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UNCERTAIN INFANT MORTALITY, LEARNING, AND LIFE-CYCLE FERTILITY*

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This article examines the links betweeninfant mortality and fertility in an environment with unobserved heterogeneity in infant mortality risk across mothers. In such an environment, replacement behavior (i.e., the fertility response to an experienced child death) might be influenced by mothers' learning about a family-specific component of infant mortality risk. I explicitly introduce learning by mothers in a dynamic stochastic model of life-cycle marital fertility, and I estimate the model's structural parameters using Malaysian panel data. The framework is used to estimate replacement rates and to correct for birth selectivity in the estimation of the relationship between infant mortality risk and "health inputs."

1. INTRODUCTION

The study of the links between infant and child mortality and fertility is useful in our efforts to understand the determinants of infant and child mortality risks, guide the design of policy interventions that seek to reduce such risks and predict the consequences of those policies for population change. In this article, I examine the links between infant mortality and fertility in an environment with heterogeneity in infant mortality risk across mothers. If fertility is correlated with components of infant mortality risk that are not observed by the researcher, a problem arises in the empirical investigation of the determinants of infant mortality risk because this correlation is a potential source of sample selection bias. Furthermore, women may also face uncertainty about an idiosyncratic component of infant mortality risks, and learning about it following the death of a child could influence subsequent fertility behavior.

In this article, I explicitly introduce learning by mothers in a dynamic stochastic model of life-cycle marital fertility behavior. The dynamic model nests a "production function" of infant survival with heterogeneity in endowments and learning about family-specific components in a nonstationary environment. I estimate the model's structural parameters using Malaysian panel data. The framework is used to obtain better estimates of fertility responses to experienced child deaths

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(replacement behavior) and to correct for birth selectivity in the estimation of the relationship between infant mortality risk and observables such as mother's education and age at birth.

The influence on fertility of infant and child mortality was first explored theoretically by economic demographers in static models of completed fertility.[2] Their goal was to explain the positive relationship between fertility and child mortality rates that was observed during the demographic transition as well as in cross-section data. However, static models abstract from the sequential nature of fertility choices. Child deaths cannot be perfectly anticipated, but many will occur when it is still possible for parents to respond by altering their fertility behavior. Ben-Porath (1976) distinguished between two types of reaction of fertility to child mortality: (1) responses to realized child deaths (replacement effects) and (2) responses to expected child mortality. As an example, consider a population of women whose goal is to have one surviving child. Suppose child mortality rates are 50% for all women, and women follow a "full replacement" strategy; that is, they have a birth and avoid any more pregnancies unless the child dies, in which case they "replace" it, and so on. If child mortality rates were to drop from 50% to 0%, the average number of births per woman would fall approximately from two to one, with no change in the average number of survivors. Conversely, suppose all women always have two births regardless of the fate of their infants. In this "zero replacement" case, the same decline in infant mortality rates would leave the average number of births unchanged; however, the average number of survivors would double. Suppose the decline in child mortality were the results of a policy intervention. If the health intervention has no effect on women's reproductive strategies, then the full impact of the program on fertility and population growth can be predicted from replacement behavior as illustrated in the example. If the reduction in expected child mortality alters women's reproductive strategies, then we have to consider the second type of responses mentioned earlier. For instance, the women's "target level" of surviving children might increase if the perception of lower mortality risk reduces the expected cost of building a family of any given size. The net effect of lower child mortality rates on fertility would be more ambiguous.

A wide range of replacement rates has been found in empirical studies.[3] Olsen (1980, 1983) and Mauskopf and Wallace (1984) estimated regressions of total births on total child deaths per woman, controlling for the feedback from births to deaths. Using data from Brazil, Mauskopf and Wallace obtained replacement rate estimates ranging from 0.44 to 0.98, increasing with the mother's years of schooling. Olsen obtained replacement rates around 0.2 for both Colombia (1980) and Malaysia (1983). The methods and data these authors used do not fully capture the dynamic nature of replacement behavior, which calls for a sequential framework. In other studies, equations that can be thought of as reduced form approximations of the decision rules of a dynamic model were estimated. Examples are Mroz and

[2] This section draws on Wolpin's (1997) survey of the literature on the determinants of infant and child mortality and its consequences for fertility.

[3] The replacement rate is the number of excess births induced by the death of a child.

Weir (1990) using 18th-century reconstituted French data, and Panis and Lillard (1993) with Malaysian data. The former authors obtained replacement rate estimates of 0.17, the latter of 0.3 on average. Olsen (1983) also reported results from waiting time to conception regressions, giving replacement estimates of 0.32 for Malaysian women. In Wolpin (1984), infant mortality was introduced in a sequential model with perfect fertility control. Estimation of the structural parameters was carried out on a sample of Malay women, and estimates of the parameters were used to compute replacement rates that, in contrast with previous studies, were found to be close to zero.

Several of the studies mentioned earlier did allow for, and found evidence of, unobserved heterogeneity in child health endowments. For instance, the estimates of Mauskopf and Wallace (1984) imply that the distributions of mother-specific infant mortality rates in Brazil had large coefficients of variation, even after controlling for mother's education and age. As noted earlier, if fertility is correlated with unobserved components of infant mortality risk, estimates of the determinants of infant mortality rates may suffer from selection bias. The correlation between fertility and mortality risk may arise even if there is no direct behavioral response of fertility to differences in mortality risk. For instance, if full replacement behavior is prevalent, then women with high infant mortality risk will tend to have more children. The selectivity bias can be corrected if estimates of the determinants of infant mortality rates are obtained jointly with a behavioral model of fertility.

A large literature exists on the determinants of infant and child mortality. Most of it has ignored the potential for birth-selectivity bias. Pitt and Rosenzweig (1989) used a reduced form model of fertility behavior and found evidence of birth selectivity bias in estimation of the determinants of birthweight in Malaysia. More attention has been paid to the closely related problem of correlation between endowments and "health inputs." Examples are Olsen and Wolpin (1983) and Rosenzweig and Wolpin (1995). They jointly estimate a statistical model of family investments in "child health inputs" and a "production function" of health outcomes. They consider unobserved heterogeneity in health endowments with child-specific and couple-specific components that are partially transmitted across generations. Their empirical framework nests different patterns of correlation between endowments and inputs that correspond to different assumptions about maternal behavior and the distribution of endowments. They find evidence that behavior in any birth interval varies with endowments and responds to previous birth outcomes.

In this article, I introduce learning by mothers about family-specific infant mortality risks in a model of fertility and infant mortality along the lines of Heckman and Willis (1976) and Wolpin (1984). Because heterogeneity seems to be an important feature of the environment, women at marriage may face uncertainty about their individual infant mortality risk. Their beliefs regarding the probability of infant deaths are allowed to depend on their own experience through Bayesian updating. Women in a given population may share initial beliefs that reflect common views held about their environment. As an example, suppose women have prior beliefs with high variance. This means they have a lot to learn about

infant mortality risks from the birth of their first child. If births are costly, those whose firstborn dies could be less willing to have another birth. The introduction of learning about the family-specific infant mortality rate enriches the framework for the study of replacement behavior. That learning of this kind might influence responses to experienced deaths was already suggested in Ben-Porath (1976). To my knowledge, this conjecture has not been investigated in theoretical or empirical models.

The dynamic structural approach I adopt is well suited for the investigation of learning effects.[4] Among the parameters I estimate are those that describe women's initial beliefs about the distribution of infant mortality risks in the population. Under rational expectations, this is the actual distribution of mortality risk "endowments." Observable characteristics such as the mother's education shift the mean of the distribution, and full information maximum likelihood estimates of these effects are corrected for the selectivity of births. Note that learning may induce correlation between fertility and the unobservable components of infant mortality risks and is therefore one potential source of selectivity bias. An important feature of the Malaysian data was the steep decline in aggregate infant mortality rates, observed even within the span of a single fertile life cycle. Therefore, a model with agents learning in a stationary mortality environment seemed inappropriate in this context. I obtain Bayesian updating formulae in an environment in which a woman's infant mortality risk may change over time. The problem is kept tractable by assuming that conditional on observables infant mortality risks of all women in the population shrink or grow by the same known factor. In this framework, women learn about a family-specific endowment from their own experience, but this endowment responds deterministically to exogenous trends and/or the family's choice of health inputs.

The rest of the article is organized as follows: Section 2 describes the data obtained from the two Malaysia Family Life Surveys. The model is presented in Section 3. The econometric implementation is discussed in Section 4 and results are reported in Section 5. Section 6 concludes.

2. THE DATA

The data are from the two Malaysian Family Life Surveys.[5] Both surveys contain retrospective life histories (marriages, fertility, child mortality, migration, employment) for the women under the age of 50 who are the primary respondents, as well as for their husbands when they were present in the household. In addition, the surveys include other household socioeconomic variables, and community and district level data (demographics, family planning services, health care, child care, water and sanitation, etc). The first survey (MFLS-1) was fielded in 1976. The second survey (MFLS-2) was fielded in 1988–89, and contains follow-up interviews with all the MFLS-1 women who were found, as well as additional samples. Fertility histories are available for ever-married women.

[4] Applications of the dynamic structural approach to demographic issues include Montgomery (1988), Hotz and Miller (1993), Ahn (1995), Carro and Mira (2006), and Todd and Wolpin (2003). In a different context, Miller (1984) estimated a structural job-matching model with Bayesian learning.

[5] Available from the Rand Corporation.

2.1. *Sample Screening.* Women from the three main ethnic groups present in Malaysia (Malays, Chinese, and Indian) were interviewed for the MFLSs. Because differences across ethnic groups are not the focus of my study but are likely to have a nontrivial incidence on fertility behavior and infant mortality risk, I restricted the sample to women from the largest group, Malays. When the two MFLSs were merged, there were 1977 different ever-married Malay women. Furthermore, I only included women with unbroken first marriages, with monogamous Malay husbands who were also in their first marriages. The restriction to women with unbroken marriages is natural given that I focus on marital fertility and do not model marriage decisions. I excluded women whose husbands were not in their first marriage because previous marriages might have resulted in financial (and other) obligations that are not incorporated in the model. There were 1421 Malay couples in unbroken first marriages. Additional screening criteria brought the estimation sample down to 1120 women.[6]

2.2. *Exogenous Variables.* For every woman in the sample we observe a vector of variables X_i, which, together with the age at marriage, we treat as exogenous determinants of fertility behavior and infant mortality risk. All elements of X are discretized, and each vector value of X is called a "type." Because estimation of the structural model nests repeated solutions of a dynamic programming problem for all values of X, discretization reduces the computational burden of estimation to the extent that the number of types is smaller than the number of women in the sample.

The variables in X are the woman's birth year, her education, district of residence, and her husband's income profile. Five birth cohorts were defined, corresponding to 1928–34, 1935–41, 1942–48, 1949–57, 1958–67, and labeled "31," "38," "45," "53," and "62," respectively. Aggregate infant mortality rates every 5 years starting in 1965 are available for each of the 78 districts in Peninsular Malaysia. That information was used to create two categories grouping high and low mortality districts, and women were assigned to them according to the district in which they lived when interviewed. As for the woman's education, I defined the categories "none," "primary," and "secondary," corresponding to 0 years of schooling, at least 1 but no more than 8 years of schooling, and 9 years of schooling or more, respectively. Finally, information obtained from all Malay husband's work histories was used in a regression of the log of real wage earnings on age, age squared, and a husband-specific fixed effect. The estimated fixed effects were grouped into three categories, high, medium, and low, defined by the 0.33 and 0.66 quantiles of the distribution, and each husband was assigned an income profile constructed using the mean fixed effect of his category.[7]

[6] Also excluded: (1) women who married before 1946, since modeling fertility decisions and expectations about current and future mortality during World War II would have complicated the problem considerably; (2) women with premarital births (which were rare); (3) women with missing information, such as the mother's education or the husband's marriage history; (4) women with very short fertility histories (<3 years); and (5) outliers in exogenous variables. See Mira (1995), for more details.

[7] The actual exogenous income profile for a woman after marriage also depends on the age gap between her husband and herself. Two age gap categories were defined, which combined with the husband's earnings categories gives six different income profiles.

TABLE 1
SAMPLE PROPORTIONS ACROSS CATEGORIES - I

	Mother's Birth Cohort					
	31	38	45	53	62	All
All: Sample size	33	111	190	444	342	1120
All: Proportion	.03	.10	.17	.40	.31	1.00
District						
High mortality	.45	.60	.55	.47	.53	.51
Low mortality	.55	.40	.45	.53	.47	.49
Education						
None	.58	.42	.19	.07	.04	.13
Primary	.39	.51	.66	.58	.33	.51
Secondary	.03	.06	.15	.34	.63	.36
Income						
Low	.73	.66	.45	.31	.17	.34
Medium	.18	.20	.34	.36	.38	.34
High	.09	.14	.21	.34	.45	.32

Table 1 shows two-way tabulations of sample sizes across categories. Earlier cohorts have less educated women, on average. An upward trend in income is also apparent. Income and education are positively correlated across the whole sample, and women in the high mortality districts tend to be less educated and have husbands with lower incomes (not shown in table).

2.3. *Demographic Histories.* Retrospective information on each pregnancy experienced by every woman in the sample was available in event history form. A woman's life after marriage was broken into 18-month-long periods, and live births were assigned to the period during which they occurred. An infant death was assigned to the same period as the birth of the child. Infant deaths were defined as stillbirths or the death of a child less than 12 months of age; this means a stillbirth was treated as a live birth *and* an infant death. Miscarriages were ignored.[8] When two live births occurred in the same 18-month period, the second one was shifted to the following period.[9] The period that includes the 45th birthday was taken to be the last fertile period. A woman's demographic history is incomplete if she was interviewed before age 45.[10]

[8] Although miscarriages contain information about the demand for children and spacing behavior, they are likely to be grossly underreported, even more so in retrospective histories. Since period length is 18 months and most miscarriages happen early in the pregnancy, most mothers can "replace" a miscarriage within the same period. It therefore seems that allowing for miscarriages would introduce unnecessary complexity into the model.

[9] Some 4.8% of births for women in the Estimation Sample were shifted, affecting 2.1% of period observations.

[10] Births beyond age 45 were ignored, but this is a rare event. Also, a few demographic histories in the sample were truncated. Reasons for truncation were widowhood, the birth of twins, or missing information.

TABLE 2
SUMMARY OF DEMOGRAPHIC HISTORIES

	Cohort					
	31	38	45	53	62	All
Sample size: Number of women	33	111	190	444	342	1120
Period-observations:						
Total	564	1910	2834	4102	1682	11092
Mean per woman	17.1	17.2	14.9	9.2	4.9	9.9
Mean age at marriage	17.7	17.6	18.4	19.7	19.2	19.0
Births	237	720	1066	1854	882	4759
Infant deaths	26	57	64	92	30	269
Infant mortality rate (%)	11.0	7.9	6.0	5.0	3.4	5.7
Test of homogenous Infant mortality risk:						
χ^2 statistic	9.59	5.54	8.64	15.71	6.51	46.00
Degrees of freedom	4	4	4	4	4	20
P-value	0.048	0.236	0.071	0.003	0.164	0.001

Table 2 gives a summary of demographic histories in the estimation sample, by birth cohort. Notice the upward trend in the age at marriage.[11] The downward trend in infant mortality rates is also apparent here, as in Figure 1, which shows aggregate infant mortality rates by district and calendar year.

Fertility in Peninsular Malaysia was high over the period covered in the MFLS Surveys. The average woman had almost seven births by age 45. Table 3 reports summary statistics for women observed at ages 27, 36, and 45 (complete histories) by cohort, education, income, and district categories. Fertility is higher for women with lower income and no schooling and for women living in high mortality districts.[12] The total number of births per woman shows a marked downward cohort trend. If we look at the number of surviving children per woman, instead of the number of live births, we observe the same patterns across categories.

The distributions of the total number of births and deaths per woman, for those with complete histories, are shown in Figure 2. Forty percent of women experienced at least one infant death. A key hypothesis of this study is that unobserved heterogeneity in infant mortality risks across women is important, and maximum-likelihood estimation of a parametric model of fertility behavior and infant survival in Section 5 will provide evidence in support of the hypothesis. To examine the hypothesis nonparametrically here, I computed the binomial distribution of the number of infant deaths each woman should experience, conditional on the number of births she had, if infant mortality risks were the same for all women.

[11] Women who married before 1946 or after 1985 were excluded from the estimation sample. Thus, sample screening increased the average age at marriage of women in the 1931 cohort and reduced it for those in the 1962 cohort.

[12] A notable exception is that for the subsample of women whose full fertility histories are observed, fertility is slightly higher in the low mortality districts. The same education, income, and district effects can be seen within cohorts, but sample sizes are smaller.

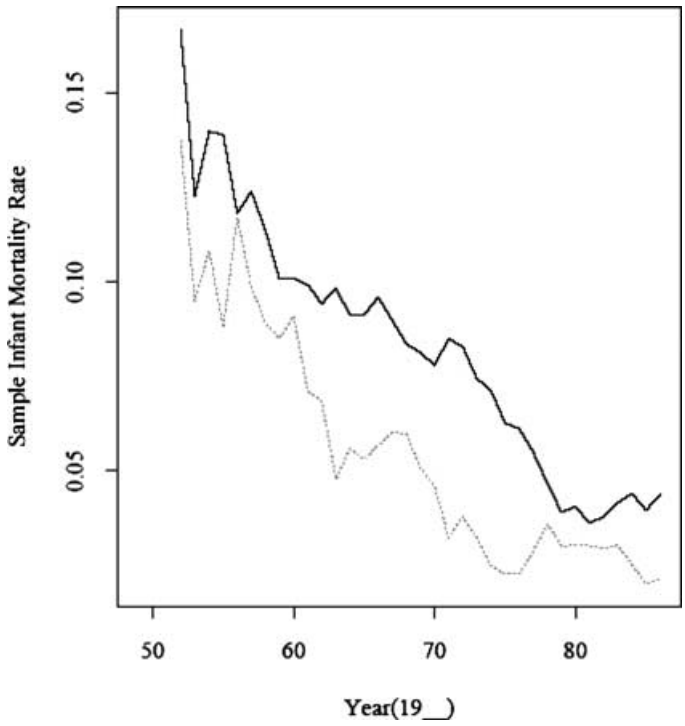

FIGURE 1

SAMPLE INFANT MORTALITY RATES, BY DISTRICT CATEGORY, 1949–86. HIGH AND LOW INFANT MORTALITY DISTRICTS, MOVING AVERAGES, 3 LAGS & 3 LEADS.

Under the assumption of homogeneity, the Bernoulli parameter common to all women in a population can be estimated quite precisely as the aggregate infant mortality rate. I estimated Bernoulli parameters conditional on the woman's birth cohort and district of residence and used sample weights to mix binomial distributions corresponding to women who had different number of births within each cohort-district type. I computed chi-squared goodness of fit statistics comparing the mixing distributions predicted under homogeneity and the actual distributions of the number of infant deaths per woman in a type.[13] Because chi-squared statistics are independent across types, they can be added to increase the power of the tests. The last rows of Table 2 report test statistics and *P*-values by cohort and for the full sample. If we consider each cohort separately, homogeneity is rejected for only two cohorts at the 5% level. However, the hypothesis that infant mortality

[13] Four outcomes are considered: 0, 1, 2, and "3 or more" infant deaths. For each cohort-district type the chi-square has two degrees of freedom. One degree of freedom has to be subtracted from the number of cells (4), and another one is lost because the type-specific Bernoulli parameter is estimated from the data.

TABLE 3
AVERAGE BIRTHS AND SURVIVING CHILDREN PER WOMAN, BY AGE AND CATEGORIES

	Age 27			Age 36			Age 45		
Category	*B*	*N*	#	*B*	*N*	#	*B*	*N*	#
Cohort									
31	3.58	3.13	31	6.32	5.65	31	7.31	6.52	29
38	3.78	3.39	103	5.80	5.35	106	6.75	6.26	84
45	3.06	2.78	179	5.03	4.74	167	5.96	5.59	51
53	2.55	2.41	403	4.55	4.33	169			0
62	2.47	2.39	223			0			0
District									
High mortality	3.01	2.77	492	5.42	5.02	248	6.45	5.82	91
Low mortality	2.56	2.43	447	4.78	4.54	225	6.79	6.44	73
Education									
None	3.60	3.27	141	6.02	5.56	111	7.34	6.72	65
Primary	2.99	2.78	491	5.09	4.74	291	6.24	5.75	89
Secondary	2.11	2.02	307	3.83	3.77	71	5.10	5.10	10
Income									
Low	3.14	2.90	330	5.43	5.06	226	6.89	6.38	100
Medium	2.85	2.67	314	5.06	4.73	142	6.33	5.79	42
High	2.34	2.22	295	4.50	4.29	105	5.82	5.41	22
All	2.79	2.61	939	5.12	4.79	473	6.60	6.10	164

NOTES: $B \equiv$ Births; $N \equiv$ Surviving children; # $\equiv$ Sample size.

risk is homogenous within types defined by the woman's birth cohort and district of residence is strongly rejected for the pooled sample.

3. THE MODEL

The dynamic structural model focuses on fertility decisions within marriage and their interaction with infant mortality.

3.1. *Fertility Choice.* Fertility decisions are modeled as the sequential binary choices of (subjective) expected utility maximizing agents, along the lines of Heckman and Willis (1976) and Wolpin (1984). Children are an irreversible durable good. The problem solved by women can be formulated as a discrete choice discrete time stochastic dynamic program. The decision horizon is the number of fertile periods after marriage, T, which is known with certainty.[14] In each period t, $t = 1, \ldots, T$, agents choose whether to have a live birth ($b_t = 1$) or not

[14] Fertility outside marriage is very low in Malaysia. The model also implicitly assumes that women do not contemplate divorce. Divorce rates are not negligible in Malaysia, but modeling the time of marriage and marital fertility jointly in a dynamic stochastic framework would add considerable complications.

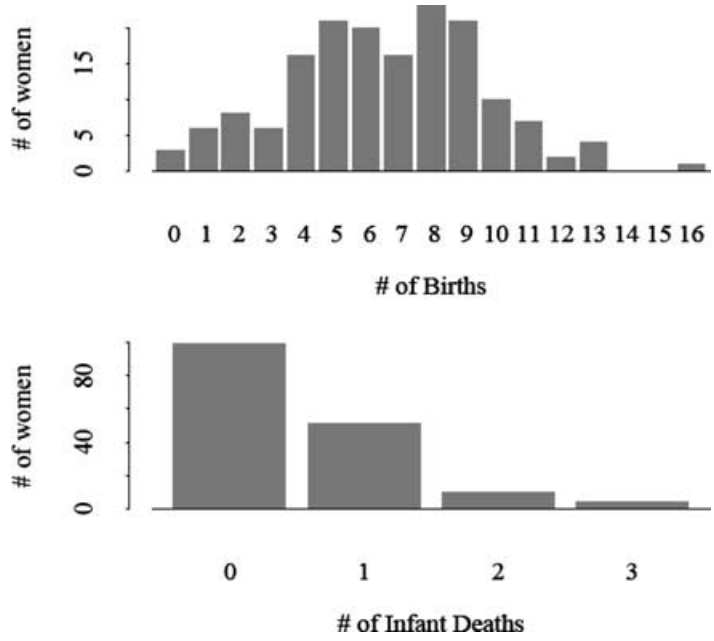

FIGURE 2

DISTRIBUTION OF THE NUMBER OF BIRTHS AND INFANT DEATHS, WOMEN WITH COMPLETE HISTORIES

($b_t = 0$). That is, fertility control is assumed perfect, but there is at most one live birth per period. The utility function is time separable:

$$\sum_{t=1}^{T} \delta^{t-1} R(c_t, N_t, b_t, \epsilon_t) + \mathrm{W}(N_T), \tag{1}$$

where $R(\cdot)$ is the period return and $\mathrm{W}(\cdot)$ the terminal value function, δ is the discount factor, c_t is consumption at t, N_t is the number of children that are alive at the end of period t, and ϵ_t is a preference shock, i.i.d. over time.

Income at t, y_t, is exogenous and deterministic and agents have perfect foresight of the life-cycle income profile.[15] It is assumed that agents cannot borrow or save. Producing children is costly. The cost of a live birth at t is the reduction in period t consumption experienced by women who choose to have a birth at t. The birth cost is defined net of contraception costs. Given the assumption of perfect fertility control, contraception costs are avoided when the woman has a live birth. Birth costs, therefore, are not restricted to be positive.[16] The (net) birth cost depends on whether a child was born the previous period and survived infancy. If no toddler

[15] Female labor supply decisions and their interaction with fertility are also ignored in order to keep the problem tractable.

[16] Independent birth cost and contraceptive cost parameters could be introduced, but they would not be separately identified from data on fertility alone.

is alive, the net cost of a birth is m_{0t}; if a toddler is alive, it is $m_{0t} + e_1$. This "toddler effect" will induce spacing of births. Cost parameters are known at time 1.

Let d_t be an infant death indicator for period t, and let $l_t = b_{t-1}(1 - d_{t-1})$ indicate survival of a child born at $t - 1$. That is, l_t will be 1 if the family includes a toddler during period t. Then consumption at t is given by

$$c_t = y_t - b_t(m_{0t} + e_1 l_t). \tag{2}$$

The period t decision is taken after the shock ϵ_t is observed. The information set at t, therefore, contains the sequences describing fertility and infant mortality up to period $t - 1$ and the time t preference shock. The optimization problem is formulated as a dynamic program in Section 4.1.

3.2. *The Mortality Environment.* Children can only die as infants. Because child mortality beyond infancy was very low in Malaysia for most of the period covered in the sample, this simplification is quite reasonable.[17] Therefore, precautionary or "hoarding" fertility does not arise in this model.[18] Conditional on birth, I assume infant deaths are generated as independent Bernoulli trials with a time-varying, mother-specific probability of death.

Let $\{p_t\}$ be the *exogenous* sequence of infant death probabilities that constitutes the mother's "endowment." Suppose a mother draws her endowment p_1 the first period after marriage from the current population distribution of Bernoulli parameters, given by a density $f_1(p)$, which under rational expectations also describes the mother's prior beliefs about her environment. Furthermore, assume that every period after marriage the endowment "shrinks" by a factor of g_t, reflecting improvements in child health care in the population. That is, $p_t = g_t p_{t-1}$, with g_t, $t = 2, \ldots, T$ a sequence of constants known to the mother.[19] Because the whole distribution of endowments is shrinking by the same factor, positions relative to the mean are not changing and mothers can still use their own experience to infer whether they are "high" or "low" infant mortality individuals within the shifting distribution. Certainly, mothers may also learn from the experience of other women in the population; the assumption here is that all such knowledge can be summarized in initial beliefs $f_1(p)$ and the sequence $\{g_t\}$ describing the

[17] Only 81 noninfant deaths are reported in the sample, out of a total of 4707 live births. The number of women who experienced a noninfant child death was 68, out of 1110.

[18] Hoarding was originally meant to describe the behavior of risk-averse parents who build a large stock of children in order to insure against late, nonreplaceable child deaths. A decline in child mortality would result in lower fertility because the insurance motive for childbearing would weaken. It is child mortality at later ages, rather than infant mortality, that induces insurance hoarding. The term "hoarding" has also been used to label all responses of fertility to the expected level of child mortality. Hoarding responses in this broader sense are not ruled out in this model. See the Introduction for an example, and Wolpin (1997) for a more detailed discussion of insurance hoarding.

[19] Perfect foresight is a strong assumption. Women presumably face uncertainty about events (such as epidemics) that shift the distribution of infant mortality rates in their environment. Ideally, information about realizations of such events would be incorporated in the updating process. However, doing so would add considerable complexity to the model.

downward trend in aggregate mortality. The focus is rather on the direct impact of learning from their own experience on fertility and replacement behavior. Modeling the process by which women obtain their priors and learn about the trend as well as about individual-specific components is beyond the scope of this article.[20]

The probability of an infant death perceived by the mother in period t is the mean of the distribution $f_t(p)$ describing her beliefs about the parameter p_t conditional on the prior $f_1(\,)$, on the sequence $\{g_t\}$, and on the mother's history of births, survivals, and deaths. That is, $\text{pr}(d_t = 1) = \int \text{pr}(d_t = 1 \mid p) \cdot df_t(p) = \int p\, df_t(p)$. To derive the model's decision rules, we need to obtain the distribution $f_t(\,)$ and its mean $\bar{p}_t$ (the "posterior mean") for every possible history. It can be shown that the exact dates of infant deaths have no effect on the kernel of the posterior; that is, the total number of infant deaths (and the number and birthdates of survivors) are sufficient statistics. This is convenient because it reduces the size of the state space.

Furthermore, I assume that the distribution from which women draw their initial endowment p_1 is in the Generalized Beta family with support $(0, k_1)$ and shape parameters (α, β). Then, the prior distributions for the parameters p_t, $t = 2, \ldots, T$ are also Generalized Beta with the same shape parameters, with supports shrinking every period by the factors g_t. That is, the support upper bounds are $k_t = g_t k_{t-1}$. This sequence of Generalized Betas describes the distribution of endowments in the population period by period, as well as the beliefs of women who have not yet had any children. Conditional on an arbitrary history of births, survivals, and/or deaths, the posterior distributions are either Generalized Betas or similar, and exact and simple updating formulae for the posterior means can be derived. Thus, the choice of Generalized Beta has several advantages: It is a closed family of distributions for p_t under our assumption that Bernoulli parameters of all women shrink by the same factor, it leads to simple updating formulae, and it is parsimonious yet quite flexible.

Proofs of the updating properties I have stated here can be found in Section B of the Appendix where I also show how the infant mortality risk perceived by a mother changes following the birth of a child. If the child dies, the subjective risk increases; if the child survives, it falls. The greater the dispersion of beliefs at the time of the birth, the more informative the outcome of the birth is. In particular, if the mean and the coefficient of variation of prior beliefs are $\bar{p}$ and c, the posterior subjective risk following a death is $p(1 + c^2)$. If the child survives, the posterior risk is $\bar{p}[1 - \frac{\bar{p}}{1-\bar{p}} c_0^2]$. The implications of updating for fertility and replacement behavior are discussed in Section 5.

The assumption that the sequence of mortality risks p_t is exogenous is simplistic to the extent that parents are aware of the effect of birth spacing, breastfeeding,

[20] Learning about the trend in the presence of individual heterogeneity is difficult because the effect of trend itself and the effect of the selectivity of fertility need to be disentangled in the aggregate data. But this cannot be done without knowledge of all individuals' decision rules and the distribution of the population across the state variables. So it seems that a model incorporating learning about mortality trends would involve a very complex fixed point problem in the space of decision rules.

and other "health input" choices on the survival probability of their children. This assumption can be relaxed by making the density $f_1(p)$ from which a woman makes her initial draw and the sequence of factors $g_t,\ t = 2, \ldots, T$ a *known deterministic* function of calendar time and health input choices. Thus, infant mortality risks of a population of women with the same history of health input choices keep shrinking or blowing up by the same factor. Therefore, with an enlarged state space, this framework can accommodate both learning about a family-specific endowment and endogenous mortality risk.

4. ECONOMETRIC IMPLEMENTATION

4.1. *Dynamic Programming Formulation and Solution.* The state vector at t includes the vector of predetermined variables $s \equiv (N, D, l, \bar{t}^N)$, where $\bar{t}^N$ is a vector of length N with the birth dates of children who survived infancy. s is discrete and belongs in a finite set S. The exogenous components of the state are the time index t, income y_t, and the preference shock ϵ_t. Following Rust (1987), I assume that there is a preference shock associated with each of the two possible choices; that is, $\epsilon_t = (\epsilon_{0t}, \epsilon_{1t})$. The shocks are independent over time and enter the period return function additively: $R(c, N, b, \epsilon) = U(c, N) + \epsilon_b$. Let (b, d) describe demographic outcomes that occur during a period, after that period's decision has been taken. Clearly, if $b = 0$, outcome $(0, 0)$ will occur with probability 1. If $b = 1$, outcomes $(1, 0)$ and $(1, 1)$ will occur with probabilities $1 - \bar{p}_t(s)$ and $\bar{p}_t(s)$, respectively, where $\bar{p}_t(s)$ is the mean of the posterior distribution of beliefs. Finally, let $V_{bt}(s, \epsilon)$ for $b = 0, 1$ denote choice-specific value functions; then the Bellman equation for this problem is

$$
\begin{aligned}
(3)\qquad V_t(s, y_t, \epsilon_t) &= \max\left[V_{0t}(s, y_t, \epsilon_t), V_{1t}(s, y_t, \epsilon_t)\right] \\
&\qquad \text{for } t = 1, \ldots, T \text{ and } s \in S(t), \text{ with} \\
V_{0t}(s, y_t, \epsilon_t) &= U(y_t, N) + \delta E_t\, V_{t+1}\big(N, D, 0, \bar{t}^N, y_{t+1}, \epsilon_{t+1}\big) + \epsilon_{0t} \\
V_{1t}(s, y_t, \epsilon_t) &= (1 - \bar{p}_t(s)) \begin{bmatrix} U\,(y_t - m_{0t} - e_1 l,\, N+1) \\ +\delta E_t\, V_{t+1}\big(N+1,\, D, 1, \bar{t}^{N+1}, y_{t+1}, \epsilon_{t+1}\big) \end{bmatrix} \\
&\quad + \bar{p}_t(s)\big[U(y_t - m_{0t} - e_1 l, N) \\
&\qquad + \delta E_t\, V_{t+1}\big(N, D+1, 0, \bar{t}^N, y_{t+1}, \epsilon_{t+1}\big)\big] + \epsilon_{1t},
\end{aligned}
$$

where $S(t) \subseteq S$ includes all states feasible at t, and period $t + 1$'s predetermined variables have been written as functions of period t's state variables and outcomes on the right-hand side. The initial state $S(1)$ is the singleton defined by $N = D = l = 0$. The terminal value function is $V_{T+1}(s, y_{t+1}, \epsilon_{t+1}) = W(N)$. Let $\bar{V}_{1t}(\cdot)$ and $\bar{V}_{0t}(\cdot)$ denote the "deterministic" component of the choice-specific value functions:

$$
V_{bt}(s, y_t, \epsilon_t) \equiv \bar{V}_{it}(s, y_t) + \epsilon_{bt},\ b = 0, 1.
$$

Because the income profile is deterministic, the expectations in (3) are actually taken over next period's preference shocks (ϵ_{t+1}) only, and the assumption that they are independent over time makes conditioning on t unnecessary. Assume that the preference shocks are i.i.d. (across alternatives) with $(0, \rho)$ extreme value distributions, with mean $\rho\gamma$ and variance $\frac{\pi^2\rho^2}{6}$. Expected value functions and choice probabilities will then have closed-form solutions:

$$E_t\, V_{t+1}(s, \epsilon) = \rho\left(\gamma + \ln \sum_{i=0}^{1} \exp \frac{\bar{V}_{it+1}(s)}{\rho}\right), \tag{4}$$

$$\mathrm{pr}_{1t}(s) = \frac{1}{1 + \exp\left(\frac{\bar{V}_{0t}(s) - \bar{V}_{1t}(s)}{\rho}\right)}, \tag{5}$$

where γ is Euler's constant, and $\mathrm{pr}_{1t}(\cdot)$ is the (state contingent) probability of choosing a birth; combining these formulae with the Bellman equation and a terminal value function, the problem is then solved by backwards induction.

4.2. *Constructing the Likelihood Function.* Suppose our sample consists of data on I women. The ith woman is observed for $\mathcal{T}_i$ periods after marriage. Information about the ith woman consists of:

- The history of live births, $F_i = \{b_{it}\}_{t=1}^{t=\mathcal{T}_i}$. Let $F_{it} = \{b_{is}\}_{s=1}^{t}$.
- The history of child deaths/survivals, $M_i = \{d_{it}\}_{t=1}^{\mathcal{T}_i}$. Let $M_{it} = \{d_{is}\}_{s=1}^{t}$.
- The vector X_i of exogenous variables, including the income profile.

The econometric specification used here will allow for unobserved heterogeneity in behavioral parameters, with discrete support as in Heckman and Singer (1984). Women with the same observed characteristics X_i may be solving optimization problems with different parameter values because they differ in some permanent characteristics that the researcher cannot observe. For the sake of clarity, I will first consider heterogeneity in the mortality environment only, given its particular importance in this article.

4.3. *Beliefs and Endowments.* Let $P_i = (p_{i1}, p_{i2}, \ldots, p_{i\mathcal{T}_i})$ be the sequence of true infant death probabilities faced by the ith woman in the sample after marriage, that is, her endowment. Let $\Gamma_i = (\alpha_i, \beta_i, k_{1i}, \ldots, k_{\mathcal{T}_i,i})$ describe her initial beliefs about the sequence p_{it} within the generalized beta family. Let Θ be the vector of utility and child rearing technology parameters. Conditional on (Θ, Γ_i, P_i), we can obtain the probability of observing the ith woman's data, $\mathrm{prob}(F_{i\mathcal{T}_i}, M_{i\mathcal{T}_i} \mid X_i)$. Iterative conditioning allows us to rewrite this probability statement as follows:

$$\begin{aligned}\mathrm{prob}(F_{i\mathcal{T}_i}, M_{i\mathcal{T}_i} \mid X_i) = \prod_{t=1}^{\mathcal{T}_i} [&\mathrm{prob}(d_{it} \mid b_{it}, F_{it-1}, M_{it-1}, X_i) \\ &\cdot \mathrm{prob}(b_{it} \mid F_{it-1}, M_{it-1}, X_i)].\end{aligned}$$

Note that conditional probability statements about d's depend on endowments P_i, but not on beliefs Γ_i. This follows from the independence of the processes generating child deaths from the preference shocks, which together with beliefs determine births. Likewise, conditional probability statements about b's depend on Γ_i, but not on P_i, and are obtained from the dynamic programming solution in (5). Therefore, conditional on (Γ_i, P_i) the likelihood factor for the ith woman can be written as follows:

$$\mathcal{L}_i(\Theta \mid \Gamma_i, P_i, X) = \mathcal{L}_{F_i}(\Theta \mid \Gamma_i; X_i) \cdot \mathcal{L}_{M_i}(P_i \mid X_i), \tag{6}$$

where $\mathcal{L}_{F_i}$ is the product of all fertility probability statements and $\mathcal{L}_{M_i}$ is the product of all mortality probability statements. To construct the unconditional likelihood, we need to specify the joint distribution from which (Γ_i, P_i) are drawn. Let us denote it by $F(\Gamma_i, P_i \mid X_i)$. The unconditional likelihood is finally

$$\mathcal{L} = \prod_{i=1}^{I} \int [\mathcal{L}_i(\Theta \mid \Gamma_i, P_i) \cdot dF(\Gamma_i, P_i \mid X_i)]. \tag{7}$$

What is the form of $F(\cdot)$, that is, the joint distribution of endowments and beliefs conditional on observed characteristics X_i? Different scenarios come to mind according to whether beliefs and endowments depend on each other and are homogenous or heterogenous within the subpopulation defined by X_i.[21] For instance, beliefs may vary reflecting the fact that an individual's endowment depends on family history, which is unobserved by the researcher. Identification of both beliefs and endowments could be problematic at this level of generality. We need to consider additional restrictions, such as the following rational expectations constraint (RE): "Conditional on Γ_i, the distribution of p_{i1} is generalized beta with support $(0, k_{1i})$ and shape parameters (α_i, β_i), and $p_{it} = p_{i,t-1} \cdot k_{ti}/k_{t-1,i}$." That is, an individual's initial prior beliefs correctly describe the distribution from which she actually draws her endowment.[22] RE restricts $F(\cdot)$ so that only a distribution over the Γ space needs to be specified. The likelihood in (7) and (6) can now be written as follows:

$$\mathcal{L} = \prod_{i=1}^{I} \int \left[\mathcal{L}_{F_i}(\Theta \mid \Gamma_i; X_i) \cdot \int \mathcal{L}_{M_i}(P_i)\, dF_{|\Gamma}(P_i \mid \Gamma_i, X_i) \right] dF_{\Gamma}(\Gamma_i \mid X_i), \tag{8}$$

where $F_{|\Gamma}$ denotes the distribution of endowments given beliefs; its functional form is given by our assumption that endowments are drawn from a generalized beta distribution. The innermost integral above gives an unconditional likelihood of the observed mortality history. Notice this integral equals the product of the conditional-on-state posterior means in (B.3) in Section B of the Appendix, the

[21] Homogeneity of beliefs or endowments implies that the corresponding marginal distribution is degenerate.

[22] This RE restriction can only be tested if belief parameters, utility parameters, and child rearing parameters are separately identified from fertility choices alone.

subjective expected mortality rates that enter the women's fertility decision problem. Therefore, we can write

$$\int \mathcal{L}_{M_i}(P_i)\, dF_{|\Gamma}(P_i; \Gamma, X_i) = \prod_{t:b_{ti}=1} [\bar{p}_t(\cdot)\, d_{ti} + (1 - \bar{p}_t(\cdot))(1 - d_{ti})]$$
$$\equiv G(M_i; \Gamma_i, X_i).$$

4.4. *Generalized Unobserved Heterogeneity.* Suppose every woman in the population belongs to one of J types unobserved by the researcher. Types may differ in their utility function or child rearing technology parameters Θ or in their beliefs Γ. Let $(\Theta_j, \Gamma_j, w_j)$ define type j, with w_j the fraction of the population of type j, and $\sum_{j=1}^{J} w_j = 1$. It is assumed that the unobserved type is statistically independent of the exogenous variables X_i. The likelihood factor for the ith woman in the sample is then

$$\mathcal{L}_i(\cdot) = \sum_{j=1}^{J} w_j \cdot \mathcal{L}_{F_i}(\Theta, \Gamma_i; X_i) \cdot G(M_i; \Gamma_j, X_i).$$

The log-likelihood for the whole sample is

$$\mathcal{L}(\{\Theta_j, \Gamma_j, w_j\}) = \sum_{i=1}^{I} \log \mathcal{L}_i. \tag{9}$$

4.5. *Identification of Learning Effects.* The hypothesis that women are Bayesian learners extends the assumption that agents are rational in an environment in which there is uncertainty about infant mortality risk. We cannot demonstrate that learning about individual-specific infant mortality risk takes place anymore than we can show that agents are rational. The structure of a model is needed in order to identify learning effects.

In this model, learning is the only reason why the survival history variables $(D, \bar{t}^N)$ are in the state vector. The model predicts that, *if* learning takes place, ceteris paribus changes in the value of those variables have an effect on birth probabilities. These effects on conditional birth probabilities are nonparametrically identified. However, it may be possible to rationalize them in other ways. Furthermore, permanent unobserved heterogeneity in preferences, birth costs, and/or beliefs may induce "spurious" dependence of birth probabilities on $(D, \bar{t}^N)$ even if no learning nor other behavioral responses are present. Without additional information, we need to rely on structure to distinguish between these and other alternative explanations of the data.[23] In this sense, learning effects are not nonparametrically identified.

[23] For instance, permanent unobserved heterogeneity in preferences for children or birth costs would induce dependence of birth probabilities on D. Also, the ages of surviving children can be recovered from their birth dates in $\bar{t}^N$. If child rearing costs are a function of the age of the child and households cannot borrow or save, the age profile of child costs may be identified from data on

Learning effects arise in the model because updating of beliefs occurs, if this has an effect on the decisions to have additional births. Ex ante identical women with the same stock of surviving children and different values of $(D, \bar{t}^N)$ perceive different costs of producing another surviving child. Note that, even if updating takes place, the size of learning effects is a complicated function of all the model's primitive parameters. But no updating will occur if the prior distributions describing beliefs are degenerate. Therefore, the null hypothesis that the variance of the (prior) distribution of endowments is zero can be used to test for the absence of learning.

5. RESULTS

Table 4 presents maximum-likelihood estimates of structural parameters. To maximize the log-likelihood (9) in the parameters of Θ and Γ, a nested numerical solution algorithm was used. The inner algorithm solves the dynamic program for the women in the sample given parameter values; the outer algorithm optimizes the log-likelihood, using a BHHH gradient method. I comment on utility, child cost, and infant mortality risk parameters in turn. For easier reference, a complete parameterization of the model can be found in Section A of the Appendix.

5.1. *Utility.* Current period utility is $U(c, N) = c + \alpha_3 N - (\alpha_4 + \alpha_6\, SEC - \alpha_7 NONE)N^2$, where *NONE* and *SEC* are indicators for the "none" and "secondary" education categories: The mother's education affects the profile of marginal utilities of additional children. In this specification, utility is linear in consumption, which implies that the husband's income category and the shape of the income profile become irrelevant.[24] As for the terminal value function, it was assumed that women live another L periods beyond T, with the same per-period utility function and discount factor applying to each of them as to the first T fertile periods. L was fixed at 20 (i.e., 30 years) for all cohorts.[25]

The signs of the coefficients on the number of surviving children and its square conform to prior expectations. Estimates of α_3 and α_4 imply that the marginal utility of additional children falls as the stock of surviving children increases. The

fertility, income, and the ages of surviving children. Ahn (1995) implemented this idea in a model with no infant mortality and no permanent unobserved heterogeneity.

[24] Ahn (1995) imposed a logarithmic in consumption utility function. Wolpin (1984) used income profiles with individual-specific intercepts instead of only three types. His estimated utility function is almost linear in consumption. When utility is linear in consumption, income does not appear in the utility differences that determine the choice probabilities. My initial specification was the more general $U(c, N) = \alpha_1 c - \alpha_2 c^2 + \alpha_3 N - (\alpha_4 + \cdots)N^2 + \alpha_5 cN$, with $\alpha_2 \geq 0$ in order to rule out convexity. In preliminary estimation iterations, α_2 went to zero and α_5 was very small. If $\alpha_2 = \alpha_5 = 0$, the full set of parameters is not formally identified. Therefore, I imposed that restriction and $\alpha_1 = \rho = 1$ for identification. See Mira (1996) for more details.

[25] Thirty years was the life expectancy at age 45 in 1979–81 for Malay women (see Swee-Hock, 1988). By 1980 only women in the earliest cohort had reached age 45. Although mortality rates will still fall after 1980, this is unlikely to have a large impact on the life expectancies of later cohorts, since mortality rates after age 45 were already at low levels.

TABLE 4

MAXIMUM-LIKELIHOOD ESTIMATES 11092 PERIOD-OBSERVATIONS, 1120 WOMEN, log-lk = −7740.0132

A. Utility and Cost Parameters (Model 3a)

Parameter and Variable		Estimate	ASE
Utility function			
$100\alpha_3$	N_t stock of children	4.380	1.317
$100\alpha_4$	N_t^2, baseline	0.1724	0.05125
$10000\alpha_6$	N_t^2, education = SEC, differential	−1.451	0.8075
$10000\alpha_7$	N_t^2, education = NONE, -differential	0.9647	0.7345
δ	discount factor	1.076	0.01047
Birth costs			
$m0$	baseline birth cost (cohort 38)	3.580	0.3346
$m31+$	cohort 31 differential	−1.079	0.2339
$m45+$	cohort 45 differential	0.3276	0.1562
$m53+$	cohort 53 differential	0.4314	0.1689
$m62+$	cohort 62 differential	1.048	0.2603
$m0+$	unobserved type +, differential	3.473	0.2488
$m0-$	unobserved type −, differential	2.597	0.2042
π_+	unobserved type +, mass	0.08881	0.01426
π_-	unobserved type −, mass	0.2993	0.02974
$m0_{12}$	age effect (slope), youngest women	0.9215	0.1377
$m0_T$	age effect (slope), oldest women	0.5937	0.08949
*marr*31	marriage effect, cohort 31	−1.072	0.3936
*marr*38	marriage effect, cohort 38	−1.068	0.2247
*marr*45	marriage effect, cohort 45	−0.6314	0.1689
*marr*53	marriage effect, cohort 53	−0.4496	0.1200
*marr*62	marriage effect, cohort 62	0.02332	0.1442
e_1	toddler birth cost, cohort 38	0.6540	0.08583
$e31_+$	toddler birth cost, cohort 31 differential	−0.240	0.1540
$e45_+$	toddler birth cost, cohort 45 differential	−0.01439	0.1034
$e53_+$	toddler birth cost, cohort 53 differential	0.3112	0.1016
$e62_+$	toddler birth cost, cohort 62 differential	0.3997	0.1358

B. Distributions of Infant Mortality Risk

Parameter (variable)	1a	1b	2a	2b	3a	3b
Generalized Beta, shape parameters						
c^2	0.7853	0.6994	0.5208	0.5495	0.4618	0.4919
(coeff. of variation)	(0.2096)	(0.2048)	(0.1567)	(0.1668)	(0.1429)	(0.1547)
S	7.7395	8.7590	2.7065	2.8304	2.0408	2.1351
(scale)	(2.9188)	(3.5083)	(1.5625)	(1.6731)	(1.2396)	(1.3780)
Upper bound of support						
f_1	–	–	–	–	−0.8671	−0.8267
(low districts, diff.)	(–)	(–)	(–)	(–)	(0.2089)	(0.2115)
f_2	1.7858	1.3563	1.0802	0.6860	0.7493	0.5185
(youngest women, diff.)	(1.0346)	(0.8433)	(0.8051)	(0.7011)	(0.4281)	(0.4718)
f_{31}	−0.1964	−0.1943	0.5208	0.4666	0.3415	0.2890
(educ=prim., diff.)	(0.3411)	(0.3454)	(0.4918)	(0.5089)	(0.3530)	(0.3697)
f_{32}	−1.0460	−1.0337	−0.1656	−0.1610	−0.1816	−0.1969
(educ=sec, diff.)	(0.3076)	(0.3046)	(0.4350)	(0.4505)	(0.3796)	(0.3899)
$f_{31} - f_{32}$	0.8496	0.8394	0.6865	0.6276	0.5231	0.4859
	(0.2761)	(0.2718)	(0.3758)	(0.3702)	(0.3031)	(0.2997)
g	–	–	0.9580	0.9605	0.9592	0.9616
(geometric trend)	(–)	(–)	(0.006424)	(0.007321)	(0.006298)	(0.007223)

Specifications labeled "a" are full information estimates, selectivity corrected.
Specifications "b" are partial information estimates that only use survival data.
Model 1 controls for mother's education.
Model 2 controls for education and the time trend.
Model 3 controls for education, time trend and district of residence.
All models control for the effect of teenage births.

ratio α_4/α_3 determines the amount of curvature in the utility function; its z-ratio, computed by the delta-method, was 19.61. Therefore, the hypothesis that the marginal utility of children is constant could be rejected.

The quadratic coefficient α_4 applies to women in the "primary" education category. α_7 is positive, which means that the marginal utility of children has a flatter profile (i.e., declines at a slower rate) for women with no schooling. However, α_6 is negative, implying that women with "secondary" education also have flatter marginal utility profiles than women with "primary" education. Thus, it is women with primary education that have the lowest fertility. However, education effects are small and their asymptotic standard errors quite large: T-ratios are -1.80 and 1.31 for α_6 and α_7, respectively, and the two are not jointly significant at the 95% level. These results may seem surprising, but other researchers using the MFLS data report similar findings.[26]

The discount factor is estimated to be greater than one, $\delta = 1.076$. This translates into a negative rate of time preference of 4.8% per year. Because the utility function is linear in consumption, δ should be interpreted as the discount factor that Malaysian women apply to future child services. In this light, a value of δ greater than one seems reasonable, implying that women put a high value on the child services that they will receive in their old age.

5.2. *Child Costs.* The life-cycle profile of birth costs was assumed piecewise linear, with a flat segment between ages 16.5 and 40.5 and two sloped segments at the tails for youngest and oldest women. The slopes ("age effects") were assumed the same for all women. Cohort effects and permanent unobserved components were also introduced, shifting the whole birth cost profile upwards or downwards by a fixed amount.

Birth costs were estimated to be positive for all women in the sample. Since the level of birth costs is not identified, they cannot be interpreted in terms of a monetary equivalent. The parameter $m0$ defines the "baseline" birth cost, that is, birth costs of cohort 38 women in the flat segment when the unobserved component is zero.

The distribution of the unobserved component had three points of support, with $m0+$ and $m0-$ defined as the differential birth cost (in absolute value) for high and low birth cost women, and π_+, π_- as the fraction of women who were high and low birth cost types, respectively. The mass at the baseline (middle) point was estimated at 60%. The birth cost was almost twice as large for women with high costs, with 9% of all women falling in that category. Thirty percent of women had low birth costs, which were only one fourth of the baseline. The source of differences between types could be, for instance, differences in fecundity that make the cost of perfect fertility control vary.

Age effects for "young" ($m0_{12}$) and "old" ($m0_T$) women were found to be highly significant. The birth cost at age 12 is almost twice as large as at age 18, for women in the baseline unobserved type. It increases by 50% from age 39 to age 43.5.

[26] See Panis and Lillard (1993). Note that the model describes behavior conditional on marriage. Education may have an effect on completed fertility through the age at marriage, which is assumed exogenous here.

All cohort effects on the life-cycle profile of birth costs were found to be significant. $m31+$, $m45+$, $m53+$, and $m62+$ were defined as the differential fixed effects, relative to cohort 38. Furthermore, notice that cohort effects increase monotonically. That is, the 1931 cohort had the lowest birth costs and the 1962 cohort had the highest. This seems consistent with an interpretation of cohort effects based on differences in the availability of contraceptive methods.[27]

If sexual intercourse does not occur before marriage, the time interval during which a woman would need to conceive in order for us to observe a birth will be shorter for the first period after marriage than for any other. Therefore, the costs of fertility control might be smaller and birth costs larger. *marr*31, *marr*38, *marr*45, *marr*53, and *marr*62 are cohort-specific marriage effects, which increase the net cost of a birth in the first period after marriage regardless of the age at marriage. They were found to be significant, and their sign was consistent with that conjecture. Furthermore, their absolute value decreased monotonically with birth cohort, which could be interpreted as evidence of a gradual increase in the frequency of premarital sex in Malaysia.

Finally, recall that the birth cost at any period depended on the presence of a (surviving) child born during the previous period. This toddler effect was allowed to vary across cohorts. In Table 4, e_1 is the increase in birth costs for women who have a surviving toddler in cohort 38; toddler effects for women in the other cohorts were defined to be $e_1 + e31_+$, $e_1 + e45_+$, $e_1 + e53_+$, and $e_1 + e62_+$. The estimated toddler effect was positive and significant for all cohorts. It increased monotonically with the birth cohort, reflecting stronger spacing behavior in the data for women of the younger cohorts.

5.3. *Beliefs and Endowments—Specification.* The shape parameters of the generalized beta distributions of infant mortality "endowments" are (α, β). These were assumed constant across types. The following reparameterization is useful. Let $R = \beta/\alpha$ and $S \equiv \alpha + \beta$; then $c^2 \equiv (\frac{R}{1+S})$ is the square of the coefficient of variation. I estimate (c^2, S). The support upper bounds k depend on calendar time and family characteristics.[28] A log–odds specification captures the effect of family characteristics and a geometric trend that of calendar time. Let k_{it} denote the bound in calendar year t for family i. Then

$$k_{it} = \frac{1}{1 + \exp(-f_{it})} \cdot g^{t-t_0},$$

where t_0 is the base year (the earliest year in which any woman in the sample married), g is a common geometric trend, and f_{it} is a linear function of dummies

[27] Between 1967 and 1971, the National Family Planning Board implemented a new program that significantly increased the availability of family planning services and contraceptive devices throughout Peninsular Malaysia. See Swee-Hock (1988).

[28] The mean of the standardized beta distribution is $\frac{\alpha}{\alpha+\beta} = \frac{1}{1+R}$, and the variance is $\frac{\alpha\beta}{(1+\alpha+\beta)(\alpha+\beta)^2}$. Changes in the upper bound of the support (k) have the same proportional effect on the mean and standard deviation of the distribution. Therefore, in this specification, the coefficient of variation is the same across all subpopulations.

indicating mother's age, mother's education, district category, and the mother's unobserved type. That is, $f_{it} = f_0 + f_1 DIST\_LO_i + f_2 YOUNG_{it} + f_{31} PRIM_i + f_{32} SEC_i + f_{41} COST\_LO_i + f_{43} COST\_HI_i$ in the most general specification. Recall that, under rational expectations, a woman's prior beliefs about the risks of infant mortality when she marries are described by the actual distribution from which she draws her endowments. So if the distributions vary across unobserved types ($f_{41} \neq 0$, $f_{43} \neq 0$), then women's beliefs are conditional on more information than the researcher has.

5.4. *Unobserved Heterogeneity and Learning Effects.* The probability of an infant death perceived by a woman is the mean of her beliefs. Her beliefs are updated every period, reflecting her own experience and the downward trend that affects all women equally. The greater the heterogeneity in endowments the more women have to learn from their own experience. In the model with $f_{41} = f_{43} = 0$, the point estimate of the coefficient of variation of the distributions of infant mortality risks is $\hat{c} = 0.68$. That is, there is substantial heterogeneity within subpopulations defined by cohort, the district type, and the woman's education. On the basis of these estimates, I carried out a variance decomposition of infant mortality risk for all women in the sample, if they were having a child at age 25. Only 31% of the variance of the Bernoulli parameter p is explained by the woman's education, district of residence and calendar time, whereas unobserved heterogeneity accounts for the remaining 69%.[29]

A likelihood ratio test rejects the hypothesis $H_0 : c^2 = 0$.[30] Note that H_0 corresponds to the joint hypothesis of no learning *and* no heterogeneity in endowments. I also estimated the extended specification in which women have more information than the researcher. In this model, the absence of learning ($c^2 = 0$) is compatible with heterogeneity in endowments unobserved to the researcher ($f_{41} \neq 0$, $f_{43} \neq 0$). The hypothesis $c^2 = 0$ is rejected again. On the contrary, the hypothesis $f_{41} = f_{42} = 0$, which corresponds to model 3 in Table 4, cannot be rejected.

When an infant death occurs, the percentage increase in the mean of beliefs is given by c^2, the square of the coefficient of variation of beliefs at the time of the child's birth. Thus, a woman whose firstborn does not survive would revise her subjective infant mortality risk upwards by 46%. When infant mortality rates are high, this could significantly increase the expected costs of raising a surviving child, reducing the willingness to have additional births. As an example, consider the model's prediction of the probability of observing a birth for a 21-year-old

[29] The estimated model of infant mortality endowments predicts (Generalized Beta) distributions of the Bernoulli parameter p for women at every period of their fertile lives. For each woman in the sample I computed the mean and the variance of these distributions given the woman's education, district of residence and the value of the trend component when the woman is 25. The variance decomposition is obtained from the sample variance and the sample mean of all women's means and variances, respectively.

[30] Twice the difference in the log-likelihood is 14.51, whereas the critical value at the 5% significance level is 2.71. Note that this test is nonstandard because $c^2 = 0$ is in the boundary of the parameter space. The distribution of the test statistic under the null is a 50–50 mixture of a mass-point at zero and a chi-squared distribution with one degree of freedom (See Gourieroux and Monfort, 1995).

woman from cohort 31. Suppose she has no schooling, baseline birth costs, lives in the high mortality environment, married at age 18, and has no surviving children. Because she is entering her third period after marriage, she may have experienced 0, 1, or 2 infant deaths. The predicted birth probabilities are 0.79, 0.75, and 0.74, respectively. This pattern illustrates learning effects: Birth probabilities decline with the number of experienced deaths. Learning effects are weaker for women living in low mortality environments: The corresponding birth probabilities for an otherwise identical woman belonging to cohort 62 would be 0.72, 0.71, and 0.71. New information about infant mortality risks leads to a reassessment of the expected costs and utility flows of a birth. Therefore, although the magnitude of learning effects depends most directly on c^2, it also depends on all other endowment, child costs, and utility parameters.

5.5. *Determinants of Infant Mortality Risk.* The lower panel of Table 4 shows different sets of estimates of infant mortality parameters. In columns labeled "a" the estimates are obtained jointly with those of utility and cost parameters; that is, they are corrected for the selectivity of births. In columns "b" the estimates are not corrected; that is, they are obtained from survival data alone.

In model 1a, only the mother's education and age shift the distribution of endowments. Women with no schooling and older than 18 are the baseline subpopulation.[31] As we would expect, the mean infant mortality risk is estimated to be lower (10%) for women with primary education and much lower (48%) for women with secondary education. But one of the three differences (none-primary) is not significant at the 5% level. If we introduce the geometric time trend (model 2a), the sign of the this difference is reversed. Furthermore, once we control for both the time trend and district in model 3a, none of the education effects remain significant at the 5% level, and the trend and district coefficients are significant. So the negative effect of mother's education on infant mortality risk in the raw data is an artifact of all other environmental and behavioral factors that underlie the negative time trend and district coefficients, combined with the positive correlation of mother's schooling with calendar time and the "low mortality district" indicator.

Model 3 is the preferred specification. Infant mortality risk "endowments" were shrinking at a rate of 4% per year.[32] Relative to the baseline population, the mean infant mortality risk was approximately 40% lower in the low infant mortality districts and 35% higher for women having a child before the age of 18, but this difference is not significant at the 5% level. Point estimates of education effects

[31] It seems doubtful that more than mean and variance of the distributions of endowments could be precisely estimated from data on births and deaths, given the limited sample size. With this specification, the mean and variance for the baseline population depend on three parameters (f_0, R, S). I normalize and set $f_0 = 0$.

[32] Note that the baseline is now women with no schooling, older than 18, and living in the high mortality districts in 1943. The results were similar when the trend was included in the log-odds specification (time and time squared). I also estimated a specification with district-education-specific geometric trends. The hypothesis of a common trend could not be rejected.

imply a 15% higher risk for women with primary education and 5% lower for women with secondary education.

5.6. *Birth Selectivity Bias.* A comparison of columns a and b in Table 4 reveals that the correction for the selectivity of births is not very important in this sample. To understand why, recall that selectivity bias can only occur if there is correlation between fertility and unobserved components of infant mortality risk. Given the model specification, there are several potential sources of correlation. If $f_{41} \neq 0$, $f_{43} \neq 0$, then fertility may respond directly to differences in the family-specific component of infant mortality risk that are taken into account by women but are not observed by the researcher. Indirect responses may arise if the unobservable components of mortality risk and birth costs are correlated. However, $f_{41} = f_{42} = 0$ could not be rejected. Even in this case, dynamic responses of fertility to infant deaths (e.g., replacement behavior) may induce correlation between fertility and components of infant mortality risk that are unobservable to both researcher and agents. If a large fraction of births are replacement births, then the distribution of endowments of women who are actually having births is upward biased. However, the experiments I report below show that although replacement rates are not negligible, replacement births are not a large fraction of total births.

As an illustration, consider a population of cohort 31 women marrying at age 18; let the distribution of endowments be that of women with no schooling in high mortality districts in 1949. The mean endowment is a mortality risk of 16.2%. The infant mortality rate observed over the whole life cycle, computed from the estimated model, would be 16.5%. The small difference is the effect of birth selectivity. Finally, note that the bias in estimates of the effect of mother's education is even smaller if selection biases in the relevant subpopulations cancel out. In our example, the mean endowment and observed infant mortality rate for a population of women with primary education would be 18.9% and 19.4%. The difference in mean endowments (2.7%) and the difference in infant mortality rates (2.9%) is almost the same.

5.7. *Assessment of Fit.* The probabilities of observing a birth predicted by the model, given parameter estimates, can be compared with the actual birth frequencies observed in the sample. Table 5 shows actual and predicted birth probability profiles, with respect to both age and duration of marriage. For example, to obtain the $t = 3$ "predicted" value in the duration table, the birth probability predicted by the model, conditional on a woman's observed type and age at marriage, was averaged over all the women who were observed in the third period after marriage. The "actual" probability is simply the fraction of those women who had a live birth in that period. Pearson χ^2 statistics were computed in order to test for equality of the actual and predicted probabilities, period by period. The differences are not statistically significant at the 5% level for all but 1 of the 22 periods. Because the specification included multiple cohort effects in birth costs, the tests were also carried out separately for all those period–cohort combinations for which at least 10 observations were available. The number of statistically significant differences and the number of tests were 1/20, 0/21, 2/20, 2/15, and 0/9, respectively, for cohorts

TABLE 5
BIRTH PROBABILITIES-FIT: AGE AND DURATION OF MARRIAGE PROFILES

Duration of Marriage					Age				
t	years	A	P	nobs	t	age	A	P	nobs
1	0	.634	.632	1120	1	12	.200	.141	10
2		.576	.578	1120	2		.290	.328	62
3		.549	.548	1120	3		.517	.503	178
4		.508	.507	1012	4		.614	.678	337
5	6	.472	.471	909	5	18	.614	.655	523
6		.438	.437	824	6		.606	.611	690
7		.408	.408	732	7		.597	.576	828
8		.380	.380	668	8		.523	.534	917
9	12	.354	.354	597	9	24	.523	.497	963
10		.330	.331	523	10		.448	.460	939
11		.310	.311	447	11		.471	.428	875
12		.290	.291	392	12		.373	.397	797
13	18	.270	.272	337	13	30	.386	.370	709
14		.253	.255	295	14		.381	.343	628
15		.234	.236	245	15		.286	.319	552
16		.208	.209	217	16		.285	.295	473
17	24	.176	.177	192	17	36	.236	.277	394
18		.040	.140	151	18		.249	.259	329
19		.102	.103	103	19		.176	.254	290
20		.071	.070	59	20		.145	.165	241
21	30	.00	.050	23	21	42	.083	.110	193
22		.167	.032	6	22		.061	.041	164

NOTES: A $\equiv$ actual frequency; P $\equiv$ predicted probability; nobs $\equiv$ number of observations.

31, 38, 45, 53, and 62. Note that the tests are fairly stringent in that predicted probabilities t periods after marriage are computed t periods ahead rather than conditional on the observed state at t. This means that the model is also used to predict the probability distribution over all possible states.[33]

Figure 3 graphs the actual and predicted birth probability profiles for the whole sample. The model captures the steep decline in birth probabilities with duration of marriage. Two effects are at work to generate this pattern: (1) Because children are a durable good, there is an incentive to have them early in the life cycle. (2) The utility function is concave in the number of surviving children. As duration of marriage increases, women have a larger stock of surviving children, and the marginal utility of an additional child falls, reducing the probability of another birth. Could a model with curvature alone fit the declining profile equally well? If δ is set to zero, dynamic effects such as (1) disappear and we are left with a sequence of static problems. To test the hypothesis that there are no dynamics, the model was reestimated imposing the restriction $\delta = 0$. The hypothesis was strongly

[33] Computing birth probability profiles as well as the measures of replacement behavior below involves fairly complex recursive calculations. Simulations were not necessary, since state transition probabilities can be computed exactly. When necessary, the calculations were repeated for every value of the observable exogenous variables and averages were obtained using sample weights. Unobserved heterogeneity also had to be dealt with. For more details, see Mira (1995).

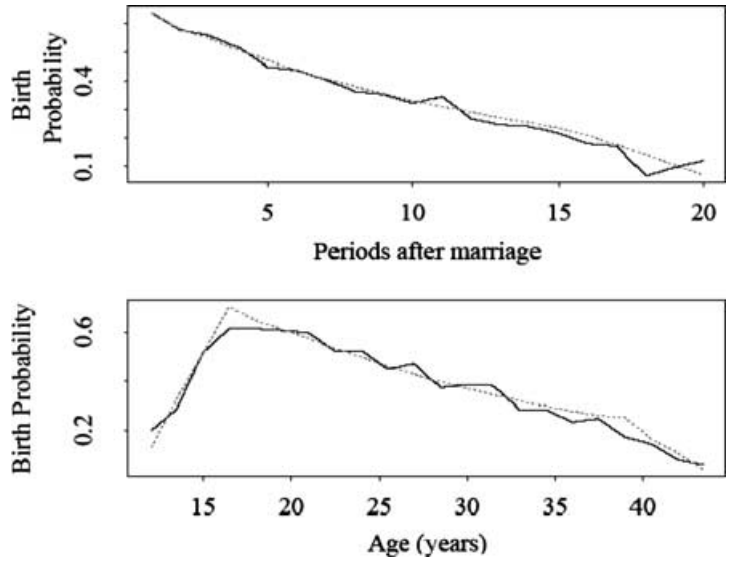

FIGURE 3

BIRTH PROBABILITY PROFILES, ACTUAL VERSUS PREDICTED

rejected at the 1% significance level. Notice that the age profile is flat between the ages of 18 and 24. The reason for this is that every period, the effect of declining duration profiles is dampened by the addition of new marriage cohorts.[34]

In Table 6, the infant mortality rates observed in the sample, by cohort and district category, are seen to be close to infant mortality rates predicted by the model. Predicted infant mortality rates are the average of the probabilities of an infant death predicted by the model for all birth episodes observed in the sample, conditional on the state variables.[35]

5.8. *Defining Replacement Rates.* To measure the response of fertility to experienced child deaths, consider the following experiment. Select two women with the same observable exogenous determinants of fertility. Suppose these two women have had identical fertility and mortality histories up to period t. The state vector is therefore the same for the two women at t. Suppose that both women have a birth, but only one of the two infants survives. If there is replacement, the woman whose child died should have higher fertility in the future. The replacement rate for a period t, state s infant death is defined as the difference in the expected number of future births:

[34] The differences between actual and predicted birth probabilities in the age profile were statistically significant at the 5% level for 5 of the 22 periods for the whole sample. Three of these larger differences (in periods 4, 5, and 19) can be attributed to the effect of kinks in the specification of birth cost profiles. By cohort, the number of significant differences and periods was 2/20, 1/21, 0/21, 4/17, 1/10.

[35] None of the differences are statistically significant at the 95% level when we compute the usual χ^2 test statistics.

TABLE 6
INFANT MORTALITY FREQUENCIES-FIT: BY DISTRICT, COHORT, AND EDUCATION

	Cohort					Education				
District	31	38	45	53	62	N	P	S	All	
High	.123	.086	.075	.062	.043	.070	.074	.052	.068	A
mortality	.115	.099	.077	.056	.042	.080	.073	.042	.068	P
	114	430	626	981	492	604	1493	561	2643	nobs
Low	.098	.069	.039	.036	.023	.076	.046	.020	.042	A
mortality	.087	.067	.044	.034	.023	.058	.048	.024	.041	P
	123	290	440	873	390	215	1120	666	2116	nobs
	.110	.079	.060	.050	.034	.072	.062	.034	.057	A
All	.100	.087	.064	.046	.034	.073	.062	.033	.057	P
	237	720	1066	1854	882	919	2613	1227	4759	nobs

NOTES: A ≡ actual frequency; P ≡ predicted probability, state-conditional; nobs ≡ number of births.

$$rep(t,s) \equiv l_{t+1}(s, b_t = 1, d_t = 1) - l_{t+1}(s, b_t = 1, d_t = 0), \tag{10}$$

where $l_t(s)$ is the expected number of future births for a woman who begins period t in state s: $l_t(s) \equiv E_t(\sum_{r=t}^{r=T} b_r \mid s)$.

The size of replacement rates depends on the following[36]: (1) preferences for family size. More specifically, on the curvature of the utility function. The death of a child reduces the number of surviving children in the family. If women with a smaller number of surviving children are more likely to have additional births, infant deaths will induce replacement responses. In this model, such "parity-dependent fertility control" occurs if the marginal utility of an additional child declines with the number of surviving children.[37] (2) The differential birth cost for women who have a toddler, e_1. If e_1 is positive, an infant death reduces the cost of a birth the following period, because no toddler will be alive. Therefore, replacement rates will increase with e_1. Notice, however, that this toddler effect operates only the period immediately following an infant death. (3) Learning effects, which reduce replacement rates as discussed in Section 5. If replacement rates were driven by the effects of parity-dependent fertility control only, they would necessarily fall in the [0, 1] interval. However, heterogeneity, learning, and toddler effects could make them negative or greater than one.

5.9. *Replacement Rates in the Sample Populations.* Replacement rates were estimated for all infant deaths that were actually observed in the sample. They range from 0% to 65%. Ceteris paribus, replacement rates increased with parity and decreased with the age of the mother. At higher parity, women have more information about their own infant mortality rates. There is less learning involved in an additional birth. With weaker learning effects, replacement rates increase.

[36] This measure of replacement is adapted from Wolpin (1984)

[37] Parity is usually defined as the number of live births a woman has experienced.

TABLE 7
AVERAGE REPLACEMENT RATES, BY COHORT

	Cohort					
	31	38	45	53	62	All
Full model (3a):						
Average replacement rate	0.31	0.32	0.33	0.35	0.36	0.35
Decomposition of completed fertility:						
$E(B)$	8.13	6.71	5.99	5.76	5.31	5.83
$E(B \mid P = 0)$	7.86	6.51	5.86	5.68	5.25	5.72
$\mathcal{R}$	0.27	0.20	0.13	0.09	0.06	0.10
No heterogeneity ($c^2 \simeq 0$) Avge. rep.	0.39	0.40	0.39	0.39	0.39	0.39
No heterogeneity, no 'toddler' effect Avge. rep.	0.36	0.37	0.35	0.33	0.32	0.33

NOTES: $E(B)$ is expected completed fertility.
$E(B \mid P = 0)$ is expected fertility conditional on zero infant mortality risk.
$\mathcal{R}$ is a measure of births due to replacement behavior.

Replacement rates generally decreased with the mother's age because children are a durable good, which reduces the value of late births.[38]

The following decomposition of expected life-cycle fertility is derived and discussed in Section C of the Appendix:

$$l_1 = E(B_T) = E(B_T \mid P = 0) + \mathcal{R}. \tag{11}$$

The first component is the average number of births that we would observe in the population if (unexpectedly) the actual probability of infant deaths were zero. The second component, $\mathcal{R}$, is a weighted sum of the replacement rates at all ages and states for which with $D = 0$ (i.e., no previous infant deaths). The weights are proportional to the probability that infant deaths will be observed in each state. The decomposition measures the contribution of replacement births to life-cycle fertility and $\mathcal{R}$ places an upper bound on the response of completed fertility to reductions in infant mortality risk through replacement effects. Loosely speaking, $\mathcal{R}$ is the number of births that we would observe if the actual mortality risk were zero, multiplied by the average infant mortality rate and by the average replacement rate.

I used the weights in $\mathcal{R}$ to compute an average of state-contingent replacement rates. The results are presented in Table 7 for each of the five birth cohorts as well as for the whole sample. The average replacement rate was 32% for cohorts with complete histories. $\mathcal{R}$ is not very large because mortality rates were already at fairly low levels for most of the sample period and because Malaysian women do not fully replace infant deaths. For instance, the average woman in cohort 31 would have had close to eight children if none had died. With an average infant

[38] See Mira (1995) for a more detailed analysis.

mortality risk of 0.125 and a replacement rate of 30%, the number of replacement births is approximately $8 \times 0.125 \times 0.3 = 0.3$.

I computed average replacement rates for two other hypothetical environments. In one of them there is no toddler effect and no room for learning; that is, e_1 is set to zero and all women have the same infant mortality endowment. In the other one, there is no heterogeneity in infant mortality rates but the toddler' effect is present. A comparison of these three measures in Table 7 suggests that replacement behavior in Malaysia is basically driven by the effects of parity-dependent control: Women replace infant deaths because the value of an additional birth is higher when they are left with fewer surviving children. Toddler and learning effects, respectively, increase and reduce replacement rates by approximately equal amounts (+5%, −4%). Recall that toddler effect may reflect the impact of interrupted breastfeeding.[39]

Replacement rates were increasing, from an average 31% for cohort 31 to an average 36% for cohort 62. The main reasons for this were the increase in the differential birth cost for women with a live toddler in the last two cohorts and the weaker learning effects for women of the more recent cohorts.[40]

Notice that the same highly structured approach yields much larger estimates of replacement rates than in Wolpin (1984).[41] Because there are several differences between the two studies in terms of both the models—which are not nested—and the samples, pinning down the source of this discrepancy is not trivial. First, my counterfactual experiments suggest that replacement responses are not driven by unobserved heterogeneity and learning, which Wolpin's model did not allow for. To confirm this, I estimated a restricted specification with no unobserved heterogeneity in mortality risk and no learning. The restricted specification was rejected, but the replacement rates it implied were very similar to those of the unrestricted model and much larger than Wolpin's. Therefore, in both my model and Wolpin's, replacement rates are determined by the pattern of estimated marginal utilities of children (decreasing and constant, respectively).

If the marginal utility of children is decreasing the ceteris paribus relationship between the birth probability and the number of surviving children (the "parity profile") should be decreasing, a particular form of state dependence. In my raw data parity profiles are never steep but they are decreasing at younger ages, flattening out gradually, and eventually becoming increasing at older ages. State dependence, combined with permanent unobserved heterogeneity in the utility or cost of children, can reconcile the model and this raw data. When I removed

[39] Olsen (1983) included breastfeeding duration data in his reduced-form equations. His replacement rate estimates of 0.32 include the effect of interrupted breastfeeding.

[40] The negative impact of learning on replacement is smaller in low mortality environments. The expected costs of infant mortality are small when the levels are low, so even relatively large adjustments in perceived infant mortality risks will have a small impact on future fertility. Also notice that, in estimation, all fertility histories were incomplete for women in the two youngest cohorts. To compute replacement rates, the model was used to predict fertility and mortality later in their life cycle. See Mira (1995) for further details.

[41] Although the samples are not identical, there are other (nonstructural) estimates of replacement rates obtained from MFLS data. Olsen (1983) reports a replacement rate of 0.32. Panis and Lillard (1993) do not compute an average, but their results are similar.

TABLE 8
COMPLETED FERTILITY AND COVARIATES

Measure	(1)	(2)	(3)	(4)	(5)	(6)
E(B)	8.76	8.70	7.87	5.88	8.52	8.88

NOTES: $E(B) \equiv$ expected births.
(1) Baseline: Infant mortality endowments constant at 1949 level, perfect foresight.
(2) Infant mortality endowments constant at 1980 level, perfect foresight.
(3) Age at marriage is 19.5.
(4) Birth costs as in cohort 62.
(5) Primary education.
(6) Secondary education.
Baseline population has birth costs as in cohort 31, no schooling, age at marriage is 16.5, and infant mortality level is sample weighted average of levels in high and low mortality districts in 1949.

controls for permanent unobserved heterogeneity in birth costs, I also obtained negligible replacement rates and the (nested) model was strongly rejected. In contrast, in Wolpin's study, introducing permanent unobserved heterogeneity in the marginal utility of children did not change estimates of curvature parameters and replacement rates.

I have explored estimation with many different specifications and data sets combining features of both studies in different ways, and I have found that the key element explaining the difference in results is the treatment of premarital observations, which Wolpin included in his sample but I did not.[42] Premarital births are very rare in Malaysia, whereas birth rates within marriage are high. Including these observations significantly reduces birth probabilities at parity zero for young women. As a result, the parity profile for young women in Wolpin's sample is hump-shaped, increasing clearly between parities 0 and 1 and decreasing slightly from parity 1. This is harder to reconcile with a decreasing marginal utility of children.

5.10. *Completed Fertility and Covariates.* In Table 8, I illustrate the effect on fertility of changes in the model's exogenous covariates. I choose as the baseline a population of cohort 31 women with no schooling, marrying at age 16.5, and facing the infant mortality risks of the late 1940s. The baseline population has women from both district categories, with weights equal to the proportions in the sample. Birth costs and infant mortality risks are heterogenous, as estimated. Column (1) shows expected fertility predicted for the baseline population: The average number of births over the life cycle would be 8.76. In counterfactual experiments (2)–(6), I compute the effect of changes in the level of infant mortality risk, age at marriage, the women's education, and "cohort" birth costs, one at a time.

[42] The other main difference in the data is that Wolpin's sample only included women from my first three cohorts. As for the specification, Wolpin included age effects in birth costs but had no toddler effect. More details are available from the author.

The population in experiment (2) faces the infant mortality risks of 1980. On the one hand, lower mortality risks reduce the number of replacement births. However, in Table 7 we saw that the scope for reductions in fertility through replacement effects was not too large, with 0.3 an approximate upper bound for women of cohort 31. Furthermore, as noted before the impact of reduced mortality on fertility cannot in general be predicted from replacement behavior alone (see Section C in the Appendix for a formal statement). The estimated model also predicts that state-conditional birth probabilities increase with the probability of child survival because lower birth costs per survivor increase the demand for survivors.[43] The net effect of lower mortality risk on completed fertility is a very small reduction.

The effect of differences in birth costs across cohorts is very large. Ceteris paribus, the increase in birth costs accounts for almost three fewer births for cohort 62 compared with cohort 31. The model also predicts an average reduction of almost one birth resulting from the 3-year increase in the age at marriage between cohorts 31 and 62. Ceteris paribus, education effects are small. Sending girls to school reduces their future fertility by 0.24 births, if they would otherwise stay at home. However, women who go on to secondary school have slightly higher fertility rates than those who do not.

Thus counterfactual experiments suggest that the contribution of the decline in infant mortality rates to the reduction in fertility in Malaysia after World War II through replacement effects was small compared with the impact of changes in exogenous variables and parameters such as the age at marriage and birth costs.

6. CONCLUSION

In this article, I study the links between infant mortality and fertility in an environment with unobserved heterogeneity in infant mortality risks across mothers. It has been suggested that replacement behavior (i.e., the fertility response to an experienced child death) might be influenced by mothers' learning about a family-specific component of infant mortality risk. However, this conjecture had not been investigated before. I explicitly introduce learning by mothers in a dynamic stochastic model of life-cycle marital fertility behavior. I estimate the model's structural parameters on a sample drawn from the Malaysia Family Life Surveys, and I use them to obtain new estimates of replacement rates. I find evidence of replacement responses, with the average replacement rate slightly above 30%. Women with fewer surviving children showed a higher propensity to have additional births, and this behavior was the main determinant of replacement behavior. Estimated learning effects are small and contribute to lower replacement rates.

The framework can also be used to obtain estimates of a "production function" of infant mortality risk that allows for unobservable family-specific components,

[43] In the model, the value of a birth depends on the difference between the birth cost and the marginal expected utility of current and future child services derived from an additional child. If the marginal expected utility term is positive, it increases as infant mortality rates fall, resulting in a higher probability of observing a birth. If the marginal expected utility term is negative, lower infant mortality rates will lead to lower birth probabilities. Given parameter estimates, the first case was prevalent.

endogenous inputs, and sample selectivity through fertility decisions and learning about the family component. As an example, I look at the effect of mother's education and teenage births on the risk of infant mortality in the Malaysian data. I find considerable heterogeneity in infant mortality risks across mothers even after I control for calendar time, mother's education, and the district of residence. The infant mortality risk is higher for less-educated mothers and for those who decide to have a child before the age of 18; however, these education and age effects do not remain significant once I control for the district of residence and the strong downward trend in mortality risk. Although replacement behavior induces a small upward bias in the mean of observed infant mortality risks, using the behavioral model to correct for the selectivity of births in this sample had a small impact on parameter estimates.

APPENDIX

A. Parameterization.

Current period utility:

$$U(c, N) = c + \alpha_3 N - (\alpha_4 + \alpha_6 SEC - \alpha_7 NONE)N^2$$

where *NONE*, *PRIM*, *SEC* are indicators for the mother's education category, c is consumption, and N is the stock of children.

Terminal value function (no estimable parameters): $\mathrm{W}(N_T) = \sum_{t=T+1}^{T+20} \delta^{t-1} \times U(c_t, N_T)$

Birth costs: Let t index 18-month-long periods in the woman's life cycle, with $t = 1$ starting at age 12, $t = 2$ at age 13.5, . . . , and $t = 22$ starting at age 43.5. Let m_t be the cost of birth in period t; then

$$m_t = m0 + (\text{cohort shifter}) + (\text{unobserved type shifter}) + (\text{ age effect}) + (\text{toddler effect}) + (\text{first-period-after-marriage effect}),$$

with

$$\text{cohort shifter} = \left\{ \begin{array}{c} 0 \text{ if cohort is 38 (baseline)} \\ m31 + \text{if cohort is 31} \\ m45 + \text{if cohort is 45} \\ m53 + \text{if cohort is 53} \\ m62 + \text{if cohort is 62} \end{array} \right\},$$

$$\text{unobserved type shifter} = \left\{ \begin{array}{c} 0 \text{ if unobserved type is baseline} \\ (\text{mass } 1 - \pi_+ - \pi_-) \\ m0 + \text{if unobserved type is + or "high" birth costs} \\ (\text{mass } \pi_+) \\ m0 - \text{if unobserved type is − or "low" birth costs} \\ (\text{mass } \pi_-) \end{array} \right\},$$

$$\text{age effect} = \left\{ \begin{array}{c} 0 \text{ if } 4 < t < 19 \\ m0_{12} \times (5 - t) \text{ if } t < 5 \\ m0_T \times (t - 18) \text{ if } t > 18 \\ \text{where } t \text{ is age} \end{array} \right\},$$

$$\text{toddler effect} = \left\{ \begin{array}{c} 0 \text{ if } b_{t-1} = 0 \\ e_1 \text{ if } b_{t-1} = 1 \text{ and cohort is 38} \\ e_1 + e31 + \text{if } b_{t-1} = 1 \text{ and cohort is 31} \\ e_1 + e45 + \text{if } b_{t-1} = 1 \text{ and cohort is 45} \\ e_1 + e53 + \text{if } b_{t-1} = 1 \text{ and cohort is 53} \\ e_1 + e62 + \text{if } b_{t-1} = 1 \text{ and cohort is 62} \end{array} \right\},$$

first-period-after-marriage effect is *marr*31 or *marr*38 or *marr*45 or *marr*53 or *marr*62, depending on cohort, if t is the first period after marriage, 0 otherwise.

Beliefs and endowments: Infant mortality risk (Bernoulli parameter) drawn from Generalized Beta distribution with shape parameters (α, β) and support $[0, k_s]$, with

$$k_s = \frac{1}{1 + \exp(-f_s)} \cdot g^{s - s_0}$$

where s is calendar year, s_0 is the base year (1943), and

$$f_s = f_1 DIST\_LO + f_2 YOUNG_s + f_{31} PRIM + f_{32} SEC + f_{41} COST\_LO + f_{43} COST\_HI$$

and the regressors are the following indicators:

$DIST\_LO = 1$ if woman lives in low mortality district

$YOUNG_s = 1$ if woman is younger than 18

$COST\_LO = 1$ if woman is of unobserved type "low"

$COST\_HI = 1$ if woman is of unobserved type "high"

Reparameterization of shape parameters: $S \equiv \alpha + \beta; c^2 \equiv (\frac{\beta/\alpha}{1 + \beta/\alpha})$.

B. Updating.

Updating the kernel of beliefs: Timing of infant deaths does not matter: As in Section 3, let $f_1(p)$ be the prior distribution describing beliefs about the infant mortality parameter in period 1; $\{g_t\}_{t=2, \ldots}$ is the sequence of factors by which p

is believed to be shrinking every period. Let $\{b_s, d_s\}_{s=1}^{t-1}$ represent an arbitrary history of demographic events experienced by a woman. What are beliefs at t? To obtain the (posterior) distribution at t, two operators are applied sequentially to the initial prior distribution: (1) updating, that is, combining current beliefs and the likelihood of experienced demographic events and (2) the "shrinking" transformation. The second operator is applied every period, whereas the first one is applied only those periods during which there is a birth. For instance, if there is no birth in period 1, the kernel of beliefs at 2 is $f_1(p/g_2)$. If there is a birth and the child survives infancy, then $f_2(p) \propto f_1(p/g_2) \cdot (1 - p/g_2)$. If there is a birth and an infant death, then $f_2(p) \propto f_1(p/g_2) \cdot p$.

Notice that the "updating" operator goes first, when applicable. Furthermore, once a likelihood or updating factor has been incorporated into beliefs, the shrinking transformation will apply to it in all subsequent periods. However, the effect of the shrinking transformation is not the same on both types of likelihood factor. When applied to a period t infant death factor, it transforms p into $(p/g_{t+s}) \cdot (1/g_{t+s})$, for $s \geq 1$. The shrinking factors will drop from the kernel. In the case of a survival, $(1 - p)$ is transformed into $(1 - p/g_{t+s})$ and the shrinking factors change the form of the kernel. This is the reason why the timing of survivals matters, whereas the timing of deaths does not.

Percentage changes in posterior means: Let $f_0(p)$ be the density describing beliefs before a birth, and $E_0 p$ and $\mathrm{var}_0 p$ its mean and variance. Let $f_1(\,)$ and $E_1 p$ be the posterior density and mean, after the birth episode. If an infant death is observed, then $f_1(p) = \frac{1}{E_0 p} f_0(p) \cdot p$, and the posterior mean is

$$E_1 p = \frac{1}{E_0 p} \int p^2 \, df_0(p) = \frac{\mathrm{var}_0 p + (E_0 p)^2}{E_0 p}.$$

Therefore, the relative change in perceived infant mortality risks following an infant death is the square of the coefficient of variation of the prior:

$$\frac{E_1 p - E_0 p}{E_0 p} = \frac{\mathrm{var}_0 p}{(E_0 p)^2} \equiv c_0^2.$$

If an infant survival is observed, then $f_1(p) = \frac{1}{1 - E_0 p} f_0(p) \, (1 - p)$; one can show that the relative change in this case is $-\frac{E_0 p}{1 - E_0 p} c_0^2$.

Posterior means with generalized beta priors: Updating formulae: I will now obtain expressions for the posterior means in the nonstationary mortality environment described in Section 3. Recall that posterior means are the (subjective) probabilities of infant deaths that appear in the Bellman equations. Let D and N be the number of infant deaths and infant survivals, respectively, that a mother has experienced. Survivals happened in periods $t_1, t_2, \ldots, t_N$. Then, the kernel of the posterior distribution of the mother's infant mortality endowment at t is

$$p^{\alpha-1+D}(k_t-p)^{\beta-1}\left(\frac{k_t}{k_{t_1}}-p\right)\cdots\left(\frac{k_t}{k_{t_N}}-p\right).$$

Let $\bar{C}_N(\alpha+D,\beta,k_{t_1},\ldots,k_{t_N},t)$ be the integral of this kernel over the range $(0, k_t)$. Then the mean of the posterior at t is

$$\frac{\bar{C}_N(\alpha+D+1,\beta,k_{t_1},\ldots,k_{t_N},t)}{\bar{C}_N(\alpha+D,\beta,k_{t_1},\ldots,k_{t_N},t)}. \tag{B.1}$$

Using the change of variables $q=k_t^{-1}p$, the integrals $\bar{C}_N(\cdot)$ can be rewritten as follows:

$$\bar{C}_N(\alpha+D,\beta,k_{t_1},\ldots,k_{t_N},t)=k_t^{\alpha+D+\beta+N-1}C_N(\alpha+D,\beta,k_{t_1},\ldots,k_{t_N}),$$

where

(B.2)

$$C_N(\alpha+D,\beta,k_{t_1},\ldots,k_{t_N})\equiv\int_0^1 q^{\alpha+d-1}(1-q)^{\beta-1}\left(\frac{1}{k_{t_1}}-q\right)\cdots\left(\frac{1}{k_{t_N}}-q\right)dq.$$

Notice $C(\cdot)$ does not depend on t. The posterior mean in (B.1) is now

$$k_t\frac{C_N(\alpha+D+1,\beta,k_{t_1},\ldots,k_{t_N})}{C_N(\alpha+D,\beta,k_{t_1},\ldots,k_{t_N})}. \tag{B.3}$$

The computation of the integrals $C_N(\cdot)$ can be performed in a simple manner using the inductive formulae that follow. Obtaining these is straightforward from the definitions (B.2).

$$\begin{aligned}C_N(\alpha+D,\beta,k_{t_1},\ldots,k_{t_N})=&\frac{1}{k_{t_N}}C_{N-1}(\alpha+D,\beta,k_{t_1},\ldots,k_{t_{N-1}})\\&-C_{N-1}(\alpha+1+D,\beta,k_{t_1},\ldots,k_{t_{N-1}})\end{aligned} \tag{B.4}$$

for $N=1,\ldots,T-1$ and $D=0,1,\ldots,T-1-N$. When $N=0$,

$$C_0(\alpha+D,\beta,-)=B(\alpha+D,\beta),$$

that is, the Beta function. Finally, notice that

$$B(\alpha+D,\beta)=\frac{\alpha+D-1}{\alpha+D-1+\beta}B(\alpha+D-1,\beta). \tag{B.5}$$

The posterior means for all possible histories, given α, β, and the sequence of k's, can be computed starting from the value of $B(\alpha,\beta)$ and applying (B.5), (B.4), and (B.3) repeatedly.

Notice that the state space the birth dates of all the children that are alive are among the state variables of the dynamic programming problem in Section 4.1. To keep the size of the state space tractable for estimation, the woman's life cycle was broken into four subperiods and only the total number of survivors born in each of the subperiods was included in the state space. Thus, when the integrals in the second factor of (B.3) are computed, the sequence of support upper bounds is approximated by a step function. Within each subperiod, k_t is assumed to be constant and equal to the k_t in middle of that subperiod. The sequence of posterior means does not become a step function, Because the full sequence of ks is still used in the first factor of (B.3).

C. Completed Fertility and Replacement Rates. In this section, I derive expressions that relate the replacement rates defined in Section 5 to the average number of births and the average number of surviving children per woman in a population. I use these expressions to formalize the links between changes in infant mortality, replacement behavior, and fertility.

Suppose for the sake of simplicity that all women in the population have the same utility and child cost parameters and the same beliefs Γ about the distribution of infant mortality risks. The only sources of heterogeneity are the unobserved infant mortality endowments, with distributions P and the realizations of permanent and transitory unobserved components of birth costs. Fertility behavior in this population is described by $\mathcal{F}$, the set of birth probabilities conditional on all possible states.

Consider the following recursive computation giving the expected number of future births for women in a given state:

$$
\begin{aligned}
l_t(s) &= [1 - \mathrm{pr}_{1t}(s)]\, l_{t+1}(s, b_t = d_t = 0) + \mathrm{pr}_{1t}(s) \\
&\quad \cdot [1 + \bar{p}_t(s) l_{t+1}(s, b_t = d_t = 1) + (1 - \bar{p}_t(s)) l_{t+1}(s, b_t = 1, d_t = 0)] \\
&= [1 - \mathrm{pr}_{1t}(s)] l_{t+1}(s, b_t = d_t = 0) + \mathrm{pr}_{1t}(s) \\
&\quad \cdot [1 + l_{t+1}(s, b_t = 1, d_t = 0) + \bar{p}_t(s) rep(t, s)]\,,
\end{aligned}
\tag{C.1}
$$

where $\mathrm{pr}_{1t}(s)$ is the probability of a birth conditional on the state, $\bar{p}_t(s)$ is the (aggregate) infant mortality rate observed for women in state s, and $rep(t, s)$ is the replacement rate. Recall that $\bar{p}_t(s)$ equals the mean of the (conditional) distribution of endowments. Note that the argument of l_{t+1} is the state at t updated with demographic events that occur between t and $t + 1$. Under Rational Expectations, beliefs Γ and the distributions of endowments P are consistent so $\bar{p}_t(s)$ is also the subjective infant mortality risk perceived by women in state s. However, it is useful to keep in mind the distinction between beliefs Γ and endowments P. In particular, fertility behavior $\mathcal{F}$ will in general depend on Γ but not on P.

For $t = 1$, (C.1) is expected life-cycle fertility; by repeated forward substitution we get

$$\begin{aligned} l_1 \equiv\ & E(B_T) = [1 - \mathrm{pr}_{11}(\cdot)]\, l_2(b_1 = d_1 = 0) \\ & + \mathrm{pr}_{11}(\cdot)\,[1 + l_2(b_1 = 1,\ d_1 = 0) + \bar{p}_1(\cdot) rep(1, \cdot)] \\ =\ & E(b_1 \mid \bar{p}_1 = 0) + [1 - \mathrm{pr}_{11}(\cdot)]\, l_2(b_1 = d_1 = 0) + \mathrm{pr}_{11}(\cdot) l_2(b_1 = 1,\ d_1 = 0) \\ & + \mathrm{pr}_{11}(\cdot)\bar{p}_1(\cdot) rep(1, \cdot) = \cdots = \\ =\ & E(B_T \mid P = 0, \mathcal{F}) + \sum_{t=1}^{T} \sum_{\{s \in S_t : D_s = 0\}} \pi(t, s \mid P = 0, \mathcal{F}) \mathrm{pr}_{1t}(s) \bar{p}_t(s) rep(t, s), \end{aligned}$$

where $\pi(t, s \mid P = 0, \mathcal{F})$ is the probability that state s will occur at t when fertility behavior is $\mathcal{F}$, conditional on the actual infant mortality endowments being zero. Thus, expected life-cycle fertility can be decomposed into the sum of two terms. The first term, which we denote by $\mathcal{B}$, is the average number of births that we would observe in a population of women with fertility behavior $\mathcal{F}$, if the actual probability of infant deaths was (unexpectedly) zero. The second term, which we denote by $\mathcal{R}$, is a weighted sum of replacement rates at all ages for women with no previous experience of infant deaths. It measures the contribution of replacement behavior to total life-cycle fertility. (Note that conditioning on $P = 0$ above is not the same as conditioning on $D = 0$ for the original population. $E(B \mid P = 0)$ does not depend on the process generating infant deaths, whereas $E(B \mid D = 0)$ does.)

Also, $\mathcal{B} = \sum_{t=1}^{T} \sum_{\{s \in S_t : D_s = 0\}} \pi(t, s \mid P = 0, \mathcal{F}) \mathrm{pr}_{1t}(s)$. If the mean infant mortality risk $\bar{p}_t(s)$ is a constant $\bar{p}$ across periods and states, then

$$E(B_T) = \mathcal{B} + \mathcal{B}\ \bar{p}\, \overline{rep},$$

where $\overline{rep} = \mathcal{R}/(\bar{p}\mathcal{B})$ is the weighted average of replacement rates across all states with $D = 0$ with weights $\pi(t, s \mid P = 0, \mathcal{F}) \mathrm{pr}_{1t}(s)$. Note that $\mathcal{B}$ and the weights $\pi(t, s \mid P = 0, \mathcal{F}) \mathrm{pr}_{1t}(s)$ depend on beliefs through fertility behavior, but not on the actual endowments. Replacement rates clearly depend on beliefs because they are a function of birth probabilities later in the life cycle. Not so obviously, they also depend on the actual endowments because these affect the relative probabilities of future demographic event histories. If we assume that, following a change in infant mortality risk, (1) beliefs stay fixed, or changes in beliefs have no effect on fertility behavior and (2) the effect of changes in endowments on replacement rates is small so we can write $\frac{d[\bar{p}\,\overline{rep}]}{d\bar{p}} = \overline{rep} + \bar{p}\frac{d\overline{rep}}{d\bar{p}} \simeq \overline{rep}$, then $d\,E(B_T) \simeq \mathcal{B}\ \overline{rep}\, d\,\bar{p}$. That is, the effect on fertility of a changes in infant mortality risk can be obtained from $\mathcal{B}$ and the average replacement rate. However, if women are rational, a reduction of infant mortality risk in the population should (eventually) be reflected in beliefs. If beliefs and fertility behavior change, the impact on fertility of reduced infant mortality risk cannot be predicted from replacement behavior alone.

Similar expressions relating replacement rates to the expected number of surviving children can also be derived. As above, we start from recursive formulation

and substitute forward to obtain

$$E(N_T) = \mathcal{B} + \sum_{t=1}^{T} \sum_{\{s \in S_t : D_s = 0\}} \pi(t, s \mid P = 0) \mathrm{pr}_{1t}(s) \bar{p}_t(s) \left(rep(t, s) - 1\right),$$

and under the same assumptions as above, $dE(N_T) \simeq \mathcal{B}\left(\overline{rep} - 1\right) d\bar{p}$.

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