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Mark Rosenzweig

T. Paul Schultz

ECONOMIC GROWTH CENTER

YALE UNIVERSITY

Box 1987, Yale Station
New Haven, Connecticut

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CONSUMER DEMAND AND HOUSEHOLD PRODUCTION:

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Mark R. Rosenzweig
University of Minnesota

and

T. Paul Schultz

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The Relationship Between Fertility and Child Mortality*

Two important demographic regularities are the strong positive correlations between birth rates and infant mortality rates across countries, communities and families at one point in time and the roughly parallel decline over time in mortality and fertility in developed countries. Within developed countries such as the United States, moreover, certain groups appear to exhibit both high fertility and high child mortality rates, for example, blacks. To the extent that public resources are allocated to improve child health and reduce child mortality as well as to help people avert unwanted births, it should be useful to understand the forces generating these noted correlations.

A number of hypotheses,with different implications for the relative efficacy of alternative health and family planning programs, have been offered to explain the association between births and child deaths. First, the associations could reflect biological relationships. Infant deaths and fertility are biologically linked in two ways: 1) a death of a child truncates breastfeeding and thus shortens the sterile period following a birth; exposure to the risk of conception increases, therefore, when infant death rates are higher, and 2) the probability of an infant's death may be biologically affected by the number of prior births;that is,birth order or the mother's cumulative fertility may directly affect the infant's health. A second hypothesis is that fertility and household investments in infant health are choices that jointly reflect the environment of the household--prices, resource constraints, health conditions. Third, parents' purposive fertility behavior may be responsive both to the anticipated exogenous prospects of child survival (hoarding) and to the realizations of infant deaths (replacement). In this paper we report estimates of both the

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biological and behavioral linkages between infant mortality and fertility in the United States, which take into account heterogeneity in health and parents' choice of health inputs.

I. Dynamic Optimizing Behavior, Health Heterogeneity and Health Production

Variations in child death rates across families may be due to exogenous differences in the health endowments of children or in the healthiness of residential areas as well as to differences in investments in children by parents. Clearly, simple associations between fertility and actual infant deaths cannot shed much light on how fertility responds to exogenous changes in the healthiness of an environment that are brought about, for example, by regulating pollution or eradicating malaria-carrying mosquitoes. Nor do simple associations indicate how family size affects infant mortality biologically. When prices and socioeconomic characteristics have been controlled, there remain unmeasured differences in tastes and in health endowments as confounding sources of the observed fertility-mortality associations. However, estimates can be obtained of both the biological relationship between fertility and infant mortality (fertility affecting infant survival) and the effect of exogenous changes in the health environment on fertility if data are available on (i) all of the important types of behavior affecting infant survival and (ii) prices and income constraints facing households.

Consider a family in which each child has a common exogenous family health "endowment" μ_i, which contains family-specific genetic and environmental attributes affecting child health. The endowment, μ_i, is assumed to be known by each family and to differ across families, and is thus one

source of health heterogeneity in the population. The health of child j at birth in family i, H_{ijo}, is given by the health production function

$$(1) \qquad H_{ijo} = \Gamma(Z_{ijo}) + \mu_i + \varepsilon_{ijo},$$

where the Z_{ijo} are prenatal inputs, including birth order, and ε_{ijo} is the stochastic component of health which is observed at the birth of the child. In the next period of the child's life, the health production function is:

$$(2) \qquad H_{ij1} = \Gamma(Z_{ijo}, Z_{ij1}, \varepsilon_{ijo}) + \mu_i + \varepsilon_{ij1},$$

where the Z_{ij1} are first-period postnatal behavior of parents (e.g., breast-feeding) and ε_{ij1} is the first-period stochastic component of health. Expression (2) embodies the assumption that the production of health is a cumulative process, with past inputs as well as past stochastic events having persistent effects.

In any dynamic optimizing model in which the health of children, and some subset of the inputs, Z_{ijk}, including family size, are arguments in the objective function, the demand for the prenatal inputs, Z_{ijo}, will be a function of prices p, income F, and μ_i, namely,

$$(3) \qquad Z_{ijo} = \psi(p, F, \mu_i).$$

The postnatal input levels, Z_{ij1}, however, will also be functions of the realized stochastic health disturbances observed at the birth of the child; that is,

$$(4) \qquad Z_{ij1} = \psi(p, F, \varepsilon_{ijo}, \mu_i).$$

In words, parents' consumption choices will reflect their awareness of the health endowments of their children. Parents will also adjust their consumption behavior to perceived exogenous changes in any one of their

children's health. For example, parents who expect to have children with a high risk of mortality (e.g., low birth weight) might seek prenatal care earlier in their pregnancies and plan to have fewer (or more) children than parents who expect to have children with greater health endowments or who are to be born in less risky environments. Moreover, for given information on endowments, parents may also prolong breastfeeding or provide more resources to children who appear to be vulnerable or who contract an illness after birth.

The remedial and anticipatory behavior of parents means that, given that both ε_{ijk} and μ_i are usually unobserved by the econometrician, the parental inputs that influence child health will not be uncorrelated with the residuals in the health production functions (1) and (2). That is, $\text{cov}(Z_{ijk}, \varepsilon_{ijk}) = 0$, $\text{cov}(Z_{ijk}, \mu_i) \neq 0$ for $k = 0, 1$ and $\text{cov}(Z_{ij1}, \varepsilon_{ij0}) \neq 0$, since the random component of the birth outcome is by definition unforeseen by the parents during pregnancy. Ordinary least squares regression methods will thus not yield consistent estimates of the parameters of the health production functions, even if all inputs are observed. The difference between the actual and predicted health outcome, based on actual inputs levels and consistent estimates of the production function parameters, would approximate the health endowment with a random error. Regressing a prenatal behavioral input, such as birth order, on this calculated residual of the health production function from either (1) or (2) yields an estimate of the relationship between changes in anticipated exogenous family health endowment (genetic and environmental) of children and the input response of parents. But since the calculated residual measures the health endowment with error, this estimate of the response of inputs to health endowment is biased toward zero. Estimates of these relationships between the health production function residuals and the input behavior of the parents after the child's birth, however, would mix the effects of "endowment" and input adjustments by parents based on new information acquired after the child's birth, since

postnatal input behavior is a function of both ε_{ijo} and μ_i, as in (4). Consequently, the pure expected endowment effects on health-related choices, such as of fertility, can only be assessed by analysis of prenatal behavior.

II. Estimation Strategies

Consistent estimates of the production function for infant mortality provide measures of the biological effect of family size on mortality as well as the information needed to estimate the effect of an exogenous change in the health environment on parental fertility behavior. The theoretical framework suggests that such consistent estimates can be obtained by estimating the demand equations for the Z_{ijk}, (3) and (4), and by using the fitted values of the Z_{ijk} in estimating the mortality production function. Prices and income, as long as they are uncorrelated with the μ_i and ε_{ijk}, serve as identifying variables, since such variables influence all health input and consumption choices, but do not directly affect mortality.

This two-stage estimation method has been applied to characterize the biological effects of parents' behavior on birthweight, gestation, and the rate of fetal growth (Rosenzweig

and Schultz). These estimates also show the significance of heterogeneity bias . However, prior work on the fertility-mortality relationship considering either heterogeneity or biological factors has not fully taken into account the stochastic-dynamic aspects of the health behavior by parents. Direct estimates of the "effect" of child mortality on fertility might be obtained using two-stage least squares, controlling for prices and income. However, such estimates (assuming that identification is theoretically justified), do not provide information on how parents would alter their fertility if the exogenous healthiness of their environment changes. Rather, the technique simulates an experiment in which parents are assigned a child death rate over which they have no control, i.e., the effect of the mortality endowment cannot be altered by changes in household resource allocations, as it can be in the real world.

The biological determinants of child mortality, such as (1) and (2), have been estimated with explicit attention to parental or regional health heterogeneity by Olsen and Wolpin. In their study, a waiting-time regression method was used to estimate a child mortality production function based on within-family differences in children's time to death and in such postnatal health inputs as breastfeeding and child spacing. However, as can be seen from (2), if H_{ij1} is taken as a latent index of survivability, differencing across children only purges out the family/area endowment, μ_f; inputs will still be correlated with the residual in the fixed family-effects model, since the child effects, ε_{ijo}, will differ across children, are observed by parents after a child's birth, and will influence parents' postnatal behavior. As a consequence, Olsen and Wolpin must assume that parents do not adjust

their input behavior in response to postnatal random shocks. The two-stage estimation approach which exploits variations in prices and income and the structure of the household demand model, however, yields production function estimates which are consistent in the presence of such dynamic behavior.

III. Fertility and Infant Mortality: Estimates of Biological and Behavioral Relationships in the United States

In this section we discuss preliminary estimates of the biological effects of birth order on infant death in the U.S. and the effects of changes in the expected exogenous component of mortality on fertility and other types of parental behavior using the two-stage demand/production estimation procedure. The data are from the 1967, 1968, and 1969 National Natality Followback Surveys (USDHEW), which provide information on national probability samples of approximately 10,000 legitimate U.S. live births. The data indicate whether or not each child has died prior to the time when the parents responded to the survey questionnaire and the interval between the child's birth date and the date of the questionnaire (average of 19 months). The number of infant deaths reported is 209. From the survey information, six forms of behavior, four prenatal, were selected as potential biological determinants of infant mortality--birth order, delay after conception in seeking medical care during the pregnancy, mother's rate of smoking while pregnant, mother's age, duration of breastfeeding, and delay by the mother after the child's birth before returning or going to work. In addition, the race of the mother and the child's sex were included as exogenous determinants of infant mortality.

To obtain the instrumental variables needed to estimate the mortality production function, state and county level information on health programs and prices were merged with the household socioeconomic and health data. The regressors in the demand equations for the six health-related behavioral variables included the schooling of the parents, the age-adjusted income of the father, local governmental health and hospital expenditures per capita, the number of hospitals and health departments with family planning services per capita, obstetrician-gynecologists per capita, prices of cigarettes and milk, the total and female unemployment rates, metropolitan area location and size, and the regional mix of employment by industry group(described more fully in Rosenzweig and Schultz). The set of variables explained a statistically significant proportion of the variance for all six inputs, with R^2s ranging from .03 for cigarette smoking to .14 for number of births and delay in obtaining prenatal medical care (reported in Appendix Table A).

The first two columns of Table 1 report the coefficients of the infant death production function estimated by OLS and two-stage least squares. Since the dependent variable is dichotomous and the residuals heteroscedastic, the reported t-values are not unbiased. Thus, while the TSLS parameter estimates should be consistent, statistical tests may be misleading.[1/] Nevertheless, the OLS and TSLS coefficient estimates differ substantially. For example, while the OLS estimates suggest that delay in prenatal care has a small and <u>negative</u> effect on infant mortality, the TSLS results confirm the anticipated finding that such delay considerably increases the probability of infant death. More importantly, the inconsistent OLS parameter estimates indicate that higher birth order is associated with higher infant mortality while the older

the mother the less likely are the chances of an infant death. The TSLS estimates suggest just the opposite--higher fertility directly lowers child mortality and delay in childbearing increases the mortality rate. Neither the OLS or TSLS estimates indicate that mother's smoking while pregnant is significantly related to the probability of child survival; this finding contrasts with other evidence that smoking reduces birthweight (Rosenzweig and Schultz). The OLS estimates suggest there is no significant mortality relationship with mother's work after her birth, while TSLS estimates indicate that if the mother works, infant mortality is less, suggesting that benefits accruing from the mother's earnings outweigh any decrease in her time in child care. Black babies have considerably higher mortality rates, even when the black-white behavioral differences in mother's age at birth, fertility, smoking, breastfeeding and employment are "controlled."

While the heteroscedascity of the residuals does not permit the conventional tests of the statistical significance of heterogeneity bias (Durbin), the striking substantive differences between the OLS and TSLS estimates suggest that parents do respond to differences in their genetic/environmental health endowments. Columns (3) through (6) of Table 1, which report the estimated effects of the production function calculated residuals on the four prenatal inputs, provide further evidence on the responses of these inputs to exogenous but anticipated differences in health endowments. These lower bound estimates of the effects of the family-specific health endowment are substantial. In particular, the results suggest that the average number of children per mother would increase by one-sixth of a child, if a child mortality rate of .1 were anticipated.

The positive associations between infant mortality

Table 1

Estimates of the Production Function for Infant Mortality

and the Effects of Anticipated Mortality on Fertility and Other Forms of Behavior

	Dependent Variables					
	Infant Mortality		Birth Order	Prenatal Care Delay	Cigarettes Smoked	Mother's Age
Estimation Technique	OLS (1)	TSLS (2)	OLS (3)	OLS (4)	OLS (5)	OLS (6)
Independent Variables (sample means)						
Residual [a] (0.00)[b]	--	--	1.57 (13.29)	-1.89 (20.2)	1.75 (3.18)	1.23 (3.46)
Birth order[c] (2.51 children)	.0129 (10.4)[d]	-.0171 (2.46)				
Prenatal Care Delay[c] (2.67 months)	-.00145 (1.16)	.0332 (3.80)				
Cigarettes Smoked[c] (4.68 cigarettes/day)	.00002 (.00)	-.00081 (.42)				
Mother's Age[c] (25.1 years)	-.00119 (2.96)	.00304 (1.81)				
Whether Breastfed[c] (.260)	-.00510 (1.28)	-.0316 (1.51)				
Whether Returned to Work[c] (.257)	-.00180 (.45)	-.106 (2.75)				
Black (0.170)	.0290 (5.87)	.0474 (4.77)				
Female (.490)	-.00742 (2.14)	-.00707 (1.83)				
Intercept	.0278 (2.02)	-.0615 (3.32)	2.50 (123)	2.67 (164)	4.66 (50.4)	25.1 (4.01)
F	26.7	10.9				
N = 8119						

[a] From TSLS infant mortality estimates

[b] Sample mean

[c] Endogenous variable

[d] Absolute value of t values in parentheses beneath regression coefficients. See text for their potential bias.

and fertility seen in the gross correlations and in our OLS estimates appear to mask a negative biological effect of birth order on infant mortality (column 2) and a substantial positive behavioral response of fertility to the anticipation of a higher mortality risk (column 3). If these response patterns are representative of historic levels, the decline in child mortality that has occurred in the last century in the developed countries could account for about a fifth of the coincident decline in fertility.

The residual estimates also indicate that the responses to a ceteris paribus exogenous increase in anticipated infant mortality are not confined to fertility behavior; mothers appear to seek prenatal medical care significantly earlier when the exogenous mortality rate is higher. In this instance the input response is compensatory, because it reduces somewhat the effect of the higher exogenous mortality. Age at birth and mother's smoking, on the other hand, appear to decrease as the survival endowment of infants increases.

Finally, we might look for a biological-environmental basis for differences in black-white fertility and other input behavior, as our evidence suggested that black mothers experience exogenously higher infant mortality rates than do white mothers. However, despite the exogenously lower survival probabilities of black infants compared to white infants, black mothers obtain prenatal medical care later than do white mothers, even though our TSLS estimates indicate that such delay increases the risk of their child's death, and that in the total population mothers with higher expectations of child mortality seek medical care earlier. Further study of this anomalous pattern of use of prenatal medical services by the black population is warranted.

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FOOTNOTE

1/ While logit estimates were calculated, the statistical properties of the two-stage logit estimates, where the first-stage equations are linear, are not well established.

Appendix Table A
Estimates of Linear Approximations of Input Demand Equations *

Independent Variables	Birth Order (1)	PreNatal Care Delay (2)	Cigarettes Smoked (3)	Mother's Age (4)	Whether Breastfed (5)	Whether Returned to Work (6)
Mother's Education:						
High School Incomplete	-.706	-.369	1.28	-2.46	-.0566	-.0642
	(8.53)	(5.57)	(3.16)	(9.76)	(2.82)	(4.15)
High School Complete	-1.02	-.736	-.408	-.711	-.0457	-.101
	(12.4)	(11.2)	(1.01)	(2.85)	(2.30)	(5.03)
College Incomplete	-1.21	-.834	-.720	-.376	.0497	-.147
	(12.6)	(10.19)	(1.53)	(1.29)	(2.13)	(6.27)
College Complete	-1.40	-.819	-.950	1.22	.146	-.207
	(12.7)	(9.29)	(1.76)	(3.64)	(5.44)	(7.66)
Father's Education:						
High School Incomplete	-1.03	-.137	.612	-2.97	-.0244	-.0275
	(13.4)	(2.22)	(1.63)	(12.7)	(1.31)	(1.46)
High School Complete	-1.24	-.315	-.049	-3.12	.0089	.00837
	(16.8)	(5.29)	(.13)	(13.8)	(.49)	(.46)
College Incomplete	-1.26	-.353	-.685	-3.24	.0889	.0191
	(14.4)	(5.02)	(1.59)	(12.1)	(4.15)	(.88)
College Complete	-.983	-.398	-.433	-1.61	.173	.0843
	(10.2)	(5.19)	(.90)	(5.52)	(7.43)	(3.58)
Log of Husband's Life Cycle Income	.0989	-.277	.371	.350	-.0400	.0496
	(3.02)	(10.6)	(2.31)	(3.50)	(5.01)	(6.15)
1967	.0792	-.479	1.46	.250	.0107	.0258
	(.55)	(4.16)	(2.07)	(.57)	(.93)	(2.21)
1968	-.019	-.162	.376	-.0071	.0050	-.0022
	(.42)	(4.42)	(1.67)	(.05)	(.45)	(.20)
Metropolitan Residence	-.107	-.162	.423	.336	.0213	.0162
	(1.70)	(3.21)	(1.37)	(1.76)	(1.40)	(1.05)
SMSA Size ($x10^9$)	-2.22	3.04	166.	77.9	34.6	1.26
	(.18)	(.29)	(2.67)	(2.01)	(.11)	(.41)
Health Expenditures	-.958	-.238	7.23	-5.48	-.0099	-.106
	(.78)	(.24)	(1.19)	(1.46)	(.03)	(.35)
Health Dept. Family Planning	-8393.	-5257.	26491.	-20068.	-1115.	-364.
	(2.32)	(1.82)	(1.49)	(1.82)	(1.26)	(.41)
Cigarette Price (x100)	33.5	-.0363	-23.7	7.09	.0845	1.30
	(3.08)	(.00)	(.44)	(.21)	(3.19)	(.48)
Cigarette Price Squared ($x10^4$)	-51.4	-.545	40.9	-.657	-.0013	-1.49
	(3.08)	(.04)	(.50)	(.13)	(3.23)	(.36)
Milk Price ($x10^3$)	.0051	-.0050	.0417	.0174	-.0057	1.32
	(.46)	(.56)	(.77)	(.52)	(2.12)	(.48)
Hospital Family Planning	-26317.	-7631.	142330.	22781.	-5230.	-2553.
	(1.82)	(.66)	(2.00)	(.52)	(1.48)	(.72)
Population Per Doctor ($x10^5$)	.883	3.67	-26.3	-11.9	-.840	-1.29
	(.28)	(1.49)	(1.74)	(1.26)	(1.12)	(1.72)
OBYGYN Per Capita	-950.	1676.	6325	7422	52.4	116.
	(.70)	(1.56)	(.96)	(1.81)	(.16)	(.35)
Manufacturing Jobs ($x10^3$)	-1.27	.539	.400	1.41	.397	.307
	(2.28)	(1.21)	(.15)	(.83)	(2.93)	(2.24)
Black	.734	.537	-2.53	.0947	-.0407	-.195
	(13.1)	(11.9)	(9.19)	(.55)	(2.97)	(14.1)
Service Jobs ($x10^3$)	-4.03	-.179	-8.26	-18.6	-.0536	.344
	(1.84)	(.10)	(.77)	(2.79)	(.10)	(.64)
Government Jobs ($x10^3$)	-2.49	-1.74	-7.26	1.34	.0948	.443
	(2.02)	(1.76)	(1.20)	(.34)	(.32)	(1.46)
General Unemployment ($x10^3$)	-.977	4.92	30.1	-17.4	-1.92	.896
	(.20)	(1.28)	(1.28)	(1.19)	(1.65)	(.76)
Female Unemployment	2.89	.242	-35.8	-2.34	4.64	-.482
	(.64)	(.07)	(1.63)	(.17)	(4.23)	(.44)
Hospital Beds Per Capita	23.3	7.56	118.	218.	-18.1	-12.1
	(1.01)	(.41)	(1.05)	(3.11)	(3.23)	(2.14)
Sales Tax on Cigarettes (x100)	-.0411	-.0596	-.295	.189	-1.90	-1.71
	(.84)	(1.52)	(1.22)	(1.26)	(1.59)	(1.42)
Infant is Female	.0422	.0233	-.0325	.188	-.0019	-.0096
	(1.10)	(.75)	(.17)	(1.61)	(.21)	(1.02)
Intercept	-1.27	6.27	4.37	22.3	-5.23	1.009
	(.70)	(4.29)	(.49)	(4.01)	(1.18)	(2.25)
R^2	.1364	.1450	.0313	.0970	.0833	.0585
F	42.6	45.7	8.72	28.9	24.5	16.7

* Absolute value of t ratios in parentheses beneath regression coefficient, although they are not unbiased in the case of the discrete dependent variables in columns (5) and (6).