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Are Selective**



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# Estimating the Determinants of Child Health When Fertility and Mortality Are Selective

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Mark M. Pitt

## ABSTRACT

*This paper estimates the determinants of child mortality and child health allowing for the possibility that samples of children are choice-based, reflecting prior selective fertility and mortality behavior. Parameter identification is the most serious practical problem in controlling for fertility and mortality selection. Identification is achieved by imposing a random-effects structure on the error correlation matrix for the set of fertility, mortality, and health behaviors. Fertility selection is found to be statistically significant in the estimation of the determinants of mortality in all 14 Sub-Saharan DHS data sets studied, and fertility and mortality selection is found to be significant in the determination of child height in Zambia. Nevertheless, most parameters are little changed when selection is accounted for.*

## I. Introduction

In recent years, a large literature has arisen which estimates the effect of household and community characteristics on the health and mortality of children in the developing world.<sup>1</sup> From these reduced-form estimates inferences are drawn concerning the efficacy of a variety of interventions, including investments in health infrastructure, on measures of child health, such as weight and

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1. For a survey of this literature see Behrman and Deolalikar (1988).

height, and on survival probabilities. This literature has consistently found that mothers' schooling is an important determinant of improved health status and survival of children.<sup>2</sup> Another literature has examined the determinants of fertility and has consistently found that women's schooling is negatively associated with fertility.<sup>3</sup> Some studies have independently estimated both the determinants of fertility and child health from the same data. Education, and many other of the covariates used in these reduced forms, including measures of health infrastructure, have an effect on child health at least at three points: (a) in the decision to have children; (b) conditional on the decision to have children, on child survival; and (c) conditional on birth and survival, health as measured at the time of the survey.<sup>4</sup> Most studies of the effects of education and health infrastructure on child health have not taken fertility decisions into account—they assume that the composition of the population of children classified by health is unrelated to prior fertility decisions. If fertility reflects the choices of women and the households in which they reside, how can the results of that choice, children, be considered as randomly drawn from the population of *potential births*? If births are not randomly drawn from the population of potential births, what are the implications for the measurement of the effects of women's schooling and program interventions on child mortality and health? If parents care about the health outcomes of potential births, then any variable, measured in the data or not, that affects the healthiness of children born will influence fertility decisions. It is the *unmeasured* determinants of health which are the potential source of bias in the estimation of the effects of education on child health using samples of children. Failure to account for the possibility that these unmeasured determinants of health affect the fertility decision, and thus the composition of those born classified by health status, will result in biased estimates of the determinants of child health.

There is substantial evidence that parents are indeed attentive to the (unmeasured) inherent healthiness (the "health endowment") and survival probabilities of potential births (Olsen and Wolpin 1983; Rosenzweig 1986; Rosenzweig and Schultz 1983). Variation in inherent healthiness of children may result from the

2. For example, T. Paul Schultz (1989) reviews some of the social science literature relating mother's schooling to decreases in the incidence of mortality, suggesting that "[the] universality of the relationship is reminiscent of the discovery in the 1960s of relative (logarithmic) wage differential associated with years of schooling" (p. 63). He suggests that an added year of maternal education is associated with a 5 to 10 percent reduction in child mortality. Cochrane, Leslie, and O'Hara (1982) review 16 studies and find a strong relationship between child mortality and mother's education. They suggest that an additional year of maternal education results in a reduction of 9/1000 in the mortality of her children. A review of the Sub-Saharan African literature is provided in Tabutin and Akoto (1992).

3. Surveys of the effects of education on fertility include Cochrane (1987), Cochrane (1983), and Cleland and Rodriguez (1987).

4. Induced and spontaneous abortion, and child fostering, could be added to the list of selective events affecting the composition of a sample of children whose health is measured at the time of a survey. In addition, if children enter a sample only if their mother resides in a sampled household, mortality of mothers may also be selective in the determination of child health and mortality. This issue may be of some consequence in the Demographic and Health Surveys of Sub-Saharan Africa used in this paper, in which high rates of maternal death, due in part to high rates of HIV infection, systematically select out many of the children of deceased mothers. This issue has recently been addressed in Bicego et al. (1995).

genetic endowments of parents as well as the health characteristics of the household and its spatial location which are not well measured in the data, including water purity, disease prevalence, and access to health facilities. Inherent healthiness and other unmeasured determinants of health will influence the fertility decision of parents who consider the health and survival outcomes of potential births in making fertility decisions. If parents are less likely to have a child when its inherent healthiness is perceived as low, we have positive birth selection. If parents are more likely to have a child when its inherent healthiness is perceived as low, we have negative birth selection. In either case, estimates of the determinants of child health or mortality based on samples of births are likely to suffer from classical sample selection bias—the distribution of the errors (which include inherent healthiness and other unobserved factors) in the child health equation is likely to be truncated by the fertility choices of parents and the nonsurvival of children to the survey date.

Consider the implication for analyzing the effect of women's schooling on the health or survival of their children. It might be that the measured effect of women's schooling on these outcomes represents nothing more than its effect on the composition of those born by inherent (unobserved) healthiness. This would result if reductions in fertility brought about by an increase in education disproportionately reduced the births of infants with low inherent healthiness. Conversely, the effect of women's schooling in enhancing child health and survival may be seriously underestimated if the reductions in fertility resulting from an increase in women's schooling disproportionately reduce the births of children with high inherent healthiness. Knowledge of the total effect on health and survival of an intervention, such as women's schooling, that affects both fertility decisions and the health of those who are born, requires estimating the parameters of both the birth selection process and the effect of the intervention on health *conditional* on being born.

Just as fertility selection may bias estimates of the determinants of child mortality and health, so too will mortality selection bias estimates of the determinants of child health. Many recent studies have used anthropometric measures of nutritional status, such as weight and height, in the analysis of child health behavior. In order to be anthropometrically measured, a child must both have been born and have survived to the survey date. Those who were born and failed to survive to the survey date are unlikely to be random draws from the population of ever-born children. It seems likely that those who are least healthy, such as those of low weight and height for their age, are most likely not to survive.

The most serious practical problem in controlling for fertility and mortality selection has been that of parameter identification. If parents care about the health outcomes of potential births, then any exogenous variable that affects health also affects the fertility decision. The sequential nature of the decision process means that there is not likely to be an exclusion restriction of the usual sort—a variable or variables that influence the endogenous regressors, birth and survival, that do not otherwise influence child health *conditional* on this regressor. Pitt and Rosenzweig (1989) rely on the nonlinearity of the choice of the bivariate normal error distribution to identify fertility-selection-corrected reduced-form models of birth weight, relying on underidentified semiparametric estimates as the basis for

a test of the validity of this error distribution. Grossman and Joyce (1990), controlling for self-selection into the resolution of pregnancies, recognize that identification is not on firm ground if based solely on distributional assumptions and identify their model with exclusion restrictions not based on any explicit model of behavior. Pitt (1995) assumes that first-births are exogenous in order to identify the determinants of child mortality conditional on fertility selection in high-fertility Sub-Saharan Africa. The idea is that if each woman in a sample of women had at least one child, one could construct a sample of children that would not be self-selected because it reflects the full distribution of women. Lee, Rosenzweig, and Pitt (forthcoming) circumvent the problem entirely by estimating structural models in which the set of instrumental variables that identify health inputs in a health production function also serve to identify the selection correction.

This paper takes an entirely new direction to disentangling the parameters of the reduced-form determinants of fertility from the parameters of the reduced-form determinants of child mortality, and the reduced-form determinants of both fertility and mortality from the determinants of health, in the joint estimation of the determinants of all three behaviors. The method requires the specification and estimation of joint multiperiod models of fertility, mortality, and health, achieving identification by an intuitive set of restrictions on the intertemporal error correlations. The data required, pregnancy histories of women and health outcomes for more than one child of some of the sampled women, are now commonly found in micro surveys with a health emphasis such as the Demographic and Health Surveys, the Malaysian and Indonesian Family Life Surveys, and the National Longitudinal Survey of Youth. Identification via covariance restrictions does not rely on difficult-to-justify exclusion restrictions or the nonlinearity of error distributions for identification, is flexible in many directions, and is applicable to the estimation of both reduced forms and structural equations.

These methods are illustrated with the estimation of reduced-form determinants of child health and mortality in a set of Sub-Saharan countries for which a Demographic and Health Survey (DHS) exists. Although these data sets do not provide a rich set of exogenous covariates, they are the most important sources of information on demographic behavior in Africa and in many other developing countries. One intent is to obtain a measure of the effect of mother's schooling on child health and mortality allowing for the potentially selective nature of fertility and mortality. The discrete (binary) nature of mortality make parameter identification based solely on the choice of an error distribution particularly difficult. The results reported below provide evidence of the statistical significance of selective fertility, although the magnitude of the bias on the estimated effect of women's schooling and age on the probability of child death prior to age two years is not large. Evidence of the statistical significance of selective fertility and mortality in the determination of child anthropometrically measured nutritional status is also presented, although, once again, the magnitude of the bias in the regression parameters is not large.

The next section of this paper sets out the problem of estimating the determinants of child health and mortality when fertility and mortality are selective in a more formal framework. The third part of the paper discusses the difficulties of statistically identifying the empirical model. Section IV presents a method of

identification based upon covariance restrictions that have intuitive appeal along with empirical results from Sub-Saharan Africa. Section V summarizes the results.

## II. Selective Fertility and the Health of Children

To examine the potentially selective nature of fertility in the estimation of the determinants of child health, consider the linear reduced-form demand equations for latent fertility,  $f^*$ , and health,  $h$ :

- (1)  $h = X_h \beta_h + \mu_h + v_h = X_h \beta_h + \varepsilon_h$
- (2)  $f^* = X_f \beta_f + \beta_{fh} \mu_h + v_f = X_f \beta_f + \varepsilon_f$ ,

where  $X_f$  and  $X_h$  are vectors of exogenous regressors, the compound error in each equation ( $\varepsilon_f$  and  $\varepsilon_h$ ) contains a heterogeneous component of health known to parents but unmeasured in the data (inherent healthiness),  $\mu_h$ , as well as non-systematic shocks  $v_f$  and  $v_h$ , and the  $\beta$ 's are parameters to be estimated representing parental responses to exogenous variables, including unmeasured heterogeneity.<sup>5</sup> Latent fertility  $f^*$  represents the unobserved intensity of reproductive effort including fecundity. If this intensity exceeds some threshold, then a birth results; otherwise it does not.

If the error terms have zero means, and the nonsystematic errors  $v_f$  and  $v_h$  are uncorrelated, then the covariance between the compound errors  $\varepsilon_f$  and  $\varepsilon_h$  is

- (3)  $\text{cov}(\varepsilon_f, \varepsilon_h) = \beta_{fh} \text{var}(\mu_h)$ .

The compound errors of the fertility and health equations are correlated, and selection bias results if parents take unobserved health heterogeneity into account when making fertility decisions, that is,  $\beta_{fh} \neq 0$ , and if such unmeasured health heterogeneity actually exists in the sampled population ( $\text{var}(\mu_h) > 0$ ).

Bias in estimating the reduced-form health equation (Equation (2)) arises because births are more likely to occur when  $\beta_{fh} \mu_h$  takes large values than when it takes small values, conditional on  $X_f$ . In the case in which all errors are distributed as joint normal, the regression function for the "population" of births (that is, the health of children conditioned on their birth) is

- (4)  $E(h|X_h, f^* > 0) = X_h \beta_h + E(\varepsilon_h|\varepsilon_f > -X_f \beta_f) = X_h \beta_h + \text{cov}(\varepsilon_h, \varepsilon_f) \lambda$ ,

where  $\lambda$  is the inverse Mill's ratio, the ratio of the density and distribution functions of the standard normal variable  $X_f \beta_f$  normalized by the standard deviation

5. These are fertility and health demand questions derived, in principle, from the standard framework of a household maximizing a utility function that includes the number and health "quality" of children, where quality is produced by the household. These "solved out" (reduced-form) demand equations are functions of only the exogenous variables of the household's optimization problem, ignoring dynamic considerations such as replacement effects. If, for example, fertility responded to the number of surviving children, then the reduced-form would have to include all past exogenous determinants, not just contemporaneous determinants. The only time-varying covariates in the data used below are calendar time and mother's age. The specification used here is meant to be representative of the fertility and health demand equations most often estimated in the literature.

of  $\varepsilon_f$ . Estimating (4) based on the sample of births without taking into account birth selection is equivalent to omitting the  $\lambda$  term in (4). As in Pitt and Rosenzweig (1989), the effect of a change in a common exogenous regressor ( $X_{fk} = X_{hk}$ ) such as women's schooling on health in a fertility-choice-based sample of births is

$$(5) \quad \frac{dE(h|X_h, f^* > 0)}{dX_k} = \beta_{hk} - \beta_{fk}(\lambda^2 + X_f\beta_f\lambda)\beta_{fh} \text{var}(\mu_h).$$

Since  $\text{var}(\mu_h) > 0$ ,  $\lambda > 0$ , and  $X_f\beta_f$  is also likely to be positive if a birth has occurred, the sign of the expression after the minus sign in (5) depends only on the signs of  $\beta_{fk}$  and  $\beta_{fh}$ . If women with lower health endowments are more likely to have children,  $\beta_{fh} < 0$  (negative birth selection), and if fertility is decreasing in women's schooling ( $\beta_{fk} < 0$ ), then, if fertility selection is not taken into account, the effect of women's schooling on health will be underestimated (the sign of the expression after the minus sign in (5) is positive).

### III. The Problem of Identification

Jointly estimating the determinants of fertility and the resulting health of those born is complicated by the difficulty in disentangling the parameters of the reduced-form determinants of fertility from the parameters of the reduced-form determinants of child mortality and health. If parents care about the health outcomes of potential births, then any exogenous variable that affects health also affects the fertility decision. Even if there were uncertainty about inherent healthiness and decisions were sequential and myopic, because the birth decision precedes the health behaviors resulting from that decision, there cannot be fewer observed or known exogenous variables influencing health than influencing the fertility decision. Conditioning on birth is required, in the case of the mortality reduced form, or on birth and survival, in the case of the weight and height reduced form, if the children whose births were averted have differing inherent healthiness or survival probabilities than those whose births were not averted. This might be the case, for example, if low-fertility women tend to have children with higher survival probabilities than higher-fertility women. The sequential nature of the decision process means that there is not likely to be an exclusion restriction of the usual sort—a variable or variables that influence the endogenous regressor, birth, that do not otherwise influence child health *conditional* on this regressor.

The discrete nature of mortality makes identification based exclusively on the choice of an error distribution problematic. The identification problem arises because all the regressors contained in  $X_f$  are also contained in  $X_h$ .<sup>6</sup> The general form of the regression equation (Equation (4)) is

$$(6) \quad E(h|X_h, f^* > 0) = X_h\beta_h + \rho g(X_f\beta_f),$$

6. The set of regressors  $X_h$  also includes variables that affect health outcomes but are not known to parents until after the fertility decision is made. Among them are the sex of the child and whether the pregnancy resulted in multiple births (twins).

where the function  $g(X_f\beta_f)$  adjusts the regression function for choice-based sampling (it is the mean of the truncated error distribution) and  $\rho$  is the correlation between the errors  $\varepsilon_f$  and  $\varepsilon_h$ . If the joint distribution of  $\varepsilon_f$  and  $\varepsilon_h$  is bivariate normal, then the function  $g(\cdot)$  is proportional to the inverse Mill's ratio  $\lambda$ , and the choice-based regression function is (4) above. For all distributions except the uniform distribution, the function  $g(\cdot)$  is nonlinear, and it is solely this nonlinearity that identifies the model.<sup>7</sup>

The fertility selection problem is more complex when the health outcome is not measured as a continuous variable. An important case of a discrete health outcome is infant or child mortality. The joint probability of observing both a birth ( $F = 1$ ) and a death ( $D = 1$ ) prior to some age is

$$(7) \quad \text{Prob}(F = 1, D = 1) = \Phi_2(X_f\beta_f, X_h\beta_h, \rho),$$

where  $\Phi_2$  represents the standard bivariate normal cumulative distribution with correlation  $\rho$ . In the discrete case, the analog to the regression function conditional on a birth (4) is the conditional probability of the mortality event, conditional on the birth event:

$$(8) \quad \text{Prob}(D = 1|F = 1) = \frac{\Phi_2(X_f\beta_f, X_h\beta_h, \rho)}{\Phi(X_f\beta_f)},$$

where  $\Phi$  is the standard univariate normal cumulative distribution. The binary probit model for mortality applied to a sample of children that ignores fertility selection maintains that  $\rho = 0$  so that  $\Phi_2(X_f\beta_f, X_h\beta_h, 0) = \Phi(X_f\beta_f) \times \Phi(X_h\beta_h)$ .

The likelihood for the bivariate discrete choice problem (with time subscripts omitted) is

$$(9) \quad L = \prod_{F=0} \Phi(-X_{fi}\beta_f) \prod_{F=1, D=1} \Phi_2(X_{fi}\beta_f, X_{hi}\beta_h, \rho) \\ \times \prod_{F=1, D=0} \Phi_2(X_{fi}\beta_f, -X_{hi}\beta_h, -\rho),$$

which has been referred to as the bivariate probit model with partial observability. The "partial observability" is the lack of information on the survival/mortality outcomes of those never born.

7. The identification problem is somewhat different when estimating the structural determinants of health (the health "production function") when fertility or mortality is selective. Lee, Rosenzweig, and Pitt (forthcoming) examine the effects of mortality selection on estimating the effects of health interventions that improve the health infrastructure, using data from India, Bangladesh, and the Philippines. They employ semi-parametric estimators to estimate (i) the effects of water and sanitation facilities on child survival and (ii) the effects of increased calorie consumption and improvements in health infrastructure on measures of children's nutritional status. The estimated health production function has child-specific calorie intake as an endogenous health input. In the estimation of a health production function, the prices of health inputs are valid instruments both for the inputs and for the survival function that controls for mortality selection. They find very little evidence of mortality selection with these data, and could not reject the hypothesis of bivariate normality. This identification strategy is only available for the estimation of structural models and not for reduced-form models of the kind most commonly applied in the literature on child health behavior. Moreover, health production function estimation requires difficult-to-obtain data on individual specific health inputs, such as food intake, which are not commonly available.



Estimating likelihood (9) with 14 DHS data sets from Sub-Saharan Africa, we were unable to identify the parameters  $\beta_h$  and  $\rho$  with sufficient confidence. The likelihoods plotted against  $\rho$  were almost flat over wide portions of  $\rho$ 's permissible range of  $\{-1, 1\}$ , although at the maximum of most likelihoods,  $\rho$  was quite distant from 0. We are not alone in finding that it is difficult to identify this bivariate probit model. Keane (1992) reports both Monte Carlo evidence and evidence using the National Longitudinal Survey of Young Men on how very difficult it is to identify multivariate probit models without exclusion or covariance restrictions. He refers to identification of multinomial probit models without restrictions as being "tenuous" or "fragile" in that the likelihood function exhibits very little variation from its maximum over a wide range of parameter values. The discrete case thus differs from the continuous case reported in Pitt and Rosenzweig (1989), in which the nonlinearity derived from assuming normally distributed errors was sufficient to identify the joint determinants of fertility and a *continuously* measured health indicator (birth weight).

#### IV. Identification with Covariance Restrictions

##### A. Random Effects Probit and the Exchangeability Property

The proposed approach to identification does not rely on arbitrary exclusion or functional form restrictions, or on the exogeneity of first-births, but rather on restrictions on error covariances that follow directly from the illustrative Model (1)–(2) set out above. The simplest form of the model is relatively easy to estimate. However, it imposes some strong restrictions on behavior. As a result, we will examine a number of related approaches that are less restrictive and extend the informational content of results in a number of interesting directions.

The restrictions on the error terms attached to the fertility and health reduced forms (Equations (1) and (2) above) at the time these equations were introduced are sufficient for identification as long as fertility and health outcomes are observed for more than one time period in the life of each woman in the sample. To elaborate, Equations (1) and (2) are rewritten below with time subscripts:

$$(10) \quad F_{it}^* = X_{fit}\beta_f + \mu_{fi} + v_{fit}$$

$$(11) \quad D_{it}^* = X_{hit}\beta_h + \mu_{hi} + v_{hit},$$

where the observed  $h$  of Equation 2 is replaced by  $D_{it}^*$ , latent mortality, corresponding to a binary realization  $D_{it} = 1$  if the infant died and  $D_{it} = 0$  if it survived.<sup>8</sup> Under the assumptions that  $E(v_{fit}, v_{hit}) = 0$ ,  $E(v_{fit}, v_{fit'}) = 0$  and  $E(v_{hit}, v_{hit'}) = 0$ , we can write the joint likelihood of births and deaths for  $T$  periods in the reproductive life of a woman as

8. The  $t$  subscript here refers to the date of the child's birth when attached to mortality, so that  $D_{it}$  is a binary indicator of whether the child of woman  $i$  born in period  $t$  is still alive at some fixed time since birth.

$$\begin{aligned}
(12) \quad & \text{Prob}(F_{i1}, F_{i2}, \dots, F_{iT}, D_{i1}, D_{i2}, \dots, D_{iT}) \\
&= \int_{-\infty}^{\infty} \int_{-\infty}^{\infty} \left[ \prod_{t=1}^T \Phi \left[ \left( \frac{X_{fit} \beta_f}{\sigma_{v_f}} + \left( \frac{\rho_f^2}{(1 - \rho_f^2)} \right)^{1/2} \bar{\mu}_{fi} \right) I_{fit} \right] \right. \\
&\quad \times \left. \prod_{t=1}^T \Phi \left[ \left( \frac{X_{hit} \beta_h}{\sigma_{v_h}} + \left( \frac{\rho_h^2}{(1 - \rho_h^2)} \right)^{1/2} \bar{\mu}_{hi} \right) I_{hit} \right] \right]^{F_{it}} \\
&\quad \times \phi_2(\bar{\mu}_{fi}, \bar{\mu}_{hi}, \rho)^{F_{it}} \times \phi_1(\bar{\mu}_{fi})^{1-F_{it}} d\bar{\mu}_{fi} d\bar{\mu}_{hi},
\end{aligned}$$

where  $\bar{\mu}_{hi} = \mu_{hi}/\sigma_{\mu_h}$ ,  $\bar{\mu}_{fi} = \mu_{fi}/\sigma_{\mu_f}$ ,  $I_{fit} = 1$  if  $F_{it} = 1$ ,  $I_{fit} = -1$  if  $F_{it} = 0$ ,  $I_{hit} = 1$  if  $D_{it} = 1$ ,  $I_{hit} = -1$  if  $D_{it} = 0$ , and where

$$\rho_f = \left( \frac{\sigma_{\mu_f}^2}{\sigma_{\mu_f}^2 + \sigma_{v_f}^2} \right)^{1/2} \quad \text{and} \quad \rho_h = \left( \frac{\sigma_{\mu_h}^2}{\sigma_{\mu_h}^2 + \sigma_{v_h}^2} \right)^{1/2}.$$

The simplification of the  $2T$ -variate probability to the form on the right-hand side of (12) results from integrating out the women-specific effects  $\mu_{fi}$  and  $\mu_{hi}$ , the only sources of correlation between the sequences of birth and death/survival events. Numerically evaluating the integrals in (12) is accomplished with fast and highly accurate Gauss-Hermite integration.<sup>9</sup>

The likelihood (12) is referred to as the random-effects bivariate probit model. The random-effects probit model was introduced into the economics literature by Heckman (1980), and the use of Gauss-Hermite quadrature to evaluate the likelihood was popularized by Butler and Moffitt (1982). The use of this model below differs from other uses in economics (Moffitt 1984) in that here the covariance restrictions of the model are used to identify an otherwise numerically unidentified model.

What restrictions on error correlations are imposed by this model and how realistic are they? Associating all correlation with a single factor ( $\mu$ ) is sufficient to yield an error correlation matrix with equicorrelation, a special case of the exchangeability property. In the case of a single set of random variables  $F_{i1}$ ,  $F_{i2}$ ,  $\dots$ ,  $F_{iT}$ , equicorrelation restricts the correlation between *any* pair of the sequence to be equal. The sequence can be shuffled or exchanged in any way, and the  $T \times T$  correlation matrix will consist of 1's on the diagonal and  $\rho_f$  in the off-diagonal. If  $F_{it}$  is fertility in time  $t$ ,  $\mu_{if}$  represents unobserved fecundity, health, and other factors that are specific to the  $i$ th woman and time invariant. Life-cycle changes in fecundity (and other factors) can be captured by introducing age effects.

In the case of the sequences of two random variables  $F_{i1}$ ,  $F_{i2}$ ,  $\dots$ ,  $F_{iT}$ , and  $D_{i1}$ ,  $D_{i2}$ ,  $\dots$ ,  $D_{iT}$ , the model imposes exchangeability within each sequence and, since the shocks  $v_{fit}$  and  $v_{hit}$  are assumed to be uncorrelated, the only source of

9. The assumption of normality for the woman-specific effects  $\mu$  in the random effects probit model (12) is not necessary for identification. This model is identified with a nonparametric distribution.

correlation between the sequences arises from the constant correlation  $\rho$  between the fertility factor  $\mu_{fi}$  and the mortality factor  $\mu_{hi}$ . The restrictions on the error are not unlike those of the standard fixed effects model with *woman-specific* effects. The equicorrelated model is substantially over-identified in that there are  $2T(2T - 1)/2 - 1$  restrictions imposed, leaving room for less restrictive specifications, some of which are suggested below.

The source of the error correlation between the fertility and mortality errors, the term  $\mu_{fi}$  and  $\mu_{hi}$ , may represent other effects, of which inherent healthiness is only one. All omitted time invariant variables will affect both fertility and subsequent mortality and health, and thus indicate selection. These might include variables related to permanent income, which is unobserved in the data used in the estimation reported below. It is likely that the richer the data set used in the estimation of the econometric model described by (12), the less important any selection bias will be.<sup>10</sup>

### ***B. The Data***

The data used in the estimation come from 14 Sub-Saharan Demographic and Health Survey (DHS) data sets.<sup>11</sup> The fertility and mortality observations for each woman consist of whether she had a birth in each 12-month period beginning with her 13th birthday, and if there was a birth, whether the child survived to its 24th month. Very few women had two pregnancies that resulted in live births within these 12-month periods. When more than one birth occurred it was most often the result of a single multiple-birth pregnancy, an exogenous event.

Table 1 presents summary statistics for the sample of births used in the empirical analysis. It provides numbers of children, variable definitions, means, and standard deviations for the children born to the women in the sample. These data are used in the child mortality portion of the various likelihoods. The likelihoods also use "woman-years" in which there was no birth as data in computing the probability of a birth in every year subsequent to each woman's 13th birthday. The number of women, woman-years, and births used in the estimation for each country is presented in Table 2.

### ***C. Estimates of the Determinants of Mortality Corrected for Selective Fertility***

Table 2 presents three sets of estimates for the determinants of death before age two years for 14 Sub-Saharan African nations using data from the Demographic and Health Surveys (DHS). The first set, labeled "fertility selected binary probit," contains standard probits in which a time-invariant woman-specific ran-

10. In addition, the  $\mu$  factors can be interpreted as subsuming a hoarding effect, that is, having more births in anticipation of a positive probability of subsequent death resulting, in part, from unobserved inherent healthiness or other omitted variables.

11. There is a substantial literature describing the collection of Demographic and Health Survey data and evaluating their quality. The most useful summary is Arnold (1991). One finding of this literature is that the timing of births is often misstated, even more so for children who subsequently died. While this raises some concern about measurement error in general, it has no clear implication for assessing the impact of fertility selection in our framework.

dom effect is not included in the likelihood; these probits are estimated from the sample of all children born to the sampled women and are thus uncorrected for the potential choice-based nature of fertility. These parameters can be compared to the results of most studies that use samples of children to study the determinants of child mortality. The reported  $t$ -statistics are corrected for the non-independence of the errors associated with woman-specific random effects.<sup>12</sup> The middle set of estimates, labeled “fertility-selected random effects binary probit,” use these same data to estimate a random effects (univariate) probit model of mortality, still uncorrected for the potential choice-based nature of fertility. The last set of estimates, labeled “selection-corrected random effects bivariate probit,” are obtained by maximizing the bivariate random effects with partial observability likelihood (12). The parameter estimates are comparable since the (composite) error is normalized to unit variance in all models.

In all possible cases, the hypothesis that the woman-specific effects  $\mu_{fi}$  and  $\mu_{hi}$  are not correlated ( $\rho = 0$ ), that is, that there is no birth selection in the determination of mortality, is rejected. In two cases (Botswana and Togo), estimates of  $\rho$  exceeded 0.99 and point estimates could not reliably be obtained. For these two countries, the estimated model imposes  $\rho = 0.99$ . All of the estimated  $\rho$ 's are positive, indicating negative birth selection (“high fertility” women are also “high mortality” women). The range in correlation between the woman-specific effects is quite large—the largest value for  $\rho$  is 0.95 in Zimbabwe and the smallest is 0.19 in Sudan—but most of them are large—all but two are greater than or equal to 0.39. In addition, all of the  $\rho_f$ 's and  $\rho_h$ 's, which are the square root of the share of the woman-specific effect in the regression error (see Equation (12)), are all significantly different from 0. The importance of woman-specific effects in the determination of child mortality was typically larger than in the determination of fertility ( $\rho_h > \rho_f$ ).

Although the selection-corrected estimates demonstrate that the fertility and mortality woman-specific effects,  $\mu_{fi}$  and  $\mu_{hi}$ , are highly correlated, the magnitude of the error correlation seems much less when expressed as the correlation between the entire fertility error ( $\epsilon_{fit} = v_{fit} + \mu_{fi}$ ) and the entire mortality regression error ( $\epsilon_{hit} = v_{hit} + \mu_{hi}$ ). If the woman-specific effects are small compared to the nonsystematic shocks  $v_{fit}$  and  $v_{hit}$ , the correlation of the total (composite) errors will be small. In this model the remaining sources of error are maintained to be uncorrelated, an assumption that is relaxed below. Since the composite errors are normalized to have unit variance,  $\rho_f$  and  $\rho_h$  are the standard deviations of the errors  $\mu_{fi}$  and  $\mu_{hi}$ , respectively, and the correlation between  $\epsilon_{fit}$  and  $\epsilon_{hit}$  is  $\bar{\rho} = \rho_f \rho_h$ . Thus, although the  $\mu$ 's are highly correlated in Zimbabwe ( $\rho = 0.96$ ), for example, the  $\epsilon$ 's are not nearly as correlated ( $\bar{\rho} = 0.05$ ) because of the relative importance of the nonsystematic errors.

Although the statistical tests of no fertility selection ( $\rho = 0$ ) are all strongly rejected, fertility selection does not seem to importantly bias estimates of the

12. The parameter covariance matrix is essentially White's heteroskedasticity-consistent covariance matrix in which the Berndt-Hall-Hausman (BHHH) component of White's formula is altered to be the cross-product of the first derivatives of the log likelihood function defined over *all* the births that each woman contributes to the sample.

**Table 1**  
*Summary Statistics for the Sample of Births*

Variable	Botswana ( <i>n</i> = 10,564)		Burundi ( <i>n</i> = 11,783)		Cameroon ( <i>n</i> = 11,404)		Ghana ( <i>n</i> = 13,965)	
	Mean	Standard Deviation	Mean	Standard Deviation	Mean	Standard Deviation	Mean	Standard Deviation
Died before 2 years	0.0619	0.2410	0.1252	0.3310	0.1182	0.3229	0.1125	0.3159
Mother's age	24.3739	5.4763	25.6110	5.5866	23.3685	5.6755	24.4130	5.8088
Mother's age squared (/100)	6.2407	2.8730	6.8713	3.0303	5.7829	2.8048	6.2973	3.0090
Years of education	4.0207	3.8829	1.0080	2.3467	3.0541	3.6104	3.1541	4.1906
Rural	0.5574	0.4967	0.8521	0.3550	0.5124	0.4999	0.7061	0.4555
Year	76.0940	6.1847	75.3904	6.4770	77.9741	6.8931	74.8556	7.0750
Male	0.4920	0.5000	0.5113	0.4999	0.5092	0.4999	0.5132	0.4998
Multiple birth	0.0104	0.1015	0.0093	0.0958	0.0181	0.1333	0.0173	0.1303

	Kenya ( <i>n</i> = 24,876)	Mali ( <i>n</i> = 12,113)	Nigeria <i>n</i> = 27,621)	Senegal ( <i>n</i> = 14,225)
Died before 2 years	0.0778	0.1883	0.1382	0.1539
Mother's age	23.8222	24.0621	24.4566	24.2432
Mother's age squared (/100)	6.0060	6.1340	6.3401	6.2184
Years of education	3.3775	3.5859	1.8035	0.8853
Rural	0.8408	0.6055	0.6649	0.6474
Year	75.9794	74.1886	77.6728	73.6696
Male	0.5085	0.5245	0.5182	0.5144
Multiple birth	0.0113	0.0115	0.0183	0.0112
	Sudan ( <i>n</i> = 25,454)	Tanzania ( <i>n</i> = 28,691)	Togo ( <i>n</i> = 10,523)	Uganda ( <i>n</i> = 15,853)
Died before 2 years	0.1027	0.1246	0.1230	0.1171
Mother's age	23.9839	24.0769	24.7289	24.1238
Mother's age squared (/100)	6.0852	6.1399	6.4557	6.0047
Years of education	1.3433	2.2598	1.0500	2.7879
Rural	0.6297	0.8399	0.7531	0.8583
Year	76.3853	77.9189	75.4219	77.5390
Male	0.5091	0.5090	0.5043	0.4939
Multiple birth	0.0139	0.0159	0.0243	0.0180

**Table 2**

*Determinants of Death before Age Two Years: Fertility-Selected Binary Probit, Binary Random Effects Probit, and Selection-Corrected Bivariate Random Effects Probit Estimates*

	Fertility-Selected Binary Probit <sup>a</sup>		Fertility-Selected Random Effects Binary Probit		Selection-Corrected Random Effects Bivariate Probit	
	Parameter	t-statistic	Parameter	t-statistic	Parameter	t-statistic
<b>Botswana</b>						
Age in years	-0.01711	(-0.771)	-0.04070	(-1.409)	-0.03152	(-1.098)
Age squared /100	0.02906	(0.704)	0.07702	(1.410)	0.05872	(1.083)
Years of education	-0.01642	(-2.860)	-0.01643	(-2.408)	-0.01694	(-2.490)
Rural	-0.00191	(-0.045)	-0.01513	(-0.297)	-0.01572	(-0.309)
Year	-0.01782	(-6.068)	-0.01731	(-4.166)	-0.01721	(-4.136)
Male	0.13605	(3.394)	0.09789	(2.202)	0.09830	(2.221)
Multiple birth	0.70666	(4.797)	0.63122	(3.754)	0.62683	(3.773)
Constant	0.02329	(0.065)	0.28866	(0.623)	0.12226	(0.265)
$\rho$	—	—	—	—	0.99000	—
$\rho$ (fertility)	—	—	0.09671	(4.992)	0.10153	(6.655)
$\rho$ (mortality)	—	—	0.41656	(10.955)	0.42905	(11.439)
<b>Burundi</b>						
Age in years	-0.02469	(-1.367)	-0.01636	(-0.753)	0.00944	(0.430)
Age squared /100	0.02737	(0.850)	0.01612	(0.404)	-0.02148	(-0.537)
Years of education	-0.00828	(-2.177)	-0.00574	(-2.261)	-0.00669	(-2.689)
Rural	0.02420	(0.532)	0.07503	(1.311)	0.06633	(1.165)
Year	-0.00941	(-4.258)	0.00122	(0.382)	-0.00205	(-0.626)
Male	0.08436	(2.769)	0.10287	(3.031)	0.10237	(3.043)
Multiple birth	0.88133	(6.871)	0.93375	(6.576)	0.93052	(6.677)
Constant	-0.08891	(-0.339)	-1.05264	(-2.947)	-1.25824	(-3.551)
$\rho$	—	—	—	—	0.73904	(7.523)
$\rho$ (fertility)	—	—	0.18153	(13.884)	0.18332	(14.184)
$\rho$ (mortality)	—	—	0.43209	(16.374)	0.44785	(16.437)
<b>Cameroon</b>						
Age in years	-0.06934	(-4.363)	-0.07772	(-4.341)	-0.07143	(-3.995)
Age squared /100	0.10408	(3.357)	0.12518	(3.637)	0.11414	(3.328)
Years of education	-0.05715	(-10.602)	-0.05106	(-7.970)	-0.05362	(-8.398)
Rural	0.04824	(1.443)	0.08469	(1.954)	0.08681	(2.004)
Year	-0.00353	(-1.596)	-0.00241	(-0.787)	-0.00326	(-1.055)
Male	0.08838	(2.802)	0.05818	(1.700)	0.05879	(1.719)
Multiple birth	0.70815	(7.394)	0.80103	(7.413)	0.79496	(7.394)
Constant	0.15269	(0.617)	0.14582	(0.472)	0.09288	(0.301)
$\rho$	—	—	—	—	0.32501	(3.730)
$\rho$ (fertility)	—	—	0.27887	(25.037)	0.27842	(24.982)
$\rho$ (mortality)	—	—	0.41674	(14.905)	0.41953	(14.728)
<b>Ghana</b>						
Age in years	-0.07079	(-5.001)	-0.09600	(-5.657)	-0.08448	(-4.953)
Age squared /100	0.12397	(4.778)	0.16224	(5.176)	0.14393	(4.587)
Years of education	-0.01676	(-4.815)	-0.01951	(-4.016)	-0.01938	(-3.997)
Rural	0.08629	(2.631)	0.07742	(1.913)	0.08850	(2.182)
Year	-0.00613	(-3.053)	0.00042	(0.151)	-0.00072	(-0.253)
Male	0.07034	(2.467)	0.05578	(1.751)	0.05720	(1.796)
Multiple birth	0.73716	(8.655)	0.83507	(8.817)	0.82812	(8.781)
Constant	0.11343	(0.533)	0.03258	(0.113)	-0.09107	(-0.316)

Table 2 (continued)

	Fertility-Selected Binary Probit <sup>a</sup>		Fertility-Selected Random Effects Binary Probit		Selection-Corrected Random Effects Bivariate Probit	
	Parameter	<i>t</i> -statistic	Parameter	<i>t</i> -statistic	Parameter	<i>t</i> -statistic
$\rho$	—	—	—	—	0.89771	(4.283)
$\rho$ (fertility)	—	—	0.09445	(5.764)	0.09644	(6.011)
$\rho$ (mortality)	—	—	0.34651	(11.977)	0.35277	(12.078)
<b>Kenya</b>						
Age in years	-0.07921	(-6.767)	-0.08510	(-5.915)	-0.07478	(-5.177)
Age squared /100	0.13386	(5.975)	0.15003	(5.520)	0.13434	(4.933)
Years of education	-0.02118	(-3.193)	-0.01645	(-6.301)	-0.01772	(-6.813)
Rural	0.03310	(0.955)	0.06600	(1.531)	0.08463	(1.957)
Year	-0.00352	(-1.717)	-0.00508	(-2.052)	-0.00711	(-2.834)
Male	0.05145	(2.188)	0.05698	(2.106)	0.05846	(2.173)
Multiple birth	0.84538	(10.455)	0.89127	(9.533)	0.88981	(9.486)
Constant	-0.06406	(-0.332)	0.04109	(0.168)	-0.00552	(-0.023)
$\rho$	—	—	—	—	0.50946	(6.679)
$\rho$ (fertility)	—	—	0.16515	(19.343)	0.16486	(19.420)
$\rho$ (mortality)	—	—	0.47546	(23.734)	0.48429	(23.775)
<b>Mali</b>						
Age in years	-0.11889	(-9.071)	-0.14045	(-9.004)	-0.13155	(-8.335)
Age squared /100	0.19517	(7.855)	0.23326	(7.862)	0.21940	(7.333)
Years of education	-0.03219	(-4.225)	-0.03122	(-3.058)	-0.03320	(-3.270)
Rural	0.23317	(7.918)	0.22728	(5.903)	0.23481	(6.096)
Year	-0.01428	(-7.590)	-0.00572	(-2.068)	-0.00672	(-2.405)
Male	0.06274	(2.323)	0.05718	(1.906)	0.05745	(1.920)
Multiple birth	0.88450	(7.993)	0.88785	(6.802)	0.88671	(6.828)
Constant	1.64974	(8.170)	1.33130	(4.969)	1.23398	(4.596)
$\rho$	—	—	—	—	0.38635	(4.541)
$\rho$ (fertility)	—	—	0.24234	(21.184)	0.24182	(21.248)
$\rho$ (mortality)	—	—	0.40421	(18.297)	0.40978	(18.321)
<b>Nigeria</b>						
Age in years	-0.02937	(-3.165)	-0.02599	(-2.338)	-0.00697	(-0.630)
Age squared /100	0.04351	(2.450)	0.03399	(1.589)	0.00915	(0.434)
Years of education	-0.03023	(-8.940)	-0.03277	(-6.791)	-0.04376	(-9.044)
Rural	0.19476	(8.453)	0.21134	(6.471)	0.19358	(5.919)
Year	-0.01172	(-8.172)	-0.00840	(-4.153)	-0.01152	(-5.629)
Male	0.09184	(4.717)	0.08727	(4.098)	0.08553	(4.082)
Multiple birth	0.80897	(13.448)	0.82875	(12.272)	0.80247	(12.056)
Constant	0.09682	(0.649)	-0.20941	(-1.034)	-0.31919	(-1.603)
$\rho$	—	—	—	—	0.64625	(14.764)
$\rho$ (fertility)	—	—	0.26641	(35.564)	0.26632	(35.970)
$\rho$ (mortality)	—	—	0.50835	(34.819)	0.53467	(35.657)
<b>Senegal</b>						
Age in years	-0.04533	(-3.241)	-0.04893	(-2.922)	-0.04104	(-2.448)
Age squared /100	0.06886	(2.629)	0.07360	(2.302)	0.06187	(1.932)
Years of education	-0.02982	(-4.650)	-0.03646	(-4.278)	-0.03705	(-4.326)
Rural	0.23469	(7.551)	0.25463	(6.476)	0.27181	(6.928)
Year	-0.01592	(-8.568)	-0.01037	(-3.954)	-0.01170	(-4.384)
Male	0.06414	(2.432)	0.04903	(1.648)	0.04977	(1.675)
Multiple birth	0.96692	(9.611)	0.87369	(7.143)	0.87846	(7.170)
Constant	0.61796	(2.919)	0.28365	(1.029)	0.21779	(0.795)
$\rho$	—	—	—	—	0.40621	(4.351)



Table 2 (continued)

	Fertility-Selected Binary Probit <sup>a</sup>		Fertility-Selected Random Effects Binary Probit		Selection-Corrected Random Effects Bivariate Probit	
	Parameter	<i>t</i> -statistic	Parameter	<i>t</i> -statistic	Parameter	<i>t</i> -statistic
$\rho$ (fertility)	—	—	0.21400	(19.533)	0.21453	(19.667)
$\rho$ (mortality)	—	—	0.37034	(15.500)	0.37330	(15.375)
Sudan						
Age in years	-0.04589	(-4.064)	-0.04774	(-3.567)	-0.04128	(-3.029)
Age squared /100	0.07458	(3.428)	0.07406	(2.873)	0.06440	(2.471)
Years of education	-0.02082	(-3.193)	-0.02700	(-4.780)	-0.02687	(-4.761)
Rural	0.05188	(2.012)	0.05019	(1.648)	0.05492	(1.802)
Year	-0.00318	(-1.900)	0.00517	(2.289)	0.00422	(1.837)
Male	0.05568	(2.565)	0.05137	(2.114)	0.05192	(2.138)
Multiple birth	0.75121	(10.352)	0.71496	(9.151)	0.71591	(9.164)
Constant	-0.44679	(-2.644)	-1.01189	(-4.627)	-1.05992	(-4.845)
$\rho$	—	—	—	—	0.18961	(2.877)
$\rho$ (fertility)	—	—	0.28223	(36.650)	0.28202	(36.712)
$\rho$ (mortality)	—	—	0.39229	(21.438)	0.39376	(21.377)
Tanzania						
Age in years	-0.05477	(-5.806)	-0.05770	(-4.839)	-0.05122	(-4.269)
Age squared /100	0.07538	(4.282)	0.08663	(3.835)	0.07613	(3.357)
Years of education	-0.02335	(-6.834)	-0.01917	(-4.263)	-0.02102	(-4.664)
Rural	0.03696	(1.327)	0.07568	(2.174)	0.08228	(2.368)
Year	-0.00247	(-1.759)	-0.00255	(-1.345)	-0.00377	(-1.950)
Male	0.05080	(2.623)	0.05442	(2.536)	0.05428	(2.536)
Multiple birth	0.88996	(14.484)	0.89583	(12.984)	0.88995	(12.873)
Constant	-0.14694	(-0.991)	-0.16072	(-0.828)	-0.18870	(-0.973)
$\rho$	—	—	—	—	0.41081	(5.738)
$\rho$ (fertility)	—	—	0.17795	(23.635)	0.17847	(23.855)
$\rho$ (mortality)	—	—	0.38594	(21.135)	0.38749	(21.038)
Togo						
Age in years	-0.01650	(-1.006)	-0.02432	(-1.156)	-0.01182	(-0.560)
Age squared /100	0.01108	(0.366)	0.02221	(0.563)	0.00201	(0.051)
Years of education	-0.03326	(-4.283)	-0.03725	(-3.834)	-0.03760	(-3.889)
Rural	0.17395	(4.250)	0.21125	(4.145)	0.22716	(4.472)
Year	-0.01214	(-5.217)	-0.01013	(-3.092)	-0.01094	(-3.296)
Male	0.05635	(1.721)	0.06111	(1.654)	0.06309	(1.709)
Multiple birth	1.01595	(12.290)	0.94904	(10.101)	0.95004	(10.131)
Constant	-0.09592	(-0.366)	-0.15329	(-0.445)	-0.31412	(-0.912)
$\rho$	—	—	—	—	0.99000	—
$\rho$ (fertility)	—	—	0.07200	(2.943)	0.08476	(5.306)
$\rho$ (mortality)	—	—	0.33965	(10.642)	0.34506	(10.926)
Uganda						
Age in years	-0.08671	(-6.467)	-0.10041	(-6.420)	-0.09541	(-6.036)
Age squared /100	0.13662	(5.406)	0.15979	(5.287)	0.15159	(4.972)
Years of education	-0.02513	(-5.354)	-0.03077	(-4.898)	-0.03236	(-5.118)
Rural	0.03454	(0.865)	0.05618	(1.068)	0.05674	(1.077)
Year	0.00065	(0.363)	0.00791	(3.285)	0.00704	(2.907)
Male	0.08245	(3.198)	0.07139	(2.454)	0.07196	(2.476)
Multiple birth	0.90106	(10.442)	0.95649	(8.563)	0.96383	(8.643)
Constant	0.00791	(0.038)	-0.34157	(-1.362)	-0.36715	(-1.467)
$\rho$	—	—	—	—	0.24223	(2.879)
$\rho$ (fertility)	—	—	0.24397	(24.896)	0.24380	(24.869)

Table 2 (continued)

	Fertility-Selected Binary Probit <sup>a</sup>		Fertility-Selected Random Effects Binary Probit		Selection-Corrected Random Effects Bivariate Probit	
	Parameter	<i>t</i> -statistic	Parameter	<i>t</i> -statistic	Parameter	<i>t</i> -statistic
$\rho$ (mortality)	—	—	0.39632	(17.867)	0.39795	(17.638)
Zambia						
Age in years	-0.07410	(-6.579)	-0.08514	(-6.205)	-0.08000	(-5.796)
Age squared /100	0.10635	(5.008)	0.12481	(4.787)	0.11689	(4.466)
Years of education	-0.03092	(-8.763)	-0.03459	(-7.396)	-0.03588	(-7.666)
Rural	0.11436	(4.699)	0.12420	(3.956)	0.12491	(3.974)
Year	0.00693	(4.515)	0.01109	(5.271)	0.00992	(4.684)
Male	0.03599	(1.653)	0.02704	(1.092)	0.02756	(1.114)
Multiple birth	0.81212	(12.189)	0.81747	(10.469)	0.81626	(10.429)
Constant	-0.57540	(-3.248)	-0.73570	(-3.295)	-0.73770	(-3.316)
$\rho$	—	—	—	—	0.42162	(4.166)
$\rho$ (fertility)	—	—	0.14080	(13.861)	0.14052	(13.864)
$\rho$ (mortality)	—	—	0.39650	(20.281)	0.40062	(20.299)
Zimbabwe						
Age in years	-0.05568	(-3.244)	-0.03528	(-1.570)	-0.01981	(-0.885)
Age squared /100	0.09069	(2.852)	0.04759	(1.129)	0.02111	(0.503)
Years of education	-0.02664	(-4.553)	-0.02245	(-3.074)	-0.02423	(-3.318)
Rural	0.25594	(5.565)	0.29756	(5.447)	0.29615	(5.476)
Year	-0.00791	(-3.311)	-0.00345	(-1.150)	-0.00449	(-1.499)
Male	0.13743	(3.967)	0.14557	(3.888)	0.14650	(3.936)
Multiple birth	0.50110	(4.903)	0.40949	(3.493)	0.41757	(3.544)
Constant	-0.23818	(-0.931)	-0.84820	(-2.433)	-1.02788	(-2.981)
$\rho$	—	—	—	—	0.95582	(5.107)
$\rho$ (fertility)	—	—	0.17420	(14.562)	0.17462	(14.737)
$\rho$ (mortality)	—	—	0.28506	(6.642)	0.28655	(6.506)

Sample sizes:

Number of

	Number of		
	Women	Births	Woman-Years
Botswana	4,359	10,564	54,537
Burundi	3,958	11,783	53,418
Cameroon	3,862	11,404	48,525
Ghana	4,477	13,965	61,379
Kenya	7,124	24,876	94,858
Mali	3,196	12,113	46,078
Nigeria	8,741	27,621	116,179
Senegal	4,406	14,225	56,267
Sudan	5,858	25,454	94,447
Tanzania	9,201	28,691	119,479
Togo	3,348	10,523	44,162
Uganda	4,716	15,853	57,811
Zambia	7,254	22,320	87,317
Zimbabwe	4,186	12,169	54,196

a. Asymptotic bootstrap covariance matrix with woman-specific effects.

effect of women's schooling on the probability of child mortality prior to age 24 months in most of the countries studied. Table 3 provides estimates of the derivative of the *conditional* probability of infant death with respect to education for the 14 countries examined. The underestimate of this derivative is largest for Nigeria. For women with no education, the selection-corrected estimates are one-third larger than the fertility-selected estimates. Overall, the effect of education on mortality is underestimated in 11 of 14 cases. It is overestimated only in Botswana, Ghana, and Sudan.<sup>13</sup>

**D. Estimating the Determinants of a Continuous Measure of Health from Fertility- and Mortality-Selected Samples: Random Effects Trivariate Probit**

The most common dependent variable in the literature on the reduced-form determinants of the health of children is some continuously measured anthropometric measure of nutritional status—weight and height possibly adjusted for age or scaled into weight-for-height or body mass index (BMI). For a child to enter into a sample of anthropometrically measured children requires that that child be born and survive to the date of the survey, both possibly selective events. If the reduced-form equation for child health  $W_{it}$  takes on a factor structure like fertility and mortality where  $\mu_{wi}$  is the woman-specific factor associated with  $W_{it}$ , and  $v_{wit}$  is a nonsystematic shock,<sup>14</sup>

$$(13) \quad W_{it} = X_{wit}\beta_w + \mu_{wi} + v_{wit},$$

the random effects probit likelihood corresponding to this problem is

$$(14) \quad \text{Prob}(F_{i1}, F_{i2}, \dots, F_{iT}, D_{i1}, D_{i2}, \dots, D_{iT}, W_{i1}, W_{i2}, \dots, W_{iT}) \\ = \int_{-\infty}^{\infty} \int_{-\infty}^{\infty} \int_{-\infty}^{\infty} \left[ \prod_{t=1}^T \Phi \left[ \left( \frac{X_{fit}\beta_f}{\sigma_{v_f}} + \left( \frac{\rho_f^2}{(1-\rho_f^2)} \right)^{1/2} \tilde{\mu}_{fi} \right) I_{fit} \right] \right. \\ \times \prod_{t=1}^T \Phi \left[ \left( \frac{X_{hit}\beta_h}{\sigma_{v_h}} + \left( \frac{\rho_h^2}{(1-\rho_h^2)} \right)^{1/2} \tilde{\mu}_{hi} \right) I_{hit} \right]^{F_{it}} \\ \times \prod_{t=1}^T \Phi \left[ \frac{W_{it} - X_{wit}\beta_w}{\sigma_{v_w}} - \left( \frac{\rho_w^2}{(1-\rho_w^2)} \right)^{1/2} \tilde{\mu}_{wi} \right]^{F_{it}(1-D_{it})} \\ \times \phi(\tilde{\mu}_{fi})^{1-F_{it}} \times \phi_2(\tilde{\mu}_{fi}, \tilde{\mu}_{hi}, \rho_{fh})^{F_{it}D_{it}} \\ \left. \times \phi_3(\tilde{\mu}_{fi}, \tilde{\mu}_{hi}, \tilde{\mu}_{wi}, \rho_{fh}, \rho_{fw}, \rho_{hw}) \right]^{F_{it}(1-D_{it})} d\tilde{\mu}_{fi} d\tilde{\mu}_{hi} d\tilde{\mu}_{wi},$$

13. In addition, in 11 of 14 countries the age effect is overestimated when fertility selection is ignored, and some of the relative magnitudes are quite large. The differences are particularly striking for Burundi and Nigeria.

14. Measures of health  $W_{it}$  can be observed at more than one point in time for the same child. The time subscript  $t$  in what follows represents the anthropometric status at the date of a survey of the child of woman  $i$  born in period  $t$ . It could alternatively represent the health status of a single child of woman  $i$  as measured at more than one point in time, indexed by  $t$ . For multiple children subject to multiple measurements, an additional subscript is required.

**Table 3**

*Derivative of the Conditional Probability of Infant Death with Respect to Education*

	Fertility-Selected	Selection-Corrected	Proportion of Infants Born to Mothers with Two or Fewer Years of Schooling Who Do Not Survive Two Years
Botswana	-0.00203	-0.00202	6.78
Burundi	-0.00119	-0.00139	11.99
Cameroon	-0.01169	-0.01224	15.15
Ghana	-0.00402	-0.00396	12.08
Kenya	-0.00253	-0.00274	9.95
Mali	-0.00861	-0.00908	19.27
Nigeria	-0.00715	-0.00958	14.77
Senegal	-0.00856	-0.00866	15.34
Sudan	-0.00506	-0.00498	10.65
Tanzania	-0.00411	-0.00444	13.13
Togo	-0.00731	-0.00734	12.23
Uganda	-0.00706	-0.00744	13.43
Zambia	-0.00829	-0.00860	14.89
Zimbabwe	-0.00348	-0.00370	9.40

where  $\phi_3(\cdot)$  is the normal trivariate density function. Estimation of the likelihood (14) involves numerical evaluation of three integrals which is computationally burdensome even on high-speed workstations.

The determinants of child weight and height corrected for the possibly selective effects of fertility and mortality were estimated with data from the Zambian DHS data set. The Zambian DHS data set provides measures of the weight and height of all children born to surveyed women in the five years prior to the survey date. Note that identification of the variance of  $\mu_w$  ( $\rho_w^2$ ) requires that there be at least two "periods" of observations for every woman. With partial observability (selection), we must have anthropometric measures for at least two surviving children for some of the sampled women and not just two years (potential birth cohorts) of fertility and mortality outcomes. In a high-fertility environment such as Zambia, there are many women with more than two surviving children born in a five-year period.

Table 4 provides estimates of the determinants of both log weight (in 100 grams) and log height (in centimeters) estimated by ordinary least squares, as is typical in most studies, and, alternatively, selection-corrected estimates obtained by maximizing the trivariate random effects probit likelihood (14). The former estimates, which ignore both fertility and mortality selection, are labeled "selected"

**Table 4**

*Determinants of Child Anthropometrics in Zambia (asymptotic t-statistics in parentheses)*

	Log Weight		Log Height	
	Selected <sup>a</sup>	Selection-Corrected	Selected <sup>a</sup>	Selection-Corrected
Age in years	0.00298 (1.064)	0.00297 (1.013)	0.00069 (0.495)	0.00076 (0.611)
Age squared /100	-0.00296 (-0.608)	-0.00282 (-0.553)	0.00006 (0.024)	-0.00006 (-0.027)
Years of education	0.00413 (5.062)	0.00406 (4.640)	0.00309 (7.208)	0.00317 (7.309)
Rural	-0.01825 (-3.170)	-0.01774 (-2.907)	-0.00920 (-3.319)	-0.00923 (-3.298)
Child age (months)	0.02453 (12.100)	0.02449 (12.503)	0.00897 (8.532)	0.00918 (10.420)
Male	0.02973 (5.743)	0.02964 (5.838)	0.00381 (1.483)	0.00301 (1.191)
Multiple birth	-0.07029 (-3.437)	-0.07075 (-3.431)	-0.03077 (-3.164)	-0.03203 (-3.465)
Child age squared /100	-0.01556 (-6.314)	-0.01533 (-6.453)	-0.00271 (-2.160)	-0.00296 (-2.811)
Constant	4.03621 (75.242)	4.03363 (75.444)	6.44170 (228.262)	6.43595 (326.743)
$\rho$ (fertility, weight/height)		-0.12881 (-0.084)		-0.12359 (-4.490)
$\rho$ (mortality, weight/height)		-0.00953 (-0.100)		-0.39175 (-14.337)
$\rho$ (fertility)		0.15628 (4.875)		0.15615 (18.368)
$\rho$ (mortality)		0.42272 (21.004)		0.42281 (26.312)
$\rho$ (weight/height)		0.59340 (16.938)		0.31803 (12.237)
$\sigma$ (weight/height)	0.13728 (91.838)	0.11024 (32.927)	0.06594 (176.954)	0.06265 (104.485)

Note: Number of children weighed and measured: 4,202.

a. Fertility- and mortality-selected asymptotic bootstrap covariance matrix with woman-specific effects.

in the table and the latter estimates are labeled “selection-corrected.” Notable in Table 4 is the lack of evidence of fertility or mortality selection in the determinants of log child weight. Both  $\rho_{fw}$  and  $\rho_{hw}$  are not significantly different from 0. However, there is evidence of fertility and mortality selection in the determinants of log child height, as both  $\rho_{fw}$  and  $\rho_{hw}$  are both negative and statistically different from 0. The implication is that there is negative birth selection (“high-fertility women have low-height children”) and negative mortality selection with respect to child height (“women whose children are more likely to die prior to age two years are more likely to have low-height children”). The estimated  $\rho_w$ , which measures the relative importance of woman-specific effects in the residual of the log height equation, is larger than  $\rho_f$  but smaller than  $\rho_h$ . Nonetheless, there does not seem to be much difference between the fertility- and mortality-selected parameters for height and the selection-corrected parameters. The bias imparted by fertility and mortality selection on anthropometric measures of child nutritional status is apparently small in this case.

#### *E. Variable Correlations of Woman-Specific Effects*

It is possible to eliminate the equicorrelation restriction and still specify structures for the error correlation matrix which preserve the relative computational simplicity of likelihoods such as (12). In particular, a  $T$ -fold joint probability can be represented by an expression with a single integral if the correlation between periods  $t$  and  $t'$ ,  $\rho(t, t')$  can be written as (Tong 1990)

$$(15) \quad \rho(t, t') = \lambda_t \lambda_{t'}, 0 < \lambda_t < 1 \quad \text{for all } t.$$

This formulation permits the share of variances ( $\rho_f^2$  and  $\rho_h^2$ ) of the (composite) error ( $\varepsilon_{fit}$  and  $\varepsilon_{hit}$ ) that is time-persistent to vary across periods. This flexibility is desirable if the importance of women’s endowments  $\mu$  change over their reproductive life-cycles. The parameters  $\lambda$  or  $\rho$  can be made parametric or nonparametric functions of any observed exogenous attribute—calendar time, woman’s age, schooling, race or ethnicity, and season.

To implement this relaxation of equicorrelation, the parameters  $\rho_f^2$  and  $\rho_h^2$  are replaced with  $\lambda_{tf}^2$  and  $\lambda_{th}^2$ , respectively, in likelihood (12). In allowing the correlation between  $\mu_{fi}$  and  $\mu_{hi}$  to vary with “time” we adopt two time-based orderings of the data. One ordering is by calendar time. The number of  $\lambda$  parameters can be quite large if there are different values for each time or age period. Two  $\lambda$  parameters,  $\lambda_{tf}$  and  $\lambda_{th}$ , need to be estimated for each period. In order to have a sufficient number of observations in each calendar year (and to save on computation), calendar time is grouped into eight categories for fertility and four categories for mortality. The latter time disaggregation is less fine because only actual births contribute to the mortality part of the likelihood while every year contributes to the fertility part of the likelihood (partial observability). The second time ordering is by woman’s age. Again, eight age categories for fertility and four age categories for mortality were chosen. Although it seems sensible to suppose that, at least in the case of woman’s age, the  $\lambda$  coefficients would exhibit smoothness, no restrictions were placed on the distribution of the  $\lambda$ ’s over “time” in the results reported.

Table 5 reports parameter estimates obtained by maximizing likelihood (12)

**Table 5**  
*Determinants of Death before Age Two Years: Selection-Corrected Bivariate Random Effects Probit Estimates with Fixed Error Correlation, Error Correlation Varying with Age and with Error Correlation Varying with Time*

	Selection-Corrected Random Effects Bivariate Probit ( $\rho$ fixed)		Selection-Corrected Random Effects Bivariate Probit ( $\rho$ varies with age)		Selection-Corrected Random Effects Bivariate Probit ( $\rho$ varies with time)	
	Parameter	t-statistic	Parameter	t-statistic	Parameter	t-statistic
Cameroon						
Age in years	-0.07143	(-3.995)	-0.07904	(-5.833)	-0.07181	(-3.976)
Age squared /100	0.11414	(3.328)	0.12383	(4.962)	0.11587	(3.347)
Years of education	-0.05362	(-8.398)	-0.05309	(-8.321)	-0.05342	(-8.233)
Rural	0.08681	(2.004)	0.08798	(2.032)	0.09067	(2.057)
Year	-0.00326	(-1.055)	-0.00227	(-0.970)	-0.00311	(-0.991)
Male	0.05879	(1.719)	0.05811	(1.706)	0.05894	(1.717)
Multiple birth	0.79496	(7.394)	0.79776	(7.385)	0.79541	(7.370)
Constant	0.09288	(0.301)	0.14563	(3.958)	0.07184	(0.232)
$\rho$	0.32501	(3.730)	0.26920	(2.943)	0.34006	(4.052)
$\rho$ (fertility)	0.27842	(24.982)				
$\rho$ (mortality)	0.41953	(14.728)				
$\lambda_f$ age: 13-16			0.18646	(7.293)	0.22828	(6.660)
$\lambda_f$ age: 17-20	time: pre-1964		0.26062	(9.207)	0.34131	(9.996)
$\lambda_f$ age: 21-24	time: 1965-69		0.30341	(9.637)	0.32118	(9.369)
$\lambda_f$ age: 25-28	time: 1970-73		0.36354	(9.724)	0.35925	(11.929)
$\lambda_f$ age: 29-32	time: 1974-77		0.39119	(8.145)	0.36866	(8.677)
$\lambda_f$ age: 33-36	time: 1978-79		0.44093	(6.798)	0.32121	(7.597)
$\lambda_f$ age: 37-40	time: 1980-81		0.27643	(2.786)	0.28657	(7.570)
$\lambda_f$ age: 41+	time: 1982-83		0.16889	(0.406)	0.13357	(4.513)

$\lambda_d$ age: 13–20	time: pre-1969	0.29831	(5.205)	0.41199	(5.536)
$\lambda_d$ age: 21–28	time: 1970–77	0.48809	(13.193)	0.43521	(8.373)
$\lambda_d$ age: 29–36	time: 1978–82	0.45389	(5.615)	0.44266	(7.943)
$\lambda_d$ age: 37 +	time: 1983 +	0.52821	(4.044)	0.39339	(6.283)
Log likelihood		–24,338.60			
Uganda		–24,310.14			
Age in years		–0.12987	(–10.684)	–0.10033	(–6.381)
Age squared /100		0.21903	(9.739)	0.16055	(5.298)
Years of education		–0.03127	(–4.945)	–0.03128	(–4.952)
Rural		0.03159	(0.607)	0.05022	(0.945)
Year		0.00299	(1.433)	0.00799	(3.170)
Male		0.06652	(2.280)	0.07119	(2.445)
Multiple birth		0.95728	(8.492)	0.96606	(8.601)
Constant		0.38390	(11.685)	–0.37379	(–1.467)
$\rho$		0.19512	(2.390)	0.15343	(2.017)
$\rho$ (fertility)					
$\rho$ (mortality)					
$\lambda_f$ age: 13–16	time: pre-1964	0.23703	(9.989)	0.16932	(7.069)
$\lambda_f$ age: 17–20	time: 1965–69	0.19380	(7.546)	0.26261	(9.312)
$\lambda_f$ age: 21–24	time: 1970–73	0.21812	(6.908)	0.29458	(9.732)
$\lambda_f$ age: 25–28	time: 1974–77	0.24712	(6.686)	0.34125	(12.528)
$\lambda_f$ age: 29–32	time: 1978–79	0.32415	(7.189)	0.33513	(9.166)
$\lambda_f$ age: 33–36	time: 1980–81	0.31313	(5.759)	0.28934	(8.437)
$\lambda_f$ age: 37–40	time: 1982–83	0.22245	(2.310)	0.26882	(7.601)
$\lambda_f$ age: 41 +	time: 1984 +	0.22763	(0.643)	0.00000	(0.000)
$\lambda_d$ age: 13–20	time: pre-1969	0.41053	(10.599)	0.46518	(11.475)
$\lambda_d$ age: 21–28	time: 1970–77	0.42623	(10.550)	0.40292	(9.052)
$\lambda_d$ age: 29–36	time: 1978–82	0.37253	(7.296)	0.35903	(7.463)
$\lambda_d$ age: 37 +	time: 1983 +	0.01589	(0.101)	0.39769	(4.480)
Log likelihood		–31,694.54			
		–31,679.47			
		–31,623.16			



with time-varying woman-specific effects for both calendar time and women's age groupings, as well as the basic selection-corrected random effects bivariate probit model as a basis of comparison. The restrictions  $\lambda_{ft} = \lambda_{ft'}$  and  $\lambda_{ht} = \lambda_{ht'}$  are rejected for age and calendar time ordered effects in both countries. For the case of age-varying  $\lambda$ 's, the likelihood ratio test statistic for Cameroon is  $\chi^2(10) = 56.92$  and for Uganda  $\chi^2(10) = 30.14$ . For the case of calendar-time-varying  $\lambda$ 's, the test statistic for Cameroon is  $\chi^2(10) = 89.92$  and for Uganda  $\chi^2(10) = 142.76$ . In both countries,  $\lambda$ 's varying with calendar time improves the log likelihood more than having  $\lambda$ 's varying with woman's age.

Not surprisingly, the parameters on the age, age squared, and time trend ("year") regressor are substantially altered when the correlation of woman-specific effects is allowed to vary with age or calendar time compared to when the correlation is fixed. For example, when  $\lambda$ 's vary by calendar time, the time trend parameter is nearly triple its magnitude than when  $\lambda$ 's vary with woman's age, but are almost identical to the random effects bivariate probit (equicorrelation) estimate. The estimated correlation between  $\mu_{ft}$  and  $\mu_{ht}$  falls in both runs, particularly with calendar-time-varying  $\lambda$ 's. The derivatives of the conditional probability of child death with respect to woman's schooling attainment do not differ much between equicorrelation and varying correlation.

#### ***F. Relaxing Exchangeability: Cohort-Specific Effects***

The restriction  $E(v_{fit}, v_{hit}) = 0$  in the bivariate model given by Equations (10) and (11) and the likelihood (12) can be relaxed while still leaving intact a variant of exchangeability. The exchangeability property in (12) is that the  $T \times T$  error correlation matrix for fertility outcomes is invariant to the ordering of time periods, as is the  $T \times T$  error correlation matrix for mortality. Define *bivariate exchangeability* as the restriction that both error correlation matrices are invariant to the order of time periods when the two errors have a fixed structure, pairing elements of the two error vectors. This property allows for cohort-specific shocks  $v_{fit}$  and  $v_{hit}$ , as well as other possible pairings of shocks, to be correlated; that is,  $E(v_{fit}, v_{hit}) \neq 0$  or  $E(v_{fit}, v_{hit'}) \neq 0$ . In the context of our problem, cohort-specific shocks allow for fertility shocks to affect the subsequent mortality probability or health of any child born in the period of the shock. Consider a fertility shock resulting from a negative (idiosyncratic) income shock, perhaps resulting from the loss of the household's crop or livestock in a way that lowers consumption (nutritional status) to a level at which fecundity temporarily falls and the parents are aware that any child born while the mother is so poorly nourished is less likely to be healthy. This shock reduces the probability of a birth in the period of the shock but might also *increase* the probability that any child born in that period will more likely suffer mortality in infancy as a result of the poor nutritional status of the mother and the reduced level of all resources available to the household. This scenario suggests that  $E(v_{fit}, v_{hit}) < 0$ ; that is, negative cohort-specific correlation. As it seems likely that the (permanent) woman-specific effects  $\mu_{fi}$  and  $\mu_{hi}$  are *positively* correlated, that is, "high fertility" women are also "high mortality" women, the error correlations for the two error components may be

of opposite sign and perhaps selection bias resulting from woman-specific effects is ameliorated or eliminated by the correlation of cohort-specific effects.

There is an alternative scenario that is perhaps more appropriate for developed nations such as the United States. In this scenario, the shock  $v_{fit}$  represents an unexpected failure of fertility planning, such as contraceptive failure. The result is an “unwanted” birth. If parents provide less care to unwanted children, then we might expect  $E(v_{fit}, v_{hit}) > 0$ ; that is, the child is more likely to not survive or more likely to have low birth weight or other measure of health status.

The likelihood for the bivariate fertility-mortality problem with the less restrictive bivariate exchangeability property is

$$\begin{aligned}
 (16) \quad & \text{Prob}(F_{i1}, F_{i2}, \dots, F_{iT}, D_{i1}, D_{i2}, \dots, D_{iT}) \\
 &= \int_{-\infty}^{\infty} \int_{-\infty}^{\infty} \left[ \prod_{t=1}^T \Phi_2 \left[ \left( \frac{X_{fit} \beta_f}{\sigma_{v_f}} + \left( \frac{\rho_f^2}{(1 - \rho_f^2)} \right)^{1/2} \tilde{\mu}_{fi} \right) I_{fit}, \right. \right. \\
 &\quad \left. \left( \frac{X_{hit} \beta_h}{\sigma_{v_h}} + \left( \frac{\rho_h^2}{(1 - \rho_h^2)} \right)^{1/2} \tilde{\mu}_{hi} \right) I_{hit}, \rho_{v_{fh}} I_{fit} I_{hit} \right]^{F_{it}} \\
 &\quad \times \prod_{t=1}^T \Phi \left[ \left( \frac{X_{fit} \beta_f}{\sigma_{v_f}} + \left( \frac{\rho_f^2}{(1 - \rho_f^2)} \right)^{1/2} \tilde{\mu}_{fi} \right) I_{fit} \right]^{1-F_{it}} \\
 &\quad \times \phi_2(\tilde{\mu}_{fi}, \tilde{\mu}_{hi}, \rho) \phi(\tilde{\mu}_{fi})^{1-F_{it}} \Big] d\tilde{\mu}_{fi} d\tilde{\mu}_{hi}.
 \end{aligned}$$

The model of a continuous (anthropometric) measure of health  $W_{it}$  selected by prior fertility and mortality outcomes given by Equations (10), (11), and (13) can also be generalized by assuming the less restrictive trivariate exchangeability property, analogous to the bivariate exchangeability property described above.

The bivariate and trivariate exchangeability properties hold not just when  $v_{fit}$  and  $v_{hit}$  are allowed to be correlated, but for any ordered pairing  $v_{fit}$  and  $v_{hit'}$ ,  $t' = t + j$ . For  $j = 1$ , we have last period's fertility shock affecting the health outcomes of the next period's birth cohort. If  $j = -1$ , health shocks to children born in year  $t - 1$  affect fertility behavior in year  $t$ . Note that exchangeability requires that if  $v_{fit}$  is correlated with  $v_{hit+k}$ , no other correlation among the  $v_{fit}$ ,  $v_{fit'}$ ,  $v_{hit}$ , and  $v_{hit'}$  is allowed. Modeling the transmission of shocks in order to maintain the exchangeability property is obviously quite limiting. Computational ease is its great appeal.<sup>15</sup> Shocks to fertility, mortality, and health may be transmitted across behavior and time periods. In that case, the individual birth and mortality events are functions of all previous shocks to the system, and the integration over the iid shocks no longer takes the form of products of normal

15. Essentially, the likelihood of the bivariate probit model with partial observability lies within the integrals of likelihood (16). It is only the nonlinearity of the normal distribution that identifies the cohort-specific correlations. However, unlike the case of the bivariate probit with partial observability, these likelihoods are not flat with respect to the cohort-specific correlation coefficients. They seem to be well identified.

distribution functions but requires numerical integration over the  $2T$  random variables. For  $T$  larger than 2 or 3, numerical integration of this type becomes difficult. In addition, the random effects need to be integrated out as well.

Table 6 provides estimates of the joint fertility-mortality model for three countries (Mali, Sudan, and Zambia) which permit cohort-specific errors to be correlated (bivariate exchangeability), adding an additional source of selection to the woman-specific effects already modeled. Table 6 provides a comparison of both the random effects bivariate probit model estimates presented in Table 2 with the bivariate model allowing for both woman-specific and cohort-specific effects and given by likelihood (16).

Cohort-specific effects are statistically significant and negative in both Mali and Sudan, and statistically significant and positive in Zambia. The positive correlation coefficient suggests that births arising in high-fertility periods have lower mortality probabilities than births arising in low-fertility periods. As noted above, this is consistent with positive income shocks (or maternal health shocks) inducing both greater fertility and a higher probability of infant survival. Obviously, this is not the case in Zambia, where the contraceptive-failure/unwanted birth scenario may underlie the negative cohort-specific correlation.

The parameters associated with regressors do not change much with the introduction of cohort correlation. Notably, the magnitude of the positive correlation between woman-specific fertility effects  $\mu_f$  and woman-specific mortality effects  $\mu_h$  falls when cohort correlation is introduced.

## V. Concluding Remarks

This paper has estimated the determinants of child mortality corrected for the selectivity of fertility, and the determinants of anthropometrically measured child health corrected for the selectivity of both fertility and mortality, in 14 Sub-Saharan countries for which Demographic and Health Survey (DHS) data are available. It differs from the usual approach of estimating reduced-form equations of child mortality from samples of children by allowing for the possibility that such samples are choice-based, reflecting prior selective fertility and mortality behavior. If parents care about the health outcomes of potential births, then any unobserved factors (heterogeneity) that affect those outcomes will influence fertility decisions. Changes in exogenous variables, including household attributes such as women's schooling and program interventions, may thus affect the survival outcomes of those born by (1) altering the composition of households, classified by inherent healthiness, who bear a child in any time period, and (2) directly altering the survival probabilities of those selected to be born. Furthermore, these exogenous variables affect the observed health of those born and surviving to the survey by altering the composition of those who survive. Fertility selection was found in all 14 data sets studied, and resulted in the underestimation of the effect of women's schooling in reducing child mortality in 11 of the 14 countries. However, this misestimation was not found to be large. Perhaps this is not surprising in a high-fertility environment like Sub-Saharan Africa.

The issue of identification of the empirical model complicates estimation that

**Table 6**

*Determinants of Death before Age Two Years: Selection-Corrected Bivariate Random Effects Probit Estimates with and without Correlated Cohort-Specific Effects*

	Selection-Corrected Random Effects Bivariate Probit		Selection-Corrected Random Effects Bivariate Probit with Correlated Cohort-Specific Effects	
Mali				
Age in years	-0.13155	(-8.335)	-0.13197	(-8.452)
Age squared /100	0.21940	(7.333)	0.21589	(7.248)
Years of education	-0.03320	(-3.270)	-0.03021	(-2.995)
Rural	0.23481	(6.096)	0.23730	(6.277)
Year	-0.00672	(-2.405)	-0.00402	(-1.383)
Male	0.05745	(1.920)	0.05844	(1.979)
Multiple birth	0.88671	(6.828)	0.86465	(6.740)
Constant	1.23398	(4.596)	1.37438	(5.204)
$\rho$	0.38635	(4.541)	0.31353	(3.241)
$\rho$ (fertility)	0.24182	(21.248)	0.24186	(21.248)
$\rho$ (mortality)	0.40978	(18.321)	0.38847	(15.718)
$\rho$ (cohort)			-0.24373	(-3.193)
Sudan				
Age in years	-0.04128	(-3.029)	-0.04348	(-3.822)
Age squared /100	0.06440	(2.471)	0.06489	(3.044)
Years of education	-0.02687	(-4.761)	-0.02476	(-4.356)
Rural	0.05492	(1.802)	0.04949	(1.653)
Year	0.00422	(1.837)	0.00631	(2.924)
Male	0.05192	(2.138)	0.05244	(2.187)
Multiple birth	0.71591	(9.164)	0.71677	(10.035)
Constant	-1.05992	(-4.845)	-0.99918	(-7.177)
$\rho$	0.18961	(2.877)	0.13542	(2.049)
$\rho$ (fertility)	0.28202	(36.712)	0.28199	(36.745)
$\rho$ (mortality)	0.39376	(21.377)	0.38669	(20.565)
$\rho$ (cohort)			-0.13870	(-2.418)
Zambia				
Age in years	-0.08000	(-5.796)	-0.07606	(-8.558)
Age squared /100	0.11689	(4.466)	0.11329	(7.151)
Years of education	-0.03588	(-7.666)	-0.03624	(-8.100)
Rural	0.12491	(3.974)	0.12384	(4.645)
Year	0.00992	(4.684)	0.00823	(4.669)
Male	0.02756	(1.114)	0.02705	(1.221)
Multiple birth	0.81626	(10.429)	0.81364	(19.088)
Constant	-0.73770	(-3.316)	-0.79663	(-16.117)
$\rho$	0.42162	(4.166)	0.43387	(9.589)
$\rho$ (fertility)	0.14052	(13.864)	0.14056	(14.441)
$\rho$ (mortality)	0.40062	(20.299)	0.40307	(22.058)
$\rho$ (cohort)			0.10691	(2.654)

incorporates selective fertility and mortality. With these data, choice of a parametric distribution for the errors was insufficient to identify a bivariate probit model of fertility and mortality. In this paper, identification was achieved by tightly parameterizing the error correlation matrix for the set of fertility, mortality, and health behaviors over all discrete time periods in the reproductive lives of sampled women. In the univariate random effects probit model with exchangeability, at least two potential-birth cohorts are required to identify the share of the fertility error variance attributable to the woman-specific random effects, a measure of the serial correlation in the fertility errors. Similarly, for any measure of child human capital, including mortality, human capital measurements at more than one point in time are required. Measurements at more than one period can take the form of (i) multiple observations on the human capital of one child, (ii) one observation on each of more than one child, or (iii) both of these. There do not need to be anthropometric data on every child who was born and survived, nor does the survival outcome need to be observed for all children who were born to women so long as there is survival data on at least two children for some women in the sample. In the DHS and other data sets with a fertility and health focus, there typically are complete pregnancy histories of all women and information on the mortality/survival outcomes of all births, but anthropometric measurements are usually taken only for children born (and surviving) in the five (or fewer) years prior to the survey date. Panel data on the health outcome of children, as opposed to single dated observations on the health outcomes of a multiple number of children per woman, can equally be used in the models specified. In this case the variable  $W_{it}$ , the health measure of the child of woman  $i$  born in period  $t$ , should be re-indexed as  $W_{i\tau}$ , the health measure as of period  $\tau$  of the child of woman  $i$  born in period  $t$  ( $\tau \geq t$ ). If there are panel data of health measures for multiple children of the mother, one can specify and identify child-specific random effects as well as mother-specific random effects.

The exchangeable trivariate random effects model of fertility, mortality, and health tightly parameterizes a  $3T(3T + 1)/2$  error covariance matrix. The number of restrictions can be relaxed in a number of directions in a way that still leaves many degrees of freedom. Two such directions are specified and estimated in this paper. One allows the error correlations to vary with the age of the mother or calendar time. The other allows for a cohort-specific effect; that is, for "shocks" to the iid component of the fertility error to affect the subsequent mortality probability of any child born in that period. Both methods significantly improved the likelihoods of the models but did not alter the regression parameters in an important way. It is difficult to find less restrictive parameterizations of the error correlation matrix that still yield the computational convenience afforded by some form of a random effects model with exchangeability. For example, if mothers learn about the innate healthiness of their children gradually, this will lead to serial correlation in the time-varying error. Allowing for general forms of serial correlation in the time-varying error requires numerical integration over the  $3T$  random variables, which becomes intractable even for small  $T$ . Future work will use simulation methods to estimate models with autoregressive error structures.

One important benefit of the identification strategy set out above is that it does not rely on arbitrary exclusion restrictions or the arbitrary choice of an error

distribution. Although the random effects models specified above assume normal distributions for the random effects and the nonsystematic errors, the distributions for these random variables need not be restricted to normal, nor do they even need to be symmetric. The covariance restrictions implied by the imposition of exchangeability (and other covariance restrictions) are alone sufficient to non-parametrically identify the model of fertility and mortality selection. Future work will estimate models such as these using semi-parametric techniques.

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