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MALNUTRITION, CHILD MORBIDITY AND THE FAMILY DECISION PROCESS

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This paper suggests a microeconomic model of the process by which infants and toddlers are subject to malnourishment, diarrhea and other illnesses in developing countries. It is econometrically estimated on a cross-section, time-series basis for 1200 children from Candelaria, Colombia. The model focuses on four issues: (i) the impact of economic constraints and intra-family resource allocation decisions on a child's nutritional and health status, (ii) the interrelationship between malnutrition, diarrhea and other diseases, (iii) the impact on health and nutritional status of specific policy interventions (maternal-child health education, food supplementation and the encouragement of breast feeding), and (iv) the importance of distinguishing between the effect of different policy variables on a child's height and weight during this period.

1. Introduction

In many developing countries, childhood malnourishment and morbidity are among the most serious burdens of underdevelopment. As such, economists have begun to evaluate the cost-effectiveness of specific nutritional supplementation and health programs.[1] Yet the formulation of such programs and the evaluation of their impact remains seriously limited. 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 measure empirically 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 participating over a 7-year period in the *Promotora* maternal-child health program in Candelaria, Colombia.[2] Three

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[1]Selowsky and Reutlinger (1976).

[2]*Promotora* is the Spanish term for the nurse-volunteers who worked in the program.

sets of issues are of central concern:

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

(2) Does a deterioration of a child's nutritional status increase its risk of illness or morbidity? Conversely, do diarrhea or other illnesses seriously weaken a child's nutritional status?

(3) What effect would such policy interventions as maternal-child health education, food supplementation or promotion of breast feeding have on a child's health or nutritional status?

Section 2 contains a detailed discussion of the model and its specification for economic analysis. Section 3 provides a description of the Promotora program and discusses the data used in the analysis. Section 4 discusses the econometric issues that arise in estimating the model, and section 5 examines the results and implications of the estimated model.

2. The model

Our model assumes that a child's nutritional and health status reflects the combined impact of basic physiological development processes, genetic factors 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. Our model is highly abstract and greatly simplifies extremely complex physiological, epidemiological and nutritional processes. Any one of the relationships implicit in the model has been the subject of substantial clinical research, and it would be impossible either to survey this research in this paper or to incorporate adequately the results of such research in our model. However, we believe that the structure of our model is not inconsistent with the basic medical literature and that the costs of simplification are outweighed by the gains from examining a more complete set of the factors influencing a child's development.[4]

The essence of the model is summarized in eqs. (1) and (2) below. Assume

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

[4]For a significant introduction to these issues, the reader is referred to Robson (1972), Scrimshaw et al. (1968), Morley (1973), Martorell (1975, 1976), and Cravioto (1976).

that for any child i in family j at time t, a physiological production function (1) describes the transformation of food inputs into the child's nutritional status, N_{ijt}, where

$$N_{ijt}=f(N_{ijt-1}, B_{ijt}, F_{ijt}, H_{ijt}), \tag{1}$$

and where B_{ijt} measures the nutrients provided through breast feeding, H_{ijt} measures the child's health status, and F_{ijt} is a vector of the quantity of other nutrient inputs. The realization of a child's natural propensity for physical growth in each phase of its development is a function of its past nutritional status (or birthweight, N_{ij0}, for a child in the neonatal period),[5] the quantity and quality of its nutrient intake, its ability to use these resources efficiently and the level of its current bodily demand for nutrients. For an infant or toddler, the latter two factors are critically influenced by a child's health status; for example, diarrhea may limit bodily absorption of nutrients, while parasites may be an additional source of demand for nutrients.[6]

Particularly in developing countries, a child's health status is the joint outcome of several factors; (i) the medical risks inherent in the quality of its external environment E_{ij}; (ii) the extent to which its parents are able to insulate a child from these risks by providing non-food inputs (clothing, medical care, shelter, parental attention, etc.), M_{ijt}, high quality parental care, L_{ijt}, and adequate nutrient intake; and (iii) its own physical ability to overcome the threat of illness without significant pain or disability. A child that is well-nourished, has not experienced any sudden adverse nutritional change, and one that was healthy in the past will have greater resistance to illness. These factors are reflected in structural equation (2):

$$H_{ijt}=g(N_{ijt}, M_{ijt}, H_{ijt-1}, \Delta N_{ijt}, E_{jt}, L_{ijt}). \tag{2}$$

In no instance can these inputs fully insulate a child from illness. In fact, it is through exposure to disease agents and illness that a child acquires a degree of subsequent natural immunity.[7] Our model only allows us to explain why some children may be excessively at risk to morbidity.

[5]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. The inclusion of N_{ijt-1} also takes account of genetic factors.

[6]Similarly, 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. See Scrimshaw et al. (1968).

[7]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 malaria prophylaxis).

In the context of economic models of household behavior,[8] one can imagine that a family decision process determines the allocation of family resources to a particular child in the form of B_{ijt}, F_{ijt}, M_{ijt} and perhaps E_{jt} (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. In maximizing utility, parental choices are subject to constraints which are both internal and external to the family. Their command over resources is limited by their wealth, the market wage rate and commodity prices. Parental age and knowledge of child-rearing will influence their efficiency in achieving a given level of child quality. Parental decisions may also be influenced by government policies.[9] Such constraints inevitably force the parents to make interpersonal judgments between themselves and their children. This may even involve conscious discrimination (e.g., for working members of the household, against girls, or against higher parity children).[10,11]

Such models suggest that parents develop decision rules concerning the allocation of parental time and family income between activities (labor, leisure and child care) and commodities, and their allocation among members of the family. In principle, a subset of these decision rules could be specified as reduced form equations for F_{ijt}, B_{ijt}, and M_{ijt}, where a child's consumption of goods and parental time is a function of price, income, parental preferences as between children, parental quality in child-rearing and the family's size and age-structure. Yet estimation of reduced form equations is not feasible, as it would require detailed data on the specific level and composition of consumption of individual children. Lacking such data, we directly estimate structural eqs. (1) and (2), using a set of instruments to proxy the factors influencing the unknown F_{ijt}, B_{ijt}, and M_{ijt} vectors. The specification of these structural equations is described below.

2.1. *Nutritional status model*

Our choice of nutritional status indicators, N_{ijt}, reflects the apparent consensus in the nutrition literature that one should differentiate the process of nutritional change into its acute and chronic manifestations. Two complementary anthropometric measures are used: a child's weight relative to its

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

[9]For example, programs in nutritional supplementation and maternal–child health education may influence parental decisions; government grain pricing policies may affect both income and the cost of food.

[10]Most of the literature does not explicitly examine discrimination between family members. An exception to this is reflected in papers by Welch (1974) and Heller (1976). In the latter, it is argued that the parents' utility function includes the number of surviving children as an argument and that the probability of survival is itself parentally influenced, if not determined.

[11]Unintended 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).

height (*WTHT*) and its height relative to its age (*HTAGE*).[12] Specifically, a child's weight, relative to the median weight for a sample of children of the same height and sex (*WTHT*), measures the degree of *acute* malnourishment associated with recent weight loss and insufficient nutrient intake and/or bodily absorption. 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 development due to *chronic* nutrient deficiency. We also include the weight for age (*WTAGE*) standard in order to facilitate comparison with other studies.[13]

The economic consequences of a low score on either of these measures are not well-established in the literature. Acute malnourishment is said to place the child at higher risk of morbidity. Stunting is clearly associated with slower cellular growth in the brain, although there is no conclusive evidence about the effect of this on mental capacity or development. Some argue that the intellectually damaging consequences of malnourishment primarily arise from the attendant apathy and listlessness that preclude a normal social and intellectual responsiveness to the environment.[14] This phenomenon may be as much the consequence of a low *WTHT* position. It is an area where further research is clearly needed.

Three principal nutrition equations are specified as follows:

$$\begin{aligned} WTHT_{tk} = N_1[&FOOD_{tk},\ NURS_{tk},\ WEANL_t,\ PR.SEVERE.DIAR._{tk},\\ &PR.MILD.DIAR._{tk},\ PR.SICKNESS_t,\\ &[FOOD{*}PR.DIAR]_{tk},\ SEX_k,\ BRTHORD,\ (\#CH<6),\\ &INTVL,\ EDMO,\ EDFA,\ AGEFA_t,\ AGEMO_t,\\ &AGEDUM_{tk},\ PROMO_{tk},\ BRTHCON,\ WTHT_{t-1},\\ &HTAGE_{t-1},\ \varepsilon_1], \end{aligned} \tag{3}$$

$$\begin{aligned} HTAGE_{tk} = N_2[&FOOD_{tk},\ NURS_{tk},\ WEANL_t,\ SEVERE.DIAR._{t-1},\\ &MILD.DIAR_{t-1},\ PR.SICKNESS_t,\\ &[FOOD{*}PR.DIAR]_{tk},\ SEX_k,\ BRTHORD,\ (\#CH<6),\\ &INTVL,\ EDMO,\ EDFA,\ AGEFA_t,\ AGEMO_t,\\ &AGEDUM_{tk},\ PROMO_{tk},\ BRTHCON,\ WTHT_{t-1},\\ &HTAGE_{t-1},\ \varepsilon_2], \end{aligned} \tag{4}$$

[12]These measures are preferred to simply using a child's weight-for-age (*WTAGE*) (i.e., its weight compared to the median weight of a large sample of children of the same age). The *WTAGE* measure is highly sensitive to family uncertainty as to a child's precise age in months and may overestimate the extent of the malnutrition problem [see Heller and Drake (1976), Waterlow (1973, 1974), and Habicht (1974)]. Other standards, for measuring nutritional status are available, including an arm circumference measure, 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.

[13]In both cases, the median score is 100. For the source of these standards and for a summary of the techniques used to develop them, see National Center for Health Statistics (1976).

[14]NAS (1973), Cravioto (1973), Cravioto in Scrimshaw and Behar (1976), and Garn (1975).

$$WTAGE_{tk} = N_3[FOOD_{tk},\ NURS_{tk},\ WEANL_t,\ PR.SEVERE.DIAR_{tk},\ PR.MILD._{tk},\ PR.SICKNESS_t,\ [FOOD*PR.DIAR]_{tk},\ SEX_k,\ BRTHORD,\ (\#CH<6),\ INTVL,\ EDMO,\ EDFA,\ AGEFA_t,\ AGEMO_t,\ AGEDUM_{tk},\ PROMO_{tk},\ BRTHCON,\ WTAGE_{t-1},\ \varepsilon_3], \quad (5)$$

where k refers to an observation on a child when it is in the kth age group. Table 1 displays the definitions of the variables used in the empirical analysis as well as their mean and standard deviation (where applicable). The variables relate specific policy issues and hypotheses about the process of nutritional change:

(1) What is the impact of varying the level and composition of nutrient intake on nutritional status at different ages? Lacking measures of the actual nutrient input to a child at a particular point in time, we must rely on the level of real family food expenditure per capita (*FOOD*)[15] and on whether the child was nursing at the time of the observation. Multiplicative dummy terms are used to differentiate the impact of food expenditure by age group (2–11, 12–23, 24–47, and over 47 months).[16] 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.[17]

Similarly, four variables have been developed to capture the effects of nursing and weaning; the percentage of a child's life nursed, for children in the first and second years of life (*PCT. of 1st yr. NURSED, PCT. of 2nd yr. NURSED*), respectively; the absolute number of months nursed for children over 24 months of age, and an additive dummy term for observations occuring during the weaning period.[18] The first two attempt to capture any

[15]It would have been preferable to adjust the size of household by the norm nutritional demands of its members.

[16]In section 4, we have described the method used in specifying the multiplicative dummy terms.

[17]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 were 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.

[18]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, those who have not been nursed during their participation in the program, and those for whom the timing of the termination of breastfeeding cannot 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 birth is assumed as the age weaned; for the former, group, complete weaning has not yet occurred.

Table 1
List of variables with means and standard deviation in parentheses.[a]

$WTHT_t$	=(100)×(ratio of a child's weight to median weight of children of same height and sex, using NAS standard) (100.27, 10.59).
$WTAGE_t$	=(100)×(ratio of a child's weight to median weight of children of same age and sex, using NAS standard) (91.49, 11.64).
$HTAGE_t$	=(100)×(ratio of a child's height to median height of children of same age and sex, using NAS standard) (95.09, 4.93).
$\Delta WTHT_t$	$=WTHT_t - WTHT_{t-1}$.
$BTHWGHT_0$	=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).
$STUNTED_{t-1}$	=1 if child's *HTAGE* score is less than 90 in period $t-1$,0 otherwise.
$MALNOUR_{t-1,k}$	=1 if a child in the kth age group has a *WTHT* score less than 90 in period $t-1$, 0 otherwise.
$FOOD_{tk}$	=Weekly real food expenditure per capita within the household in period t in 1968 pesos (253.5, 156.3).
WEANL	=1 if observation is during or directly after weaning (the latter, within 3 months) (0.23, 0.42).
$NURS_{tk}$	=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 ≧24 months).
PCT. OF 1ST YR. NURSED	=For children <12 months, fraction of its age it has been nursed; for all others, 0 (0.80, 0.29 on sample of children aged 0–11 months).
PCT. OF 2ND YR. NURSED	=For children with 12 months ≦age<24 months, fraction of its age it has been nursed; for all others, 0 (0.49, 0.30 on children aged 12–23 months).
AGE WEANED	=Number of months before child was weaned (10.71, 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 previous child, $INTVL=72$ (46.4, 22.31).
$\#CH<6$	=Total number of children in the household of age less than 72 months (2.33, 0.96).
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, 0.76).
EDMO	=Educational level of the mother (as above) (2.79, 0.73).
AGFA	=Age of father in years (35.52, 8.47).
AGMO	=Age of mother in years (29.06, 6.50).
BRTHCON	=1 if mother used the pill or IUD for birth control; 0 otherwise (0.38, 0.48).

Table 1 (*continued*)
List of variables with means and standard deviation in parentheses.[a]

$PROMO_{tk}$	= Percentage of child's life it was in the Promotora program, at time t for child of age k (0.84, 0.24).
DPT	= 1 if child ever received a DPT immunization; 0 otherwise (0.14, 0.19).
POLIO	= 1 if child ever received a polio immunization; 0 otherwise (0.10, 0.17).
$INCOME_t$	= Monthly family income in 1000 real 1968 pesos (1253.1, 950.7).
$YEAR_t$	= Calendar year associated with observation in period t.
PRENAT	= 1 if mother ever received prenatal care; 0 otherwise (0.31, 0.46).
$SICKNESS_t$	= 1 if child was ill since previous visit by promotora: 0 otherwise (0.24, 0.43).
PR. SICKNESS	= Estimated probability that child was ill since previous visit by promotora (0.25, 0.08).
$DAYS\ SICK_t$	= 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 (0.08, 0.27).
$PR.\ MILD\ DIAR_{tk}$	= Estimated probability that child had a mild diarrhea episode during month preceding visit in time t (0.07, 0.04) 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 (0.08, 0.27).
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 (0.07, 0.03).
$[(FOOD)\ (PR.\ DIAR)]_{tk}$	= 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.

[a]We have omitted the ith term for child and jth term for family where it is clear. The t subscript refers to the current observation on any child. The spacing between t and preceding and subsequent observations is child-specific.

non-linearity over time in the impact of nursing.[19] The third evaluates whether the length of time a child is nursed has any residual impact on the child's nutritional status after it has been weaned. The fourth attempts to isolate any heightened risk to nutritional status during the weaning period.

(2) If a model of nutritional status did not take account of a child's health status, the coefficients of nutrient variables would be seriously biased. The impact of nutrients is contingent on the efficiency of nutrient utilization, and this will be affected by a child's health. Since the number and timing of our observations on any particular child varies considerably, it is impossible to reconstruct a child's medical history. This is exacerbated by the fact that our survey indicates only the duration of a diarrheal episode in the month preceding the promotora visit. The lack of refinement in these morbidity measures obviously weakens the explanatory power of the model. Even these variables are themselves subject to significant recall inaccuracy. Recent studies suggest that parental recall of diarrheal episodes more than 15 days past is subject to considerable error.[20]

For our model, we have identified three illness variables: whether or not the child had (i) a mild diarrheal episode, lasting one to four days during the previous month ($MILD.DIAR_{tk}$), (ii) a severe diarrheal episode lasting more than 4 days ($SEVERE.DIAR._{tk}$), and (iii) any other illness since the previous visit of the promotora ($SICKNESS_t$).[21,22] As a consequence of the econometric estimation procedure used, instruments for these three variables are created that indicate the *probability* of a mild diarrhea episode (*PR.MILD.DIAR.*), the probability of a severe diarrhea episode (*PR.SEVERE.DIAR.*), and the probability of another illness episode (*PR.SICKNESS*) during the aforementioned time periods. The distinction between a mild and severe diarrheal episode reflects the bimodal distribution of the data, our own doubts that a finer distinction can be reliably drawn from the data, and the likely correspondence between these measures and toxic and infectious diarrhea, respectively.[23] Severe episodes should have a

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

[20]In fact, studies have shown that recall accuracy declines significantly after one week [Woodward (1973)].

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

[22]The probability of sickness for a child in Candelaria is high. The probability of a mild and severe diarrhea episode is approximately 0.075 and 0.08 respectively, during any month in the first year of a child's life and 0.095 and 0.097, respectively, during any month in the second year. The probability of other kinds of illness (principally respiratory) is 0.24.

[23]The distribution of diarrhea in the previous month, by days of duration, is as follows: 0 days – 85.1%, 1 day – 0.7%, 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%.

more deleterious effect on nutritional status, particularly *WTHT*. However, it should be noted that even if a child registers one of these disease episodes, it may be consistent with alternative medical problems of varying degrees of severity, each with a different impact on nutritional status. Thus, the statistical impact of any illness variable is likely to reflect the composite effect of the underlying disease problems.

It is important to specify accurately the dynamics of the impact of any illness episode. Whereas diarrhea or illness may have an immediate effect on a child's weight, and thus its *WTHT* or *WTAGE* score, it is likely to influence a child's height only over the medium term. In our *HTAGE* model, we test for the impact of a severe or mild level of diarrhea in the month preceding the $t-1$st observation. As above, multiplicative dummy variables are used to measure the differential impact of diarrhea across age groups.

To measure the impact of diarrhea on the body's food utilization efficiency, we introduce a nonlinear variable, $[(FOOD)\ (PR.DIAR.)]_{tk}$ where $[PR.DIAR._{tk}]$ 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 implicit in the product of these two variables. The higher the likelihood of diarrhea, the weaker will be the impact of any dollar of food expenditure, thus yielding a negative coefficient for this variable. Yet in the context of a given probability of diarrhea, the greater the food expenditure, the smaller the negative nutritional impact of the diarrhea. The sign and significance of the coefficient suggests which of these two relationships is the dominant one, though both may be present and significant.

(3) It is possible that parents 'discriminate' between children in their allocation of resources and time (e.g., against girls or higher birth order children). The model tests the hypothesis that the sex (*SEX*) or birth order (*BRTHORD*) of a child has an influence on its nutritional status.[24]

(4) Parental ability 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 (*INTVL*) may be deprived of their proportionate share of family resources. A child in a family with a large number of young siblings under 6 ($\#CH<6$) may also obtain fewer resources. Both factors should be reflected in a lowered nutritional status.

(5) The nutritional impact of a given level of food expenditure is not independent of a parent's knowledge of personal and food hygiene. Since parental human capital accumulates either through experience or through

[24]The NAS standards are ostensibly sex-specific. Although there may be some tendency for birth order to influence a child's neonatal nutritional status, one would not expect such differences to be compelling beyond the first year of life.

education, we include measures of the formal educational level of mothers and fathers (*EDMO*, *EDFA*) and of the age of the mother and father (*AGEFA*, *AGEMO*). One might hypothesize some ambiguity in the impact of these variables. Age is not only correlated with experience but with diminished physical energy, possible fatalism and a lower potential rate of return on investments in child quality. Children of older mothers are likely to be of lower birthweight. Higher education may promote values detrimental to child health (e.g., early weaning and improper use of bottled milk).[25]

(6) Though our nutritional measures either correct for age or are age independent, additive age dummies ($AGEDUM_{tk}$) for each of the first four years of life were included to measure any trends in nutritional scores over a child's early years.[26] 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.[27]

(7) To assess the Promotora program's impact, we have developed a measure of the degree of program participation. Since a child could have entered the program at any point in its life,[28] the fraction of its life in the 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.[29] To capture both effects, we create multiplicative dummy terms measuring 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 ($PROMO_{tk}$). Since the Promotora program also disseminated family planning information, we hypothesize that parental use of modern birth control methods (*BRTHCON*) proxies the degree and enthusiasm of their participation in the program.

(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. Similarly, genetic factors, as proxied by parental height or maternal stature, are likely to have significant explanatory power, particularly in the *HTAGE* equation.[30] Unfortunately, our study lacked parental physical attribute measures. Lacking these, the lagged dependent variable

[25]See Wray and Aguirre (1969).

[26]Such dummies take on the value 1 if a child is within a given age bracket, zero otherwise.

[27]Drake and Fajardo (1976).

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

[29]The 'number of months' in the program is collinear with age.

[30]Tanner has documented that the significance attributed to anthropometric measures may be modified if appropriate corrections for parental height are introduced. Tanner (1966).

had to serve as a correlated measure of genetic background. Since the timing of an observation was determined by when the promotora visited the child's family, it is necessary to ensure 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.[31]

2.2. Health status model

Although it would be useful to specify (2) by examining the risks of illness from the separate disease agents to which a child is commonly exposed, we have noted that our data is limited to information on the duration of diarrhea episodes and of the occurrence of other kinds of illness since the previous visit by the promotora. Both measures of illness carry a heavy subjective element, depending upon the accuracy of parental recall and differing perceptions of 'abnormality' in healthy status. Medical problems without obvious or unusual symptoms may pass unnoticed by the parent. Given the questionable accuracy of the data, we limited ourselves to three dichotomous dependent variables: $MILD.DIAR_{tk}$, $SEVERE.DIAR_{tk}$, and $SICKNESS_t$ as described above and in table 1.

Despite its frequency, the etiology of diarrhea disease remains unclear. Morley comments that 'the diarrheal stool has been a happy but rather barren hunting ground for microbiologists for many years'.[32] 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 are presumed to be most susceptible during weaning. Their exposure is greatest (as they use their mouth to identify objects and because of their newly found mobility). At that time, their level of immunity to new pathogenic agents is lowest and their vulnerability to substitution of an inadequate and imbalanced diet greatest. Potential exposure to feces-borne disease agents is most serious in environments with inadequate waste disposal methods and/or low volumes of water usage, since this influences both the presence and density of disease agents. Malnourished children are most at risk since there is evidence that their cellular immunity is lower because of the impact of malnourishment on the thymus gland.[33]

The specification of the structural equations to explain the occurrence of a mild or severe diarrhea episode in (6) below reflects both these con-

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

[32]Morley (1973).

[33]Ibid.

siderations and some of our earlier hypotheses concerning the impact of economic and demographic factors on health status. Our specification for the occurrence of other illnesses in (7) is assumed identical to (6),[34]

$$\left.\begin{matrix} MILD.DIAR_t \\ SEVERE.DIAR_t \end{matrix}\right\} = [MALNOUR_{t-1},\ STUNTED_{t-1},\ BTHWGHT,\ \Delta WTHT_{t,t-1},\ INCOME_t,\ EDMO,\ EDFA,\ AGEFA,\ AGEMO,\ SEX,\ BRTHORD,\ (\#CH<6),\ INTVL,\ NURS_{tk},\ WEANL_t,\ DPT,\ POLIO,\ PRENAT,\ BRTHCON,\ PR.SICKNESS,\ AGEDUM_{tk},\ PROMO_{tk},\ YEAR,\ \varepsilon_4], \quad (6)$$

$$SICKNESS_t = [MALNOUR_{t-1,k},\ STUNTED_{t-1},\ BTHWGHT,\ \Delta WTHT_{t,t-1},\ INCOME_t,\ EDMO,\ EDFA,\ AGEMO,\ AGEFA,\ SEX,\ BRTHORD,\ (\#CH<6),\ INTVL,\ NURS_{tk},\ WEANL_t,\ DPT,\ POLIO,\ PRENAT,\ BRTHCON,\ AGEDUM_{tk},\ PROMO_{tk},\ YEAR,\ \varepsilon_5]. \quad (7)$$

(1) Poor nutritional status may heighten the risk of illness. Two dummy variables are included to distinguish children with *WTHT* or *HTAGE* scores lower than 90 in the previous period ($MALNOUR_{t-1,k}$, $STUNTED_{t-1}$). For infants, birthweight (*BTHWGHT*) is probably the best indicator of initial nutritional status. After the first year, the effect of birthweight declines relative to current nutritional status.

(2) *Changes* in weight or height are often argued to be useful clinical indicators of a child's risk of morbidity. A child with a high *WTHT* or *HTAGE* score 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.[35] Since it is a sudden weight change that is the primary source of concern, only a change in *WTHT* percentile position has been included. In the structural equation system, $\Delta WTHT_{t,t-1}$ is endogenous.

(3) The ability of parents to provide inputs M_{ijt} to insulate a child from the risks of morbidity is measured by real family income per capita ($INCOME_t$). Differences in parental child-rearing efficiency are proxied by the educational level and age of the parents.

(4) Parental priorities in allocating family resources are 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.

[34]The specifications for the mild and severe diarrhea equations are identical.

[35]Morley (1973).

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

(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 (*DPT, POLIO*); (ii) whether a mother received any prenatal care (*PRENAT*); and (iii) whether she uses a modern method of birth control (*BRTHCON*).

(7) Since the onset of diarrhea may be induced by other illnesses, the specifications of the diarrheal equations include an estimate of the probability of sickness for the child.

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

(9) The impact of the Promotora program on child health is tested using 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 change over time in the quality of the environment arising from program activities or other factors.

2.3. Parental decisions on nutrient intake

We also use the framework of parental choice to explain: (i) the age at which the child is weaned from breast feeding, and (ii) the level of food expenditure per capita in the household. The length of time a child is breast fed reflects both cultural and economic considerations. In many developing countries, modernization is accompanied by reduced reliance on breast feeding. The higher the opportunity cost of the mother's time, the greater the effect since it raises the price of breast feeding relative to bottle-feeding. The higher the income and education levels of a family, ceteris paribus, the earlier the age of weaning. Since the Promotora programs stressed breast feeding, one would expect a positive correlation between program participation and age at weaning. The mother's physiological capacity to breast feed would influence the age of weaning, and this might be negatively related to a child's birth order or maternal 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

$$AGE\ WEANED = [INCOME_t, PROMO_t, PRENATAL, BRTHORD, SEX, AGEMO, INTVL, EDMO, EDFA, (\#CH<6), \varepsilon_6]. \quad (8)$$

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 non-food expenditure. A conventional demand equation of the following form is used:

$$FOOD = \beta_0 INCOME^{\beta 1}\ (\#CH<6)^{\beta 2}\ (EDMO)^{\beta 3}\ (EDFA)^{\beta 4}\ \varepsilon_7.$$

Dividing by *INCOME* and taking logarithms, this was estimated as

$$\ln\frac{FOOD}{INCOME} = \ln(\beta_0) + (\beta_1 - 1)\ln INCOME + \beta_2 \ln(\#CH<6) + \beta_3 \ln(EDMO) + \beta_4 \ln(EDFA) + \ln\varepsilon_7, \quad (9)$$

where β_1 is the income elasticity for food expenditure in the household.

3. The data

Even though this is a highly simplified model of health and nutritional status, the data requirements for its estimation are substantial, particularly in the context of a developing country. It requires longitudinal data on the physical development and health status of the child, data on nutritional inputs as well as detailed longitudinal socioeconomic and demographic data on the characteristics of the family in which the child is a member. The Candelaria data base is unusual in that it satifies these requirements.

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, aged 16 to 21 and with at least five years of primary school education, were given six months of training. All families with children less than six years of age were visited every two

months. The promotoras provided information on nutrition, hygiene and 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. The promotoras regularly collected demographic, medical and socioeconomic data on both the family and the individual child.

At the time the family entered the Promotora program, socioeconomic data were obtained on each family, along with data on the health status of each child. Thereafter, health and nutritional status data on the children were obtained during 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. Our sample consists of multiple observations on 1,270 children[36] made between 1970 and 1974. There are one to five observations per child, with an average of three (taking account of a one period lag). A considerable amount of effort was invested in 'cleaning' this survey data, either by eliminating clearly spurious or logically inconsistent data or by referring to source documents to correct questionable answers.[37]

Candelaria is a small town 30 kilometers from Cali. It is a transient community, principally composed of migrants from the outlying rural areas who ultimately emigrate to Cali or other larger urban areas. This is reflected by an annual population growth rate of 10%, despite an estimated outmigration rate of 17%. The principal sources of employment are the sugar cane plantations and sugar processing factories. Although there is some home production of vegetables and other foodstuffs, most of the population appears to depend on cash income for survival. The average monthly income level in the community is less than U.S. $30.00.

Candelaria is unusual for its proximity to an active and innovative medical research and educational center, one which had an operational rural health program for 6 to 10 years prior to the inception of the Promotora program.[38] In the Candelaria municipality proper, 67.7% of the homes inside the city had sanitary waste facilities and 90.9% had piped water inside their homes by 1964.[39] Consequently, the rate of malnourishment in Candelaria is

[36]Our sample was chosen from a larger data set containing observations on over 80% of all children under six years of age in the town. Observations of children during 1968 and 1969 were excluded since they lacked information on a child's height. For a more detailed description of Candelaria and of the Promotora program, see Drake and Fajardo (1976).

[37]Several computer programs were written which performed range comparisons and logical consistency checks among different variables. A full description of this methodology is contained in the report on Community Level Nutrition Interventions, available through Community Systems Foundation, Ann Arbor, MI.

[38]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 mothers). Mothers in groups of 25 to 30 took part in seven educational presentations

[39]These numbers are likely to be overestimates for the Promotora client population, since the latter are drawn from a wider area.

lower than the national average for Colombia; only 30% of its children were malnourished in 1963 on a *WTAGE* standard compared to 55.6% nationally in 1965.

The absence of severe, widespread malnutrition in the Candelaria area is also clear from our sample. Although 32% of the children in the sample had *WTAGE* scores below 85, more than 80% of the children had a *WTHT* score above 85. In fact, despite a mean *WTAGE* score of 92.0 for the sample, the mean *WTHT* score was 100.3. Although the Candelaria population is short relative to the N.A.S. standard, with a mean *HTAGE* score of 95.5,[40] significant stunting is even more limited than acute malnourishment. Only 3% of observations are on children scoring below 85 in terms of *HTAGE*. The implications of using such a sample must be noted. The fact that the sample is not heavily weighted with extreme cases of malnutrition implies that our model is likely to be inefficient in estimating the detailed relationships between malnutrition and morbidity among the severely malnourished. Conversely, because there are many children in the sample with normal as well as abnormal development patterns, our data are more likely to capture the variables that *do* differentiate those children that are ill or malnourished. Nevertheless, it is clear that the results should be interpreted and applied with caution.

4. Econometric issues

The estimation of the model requires three principal adjustments for deviations from the assumptions of the classical regression model (OLS). First, OLS estimation would be inefficient for the three dependent variables which are dichotomous, due to heteroscedasticity, and misleading for predictive purposes, since there is no guarantee that the predicted Y would be in the interval (0, 1). A maximum likelihood estimation procedure – logit – of the form

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

is used where, for example, $P(Y_{it}=1)$ is the probability that the ith child had a severe diarrhea episode in period t.[41]

Second, the model 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 – *SEVERE*

[40]It is quite possible that if parental height were taken into account, the magnitude of the alleged deficiency would decrease and possibly disappear.

[41]For a fuller discussion of the problems of estimating equations with dichotomous dependent variables, see Kmenta (1971) and DuMouschel (n.d.).

DIAR, *MILD DIAR.*, and *SICKNESS* – in (3), (4), and (5) are estimated from a first stage logit estimation on the entire set of exogenous variables. These instruments measure the *predicted probability* of particular disease episodes.[42]

Finally, one might expect considerable non-linearity 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 non-linearity with age. Much of the remaining non-linearity relates to changes in potential structural coefficients as a child progresses in age; this is dealt with through multiplicative dummy terms (see footnote i of table 2). Since it is the first three years that are the most critical for a child's nutritional development and its risk of illness, the non-linearities 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.

5. Estimation results

Table 2 displays the econometric estimates of the structural model of nutritional status of (3), (4) and (5); and table 3 presents estimates of the model of health status, of the determinants of the age of weaning and of the level of food expenditure. To assess the impact of policies on nutritionally vulnerable children, the model is also estimated on the restricted sample of children that have ever suffered any degree of malnourishment.[43]

Interactions of health and nutritional status. Our results confirm that the timing and severity of a diarrheal episode determine its nutritional impact. From table 2, eq. (2) severe diarrhea in the early part of the first year of life has a highly significant, negative effect on a child's structural development in the course of that year, with a loss of almost five points in *HTAGE*.[44] This might explain the unusual positive coefficient for severe diarrhea in terms of *WTHT* score in the first year, since the child's weight may be less adversely

[42]Since the data set represents a pooling of cross section and time series observations, tests for autocorrelation were made by estimating a first order autocorrelation coefficient. In the equations for diarrhea and sickness, ρ is low, ranging from -0.01 to $+0.03$. 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 in the nutritional status equations.

[43]For the *WTHT*, *WTAGE* and *HTAGE* equations, the samples include all observations on any child that has ever had (i) a *WTHT* score less than 85, (ii) a *WTAGE* score less than 85, and (iii) a *HTAGE* score less than 87, respectively. The results are reported in tables 2 and 3.

[44]From Heller (1976) the contemporaneous effect of a severe diarrhea episode in the first six months of life on *HTAGE* is not significant. In the *HTAGE* equation [table 2, eq. (2)], we capture the effect on diarrhea in the *previous* period. Thus, for infants, we are observing the effect on *HTAGE* in the latter half of a child's first year.

Table 2

Determinants of nutritional status[c] – Entire, stunted and malnourished samples: Structural equations.

Exogenous (X) or endogenous (N)	Dependent variables / Independent variables	Total sample			Stunted or malnourished group		
		Weight for height (coefficient) (1)	Height for age (coefficient) (2)	Weight for age (coefficient) (3)	Weight for[a] height (coefficient) (4)	Height for[b] age (coefficient) (5)	Weight for age (coefficient) (6)
	All children						
N	Pr. sickness	7.37**	−4.82**	−2.74	9.05	−7.18*	−15.09**
X	Birth order	−0.30**	−0.05*	−0.35**	−0.35*	0.19*	−0.33**
X	# Children less than 6 yrs.	−0.03	−0.13*	−0.29*	0.08	0.08	−0.26
X	Weanling observation	−1.84**	0.26	−0.42	0.29	1.20*	0.96*
X	Education level – mother	0.86**	−0.20**	0.23	0.55	−0.72**	−0.16
X	Education level – father	−0.06	0.12	0.34*	−0.12	0.13	0.78**
X	Father's age	0.10**	−0.01*	0.03	0.11*	−0.06*	0.06*
X	Mother's age	−0.08**	0.03**	0.03	−0.16	0.02	0.09*
X	Modern birth control use	0.22	0.06	0.32	1.03	−0.37	−0.66*
X	Birth interval	0.02*	0.0	0.01*	0.05*	0.02**	0.01
X	Pct. weight for height$_{t-1}$	0.49**	0.07**		0.32**	0.10**	
X	Pct. weight for age$_{t-1}$			0.66**			0.50**
X	Pct. height for age$_{t-1}$	0.14**	0.72**		0.15*	0.61**	
	Age: 0–11 months[f]						
N	Prob. sev. diarr. episode	16.52*		2.85	29.77*		8.05
N	Prob. mild diarr. episode	−6.1		10.65	43.9		17.5
N	(Food exp) (prob. diar)[d]	0.35*	−0.14*	−0.12	−0.86*	−0.55*	−0.90**
X	Sev. diar. episode$_{t-1}$		−4.95**				
X	Mild diar. episode$_{t-1}$		0.75*				
X	Pct. of 1st yr. nursed	5.01**	−0.18	3.59**	5.17*	−0.40	4.0**
X	Food exp/capita	−0.08	0.04	0.02	0.04*	0.11	0.21**
X	Age	34.40**	17.97**	28.58**	29.41**	22.5**	39.17**
X	% life in Promotora Prog.	0.46	0.09**	−0.34	19.71**	0.67**	1.5*
X	Sex (boy = 0, girl = 1)	0.19*	0.41	−2.24*	−3.86*	−0.03	−3.48*

Table 2 (*continued*)

Determinants of nutritional status[e] – Entire, stunted and malnourished samples: Structural equations.

Exogenous (X) or endogenous (N)	Dependent variables / Independent variables	Total sample			Stunted or malnourished group		
		Weight for height (coefficient) (1)	Height for age (coefficient) (2)	Weight for age (coefficient) (3)	Weight for[a] height (coefficient) (4)	Height for[b] age (coefficient) (5)	Weight for[c] age (coefficient) (6)
	Age: 12–23 months[f]						
N	Prob. sev. diar. episode	−13.68*		−15.38	−30.04		−20.28
N	Prob. mild diar. episode	40.90*		10.95	7.06		2.50
N	(Food exp.) (pr. diar.)[d]	0.22*	−0.15**	−0.16	−0.21	−1.08**	−0.75**
X	Sev. diar. episode$_{t-1}$		0.39*				
X	Mild diar. episode$_{t-1}$		0.39*				
X	Pct. of 2nd yr. nursed	0.26	−1.27**	−3.13**	−2.54	−1.90	−4.43**
X	Food exp./capita	−0.03	0.05*	0.06	0.05*	0.20*	0.25**
X	Age	32.22**	19.42**	29.91**	39.23**	26.91**	42.23**
X	% life in Promotora prog.	−5.04**	2.22	0.33	1.15**	3.02	2.07
X	Sex (boy = 0, girl = 1)	0.54*	0.34	0.31	1.53	−0.25	−0.51*
	Age: 24–72 months[f]						
N	Prob. sev. diar. episode	−25.31*		−10.63	−45.16		−8.52
N	Prob. mild diar. episode	11.90		−9.84	50.85		−21.37
N	(Food exp.) (pr. diar.) 24–48 mo.[d]	−0.02	−0.19*	−0.35*	−0.08	0.85*	−1.29**
X	Sev. diar. episode$_{t-1}$		−0.22			−1.05*	
X	Mild diar. episode$_{t-1}$		−0.22				

X	Months nursed	0.05*	0.01	0.03	0.01	0.08**	0.05
X	Food exp./capita 24–48 mo.	−0.04	0.06**	0.08*	−0.06*	0.22*	0.22*
X	Food exp./capita 48–72 mo.	−0.04	0.02	0.01	−0.20*	0.02	−0.02
X	Age: 36–48 mo.	33.57**	19.96**	29.96*	30.95**	24.29*	42.78**
X	Age: 48–72 mo.	31.50**	20.34**	28.71*	31.90**	26.99*	42.91**
X	% life in Promotora prog., 24–36 mo.	−0.41	1.29	0.11	2.48**	4.17	−1.10
X	% life in Promotora prog., 37–72 mo.	−0.64	0.70*	0.16	19.36**	1.72	1.5
X	Sex (boy=0, girl=1),						
	R^2	0.35	0.62	0.61	0.25	0.59	0.44
	N	2194	2230	2153	547	332	1218
	Estimation procedure	TSLS	TSLS	TSLS	TSLS	TSLS	TSLS

[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%.
[d]This variable is the product of real weekly food expenditure and the probability of diarrhea in a particular time period.
[e]Significance level of 80% for figures with one asterisk. Significance level of 95% for figures with two asterisks.
[f]These age-specific 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 table, we have already made the above additions for all the multiplicative dummy expressions in order to facilitate the interpretation of the results. The statistic on significance displayed is that corresponding to β_A, β_B or β_C, etc. For any particular variable, the variable relating to the oldest age group is equivalent to variable A in the above example.

Table 3

Determinants of (i) episodes of mild and severe diarrhea and of other illnesses[c], and (ii) parental food decisions.

Dependent variables / Independent variables	Morbidity variables			Parental food decisions[a]	
	Mild diarrheal disease (coefficient)	Severe diarrheal disease (coefficient)	Days with illness (coefficient)	Age weaned (coefficient)	$\left(\frac{\text{Food expenditure}}{\text{Income}}\right)^{a}$ (coefficient)
Endogenous					
Pr. sickness	−0.26*	0.19*			
Change in percentile position, weight for height	0.23**	−0.14	0.02		
Exogenous					
Sex (boys = 0, girls = 1)	0.04	−0.27	0.25	−0.20	
Birth order	0.02	−0.09**	0.03	−0.55**	
# children less than 6 yrs.	0.02	−0.17*	−0.02	1.41**	0.10**
Education level – mother	−0.17	0.13	−0.15**	−0.67*	−0.01
Education level – father	0.21	−0.07	0.08	0.33	−0.51**
Father's age	−0.01	0.02	0.0		
Mother's age	0.02	−0.02	−0.02	0.39**	
Modern birth control use	0.18	0.10	−0.17		
Birth interval	−0.26	−0.0	0.0	0.09**	
Weaning observation	0.49**	−0.04	−0.19		
% life in Promotora program[b]					
0–11 mo.	0.59	−0.01	−0.07	−9.65**	
12–23 mo.	0.70	−0.32	−0.41		
>24 mo.	−0.88	−0.03	0.24		
Girls	−0.69	−0.54	0.56		
DPT immunization	0.71	0.30	−0.19		
Polio immunization	−1.01*	−0.64	0.38		

Income per capita	0.40**	−0.10	0.20	−1.3**	−0.50**[d]
Prenatal care to mother	0.18	0.02	−0.14**	0.81	
Birth weight, children < 12 mo.	0.50*	−0.37	0.08		
Stunted$_{t-1}$	0.29*	0.28*	0.23*		
Malnourished, 6–24 mo.$_{t-1}$	0.04	0.48	0.12		
Malnourished$_{t-1}$	−0.0	0.01	0.34**		
Year of observation	0.11*	0.09	0.30**		
Months nursed, children >24 mo.	−0.02	0.01	0.01		
Pct. of 1st yr. nursed	0.57	0.59	0.26		
Pct. of 2nd yr. nursed	−1.13	0.39	0.05		
Age dummy[b]					
0–11 mo.	−12.42**	−6.83*	−23.78**		
12–23 mo.	−10.79**	−8.49*	−23.20**		
24–35 mo.	−10.41**	−8.72**	−23.63**		
36–48 mo.	−11.23**	−9.06*	−23.46**		
48–72 mo.	−11.06**	−9.62*	−23.85**		
R^2	0.04	0.04	0.01	0.21	0.32
Predictive power	0.68	0.77	0.59		
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.
[b]See footnote f of table 2.
[c]Significance level of 80% for figures with one asterisk. Significance level of 90% for figures with two asterisks.
[d]In this equation, income is measured in 1968 pesos.

affected than its height. This is supported by the negligible change in *WTAGE* score that is associated with severe diarrhea in the first year. With a slowing of growth, the *WTHT* score 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* score (by 0.75 points) and a lower *WTHT* score.

After the first year, a severe diarrheal episode is highly deleterious for a child's *WTHT* score, with a potential loss of 1 to 2 points. A mild diarrheal episode does not appear to lower a child's *WTHT* score significantly. Diarrhea is also of minor consequence for a child's *HTAGE* score at this time (even if one considers the indirect effect on *HTAGE* caused by the effect of a severe diarrheal episode on *WTHT*). These results tend to validate the econometric model as they are consistent with the results found in the literature on malnutrition.

Policies to reduce the likelihood of severe diarrhea would have a stronger impact on a nutritionally vulnerable child. Even more than with normal children, severe diarrhea *after* the first year will lower the acutely malnourished child's *WTHT* score by as much as 4.5 points [table 2, eq. (4)]. The effect of a past history of severe diarrhea is to reduce a stunted child's *HTAGE* score by 1 point. As above, a mild diarrhea episode is not clearly adverse to a child's nutritional status.

The most damaging effect of other illnesses is with respect to a child's long-term structural development, with a significant decline in *HTAGE* score. The attendant rise in *WTHT* score again suggests that in the event of illness, the body channels nutrients to the higher priority of maintaining body weight at the expense of normal structual development. Although the *WTHT* score rises, *WTAGE* score falls, though *less* than *HTAGE*.

Malnourished children are also at higher risk of further illness [table 3, eq. (3)]. For a child in the critical age of 6 to 24 months, a low *WTHT* score in the previous period raises the probability of a severe diarrhea episode (table 3, eq. (2)]. Similarly, stunted children are more susceptible to both *mild* and *severe* diarrheal episodes. Equally interesting, there is an inverse (direct) relationship between a change in *WTHT* score and the probability of severe (mild) diarrhea. Since severe diarrhea is the principal determinant of a lowered *WTHT* score, the self-reinforcing effect of lowering diarrhea and improved nutritional status is clear 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.

Finally, the coefficient of the nonlinear term $(FOOD*PR.DIAR.)_{it}$ supports the argument that poor health not only directly weakens a child's nutritional status, but also reduces the efficiency of nutrient use. The variable's coefficient is negative in the *HTAGE* equations, 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 greater food expenditure at this time may weaken the adverse effect of diarrhea by channelling nutrients to maintain body weight, at the expense of structural development. For children at risk of acute and chronic malnourishment, even this displacement is unsuccessful. The negative coefficient $[FOOD*PR.DIAR.]_{tk}$ in both equations suggests the dominant negative effect of diarrhea on the impact of food expenditure.

Dynamics of nutritional change. First, it is important to note that a large proportion of the variance among children in nutritional status in the first seven months of life is not explained by our socioeconomic or health factors. In Heller–Drake (1976b), we attempted to explain the nutritional scores of children aged 1 to 7 months, and obtained an R^2 of no more than 0.05. These equations did not include variables proxying a child's genetic inheritance (such as parental height or stature), and it is likely that such variables would explain much of this variation. Similarly, it is not surprising that there is a positive correlation between the *WTHT* and *HTAGE* scores 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* scores 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: 0.32 relative to 0.50 (table 2, eq. (4)). An acutely malnourished child has far greater potential for variability in its *WTHT* score.

Second, the results indicate that a child's *HTAGE* is positively correlated with its *WTHT* in the previous period and vice versa. Thus, nutritional programs may promote improvements in *WTHT* score for their dynamic impact on structural development. This effect is greatest for the stunted group, with a coefficient of 0.10 relative to 0.07 [table 2, eqs. (2) and (5)].

Some evidence also emerges on the hypothesis of a 'natural' history of a child's nutritional and health status. Examining the age dummy terms in the structural equations, *no* systematic pattern emerges across age groups. 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* score 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.

The impact of nutrient sources. The relative impact of breast feeding and food expenditure clearly changes in the early years of a child's development. In the first year, a child that is breastfed over the entire year will emerge with a highly significant difference in its *WTHT* score, relative to a child that has been weaned immediately. For example, for every month breastfed in the

first year, a child gains more than 0.4 points in its *WTHT* score (up to the 5.01 points associated with breast feeding during the entire period) [table 2, eq. (1)]. Per capita food expenditure has a negative, though insignificant, impact during the first year. If a child were immediately weaned at birth and consumed the mean weekly food expenditure of 253 pesos for the sample, this would suggest a *WTHT* percentile position 1.97 points lower. From earlier estimates on the age group 1 to 7 months, the effect is strongest during this period [Heller–Drake (1976)], 7.65 points, 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 *HTAGE* score is insensitive to these alternative nutrient sources during the first year.

After the first year, breast feeding becomes significantly less important for a child's *WTHT* score and has a dampening effect on the child's *HTAGE* score (with a maximum loss of -1.26 points in *HTAGE* for a child that has not been fully weaned by the end of its second year). The negative effect is more striking in terms of *WTAGE* (with a maximum potential loss of approximately -4.06 points). For older children (over 2 years), nursing has a long-term legacy of 0.05 points on the *WTHT* score for each month nursed.

Surprisingly, the level of family food expenditure per capita is not a critical factor in determining nutritional status variations, at least for the entire sample of children. Although the coefficient of $FOOD_{tk}$ in the *HTAGE* equation is statistically significant, even a level of food expenditure per capita one standard deviation above the average for the sample would yield no more than a 1.5 point difference in *HTAGE* score. As expected, nutritional scores decline during the weaning period, but as implied above, only in terms of the *WTHT* score.

For an acutely malnourished child (low *WTHT* scores), the appropriate pattern of nutritional supplementation is clear from table 2. Breast feeding in the first year is at least as decisive as above.[45] In the second year, weaning of a malnourished child becomes *more* imperative than for the entire sample, as the coefficient on breast feeding becomes sharply negative. The positive effect of food expenditure during this and subsequent periods is not large; a one standard deviation change in weekly food expenditure (150 pesos) has no more than a 0.75 point impact in *WTHT* score.

For a stunted child (low *HTAGE* score), the effect of breast feeding 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, eqs. (2) and (5)]. $FOOD_{tk}$ is relatively more[46]

[45]Estimates of reduced form equations suggest that each additional month of nursing is worth 0.7 points (as compared with 0.53 for the entire sample) [Heller and Drake (1976)].

[46]The reduced form coefficients suggest a potential net change half as large, but still this remains significant [Heller and Drake (1976)].

decisive for the stunted group; in the second and third years, a one standard deviation increase in weekly food expenditure can raise the *HTAGE* position as much as 3 points (relative to only approximately 0.45 for the entire sample).

Two additional points should be noted. The results do not suggest 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. Breast feeding during the entire first year contributes half a centimeter to height. The results of table 2 capture changes relative to a norm. Second, the advantage of differentiating between *WTHT* and *HTAGE* emerges from the results. Eq. (3) of table 2 suggests that breast feeding in the second year lowers *WTAGE* whereas the results of eqs. (1) and (2) in table 2 indicate that this occurs only because it causes stunting, not because the child's *WTHT* score is adversely affected. For the same reason, the *WTAGE* variable obscures the contribution of food to height as opposed to weight.

Finally, is a child's health status affected by whether it is breastfed or at the time of weaning? A child that is breastfed through the second year has a significantly lower likelihood of mild diarrhea [table 3, eq. (1)]. This may simply reflect that delayed weaning postpones exposure to food-borne illnesses.[47] The weaning period is clearly a period of increased sensitivity to diarrhea, though not to other illnesses.

Promotora program. The results suggest that the principal impact of the Promotora program was on long-term nutritional status (*HTAGE*), with the strongest impact on children in their second year of life. The impact of a high proportion of a child's life in the Promotora program on its *HTAGE* score is only 0.09 points for a one-year old compared with 2.2, 1.2 and 0.7 for children in the next three years, respectively. The short-term effect on *WTHT* or *WTAGE* is generally insiginificant; when it is significant, it is 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 from *WTAGE* in the sample considered.[48]

[47]This is also borne out by the difference in mean probability of diarrhea according to whether a child is breastfed. In the first two years of life the probability of mild and severe diarrhea while nursed is 0.072 and 0.064, respectively; for a child that is weaned, these probabilities are 0.09 and 0.10, respectively. These are monthly probabilities of diarrhea.

[48]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. Much of the relevant change in nutritional status inspired by the program may have occurred during that period. 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

In terms of health, participation in the Promotora programs *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 the level of risk to both kinds of diarrhea.

Medical programs. 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 significantly) *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. Use of health services, receipt of prenatal care by the mother, and use of modern birth control methods all reduce the probability of other illnesses. The policy implications of these results should not be overstated, since we could only weakly measure the quality and quantity of health services consumed by the family.

5.1. *Economic constraints and family decisions affecting nutritional and health status*

Parental income.[49] Our model suggests family income influences a child's nutritional status through its effect on nutrient intake, and health status through the purchase of goods and services. As might be expected, food expenditure rises less rapidly than income, with an income elasticity $(1+\beta_{INCOME})$ of 0.50 [table 3, eq. (5)]. Thus, substantial leakage 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 age of weaning is accelerated as income rises. This may reflect the effect of a higher opportunity cost of the mother's time and/or that breast milk may be considered an 'inferior' good. Every additional thousand pesos of monthly income would reduce the nursing period by 1.3 months.

The net effect of income on nutritional status, as estimated from the reduced form equations (not shown),[50] is positive, but quantitatively small. For the entire sample, a thousand peso increase in income raises *HTAGE* and *WTAGE* scores by 0.3 and 0.9 points, respectively. Increased income has little discernable effect on children already stunted, though it has an input on

problems and it is possible that a negative correlation between program participation and nutritional status may reflect this.

[49]Mean real monthly per capita income is 1253 pesos, with a standard deviation of 971 pesos.

[50]Heller and Drake (1976, p. 27).

children at risk of low *WTHT* scores (with an income elasticity on *WTHT* score of 1.4%).

Finally, by allowing greater parentally provided inputs higher income significantly reduces the child's probability of mild diarrhea, with an elasticity of 0.47. On the other hand, it raises the probability of other illnesses, but at a lower level of significance ($t=1.23$) and elasticity (0.188).

Competition for family resources. The results indicate that child competition adversely affects nutritional status, though not health status. First, nutrient intake per child falls. Although the level of family food expenditure rises with the number of young children, the elasticity is low: 0.10. 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 greater the number of competing children under age 6, the lower the *HTAGE* score [table 2, eqs. (1) to (3)]. Family planning programs aimed at wider spacing between a child and an earlier sibling may both lead to a longer period of breast feeding[51] and may raise *WTHT* and *WTAGE* scores, though the maximum differential is only 1 point.[52] 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 0.045 points and *HTAGE* by 0.024 points. Both effects are larger than for the entire child population sample.

Parental discrimination across children. Our hypothesis that higher birth order children are discriminated against is confirmed. 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 0.5 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.[53] This lends support to the cause not being a lower weight new-born, but rather intrafamilial discrimination. Later parity children will also receive one-half month less nursing than the preceding child [table 3, eq. (4)]. This is after correction for the effect of the age of mother on nursing.[54]

Our results are not conclusive on whether there is a bias against baby girls or not. Despite the fact that the anthropometric standards are sex-specific,

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

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

[53]In the sample of children aged 0–6 months, birth order has no effect on nutritional status. Heller and Drake (1976, p. 26).

[54]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.

girls are clearly lower in *WTAGE* [table 2, eq. (3)] by as much as 2 to 3 points at the end of the first year of life, with the differential narrowing thereafter to 1.2–1.7 points.[55] In the first year this reflects a slightly higher *HTAGE* position and a lower *WTHT* position; thereafter, possibly after weaning, their *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.[56, 57]

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 clear from the results. First, more educated parents[58] 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, 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 appear to have higher *WTHT* and lower *HTAGE* scores. 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* score, 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 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

[55]This is from the reduced form equations [Heller-Drake (1976)].

[56]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.

[57]In eq. (6) and (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.

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

evaluating the risks of diarrheal disease or of malnourishment in maternal-child health programs.

6. 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, the data subject to considerable imprecision, and the methodological problems of estimating such a model great, the ambiguity of some of our results is not surprising. In fact, the clarity of many of the results is itself unusual given these problems. However, the following insights are of sufficient heuristic value such as to warrant *further* policy analysis in order to verify the accuracy of these estimates.

(i) The character of nutrient intake in the first two years of life is pivotal. Insufficient breast feeding in the first year sharply lowers the child's current *WTHT* score, with an equally serious dynamic impact on its *HTAGE* score. In fact, breast feeding 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 breast feeding 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 breast feeding is 9–11 months within the sample. This suggests that family characteristics which imply significantly lower or higher periods of breast feeding 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 decline.

(ii) Our model affirms the importance of differentiating between a child's *WTHT* and *HTAGE*. 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 incorporate this distinction into their analyses of the social costs of malnutrition. If these costs were to arise principally 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, *once they have occurred.*

(iv) Although our specification of the health status model includes a substantial number of *potentially* relevant variables, our understanding of the factors that contribute to a diarrhea episode's occurrence remain limited ($r^2 = 0.02$–0.03). 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. Absence of measures of environmental quality 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) 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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