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Demand for Child Health and Fertility in Rural India

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Abstract

The determinants of child health, mortality, and fertility are analyzed within a household production framework which incorporates the child's genetic health endowment along with relevant prices. Child height-for-age and weight-for-height are the proxies for child health in the estimated equations. But because child stature reflects both genetic and environmental factors, a proxy for genetic endowment is included, namely, the mother's height and weight-for-height. The empirical results confirm the conventional wisdom of the health literature that child height represents cumulative health, and child weight-for-height, transitory health. This confirmation follows from the result that child height is determined by measures of the longer-term inherited income and health position of the household, namely, father's schooling and household assets. Child weight-for-height is determined by mother's schooling, wage and weight-for-height, and father's wage. Thus, the use of the two indicators in any evaluation of child health in some sense complement each other.

DEMAND FOR CHILD HEALTH AND FERTILITY IN RURAL INDIA*

1. Introduction

Child health and survival are important issues in rural India. High fertility, child mortality, and morbidity have direct implications for both the productivity and well-being of the population, and, in the aggregate, for the economic development and growth of the region. The economic, technological and institutional determinants of child health and fertility are analyzed here within a household production framework which incorporates the effects of the child's genetic health endowment as well as the child's earnings, which may be a relatively important component of family earnings (Part 2). Data collected under the auspices of the International Crops Research Institute in the Semi-Arid Tropics (ICRISAT) are analyzed. Retrospective fertility histories of 401 women , and anthropometric measurements of the parents and children in the six village sample collected by investigators associated with or sponsored by ICRISAT complements community and household level information (Part 3).

Child height-for-age and weight-for-height are the proxies for child health in the estimated equations, because it is well known that child growth reflects the confluence of genetic and environmental (nutrition, exposure to infection and disease) factors. By controlling for the genetic health endowment, better measures of the effect of other health and nutritional inputs on child height and weight-for-height are estimated. This genetic health endowment of the child is proxied by the parent's height and weight-for-height, to obtain more efficient estimates (Part 4).

Joint estimation of the household demand for numbers of children and health of children illuminates why price effects cannot generally be derived from the theoretical model (Part 5). While the empirical results imply that

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numbers and health of children are complements, and schooling and health of children are substitutes, and generally, confirm that anthropometric indicators help us to understand investments in children (numbers, health and schooling), it is important that these results be replicated with other Indian data and data from other countries before drawing any specific policy recommendations.

2.Theory

The determinants of fertility and child health are examined here within the framework of the household production model. Essentially, the model posits that parents derive satisfaction from numbers and the health of their children. Their problem is to maximize their utility subject to the numerous income, time and production constraints facing them.

The budget constraint assures that full household income does not exceed household expenditures. The time budget constraints assure that the time spent in the market and household activities does not exceed total time. There are three other less conventional constraints peculier to the application of the new household economics to rural agricultural households. In such households, the production function for crops or animal products, say, becomes important to the analysis. Time of the family members, parents and children alike, are combined with various household productive assets, like land, livestock, and farm tools, and less tangible, more qualitative, assets like the managerial efficiency of the farmer, to produce the goods the household sells in the market. The fourth constraint is the biological child birth production function. Parent's consumption, environmental quality, i.e., water and sewage facilities, as well as parent's genetic qualities determine fecundity. Fecundity and contraception are combined to realize a desired family size. The fifth constraint is the production function for child health. There are several levels of inputs: individual, household and community, that are combined in the estimating equations in a linear fashion, thus ignoring or approximating a more complicated interactive mechanism (Martorell (1982)). The child's genetic endowment, namely, a predisposition to a certain health status, or a predisposition to infectious diseases, along with the child's nutritional intake, and his household and village environment, are combined to produce child health and nutritional status. Parent's effective time should also be included in the production of their child's health. The literature suggests how parent's education affects their child's health: more educated parents are more aware of preventative and curatuve health and nutrition measures, or they are less fatalistic about illnesses, and thus, more likely to seek medical attention for sick children, etc. (Wolfe and Behrman (1980)).[1]

A simple model in which the production functions for the household commodities included in the household's utility function, namely numbers (N) and health (H) of the children, are linearly homogeneous, in order to be able to write down cost functions linear in prices and wages,

1a) $P_N = p_N x_N + w_M t_{MN}$

1b) $P_H = p_H x_H(e) + Hwt_{KH}(e) + w_M t_{MH}(e) + w_F t_{FH}(e)$

p_i are the prices of the market goods that are used in the production of household commodity i, t and x are the marginal input coefficients of the time and goods inputs, for j = N, H, L and i = K, M, F. K indexes each child in the family, M is mother and F, father.

In this simple model, the time of the mother, t_{MH}, father, t_{FH}, and children, t_{KH}, are used to produce child health, while only the time of the mother is used to produce numbers, t_{MN}, and the time of the child is used to produce leisure. w_M and w_F are the wage of the mother and father respectively, and Hw, the child wage which is assumed proportional to the level of their health "capital." w then is the rental rate per unit of health. v is unearned income of the household. The average health endowment of the children, e, is assumed to complement all factor inputs in the production of health, namely time of the parents and children, and a bundle of purchased health goods, x, such that dx/de, $dt_{iH}/de > 0$. Bundles of market goods used in the production of N and L as well, purchased at market prices p_N and p_L are likewise inputs. The time constraints facing the household,

2a) $T_{KM} + T_{KH} + T_{KL} = 1$

2b) $T_{MN} + T_{MH} + T_{MM} + T_{ML} = 1$

2c) $T_{FH} + T_{FM} + T_{FL} = 1,$

prescribe child market work as T_{KM} units of time. (Leisure is henceforth not explicitly considered.)

Full household income must equal expenditures,

3) $HwN + w_M + w_F + v = P_N N + NP_H H$.

where v is unearned household income.

Maximization of household utility, subject to the production (1), time (2) and budget (3) constraints yields the following commodity shadow prices that are functions of the optimal bundle of numbers and health of children.[2]

4a) $\Pi_N = -wH + P_N + HP_H$

4b) $\Pi_H = -wN + Np_Hx_H + 2NwHt_{KH}$

Under certain conditions (Pollak and Wachter (1977)), the commodity demand equations can be written, not in terms of endogenous shadow prices, but rather in terms of the exogenous factors, namely, wages of the parents and children, prices of the purchased goods used in the production of health and fertility, the child's genetic endowment, and the capital endowment of the household (source of the flow of unearned income).

5a) $N = N(w, w_M, w_F, v, p_N, p_H, e)$

5b) $H = H(w, w_M, w_F, v, p_N, p_H, e)$

Thus, the estimating equations are part of a system of reduced form demand equations conditioned on the same set of exogenous variables. The signs of the compensated price effects can only be determined if restrictions are placed on the general model, but even with the convenient assumptions of additive separability of the parent's utility function and goods normality,[3] only one effect can be unambiguously signed. Since an increase in unearned income produces no offsetting substitution effect, the effect on the demand for both numbers and health of children is positive.

Other price effects cannot be determined. Since child wages are both a component of the opportunity cost and a reflection of the return to the child's health, the effect on the demand for child health of an increase in child wages is ambiguous in sign. Similarly, the demand effect of the parent's wages cannot be predicted, because the wages enter into the production cost of the commodities (numbers and health) as well as determine the income of the household. Although these price effects cannot be unambigously predicted by the theory, the empirical results may illuminate the regularities.

3.Data and Specification

Two empirical issues are addressed: 1) the estimating equation must be specified, and 2) empirical measures for the endogenous (fertility and child health) as well as the exogenous (wages, prices and endowment) variables of the theoretical model must be chosen. Child mortality is estimated as a check on consistency, in the sense that mortality is the lower bound to child health.

The reduced-form demand equation for numbers and health of children were derived from the first order conditions, and can be written,

5a) $N = N(w, w_F, w_M, v, p_N, p_H, p_L, e)$

5b) $H = H(w, w_F, w_M, v, p_N, p_H, p_L, e)$

The prices of the time of children, mother and father, are w, w_M, and w_F respectively, and p_N, p_H, and p_L, the prices of the purchased goods. The genetic health endowment of the children is e.

The estimating equations are linear combinations of the conditioning varibles, called Z hereafter,

6a) $N_j = Z_j + u_j$

6b) $H_{ij} = Z_{ij} + (f_j + c_{ij})$,

where i indexes children within the household, and j, the household. Because the equations are conditioned on the same set of variables, single equation OLS methods are appropriate, if error terms among children within the household are uncorrelated. Correcting for such a correlation in the child health regressions involves a simple transformation of the information matrix, (H_{ij}, Z_{ij}), that accounts for a specific kind of correlation that may arise from omitted household background varibles, that would be the same for all children within a household, f_j (Hausman (1980), Maddala (1978), Judge, et. al (1981)) (see footnote 24). Thus, $f_j + c_{ij}$ is the error term in the health equation, and u_j in the fertility equation.

The data analyzed are cross-sectional data from six villages in semi-arid tropical India collected by ICRISAT from 1975 to 1979. The villages were selected to be representative of all the villages in their respective talukas (districts) with regard to forty agronomic, climatic, and social varible, such as the modal value of land-use and cropping patterns, extent of irrigation, population size and livestock, etc. There are electricity and wells in all six villages, private medical practitioners in two, and a primary health care center in one. There are primary schools in all the villages, but upper primary schools in only four and high schools in two. In each village, ten households were randomly chosen from four (stratified) landholding-size groups: landless, and small, medium and large landholding farmers.[4]

Retrospective fertility histories of 400 women, and anthropometric measurements of the parents and children were collected by independent investigatores sponsored by ICRISAT, so that the data had to be combed to insure correct matching with the original ICRISAT sample.

The sample analyzed here consists of 169 women, representing primary families with schoolaged children (children between the ages of 6 and 19). At most, 133 subsidiary families in the extended family households, that is, families headed by children or siblings of the head of the household, were excluded, as were families headed by widows, widowers, and families in which the parents were separated or divorced.

The 354 school-aged children of these women comprise the sample in subsequent child health regressions.[5] Pre-school age children were excluded because of my interest in the simultaneous schooling and health choice, and as well, because of the question of the most appropriate measure of attained child health in a sample of children most of whom have not grown up. A reading of the health literature (noted below) indicates that the stature of the child, here used as the proxy for health, is determined for the child in the preschool years. There is experimental evidence that the critical periods of brain growth occur in the first years of the child's life, and when the child is severely deprived of nutrition in these years, mental capacity is irremedially stunted . The child's body may be able to catch up when the nutritional environment is improved, provided the child has not been severely deprived in critical periods of growth. Thus, the position may be taken that mental and physical health may be determined in some sense in preschool years, so that an hropometric measures of children beyond preschool age represents their lifetime health status.

Actual fertility, namely the number of children ever born to the women, is the dependent variable in the fertility regression equation.[6] The ratio of actual mortality, the number of children who had died, to actual fertility is the dependent variable in the child mortality regression equation. Age of the mother, and age squared, are included to purge the dependent variable of age and cohort effects, so that the dependent variable can be considered to be a good proxy for the completed or desired fertility of the woman. The cohort effects are apparent in the summary tables (Tables 1 and 2). The village sample appears to be undergoing fertility decline. The oldest cohort of women (cohort 9, column 4) stopped bearing children at a later age than the next older cohort (cohort 8) — 37 years of age versus 34. Age effects are a problem because of the inclusion of women who obviously are at risk of

TABLE 1

LAST BIRTH EXPERIENCE (THE ICRISAT DATA, RURAL INDIA)

Cohort*	Mean Age	Birth in Year	Age Last Birth	Date Last Birth (mo. prior)	Women Pregnant	Women Breastfdng.	Last Child Alive	Sample Size (Women Childless)
1 (ages 10-14)	11.8 (1.5)	0	0	0	0	0	0	10 (10)
2 (ages 15-19)	17.4 (1.3)	.18(.39)	17.05(1.1)	.68(.8)	.18(.4)	.31(.5)	.89(.3)	49 (30)
3 (ages 20-24)	21.8 (1.4)	.37(.5)	20.48(1.6)	1.24(1.4)	.15(.4)	.54(.5)	.82(.4)	41 (8)
4 (ages 25-29)	26.5 (1.7)	.16(.4)	23.94(2.5)	2.58(2.1)	.16(.4)	.45(.5)	.88(.3)	37 (4)
5 (ages 30-34)	31.8 (1.4)	.08(.3)	26.75(3.5)	5.06(3.5)	.03(.2)	.32(.5)	.97(.2)	37 (1)
6 (ages 35-39)	36.7 (1.6)	.07(.3)	29.76(4.2)	7.05(4.3)	.02(.1)	.14(.3)	.97(.2)	42 (4)
7 (ages 40-44)	42.3 (1.5)	0	31.67(5.6)	10.74(5.5)	0	.02(.2)	.92(.3)	41 (2)
8 (ages 45-49)	46.0 (1.3)	.05(.2)	34.62(6.7)	11.32(6.6)	0	.11(.3)	.91(.3)	37 (3)
9 (over 50)	60.2 (8.6)	0	37.10(9.6)	23.03(9.8)	0	0	.82(.4)	98 (5)

*Birth cohort of the mother

Standard deviations in parentheses

TABLE 2

CUMULATIVE FERTILITY AND FAMILY SIZE (THE ICRISAT DATA, RURAL INDIA)

Cohort*	Mean Age	Sons Born	Daughters Born	Children Born	Sons Living	Daughters Living	Children Living	Sample Size (Childless)
1 (ages 10-14)	11.8 (1.5)	0	0	0	0	0	0	10 (10)
2 (ages 15-19)	17.4 (1.3)	.22(.4)	.14(.4)	.37(.5)	.20(.4)	.12(.3)	.33(.5)	49 (30)
3 (ages 20-24)	21.8 (1.4)	.75(.8)	.71(.9)	1.46(1.2)	.56(.7)	.56(.7)	1.12(.9)	41 (8)
4 (ages 25-29)	26.5 (1.7)	1.35(1.2)	1.11(1.1)	2.45(1.4)	1.11(1.0)	.89(1.0)	2.00(1.2)	37 (4)
5 (ages 30-34)	31.8 (1.4)	2.22(1.2)	2.03(1.2)	4.24(1.9)	1.59(.8)	1.43(1.0)	3.03(1.3)	37 (1)
6 (ages 35-39)	36.7 (1.6)	2.40(1.7)	2.40(1.7)	4.81(2.7)	1.79(1.3)	1.74(1.2)	3.52(2.0)	42 (4)
7 (ages 40-44)	42.3 (1.5)	3.26(1.9)	2.44(1.7)	5.70(2.8)	2.10(1.5)	1.71(1.4)	3.80(2.1)	41 (2)
8 (ages 45-49)	46.0 (1.3)	3.11(2.1)	2.86(2.0)	5.97(3.6)	2.14(1.4)	1.89(1.4)	4.03(2.3)	37 (3)
9 (over 50)	60.2 (8.6)	3.23(2.0)	3.23(2.0)	6.47(3.0)	1.95(1.2)	1.80(1.5)	3.74(2.0)	98 (5)

*Birth cohort of the mother
Standard deviations in parentheses

childbearing, namely those less than fifty years of age, for the main reason of enhancing the sample size. Hence, actual fertility is standardized by inclusion of the age of the mother and her age squared in the regression equation. According to the proposed model of the interrelated system of household demands, the residual variation in actual fertility is determined by wages, prices and the genetic endowment.

Analogously, if children who have not attained full stature are excluded from the sample, the data in the remaining sample would not support estimation. Moreover, children who are no longer living with their parents are not observed. Therefore, health status is proxied by two anthropometric measures observed for children currently living with their parents: height for age and weight-for-height. Age and sex interaction variables are included in the regressions to purge the dependent variables of the age and sex effects in the data.[7] Namely, some children are still growing, and boys and girls may grow at different rates. Stature and rates of growth reportedly differ between Indian boys and girls (Eveleth and Tanner (1980)), i.e. boys generally mature at later ages, and grow to a taller height, with greater weight-for-height, than girls. More direct data on health status are not available to allow us to get beyond the anthropometric indicators of child health.

Height and weight-for-height reflect the nutritional environment of the child to the age of the child when measured by ICRISAT. Substandard height suggests that the child suffered mild to moderate malnutrition--the response of the body to a mildly stressful nutritional environment is diminished growth, a phenomenon called nutritional "stunting." Weight-for-height is not as accurate an indicator of chronic mild malnutrition. Rather the child's body may be able to maintain a "normal" weight-for-height relationship in the range of mild to moderate malnutrition. But in the case of severe malnutrition, when even the basal metabolic requirements are not covered by the energy intake, the body's reserve of fat and muscle must be used and the child's weight-for-height deteriorates. The phenomenon is called "wasting."[8]

However, the sample of Indian children reveals that many who are stunted have not been able to maintain a normal weight-for-height ratio (Table 3).[9] Indeed, the incidence of wasting and stunting is high in all age groups. One would expect in normal populations one-sixth of the children to be less than one standard deviation below the mean, and five-sixths "normal." Instead, one-sixth of the Indian children are normal and about half are stunted and wasted.[10]

There are two statistical problems inherent in the use of the age-specific anthropometric measures,[11] aside from the possibility of errors in the measurement of age. 1) the possibility of differential growth rates across the different children, and 2) the possibility of the "short" draw from the gene pool. Any child may be less than the village average height for his age, not because of malnutrition, but because he is either a late developer (as distinct from the concern that the longer growth period is itself the result of deficient nutrition), or because his potential stature is limited by genetic makeup. Thus, downward deviation from the village average may not necessarily be unhealthy, and true health status may be understated for some.[12] The steps taken to resolve these problems are tied with the problem of finding an empirical counterpart to the theoretical genetic concept of initial health endowment, e, in equations 1 and 5. In the theoretical model, recall that the outcomes of child health, and in general, numbers and quality of children, are conditioned on wages, prices and initial health endowment. In particular, initial health endowment is assumed to complement other inputs of time and health goods in the production of child health.

Table 3

Malnutrition in the Six Villages

Age	Stunted and Wasted	Stunted Not Wasted	Wasted Not Stunted	Normal
		%		
0-5	47.3	20.2	17.1	15.5
6-11	69.0	5.3	17.7	8.4
12-15	68.6	4.8	16.1	10.5
16-19	46.2	8.8	27.5	17.6
over 19	41.4	17.6	25.5	15.5

(1) The cross-tabulation of wasting and stunting follows the methods used by Waterlow (1972), where stunting is indicated by a height for age that is less than 90% (more than 1 standard deviation) the height of the Indian referene child of the same age and sex. Wasting is indicated by weight for height that is less than 90% of the weight for height of the referene child.

Genetic endowment of the child, although unobserved, has been proxied by the height of the mother or father.[13] This formulation is based on the recognition that the family gene pool is more restricted than the population (village) gene pool. The child's random draw from the family gene pool, however, must be subsumed in the random error term of the regression, because the child health data are cross-sectional, not longitudinal.[14] Obviously, a complex, non-linear and interactive mechanism that characterizes the transmission of genetic material from parent to child, has been here radically simplified.

Use of the proxy creates its own problems. If height of the parents reflects the parent's childhood environment, and to the extent that the environment persists intergenerationally (not unlikely in the rural Indian villages), then inclusion of the parent's height varibles will bias downward the coefficients of the variables that are used to characterize the household's current environment. Indeed, father's height is significantly correlated with the value of the land owned by the household and with the father's schooling. On the other hand, mother's height is uncorrelated with the household value of land. In these villages, a wife generally lives with the family of her husband and is economically dependent on them. Thus, the father's height is excluded, and the mother's height included, as a better proxy for the child's genetic endowment, in the sense that it will be less correlated with current environmental condition of the household. Mother's weight-for-height is included to represent the prenatal intrauterine environment of the child, and may be associated with the postnatal indices of child health inputs and child health outcomes. On the other hand, if weight-for-height of the mother and father (uncorrelated with father's own schooling or household assets) were to effect their child's health status symmetrically, a genetic effect analagous to the effect found by Coate (1980) might be induced.[15] Complete omission of the proxies for the genetic effect would lead to an autocorrelation of errors across children within a family, because the proxied genetic component is common to all the children in the same family.[16] The estimator would still be unbiased, but inefficient, and the estimates of error variances would be biased downward, so that estimated confidence intervals on slope coefficients would be understated.

The effect of the mother's height on the child's height and weight-for-height are estimated separately for boys and girls. While theory does not prescribe the sign of the effect of genetic endowment on the demand for children or child health, symmetric effects are expected if mother's height represents only genetically transmitted effects. Asymmetry for boys and girls may be evidence of discriminatory intra-family allocation of food and other health inputs.

Just as proxies were constructed for genetic endowment, fertility and child health, proxies must be constructed for the wages and prices of purchased goods that determine the household demand for fertility and child health. A vector of family agricultural inputs--land, livestock and farm buildings, is included as a proxy for the child's wage that may be positively associated with the productivity of child labor at the margin. There is some evidence that the productivity of girls in the villages is associated with land while productivity of boys is related to holdings of livestock.[17,18] Whether or not the more productive child enjoys better health depends in part on whether or not the perceived returns to his health, in the form of enhanced lifetime earnings as well as the parent's enjoyment of healthy children, exceeds the opportunity cost of the household's investment.

Simons and Bernstein (1981) found holdings of large animals to be a significant correlate with male neonatal mortality in a sample of children from villages in Uttar Pradesh state, in Northern India. It is possible that value of livestock holdings may capture an analogous effect in the child health regressions, rather than the enhanced productivity of the child.

Parent's wages must be imputed because some of the parents do not participate in the hired labor market. If the observed market wage rate did not represent the marginal productivity of the representative adult, then the imputed wages would contain a selectivity bias. To correct for this bias, the participation of the parents is first conditioned on a vector of exogenous household productive assets--land and farm buildings,[19] as well as own schooling, spouse's schooling, age and age squared, and community level variables that characterize labor demand--ratio of irrigated land to total cropped area, distance from a large town, and whether or not cotton is grown in the village. The bias arising from self-selection into the hired wage labor market was simply corrected by including the inverse of the predicted probability of participation from the hired labor participation regressions in the wage regressions (Tables A3 through A6 in the appendix).[20]

The selectivity correction term is significant and negative only in the mother's slack season wage regression, indicating that an increase in the probability of participation in the slack season is associated with higher wages. As landholdings decrease, for example, the mother's market participation increases, and her imputed wage increases. On the other hand, mother's peak season wages may be imputed without bias using the regression coefficients from the wage regression (1 in Table A5 in the appendix), where her wages are primarily explained by her own schooling and the irrigated land ratio. Similarly, the selectivity correction term was insignificant in both the father's peak and slack period wage regressions(1 and 2 in Table A4 in the appendix).

While the theoretical predictions about the effects of parent's wages are ambiguous, a negative sign on the coefficients would imply that the price effects offset the positive income effects of an increase in either parent's wages. Higher female wages, for example, are associated with higher participation, and may be associated with lower indices of child health because the child therefore receives less attention from his mother that is not offset by other child care inputs.21

Parent's schooling represents the parent's efficiency in the production of child health that may not be captured by wages. A positive sign would support the hypothesis that the nonmarket productivity of education exceeded the market productivity effects. Levels of attained schooling, rather than years, is used because levels contain information about school achievement.22 Only one level is identified, namely whether or not the parent had received any formal schooling, because there is little variation across the parents. 86 percent of the mothers and 30 percent of the fathers are illiterate.

4.Empirical Findings

Least squares methods were used to estimate the fertility, mortality, and child height and child weight-for-height equations (Tables 4 and 5).23 The discussion focuses on the variables with the most obvious economic content, namely, parent's schooling, wages, and assets, and, as well, the variables that control for biological effects, especially, mother's health status.24

Figures 1 and 2 illustrate the growth patterns predicted by the series of regressions, where the solid line traces out the regression of child height and weight-for-height on the only biological variables, sex and age (up to the

Table 4
LEAST SQUARES ESTIMATES: DETERMINANTS OF FERTILITY, CHILD MORTALITY, AND CHILD HEALTH IN RURAL INDIA
(t-statistics in parentheses)

	(1) Fertility	(2) Mortality	(3) Child Height	(4) Child Weight/Height
Intercept	-11.53 (-9.40)	-.975 (-.87)	156.09 (.98)	.579 (1.19)
Mother's Schooling	.799 (.77)	-.196 (-2.07)	1.821 (.74)	.0175 (2.32)
Father's Schooling	-.424 (-.87)	-.079 (-1.79)	-2.110 (-1.72)	-.00160 (-.043)
Mother's Wage [1]	-1.035 (-.39)	.337 (1.40)	3.261 (.51)	-.0107 (-.55)
Father's Wage [1]	2.443 (1.92)	.296 (2.56)	-3.228 (-1.21)	-.0151 (-1.86)
Landholder	.166 (.31)	.031 (.63)	1.194 (.88)	-.0021 (-.51)
Land	1.171 (.70)	-.00651 (-.42)	-2.223 (-.62)	.000396 (.36)
Livestock	.748 (-.51)	-.000493 (-.003)	6.961 (2.47)	.00565 (.65)
Buildings	.0425 (.06)	-.0312 (-.44)	-2.75 (-1.87)	-.00588 (-1.31)
Mother's Height [2]	-2.816 (-.43)	-.843 (-1.41)	41.923 (2.72)	-.0170 (.36)
Mother's Weight/Height	2.35 1.06)	.067 (.33)	-3.571 (-.67)	1.043 (2.68)
Mother's Age	.394 (1.76)	.0166 (.81)	.	.
Mother's Age Squared	-.0023 (-.93)	-.00011 (-.88)	.	.
R^2	.2946	.1937	.8463	.1620
Degrees Freedom	112	112	272	272
SSE	554.96	4.58	17394.4	.792

[1] The wages are imputed wages. See the text, part 3.

[2] Mother's height is the standardized (by the Indian standard), as is mother's weight -for-height.

Table 5

Household Statistics

Variable Definition	Mean	St. Dev.
Endogenous		
Children ever born to the woman	6.32	2.55
Number of children that died	2.09	1.82
Proportion of children dead to children ever born	.29	.21
Father's peak season wage (imputed) rps	3.22	.26
Mother's peak season wage (imputed) in rps	1.80	.14
Father's slack season wage (imputed)	.57	.41
Mother's slack season wage (imputed)	1.72	.25
Weight of child (in kilograms)	27.0	10.7
Height of child (in centimeter)	131.0	19.4
Weight for height of child in kg/cm	.20	.05
Exogenous		
Age of mother (years)	41.1	8.2
Schooling of mother (Dummy)	.13	.30
Schooling of father (Dummy)	.29	.46
Height of mother (in cms)	148.6	5.2
Weight for height of mother (in kg/cm)	.27	.03
Height of father (in cms)	160.5	7.9
Weight for height of father (kg/cm)	.30	.04
Land owned by the household	15.57	21.71
Livestock (1000 rupees)	1.72	2.34
Craft and farm tools (1000 rupees)	3.38	4.68
Price of schooling (proportion of mother's childbearing years during which there was a village school)	.97	.12
Price of healthcare — distance to the nearest marketing town (kilometers)	13.5	10.7
Proportion of village land that is irrigated (ILR)	11.0	9.6
Average rainfall in taluka (cms)		
NUMBER OF HOUSEHOLDS	145	

GROWTH PROFILES
HEIGHT

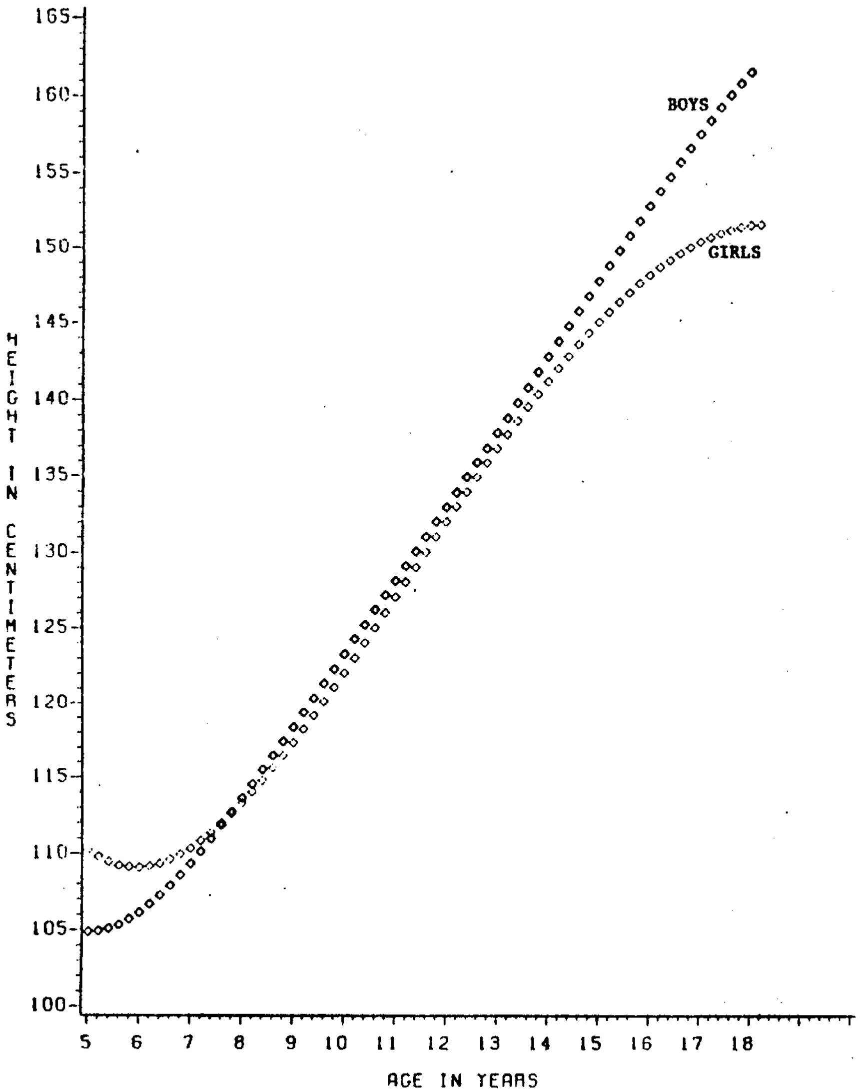

FIGURE 1

GROWTH PROFILES
WEIGHT-FOR-HEIGHT

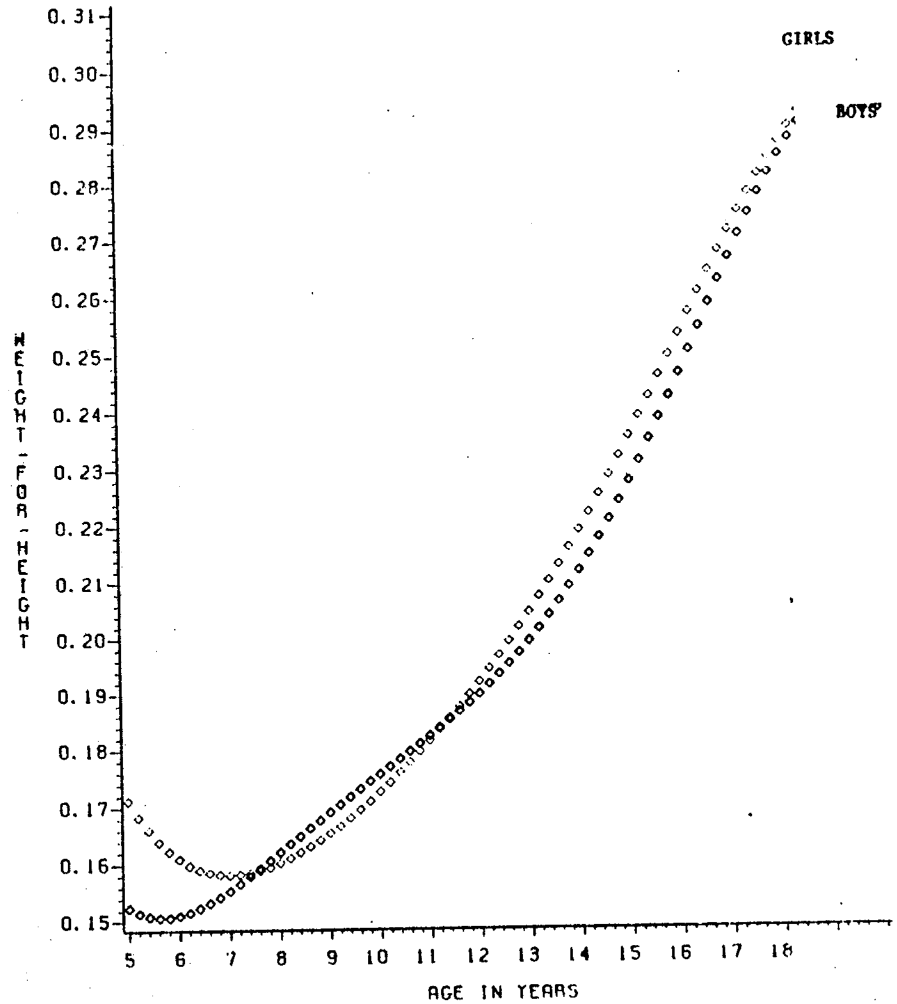

FIGURE 2

third polynomial term for height, and the fifth for weight-for-height).25 The dotted line corresponds to the regression including mother's height and weight-for-height, and the dashed line including wages and prices as well as age. The profiles show that height of boys and girls diverges around age 10. Girls have attained full height by age 16, while boys are growing up to the age of 19. These profiles mirror observed growth patterns of Indian girls and boys reported by Eveleth and Tanner (1980).

The symmetry of the effects of mother's height on the height and weight-for-height of her children, as well as symmetry of the effects across boys and girls (summarized in Table 6 in elasticity form to facilitate comparison across equations and across children by sex) gives credence to the hypothesis that mother's height represents genetic health endowment. F-tests at the .05 level of confidence confirmed the symmetry: the data do not support the hypothesis that the effects differ between boys and girls.

By the same logic, the asymmetry of the effects of weight-for-height of the mother and father on the weight-for-height of their children, with mother's weight-for-height being significant, and father's, insignificant. This pattern suggests that mother's weight-for-height the socioeconomic environment, rather than genetically transmitted predisposition to a certain weight-for-height. Thus, if mother's weight-for-height reflects intra-household food distribution practices, one may conclude that nutritional discrimination between children by sex does not occur in these households. 26,27

Furthermore, the effects of mother's weight-for-height was associated with higher fertility, albeit marginally significant. There are increased demands on the fat and protein stored in the mother's body during pregnancy , and the mother with the greater weight-for-height (nutritional or health status) may be better able to meet these demands, and bring her pregnancy to fruition(Lechtig, et. al. (1979)).[28]

In general, there are gains to be realized from considering the system of household demands, including the possibility of clarifying the nature of the proxies because the effects should be consistent across equations. For example, a negative impact of livestock in the mortality and child health regressions would confirm that livestock transmit tetanus or other diseases (Simons and Bernstein (1980)). On the other hand, a negative effect on child mortality, and a positive effect on child health might point to a selection problem: only those children most resistent to infection and disease, because of superior genetic or nutritional inputs (Martorell (1982)), survive. The only story supported by the ICRISAT data is that the household with healthy children tends to have cattle, and fewer farm buildings. The effects of the dissaggregated asset categories are important only in the child height regressions, but of different signs.

The significant correlation of father's (imputed) wage and fertility is in line with, for example, Khan and Sirageldin (1980) empirical analysis of fertility in a sample of rural Pakistani villages. Father's personal characteristics, rather than those of the mother, determine fertility.

The effect of father's (imputed) wage is consistent across the child weight-for-height and mortality equations: the higher his wage, the lesser child weight-for-height, and the higher child mortality. The connection between (lower) child weight-for-height and (higher) mortality is intuitive, but the connection between (higher) wage and (lower) weight-for-height, less so. Consideration of the effect of father's schooling on child height and mortality may illuminate the answer. Father's wage may represent the more transitory component of household income, and father's schooling, the permanent component. Child survival may be normal with respect to permanent income, and child height appears to be inferior, because the greater number of survivors, the greater the chance of chronic (over the long run) regime of mild malnutrition. In the short run, the higher the father's wage, the more food given to sustain him, and the less food left for children. Deprivation, even for a short time, may significantly lower on the marginal child's survivability.

As mother's schooling reflects her efficiency in the production of healthy children, perhaps through a better understanding of how to use medical facilities, then her schooling would be negatively associated with the household price of health. The positive effect of mother's schooling on child weight-for-height, and the negative effect on child mortality,[29] could then be interpreted as an own-price effect. Another story suggests that the mother may be in a better position to bargain for nutritional and health inputs for her children.[30]

All in all, the results confirm the conventional wisdom of the health literature that child height represents cumulative health, and child weight-for-height, transitory health, and thus the use of the two indicators in any evaluation of child health complement each other. This conclusion follows from the result that child height is determined by father's schooling and household assets, as well as mother's height, that is, in some sense, by the longer-term inherited income and health position of the household. Child weight-for-height is determined by mother's schooling, wage and weight-for-height, as well as father's wage, but not by mother's height. The significant determinants of child mortality parallel those of child weight-for-height, but in the final analysis, the data are not strong enough or the sample is too small to draw many firm conclusions about child health, and fewer about the determinants of fertility and child mortality.

5. Summary

Joint estimation of the determinants of fertility and child health illustrates the useful application of the theoretical framework (Part 2), as well as its limitations. Even when genetic health endowment and earnings of children affect the structure of household commodity shadow prices, the only unambiguous prediction of the model is the positive effect of unearned income, which follows from the standard assumption that these household commodities are normal goods. Although statistical and conceptual problems arise from the proxy measures of the wages and prices that theoretically condition the demand for fertility and child health (Part 3), certain net effects can be empirically signed (Part 4).

Although the results confirm that anthropometric indicators may enhance our understanding of investments in children, it is important to replicate these results with other data from other countries, precisely because the signs of the cross price effects cannot be derived from the theory, and can only be inferred from empirical analysis. While certain patterns in the results are consistent and intuitive, the data apparently cannot support estimation of the proposed system of household demands, so recourse to other and probably larger samples from low income communities is necessary.

Footnotes

[1] When schooling is included in this system of child quality and quantity, another production constraint, namely the household production function for schooling, becomes important. It may be interesting to incorporate the complementary effect of health on schooling attainment, in the popularly recognized sense that the malnourished morbid child is the listless student. This is distinct from imposing complementarity of schooling and health in the household's utility function.

[2]Generally, shadow prices, as distinct from market prices, include the value of the time of the individual used in consuming or producing the commodity, as well as the cost of the market goods (inputs into this consumption or production process). For example, the shadow price of child health may be comprised of the cost of food and medical care, and the cost of the time the mother spends in feeding the child, or in taking the child to the healthcare center as well.

[3]Several restrictions have already been imposed, namely, the complementarity of the genetic health endowment, e, and the health inputs.

[4]The landless laborer households are distinguished as those working regularly for a daily wage, or that farm less than .2 hectares of land. Landholding households farm at least .2 hectares of their own or rented land, and others than the household head may be salaried or otherwise regularly employed workers.

[5]Secondary selection criteria are summarized in Tables A1 and A2. Basically, observtions were lost because of the lack of correspondence between the fertility and health surveys. Fertility data was not collected for every

household surveyed by the health workers.

[6]Cumulative and last birth experience of the total sample of 401 women surveyed in the fertility survey are described in Tables 1 and 2. Noteworthy are the completed family size, constructed as the family size of women over 45 who are presumed to be beyond childbearing age,the high proportion of children dead to children ever born, around 2 to 6, and the similarity of mortality rates across boys and girls. Because the data is recall data, it is important to consider the reasonableness of the statistics.

[7] Sex and household asset interaction variables and sex and parent's health interaction variables were included in the regression, at different unreported stages, to separate the biological from the economic content of sex. [8]There is evidence that the body may be able to catch up when the nutritional environment is improved, provided that the child has not been severely deprived in the critical periods of growth. Because the periods of growth—infancy and adolescent post-pubescence, show child specificity, it is not possible to predict, say, that nutritional supplementation to children over twelve would be effective. Kwashiokor and maramus are forms of wasting, the former due to acute protein deficiency, the latter to acute protein and calorie deficiency. The terms "stunting" and "wasting" were used by Waterlow (1972).

[9]There is a literature on the sensitivity of the various anthropometric measures, where the metric is the underlying true health or nutritional status of the target population of health and nutritional improvement programs. Martorell (1982) for example found that weight for height and weight were not sensitive indicators of the improvement in the nutritional status of children in rural Guatemalan villages, as the children were only suffering from mild to moderate malnutrition, and were able to maintain normal weight-for-height over the range of mild malnutrition to improved nutrition. Habicht (1980) found

that weight for height might also deteriorate when the nutrition was improved as children "caught up" to a more normal height. Martorell found tht height was the most sensitive indicator of the improvement in the nutritional status of Guatemalan pre-schoolers.

[10]The calculation that 1 standard deviation is approximately 10% of the median is well known. Waterlow (1977) constructed an alternative measure to the percentile or percentage of median measure of wasting and stunting based on the exact calculation of standard deviation. Of course, construction of the critical region of the standard normal distribution is textbook knowlege.

[11]Weight-for-height has the advantage of being age independent, except as puberty is approached or at extreme values (Waterlow (1976)). But use of weight-for-height, of course, masks the occurrance of stunting suffered by the child, which is common in all six villages. Futhermore, to anticipate the empirical findings, weight-for-height was associated with age.

[12]Habicht and Butz (1979) note that the smaller the child in a population, the greater the probability that the child's growth was stunted for genetic reasons.

[13]Chernichovsky and Kielmann (1979) also used height of the mother as a proxy for genetic endowment in their analysis of diet, health and growth of pre-schoolers in 14 villages in rural Punjab.

[14]Multiple observations on the anthropometric measures, i.e. measuring height and weight at, say, monthly intervals, would enable one to control for child specific influences using either variance components or first differencing estimation methods.

[15]Coate (1980) found that tricep skinfold of mother and father were positively associated with the skinfold of their children, indicating that the tendancy to develop adipose (fat) tissue was transmitted from parents to children

(Coate controlled for the effects of similar environment, that is, diet). Accordingly, if weight-for-height of the mother and father were to effect the weight-for-height of their children symmetrically, one might conclude tht weight-for-height was a component of the genetic health endowment.

[16]There still remains the possibility of missing mother or family specific variables. Variance components methods were used to control for the possibly missing variables, but it was found that the family specific component of the residual was insignificant (footnote 24 elaborates on this point).

[17]Participation of the children in the hired labor market was first conditioned on a vector of presumable exogenous productive farm assets, namely, land, livestock and buildings. Regression results showed that the size and direction of the participation effects vary across boys and girls. For example, the elasticity of girl's participation (in slack season) with respect to the value of land owned is almost twice that of boy's participation. The elasticity of the boy's participation with respect to livestock is negative, and that of the girl's is insignificantly positive, while the elasticity of boy's participation with respect to buildings is positive and that of girl's, insignificant. These results are reasonable for a market where certain tasks appear to be systematically assigned to girls, and others, to boys. When the linear selection term (one minus the predicted probability of participation) (see text) was included in a wage slack season wage regression for boys, it was significant and positive, indicating that higher participation is associated with lower wages. These imputed wages were tried in the child health and fertility regressions, but were insignificant, perhaps because of the lack of information in the data—only 56 of 323 children worked for a wage.
Another proxy for the child wage would be the village level (average) wage paid to children, but it was not used in light of the fact that the participation

rate (days worked per year) was small for even the fifty-six children who participated in the mrket in 1976 and 1977.

[18]It is, of course, possible that family assets respond to the number, age and sex of surviving offspring. The true model would include portfolio choice as an endogenous variable.

[19]Rosenzweig (1980) postulated that farm-earnings assets would be negatively associated with off-farm work of family members in landholding households. Own-farm production is explicitly modelled in his paper.

[20]The selectivity bias framework is Olsen's (1980). The main drawback is that the selectivity correction term is a linear combination of some of the regressors in the wage regression, and the participation regression was estimated using OLS methods, which are less appropriate for qualitative dependent variable regressions than logit or probit, but Olsen maintains that the bias is in the direction of accepting the hypothesis of selectivity bias. Using Olsen's method, the hypothesis of selectivity bias was rejected, and the imputed wages of the parents are estimted from conventional wage regressions.

[21]DaVanzo and Butz (1978) suggest that it is the quality and not the quantity of time of the mother that is represented by her schooling and wage.

[22]There is a potential errors-in-variables problem arising from the incidence of migration of women with young children in these villages. There is no information on the duration of household residence in the village. If the incidence of migration over the five years of the survey is any indication of the likelihood of migration, then we might assert tht the problem is small. Only ten of the original 240 households surveyed migrated from another village. Dhar (1980) found thta 29.7 percent of the adult female population in India in 1971 were intra-district migrants, i.e. their last place of residence differed from their current, however, most of this migration occurred upon marriage.

23 Fuller and Battese (1973) show that when the error is comprised of a random components and a component that is the same for all children, say, within a family group, then a transformation of the regression equation,

$$Y_{ij} = \beta x_{ij} + f_j + c_{ij} \quad ,$$

gives us the estimating equation,

$$Y_{ij} - \alpha \bar{Y}_i = (1 - \alpha)\bar{\beta}_1 + \sum_{K=2} \beta_K (x_{ki} - \bar{x}_{ki.}) + c_{ij},$$

where $\alpha = 1 - \sigma_c / \sigma_1$ and the v_{ij} are homoskedastic and uncorrelated and $Y_i = \sum_{j=1}^{J} y_{ij}$. OLS methods yields consistent estimators, when σ_c^2, σ_f^2, and σ_1^2 are known, where σ_c^2 is the random component of variance, σ_f^2 is the group-specific component of variance, and $\sigma_1^2 = \sigma_c^2 + \sigma_f^2 N$.

When σ_c^2 and σ_f^2 are unknown, unbiased estimates can be obtained (Swamy and Aurora (1972)).

When the transformtion was applied to the information matrix for child height and weight-for-height, σ_f^2 was negative, and so α was set to 0, as prescribed by Maddala (cited in Judge, et.al (1982)).

In this case, OLS and the GLS estimators were equivalent because the family-specific component of variance was insignificant, indicating that household background variables were not omitted from the specification.

24 Unfortunately, information on birthweight, interpregnancy intervals, and breastfeeding practices—the most important correlates in the Butz, DaVanzo and Habicht (1982) study of infant mortality, were either not available in the ICRISAT data, or were available but unreliable (the breastfeeding data).

25 Age, age squared, etc. to the fifth polynomial were found to be jointly significant in the empirical specification of child weight-for-height, while only age, age squared and age cubed were jointly significant in child height.

[26] Lectig, et.al. (1979) suggest that the nutritional status of the mother during pregnancy, as measured by her weight gain, is associated with the birthweight of her infant. Beaton and Benzoa found that mothers who were stunted (in height) due to infant or adolescent malnutrition, had low birthweight babies, and they further found that low birthweight infants suffered a higher risk of protein energy malnutrition. It is not clear that Beaton and Benzoa controlled for mother's nutriture during her pregnancy, nor do they attribute the cause of the higher observed risk of PEM on the part of the child to either environment or genetic inheritance. What is relevant to the paper is that both Lectig and Beaton and Balzoa suggests that birthweight may mediate mother's nutritional status and the subsequent status of her children.

[27]Ryan, et. al. (1981) observes that men in these villages apparently receive a premium in food, but that men perform the most physically demanding tasks—ploughing with bullocks, for example, so that they burn up extra calories. This may account for the insignifant correlation between father's and child's weight-for-height.

It would be interesting to analyze the nutritional survey, wherein the food intake of the household members on four days over a year is described in terms of the nutrient components of the food, in order to confirm the result.

[29] The effect may be analogous to the biological effect hypothesized by Frisch (1974), namely, the fat to body weight ratio affect ovulation and fecundity. Lechtig, et. al (1976) also maintain that the nutritional status of the mother affects birthweight.

[30]The association between mother's schooling and postneonatal infant mortality is exemplified in Simmons and Bernstein (1981) and Butz, DaVanzo and Habicht (1982). Simmons and Bernstein found that although parental schooling

insignificantly affects neonatal (up to 1 month) tetanus and non-tetanus mortality in a sample drawn from 120 villages in northern India, mother's schooling significantly decreased the risk of post-neonatal (1 to 12 months in age) mortality of girl children. Using data from the Malaysian Life Survey, Butz, et.al., found that mother's schooling significantly reduces the risk of death (both attributable and relative) in post-neonatal infants, and went on to conclude that the effect of mother's schooling was mediated through mother's age and birthweight (the proximate biological influences). Further, DaVanzo and Lee (1978) confirm conventional wisdom, and conclude that mother's schooling is related to the quality not the quantity of the time that the mother spends caring for her children.

[31] Price of health care was proxied by both the distance to the nearest marketing town in preliminary regressions. The presence of a primary health care clinic in the village was found to be significantly correlated with the distance to the nearest town--there is a clinic in the village that is located farthest from a town. Moreover, the private medical practitioners located themselves in the two villages nearest marketing towns. Hence, only distance to the nearest town was included in the regressions. The interpretation on the proxy is that the further the parent and child must travel to obtain healthcare, the higher the price. In the household production model, the price of health is proportional to the price of the time spent in production. Alternatively, the average level of rainfall or the proportion of irrigated land in the village may proxy for the price of healthcare, because the variables may capture increased exposure of the children to waterborn diseases. In the preliminary results, the proxies were insignificant.

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Appendix

Table A1
Sample Selection Criteria for the Analysis of Fertility

Reason for Inclusion (or Exclusion)	Number of Families	Number of Households
Families in 233 households in 6 villages	400	233
Mother was dead	-8	229
Families with observations on the mothers (some households consisted of several women, and widowers together)		
Father was dead	-69	
Father and mother were separated	-8	
Father and mother were divorced	-2	
Families with mother and father	312	207
Subsidiary families	-133	
Primary families	179	179
Childless primary families	-7	
Children between 6 and 19 years of age were not observed	-22	
Parents and children between 6 and 19 observed in the household	144	
Missing observation on assets	-1	
Missing observation on parent's health	-13	
WORKING SAMPLE	130	130

Table A2
Sample Selection Criteria for the Analysis of Child Health

Reason for Inclusion (or Exclusion)	Number of Children	Number of Households
Children from primary families, i.e. father is the head of house (1)	764	
Missing observations on Child Health (2)	-209	
Children under 6 or over 20 years old	-179	
Children between 6 and 19	376	167

Children from families excluded from the Fertility Survey (3)	-55	-26
Missing observation on Assets	-1	-1
Missing observations on Parent's Health	-37	-10
WORKING SAMPLE	283	130

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(1) These children are distinguished form children whose fathers are say sons or brothers of the head of house, herein referred to as children from primary, distinct from subsidiary families in the household.

(2) The sample households were surveyed in 1977-1978 by a team of medical persons. Apparently, not every member in the household that had been enumerated in the ongoing census was

(3) The fertility data was collected more than one year after the health and nutrition data. Again, not every member nor every household surveyed by the original census takers, nor the health investigators, was surveyed by the fertility investigators. The household may have left the village, permanently or just temporarily.

TABLE A3
FATHER'S PARTICIPATION REGRESSIONS
PEAK AND SLACK SEASONS, 1976-1977

	Peak Season	Slack Season
Constant	.083 (.11)	.51 (.64)
Age	.015 (.45)	-.0086 (-.26)
Age squared	-.00024 (-.71)	-.00038 (-.11)
Schooling of Wife	-.17 (-1.31)	-.26 (-1.90)
Own Schooling	-.045 (.51)	.056 (.63)
Land	-.047 (-2.10)	-.044 (-1.95)
Buildings	-.085 (-.76)	-.054 (-.48)
Caste Rank	.0095 (.49)	.035 (1.76)
Irrigated Land Ratio	.0032 (.57)	-.077 (-.0014)
Cotton Growing	.26 (2.30)	.13 (1.13)
Distance to Town	.0027 (.68)	.0018 (.45)
R-squared	.2137	.1899
Number of Obs.	144	144
Predicted Value Actual Value	.28	.28

Dependent variable is a dummy, 1 if father participated, 0 otherwise.
t-statistics in parentheses.

TABLE A4
FATHER'S WAGE REGRESSIONS
PEAK AND SLACK SEASON DAILY WAGES 1976-1977

	Daily Peak Season Wage		Daily Slack Season Wage	
Constant	1.72 (1.48)	1.83 (1.72)	.81 (.85)	.60 (.63)
Age	-.019 (-.39)	-.020(-.42)	.020(.50)	.010(.25)
Age Squared	.0006(.28)	.00017(.32)	-.0002(-.5)	-.0001(-.3)
Own School	-.016(-.15)	-.0037(-.4)	.075(.60)	.12(.96)
Irrigated LandRatio	-.0037(-.6)	-.0045(-.7)	-.0075(-.9)	-.0085(-1.0)
Cotton Grower	.053(.26)	.0098(.1)	.031(.20)	.07(.45)
Selectivity Correction	.15(.26)	---	----	85(1.19
R squared	.1318	.1300	.1103	.1468
No. obs.	41	41	41	41
(1-p)	.56	--	--	.58

Dependent variable is the natural logarithm of the daily wage in peak and slack season.

TABLE A5
MOTHER'S PARTICIPATION REGRESSIONS
PEAK AND SLACK SEASON, 1976-1977

	Peak Season	Slack Season
Constant	.90 (1.55)	.40 (.70)
Age	-.03 (-1.23)	-.008 (-.32)
Age Squared	.00032(.99)	.00006(.18)
Own Schooling	-.19 (-1.53)	-.30 (-2.37)
Schooling of Husband	.012 (.14)	-.030 (-.35)
Land	-.086 (-4.04)	-.080 (-3.75)
Buildings	-.043 (-.40)	-.032 (-.30)
Caste Rank	.055 (2.96)	.049 (2.65)
Irrigated Land Ratio	.017 (3.21)	.018 (3.35)
Cotton Grower	.38 (3.66)	.34 (3.30)
Distance to Town	.0085 (2.30)	.0091 (2.45)
R-squared	.4056	.4079
No. Obs.	144	144
Actual Value of Dependent Var.	.57	.58
Predicted Value		

Estimated by OLS methods.
t-statistics in parentheses.
Dependent variable is the same as in footnote to Table A3.

TABLE A6
MOTHER'S WAGE REGRESSIONS
PEAK AND SLACK SEASON DAILY WAGES 1976-1977

	DAILY WAGE Peak Season (1)		Slack Season (2)	
Constant	.43(1.1)	.47(1.3)	.59(3.0)	.56(2.7)
Age	.001(.1)	-.003(-.1)	.02(1.8)	.02(1.6)
Age squared	.00002(.1)	.00006(.2)	-.0002(-1.8)	-.0002(-1.6)
Own Schooling	.2(1.7)	.18(1.6)	.03(.2)	-.05(-.4)
Irrigated Land Ratio	.006(1.7)	.007(2.4)	-.008(-4.0)	-.006(-3.3)
Cotton Grower	.007(.1)	.03(.4)	-.2(-5.3)	-.2(-4.8)
Distance to Town	.002(.6)	.002(.7)	-.02(-12.2)	-.2(-11.7)
Selectivity Correction	-.10(-.6)	---	-.2(-2.1)	---
R-squared	.1448	.1410	.7034	.6862
No.Obs.	82	82	83	83
Mean Value of Dependent Var.	.26	.26	.25	.25

Dependent variable is the same as defined in footnote to Table A4.