1.33
Kenya[5]	6.37	6.51	1.02
Philippines[3]	5.43	7.29	1.30

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[1]All rates are for subpopulations of the various countries. For full particulars each study should be consulted.

[2]Ben-Porath (1973)

[3]Boulier and Rosenzweig

[4]See section III below, rural women 35-39.

[5]Wallace (1979).

B. Mortality Rate Regressions

One method which is often used to avoid the bias implicit in the use of d_i is to use the realized mortality rate d_i/n_i as a regressor and then use the derivative

$$\frac{d(n_i)}{d(d_i)} = \frac{n_i d(n_i)/d(d_i/n_i)}{n_i^2 + d_i d(n_i)/d(d_i/n_i)}$$

and evaluate the derivative at the sample mean which roughly amounts to dividing the coefficient of d_i/n_i by $\bar{n}$. In order to facilitate the analysis, let us use the series expansion for d_i/n_i using $n_i = \bar{n} + u_i$

$$\frac{d_i}{n_i} = [p(\bar{n} + u_i) + \varepsilon_i] \left[\frac{1}{\bar{n}} - \frac{u_i}{\bar{n}^2} + \frac{u_i^2}{\bar{n}^3} - \frac{u_i^3}{\bar{n}^4} + \ldots\right]$$

$$= p + \frac{\varepsilon_i}{\bar{n}} - \frac{u_i \varepsilon_i}{\bar{n}^2} + \frac{u_i^2 \varepsilon_i}{\bar{n}^3} \quad \ldots$$

where ε_i is again the error term in the binomial death model. This expansion is valid only for $u_i < \bar{n}$, which is not teribly restrictive.

The covariance of u_i and d_i/n_i is then

$$E(u_i d_i/n_i) = E\left(pu_i + \frac{u_i \varepsilon_i}{\bar{n}} - \frac{u_i^2 \varepsilon_i}{\bar{n}^2} + \frac{u_i^3 \varepsilon_i}{\bar{n}^3} - \ldots\right).$$

Now

$$E(u_i^s \varepsilon_i) = \int_{u_i} u_i^s \left[\int_{\varepsilon_i} \varepsilon_i f(\varepsilon_i | u_i)\, d\varepsilon_i\right] g(u_i)\, du_i = 0 \qquad (2.3)$$

and the term in the brackets is zero since the conditional distribution of ε_i given u_i is binomial and the mean of this condition expectation is zero. If we estimate

$$n_i = \bar{n} + R(d_i/n_i - \overline{d/n}) + u_i'$$

by OLS, where $\overline{d/n}$ is the sample mean of d_i/n_i, when (1.1) is the correct specification then

$$\text{plim } (\hat{R}) = \alpha^2 \text{plim } [\hat{r}/\hat{B}]$$

where $\hat{B}$ is the coefficient for the regression of d_i/n_i on d_i which is

$$\hat{B} = \frac{\text{cov } (d_i, d_i/n_i)}{\text{var } (d_i)} .$$

and α is the correlation between d_i and d_i/n_i which is typically about 0.85.

Now

$$\text{cov } (d_i , d_i/n_i) = E\left[(pu_i + \varepsilon_i)(d_i/n_i)\right]$$

$$= E\,[p^2 u_i + p\varepsilon_i + pu_i\varepsilon_i/\bar{n} + \varepsilon_i{}^2/\bar{n}$$

$$-pu_i{}^2\varepsilon_i/\bar{n}^2 - u_i\varepsilon_i{}^2/\bar{n}^2 + pu_i{}^3\varepsilon_i/\bar{n}^3 + u_i{}^2\varepsilon_i{}^2/\bar{n}^4 - \ldots]$$

$$= E(\varepsilon_i{}^2/\bar{n}) - E(u_i\varepsilon_i{}^2/\bar{n}^2)$$

where the last equality is an approximation, omitting terms with $\bar{n}^{3}$ or more in the denominator. The other terms have zero expectation from (2.3). These last two expectations are evaluated in lemma 2 and 3 respectively in the appendix. Substituting we find

$$\hat{B} = \frac{(1-p-pr) \ / \ (1-\mathrm{var}\ (n) \ / \ \bar{n}^{2})}{\bar{n}\ (1-p-pr) + p\ \mathrm{Var}\ (n)}$$

which is roughly $1/\bar{n}$ for typical values of p, $\bar{n}$ and Var (n).

The use of d_i/n_i in place of d_i produces, approximately,

$$\mathrm{plim}\ (\hat{R}) = \alpha^2 \bar{n}\ \mathrm{plim}\ (\hat{r})$$

and so dividing $\hat{R}$ by $\alpha^2 n$ leaves us no better off than before even though d_i/n_i is uncorrelated with u_i.

If the true relation connecting fertility and mortality were

$$n_i = \bar{n} + R(d_i/n_i - \overline{d/n}) + u_i'$$

then the rate regression would be correctly specified. The objection to using the observed mortality rate is twofold. First, if d_i rather than d_i/n_i is the correct specification nothing has been gained. Second, the d_i specification is more plausible because it directly models the behavioral issue of the impact of an additional death on fertility. While the rate specification requires the last child to be born before the final response to mortality is made, the d_i specification captures the intuitively pleasing idea of the family following a sequential strategy of adjustment to child mortality.

The primary virtue of d_i/n_i is that it makes an excellent instrumental variable since it is very highly correlated with d_i and is at the same time uncorrelated with u_i , as we have shown. This means an instrumental variables regression is an alternative to the use of (2.2) and will yield consistent estimators so long as p_i is not correlated with u_i . It is this general problem to which we turn next.

C. Random Mortality Rates

The assumption that p_i , the mortality rate, is constant for all women is rather strong. Some heterogeneity is to be expected if only due to physiological factors. Once we allow for random mortality rates we must also consider the possibility that the mortality rate is correlated with fertility. Allowing for such a correlation makes it necessary to estimate this additional parameter. The stochastic structure of the model allows the correlation to be estimated. This correlation may be due to either unobservable random efforts or observable traits which affect both fertility and mortality. Since we are not concerned with the determinants of p_i the source of the correlation is irrelevant to the central issue here.

When p_i is random, our expression for d_i becomes

$$d_i = p_i\bar{n} + \frac{p_i u_i}{1-p_i r} + \frac{\varepsilon_i}{1-pr}$$

and if $p_i r$ is small, this can be simplified to

$$d_i = p_i\bar{n} + p_i u_i + \varepsilon_i$$

Now plim $(\hat{r}) = r + \text{cov}(d_i u_i)/\text{var}(d_i)$ which must be evaluated under the assumption p_i is random with mean $\bar{p}$. Both $\text{var}(d_i)$ and $\text{cov}(u_i d_i)$ involve moments of order greater than two, so plim $(\hat{r})$ depends upon the joint distribution of n_i and p_i except in the special case where they are independent. Now

$$\text{cov}(u_i d_i) = E(p_i u_i \bar{n} + p_i u_i^2 + \varepsilon_i u_i)$$

$$= E(p_i n_i^2 - \bar{n} n_i p_i) \qquad (2.4)$$

where we have used $E(\varepsilon_i u_i) = 0$ from (2.3) and $n_i = \bar{n} + u_i$.

From lemma 4 we have

$$\mathrm{var}(d_i) = E(n_i p_i - n_i p_i^{\,2} + n_i^{\,2} p_i^{\,2}) - [E(n_i p_i)]^2 \qquad (2.5)$$

When n_i and p_i are independent we have

$$\mathrm{plim}\ (\hat{r}) = r + \frac{1}{\bar{p} + (1-\bar{p})\bar{n}/\sigma_n^2 + \sigma_p^2[1 + (\bar{n}^2-n)/\sigma_n^2]/\bar{p}}$$

and comparing this to (2.2) we see if $r=0$ the bias in $\hat{r}$ is reduced when p_i is random and uncorrelated with u_i. The random element of p_i increases the variance of d_i leaving the covariance between d_i and u_i unchanged. As noted above, if d_i did not measure n_i with error, then plim ($\hat{r}$) would be roughly $1/\bar{p}$; the presence of more measurement error in d_i due to the random nature of p_i reduces the extent of the bias towards $1/\bar{p}$.

When n is held constant (2.5) reduces to

$$\mathrm{var}(d_i|n) = n\bar{p}(1-\bar{p}) + \sigma_{p|n}^2(n^2-n)$$

which gives the variance in deaths for a given parity as a funtion of the mean and variance of the mortality rate for that parity. For each parity $\mathrm{var}(d_i|n)$ can be calculated so we can solve for $\sigma_{p|n}^2$, the within parity variance in p_i given n. This gives us a second relation

$$\sigma_{p|n}^2 = (1-\rho^2)\sigma_p^2 \qquad (2.6)$$

which, together with (2.5) can be solved for σ_p^2 and ρ , and we can then evaluate plim ($\hat{r}$). If n_i and p_i are not independent, plim ($\hat{r}$) will depend upon the form of the bivariate distribution on n_i and p_i because of the presence of high order moments. While n_i is discrete it is convenient to use a continuous approximation. The distribution of n_i is skewed right, but based upon the Box-Cox [1969] analysis its distribution is somewhere between normal and log-normal, being somewhat closer to the former. By contrast, we do not directly observe realized values of p_i , but since the mean of p_i is roughly equal to the within parity standard deviation, it is clear p_i cannot be normal. We will proceed under the assumption that p_i is log-normally distributed. In order to make the evaluation of the higher order moments easier, we will consider two joint distributions 1) log (p_i) and n_i are bivariate normal and 2) log (p_i) and log (n_i) are bivariate normal. Occasionally two roots are produced by (2.5) and (2.6), this occurring more often in the normal log-normal case than in the bivariate log-normal case. In all cases the second root produces an estimate of the standard deviation of the mortality rate across the population which is implausibly large, say 0.5, or five times the within parity standard deviation. Because this second root only occasionally occurs, and when it does produces anomalous results, we view it as a numerical artifact and of no substantive interest.

D. Extension to Multivariate Regression

When additional explanatory variables enter the regression, the above results must be slightly modified. Let us call this set of regressors x . Instead of $\bar{n}$, we must use the mean of n_i given x_i . Likewise Var(n) and Var(d) give way to the conditional variances of n and d given x . These conditional variances are simply the unexplained variance from regressions of n and d on x, respectively. Since $\bar{n}$ gives way to $E(n_i|x_i)$, our probability limits take on different values for different values of x_i . In the case where the x_i are fixed in repeated sampling, the appropriate probability limit would involve the separate probability limits which result for different x_i . A simpler but inexact alternative is to evaluate the probability limits at the sample mean of x_i , which would result in simply using $\bar{n}$. The preceeding formulae hold so long as the unconditional variances of n and d are replaced by their conditional variances given x_i.

III. Empirical Application

A. Direct Replacement of Deaths

The method in section II is applied here to the 1973 Colombia Census Public Use Sample. In table 2 we present the summary statistics and raw regression coefficients for wives grouped by age and urban versus rural location. Only the mortality coefficients are given even when other regressors are used.[2] When the instrumental variables method is used, the full set of exogenous variables and d_i/n_i are used as instruments. All women are married with husband present; only live births are considered. In table 3 we show a summary of fertility and mortality for rural wives 35-39.

In table 3 we observe a tendency for the mortality rate to rise with higher parities while the standard deviation of the mortality rate for wives with the same parity is roughly the same across all parities. This same pattern is repeated in the other groups we consider. The constancy of $\sigma_{p|n}$ indicates our simple structure with a cross-sectional variance for p , which is equal for all parities, is roughly correct. The large values for the standard deviation of p for given parity are also powerful evidence that p cannot be considered a fixed parameter. We have not attempted to determine whether the exogenous regressors explain all or part of the variation in p . If p is constant across wives,

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[2]The regressors consist of a set of dummy variables representing schooling categories for the wife and husband as well as a set of regional dummies.

TABLE 2

Summary Statistics and Regression Coefficients for Colombian Women

	Urban 35-39	Rural 35-39	Urban 40-44	Rural 40-44	Urban 45-49	Rural 45-49
Descriptive Statistics						
$E(n_i)$	5.69	7.36	6.63	8.26	7.15	8.61
$var(n_i)$	8.84	10.25	12.19	13.33	14.83	15.99
$var(n_i \mid x_i)$	7.76	9.80	11.20	12.96	13.68	15.29
mean mortality rate	0.0932	0.1503	0.1124	0.1747	0.1359	0.1888
$var(p \mid n)$	0.008742	0.01392	0.01319	0.01837	0.01360	0.01936
$var(d_i)$	1.47	2.76	2.18	3.81	3.09	4.70
$var(d_i \mid x)$	1.37	2.66	2.07	3.64	2.89	4.49
Number of observations	7612	4562	6007	3575	4456	2635
Regression Coefficients						
d_i OLS x out	1.26 (R^2= .26)	1.01 (R^2= .27)	1.24 (R^2= .28)	0.99 (R^2= .28)	1.27 (R^2= .34)	1.06 (R^2= .33)
d_i OLS x in	1.13 (R^2= .32)	0.98 (R^2= .30)	1.16 (R^2= .31)	1.00 (R^2= .30)	1.21 (R^2= .36)	1.04 (R^2= .35)
d_i IV x out	0.93	0.61	0.80	0.55	0.86	0.57
d_i IV x in	0.71	0.55	0.64	0.52	0.72	0.49
d_i/n_i x out	6.30 (R^2= .09)	4.81 (R^2= .07)	5.40 (R^2= .07)	4.54 (R^2= .06)	6.70 (R^2= .09)	4.55 (R^2= .05)
d_i/n_i x in	5.00 (R^2= .18)	4.46 (R^2= .11)	4.61 (R^2= .13)	4.43 (R^2= .09)	5.80 (R^2= .15)	4.25 (R^2= .10)

Table 3

Fertility and Mortality Summary
Rural Wives 35-39

Parity	No. Wives	Mortality Rate	$\sigma_{p\|n}$
1	114	.07018	n.a.
2	194	.07474	.0994
3	250	.08533	.107
4	324	.07639	.103
5	452	.09513	.0942
6	517	.1006	.113
7	547	.1097	.113
8	527	.1369	.135
9	486	.1369	.123
10	408	.1667	.135
11	255	.1512	.113
12	232	.2274	.141
13	127	.2483	.111
14	64	.2891	.162
15	30	.2022	.131
16	14	.2857	.142
17	11	.4759	.155
18	7	.3333	.101
19	3	.5789	negative

TABLE 4

Stochastic Structure and Estimates of Replacement Rates[1]

Assumed Structure and Parameters Estimated	Urban 35-39	Rural 35-39	Urban 40-44	Rural 40-44	Urban 45-49	Rural 45-49
p_i constant						
r (x out)	-0.22	-0.31	-0.44	-0.47	-0.54	-0.53
r (x in)	-0.18	-0.28	-0.41	-0.42	-0.49	-0.51
predicted $var(d_i)$	0.56	1.17	0.82	1.59	1.11	1.89
p_i random, independent of n_i						
r (x out)	0.31	0.23	0.31	0.20	0.21	0.19
r (x in)	0.27	0.22	0.29	0.23	0.20	0.20
predicted $var(d_i)$	0.87	1.97	1.47	2.94	1.91	3.47
predicted $var(d_i \mid x_i)$	0.85	1.95	1.44	2.93	1.89	3.43
p_i random, $\rho \neq 0$, x out						
$\log(p_i)$ and n_i bivariate normal						
r	0.24	0.14	0.27	0.14	0.22	0.15
h	1.66	0.89	1.18	0.63	1.24	0.71
ρ	0.32	0.27	0.27	0.20	0.30	0.23
σ_p	0.10	0.13	0.12	0.14	0.13	0.15
$\log(p_i)$ and $\log(n_i)$ bivariate normal						
r	0.16	0.10	0.21	0.12	0.16	0.12
h	1.41	0.84	0.83	0.51	1.12	0.60
ρ	0.27	0.23	0.19	0.16	0.25	0.18
σ_p	0.10	0.12	0.12	0.14	0.12	0.14
p random, $\rho \neq 0$, x in						
$\log(p_i)$ and n_i bivariate normal						
r	0.17	0.13	0.23	0.17	0.19	0.16
h	1.52	0.83	0.97	0.56	1.15	0.64
ρ	0.31	0.25	0.23	0.18	0.29	0.21
σ_p	0.10	0.13	0.12	0.14	0.13	0.15
$\log(p_i)$ and $\log(n_i)$ bivariate normal						
r	0.10	0.10	0.18	0.15	0.13	0.13
h	1.27	0.80	0.75	0.44	1.00	0.55
ρ	0.26	0.22	0.18	0.14	0.23	0.17
σ_p	0.10	0.12	0.12	0.14	0.12	0.14

[1] t-statistics have not been provided for the various values of $\hat{r}$. Sample sizes are so large $\hat{r}$ is always significantly different from zero. The correct standard errors for $\hat{r}$ range from 0.02 to 0.03.

the variance of child deaths for rural wives 35-39 should be $\bar{n}\bar{p}(1-\bar{p}) + \bar{p}^2 var(n)$. Substituting $\bar{n} = 7.36$, $\bar{p} = 0.1503$ and $var(n) = 10.25$, makes the predicted value of $var(d_i)$ be 1.17 if there is no variance in the mortality rate across wives. Since the sample variance of d_i is 2.76, which is over twice as large as predicted with nonrandom p , we are forced to concede that p is random. Later in this section we will examine whether the random mortality rate is correlated with fertility.

In table 4 the various OLS coefficients in table 2 are corrected for bias under a variety of assumptions about the true model. One property of the empirical results is that the presence of other regressors makes little difference in the estimates of replacement since the signs and magnitudes of $\hat{r}$ are unchanged. While this result need not generalize across all data sets, it at least suggests that researchers may interpret existing regressions using the simpler formulae in the absence of regressors. Another common thread running through the results is that treating p as constant for all women gives the lowest value to r . As mentioned in section II Wallace has derived a correction which uses as the dependent variable not n_i but rather $n_i - E(n_i|d_i)$ where $E(n_i|d_i)$ is calculated using the empirical frequency function for n together with the assumption that deaths follow a simple binomial model with fixed p . Table 5 compares the replacement result produced by the Wallace method with those obtained by the method here with p fixed.

Table 5

Comparison of Replacement Estimates

Sample	Wallace Correction	OLS Correction
Colombia 35-39 rural	-0.22	-0.31
Colombia 35-39 urban	-0.11	-0.22
Kenya, older women	0.07	0.06

The results are fairly close, but both sets of results must be rejected for the Colombian data since the assumption of constant p is untenable.

If we assume p is random and independent of n , then the replacement rate is around 0.25. When we assume p_i is random but independent of n_i , the predicted variance of d_i is closer to the realized value than when we assume p_i fixed, but it is not close enough so that we could accept the null hypothesis that independence holds. With the large samples used here the standard deviation of the sample variance of d_i will be roughly 0.05.[3]

Since we cannot accept the simple model based upon $\rho=0$, we are forced to solve the two nonlinear equations (2.5) and (2.6) for ρ and σ_p . The numerical solution to these equations can be obtained quite easily either by trial and error or by using a computer. The expressions in Lemma 5 in the Appendix enable us to obtain the required higher order moments for n_i and p_i . This has been done in Table 4 allowing n_i to be either normally or log-normally distributed while p_i is log-normally distributed. n_i is more nearly normal, but both distributions yield nearly identical results.

Because the death of an infant interrupts lactation, there may be a spurious effect similar to replacement imparted by a shorter post-partem period of sterility. Preston (1975) has hypothesized that this effect is minor in Latin America where the duration of breast feeding is relatively short, say three to seven months compared to an average birth interval of thirty months. If these

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[3]This is based on the well known formula $var(s^2) = 3\sigma^4$ where s^2 is the sample variance of a normal variable with population variance σ^2 . While the distribution of d_i with random n and p is not normal, we would still have to reject $\rho=0$ even if the standard deviation of the sample variance were several times as large as the normal formula suggests.

sterility effects produce a replacement effect of 0.1, then the behavioral replacement response to a child death is roughly 0.08. However, if breast-feeding is used for control over fertility, its effect is not spurious but behavioral which would make 0.18 the proper estimate of direct replacement.

B. Replacement by Hoarding

As mentioned in section II fertility hoarding is another possible response to the uncertainties produced by mortality. If each couple in our sample knows its specific mortality rate, then we may use the estimated correlation between p_i and n_i to infer the hoarding response to mortality, that is dn_i/dp_i. This effect can be estimated as $\hat{\gamma}=\rho\sigma_n/\sigma_p$ so long as $r^2 var(d_i)$ is small relative to $var(n_i)$, which it is. If we divide $\hat{\gamma}$ by $\bar{n}$ we can approximate h--the hoarding response to an additional death (see sections II A and B). This coefficient is also shown in table 4. Because the solutions for ρ and σ_p were obtained from a highly nonlinear equation, it is most difficult to attach standard errors to $\hat{h}$. If hoarding accounts for even half of the correlation between n_i and p_i it is roughly twice as important as direct death related replacement in offsetting mortality.

When we speak of replacement, we should bear in mind that couples need not have extra births at the end of the reproductive period to offset past deaths. Instead, replacement may be implemented by adjusting the timing of the last birth. In the absence of any method for adjusting the probability of a birth, replacement would be impossible and hoarding would be the only response. If it is easier to adjust fecundity downward when the couple is older (abstinance, vasectomy, etc.) than upward, the final adjustment to fertility will more likely involve a decision to stop bearing children rather than a decision to bear children more quickly to replace deaths. If couples do not hoard children in anticipation of mortality but instead use direct replacement, say by means of sterilization as soon as desired fertility is reached, it will appear as if they hoard when we observe them before the completion of fertility. Such a couple with a large p_i will bear children at a rapid pace until reaching their target. Their apparent high fertility early in life mimicks hoarding. The fact that the couple has an additional birth for each additional death will only be apparent from their behavior at the end of their life. At middle age a pure hoarding strategy may be difficult to distinguish from a pure replacement strategy consisting of initial hoarding with terminal contraception. This may explain why hoarding appears to be more important in the youngest age group in table 4.

Even though the correlation between fertility and mortality rates remains large even when we control for education and region; we cannot be certain this correlation reflects hoarding. In order for this correlation to reflect hoarding, couples must have information about their particular value of p_i. If family and/or local history explain nontrivial amounts of the variation in p_i then it is reasonable to assume couples do have sufficient information to construct a hoarding strategy. There may be hoarding in response to the level of $\bar{p}$, but we cannot separate this from the overall level of desired fertility on the basis of cross-sectional data.

IV. Summary

In conclusion, we note that the corrections described here make is possible to estimate the extent of direct replacement of children who die using a linear regression of fertility on child mortality. A variety of corrections is given depending upon the stochastic structure of the mortality process. While some of the calculations are specialized, none is terribly complex. For the Colombian data examined here direct replacement is present but of modest magnitude. There appears to be substantial individual specific variation in the mortality rate, and this variation is positively correlated with fertility. The theoretical variance of d_i is shown to be a function of the mean and variance of both the mortality rate and children ever born as well as the correlation between fertility and the mortality rate. It is possible to test restrictions on the mortality process such as p_i fixed or p_i random and uncorrelated with n_i by testing the implied restrictions on the theoretical variance of d_i. These tests should be carried out even if other estimation strategies are used since a correlation between the individual specific mortality rate and fertility will bias almost any estimator of the replacement effect. Such bias can easily be taken into account using the estimator developed here. Existing studies of replacement using parity progression ratios or stopping probabilities implicitly assume the mortality rate is independent of fertility. The empirical work here demonstrates this assumption is incorrect for Colombia, which suggests these existing studies may be unreliable. If parity progression ratios or stopping probabilities are to be used to study replacement, then the methods must be reformulated to take into account correlation between fertility and the mortality rate.

Appendix

Let d_i be the number of successes from n_i Bernoulli trials each with probability of success π. The distribution of d_i given n_i is binomial.

Lemma 1: $\mathrm{Var}(d_i) = E(n_i)\pi(1-\pi) + \pi^2 \mathrm{Var}(n_i)$

$$\mathrm{Var}(d_i) = E(d_i^2) - E(d_i)^2$$

$$E(d_i) = \int_{n_i} \sum_d d\, f(d|n_i)\, g(n_i)\, dn_i$$

$$= \int_{n_i} n_i\, \pi\, g(n_i)\, dn_i = \pi\, E(n_i)$$

$$E(d_i^2) = \int_{n_i} \sum_d d^2\, f(d|n_i)\, g(n_i)\, dn_i$$

$$= \int_{n_i} (n_i\pi(1-\pi) + n_i^2\pi^2)\, g(n_i)\, dn_i$$

$$= E(n_i)\pi(1-\pi) + \pi^2\, [\mathrm{Var}(n_i) + E(n_i)^2]$$

The result follows immediately.

If $E(n_i) = \bar{n}$, and $n_i = \bar{n} + u_i$ then $d_i = \pi\bar{n} + \pi u_i + \varepsilon_i$.

Lemma 2:

The unconditional variance of ε_i is $\bar{n}\pi(1-\pi)$

$$\mathrm{Var}(\varepsilon_i) = \int_{u_i} \int_{\varepsilon_i} \varepsilon_i^2\, f(\varepsilon_i|n_i)\, g(u_i)\, d\varepsilon_i\, du_i$$

$$= \int_{u_i} [\int_{\varepsilon_i} \varepsilon_i^2\, f(\varepsilon_i|n_i)\, d\varepsilon_i]\, g(n_i)\, du_i$$

The term in the brackets is the conditional variance of ε_i given u_i which is $(\bar{n} + u_i)\, \pi\, (1-\pi)$ so $\mathrm{Var}(\varepsilon_i) = \int_{u_i} (\bar{n} + u_i)\pi(1-\pi)\, g(u_i)\, du_i$

$= \bar{n}\pi(1-\pi)$.

Lemma 3: $E(\varepsilon_i{}^2 u_i) = Var(u_i)\pi(1-\pi)$

$$E(\varepsilon_i{}^2 u_i) = \int_{u_i} u_i \left[\int_{\varepsilon_i} \varepsilon_i^2\, f(\varepsilon_i|u_i)\, d\varepsilon_i\right] g(u_i)\, du_i$$

$$= \int_{u_i} (\bar{n}u_i + u_i{}^2)\pi(1-\pi)\, g(u_i)\, du_i$$

$$= Var(u_i)\pi(1-\pi)$$

Lemma 4: Let π_i and n_i be jointly distributed with higher order moments which follow the bivariate normal pattern. Then

$$Var(d_i) = \bar{n}\,\bar{\pi}(1-\bar{\pi}) + (1+\rho^2)\,\sigma_\pi^2\,\sigma_n^2 + \sigma_\pi^2\,(\bar{n}^2 - \bar{n})$$

$$+ \sigma_n^2\pi^2 + \rho\sigma_\pi\sigma_n\,(1-2\bar{\pi} + 2\bar{n}\bar{\pi})$$

We have

$$E(d_i) = \int_n \int_\pi d_i\; f(d_i|n_i,\pi_i)\; g(n_i,\pi_i)\; d\pi_i\, dn_i$$

$$= E(n_i\pi_i) = \bar{n}\,\bar{\pi} + \rho\sigma_\pi\sigma_n$$

$$E(d_i{}^2) = \int_n \int_\pi d_i{}^2\; f(d_i|n_i,\pi_i)\; g(n_i,\pi_i)\; d\pi_i\; dn_i$$

$$= E(n_i\pi_i - n_i\pi_i{}^2 + n_i{}^2\pi_i{}^2)$$

$$= \bar{n}\,\bar{\pi} + \rho\sigma_\pi\sigma_n - \sigma_\pi^2\bar{n} - \bar{\pi}^2\bar{n} - 2\bar{\pi}\rho\sigma_\pi\sigma_n + \sigma_\pi^2\sigma_n^2$$

$$+ \sigma_n^2\bar{\pi}^2 + \bar{n}^2\sigma^2_\pi + \bar{n}^2\bar{\pi}^2 + 2\rho^2\sigma_\pi^2\sigma_n^2 + 4\bar{n}\bar{\pi}\rho\sigma_n\sigma_\pi$$

from which the result follows. Note that as a corollary when we condition upon n_i we replace $g(n_i,\pi_i)$ with $h(\pi_i|n_i)$ and obtain

$$Var(d_i|n_i) = n_i\bar{\pi}_{n_i}(1-\bar{\pi}_{n_i}) + \sigma^2_{\pi|n_i}(n_i^2 - n_i)$$

where $\bar{\pi}_{n_i}$ is $E(\pi_i|n_i)$ and $\sigma^2_{\pi|n_i}$ is $Var(\pi_i|n_i)$

Lemma 5: If we assume ln(X) and ln(Y) follow a bivariate normal density, then the moments of X and Y are

$$E(X^r Y^s) = \exp[r\mu_x + s\mu_y + 1/2(r^2\sigma_x^2 + s^2\sigma_y^2 + 2rs\rho\sigma_x\sigma_y)]$$

If X and ln(Y) are joint normal the joint moments $E(X^r Y^s)$ can be obtained by differentiating the moment generating function

$$M(t) = \exp[t\mu_x + s\mu_y + 1/2(t^2\sigma_x^2 + s^2\sigma_y^2 + 2ts\rho\sigma_x\sigma_y)]$$

r times with respect to t and then setting t=0 . Note that ρ is not the correlation between X and Y . Because ln(Y) is normal, Y is a nonlinear transform of a normal variable. This means the correlation between Y and X is restricted to be considerably under one in absolute value since the correlation is a measure of linear association.

The derivation of these results follows from direct integration.

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