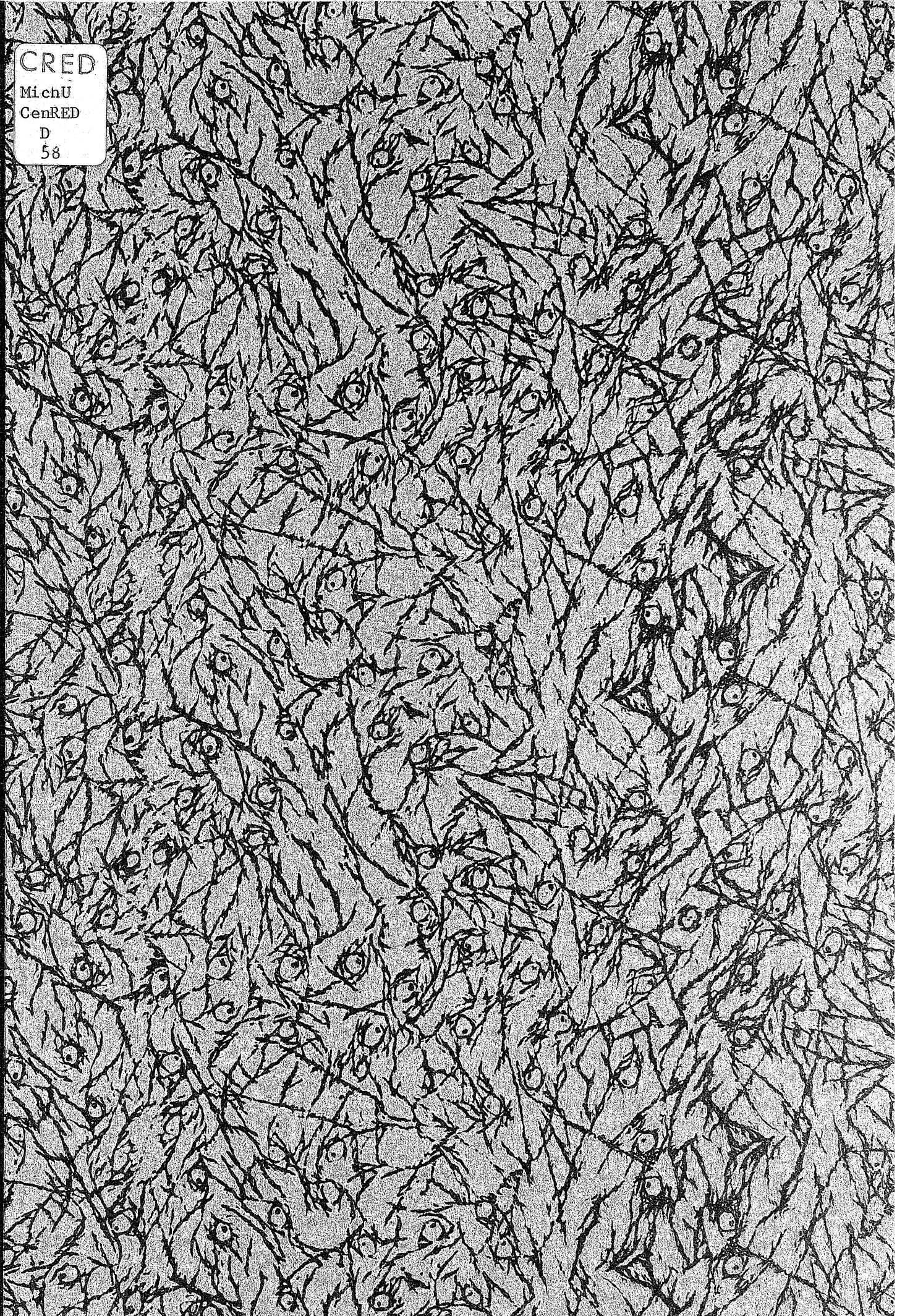

Malnutrition, Child Morbidity and the Family Decision Process

by

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ABSTRACT

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This paper developes an econometric model of the nutritional and health status of pre-school children in developing countries. The model is addressed to three central issues: (1) What are the characteristics of the interaction between a child's nutritional status and its degree of risk to morbidity, particularly diarrhea? (2) What is the effect of alternative kinds of nutrient inputs on a child's nutritional status (e.g. nursing vs food expenditure)? (3) Is a child's nutritional and health status influenced by economic constraints on the family and its intra-family resource allocation decisions? The model is estimated on observations on the health and nutritional status of approximately 1200 children who participated over a 7 year period in the Promotora maternal-child program in Candelaria, Colombia. The results suggest that: (1) severe diarrhea substantially weakens a child's nutritional status, (2) acutely malnourished and stunted children are more susceptible to diarrhea (3) nursing in the first year is pivotal to a child's nutritional status, (4) one may view malnutrition and poor health as the consequence of family processes. The results support the hypothesis that both child competition and parental discrimination adversely influence nutritional status.

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Cet exposé développe un modèle économétrique du statut alimentaire et de la santé des enfants d'âge pré-scolaire dans les pays en voie de développement. Le modèle concerne trois questions centrales: (1) Quelles sont les caractéristiques de l'intéraction entre le statut alimentaire d'un enfant et le degré de son risque de morbidité particulièrement diarrhée? (2) Quel est l'effet des différentes sortes de consommations d'aliments sur le statut alimentaire d'un enfant (par exemple frais de nourrice par rapport aux dépenses alimentaires)? (3) Les conditions alimentaires et de santé d'un enfant sont-elles influencées par des contraintes économiques subies par la famille et par les décisions inter-familiales concernant l'allocation des ressources? Le modèle est évalué d'aprés des observations sur le statut alimentaire et de la santé d'environ 1200 enfants qui ont participé pendant une période de 7 années au programme de santé mère-enfant Promotora à Candelaria en Colombie. Les résultats suggèrent que: (1) une sévère diarrhée affaiblit substantiellement le statut alimentaire d'un enfant, (2) les enfants extrêmement mal nourris et chétifs sont plus susceptibles d'avoir la diarrhée, la prise en charge par une nourrice dans la première année est une période critique pour le statut alimentaire d'un enfant, (4) on peut considérer la malnutrition et la mauvaise santé comme la conséquence des facteurs de décision familiale. Les résultats corroborent l'hypothèse que, et la compétition entre enfants, et la discrimination parentale, influencent défavorablement le statut alimentaire.

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MALNUTRITION, CHILD MORBIDITY AND THE FAMILY DECISION PROCESS*

by Peter S. Heller and William D. Drake

I. Introduction

In many developing countries, childhood malnourishment and morbidity are among the most serious burdens of underdevelopment. Their social cost in resources and welfare is perceived as both unnecessary and intolerable, and as a consequence, the promotion of child health is often accorded one of the highest nominal priorities among social development targets. There is a considerable literature by nutritionists and medical scholars on the characteristics and interaction of malnourishment and disease in developing countries. Economists have also begun to evaluate the cost-effectiveness of specific nutritional supplementation or health programs. Yet the formulation of such programs and the evaluation of their impact remains seriously limited. In part, this reflects the complexity of the process of nutritional change. Yet most studies lack a coherent model of the forces influencing change in a child's nutritional and health status, give only limited consideration to socioeconomic variables, and lack sufficient data over time to empirically measure the impact of specific policies.

In this paper, we estimate an econometric model of the nutritional and health status of pre-school children. Our data are drawn from observations on approximately 1200 children who participated over a 7-year period in the Promotora maternal-child health program in Candelaria, Columbia.[1] Three considerations dictated the specification of the model. First, any model of the dynamics and interaction of malnourishment has to be in accord with the understanding of these issues in the medical and nutritional literature.

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[1] Promotora is the Spanish term for the nurse volunteers who worked in the program.

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Second, it should incorporate the economic perspective that a family's health status is strongly influenced by economic constraints, and by family decisions concerning fertility, intra-family resource allocation, work and child care.[1] The model tests the hypothesis that socioeconomic factors channel a child into different degrees of medical and nutritional risk. Third, the model strives for sufficient specificity to generate policy-relevant implications for nutritional and health programs in developing countries. In Section II, we provide a detailed discussion of the model and its specification for econometric analysis.

Section III provides a description of the Promotora program, discusses the data used in the analysis and the limitations on the generality of our results. Section IV discusses econometric issues in estimating the model. In Section V, we examine the results and implications of the estimated model. Three sets of issues are of central concern:

(1) Can we statistically discern the interaction of a child's nutritional status with its degree of risk to morbidity? For example, does an episode of diarrhea or another illness seriously weaken the child's nutritional status, and does this further raise its risk of morbidity?

(2) What effect would potential policy interventions have on a child's health of nutritional status? For example, what is the impact of: (i) breastfeeding relative to other sources of nutrient intake? (ii) the implementation of a Promotora program or another preventive health program? What kinds of policies will be successful in ameliorating the nutritional status of children who already fall below a minimum nutritional level?

(3) Is a child's nutritional and health status influenced by economic constraints on the family and its intra-family resource allocation decisions? For example, what factors determine the level of food expenditure and the decision to nurse? Is there evidence of parental discrimination between children or of the adverse effect of fertility on the allocation of resources to each child? Is the quality of parental care an important factor in predicting the child's nutritional or health status?

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[1]For examples of this view, see Heller (1976), Welch (1974), Willis (1973), and Grossman (1972).

II. The Model

Our model is based on the assumption that a child's nutritional and health status reflects the combined impact of basic physiological development processes and of economic decisions made by the family, within the context of a given environment. The latter determines the quality and quantity of resources devoted to a child over the course of its early development.

The realization of a child's natural propensity for physical growth is constrained by whether it obtains an adequate quantity of different nutrient sources at each phase of its development path[1] and by whether its body is able to utilize these nutrients efficiently for physical growth. The quality and quantity of nutrients are constrained by the availability of family time and monetary resources, and are influenced by parental knowledge and cultural attitudes, by the degree of competition within the family for these resources, and by the priority attached to an individual child by the parents.

The efficiency of nutrient utilization is critically influenced by a child's health status. Some childhood illnesses, such as diarrhea, may limit the body's absorption of nutrients. Parasitic illnesses common to many developing countries (LDC's) may constitute an independent source of demand for nutrient resources within a child's body. Episodes of fever increase the nutrient requirements for normal bodily development. A child's health status may also influence the level of nutrient input; in some cultures, parents respond to the symptoms of illness by the temporary withdrawal of normal food and water intake.

Similarly, a child's health status is responsive to parental decisions. Any environment is associated with specific probabilities of contracting different diseases. In developed countries, the general level of morbidity risk is lower, due to the higher quality of the external environment and the smaller number and lesser virulence of disease agents. In less developed countries, this "public health externality" is less apparent, and the degree of insulation of a child from these risks and its capacity to overcome illness without significant pain and disability will be more of a function of

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[1] This does not imply that a child cannot partially "catch up" from a period of inadequate nutritional development [Morley (1973)].

its family situation. In part, this relates to whether the parents provide adequate clothing and shelter, are hygienic in the preparation of food and the disposal of wastes, and take advantage of available preventive and curative medical facilities. Yet the decisions that influence a child's nutritional status are also important. A malnourished child is more vulnerable to infection.

In no instance can these inputs fully insulate a child from illness. Virtually all children will be ill with diarrhea or other diseases at some point. In fact, it is through exposure to disease agents and through limited duration illnesses that a child acquires a degree of subsequent natural immunity.[1] Thus, our model only allows us to explain why some children are excessively at risk to morbidity.

The essence of the model is summarized in equations (1) and (2). Assume that for any child $\underline{i}$ in family $\underline{j}$ at time $\underline{t}$, a physiological production function explains the transformation of food and nonfood inputs into the child's nutritional status, N_{ijt}, and health status, H_{ijt}:

$$N_{ijt} = f(N_{ijt-1}, B_{ijt}, q_{ijt}\pi_{ijt}, H_{ijt}) \quad \text{and} \tag{1}$$

$$H_{ijt} = g(N_{ijt}, M_{ijt}, H_{ijt-1}, \Delta N_{ijt}, E), \tag{2}$$

where B_{ijt} measures the nutrients provided through nursing (breast-feeding), q_{ijt} is a vector of the quantity of other kinds of nutrient inputs, π_{ijt} is a vector of weights for the nutritional quality of each element of q_{ijt}, M_{ijt} is a vector of nonfood inputs to the child (parental attention, clothing, medical care, shelter, etc.) and E is a measure of environmental quality.

In (1), a child's nutritional status in period t is a function of its past nutritional status (or birthweight, N_{ij0}, for a child in the neonatal period),[2] the quantity and quality of nutrient inputs, its ability to use

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[1] In fact, it is a matter of debate among tropical pediatricians whether it is advisable to shield the child fully from the risks of particular diseases for fear that as a consequence the child will never build up any natural immunity (e.g. the debate on the advisability of maleria prophylaxis in the Danfa Project in Ghana). (Morley (1973)),

[2] In specifying an equation for N_{ij0}, the child's weight at birth, the arguments would relate as much to the nutritional and health status of the mother and the quantity and quality of her nutrient intake. We attempted to estimate an econometric model of child birthweight from our sample, but were precluded by the absence of precise birthweight data.

these resources efficiently and the level of current bodily demand for nutrients. For an infant or toddler, the latter two factors are functions of the child's health status. In (2), the health status of a child in a given quality of environment E is a function of whether it is well-nourished, of its past health status and by the extent to which nonfood inputs M_{ijt} insulate the child from health risks. These physiological production functions are presumed generalizable across children.

In the content of economic models of household behavior,[1] a family decision process determines B_{ijt}, $(q_{ijt}\pi_{ijt})$, M_{ijt} and perhaps E (through migration). Parents are assumed to derive utility over their lifetime from the number and quality of surviving children as well as from their own consumption of leisure and commodities. Parental choices are subject to constraints which are both internal and external to the family. At any point in time, their command over resources is limited by their wealth, the market wage rate and commodity prices. Parental age (l_{jt}) and knowledge of child-rearing (K_{jt}) will influence their efficiency in achieving a given level of child quality. Family decisions are also made within a given policy environment, and this may influence the constraints on parents, as well as the relevant market parameters.[2]

Usually in models of this kind, parents are assumed not to differentiate between household members in allocating resources.[3] Yet it can be argued that parents are forced by the pressure of limited income to make interpersonal judgments between themselves and their children. Most if not all of these decisions are made without malice or forethought.[4] Yet this may involve conscious discrimination; e.g. for working members of the household, against girls or against higher parity children.

In maximizing utility for the family, parents develop a set of decision rules concerning their allocation of time and of income as between activities,

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[1] See Heller (1976) and Willis (1973).

[2] For example, if a policy program were introduced which involved nutritional supplementation of children under age 6, this would influence parental decisions; similarly, the promotion of a program may influence parental child rearing efficiency.

[3] An exception to this is reflected in papers by Welch (1974) and Heller (1976).

[4] In another paper by the author (Heller 1976), it is argued that the parent's utility functions include the number of surviving children as arguments, and that the probability of survival is itself parentally influenced if not determined.

[5] Unconscious physiological discrimination may occur against higher parity children; e.g. if the quality of breast milk is lower in nutrient value, or the attention of older parents is less.

commodities and members of the family. We hypothesize that the following reduced form equations for $q_{ijt}\pi_{ijt}$ (hereafter F_{ijt}), B_{ijt} and M_{ijt} embody a subset of these rules:

$$F_{ijt} = F(y_{jt}, n_{jt}, a_{jt}, P_{Fjt}, s_{ij}, b_{ij}, l_{ij}, K_{jt}) \quad (3)$$

$$B_{ijt} = B(y_{jt}, n_{jt}, a_{jt}, P_{Fjt}, s_{ij}, b_{ij}, l_{ijt}, K_{jt}, w_{jt}) \quad (4)$$

$$M_{ijt} = M(y_{jt}, n_{jt}, a_{jt}, s_{ij}, b_{ij}, P_{Mjt}, l_{ijt}, K_{jt}) \quad (5)$$

where y_{jt} is the level of family j's income at time t, n_{jt} is the number of living children competing for resources within the family, a_{jt} is the number of adult members of the household, P_{Fjt} is the cost to the family of alternative food resources, w_{jt} is the mother's opportunity cost of time in the market, s_{ij} is the sex of the child, b_{ij} is the birth order of the child, and P_{Mjt} is the cost of nonfood inputs.

Estimation of the reduced form equations (3) to (5) is often infeasible. It requires data on the level of food and nonfood inputs directly to a child. Usually only aggregative family data are available. Although it is often possible to learn whether a child is nursing, it is hard to characterize the extent to which it has been weaned. This suggests direct estimation of the structural equations (1) and (2) so that the arguments in equations (3) to (5) become instruments for the F_{ijt}, B_{ijt} and M_{ijt} vectors. As described below, the formal specification of our econometric model closely follows the conceptual framework of equations (1) through (5).

Nutritional Status Model

Our choice of nutritional status indicators reflects the apparent consensus in the nutrition literature that differentiates the process of nutritional change into its acute and chronic manifestations. Specifically, until recently nutritionists have used a child's weight-for-age (WTAGE) as an anthropometric measure of nutritional status. A child's weight is compared with the median weight of a large sample of children of the same age. If its weight falls below 85 to 90% of this standard, it is considered "mildly"

malnourished. Lower percentile positions are associated with "moderate" and "severe" levels of malnourishment.[1]

Two problems have commonly arisen with this standard. First, the age of a child is often only imprecisely known or "culturally unavailable" in the rural areas of developing countries. Second, it fails to distinguish betwen (1) children who were stunted at some point in their physical development, and are thus of low weight for their age because they are short, and (2) children of normal height who suffer a sudden weight loss.

In the process of malnourishment, a child's physical growth may become stunted, i.e. significantly low in height for its age. However, once stunted, a child's physical development may thereafter proceed quite normally if nutrient intake proves sufficient, although always at lower absolute height and weight levels. Though low in WTAGE, such a child may not be even mildly malnourished in the sense of insufficient nutrients relative to bodily requirements, nor at risk to the principal effects of malnourishment, such as greater morbidity and possible retardation in mental and psychosocial development.[2,3] These criticisms have led to the distinction between a child's weight-for-height (WTHT), and height-for-age (HTAGE).

A child's weight relative to the median weight for a sample of children of the same height and sex (WTHT) provides a measure of the acute malnourishment associated with recent weight loss and insufficient nutrient intake and/or bodily absorption. This standard is age-independent and thus insensitive to errors in family estimates of child age. A child's height relative to the median height for a sample of children of the same age and sex (HTAGE) provides a measure of whether a child is physically stunted in its structural

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[1] Alternative weight-for-age standards have been used, with the Gomez (Mexico) and Harvard standards being most common. In general, the range for mild malnourishment is 75 to 90, moderate 60 to 75 and severe, <60 in terms of percentile position. Other kinds of standards for measuring nutritional status are also available, including arm circumference measures, clinical tests for the oedema and marasmus rates, and laboratory tests for hemoglobin and serum albumin levels. See Zerfas et. al. (1975) for a survey of such standards.

[2] Waterlow (1973), (1974), Habicht (1974).

[3] Most economists have also used a weight-for-age standard. See Selowsky (1971), Selowsky and Taylor (1973), and Berg (1973). This will lead to erroneous judgments concerning the magnitude of "malnourishment" within a population or of the effect of a change in policy.

development due to chronic or long-term nutrient deficiency. The standards recently recommended by the National Academy of Sciences (N.A.S.) have been used in our analysis.[1] The percentile positions of a child are:

$$WTHT_{ijt} = \frac{Weight_{ijt}}{\tilde{Weight}\ (Height_{ijt})}$$

$$HTAGE_{ijt} = \frac{Height_{ijt}}{\tilde{Height}\ (Age_{ijt})}$$

$$WTAGE_{ijt} = \frac{Weight_{ijt}}{\tilde{Weight}\ (Age_{ijt})}$$

where an $\sim$ represents a norm weight or height which is a function of the sex and either the age or height of the child, based on the N.A.S. standard. In our modelling we have included the WTAGE standard as well for comparative purposes.

The importance of this distinction can be noted immediately by comparing the percentage of children in our sample that have fallen below a given percentile position on the three standards. A substantial fraction--32%--of the Candelaria population have fallen below the 85th percentile on WTAGE. In part, this may reflect that the recommended standard of the N.A.S. is based on a more affluent population than the Candelaria group. Yet this proves a misleading statistic on malnourishment because more than 80% of these children have a weight which, relative to their height, is above the 85th percentile. In fact, although the mean WTAGE percentile position was 92.0, the mean of the WTHT position was 100.27. Although the Candelaria population is short relative to the N.A.S. standard, with a mean of 95.5%, significant stunting is even more limited than acute malnourishment. Only 3% of observations are on children below the 85th percentile in HTAGE.

Neither the economic consequences of a low percentile position for either of these measures nor the process by which the two malnourishment

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[1]The source of the data is listed and the techniques used to develop these measures are summarized in National Center for Health Statistics (1976).

phenomena occur are well-established in the literature. Stunting is clearly associated with equally slow cellular growth in the brain, although there is no conclusive evidence on the effect this has on mental capacity or development. Others have argued that the intellectually damaging consequences of malnourishment arise from the resulting apathy and listlessness that preclude a normal learning and intellectual response to the environment.[1] This might be the consequence of a low WTHT position. It is an area where further research is clearly needed.

The three principal nutrition equations, derived from (1), (3), (4), and (5), are specified in (6) through (8) below:

$$\begin{aligned} WTHT_{tk} = {} & \beta_{10} + \beta_{11}FOOD_{tk} + \beta_{12}NURS_{tk} + \beta_{13}WEANL_{t} + \beta_{14}PR.SEVERE.DIAR_{tk} \\ & + \beta_{15}PR.MILD.DIAR_{tk} + \beta_{16}PR.SICKNESS_{t} + \beta_{17}[FOOD*PR.DIAR]_{tk} \\ & + \beta_{18}SEX_{k} + \beta_{19}BRTHORD + \beta_{1,10}(\#CH<6) + \beta_{1,11}INTVL + \beta_{1,12}EDMO \\ & + \beta_{1,12}EDFA + \beta_{1,14}AGEFA_{t} + \beta_{1,15}AGEMO_{t} + \beta_{1,16}AGEDUM_{tk} \\ & + \beta_{1,17}PROMO_{tk} + \beta_{1,18}BTHCON + \beta_{1,19}WTHT_{t-1} + \beta_{1,20}HTAGE_{t-1} + \varepsilon_{1}, \end{aligned} \tag{6}$$

$$\begin{aligned} HTAGE_{tk} = {} & \beta_{20} + \beta_{21}FOOD_{tk} + \beta_{22}NURS_{tk} + \beta_{23}WEANL_{t} + \beta_{24}SEVERE.DIAR_{t-1,k} \\ & + \beta_{25}MILD.DIAR_{t-1,k} + \beta_{26}PR.\ SICKNESS_{t} + \beta_{27}[FOOD*PR.DIAR]_{tk} \\ & + \beta_{28}SEX_{k} + \beta_{29}BRTHORD + \beta_{2,10}(\#CH<6) + \beta_{2,11}INTVL. + \beta_{2,12}EDMO \\ & + \beta_{2,13}EDFA + \beta_{2,14}AGEFA_{t} + \beta_{2,15}AGEMO_{t} + \beta_{2,16}AGEDUM_{tk} \\ & + \beta_{2,17}PROMO_{tk} + \beta_{2,18}BTHCON + \beta_{2,19}WTHT_{t-1} + \beta_{2,20}HTAGE_{t-1} + \varepsilon_{2} \end{aligned} \tag{7}$$

and

$$\begin{aligned} WTAGE_{tk} = {} & \beta_{30} + \beta_{31}FOOD_{tk} + \beta_{32}NURS_{tk} + \beta_{33}WEANL_{t} + \beta_{34}PR.SEVERE.DIAR_{tk} \\ & + \beta_{35}PR.MILD.DIAR_{tk} + \beta_{36}PR.SICKNESS_{t} + \beta_{37}[FOOD*PR.DIAR]_{tk} \\ & + \beta_{38}SEX_{k} + \beta_{39}BRTHORD + \beta_{3,10}(\#CH<6) + \beta_{3,11}INTVL + \beta_{3,12}EDMO \\ & + \beta_{3,13}EDFA + \beta_{3,14}AGEFA_{t} + \beta_{3,15}AGEMO_{t} + \beta_{3,16}AGEDUM_{tk} \\ & + \beta_{3,17}PROMO_{tk} + \beta_{3,18}BTHCON + \beta_{3,19}WTAGE_{t-1} + \varepsilon_{3} \end{aligned} \tag{8}$$

where $\underline{k}$ refers to an observation on a child when it is in the k^{th} age group. Table 1 displays the definitions of the variables used in the empirical

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[1] N.A.S. (1973); Cravioto (1973); Cravioto in Scrimshaw & Béhar (1976); Garn(1975)

List of Variables with Means and Standard Deviation in parentheses*

$WTHT_t$	= percentile position for ith child's weight relative to its height, on basis of NAS standard in period t. (100.27, 10.59)
$WTAGE_t$	= percentile position for ith child's weight relative to its age, on basis of NAS standard in period t. (91.49, 11.64)
$HTAGE_t$	= percentile position for ith child's height relative to its age, on basis of NAS standard in period t. (95.09, 4.93)
$\Delta WTHT_t$	= Change in percentile position, weight for height $(WTHT_t - WTHT_{t-1})$
$BTHWGHT_0$	= weight of child for earliest observation before age 2 months, if child is 0-11 months; necessary to omit observation if child is 0-11 months with no birthweight data; 0 otherwise. (in 1000 grams)
$STUNTED_t$	= 1 if child's height for age percentile position was less than 90th percentile in period t-1, 0 otherwise.
$MALNOUR_{tk}$	= 1 if child's weight for height percentile position was less than 90th percentile in period t-1, 0 otherwise for a child in the kth age group.
$FOOD_{tk}$	= weekly real food expenditure per capita within the household in period t in 1968 pesos. (25.35, 15.63)
WEANL	= 1 if observation is during or directly after weaning (the latter, within 3 months). (.23, .42)
$NURS_{tk}$	= includes the variables (i) Months Nursed, (ii) Pct. of 1st year nursed, (iii) Pct. of 2nd year nursed and (iv) WEANL, as defined above
MONTHS NURSED	= for children over 23 months, age of child at which it terminated nursing; 0 otherwise. (9.05, 6.53) (11.69, 7.85 on sample of children, aged $\geq$ 24 months).
PCT. OF 1ST YR. NURSED	= for children < 12 months, fraction of its age it has been nursed; for all others, 0. (.80, .29 on sample of children aged 0-11 months)
PCT. OF 2ND YR. NURSED	= for children with 12 months $\leq$age <24 months, fraction of its age it has been nursed; for all others, 0. (.49, .30 on children aged 12-23 months)
AGE WEANED	= number of months before child was weaned. (10.75, 7.15)
SEX	= sex of child (boys = 0, girls = 1).
BRTHORD	= birth order of the child within the household. (4.11, 2.54)
INTVL	= number of months between age of child and its predecessor; if no pervious child, INTVL = 72. (46.4, 22.31)
#CH<6	= total number of children in the household of age less than 72 months. (2.33, .96)
EDFA	= educational level of the father (1=illiterate, 2=read & write only, 3=primary school (grades 1-5), 4=secondary school (grades 6-11), 5=technical, (2.78, .76)
EDMO	= education level of the mother (as above). (2.79, .73)
AGFA	= age of father in years (35.52, 8.47)
AGMO	= age of mother in years (29.06, 6.50)
BTHCON	= 1 if mother used the pill or IUD for birth control; 0 otherwise. (.38, .48)
$PROMO_{tk}$	= percentage of child's life it was in the Promotora program, at time t for child of age k. (.84, .24)
DPT	= 1 if child ever received a DPT immunization; 0 otherwise. (.14, .19)
POLIO	= 1 if child ever received a polio immunization; 0 otherwise. (.10, .17)
$INCOME_t$	= monthly family income in pesos in real 1968 (125.31, 95.07)
$YEAR_t$	= calendar year associated with observation in period t.
PRENAT	= 1 if mother ever received prenatal care; 0 otherwise. (.31, .46)
$SICKNESS_t$	= 1 if child was ill since previous visit by Promotora; 0 otherwise. (.24, .43)
PR. SICKNESS	= estimated probability that child was ill since previous visit by Promotora. (.25, .08)
$DAYS\ SICK_t$	= number of days sick since previous visit by Promotora.
$MILD\ DIAR_{tk}$	= 1 if child had 1-4 days of diarrhea during the month preceding visit in time t, 0 otherwise, where diarrhea is defined as more than 3 loose stools per day; for a child in kth age group (.08, .27)
PR. MILD $DIAR_{tk}$	= estimated probability that child had a mild diarrhea episode during month preceding visit in time t. (.07, .04) for a child in kth age group
$SEVERE\ DIAR_{tk}$	= 1 if child has had more than 4 days of diarrhea during the month preceding visit in time t; 0 otherwise, for a child in kth age group (.08, .27)
PR. SEVERE DIAR	= estimated probability that child had a severe diarrhea episode during month preceding visit in time t, for a child in kth age group (.07, .03).
$(FOOD\text{-}PR.\ DIAR)_{tk}$	= product of weekly food expenditure per capita in the household and the probability of a diarrhea episode in time t for a child in the kth age bracket.
$AGEDUM_{tk}$	= this includes dummy variables for (i) age, 0-11 months, (ii) age 12-23 months, (iii) age 24-35 months, (iv) age 36-48 months, (v) age 49-72 months, as described below.
AGE, 12-23 MONTHS	= 1 if child is aged 12-23 months; 0 otherwise.
AGE, 24-35 MONTHS	= 1 if child is aged 24-35 months; 0 otherwise.
AGE, 36-48 MONTHS	= 1 if child is aged 36-48 months; 0 otherwise.
AGE, 49-72 MONTHS	= 1 if child is aged 49-72 months; 0 otherwise.
AGE, 0-11 MONTHS	= 1 if child is aged 0-11 months; 0 otherwise.
$\Delta AGE_{t,\ t-1}$	= change in age in months between period t and t-1 for given child.

*We've omitted the ith term for child and jth term for family where it is ... spacing between t and preceding and subsequent observations is child-s... The t subscript refers to the current observation on any child. The

analysis as well as their mean and standard deviation (where applicable). The variables fall into several principal categories related to specific policy issues and hypotheses about the process of nutritional change.

(1) What is the impact of alternative nutrient sources on the process of nutritional development, and at what ages do each prove most influential? Ideally, one would want measures of the actual nutrient input to a child at a particular point in time. Unfortunately, we only have information on the level of food expenditure within the family and on whether the child was nursing at the time of the observation. Two sets of variables are used in the model. The first measures the level of real food expenditure per capita, where the divisor is simply the size of the household. Multiplicative dummy variables are used to measure the impact of food expenditure at ages 2-11 months, 12-23 months, 24-47 months, and over 47 months.[1] Although this is a reasonable proxy for food intake for a weaned child, it is not as accurate as would be desired for policy analysis for the entire sample.[2]

Similarly, four variables have been developed to capture the short and long term effects of nursing and weaning: (i) the percentage of a child's life nursed for children aged 0-11 months (0. otherwise), (ii) the percentage of a child's life nursed for children 12-23 months (0. otherwise), (iii) the number of months nursed for children aged 24 months and over, and (iv) a dummy variable equalling 1 if an observation on a child occurs in a period in which it is being weaned.[3] Variables (i) and (ii) attempt to

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[1] In Section IV, we have described the method used in specifying the multiplicative dummy terms.

[2] There is a legitimate problem of interpretation of the food expenditure variable. The variable measures real food expenditure in the household, where the divisor is simply the number of persons in the household. If a child were only breast fed at time t, food would only be a factor influencing its nutritional status to the extent that the quality and quantity of milk is contingent on the level of maternal nutrient intake. The divisor would therefore only be accurate if the mother's consumption was twice as large as other household members. After weaning, our measure is accurate if one assumes equal sharing of food within the household. The problem is rendered more complex where children are only partially weaned.

[3] The data in our sample included information on whether a child was nursing at the time of the visit. Given multiple observations on each child, there are three classes of children--those who are still nursing,

capture any nonlinearity over time in the impact of nursing.[1] Variable (iii) measures whether there is any legacy to a weaned child of a long period of nursing. Variable (iv) attempts to measure the heightened risk of a decline in nutritional status associated with the weaning period.

(2) It is well-known that a child's nutritional status may be impaired by illness.[2] Each day of a diarrhea episode costs a child one to two days of nutrients and leads to the development of a negative calcium and caloric balance. Children who are ill have a weakened ability to absorb nutrients efficiently. It is critical that any model of nutritional status take account of a child's health status. By its omission, the coefficients of variables measuring nutrient intake would be seriously biased, since their impact is contingent on the efficiency of nutrient consumption. Since the number and timing of the observations in our sample was not carefully controlled, it proved impossible to reconstruct the child's medical history at any given point in time. Our data do enable us to differentiate (i) whether a child had had an episode of diarrhea that was mild (1-4 days) or severe (> 4 days) in its duration during the month preceding the observation, and (ii) whether it had had any other illness since the previous visit of the Promotora, on which we have information (on average, 6 months earlier).[3] The probability of illness for a child in Candelaria is high. The probability of a mild and severe diarrhea episode is approximately .075 and .080, respectively, during the first year of a child's life and .095

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those who, according to our observations have never been nursed, and those for whom the timing of the termination of breastfeeding can be established. For the last group, the midpoint age between the nursing and off-nursing observations is taken as the age weaned; for the middle group, the midpoint age between our earliest observation on the child and the birth is assumed as the age weaned; for the former group, complete weaning has not yet occurred.

[1] We did not simply use the "number of months nursed" since it is likely to be highly collinear with age.

[2] Scrimshaw, et al. (1968).

[3] Each observation on a child was taken at the time of a visit by the promotora to the child's family (as discussed in Section III). Although visits were made every two months, our data includes a sample of observations chosen to be approximately 6 months apart.

and .097, respectively, during the second year.[1] The probability of other kinds of illness is .246.

It is important to accurately specify the dynamics of the impact of an illness episode. An episode of diarrhea or another illness is likely to have an immediate effect on a child's weight, and be reflected in the child's WTHT or WTAGE percentile position. However, it is unlikely that diarrhea in the month preceding an observation would adversely influence a child's height. In our HTAGE model, we test for the impact of a severe or mild level of diarrhea in the month preceding the t-1st observation. It is also of policy interest whether the impact of diarrhea changes at different ages, and again, multiplicative dummy variables are used to capture any possible nonlinearities in effect.

What is the impact of poor health status on the role of nutrient input? To measure this, we introduce a nonlinear term equalling $[Food*Pr.Diarr]_{tk}$ where $[Pr.Diarr_{tk}]$ is the estimated probability that a child will have a diarrhea episode in time t for a child of age k. There are two opposing processes implicitly reflected in this variable. One would expect that diarrhea would cut into whatever impact food expenditure would have on the child's nutritional status, thus yielding a negative coefficient for this variable. Yet in the context of diarrhea, the greater the food expenditure, the smaller the negative impact of diarrhea on nutritional status. The sign and significance of the coefficient suggests which of these two relationships is dominant, though both may be present and significant.

(3) Parental control over resource allocation implies the possibility of parental "discrimination" among children. An egalitarian hypothesis would suggest that the sex or birth order of a child would not have a long-term influence on the child's nutritional status or on the intra-family allocation of resources.[2] Two variables test for discrimination: (i) a dummy term for the sex of a child and (ii) the birth order of a child within the household.

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[1]The distribution of diarrhea by days of duration may be summarized: 0 days--85.1%; 1 day--.7%; 2 days--2.1%; 3 days--2.4%; 4 days--1.4%; 5-10 days--5.0%; 11-20 days--1.6%; 21-"90" days--1.4%.

[2]This assumes that our anthropometric measures have adequately taken account of normal differences in development between the sexes. There is no reason to anticipate such bias in the N.A.S. data. There may be some tendency for birth order to influence the child's neonatal nutritional status. In any case, one would not expect that these differences would be compelling over a long period in the child's life.

(4) The ability of parents to provide adequate resources and attention for any child is weakened by the extent of implicit competition with other siblings. Children for whom the birth interval with a preceding child is particularly short are presumably more at risk of lessened resources, partly because of the lack of time and attention. A child in a family with a large number of siblings would also obtain fewer resources, and this should be reflected in its nutritional status. Two variables test the nutritional consequences of a high fertility household: (i) the number of children under age 6, (ii) the interval between a child and predecessor.

(5) The quality of parental input is implicitly the focus of many nutritional and preventive health programs. Parental human capital accumulates either through parental experience or through various channels of education. We include measures of the formal educational level of mothers and fathers and of the age of the mother and father. Both sets of variables lend themselves to some ambiguity in their hypothesized impact. Age is not only correlated with experience but with diminished physical energy and possible fatalism or lessened concern for the quality of the children. Children of older mothers have the short-term disadvantage of a lower average birthweight. Higher education may promote values which are detrimental to child health (bottlefeeding being the most common example).[1]

(6) Notwithstanding the fact that our nutritional indicators either correct for age or are age independent, age dummies for each of the first four years of life were included to measure any tendency for change in percentile positions over the child's early years. This provides a more accurate test of the hypothesis of an earlier study that there is a "natural history" of nutritional development reflected by a fall in nutritional status after the first 12 months with a rise after 48 months.[2]

(7) Since all observations are on participants in the Promotora program, how can we differentiate the degree of program participation across children in order to assess the program's impact? Since a child could have entered the program at any point in its life,[3] the fraction of its life in the

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[1]This has been discussed in a paper by Wray and Aguirre (1969).

[2]Drake and Fajardo (1976).

[3]Although in principle children enter the program at birth, children of new migrant families and others not previously reached by the program entered later.

program, for children of the same age, is one possible measure of the degree of participation. Yet across children of different ages, one might expect that the longer a child has been in the program, the more beneficial the impact.[1] To capture both effects, we apply multiplicative dummies to test the nutritional impact of the fraction of the child's life in the program, according to whether the child is aged one, two, three or four or more years. Since the Promotora program also disseminates family planning information, it is hypothesized that parental use of modern birth control methods proxies the extent and enthusiasm of their participation in the program. A dummy variable tests this hypothesis.

(8) The process of nutritional change is a dynamic process. At any point in time, a child's nutritional status is not likely to change dramatically from the previous period, and thus we include the lagged dependent variable.[2] Since the timing of an observation was determined by when the Promotora visited the child's family, it is necessary to essure some comparability in the time frame between visits. It would be misleading if the gap between the t and $(t-1)^{st}$ observation of two children differed widely (e.g., 2 vs. 24 months). Thus, one period lags are taken only when the time difference between observations is 3 to 9 months.[3]

Health Status Model

Although it would be more useful to test our model in (2)-(5) by examining the risks of illness from the separate disease agents to which a child is commonly exposed, our data do not allow this. However, we have information on a primary contributing factor to poor nutritional status, viz., diarrhea. This includes information on the number of days the child was sick with diarrhea in the preceding month. In addition, we have data on the total number of days of any kind of illness since the previous visit by the promotora, a period of approximately 6 months.

Both measures of illness carry a heavy subjective element, depending on the accuracy of parental recall and variable criteria for "abnormality"

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[1] The "number of months" in the program is collinear with age.

[2] The econometric implications of this are discussed in Section IV.

[3] The average interval between observations is 5.4 months, with a standard deviation of 1.96 months.

in health status.[1] Medical problems that are without telling or unusual symptoms may pass unnoticed by the parent. Since the information on the number of days of diarrhea and of sickness are of questionable accuracy, we limited ourselves to estimating a model of three dichotomous dependent variables: (i) whether or not the child had a mild episode of diarrhea in the preceding month ($\leq$ 4 days episode), (ii) whether or not the child had a severe level of diarrhea in the preceding month ($\geq$ 5 days) and (iii) whether or not the child was ill since the previous visit of the promotora.

Despite its frequency, the etiology of diarrheal disease is not fully established. As Morley puts it, "the diarrheal stool has been a happy but rather barren hunting ground for microbiologists for many years." No single pathogenic agent is primarily responsible, and only in a quarter of cases can any specific pathogen be isolated at all. Discussions of the disease inevitably focus on circumstances in which the risk is greatest.

Children in the weanling period are argued to be the most susceptible. Their exposure is greatest (as they use their mouth to identify objects and because of their newly found mobility). Their degree of immunity to new pathogenic agents is low, and their vulnerability to substitution of an inadequate and imbalanced diet is greatest. Potential exposure to faeces-borne disease agents remains greatest in environments with inadequate waste disposal methods and/or low volumes of water usage, since this will influence the presence and density of disease agents. Malnourished children are at the greatest risk since there is evidence that their cellular immunity is less because of the impact of the malnourishment on the thymus gland.[2]

The specification of the structural equations to explain the occurrence of a mild or severe diarrhea episode in (9) below reflects these considerations but also tests some of our earlier hypotheses concerning the impact of economic factors on health status.[3] Since our data do not differentiate the other kinds of illnesses experienced in our sample, our specification of our model for the occurrence of a child's sickness in (10) is assumed

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[1] Studies have shown that recall accuracy declines significantly after one week (Woodward ( )).

[2] Morley (1973).

[3] The specification for the mild and severe diarrhea equations are identical.

identical to the model in (9).

$$\left\{\begin{matrix}\text{MILD DIAR}_t \\ \text{SEVERE DIAR}_t\end{matrix}\right\} = \beta_{40} + \beta_{41}\text{MALNOUR}_{t-1,k} + \beta_{42}\text{STUNTED}_{t-1} + \beta_{43}\text{BTHWGHT} \quad (9)$$
$$+ \beta_{44}\Delta\text{WTHT}_{t,t-1} + \beta_{45}\text{INCOME}_t + \beta_{46}\text{EDMO} + \beta_{47}\text{EDFA}$$
$$+ \beta_{48}\text{AGEFA} + \beta_{49}\text{AGEMO} + \beta_{4,10}\text{SEX} + \beta_{4,11}\text{BRTHORD}$$
$$+ \beta_{4,12}(\#\text{CH}<6) + \beta_{4,13}\text{INTVL} + \beta_{4,14}\text{NURS}_{tk}$$
$$+ \beta_{4,15}\text{WEANL}_t + \beta_{4,16}\text{DPT} + \beta_{4,17}\text{POLIO} + \beta_{4,18}\text{PRENAT}$$
$$+ \beta_{4,19}\text{BTHCON} + \beta_{4,20}\text{PR.SICKNESS} + \beta_{4,21}\text{AGEDUM}_{tk}$$
$$+ \beta_{4,22}\text{PROMO}_{tk} + \beta_{4,23}\text{YEAR} + \varepsilon_4$$

$$\text{SICKNESS}_t = \beta_{50} + \beta_{51}\text{MALNOUR}_{t-1,k} + \beta_{52}\text{STUNTED}_{t-1} + \beta_{53}\text{BTHWGHT} \quad (10)$$
$$+ \beta_{54}\Delta\text{WTHT}_{t,t-1} + \beta_{55}\text{INCOME}_t + \beta_{56}\text{EDMO} + \beta_{57}\text{EDFA}$$
$$+ \beta_{58}\text{AGEFA} + \beta_{59}\text{AGEMO} + \beta_{5,10}\text{SEX} + \beta_{5,11}\text{BRTHORD}$$
$$+ \beta_{5,12}(\#\text{CH}<6) + \beta_{5,13}\text{INTVL} + \beta_{5,14}\text{NURS}_{tk}$$
$$+ \beta_{5,15}\text{WEANL}_t + \beta_{5,16}\text{DPT} + \beta_{5,17}\text{POLIO} + \beta_{5,18}\text{PRENAT}$$
$$+ \beta_{5,19}\text{BTHCON} + \beta_{5,20}\text{AGEDUM}_{tk} + \beta_{5,21}\text{PROMO}_{tk}$$
$$+ \beta_{5,22}\text{YEAR} + \varepsilon_5$$

For this specification:

(1) the literature suggests that poor nutritional status may heighten the risk of illness. Two dummy variables are introduced which equal one only if a child's WTHT or HTAGE percentile position was less than 90% in the previous period. For infants, birthweight is probably the best indicator of initial nutritional status. A variable equalling birthweight for children under 12 months is introduced. After the first year, the legacy of the birthweight is likely to be minor relative to current nutritional status.

(2) Some pediatricians argue that changes in weight or height are useful clinical indicators of a child's risk of morbidity. A child high in its WTHT or HTAGE position but experiencing a significant slowing in the rate of growth in weight or height may be clinically more at risk to illness than a child with a low percentile position that is stable or exhibiting improvement.[1] Since it is a sudden weight change that is the primary source

[1]Morley (1973).

of concern, only a change in WTHT percentile position has been included. In the structural equation system, $\Delta WTHT_{t,t-1}$ is an endogenous variable.

(3) The ability of parents to insulate a child from the risks of morbidity is measured by real family income per capita. Differences in parental efficiency are proxied by the educational level and age of the parents.

(4) The role of parental priorities in the allocation of family resources is again tested by the sex and birth order variables; the effect of family competition by variables measuring the birth interval and the number of siblings under age 6.

(5) A child is often exposed to illness through the poor quality of food and its unhygienic preparation. We would hypothesize this exposure to be lowest during the period the child is breastfed. A dummy variable is used to evaluate the increased sensitivity of the child to diarrhea and other diseases during the weaning period.

(6) The receptivity of parents to preventive medical services may be indicative of their attitude toward hygiene and health care. Several proxy indicators are available: (i) whether a child received a D.P.T. or polio immunization; (ii) whether a mother received any prenatal care and (iii) whether she uses a modern method of birth control.

(7) Since the onset of diarrhea is often induced by other illnesses, we include in the diarrheal equations an estimate of the probability of sickness for the child.

(8) Dummy variables for a child's age test for any nonlinear reduction in the risk of morbidity obtained through exposure to pathogenic agents over its life.

(9) The impact of the Promotora program on child health is tested by applying the indicators of program participation discussed above.

(10) Finally, the occurrence of diarrhea or illness is likely to be seasonally correlated. Since we cannot date each observation, this cannot be tested in the model. A calendar year variable is introduced to capture any secular change in the quality of the environment arising from program activities or other factors.

Parental Decisions on Nutrient Intake

Finally, we may use the framework of parental choice to explain two critical parental decisions in the child's life: (i) the age at which the

child is weaned and (ii) the level of food expenditure per capita in the household. The length of time a child is breastfed will reflect both cultural and economic considerations. In most developing countries, modernization breeds contempt for breast-feeding. This bias is reinforced the higher the opportunity cost of the mother's time, since it raises the price of nursing relative to bottle-feeding. Thus, we would expect an earlier age of weaning for children in families with higher income levels and more education, in the absence of a countervailing pro-nursing campaign.

Second, since a common message in the Promotora and other preventive health programs is the importance of nursing, one would expect a positive correlation between program participation and age at weaning. Again, three indicators of participation are used (percentage of child's life in the Promotora program, use of modern birth control methods and receipt of prenatal care by the mother). Finally, the mother's physiological capacity to nurse would influence the age at weaning, and this might be negatively related to a child's birth order or mother's age.

This model is estimated on the set of observations taken at the time of weaning for all children for whom precise weaning data could be derived. The model specification is:

$$\begin{aligned}\text{Aged Weaned} = {} & \beta_{60} + \beta_{61}\text{INCOME}_t + \beta_{62}\text{PROMO}_t + \beta_{63}\text{PRENATAL} + \beta_{64}\text{BRTHORD} \\ & + \beta_{65}\text{SEX} + \beta_{66}\text{AGEMO} + \beta_{67}\text{INTVL} + \beta_{68}\text{EDMO} + \beta_{69}\text{EDFA} \\ & + \beta_{6,10}(\#\text{CH}<6) + \varepsilon_6.\end{aligned} \tag{11}$$

A model that explains the level of real food expenditure per capita in the household is important for policy analysis of some nutritional program alternatives. Evaluation of any program that focuses on income transfers (and possibly even nutritional supplementation) must measure the potential leakage that will emerge between the growth in income and ultimate changes in nutritional status. This requires an estimate of the initial leakage of income to nonfood expenditure. A conventional demand equation of the form:

$$\text{Food Expenditure} = \beta_{70}\text{INCOME}^{\beta_{71}}(\#\text{CH}<6)^{\beta_{72}}(\text{EDMO})^{\beta_{73}}(\text{EDFA})^{\beta_{74}}\varepsilon_7 \tag{12}$$

is used. Dividing by INCOME and taking logarithms, this was estimated as

$$\begin{aligned}\ln\frac{\text{Food}}{\text{INCOME}} = {} & \ln(\alpha_{70}) + (\beta_{71}-1)\ln\text{INCOME} + \beta_{72}\ln(\#\text{CH}<6) + \beta_{73}\ln(\text{EDMO}) \\ & + \beta_{94}\ln(\text{EDFA}) + \ln\varepsilon_7\end{aligned} \tag{13}$$

where β_{71} is the income elasticity for food expenditure.

III. The Data[1]

Our data consists of multiple observations on 1,270 children from the town of Candelaria, Colombia (population, approximately 8,000). The observations were made between 1968 and 1974 while the children participated in the Promotora program. Our sample was chosen from a larger data set containing observations on over 80% of all children under six years and their families in the town. This section will briefly describe the Promotora program, the characteristics of the Candelaria population and the criteria applied to choose the subsample of observations used in our analysis. Since the results are undoubtedly sensitive to the particular population sample studied, the general applicability of the policy implications to be drawn must not be overstated.

Setting

Candelaria is a small town 30 kilometers from Cali. It is a transient community, containing a large number of migrants from the outlying rural areas who reside in Candelaria only temporarily before further migration to Cali and other larger urban areas. This is reflected by an annual population growth rate of 10%, despite an estimated outmigration rate of 17%.[2] The bulk of employment is derived from the sugar cane plantations or sugar processing factories. Although there is some home production of vegetables and other foodstuffs, most of the population appears to depend on cash incomes for survival.

Candelaria is not a typical LDC community. It is neither fully rural nor urban. It is only 30 kilometers from a city which has an active and innovative medical research and educational center, and which had a rural health program as an offshoot of the latter for 6 to 10 years prior to the inception of the Promotora program.[3] As a result of municipal water and

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[1]The description of Candelaria and of the Promotora program is drawn from Drake and Fajardo (1976).

[2]The outmigration estimate is from operating statistics of the Promotora program.

[3]For example, for a 14 month period beginning in 1964, weekly food supplements were distributed to each person of nutritionally vulnerable age (pre-school children, pregnant and nursing moethers). Mothers in groups of 25-30 took part in seven educational presentations.

sewage programs, 67.7% of the homes inside the city had sanitary waste facilities and 90.9% had piped water inside their homes by 1964.

These numbers are likely to be overestimates for the Promotora client population, since the latter is drawn from a wider area. Nevertheless, it does suggest that it is not an environmentally backward area. The impact of this on the malnourishment and morbidity rates cannot be accurately determined but a 1963 survey conducted prior to the supplementation program indicated a malnourishment rate of 40.8% compared to the 55.6% shown in the 1965 national survey, using a WTAGE standard. As a consequence, by the time Promotora was initiated, the children of Candelaria were already comparatively healthy by rural Latin American standards. In 1968, the Promotora program revealed a malnourishment rate of roughly 30% on a WTAGE standard.[1] Finally, although it has many transient residents, as a community it is accustomed to projects being conducted by outsiders.

Despite these limitations on the "typicality" of Candelaria, it would be foolish to disregard the unusual volume of demographic, medical, socioeconomic and nutritional data that has been collected there. It is still a poor community, with average monthly income less than U.S. $30.00. Although the malnourishment rate is not at famine levels, it is high enough that there is still much one may learn about the process of nutritional change.

The Promotora program was established in 1968 to provide home-based preventive and maternal-child health services. Its major objective was to prevent childhood diseases as well as to reach children before illness had progressed to a point requiring extensive and costly medical treatment. Ten volunteer health workers--promotoras--between the ages of 16 and 21 and with at least five years of primary school education were given six months of training. Their task was to visit all families with children less than six years of age every two months. The volunteers provided education on nutrition, hygiene and on the role of modern medical services, gathered data on each child's height and weight and referred sick children to a health center staffed by paramedical workers. Pregnant and lactating mothers were also instructed in pre-natal and post-natal care. Finally, the promotoras regularly collected demographic, medical and socioeconomic data on both the family and the individual child.

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[1] A. Pradilla (1973).

At the time of first participation in the program, family socioeconomic and data were obtained along with the health status of each child under six years of age. Thereafter, health and nutritional status data on the children were obtained on each visit of the Promotora to the family. In addition, there was an annual update on the family's composition, monthly income, weekly food expenditure and method of birth control.

The Sample

Although there were 9,800 observations on approximately 1,900 children from approximately 1,100 families, a smaller sample has been drawn for the purposes of our analysis. In the first two years of the program, height data was not collected, and thus it was impossible to estimate WTHT or HTAGE. Only observations for which height information is available are used in our analysis. This involves 5,350 observations on approximately 1,300 children. Since at least one period lagged variables are used in the analysis, this pares the sample further.[1] The number of observations per child ranges from one to five, with an average of three (taking account of a one period lag).

IV. Econometric Issues

The estimation of the model requires three principal adjustments for deviations from the assumptions of ordinary least squares. First, three of our dependent variables--the occurrence of severe and mild diarrhea, and of sickness, are dichotomous. For these variables OLS estimation would be inefficient, due to heteroscedasticity, and misleading for predictive purposes (since there is no guarantee the predicted $\hat{y}$ would be in the interval (0,1). A maximum likelihood estimation procedure--logit--of the form

$$\log\left[\frac{P(Y_{it}=1)}{(1-P_{it}(y=1))}\right] = \alpha_0 + \alpha_1 X_{1it} + \alpha_2 X_{2it} + \ldots + \alpha_n X_{nit} + \varepsilon_{it}$$

is used, where, for example, $P(Y_{it}=1)$ is the probability that the i^{th} child

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[1] A further paring occurred by eliminating all cases where weekly food expenditure was recorded as less than or equal to zero. This occurred in cases where the family lived with another family or an extended family.

will have a severe diarrhea episode in period t.[1] Second, the model as specified, suggests the possibility of simultaneous equations bias, and thus the structural equations are estimated using two stage least squares (TSLS). Instruments for the included endogenous variables of mild and severe diarrhea episodes and sickness in the nutritional status equations are estimated from a first stage logit estimation on the entire set of exogenous variables. These instruments measure the predicted likelihood of a particular disease episode.

Third, the data set is a pooling of cross section and time series observations. In order to test for autocorrelation, estimates of ρ were obtained by estimating

$$\hat{\varepsilon}_{it} = \rho\hat{\varepsilon}_{i,t-1} + u_{it}$$

for each of the structural equations, where ε_{it} is the estimated residual from the second stage of the TSLS estimations, u_{it} is assumed normally distributed with zero mean, constant variance σ_u^2 for all i, $E(\varepsilon_{it}\varepsilon_{i't}) = 0$ $(i \neq i')$ and ρ constant for all i. In the equations for diarrhea and sickness, the first order autocorrelation coefficient is low--ρ is in the range of -.01 and +.03 respectively. Autocorrelation is apparent in the nutritional status equations but only when the equations are specified <u>without</u> a lagged endogenous variable. Inclusion of the latter in the specification virtually eliminates autocorrelation at a 90% confidence level.[2]

Finally, one would expect considerable nonlinearity in many of the relationships of the model. An important motivation for the use of an anthropometric standard rather than the absolute level of weight and height is to adjust for nonlinearity with age. Much of the remaining nonlinearity relates to changes in potential structural coefficients as a child progresses in age,

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[1]For a fuller discussion of the problems of estimating equations with dichotomous dependent variables-see J. Kmenta, <u>Elements of Econometrics</u>, pp. 462-463, and W. DuMouchel, <u>The Regression of A Dichotomous Variable</u> (The University of Michigan, Institute of Social Research, unpublished).

[2]Without the lagged endogenous variable, the estimate of ρ is approximately .32, .50, and .50 in the WTHT, WTAGE, HTAGE equations, respectively. Inclusion of the lagged endogenous variable lowers these estimates to -.017, .0008, and -.0018 respectively. Using the test suggested by Durbin (1970) for autocorrelation when some regressors are lagged dependent variables, one may reject the possibility of autocorrelation at a 90% confidence level.

and this is dealt with through multiplicative dummies. Since it is the first three years that are the most critical for a child's nutritional development and its risk of illness, the nonlinearities during this period are particularly important. The cut-off ages are 12, 24, 36 and occasionally 48 months. This suggests the possibility of some bias in our coefficients if the cut-off ages are incorrect.

Our methodology may be illustrated for the instruments of the included endogenous variable (PR.SEV.DIAR). Three variables were included in the specification with values equalling: (i) (PR.SEV.DIAR) with coefficient α_1, (ii) (PR.SEV.DIAR) if the child is aged 0 to 11 months, 0. otherwise, with coefficient α_2; (iii) (PR.SEV.DIAR) if the child is aged 12 to 23 months, 0. otherwise, with coefficient α_3. The coefficient for α_1 measures the marginal effect of an increase in the likelihood of severe diarrhea for a child aged 24 to 72 months. The marginal effect of an increase in the likelihood of severe diarrhea for a child aged 0 to 11 months is $(\alpha_1 + \alpha_2)$. The marginal effect for a child aged 12 to 23 months is $(\alpha_1 + \alpha_3)$. Although the estimated coefficients were α_1, α_2 and α_3, the coefficients α_1, $(\alpha_1 + \alpha_2)$ and $(\alpha_1 + \alpha_3)$ are presented in Tables 2-5 to facilitate interpretation of the results. In the tables, the t statistic displayed corresponds to the estimated coefficients α_1, α_2 and α_3, respectively, and for any given set of multiplicative dummies, the nonstarred term corresponds to variable (i) in the above example.

V. Model Results

In this section, Table 2 displays the results from an econometric estimation of the structural model of nutritional status of (6), (7) and (8). Since the latter specification includes a lagged dependent variable, it does not allow us to isolate those initial factors which channel a child toward a particular nutritional status. Table 3 displays estimates of the model where our sample is the first observation on each child, providing that the observation is within the age bracket of 1 to 7 months. Table 4 reports the results of estimating the reduced form equations of the nutritional status model. Table 5 presents the estimates of the model of health status, of the determinants of the age of weaning and of the level of food expenditure.

In order to assess the impact of policies on children identified as

Table 2

DETERMINANTS OF NUTRITIONAL STATUS: ENTIRE, STUNTED AND MALNOURISHED SAMPLES: STRUCTURAL EQUATIONS

	Total Sample						Stunted or Malnourished Group					
	Weight for Height		Height for Age		Weight for Age		Weight for Height[a]		Height for Age[b]		Weight for Age[c]	
Variables	Coeff	t	Coeff	t	Coeff	t	Coeff	t	Coeff	t	Coeff	t
Equation Number	1		2		3		4		5		6	
Endogenous												
Pr. Sickness	7.374	2.01	-4.821	-3.92	-2.739	-.92	9.054	.90	-7.175	-1.53	-15.085	-3.46
Pr. Sev. Diar. Episode, 0-11 mo.*	16.517	1.71			2.854	.67	29.769	1.38			8.047	.62
Pr. Sev. Diar. Episode, 12-23 mo.*	-13.680	.63			-15.378	-.31	-30.040	.35			-20.279	-.58
Pr. Sev. Diar. Episode, > 24 mo.	-25.310	-1.48			-10.634	-.75	-45.157	-1.03			-8.522	-.44
Pr. Mild Diar. Episode, 0-11 mo.*	-6.116	-.84			10.650	1.17	43.906	-.12			17.466	1.54
Pr. Mild Diar. Episode, 12-24 mo.*	40.897	1.53			10.949	1.34	7.060	.13			2.495	.84
Pr. Mild Diar. Episode, > 24 mo.	11.899	.75			-9.842	-.76	50.851	1.04			-21.366	-1.13
(Food exp.)(Pr. Diar.) 2-11 mo.[d]	.350	1.35	-.144	-1.71	-.123	-.58	-.860	-1.21	-.552	-1.40	-.900	-2.44
(Food exp.)(Pr. Diar.) 12-23 mo.[d]	.218	1.26	-.147	-2.45	-.161	-1.14	-.214	-.39	-1.083	-2.75	-.745	-2.71
(Food exp.)(Pr. Diar.) 24-48 mo.[d]	-.019	-.06	-.188	-1.81	-.348	-1.37	-.078	-.09	.847	-1.47	-1.287	-3.03
Exogenous												
Sev. Diar. Episode, 0-11 mo.*$_{t-1}$			-4.948	-4.65								
Sev. Diar. Episode, 12-24 mo.*$_{t-1}$			.388	1.21								
Sev. Diar. Episode, > 24 mo.$_{t-1}$			-.218	-.68					-1.053	-1.43		
Mild Diar. Episode, 0-11 mo.*$_{t-1}$			.748	1.24								
Mild Diar. Episode, 12-24 mo.*$_{t-1}$			.386	1.15								
Mild Diar. Episode, > 24 mo.$_{t-1}$			-.224	-.60					1.137	1.27		
Months nursed, children > 24 mo.	.054	1.41	.005	.42	.026	.84	.012	.13	.079	1.91	.047	1.07
Pct. of 1st yr. nursed	5.010	3.61	-.181	-.38	3.591	3.16	5.174	1.85	-.399	-.26	4.004	2.68
Pct. of 2nd yr. nursed	.262	.20	-1.266	-2.96	-3.132	-2.95	-2.538	-.82	-1.901	-1.14	-4.427	-2.99
Food exp/capita 2-11 mo.*	-.079	-.54	.038	.85	.019	.10	.037	1.30	.113	.86	.205	2.68
Food exp/capita 12-23 mo.*	-.034	.14	.045	1.33	.062	1.01	.048	1.36	.200	1.62	.251	3.49
Food exp/capita 24-48 mo.*	-.043	-.01	.055	1.90	.077	1.37	-.061	.79	.224	1.87	.218	3.26
Food exp/capita 48-72 mo.	-.042	-.85	.018	1.06	.013	.30	-.199	-1.27	.018	.21	-.018	-.28
Birth order	-.298	-2.56	-.052	-1.37	-.347	-3.61	-.349	-1.24	.188	1.38	-.328	-2.54
# children less than 6 yrs.	-.026	-.10	-.128	-1.52	-.294	-1.37	.082	.13	.083	.29	-.264	-.90
Age: 0-11 mo.*	34.383	.95	17.974	-2.65	28.581	-.05	29.408	-.35	22.520	-1.47	39.166	-1.06
Age: 12-23 mo.*	32.215	.24	19.420	-1.03	29.905	.48	39.233	.62	26.907	-.02	42.229	-.17
Age: 24-35 mo.*	33.345	.79	19.914	-.61	31.688	1.54	44.465	1.24	22.432	-1.56	46.582	1.26
Age: 36-48 mo.*	33.572	1.78	19.963	-.98	29.958	1.27	30.946	-.29	24.287	-1.74	42.780	-.10
Age: 48-72 mo.	31.498	5.76	20.344	11.29	28.708	10.71	31.900	2.06	26.985	4.01	42.908	10.84
Weanling observation	-1.837	-2.61	.262	1.12	-.421	-.73	.285	.17	1.195	1.42	.959	1.18
%Life in Promotora Prog., 0-11 mo.*	-.464	.34	.089	-3.42	-.343	-1.13	19.709	.25	.673	-1.86	.623	-1.43
%Life in Promotora Prog., 12-23 mo.*	-5.039	-2.16	2.217	2.22	.331	.10	1.150	-1.84	3.018	.55	2.074	.24
%Life in Promotora Prog., 24-36 mo.*	-.413	.11	1.286	.86	.111	-.03	2.476	-1.74	4.170	.85	-1.103	-1.00
%Life in Promotora Prog., 37-72 mo.	-.643	-.46	.699	1.60	.155	.13	19.360	2.15	1.716	1.07	1.499	.88
Educ. level-mother	.861	2.71	-.200	-1.89	.233	.90	.549	.70	-.723	-1.83	-.162	-.45
Educ. level-father	-.064	-.21	.117	1.14	.336	1.33	-.117	-.14	.133	.43	.777	2.31
Father's age	.097	2.84	-.014	-1.31	.032	1.17	.114	1.30	-.062	-1.47	.059	1.41
Mother's age	-.083	-1.62	.032	2.00	.027	.65	-.156	-1.22	.020	.35	.091	1.56
Modern Birth Control Use	.221	.51	.056	-.38	.321	.65	1.026	.95	-.370	-.69	-.656	-1.29
Sex (Boy=0, Girl=1), 0-11 mo.*	.189	-.43	.408	1.18	-2.241	-2.00	-3.860	-1.64	-.032	-.46	-3.480	-1.83
Sex (Boy=0, Girl=1), 12-23 mo.*	.543	-.17	-.336	-1.04	.313	.90	1.533	.56	-.251	-.80	-.512	.63
Sex (Boy=0, Girl=1), > 24 mo.	.688	1.29	-.033	-.19	-.341	-.77	.288	.19	.511	.87	-1.142	-1.84
Birth interval	.015	1.48	.976 - 3	.28	.014	1.68	.045	1.73	.024	2.01	.008	.66
Pct. Wt. for Height$_{t-1}$	.493	27.07	.071	11.73			.323	7.12	.100	6.26		
Pct. Wt. for Age$_{t-1}$					.660	49.39					.498	22.78
Pct. Ht. for Age$_{t-1}$	.144	3.60	.720	52.122			.145	1.63	.606	11.95		
R^2	.347		.617		.611		.247		.558		.438	
N	2194		2230		2153		547		332		1218	
Estimation Procedure	TSLS		TSLS		TSLS		TSLS		TSLS		TSLS	

* These variables were estimated through the use of multiplicative dummy terms. For example, for the variable "pr. severe diarrheal episode," three variables were included: (i) var A = pr. severe diarrheal episode; (ii) var B = var A, where age = 0-11 months, 0 otherwise; (iii) var C = var A, where age = 12-23 months, 0 otherwise. The marginal coefficient for (i) the pr. severe diarrheal episode for a child aged 24-72 months equals β_A the coefficient of variable A; (ii) the pr. severe diarrheal episode for a child aged 0-11 months equals $\beta_A + \beta_B$; and (iii) the pr. severe diarrheal episode for a child aged 12-23 months, equals $\beta_A + \beta_C$. In presenting the results in this and subsequent tables, we have already made the above additions for all multiplicative dummy expressions in order to facilitate the interpretation of the results. The "t" statistic displayed is that corresponding to β_A, β_B or β_C, etc. For any given set of multiplicative dummies, the nonstarred term is equivalent to variable A in the above example. Thus, to judge the significance of a starred coefficient value, one would have to examine its own "t" statistic and that for the nonstarred coefficient.

[a] This is the sample of children who have ever had a WTHT percentile position < 85%.

[b] This is the sample of children who have ever had a HTAGE percentile position < 85%.

[c] This is the sample of children who have ever had a WTAGE percentile position < 85%.

[d] This variable is the product of real weekly food expenditure and the estimated instrument of the probability of diarrhea for a child in age group k.

Table 3

DETERMINANTS OF NUTRITIONAL STATUS: FIRST OBSERVATION ON CHILDREN AGED 0-7 MONTHS

Variables	Weight for Height Coeff	t	Height for Age Coeff	t	Weight for Age Coeff	t
Equation Number	1		2		3	
Endogenous						
Pr. Sickness	36.37	.74	-6.78	-.35	64.33	1.14
Pr. Sev. Diar. Episode	36.91	.74	19.97	1.00	31.24	.55
Pr. Mild Diar. Episode	-41.72	-.81	11.74	.61	5.51	.09
Exogenous						
Pct. of life nursed	7.65	1.73	-.15	-.86	8.07	1.59
Food exp./capita	-.01	-.29	-.00	-.11	-.02	-.36
Sex	2.16	.93	.63	.69	6.39	2.38
Birth order	-.07	-.12	.08	.34	.41	.60
# children less than 6 years	-1.26	-1.25	-.21	-.52	-1.47	-1.26
Educ. level-mother	.58	.50	-.80	-1.70	-1.26	-.93
Educ. level-father	3.62	1.60	.54	.61	5.34	2.05
Father's age	.08	.34	.02	.26	.11	.39
Mother's age	-.11	-.61	.03	.07	-.07	-.33
Modern birth control use	-5.93	-.70	1.25	.93	-2.33	-.59
Birth interval	-.10	-1.40	.00	.16	-.10	-1.12
Constant	85.54	4.83	94.78	36.93	65.21	3.19
R^2	.04		.03		.05	
N	533		533		533	
Estimation Procedure	TSLS		TSLS		TSLS	

Table 4

DETERMINANTS OF NUTRITIONAL STATUS: ENTIRE, STUNTED & MALNOURISHED SAMPLES: REDUCED FORM EQUATIONS

Variables	Normal Group						Stunted or Malnourished Group					
	WTHT		HTAGE		WTAGE		WTHT [a]		HTAGE [b]		WTAGE [c]	
	Coeff	t	Coeff	t	Coeff	t	Coeff	t	Coeff	t	Coeff	t
Equation Number	1		2		3		4		5		6	
Months nursed, children>24 mo.	.034	.96	.004	.35	.034	1.18	-.037	-.42	.052	1.26	.040	1.00
Pct. of 1st yr. nursed	6.464	4.67	.776	1.59	7.180	6.46	8.484	3.00	1.031	.61	9.109	6.25
Pct. of 2nd yr. nursed	-.602	-.50	-1.532	-3.65	-3.597	-3.77	-4.646	-1.62	-2.792	-1.78	-4.291	-3.28
Food exp.per capita, 2-11 mo.*	.006	.51	.017	1.08	.049	1.56	-.032	1.35	.071	1.12	.098	3.05
Food exp.per capita, 12-24 mo.*	-.011	.23	-.008	-.23	-.026	-.17	-.058	1.21	-.019	.19	-.043	.95
Food exp.per capita, 24-36 mo.*	-.027	.11	.015	.87	.001	.53	-.078	1.12	.087	1.50	-.016	1.50
Food exp.per capita, 36-72 mo.	-.022	-.45	-.004	-.21	-.019	-.47	-.236	-1.61	-.036	-.41	-.099	-1.68
Sex (Boys=0, Girls=1), 0-11 mo.*	-1.633	-1.12	.165	1.60	-3.440	-1.92	1.075	-1.32	-.950	-.90	-1.644	-.95
Sex (Boys=0, Girls=1), 12-23 mo.*	-.424	.01	-.718	-.96	-1.250	.81	5.433	.68	-.562	-.74	.303	.97
Sex (Boys=0, Girls=1), >24 mo.	-.428	-.32	-.422	-.93	-1.783	-1.65	4.090	.67	.143	.08	-.555	-.35
Birth order	.200	-1.87	-.062	-1.63	-.288	-3.36	-.257	-1.06	.072	.50	-.318	-2.79
# children less than 6 years	.037	.16	-.046	-.55	-.055	-.30	.566	1.02	.038	.14	.243	.95
Age: 0-11 mo.*	38.682	3.29	17.299	-.07	35.972	4.78	25.133	1.13	21.551	.53	38.772	3.05
Age: 12-23 mo.*	32.308	.98	16.582	-.89	27.230	.80	26.241	.74	19.960	.04	30.281	.03
Age: 24-35 mo.*	31.452	.81	16.852	-.73	27.112	.93	32.691	1.50	14.360	-1.88	30.720	.23
Age: 36-48 mo.*	30.953	1.00	17.006	-.92	26.489	.97	15.397	-1.01	17.104	-1.76	29.972	-.20
Age: 48-72 mo.	29.858	5.55	17.361	9.18	25.638	9.59	18.438	1.09	19.838	3.11	30.205	7.91
Weanling observation	-1.331	-2.17	.367	1.70	-.296	-.60	.541	.39	.799	1.03	.836	1.25
%Life in Promotora Prog., 0-11 mo.*	-1.680	.67	-.146	-2.44	-1.237	-.79	18.696	.87	-.357	-1.08	.999	-.83
%Life in Promotora Prog., 12-23 mo.*	-4.737	-1.43	1.973	2.53	.005	.61	1.221	-1.70	1.178	.41	1.949	.23
%Life in Promotora Prog., 24-36 mo.*	-2.708	-.36	1.078	1.19	-.380	.35	-2.128	-2.03	3.982	1.33	1.387	-.03
%Life in Promotora Prog., 36-72 mo.	-2.010	-1.39	.276	.54	-.927	-.80	17.632	1.75	.210	.12	1.451	.89
%Life in Promotora Prog., girls	-1.246	.50	.531	.98	.602	1.24	13.345	-.67	.750	.26	.883	-.32
Educ. level-mother	.496	1.80	-.074	-.76	.303	1.37	-.183	-.30	-.327	-1.01	.293	.99
Educ. level-father	.315	1.13	-.054	-.55	.106	.47	.786	1.04	-.046	-.15	.328	1.12
Father's age	.061	2.14	-.007	-.67	.036	1.60	.111	1.56	-.030	-.80	.102	3.06
Mother's age	-.023	-.52	.020	1.30	.030	.86	-.066	-.61	-.014	.25	.046	.98
Modern birth control use	.212	.55	.101	.74	.399	1.30	.723	.80	.196	.41	.219	.52
Birth interval	.021	2.18	-.001	-.31	.017	2.26	.069	3.10	.015	1.23	.005	.49
Prenatal care to mother	.226	.01	-.361	-2.55	-.775	-2.41	.121	.12	-.866	-1.76	-.770	-1.78
DPT immuniz.	.366	.24	-.073	-.13	.185	.15	3.256	.78	-1.998	-.92	-.168	-.10
Polio immuniz.	-.411	-.25	-.464	-.79	-1.090	-.82	-5.318	-1.16	2.182	.88	-1.560	-.86
Income per capita	.003	.97	.003	2.79	.009	3.61	.016	1.57	-.004	-.46	.020	4.14
Birth weight,children < 12 mo.	-2.000	3.71	-.942	-6.24	-4.000	-10.13	-3.000	-2.81	-2.000	-2.82	-5.000	-9.12
Mild diar. episode, 0-11 mo.$_{t-1}$*	-1.882	-.69	.660	1.05	.521	.69						
Mild diar. episode, 12-23 mo.$_{t-1}$*	.261	.40	.416	1.10	.752	1.22	} -.796	-.47	} 1.312	1.46	} .672	.90
Mild diar. episode, > 24 mo.$_{t-1}$	-.342	-.33	-.162	-.44	-.708	-.84						
Sev. diar. episode, 0-11 mo.$_{t-1}$*	16.484	5.20	-4.563	-4.34	.905	.11						
Sev. diar. episode, 12-23 mo.$_{t-1}$*	1.549	.18	.302	.97	2.058	1.25	} 4.476	2.91	} .969	-1.32	} 1.223	1.65
Sev. diar. episode, >24 mo.$_{t-1}$	1.289	1.43	-.181	-.57	.645	.89						
Pct., weight for height$_{t-1}$	.510	29.07	.077	12.53			.350	8.46	.107	6.59	.567	27.324
Pct., weight for age,$_{t-1}$					.704	53.643						
Pct., height for age$_{t-1}$	.164	4.13	.746	53.46			.189	2.15	.689	15.11		
R^2	.35612		.62436		.63253						.47431	
N	2230		2230		2230						1261	
Estimation Procedure	OLS		OLS		OLS		OLS		OLS		OLS	

[a] This is the sample of children who have ever had a WTHT percentile position < 85%.

[b] This is the sample of children who have ever had a HTAGE percentile position < 87%.

[c] This is the sample of children who have ever had a WTAGE percentile position < 85%.

*See *footnote on Table 2.

Table 5

DETERMINANTS OF (i) EPISODES OF MILD AND SEVERE DIARRHEA & OF OTHER ILLNESSES AND OF (ii) PARENTAL FOOD DECISIONS

Variable	Mild Diarrheal Disease Coeff	β/S.E.	Severe Diarrheal Disease Coeff	β/S.E.	Days with illness Coeff	β/S.E.	Age weaned. Coeff	t	Food Expenditure[a/] / Income Coeff	t
Equation number	1		2		3		4			
Endogenous										
Pr. Sickness	-.263	-1.46	.189	1.46						
Change in percentile position, Weight for Height	.232	1.85	-.141	-1.09	.024	.28				
Exogenous										
Sex (Boys=0; Girls=1)	.040	.06	-.274	-.40	.246	.67	-.195	-.29		
Birth Order	.020	.38	-.093	-1.78	.026	.83	-.550	-2.51		
# children less than 6 years	.020	.24	-.170	-1.60	-.020	-.30	-1.406	-3.58	.101	9.26[a/]
Educ. level-mother	-.174	-1.31	.130	.96	-.153	-1.86	-.674	-1.38	-.011	-.80[a/]
Educ. level-father	.212	1.49	-.070	-.51	.079	.92	.331	.64	-.506	3.82[a/]
Father's Age	-.010	-.73	.015	1.19	.004	.53				
Mother's Age	.022	1.11	-.016	-.82	.008	.68	.389	4.64		
Modern birth control use	.176	.96	.097	.54	-.169	-1.53				
Birth interval	-.259	-.60	-.001	-.24	.001	.39	.092	2.77		
Weanling observation	.487	1.72	-.037	-.12	-.188	-.97				
%Life in Promotora Prog., 0-11 mo.*	.592	1.17	-.012	.06	-.068	.09	-9.651	-5.38		
%Life in Promotora Prog., 12-23 mo.*	.696	1.78	-.315	-.33	-.405	-.31				
%Life in Promotora Prog., > 24 mo.	-.881	-1.28	-.025	.04	.242	.64				
%Life in Promotora Prog., girls	-.693	.26	-.542	.72	.561	-.76				
DPT immuniz.	.705	1.03	.304	.43	-.187	-.42				
Polio immuniz.	-1.012	-1.29	-.636	-.79	.378	.79				
Income per capita	-.004	-1.81	-.001	-.21	.002	1.23	-.013	-2.92	-.504	-40.89[a/]
Prenatal care to mother	.180	.97	.020	.11	-.143	-1.23	.811	1.08		
Birth weight children < 12 mo.	.499	1.37	-.370	-.99	.077	.31				
$Stunted_{t-1}$	.287	1.20	.283	1.23	.227	1.53				
$Malnourished, 6-24\ mo._{t-1}$	.038	.08	.481	1.13	.122	.78				
$Malnourished_{t-1}$*	-.001	-.28	.01	-.04	.34	2.06				
Year of observation	.113	1.43	.088	1.11	.296	5.81				
Months nursed, children > 24 mo.	-.022	-1.10	.009	.54	.005	.47				
Pct. of 1st yr. nursed	-.568	-.83	.587	.81	.259	.54				
Pct. of 2nd yr. nursed	-1.128	-1.83	.389	.62	.050	.12				
Age: 0-11 mo.*	-12.42	-.69	-6.83	1.36	-23.78	.05				
Age: 12-23 mo.*	-10.79	.31	-8.49	1.33	-23.20	1.24				
Age: 24-35 mo.*	-10.41	1.53	-8.72	2.26	-23.63	.98				
Age: 36-48 mo.*	-11.23	-.36	-9.06	1.45	-23.46	1.79				
Age: 48-72 mo.	-11.06	-1.87	-9.62	-1.60	-23.85	-6.21				
R^2	.04		.037		.014		.20984		.31920	
Predictive power	.678260		.77343		.58750					
N	2166		2167		2201		354		3887	
Estimation Procedure	LOGIT		LOGIT		LOGIT		OLS		OLS	

[a/] for (food expenditure/income) equation, all variables are expressed in natural logarithms.

* See *footnote on Table 2

at risk of malnourishment, the model is also estimated for the restricted sample of children that have ever suffered any degree of malnourishment. For the WTHT, WTAGE and HTAGE equations, the samples include all observations on any child that has ever been less than (i) the 85th percentile in WTHT, (ii) the 85th percentile in WTAGE and (iii) the 87th percentile in HTAGE, respectively. The results are reported in Tables 2 and 4, and allow a comparison of the structural and reduced form coefficients for the stunted and acutely malnourished samples relative to the entire sample.

Interactions of Health and Nutritional Status and the Dynamics of Nutritional Change

Our results confirm that the timing and severity of a diarrheal episode largely determine the character of its nutritional impact. From Table 2, eqn. 2, the effect of severe diarrhea in the first year is particularly interesting. If it occurs early in that time period, it has a highly significant negative effect on a child's structural development in the latter half of the first year. The child loses almost five percentile points in its HTAGE position over a period of approximately 6 months.[1] This might explain the unusual positive coefficient for severe diarrhea in terms of WTHT position in the first year, since the child's weight may be less adversely affected than its height. This is supported by the negligible change in WTAGE position that is associated with severe diarrhea in the first year. With a slowing of growth, the WTHT position rises. On the other hand, a mild diarrhea episode is of far less concern. In fact, children with mild diarrhea have a slightly higher HTAGE position (by .75 percentile points) and a lower WTHT position.

After the first year, a severe diarrheal episode is highly deleterious for a child's WTHT percentile position, with a potential loss of 1 to 2 points. A mild diarrheal episode does not appear to lower a child's WTHT position significantly. Diarrhea is also of only minor consequence for a child's HTAGE position at this time (even if one considers the indirect effect on HTAGE caused by the effect of a severe diarrheal episode on WTHT (Table 4)). In summary, the results suggest that it is primarily a severe

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[1] From Table 3, eqn. 2, the contemporaneous effect of the severe diarrhea episode is not significant. In the HTAGE equation (Table 2, eqn. 2), we are capturing the effect of diarrhea in the previous period, and thus for infants, we are observing the effect on HTAGE in the latter half of a child's first year.

diarrheal episode which proves most damaging to a child's nutritional status.

Policies to reduce the likelihood of severe diarrhea would have a stronger impact on a child nutritionally at risk. Even more than with normal children, severe diarrhea after the first year will lower the acutely malnourished child's WTHT position by as much as 4.5 points (Table 2, eqn. 4). The effect of a past history of severe diarrhea is to reduce a stunted child's HTAGE position by 1 percentile point. As above, the effect of mild diarrheal episodes does not appear particularly adverse to a child's nutritional status.

The most damaging effect of nondiarrheal illnesses is with respect to a child's long-term structural development. A higher likelihood of sickness for a child leads to a significant decline in HTAGE position. The attendant rise in WTHT position may again suggest that illness operates to channel nutrients to the higher priority of maintaining body weight at the expense of normal structural development. Although WTHT position rises, WTAGE position falls, though less than HTAGE.

The effect of existing acute malnutrition or stunting on the likelihood of diarrhea and other illnesses is also consistent with our earlier hypotheses. A worsened nutritional status clearly raises the likelihood of illness (Table 5, eqn. 3). A low WTHT percentile position in the previous period raises the probability of a severe diarrhea episode for a child in the critical age of 6 to 24 months (Table 5, eqn. 2). Similarly, stunted children are also more susceptible to both mild and severe diarrheal episodes.

Equally interesting is that a positive (negative) change in percentile WTHT position lowers (raises) the probability of severe diarrhea but raises (lowers) that of mild diarrhea. Since it is severe diarrhea that is the principal contributor to a loss in WTHT, the self-reinforcing effect of lowering diarrhea and improved nutritional status clearly emerges from the results. Conversely, the emergence of an adverse nutritional status contributes to the likelihood of diarrhea. Low birthweight increases (reduces) the likelihood of severe (mild) diarrhea in the first year.

The results support our argument that poor health not only directly weakens a child's nutritional status, but also reduces the efficiency of nutrient use. This was tested through the nonlinear term $[FOOD*PR.DIAR]_{tk}$. The variable coefficient is negative for a child's long term development (HTAGE), suggesting that diarrhea reduces the coefficient of the food expenditure variable. In the WTHT equation, positive coefficients emerge in both

the first and second years. This suggests that a greater level of food expenditure at this time can weaken the adverse effect of diarrhea by channeling nutrients to maintain body weight, at the expense of long-term structural development. For children at risk of acute and chronic malnourishment, even this displacement is unsuccessful. The $(FOOD*PR.DIAR)_{tk}$ term now has a consistently negative coefficient for each group. The negative effect of diarrhea on the impact of food expenditure on nutritional status is dominant.

Dynamics of Nutritional Change: First, the results of Table 3 indicate that a large proportion of the variance among children in nutritional status in the first seven months is not easily explained by socioeconomic or health factors. Genetic differences clearly dominate. Second, it is not surprising that there is a positive correlation betwenn the WTHT and HTAGE positions of a child in the current and previous periods. For the entire sample, 49% and 72%, respectively, of the previous period's WTHT and HTAGE positions are carried over to the current period. For the acutely malnourished group, the coefficient on $WTHT_{t-1}$ is significantly lower than for the entire sample: .32 relative to .50 (Table 2, eqn. 4). An acutely malnourished child has far greater potential for variability in its WTHT position. Third, the results also indicate that a child's HTAGE is positively correlated with its WTHT in the previous period and vice versa. This is obvious but nonetheless important. Although a child's HTAGE percentile position is perhaps more critical in terms of its long-term development, nutritional programs may promote improvements in WTHT position for their dynamic impact on long-term nutritional status. For the stunted group, this effect is even higher, with a coefficient of .10 relative to .07 (Table 2, eqn. 2 and 5).

Some evidence also emerges on the hypothesis of a "natural" history of a child's nutritional and health status as discussed earlier. If we examine the age dummy terms in the structural equations, no systematic pattern emerges in terms of a child's WTHT or HTAGE position as it ages. From the statistical significance of the marginal dummy terms (up to age 48 months), there may be a slight increase in the WTHT intercept in the fourth year (from 31.5 to 33.5), a slight rise in HTAGE position over time (from 18.0 in the first year to 19.4 in the second and 20.3 thereafter) and a peaking in WTAGE in the third year. Yet the basic lack of change in the

structural coefficients is not too surprising, since by definition the standards are corrected for age.

How can these results be reconciled with the strong earlier evidence that mean nutritional status falls after the first 12 months?[1] Perhaps the answer may be found in the reduced form equation results, which capture the net effect of age on the nutritional indicators. From Table 4, a clear fall in WTHT and WTAGE occurs after 12 months with a slight decline thereafter; the HTAGE standard is invariant to age. In summary, the structural equation results suggest that once the standards are used to correct a child's size for age (WTHT is an age-independent measure), no further age pattern emerges.

The Impact of Policy Variables: Alternative Nutrient Sources, Medical Care and the Promotora Program

Nutrient Sources: The relative impact of nursing and food expenditure clearly changes in the early years of a child's development. In the first year, a child that is nursed over the entire year will emerge with a highly significant difference in percentile position in weight-for-height (WTHT) relative to a child receiving only food expenditure. For example, for every month nursed in the first year, a child gains more than .4 points on a percentile basis in WTHT (up to the 5.01 associated with nursing the whole period) (Table 2, eqn. 1) whereas food expenditure has a negative though insignificant impact during the first year. If a child were fully weaned and consumed the mean weekly food expenditure of 25 pesos for the sample, this would suggest a WTHT percentile position 1.97 points lower. The nursing effect is strongest in the first eight months (Table 3, eqn. 1) and by inference, must dampen in order to yield a coefficient of 5.01 for the entire first year. On the other hand, a child's height-for-age (HTAGE) percentile position is relatively insensitive to nursing or food intake during the first year, with coefficients that are actually negative though insignificant (Table 3, eqn. 2; Table 5, eqn. 2).

After the first year, there is a reversal which is equally significant. Though nursing becomes of minor importance for WTHT development, it has a slightly depressing effect on the child's long-term structural development (HTAGE), with a maximum loss of -1.26 percentile points for a child that has not been weaned by its second year. The effect is even more striking in terms of WTAGE (with a maximum potential loss of approximately -4.06 in

[1] Drake & Fajardo (1976)

the percentile position). For older children (over 2 years), nursing has a long-term legacy of .05 percentile points on WTHT for each month nursed.

The level of food expenditure proves not to be of critical importance for the rate of physical development, at least for the entire sample of children. Although the coefficient of the food variable on HTAGE is statistically significant, even a level of food expenditure one standard deviation above the average for the sample would yield no more than a 1.5 point percentile difference in HTAGE position. As one would expect, the weaning period has a negative impact, but as implied above, only on the WTHT percentile position.

For an acutely malnourished child, the pattern of optimal nutritional supplementation emerges quite clearly from the results of Tables 2 and 4. Nursing in the first year is at least as decisive as observed above, and the reduced form equations of Table 4 suggest that each additional month of nursing is worth .7 percentile points (as compared with .53 for the entire sample). After the second year, weaning of a malnourished child becomes more imperative than for the entire sample, as the coefficient on breastfeeding becomes sharply negative. The effect of food expenditure during this and subsequent periods is ambiguous in sign, but in any case not of large magnitude. The structural (reduced form) equations suggest a slight positive (negative) impact, but in either case a one standard deviation change in weekly food expenditure (15 pesos) has no more than a .75 point impact in percentile position, positive or negative.

For a stunted child, the effect of nursing is not statistically significant until the second year and then only adversely. The negative coefficient on HTAGE for this group is -1.90 relative to -1.27 for the entire sample (Table 2, eqns. 2,5). Food expenditure has a much more decisive relative effect for the stunted group. The structural equations (Table 2) suggest that in the second and third years, a one standard deviation increase in weekly food expenditure can raise the HTAGE position as much as 3 percentile points (relative to only approximately .45 for the entire sample). The reduced form coefficients suggest a potential net change half as large, but still this remains significant.

What is the effect of the nursing and weaning process on a child's health status? A child that is nursed in the second year has a significantly lower likelihood of mild diarrhea (Table 5, eqn. 1). This may simply

reflect that it puts off weaning and adverse exposure to food-borne illnesses.[1] The weaning period is clearly a period of increased sensitivity to diarrhea, though not to other illnesses.

Two additional points are worthy of note. The results do not imply that food expenditure has no influence on a child's absolute weight or height. For example, earlier results (not shown) suggest that food expenditure per capita in the first year has a small positive effect on weight and height. Nursing during the entire first year contributes half a centimeter to height. The results of Tables 2 and 3 are more relevant because they capture these changes relative to a norm.

Second, the advantage of differentiating between WTHT and HTAGE as opposed to relying solely on a HTAGE standard emerges from these results. Equation 3 of Table 2 suggests that nursing in the second year lowers WTAGE whereas the results of equations 1 and 2 (Table 2) indicate that this occurs only because it causes stunting, not because the child's WTHT position is adversely affected. For the same reason, the WTAGE variable obscures the contribution of food to height as opposed to weight.

Promotora Program: The results suggest that the principal impact of the Promotora program is on long-term nutrition status (HTAGE), with the strongest impact on children in their second year of life. The impact of a high fraction of a child's life in the Promotora program on its HTAGE position is only .09 points for a one-year old compared with 2.2, 1.24, and .7 for children in the next three years, respectively. The short-term effect on WTHT or WTAGE is generally insignificant, and when significant, clearly negative. Again this may reflect the program's impact on the child's height relative to its weight, thus leading to an increase in HTAGE and a corresponding decrease in WTHT position as a consequence. No significant change is observed for WTAGE in the sample considered.

For children at risk of malnourishment, the impact of the Promotora program remains ambiguous. From Table 4, our estimates would suggest an extremely substantial effect on an acturely malnourished child's WTHT but

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[1]This is also borne out by the difference in mean probability of diarrhea according to whether a child is nursed. In the first two years of life the probability of mild and severe diarrhea while nursed is .072 and .064, respectively; for a child that is weaned, these probabilities are .09 and .10, respectively. These are monthly probabilities of diarrhea.

only for children under age 1 or over age 3 (β = 18.7). The program's effect drops sharply (β = 1.2) in the second year and is actually negative during the third year. For the stunted group, the program has a clear impact, but only for children over age 2. This suggests that some acceleration of growth through health education is feasible but the explanation of the effect's specificity to these age groups is unclear.

In terms of health, participation in the Promotora program lowers the likelihood of diarrhea in the first and third years of life, but raises it during the second. It has little effect on the probability of other illnesses. It was also hypothesized that over time, general improvements in the sanitary situation may have occurred. This is possible, but it is not captured by a calendar year term, which is clearly positive for both kinds of diarrhea.

There are three possible explanations for the ambiguity of the results concerning the Promotora program's impact. First, we were forced to omit data on the first two years of the program because height data were not collected. It has been argued that much of the relevant change in nutritional status inspired by the program occurred during that period.[1] Second, our variables controlled for the percentage of a child's life spent in the program, not the amount of time the family was enrolled. The latter may have been the more relevant variable. Third, the Promotora program placed considerable stress on reaching out to families with nutritional and health problems and it is possible that a negative correlation between program participation and nutritional status may reflect this.

Medical Programs: Finally, our results indicate that exposure to and utilization of health services is of mixed value in reducing the risk of illness. Families that use modern contraceptive techniques or receive prenatal care have a slightly (though not significant) higher risk to diarrhea. While children with polio immunizations have a lower probability of both kinds of diarrhea, those that have received a DPT immunization are at higher risk. A child with both immunizations is at lower risk. The use of health services lowers the probability of other sicknesses (Table 5, eqn. 3). The receipt of prenatal care to the mother, and the use of modern birth control methods both reduce the chance of sickness. The policy implications of these

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[1] Drake and Fajardo (1976).

results should not be overstated, since our explanatory variables only weakly measure the quality and quantity of health services received by the family.

Economic Determinants of Nutritional and Health Status

Our model asserted that economic constraints and family decisions will influence a child's nutritional and health status. In addition to directly incorporating these factors into our structural equations (6) through (10), we have also examined their impact on two critical parental decisions: the age of weaning and the level of family food expenditure (Table 5, eqns. 4,5).

Parental Income:[1] Our model suggested that the level of family income influences a child's nutritional status, through its effect on nutrient intake, and health status through the purchase of goods and services. The significant relationship observed between food expenditure and income (Table 5, eqn. 5) is not surprising. As might be expected, food expenditure rises less rapidly than income, with an income elasticity of food expenditure: .496 (equalling $1 + \beta_{INCOME}$) (Table 5, eqn. 5). This suggests the substantial leakage that would arise in any income supplementation scheme in terms of its impact on family food expenditure, even before one considers its division within the family. Less obvious, but equally important, the time of weaning is accelerated as income rises. This may reflect the effect of a higher opportunity cost of the mother's time and that nursing may be an "inferior" good. Every additional hundred pesos of monthly income reduces the length of the nursing period by 1.3 months.

The net effect of income on nutritional status, as estimated from the reduced form equations, is positive (Table 4), but quantitatively small. For the entire sample, it leads to higher levels of HTAGE and WTAGE. Yet for the malnourished group, increased income has very little impact on children already at risk to stunting. It operates primarily to improve the WTHT and WTAGE position. Moreover, in both cases, the elasticity of income on nutritional status is small. The income elasticity of an acutely malnourished child's percentile WTHT position is only 1.4%.

Finally, by allowing greater parentally provided inputs, higher income significantly reduces the child's probability of mild diarrhea, with an

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[1] Mean real monthly per capita income is 125 pesos, with a standard deviation of 97.1 pesos.

elasticity of .467. On the other hand, it raises the probability of other illnesses, but at a lower level of significance (t = 1.23) and elasticity (.188).

Competition for Family Resources: The results indicate that child competition may have an adverse impact on nutritional status. First, nutrient intake per child falls. Although the level of family food expenditure rises with the number of young children, the elasticity is small: .10. Whether for physiological or economic reasons, the age of weaning is also accelerated by 1.4 months for each additional child under age 6 in the family.

Second, the structural equation results indicate that the greater the number of competing children under age 6, the lower the HTAGE percentile position (Table 2, eqns. 1 to 3), though it is not significant in the reduced form equation. Family planning programs aimed at wider spacing between children may have an impact on nutritional status. Short intervals between a child and an earlier sibling will lead to a lower WTHT and WTAGE position, though the maximum differential is only 1 point.[1] It will also lead to a longer nursing span.[2] Shorter intervals have a differentially worse impact on the nutritional status of both malnourished and stunted children. Each additional month's interval between a child and its predecessor raises the former's WTHT position by .07 points and HTAGE by .015 points. Both effects are larger than for the entire child population sample. Finally, in terms of health status, child competition and birth spacing both prove statistically insignificant in influencing the probability of illness. In fact, the results suggest that children with a large number of young siblings may have a lower probability of severe diarrhea.

Parental Discrimination Across Children: Our results are not conclusive on whether there is a bias against baby girls in nutritional status. Despite the fact that the anthropometric standards are sex-specific, girls are clearly lower in WTAGE (Table 4, eqn. 3; Table 2, eqn. 3) by as much as 3.44 points at the end of the first year of life, with the differential

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[1]This has been suggested in an earlier paper by Wray and Aguirre (1969).

[2]This may arise if there were a high correlation between the length of nursing of a given child and its predecessor.

narrowing thereafter to 1.2 - 1.7 points. The reduced form equations suggest that in the first year this reflects a slightly higher HTAGE position and a lower WTHT position; thereafter, possibly after weaning, their WTHT and HTAGE positions are lower though these results are not significant. In an earlier estimation, we indirectly tested the hypothesis of discrimination against girls in the allocation of a given level of family food expenditure, but could not find supporting evidence.[1,2]

Our hypothesis that higher birth order children are discriminated against receives stronger statistical confirmation. Both the structural and reduced form equations indicate that the highest birth order child in the sample--nine--will be approximately 3 percentile points worse off than the first baby in a family for WTHT and WTAGE and .5 points lower in HTAGE. Although it is possible this simply reflects the physiological consequences of being of higher birth order, it is interesting that this differential only emerges in the latter half of the first year. This lends support to the cause not being a weak new-born but rather intrafamilial discrimination. No differential is indicated in the results of Table 3. Later parity children will also receive one-half month less nursing than the preceding child (Table 5, eqn. 4). This is after correction for the effect of the age of mother on nursing.[3]

Parental discrimination does not appear to be present as a factor influencing the probability of illness. The sex of child is not a statistically significant factor in determining its health status. As for birth order, the results indicate that earlier children are more at risk, though this result is significant only for severe diarrhea episodes.

Parental Quality: The causal relation between parental quality and nutritional or health status is not altogether clear from the results.

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[1]In an earlier study using the Gomez standard for WTAGE, Drake and Fajardo (1969, p. 15) note a dramatic decrease in the female relative to male malnourishment rate.

[2]In equations (6) to (8) we tested whether a given level of family food expenditure per capita had a differentially greater impact on the nutritional status of boys relative to girls. No significant difference in the effect of food emerged.

[3]Surprisingly, the age of the mother is positively correlated with the period of nursing. This may reflect some collinearity between the educational level and age of mother in the sample.

First, more educated parents[1] do not spend more on food for their family, ceteris paribus. Not surprisingly, maternal education is negatively correlated with the length of nursing. Women with secondary education will nurse almost 3.5 months less than an uneducated woman.

Second, the results of the structural and the reduced form equations suggest that to the extent that more parental education has a statistically significant impact, it biases a child toward a high WTHT-low HTAGE position. Children of educated mothers and fathers appear to be of higher WTHT and lower HTAGE in their first seven months of life (Table 3, eqns. 1 and 2). Third, greater maternal education lowers the likelihood of mild diarrhea and of other illnesses but increases it for severe diarrhea (although the former effects are significant at a higher confidence level); paternal education operates in a completely opposite way. Fourth, children of older mothers and young fathers appear high in their HTAGE position, low in WTHT, have the highest probability of mild diarrhea but the lowest probability of severe diarrhea. This may reflect the optimal combination of maternal child-rearing experience and paternal energy for income-earning in plantation agriculture. In general, education and age together account for a maximum variation of 3.5 percentile points for any child. Since the causal mechanisms involved are not easily identified, the policy implications must be considered as vague. However, the results may serve as clinically useful rules of thumb for evaluating the risks of diarrheal disease or of malnourishment in maternal-child health programs.

VI. Conclusion

This paper has attempted to model the process of nutritional change and the development of risk of childhood morbidity. Since both processes are highly complex and the methodological problems of estimating such a model sufficiently great, the ambiguity of some of the results concerning the model's hypotheses is not surprising. However, the econometric model does provide some insights which are strong enough to warrant further policy analysis, and which we shall summarize in this section.

(i) The quality of nutrient intake in the first two years of life is

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[1] In our sample, mothers and fathers receive no more than 6 and 9 years of education, respectively.

pivotal. Insufficient nursing in the first year sharply lowers the child's contemporary WTHT position, with an equally serious dynamic impact on the child's HTAGE. In fact, the results of Table 3 suggest that nursing is one of the principal differentiating factors across children in their first seven months. These effects are even stronger for malnourished infants. The beneficial effects of nursing clearly taper off near the end of the first year and actually become deleterious to the child's HTAGE beyond this point. The average period of nursing is 9-11 months within the sample. This suggests that family characteristics which imply significantly lower or higher periods of nursing ought to call forth further maternal-child health education. Surprisingly, the level of food expenditure does not exert a significant impact on nutritional status, except in the sense that if a child is not weaned by the end of the first year, his nutritional status will fall.

(ii) Our model strongly affirms the importance of differentiating between a child's WTHT and HTAGE. As the results of Table 2 suggest, a WTAGE measure blurs the character of nutritional development, since it is the composite of the former two measures. Policies aimed at raising a child's HTAGE may not be the same as those designed to influence WTHT. In the past, economists have failed to make this differentiation in their analyses of the social costs of malnutrition. If these costs were to principally arise from a low position on only one of these measures (for example, WTHT), policies that focussed on raising the WTAGE measure would not necessarily be cost-effective.

(iii) Our results confirm the interdependence of poor nutritional status and poor health. Episodes of severe diarrhea clearly worsen a child's nutritional status, particularly in the first two years. Other kinds of illnesses contribute to the "stunting" of a child. For children who are either stunted or acutely malnourished, diarrhea sharply weakens whatever positive impact food expenditure has on nutritional status. Conversely, children who are malnourished are at greater risk to severe diarrhea and to other illnesses. Although our results do not yield any clear policy remedies for lowering the risk of severe diarrhea, they do suggest the importance of minimizing the adverse nutritional impact of such illnesses.

(iv) Despite the fact that our specification of the health status

model includes a substantial number of potentially relevant variables, our correlation coefficient is remarkably low (r^2 = .02 - .03). In particular, our understanding of the factors that contribute to a diarrhea episode's occurrence remain limited. This may arise for several reasons. Since our data only captures episodes that occurred in the previous month, and recall error for another person is also likely, it is possible that much of the diarrhea experienced by the population is missed, and this adds to the potential error involved. The unavailability of environmental quality measures also must contribute to the low explanatory power. Another possible explanation is that the observed diarrheal rate in Candelaria is relatively low--14%--compared to many other Latin American communities of comparable socioeconomic status, perhaps due to the environmental improvements that preceded the onset of the Promotora program. Perhaps the range of experience captured by both our dependent and explanatory variables is too limited to fully measure the impact of the latter. Though some of the residual cases are still explained by socioeconomic factors, the majority of episodes are random occurrences that are normal in any child's development. Thus, our results may not be extremely useful for measuring the impact of policies for communities where diarrhea is more endemic.

(v) Nevertheless, one of the more interesting results of the health status model is that many of the factors which increase the likelihood of <u>mild</u> diarrhea are not the ones which increase the likelihood of <u>severe</u> diarrhea (Table 5, eqns. 1,2).

(vi) The model does provide support for viewing malnutrition and poor health as the consequence of family decision processes. The level and quality of nutrient intake are clearly affected by parental income and education. The results support the hypothesis that both child competition and parental discrimination adversely influence nutritional status. This suggests that an additional benefit from family planning programs would be an improvement in the nutritional status of children.

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