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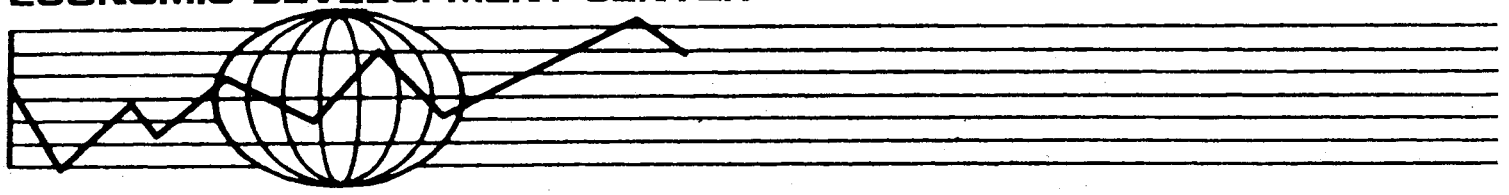

HETEROGENEITY, INTRAFAMILY DISTRIBUTION AND CHILD HEALTH

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A dynamic model of intrahousehold allocative behavior incorporating variations in and uncertainty about individual child characteristics is formulated to ascertain how both the timing of childbearing and child-specific allocations vary with exogenously-determined household and child-specific health characteristics and to assess the implications of such dynamic behavior for the estimation of the behavioral determinants of child health. Longitudinal data on children and households from a village in Colombia are used to compare the sensitivity of estimates to assumptions about heterogeneity with respect to birth spacing and timing, per-capita food consumption, innoculations, and the incidence of breastfeeding on the age-standardized weight of children at two life-cycle points and to estimate intra-family and inter-family resource allocation and fertility responses to inherent health variations.

The estimation of the effects of household resources on the survival, health and well-being of children has been a central concern in the demographic, economic and medical literatures, (e.g. Heller and Drake, 1979; Olsen and Wolpin, 1983; DaVanzo, Butz and Habricht, 1983). One of the potential problems in obtaining estimates of the effects of such behavioral inputs as maternal age of childbearing, breastfeeding, and use of medical services on measures of child health is the existence of health-related factors known to or affecting parental decision makers but unobserved by the researcher. Variations in such unobserved factors (heterogeneity) in the sample population may provide misleading estimates of the causal relationships among parental choices and observed health outcomes. Yet few studies have been attentive to this problem.

There are two distinct sources of heterogeneity, with different implications for statistical treatment. First, there may be across-household variation in the health environment in which allocative decisions are made -- mosquito infestation, sanitary conditions -- or in the inherent healthiness of parents, some of which is transmitted genetically to offspring. If parents take into consideration these household factors in their allocative decisions; for example, if households in healthier environments choose to have fewer children or to space them more widely, then the observed association between variations in such variables and measures of child health will overstate their consequences for child health. Use of information on siblings and a household fixed effect procedure circumvents this problem, given the invariance of these household health unobservables. However, only one study of the behavioral determinants of

child health has used this procedure (Olsen and Wolpin, 1983), where the importance of this type of heterogeneity is demonstrated.

A second source of heterogeneity arises from variations in the inherent qualities of children born within a family. Differences among children in healthiness or skills may affect how parents allocate resources across their offspring as well as parental fertility decisions. For example, it is well-known that an infant's intake of breastmilk depends on its ability to suckle; immature or ill infants may thus be breastfed less or not at all, leading to an upward bias in the estimation of the effects of breast-feeding on infant survival or nutritional status. The death of an infant may lead to a more closely-spaced subsequent child (the so-called replacement effect), with deleterious consequences for that child's health.

No studies of child health have attempted to deal with both intra and inter household heterogeneity. Rosenzweig and Schultz (1983) use an instrumental procedure to obtain estimates of the behavioral determinants of birthweight; however, their study assumes that parental schooling levels and husband's income are orthogonal to the unobserved factors associated with child health. If more educated and wealthier parents are also healthier and thus have inherently healthier children, however, their estimates will be inconsistent. The Olsen and Wolpin study ignores any responsiveness of parental allocations to variations in the healthiness of individual children.

Little empirical evidence exists on how resources are allocated across family members as a function of their "endowments," (Rosenzweig and Schultz, 1982). The existing theoretical literature on intrahousehold allocations (Becker and Tomes, 1976; Behrman et al., 1982; Sheshinski and Weiss, 1982) is deficient in providing insights into how parents respond to exogenous

variations in the inherent qualities of children, and thus on the direction of bias, if any, in studies ignoring such behavior and/or heterogeneity, chiefly because they assume that the qualities of all children are known by parents in advance, prior to their birth. However, early and important decisions about resource allocations to children cannot be fully informed about the characteristics of children yet unborn; such decisions are inherently dynamic and sequential (Wolpin, 1984).

In this paper, we formulate an illustrative dynamic model of intra-household allocative behavior incorporating variations in and uncertainty about individual child characteristics. The model is used to show how both the timing of childbearing and child-specific allocations vary with both household and child-specific health endowments. In part 2, we discuss the implications of the model for estimation of the behavioral determinants of child health and we use the information restrictions in the model associated with the sequencing of births to develop an estimation procedure which takes into account both intra and inter household heterogeneity. In part 3, longitudinal data on children and households from a village in Colombia are described and used to compare estimates of the effects of birth order, birth spacing and timing, per-capita food consumption, innoculations, and the incidence of breastfeeding on the age-standardized weight of children at two life-cycle points, at birth and within six months after birth. The estimates, obtained using ordinary least squares, a family fixed procedure, and the new procedure suggest the sensitivity of estimates to assumptions about heterogeneity and parental behavior. In particular, those procedures which ignore heterogeneity understate importantly the effects of birth order and birth spacing but overstate

the effects of breastfeeding. The consistent estimates obtained in part 3 are used in part 4 to compute estimates of the health endowments of individual children and of households and to estimate the effects of variations in such endowments on the behavioral variables. These estimates indicate that healthier households, for given income, have more children and more closely-spaced children but consume no more food per-capita than do less well-endowed households. These results imply that households tend to reduce interfamily inequalities in child health. However, while the estimates suggest that parents are more likely to have a subsequent child quickly the more healthy is the prior (surviving) child, they are more likely to breastfeed an inherently healthier child.

1. The Model

a. Heterogeneity, the Health Technology and Information Restrictions

Assume that the health at birth h^0 of a child born to a particular family depends on its birth order, the timing of preceding births, the age of the mother at its birth, and prenatal child-specific resources. For child of order i, the (log) of health at birth is assumed to be given by

$$(1)\quad \log h^0_{i,t_i} = \gamma^0_1 t_i + \sum_{k=1}^{i-1} \gamma^0_{2,k} n_{i-k} \cdot (t_i - t_{i-k}) + \gamma^0_3 i + Z^P_i \gamma^0_4 + \mu + \varepsilon_i + v^0_i$$

where h^0_{i,t_i} is the health at birth of the child or order i born to a mother at age t_i, n_{i-k} is equal to one if a child of order i-k is born at mother's age t_{i-k}, and Z^P_i are a vector of prenatal inputs to child of order i. The randomness in observed initial health is due to a family health endowment common to all children within a family (μ), a child-specific health endowment common to all ages of a particular child (ε_i), and a purely random

serially uncorrelated draw (V_i^0). The following assumptions are made about these error components: $E(\mu_j\mu_k) = \sigma_\mu^2$ $(j=k) = 0$ $(j \neq k)$; $E(\varepsilon_{ij}\varepsilon_{k\ell}) = \sigma_\varepsilon^2$ $(i=k,\ j=\ell) = 0$ $(i\neq k,\ j\neq\ell)$; $E(V_{ij}^0\ V_{k\ell}^0) = \sigma_v^2$ $(i=k, j=\ell) = 0$ $(i\neq k, j\neq\ell)$; $E(\mu_j\varepsilon_{k\ell}) = E(\mu_j V_{k\ell}^0) = E(\varepsilon_{j\ell}V_{km}^0) = 0$ $\forall_{j,k,\ell,m}$.

The health of child i at any age, a, after its birth may depend on the timing of the births of subsequent children (if any) and on post-natal resources Z_i^a allocated to it; thus, for child i

$$(2)\quad \log h_{i,t_i}^a = \gamma_1^a t_i + \sum_{k=1}^{i-1} \gamma_{2,k}^a n_{i-k}\cdot (t_i - t_{i-k}) + \sum_{k=1}^{\tau} \theta_{2,k}^a n_{t_{i+k}}\cdot(t_{i+k} - t_i) + \gamma_3^a i + Z_i^P \gamma_4^a + \sum_{k=1}^{a} Z^k \gamma_{5,k}^a + \mu + \varepsilon_i + V_i^a$$

Notice that prior inputs are assumed to potentially affect the stock of health at any age and that such inputs may not have uniform effects at all ages. Note also that, due to the logarithmic specification, the effects of all inputs on the level of a child's health depend on the magnitude of the child's health endowment, composed of the elements μ, ε_i and V_i.

Equations (1) and (2) describe the production technology relating the timing of births in the household and child-specific resources to a child's health at its birth and later in its life. Use of least squares or other single equation procedures to estimate the health technology parameters in (1) and (2) will yield unbiased estimates of these parameters only if the "inputs" are uncorrelated with both the household and child-specific endowments unobserved by the econometrician. The direction of the biases will in turn depend on whether the parents, when making their decisions about each of the inputs, observe the endowments, or components of them, and how such decisions are affected by such knowledge.

The sequential ordering of births places some important restrictions on parental information. The decision concerning when to have child i cannot, for example, depend on its child-specific endowment ε_i, which only becomes known after its birth, but may be informed by the household's health environment μ and may also depend on the perceived healthiness of prior children (ε_{i-1}). However, decisions about the level of post-birth resources Z_i^a allocated to child i may depend on (and will certainly be informed by) the initial healthiness of the child as well as on the healthiness of all prior children.

b. Parental Resource Allocations to Children

Given the existence of parental perceptions about the health environment in which they reside and about the individual, inherent traits of their children, little can be said a priori about how such information affects parental resource allocations to children without specifying parental objectives and constraints. Such a behavioral theory should also incorporate the biological characteristics describing the consequences of allocative decisions, as in (1) and (2), and the information constraints associated with the sequencing of births. To obtain some insights into how differences in healthiness across households and how differences in healthiness across children within households affect household allocative decisions, and thus how single equation estimates of biological relationships involving endogenous parental decisions in the presence of heterogeneity may be biased, we formulate a simple dynamic model.

Assume that the parents in each life-cycle period maximize the expected value of an intertemporally separable utility function that has as arguments

the mean H_t of the "final" child health outcomes of children in the household, the number of children in the household and a commodity X. Final child health is achieved at some arbitrary age A of the child, i.e., it is h^A_{i,t_i} for child of order i. Parents thus care both about the health and number M_t of their children, where $M_t = M_{t-1} + n_t$. The parents' problem is described by

$$(3) \quad \max_{Z_t, n_t} E\left[\sum_{\ell=t}^{T} \delta^{\ell-1} U(H_\ell, M_\ell, X_\ell)\right]$$

subject to the

per-period income constraint, which must be satisfied in each period,

$$(4) \quad F_t = w_t Z_t + X_t + P_t n_t,$$

where F_t = income, w_t = cost of a unit of resource Z, P_t = price of having a child; and to the "final" health equation (2) at a = A. Parents thus choose whether to have a child in each period and how much Z to allocate to that child after it is born and to all other children who have not yet reached their "final" health stock based on the information set Ω they have at the beginning of the period. Thus at the onset of period t, for example, parents know the household endowment μ, all their past decisions, the health technology (2), and the individual endowments (and thus health outcomes) of all prior children; they do not know the child-specific endowment ε_i of children to be born in t or after period t.

To simplify the model, assume that the decision horizon has four periods; children can be born at the beginning of period two, three, or four and health inputs are required only for one (the first) period of the child's life. Thus in the last period (four) only the level of Z for a child to be born in period four needs to be determined if the household had decided

during period three to have a child in period four. The technology of final health production is described by equation (2), except that, for simplicity, we will ignore all prenatal inputs except those associated with the spacing of births. The information sets associated with the beginning of each successive period are thus: $\Omega_1 = \{\mu; \Gamma\}$ $\Omega_2 = \{\mu, \varepsilon_1, n_1; \Gamma\}$, $\Omega_3 = \{\mu, \varepsilon_1, \varepsilon_2, n_1, n_2, Z_1, ; \Gamma\}$, $\Omega_4 = \{\mu, \varepsilon_1, \varepsilon_2, \varepsilon_3, n_1, n_2, n_3, Z_1, Z_2; \Gamma\}$, where Γ represents the technology parameters.

To further simplify, assume that utility in each period is linear quadratic; thus in period four

$$(5) \quad U_4 = \alpha_1 H_4 - \alpha_2 (H_4)^2 + \beta_1 X_4 - \beta_2 (X_4)^2 + \delta M_4$$

Also assume for (innocuous) simplicity, that Z is a dichotomous variable, e.g., breastfeeding, taking on the value of 1 if Z is allocated to child i and the value of zero if it is not.

In such dynamic, forward-looking problems, it is not generally feasible to derive analytically the parental decisions rules for n_t and Z_t in any period (Wolpin (1984)). However, comparative statics can be performed readily for the fourth (final) period decision, when, in this case parents have full information about endowments. That is, the effects of the endowments of the children on the allocation of resources to the last child can be discerned in terms of the structural technological and preference parameters of the model.

Assume that it is optimal to have a child born in period four. Then at the beginning of the fourth period, the parents compare expected utility with Z = 1 to expected utility with Z = 0, given their information set Ω_4;

the difference in expected utilities J_4 is:

$$(6)\quad J_4 = E_4(U_4|Z = 1;\ \Omega_4) - E_4(U_4|Z = 0;\ \Omega_4).$$

Only if $J_4 > 0$ will Z be provided to this child.

In order to calculate J_4 explicitly it is necessary to make a distributional assumption about the random term V_i. If V_i is assumed to be normal with mean zero and variance σ_v^2, then the expected value of the health of child i to be born and breastfed in period four is given by:

$$(7)\quad E_4(h^A_{i,4}|Z = 1;\Omega_4)=\exp(\gamma^A_1 4 + \sum_{j=1}^{2} \gamma^A_2 n_j(4-t_j) + \gamma^A_3 i + \gamma^A_5 + \mu + \varepsilon_i + 1/2\ \sigma_v^2)$$

where γ^A_5 vanishes if child i is not breastfed. Let that part of (7) which contains all health determinants (inclusive of endowments) except Z be given by h^*_i; algebraic manipulation yields the following expression for J_4:

$$(8)\quad J_4 = \frac{h^*_{i,4}}{M_4}\ [\alpha_1(e^{\gamma 5}- 1) - \frac{\alpha_2}{M_4}\ \{\frac{h^*_{i,4}}{M_4}\ (e^{2\gamma 5} - 1) + 2(h^A_1 n_1 + h^A_2 n_2)\ (e^{\gamma 5} - 1)\}]$$

$$+ w_4\ (\beta_2(2F_4 - w_4) - \beta_1)$$

The effect of a change in the child-specific endowment of child i born in period four on the value of J_4 for a family with any given prior allocations of n and Z is thus given by:

$$(9)\quad \frac{\partial J_4}{\partial \varepsilon_i} = \frac{h^*_{i,4}}{M_4}\ [\alpha_1\ (e^{\gamma 5}-1) - \frac{\alpha_2}{M_4}\ \{2(e^{\gamma 5}-1)(h^A_1 n_1 + h^A_2 n_2) + \frac{h^*_{i,4}}{M_4}\ (e^{2\gamma 5}-1)\}],$$

where it will be recalled that $n_i = 0$ if no prior child is born.
Expression (9) cannot be signed, as there are two opposing forces at work -- an increase in the child's endowment, given the technology described by (2),

raises the return to the resource Z and increases J_4. This positive substitution effect is embodied in the first term in (9). On the other hand, an increase in the child's endowment raises mean health directly and, given diminishing marginal utility as embodied in the parameter α_2 in (5), induces "wealthier" parents to spend their endowment on other resources. The sign and magnitude of (9) thus depend on both technology and preferences. Indeed, if the health technology were linear (rather than loglinear), it can be easily shown that the first term in (9) would vanish. Thus, in the case where endowments do not affect the productivity of inputs, more endowed children are likely to receive fewer resources; intrafamily behavior would tend to be equalizing or compensatory. When endowments augment resource returns, as in (2), the effects of intrafamily variations in child endowments on the allocation of resources across children cannot be known a priori.[1]

The effects of endowment variations across families on the allocation of resources to children are even more complex. The effect of a change in the family endowment μ on the likelihood that a child born in the last period receives resource Z consists of two effects. The first is given by expression (9); an increase in μ increases the last child's endowment and thus, for given prior fertility and health decisions, induces the substitution and wealth effects discussed. However, families with different endowments will not in general have identical fertility patterns and will not have invested identical resources across all prior children. Prior fertility and other investment decisions affect the direction of the family endowment effect on the likelihood that the last child receives resource Z, from (8), to the extent that i) the child's own health is affected (via prior spacing

decisions) and ii) mean child health levels (h_1^A, h_2^A) are altered. If, for example, variations in the household health endowment μ affected only the allocation of Z (no fertility responses), then the effect of variation in μ on the probability that the last child receives resource Z is given by:

$$(10)\quad \frac{\partial J_4}{\partial \mu} = \frac{\partial J_4}{\partial \epsilon_i} - 2\alpha_2 \frac{h^*_{i,4}}{M_4} \left(h_1 \frac{dh_1^A}{d\mu} + \frac{dh_2^A}{d\mu} - n_2\right)$$

where $dh_i^A/d\mu$ is the total effect of a change in μ on prior children's health inclusive of resource allocations. As can be seen, if more endowed families have healthier children (even if they invest less in them), then the effects of interfamily variation in endowments on the probability that the last child receives resource Z will be algebraically less (more negative) than the effect due to intrafamily endowment variation. This is because well-endowed families, given taste homogeneity, will receive less utility from any additions to mean child health than will less-endowed families.

In general, households with different endowment levels will exhibit different patterns of fertility and resource allocative behavior. To ascertain the effects of endowment variations on the complete life-cycle behavior of families and thus on the last period decision would require enormously complex calculations even in the simple dynamic model. For example, to solve for the effects of prior children's endowments on the decisions concerning whether to have a child in the third period and whether to breastfeed the child born in the second period (if it exists) requires a computation which must take into account the probability distribution of the third period child's endowment and the optimal fourth-period parental responses just discussed. At the beginning of the third period, parents must compare expected future utilities associated with their alternative fertility

choices and with their allocations of Z for all combinations of alternative choices in periods three and four, i.e., to discern whether J_3 is positive or negative; where J_3 is:

$$(11)\quad J_3 = \max \{E_3(U|n_3 = 1, Z_2 = 1; \Omega_3), E_3(U|n_3 = 0, Z_2 = 1; \Omega_3), E_3(U|n_3 = 1, Z_2 = 0, \Omega_3), E_3(U|n_3 = 0, Z_2 = 0; \Omega_3)\}$$

$$\text{where } E_3(U|n_3 = 1, Z_2 = 1; \Omega_3) = E_3(U_3|n_3 = 1, Z_2 = 1) + E_3\{\max [E_4(U_4|Z_3 = 1, n_3 = 1, Z_2 = 1), E_3(U_4|Z_3 = 0, n_3 = 1, Z_2 = 1)]\}$$

and E_3 is the expectation operator, given information at the beginning of period three. While no precise predictions can be derived from (11), the results indicate that both the timing of childbearing (and thus intervals between births) and the allocation of resources across children will generally depend differentially on the household's health environment (or parental endowments) and on the individual endowed healthiness of the children.

2. Estimating the Effects of Parental Choices on Child Health Outcomes and the Effects of Endowment Heterogeneity

The principal impediment to both achieving consistent estimates of health equations such as (1) and (2) and of parental responses to endowment differences among children is the absence of direct information on endowments. With neither the family endowments nor the child-specific endowments observable to researchers, it is clear from either static or dynamic intrafamily optimizing models that the right-hand-side health inputs in (1) and (2) will be correlated with the health "residuals" containing both the unobserved μ and the child-specific endowment. Least squares estimates of the γs will thus be biased.

Two procedures have been employed to circumvent the potential biases arising from endowment heterogeneity. Olsen and Wolpin (1983) employ data

on siblings and use a family fixed effect estimation procedure (FFE) to estimate a child mortality function. However, their procedures, which demonstrate the sensitivity of results to estimation techniques, purges only the family endowment component μ from the residual; their study implicitly assumes that parents do not respond to child-specific traits (they thus rule out, for example, "replacement" effects.). Rosenzweig and Schultz (1983) employ two-stage least squares to estimate a birthweight equation. Their procedure assumes, however, that household or child health endowments are orthogonal to parental characteristics such as schooling and income, an assumption that will be tested (and rejected) below.

The information restrictions of the dynamic model associated with the sequencing of births suggest that consistent estimates of the input effects Γ can be obtained from data on siblings by using both "lagged" inputs, from <u>older</u> siblings, and parental characteristics as instruments in a fixed effect procedure. In particular, since the information set of parents at time t in family j cannot include the child-specific attributes ε_{ij} of children yet unborn, the following covariance restrictions are implied: cov $(Z^t_{ij}, \varepsilon^\tau_{kj}) = 0$, $t < \tau$, $i < k$; cov $(Z_{ij}, \varepsilon_{kj}) \neq 0$ $i \geq k$, where the superscript refers to time period; i.e., investments in child i at time t cannot be a function of child k's endowment ε_{kj} as long as they occur prior to child k's birth; Z_{kj} can be a function of both ε_{ij} and ε_{kj}.

Since the decision concerning <u>when</u> to have a child must be made in the absence of information on that child's specific endowments, sequencing additionally implies that cov $(n_{ij}, \varepsilon_{kj}) = 0$ $i \leq k$. This means that to estimate health outcome equations, all prenatal variables associated with child i will be appropriate instruments for <u>differences</u> in spacing and other prenatal inputs across child i and child i + 1. To see this, consider the birth outcome

difference equation for children one and two, from (1), with post-birth spacing variables appropriately deleted:

$$(12)\quad \tilde{lnh}^{0}_{2j} = (\gamma_1 + \gamma_2)\,\tilde{t}_{2j} + \gamma_4\,\tilde{Z}^{P}_{2j} + \tilde{\varepsilon}_{2j} + \tilde{v}^{0}_{2j}$$

where $\tilde{Y}_{2j} = Y_{2j} - Y_{1j}$.

As noted, OLS estimation of (12), equivalent to the family fixed effect or "sibling" difference method, would yield biased estimates of the γs, since $\tilde{t}_{2j}$ would be correlated with $\tilde{\varepsilon}_{2j}$, containing ε_{1j}. However, since t_{1j} and Z^{P}_{1j} are not correlated with either the unforeseen child specific endowments ε_{2j} or ε_{1j}, but are likely to be correlated with $\tilde{t}_{2j}$ and $\tilde{Z}_{2j}$, these lagged level variables are suitable instruments for (12) as well as the relevant difference equations for the post-birth health production technology in (2). Moreover, since the family component of the child's health endowment (the health environment, unobserved traits passed on from parents to children) is purged from (12), parental characteristics can also be used as instruments, since such characteristics (schooling, income) are unlikely to be correlated with the deviations of individual child traits among the offspring.

With appropriate information on birth outcomes, measures of child health, parental characteristics, and a family birth history, consistent estimates of the effects of maternal age, birth order, birth spacing and other parental inputs on health outcomes as well as of child endowments can thus be obtained using the lagged instrumental fixed effect (LIFE) procedure from families who have as few as two children. Since the residuals from such consistently-estimated birth outcome equations contain the child and family-specific endowment components, it is also possible to estimate the responses of the timing of births and the allocation of resources to individual children to those "initial" endowment components, if there are no missing child-invariant inputs (to estimate the effects of changes in μ_j) or missing child-specific inputs (to estimate child-specific endowment responses).

3. The Data

To implement the LIFE estimation procedure and to test for the importance of inter and intrafamily endowment effects in determining the timing of fertility and the allocation of resources among children, data are needed on parental characteristics, on demographic histories, and on health outcomees for multiple children within a household. We employ a unique data set from Candelaria, Colombia. These data were collected over a seven year period, from 1968 to 1974, to evaluate the impact of a program designed to provide child health services in all households in the town in which there were any children under the age of six. The services were provided by promotoras, who, at each visit (approximately every two months), also collected demographic and medical data on the individual children and parents. The data provide longitudinal information on the weight of all children under six during the entire survey period as well as information on such health inputs as innoculations (DPT) and breastfeeding.[2] There are also annual data on monthly food expenditures and family composition as well as basic socioeconomic information on parents, collected at the onset of the program. These data were analyzed by Heller and Drake (1979), who employed procedures which did not take into account any form of heterogeneity or dynamic behavior.[3]

To estimate the birth outcome equation (1) and post-birth health equation (2), we selected a subsample of 109 households in which at least two children were born during the seven-year program. For this subsample, information is thus available on health status at birth and on early post-birth input allocations for two or more siblings. The sample size is 238 children. An advantage of the data set is that none of the information was

collected retrospectively; thus the results obtained are not subject to recall error. However, the need for two or more siblings clearly results in a choice-based sample (households with higher fertility) and a relatively small sample size.[4]

We employ as a measure of health status the child's weight standardized for his or her age (in months) observed at birth and within six months after birth (the first post-birth observation).[5] The estimating equations are:

$$(13)\quad WT^{0}_{ij} = \gamma^{0}\varepsilon_{ij}\mu^{0}_{j}\, age^{\gamma^{0}_{1}}_{ij}\, int^{\gamma^{0}_{2}}_{ij}\, order^{\gamma^{0}_{3}}_{ij}\, food^{\gamma^{0}_{4}}_{ij}\, e^{\gamma^{0}_{5}\, sex_{ij}}$$

$$(14)\quad WT_{ij} = \gamma\varepsilon_{ij}\mu_{j}\, age^{\gamma_{1}}_{ij}\, int^{\gamma_{2}}_{ij}\, order^{\gamma_{3}}_{ij}\, food^{\gamma_{4}}_{ij}\, e^{\gamma_{5}\, sex_{ij} + \gamma_{6}\, DPT_{ij} + \gamma_{7}\, bf_{ij}}$$

where age_{ij} = maternal age at birth (of child i in family j), int_{ij} = prior interval, $order_{ij}$ = birth order, $food_{ij}$ = per-capita monthly food expenditure in household, sex_{ij} = 1 if the child is male, DPT_{ij} = 1 if child innoculated against DPT, and bf_{ij} = 1 if child breastfed.

Table 1 provides descriptive statistics on the sample children and households. The first two columns pertain to the sample of households who had at least two children during the Promotora program; the second two columns refer to the households who had one or more children born during the seven-year survey period.[6] This sample will be used to estimate the effects of endowment variations on parental decisions. All but the food expenditure variable of the set of household variables are used as instruments in obtaining the LIFE estimates of (13) and (14) along with the lagged maternal age at birth, birth order, and interval variables.

Table 1

Descriptive Statistics: Two-Child and One-Child Samples

Sample Variable	At Least Two Children Mean	S.D.	At Least One Child Mean	S.D.
Children	All Children		First Children	
Normalized Weight	.985	.186	.988	.192
Birth Order	5.29	2.86	4.62	2.85
Maternal Age at Birth (years)	27.6	5.98	27.3	6.32
Prior Interval (months)	23.5	14.7	27.6	19.5
Number of Older Siblings < 6	2.94	.877	2.40	.877
Breastfed	.885	.320	.888	.100
Innoculated (DPT)	.219	.415	.263	.189
Sex (male = 1)	.529	.500	.520	.500
Sample Size	238		383	
Families				
Years of Schooling - Mother	2.41	1.68	2.49	1.65
No Schooling - Mother	.211	.409	.179	.385
Monthly Income (pesos)	884	226	892	254
Per-Capita Food Expenditure	31.6	13.9	33.2	18.8
Enrolled in Family Planning Program	.0361	.188	.0493	.217
Sample Size	109		223	

4. Empirical Results: Behavioral Determinants of Normalized Weight

Because the sample selection rule may introduce bias into the least squares estimates of the weight-for-age equations (13) and (14) in addition to that resulting from health heterogeneity, a selection correction procedure was employed in which the determinants of the probability that the household was selected was first estimated as a function of the household characteristics.[7] These estimates were then used to predict the probability of sample inclusion for the sub-sample from which the γ estimates are obtained (Olsen, 1983). Because the family fixed effect and LIFE procedures purge out all household-level variables, no selection-correction variable is included when these procedures are used. All estimates, of course, pertain to children who lived for at least three to six months. In addition, to exploit estimation efficiencies, the two age-specific weight equations are estimated jointly as a system.

Table 2 reports estimates of the parameters of the normalized weight equations, obtained using seemingly-unrelated-regression (SUR), the family fixed effect procedure (FFE) and the lagged instrumental fixed effect technique (LIFE). Both the FFE method, which "corrects" for interfamily heterogeneity and within-family child-invariant omitted variables, and the LIFE method, which avoids as well biases associated with intrafamily heterogeneity, yield results which differ from those obtained using SUR and from each other. In particular, the negative effect of birth order on weight at birth appears to be understated significantly by both the SUR and FFE methods compared to the instrumental method -- the LIFE birth order coefficient in absolute value is double that provided by the FFE method and almost three-fold

Table 2

Behavioral Determinants of Log of Normalized Weight: At Birth and Within 6 Months After Birth

Estimation Procedure/Input	SUR (1)	SUR (2)	FFE (1)	FFE (2)	LIFE (1)	LIFE (2)
Sex (Male = 1)	-.0407 (1.59)[a]	-.0434 (2.06)	-.0425 (1.30)	-.0291 (1.25)	-.0410 (1.25)	-.0341 (1.26)
Maternal Age at Birth[c,d]	.0665 (0.82)	.0460 (0.69)	.310 (0.69)	.147 (0.45)	.761 (1.35)	-.488 (1.03)
Prior Interval[c,d]	.0404 (2.33)	.0306 (2.14)	.0501 (2.01)	.0311 (1.73)	.0563 (2.08)	.0224 (0.92)
Birth Order[c,d]	-.0842 (2.88)	-.0726 (2.96)	-.120 (1.14)	-.0853 (1.13)	-.244 (1.83)	-.0230 (0.21)
Breastfed [d]	-	.0316 (1.12)	-	-.0106 (0.24)	-	-.0358 (0.35)
Innoculated [d]	-	.0259 (1.22)	-	.0364 (1.29)	-	.0598 (1.15)
Food Per-Capita[c,d]	.0003 (0.25)	.0284 (1.23)	.00208 (0.31)	.0119 (0.28)	.00130 (0.08)	.133 (1.69)
λ[e]	-.164 (0.88)	-.265 (1.72)	-	-	-	-
Intercept	-.215 (0.85)	-.264 (1.09)	-	-	-	-
R^2	.056[b]	.092[b]	-	-	-	-
n	238		238		238	

a. Asymptotic t-values in parentheses.

b. From OLS regression.

c. Log of variable.

d. Endogenous variable.

e. Selection-correction variable.

larger than the SUR estimate of the birth order effect. The positive effect of the size of the interval preceding a birth on birthweight, statistically significant under all procedures, is ten percent greater when the LIFE method is employed compared to using the FFE method and is almost 49 percent higher than the corresponding SUR estimate. Moreover, while the SUR estimates suggest that children who are breastfed experience (marginally significantly) greater weight gains, the breastfeeding coefficients are neither positive nor significant when estimated with either the family fixed effect or LIFE methods. While this result does not necessarily imply that breastfeeding is ineffective (since the effect of breastfeeding depends on its duration and intensity and breastfeeding may augment survival), the estimates suggest that inattention to heterogeneity overstates the effects of breastfeeding incidence on children's weight and understates the effects of interval, length and birth order.[8] Moreover, the effects of household food consumption per-capita, and to a lesser extent, of innoculations appear also to be understated using either the SUR or FFE methods; but neglect of heterogeneity across and within households appears to lead to an overestimate of the persistent effects of birth order and birth intervals on post-birth weight.

While many of the individual coefficients are not measured with much precision, application of the Wu/Hausman test indicates rejection of the hypotheses that the behavioral inputs are uncorrelated with the residuals in the equations estimated by the SUR and FFE methods at the five percent level (F-test). Heterogeneity both within and across the sample households appears to be affecting the sample variation in the inputs and thus the estimated coefficients. Moreover, the magnitudes of the consistently-estimated effects (from the LIFE estimates) of some of the variables on weight

are not trivial -- increasing the mean birth interval from two to four years increases weight at birth by 16 percent (interval plus age effect); an increase in monthly per-capita food intake by 20 percent and early innoculation against diptheria, polio or tetanus raises weight-for-age within six months after birth by 2.6 percent and six percent respectively.

5. Empirical Results: Intra and Interhousehold Heterogeneity and Household Resource Allocations

As noted, the residuals $\hat{\eta}^a_{ij}$, obtained by subtracting the predicted standardized weight values based on the consistently estimated (LIFE) parameters from actual standardized weight values, contain the child-invariant household endowment, the child specific endowment, and a random error. By averaging the $\hat{\eta}^a_{ij}$ over all children i for the two periods in a family j, a consistent estimate of the family "effect" for family j $\hat{\mu}_j$ may be obtained since $\underset{i\to\infty}{\text{plim}}\ (\mu_j + \varepsilon_{ij} + V^a_{ij}) = \mu_j$. Child-specific deviations of the $\hat{\eta}^a_{ij}$ from $\hat{\mu}_j$ averaged over two periods provide an estimate of the child-specific effects $\hat{\varepsilon}_{ij}$ for family j.

Interpretations of each of the two residual components μ and ε_{ij} (plus random measurement error) as endowments/requires different assumptions about the completeness of the set of health inputs in (13) and (14). The family effect, $\hat{\mu}_j$, will unambiguously represent the exogenous health endowment of the family only if there are no omitted child-invariant endogenous variables in (13) or (14), a strong assumption. The violation of this assumption does not, of course, mean that the FFE or LIFE estimates of the γs are inconsistent (that must be due to (optimizing) behavior with respect to the child-varying inputs based on household information about the ε_{ij}). Rather, variations in $\hat{\mu}_j$ may then be due to interfamily variations in unobserved endogenous inputs and thus may reflect interfamily heterogeneity in both preferences and endowments. The

residually-estimated ε_{ij}s, however, will represent child-specific endowments/ plus random measurement error to the extent that there are no important inputs which vary across children within a family, a weaker assumption. The associations between the $\hat{\varepsilon}_{ij}$ and family allocation decisions may correspond more to endowment effects than will the associations between the $\hat{\mu}_j$ and such household behavior.[9]

To estimate how variations in the health endowments of households are related to the across-household variations in fertility and household per-capita nutritional intake, we regressed the number of children less than six years of age, children ever born and monthly per-capita food consumption at the start of the survey period (1968), and maternal age at the birth of the (first) child born during the sample period on the computed household health endowment and a set of parental socioeconomic variables including the mother's schooling attainment and predicted family income based on the father's schooling, age and occupation.[10] Because exclusion of households who had less than two children during the survey period from the sample would obviously impart bias to these fertility and consumption estimates, we employed the augmented sample of households, including as well those who had only one child in the seven-year survey period. To compute the household and child-specific health endowments for the "one-child" households, we first regressed the estimated household endowments $\hat{\mu}_j$ on the total child residuals $\hat{\eta}_{ij}$ using the two-plus child sample. The estimates were then used to predict the household and child-specific endowments based on the child residuals (or total child endowments) computed from the information on the relevant life-cycle weight and input variables for each of the children born during the survey period in the "one-child" household sample (using the LIFE estimates of Table 2).

Table 3 reports the regressions employing the computed and estimated household endowments for the augmented sample. As can be seen, differences among households in the inherent average healthiness of their children is significantly correlated with inter-household differences in the pace and magnitude of fertility--households with better-endowed children exhibit significantly higher cumulative fertility and tend to have births significantly earlier; such households do not, however, consume significantly different levels of food per capita, controlling for the schooling attainment of the mother and income. Since such fertility behavior, given the estimates of Table 2, tends unambiguously to diminish the nutritional status of children at birth, the results imply that children born in healthier households, net of family inputs and income, tend to receive less favorable inputs. Inherent across-household inequalities in children's healthiness appear to be reduced by household fertility behavior.

endowment (which are biased to zero)
The /point estimates/indicate that in households in which children on average are ten percent heavier at birth than average children in the town population (net of parental resources), the number of children ever born is higher by about one-half child and the mother accelerated the timing of the first birth during the survey period by over one year. The LIFE estimates of Table 2 suggest that such adjustments in fertility behavior would reduce weight at birth by 5.4 percent. About one-half of the initial weight advantage is thus erased due to fertility responses to family health endowment variation; children in high-μ households retain their inherent advantage on net ($dh/d\mu > 0$ in equation (10)).

The vector of socioeconomic variables is also significantly correlated with each of the fertility and food consumption variables in Table 3. The set of parental variables is also , however, significantly correlated with the computed family health endowment (five percent significance level).[11]

Table 3

Family Endowments, Fertility and Per-Capita

Food Expenditure

Variable	Children Ever Born	Maternal Age at Birth	Children Under Six	Per-Capita Food Expenditure
Family Health Endowment	.992 (1.67)[a]	-10.8 (9.42)	.359 (1.90)	.309 (0.08)
Income ($x10^{-3}$)	-.818 (0.80)	-.681 (0.36)	-.176 (0.55)	20.5 (3.06)
Schooling of Mother	-.286 (2.28)	-.620 (2.67)	.00629 (0.16)	1.34 (1.64)
Family Planning	-.818 (0.80)	.965 (0.51)	-.146 (0.46)	-2.69 (0.40)
Intercept	6.26 (6.90)	26.82 (15.8)	2.63 (9.27)	11.9 (2.01)
R^2	.055	.362	.021	.074
d.f.	218	218	218	218

a. t-values in parentheses.

This latter result implies that "healthiness" net of parental resources is not distributed randomly across the population with respect to either the educational attainment of parents or income. Thus, estimates of income or schooling effects on fertility and other health-related variables which ignore health heterogeneity across households may also be biased.[12] Indeed, the health endowment has a stronger relationship with the fertility variables than does income. The estimates indicate that, for given health endowments, income is not significantly associated with the number or spacing of children; however, higher income families consume more food per-capita. These estimates suggest that interventions that improve the health environment may induce somewhat higher fertility levels; however, income-augmenting projects would appear to have little effect on fertility. Moreover, mothers with higher levels of schooling have significantly lower family size, although they tend to have children earlier. Since only eleven of the 223 sample households contained a mother who was enrolled in the family planning program, no precise estimates can be obtained of the effects of this program; however, the relevant coefficient signs suggest that the program may be lowering fertility.

The estimates of Table 3 suggest that observationally identical households with differing health endowments exhibit significantly different fertility behavior, such that inherently healthier children appear to receive less favorable allocations. To ascertain if within-household disparities in child health endowments are exacerbated or lessened by intra-family parental allocative behavior, we estimated the effects of variations in two child-specific endowments--the health endowment as measured by $\hat{\varepsilon}_{ij}$ and the gender of the first child born in the sample period--on the subsequent fertility behavior of the parents and on the probabilities that the child is

breastfed and/or receives the DPT vaccine. Table 4 reports the maximum likelihood logit estimates of the probabilities of a subsequent short fertility interval (within three years after the birth of the first sample child), of the child being breastfed, and of the child being provided the DPT innoculation, as functions of the two child-specific endowments, the household endowment and the socioeconomic variables. These estimates indicate that while the set of socioeconomic variables is not statistically significantly related to the dependent variable in any equation, resource allocations within the household do respond to exogenous variations in the characteristics of children, although not uniformly. In particular, children with higher-than-average health endowments within the family are significantly more likely to have a more closely-spaced younger sibling than their less well-endowed siblings, but are also more likely to be breastfed. This latter result suggests why use of the single equation procedure may have overstated the "effect" of breastfeeding incidence on child weight in Table 2; as indicated in the model, evidently the returns to breastfeeding depend positively on the inherent healthiness of the child. On the other hand, the closer spacing following the birth of a healthier-than-average (or expected) child may reflect mainly an "income" effect, with parents "spending" their additional unanticipated wealth (endowment) on additional or more rapidly-accumulated children. Finally, despite boys having a weight disadvantage at birth (Table 2), neither subsequent spacing nor the probability of a child receiving breastmilk appears to be related to gender; innoculations, however, appear to be provided to boys more often than to girls but to be orthogonal to health endowments measured by weight-for-age.

Table 4

Maximum Likelihoood Logit Estimates: Family and Child-Specific Endowment Effects on Post-Birth Interval, Breastfeeding, Innoculation

Variable	Short Interval	Breastfed	Innoculated
Child Endowment	7.91	2.82	.244
	(5.45)	(1.44)	(0.55)
Family Endowment	-1.19	.416	.430
	(2.61)	(0.54)	(0.30)
Sex of Child (male=1)	-.175	.0261	.767
	(0.51)	(0.01)	(2.33)
Income ($\times 10^{-3}$)	-.537	.863	.448
	(0.54)	(0.74)	(0.61)
Schooling of Mother	-.0684	-.184	.0505
	(0.75)	(1.34)	(0.51)
Family Planning	-.981	.218	.848
	(1.14)	(0.12)	(1.34)
Intercept	.605	2.09	-2.79
	(0.68)	(1.85)	(2.94)
d.f.	217	217	217

a. Asymptotic t-values in parentheses.

6. Conclusion

While there is a large scientific literature concerned with the child health consequences of household decisions, interest in the determinants of household decision-making over time has just begun. Few empirical studies of health have thus taken into consideration parental dynamic behavior. In this paper, we have formulated a simple dynamic model incorporating uncertainty to demonstrate the complexity of household decision rules concerning the allocation of resources to and across children when there is both unanticipated and sequential variation in child traits within the family and variation in healthiness across households. Estimates of the effects of the timing and level of fertility, use of medical services, food consumption, and breastfeeding on early measures of childrens' nutritional status were obtained based on an estimation procedure informed by the dynamic model. These estimates were compared to estimates obtained using procedures which ignore either or both intrafamily health heterogeneity and parental adjustments to child-specific-health shocks.

The results, obtained from a longitudinal sample of households in Colombia, suggested that, consistent with the model, parental behavior appears to respond to unanticipated health outcomes among children and is also significantly associated with more persistent health factors, unrecorded in the data, that vary across households. As a consequence, estimates of the child health effects of parental decisions, or the fertility effects of child mortality, ignoring the behavioral consequences of inter and intra-family heterogeneity would appear to be biased. In particular, our results indicated that single-equation or family fixed effect techniques underestimate the negative consequences for birthweight of high fertility and short birth intervals, but overstate them

for post-birth weight. Moreover, as an evident consequence of inherently healthier children being more likely to be breastfed, the estimates neglecting heterogeneity appear to overstate the positive effects of breastfeeding.

Estimates of the effects of within-household and across household variation in endowments also appeared to suggest that i) the "endowed" healthiness of households net of parental resources allocated to children was a more important determinant of fertility behavior than income, with healthier households evidently having more children at earlier ages, and ii) within households, healther surviving children are more likely to be followed by a closely-spaced, subsequent child and to be breastfed. These results imply that existing estimates of fertility responses to child mortality confound intra and interhousehold endowment effects.

A cost of our estimation procedure, which makes use of longitudinal information on multiple children within a household to obtain production function estimates immune to missing household-level information and the existence of dynamic adjustments by parents, is low sample size and consequent loss of estimation precision. Our results imply, however, that cross-sectional samples taken from populations with little observed variation in exogenous variables (excluding parental characteristics), no matter how large or detailed, would be inadequate for obtaining consistent estimates of the consequences of parental resource allocations or of fertility behavior for child health or mortality. Moreover, longitudinal data on single children (no siblings) may also be inadequate, to the extent that there is little intertemporal variability in exogenous variables and, net of child-specific fixed effects, serial correlation in endowments over

time for a child is important relative to serial correlation of endowments across siblings net of both family and child-specific endowments. Finally, while we have estimated directly the parameters describing the health technology, no attempt was made to estimate the parameters characterizing parental preferences, thus our estimates pertaining to parental responses to within and across household endowment variation are merely first-order approximations to family behavior rules, and are subject to the usual caveats about reduced form estimates.

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Footnotes

1. Additional ambiguity results when parents are also directly concerned about health disparities across their children. Assume that the variance in child-specific health outcomes enters linearly in the quadratic utility function (5) with a coefficient of $-\alpha_3$. Then the following ambiguously-signed term is added to expression (9):

$$(9') \quad \frac{2\alpha_3}{M_4^2} h^*_{i,4} \left[(h_1^A n_1 + h_2^A n_2)(e^{\gamma 5} - 1) - h^*_{i,4} (M_4 e^{\gamma 5} - 1) \right]$$

$$= - \frac{2\alpha_3}{M_4} h^*_{i,4} \left[e^{\gamma 5} (h^*_{i,4} - H_4) + H_4 - h^*_{i,4} (2 - e^{\gamma 5})/M_4 \right]$$

The sign of (9') depends in part on whether the health of the final-period child net of the effect of the Z resource exceeds the mean health of all children inclusive of prior resources. Since less-endowed prior children may have greater health outcomes than does a subsequent child, due, for example, to negative maternal age and birth order effects, with inequality-averse parents it is thus possible, even when the health technology is linear, for a better-endowed last child to receive resource Z.

2. Height information was also collected, but only after two years of the program had elapsed. Restriction of our sub-sample (described below) to children with both height and weight information would have reduced the sample size by 40 percent.

3. Indeed, their specifications yield results that are not interpretable as estimates of either technology or preferences; the usefulness of their partial correlations is unclear.

4. We test for selectivity below. Note that if we had solved the dynamic model for both resource and fertility decisions, such a correction would be automatic in a full maximum likelihood approach.

5. Weight at birth has been shown to be a significant predictor of physical growth, development and morbidity; see for example Chernichovsky and Coate (1980) and Beck and van den Berg (1975). No study of the consequences of early child health status variables has taken into account heterogeneity, however.

6. There were 640 households in the original data containing a mother of childbearing age with children less than age 7 sometime during the sample period and with no missing information on the relevant variables used in the analysis. Of these, 223 had at least one child born during the sample period for which the relevant data were recorded. Because of village immigration and outmigration during the 7-year period the mean number of years of sample exposure for households is 3.8. All but 10 of the 109 households bearing two or more children were in the sample the full 7 years.

7. The sample selection equation included all of the family-level variables listed in Table 1, excluding per-capita food expenditure but including the ages of the mother and father in 1968, when the promotora program began. Not surprisingly, maternal age in 1968 and family planning enrollment were the two most significant determinants of sample inclusion; both variables were negatively associated with the probability of meeting the sample criteria.

8. The breastfeeding results are similar to those reported in Olsen and Wolpin (1983); correction for across-household heterogeneity reduced significantly the apparent positive breastfeeding effect on child survival.

9. Olsen and Wolpin (1983) and Rosenzweig and Schultz (1983a and 1983b) also employ production function residuals to estimate behavioral responses to health endowments. None of these studies distinguish between adjustments to unanticipated child-specific shocks and inter-family endowment heterogeneity. Olsen (1983) attempts to decompose the child-specific (mortality) production function residual into the relevant child and family components and to estimate the fertility response to an unanticipated child death. However, his production function estimates are obtained using the family fixed effect method, which assumes the absence of intrafamily responses. His finding of a significant "replacement" effect indicates that his estimates and those of Wolpin and Olsen are thus inconsistent.

10. The first-stage income estimates are:

$$\begin{aligned} \text{income} = {} & 956 - \underset{(5.14)}{10.1}\ \text{agefather} + \underset{(1.26)}{.146}\ (\text{agefather})^2 - \underset{(1.92)}{363}\ (\text{agefather missing}) \\ & + \underset{(3.44)}{45.8}\ (\text{schoolfather}) - \underset{(1.79)}{104}\ (\text{father} = \text{manual laborer}) \\ & + \underset{(6.95)}{522}\ (\text{father} = \text{clerical worker}) + \underset{(1.47)}{18.8}\ (\text{schoolmother}) \\ & - \underset{(0.30)}{2.46}\ (\text{agemother}) \end{aligned}$$

11. Households with a higher health endowment had significantly lower income ($t=2.32$) but contained fathers with marginally significantly higher schooling attainment ($t=1.45$). The schooling attainment of the mother was not statistically significantly related to the household health fixed effect.

12. Wolfe and Behrman (1983) suggest that estimates of income effects on child health may be misleading due to the existence of other family

endowments. Their data do not permit estimates of interfamily health heterogeneity. Our results (Table 3 and fn. 8) imply that estimated income effects on fertility obtained without controlling for health endowments would be negatively biased and those for maternal age at birth positively biased; the estimated income elasticity for food is not sensitive to health heterogeneity, however.