A RAND NOTE

BIOLOGICAL AND BEHAVIORAL INFLUENCES
ON THE MORTALITY OF MALAYSIAN INFANTS

William P. Butz, Julie DaVanzo,
Jean-Pierre Habicht

April 1982

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The Agency for International Development

Rand
SANTA MONICA, CA. 90406

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PREFACE

This Note is a publication of The Rand Corporation as well as of the Cornell University Agricultural Experiment Station, Division of Nutritional Sciences. The research upon which it is based was funded by Grant No. AID/otr-1744 from the U.S. Agency for International Development to The Rand Corporation. This grant supported research on breastfeeding, contraceptive use, birthspacing, and infant mortality in Malaysia. The survey data analyzed were collected under an earlier contract, No. AID/pha-1057, between AID and Rand.

This Note should be of interest to researchers and policymakers concerned with the causes of infant mortality in Malaysia and other less developed countries. It should particularly interest persons concerned with understanding how the joint mortality influences of biological and behavioral factors change over the infant's first year of life.

William P. Butz and Julie DaVanzo are economists on the staff of The Rand Corporation. Jean-Pierre Habicht is James Jamison Professor of Nutritional Epidemiology at Cornell University. Their order of authorship was determined alphabetically.

SUMMARY

This study examines the determinants of infant mortality variations in Peninsular Malaysia. It considers proximate biological correlates of mortality as well as family characteristics and behavior, and inspects the degree to which some of these latter factors have their effects indirectly through more proximate factors. It assesses how these influences and interactions change in importance through successive subperiods of the first year of an infant's life.

The analysis is based on data from the 1976-77 Malaysian Family Life Survey (MFLS). The data are primarily retrospective, largely drawn from subject-reported questionnaires. They document proximate biological correlates of infant mortality, as well as many family-level and community correlates. Included are most of the influences commonly cited as affecting infant mortality: maternal education, socioeconomic class, age, birthspacing, and prior reproductive loss; availability of health services; and infant's sex, breastfeeding and type of weaning food, birthweight, and birth order. In addition, the analyses include proxies for exposure to respiratory and gastrointestinal diseases, measures of the mother's availability for child care and of household composition, child's year of birth, ethnicity, and a measure of rurality.

The estimates derive from a linear probability model estimated by ordinary least squares. This model is appropriate for processes that influence mortality through attributable risk. For comparison, we also show logit estimates, appropriate for estimating relative risk.

These data, despite being mothers' reports of events many years earlier, produce many statistical associations with infant mortality that are consistent with the clinical and epidemiological literature. Examples are the elevated mortality risk associated with very young maternal age, low birthweight, short previous birth interval, and male sex of the child. These corroborations with clinical data suggest that retrospective data can yield valid conclusions about influences on infant mortality. This paper moves beyond these findings to investigate other less well-known interrelationships with infant mortality:

- Biological factors such as low birthweight are more important early in the first year of life, while such behavioral and environmental factors as mother's education or types of water and sanitation system are more important later.
- When proximate mortality determinants are not controlled, Indians' infant mortality is not significantly different from Malays'. When the more proximate correlates are controlled, Indian infant mortality is significantly lower than Malays'. This is because Indians have lower birthweights and shorter intervals preceding their births.
- When no other variables or only the biological attributes at birth are controlled, rural babies are significantly more likely to die in the first week and last six months of infancy. But when other biological and family influences are controlled, no rural/urban differentials emerge. Hence, it is not rurality *per se*, but postnatal biological influences (e.g., type of water) and indirect family influences (e.g., mother's

- Many babies' breastfeeding is short because they died rather than vice versa. When this source of spurious correlation is removed, the estimated effects of breastfeeding on infant mortality decrease significantly. Previous studies have not removed this source of bias and have hence overestimated the effects of breastfeeding, sometimes drastically. We find that length of breastfeeding (especially unsupplemented breastfeeding) is nevertheless one of the strongest correlates of infant mortality, but no stronger than several other factors that public programs can influence.
- Presence of piped water or toilet sanitation is most important in reducing infant mortality for women who breastfeed little or not at all. This importance is less where breastfeeding is initiated and maintained for a month. Presence of toilets is more important than piped water in nearly every instance.
- Breastfeeding declines are less harmful in households with either toilets or piped water and much less harmful in households with both. In actuality, women in this Malaysian sample who breastfeed very little or not at all tend disproportionately to live in houses with modern water and sanitation facilities. The study suggests that their infants suffer little mortality risk on average from the lack of breastfeeding. Much more dangerous is lack of breastfeeding in communities without these modern facilities, where water used in mixing alternative foods is more likely to be contaminated.

These new findings have specific implications for risk screening programs and for direct interventions to reduce infant mortality, which are discussed.

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I. INTRODUCTION

Infant mortality is widely used as a summary measure of socioeconomic development and well-being and is included in almost all composite indicators used to measure welfare. Often, it is even factored in twice (Morris, 1979; Grant, 1981)--once directly and once again as life expectancy, of which it is a major component. This study examines the determinants of infant mortality variations in Peninsular Malaysia. Its goals are (1) to identify which infant and family characteristics are most conducive or detrimental to infant survival; these are candidate targets for interventions; and (2) to improve the effectiveness of these or other interventions by specifying screening characteristics of high-risk populations, to whom interventions could be effectively targeted.

It is generally known (Klein, 1980; United Nations, 1973) that infant mortality is influenced by characteristics of the newborn, such as birthweight, and of the mother, such as her nutrition, literacy, socioeconomic class, marital status, smoking, use of health services, extremes of reproductive age, high parity, and prior reproductive loss.[1] How these different influences interact in affecting mortality outcomes is often unclear, however, both logically and empirically.

Two premises underlie our attempt to clarify some of these relationships in this paper. The first is that the causes of death and their determinants change as an infant ages and that analysis of the determinants of infant mortality should therefore be disaggregated over

[1] See United Nations (1973) for a succinct review of the evidence regarding the correlates of infant mortality.

subperiods of the first year of life. The second premise is that although the ultimate cause of death is biological, the determinants of the fatal biological factor may be a chain of biological and behavioral factors (Butz and Habicht, 1976). We therefore consider both proximate biological correlates of mortality as well as family characteristics and behavior, and inspect the degree to which some of these latter factors have their effects indirectly through changes in more proximate factors. Combining these two premises, we consider how these influences and interactions change in importance through successive subperiods of the first year of an infant's life.

To address these issues we use data from the Malaysian Family Life Survey (MFLS). The data are primarily retrospective, largely drawn from subject-reported questionnaires. They document proximate biological correlates of infant mortality, as well as many family-level and community correlates.

With these data we can move only part way toward our goals, far enough in many cases to suggest that there is something about particular factors that is associated with significantly higher (or lower) mortality, but usually not so far as to be completely confident of the causal relationships and their underlying mechanisms. Most of our specific findings are therefore suggestions, some strong and others tentative, that particular factors now be examined in detail. Special investigations, preferably with actual trial interventions and appropriate controls (Habicht and Butz, 1980), can then focus on these, out of the myriad interventions possible, and thereby determine which will be truly effective and at what cost.

Section II describes the Malaysian Family Life Survey data set we have used and how these data were structured for our analyses. Section III presents and discusses our main empirical results. Section IV explores the relation between infants' feeding and their mortality in more depth. The Note concludes, in Section V, with a discussion of implications for research, programs, and policy.

II. DATA, SAMPLES, AND ESTIMATION ISSUES

DATA

We use data from the 1976-77 Malaysian Family Life Survey (MFLS).[2] This population-based probability-sample survey comprised three rounds in the same panel of households, four months apart, beginning in August 1976. The sample consists of randomly-selected private households that each contained at least one ever-married woman less than 50 years old at the initial visit. A total of 1262 households (88 percent of the eligible probability sample) completed Round 1 of the survey. These households are contained in 52 primary sampling areas in Peninsular Malaysia. Forty-nine of these areas were randomly selected; the other three were purposively selected to give additional representation to Indian households and households in fishing communities.

The key questionnaire for this analysis is the Round-1 Female Retrospective Life History questionnaire (MF2), which includes life events as early as the mid 1940s for some respondents. A few items also came from the Female Attitudes and Expectations questionnaire (MF7). Questionnaire MF2 includes a complete life history of all of a woman's pregnancies and related events. For each pregnancy the woman was asked the date and type of outcome (live birth, stillbirth, miscarriage, or abortion). For each live birth, information was collected on birthweight, lengths of full (unsupplemented) and supplemented breastfeeding, whether the child died and, if so, the date of death.[3] The retrospective data also contain a residential history, including

[2] For more information about the MFLS, see Butz and DaVanzo (1978).

[3] Some mothers were unable to report the exact death dates of

characteristics of houses where respondents have lived (such as type of toilet, with whom lived). The questionnaire MF7 includes information on birthplaces of each woman's first and most recent births and on the first weaning or supplemental food given to those children.

The data from these two questionnaires include most of those influences commonly cited as affecting mortality (Klein, 1980; United Nations, 1973) (maternal education, socioeconomic class, age, parity, prior reproductive loss, availability of health services, and infant sex and birthweight). Maternal smoking and illegitimacy are not examined because they are both rare in Malaysia[4], and data on them were not collected. Maternal nutrition is not included, although its contribution to birthweight in these data is discussed in detail in a related paper (DaVanzo, Habicht, and Butz, 1981) and is mentioned here when appropriate. Respiratory and gastrointestinal morbidity are not measured directly. For respiratory diseases, exposure to infection is estimated from a measure of persons per room; for gastrointestinal diseases, exposure is estimated from data about household water and sewage provisions. Within the past two decades some researchers have also considered birthspacing (reviewed in Wray, 1971), breastfeeding and type of weaning food (reviewed in Jelliffe and Jelliffe, 1978), and desire for having this baby (Scrimshaw, 1978) as important mortality determinants. We consider all four. In addition, year of birth, ethnicity, and a measure of rurality are included because of their demonstrated association with Malaysian infant mortality (Government of

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their deceased children. Using other information in the survey, we were able to place all of these deaths within the age intervals examined in this study.

[4] Personal letter dated September 17, 1981 from Khairuddin Yusof, M.D., Professor of Social Obstetrics, University of Malaya.

Malaysia, various years). Measures of mother's availability for child care, such as hours of work, distance to work, and occupation are also considered. Finally, we include measures of household composition as proxies for demands and resources not identified by other measures.

The reliability and validity of any retrospectively reported data on infant mortality and associated life events are usually open to serious question. In this case, however, Haaga (1982) has investigated important aspects of these issues for the MFLS. He finds that the Chinese-Malay and male-female differences, and the secular trends, in infant mortality in the MFLS data are generally similar to those indicated by Malaysian vital statistics.[5] Figure 1, for example, compares trends in infant mortality rates calculated from the MFLS and Malaysian Vital Statistics. These comparisons show no evidence for decreased reporting of mortality events in the distant past.

As is true in many surveys, the self-reported breastfeeding data exhibit considerable heaping at lengths of 6, 12, 18, and 24 months. Haaga (1982) shows that the source is in the reporting rather than actual breastfeeding norms. Removing these heaped observations from the sample does not notably change the breastfeeding coefficients or their statistical significance in this analysis. Haaga has also investigated the quality of several other parts of the data used in this analysis, finding most of them to be adequate to support multivariate analysis.

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[5] An exception is for Indians in the MFLS sample, whose infant mortality is higher than that indicated by vital statistics for the entire population of Malaysian Indians. The MFLS Indian sample is relatively small.

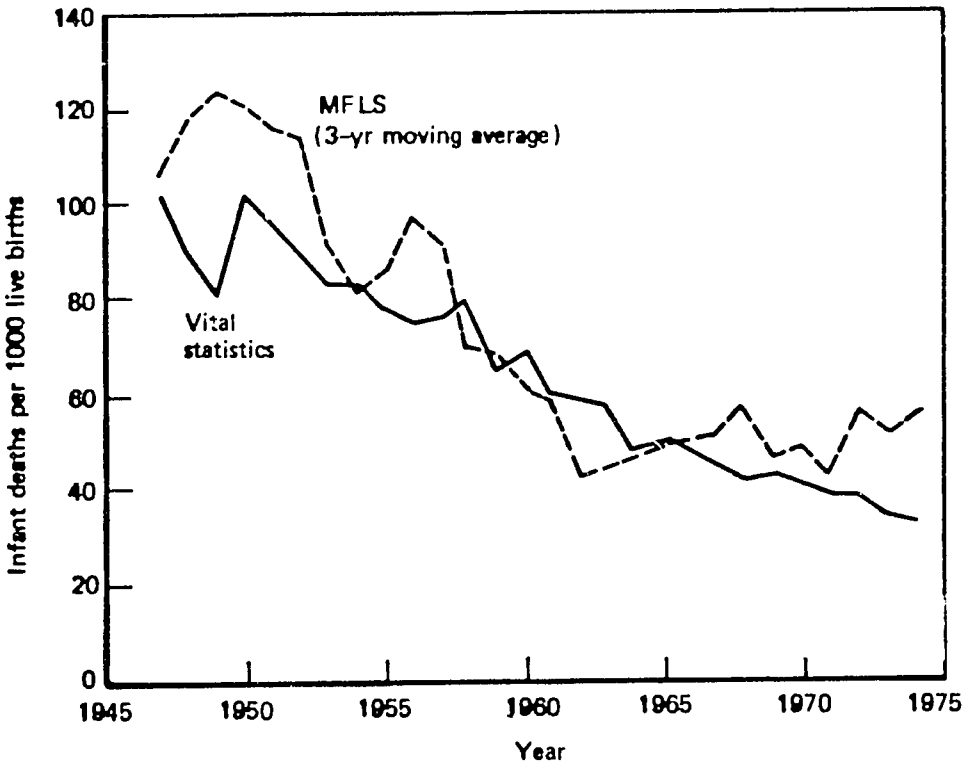

Fig. 1 – Trends in infant mortality rates: Comparison of MFLS and Malaysian vital statistics

ESTIMATION ISSUES

The principal weakness of these data lies not in their reliability but in the small number of mortality events, relative to the inferences we desire to make about them. Malaysian infant mortality rates are relatively low for a developing country. The total sample used in this analysis contains 5573 live singleton births, of whom 270 (48 deaths per 1000 live births) died in infancy. These numbers are adequate for studying the determinants of infant mortality, but less so for investigating mortality during subperiods of the first year of life.

Some of the mortality determinants we investigate are related to general public health conditions that affect the mortality risk of most infants subjected to these influences. Logit analysis is appropriate for these factors because it is a multivariate method for estimating relative risk. The logit coefficients are the natural logarithms of the relative odds by which the determinants of mortality increase the risk of dying. When, as in this paper, the relative odds are low, they approximate the relative risks, which are intuitively easier to interpret (McMahon and Pugh, 1970). For example, in a regression that controls for other mortality correlates, the logit coefficient on living in a house with a toilet is -.425. This means that the presence of a toilet reduces the logarithm of the odds ratio by 42 percent, and that a baby living in a house with a toilet is about 2/3 (antilog -.425 = .65) as likely to die in infancy as is one living in a house without a toilet, other things the same.

Some of the determinants, on the other hand, affect only a subportion of children exposed. The appropriate description of these effects is through attributable risk, which is appropriately analyzed using a linear probability model such as ordinary least squares (OLS). The OLS coefficient indicating the reduction in attributable risk from living in a house with a toilet is -.029. There are 29 less infant deaths per thousand babies living in houses with toilets compared with those living in houses without toilets, other things the same.

Our small samples of mortality events, combined with relatively large numbers of variables and concentrations of mortality events at particular values of some variables, make logit estimation impossible

except in the largest sample, that for infant mortality as a whole. The estimates reported below therefore derive from a linear probability model estimated by OLS. For comparison, Table 4 shows a logit regression of the most complete specification on the entire infant mortality sample. Almost all inferences about the relative strengths of association among the determinants of infant mortality remain the same. Trussell and Preston (1981) show that this similarity between OLS and logit should be expected and, further, that OLS analyses result in inferences similar to those from a hazards model.

Our strategy is to investigate mortality determinants in successive subperiods of infancy, using a life table approach. For each subperiod we use a sample of those who survived until the beginning of the subperiod and who could have survived until the end of the subperiod.[6] With a very large sample, one could move through the first year by weeks (indeed by days if it would serve a purpose). With our sample size, we have managed only a much more aggregated scheme by disaggregating the most in the first month of life, where we treat separately the first week and the remainder of the first month. Thereafter, we aggregate and examine the probabilities of infant deaths in two periods: months 2 through 6, and months 7 through 12. This disaggregation is probably compatible with the speed of change in the structure of mortality determinants. Nonetheless, coefficient estimates in these data sometimes move erratically from period to period during infancy, suggesting more the dominance of small sample variations (in terms of number of deaths) than a changing underlying structure of mortality

[6] For example, in the regression explaining mortality in the seventh through twelfth months of infancy, we exclude from the sample babies born in the twelve months prior to the survey.

determinants. Accordingly, one must at points judge whether to believe an isolated significant estimate or a deviation from a broader pattern. Our criteria are straightforward: We place more credence in a coefficient pattern if it changes smoothly across infant age groups, if it reflects a biological or behavioral mechanism known or suspected in similar populations, and if it emerges also in the regressions explaining all of infant mortality, where the number of deaths is largest. In most instances the patterns are clear, but in some we and the reader must judge.

Some of the variables considered below show a declining association with mortality over the course of infancy. There are two possible biological interpretations for this pattern. One, the phenomenon of sample heterogeneity,[7] is that infants most susceptible to a fatal factor die early leaving the less susceptible infants to survive. The other interpretation is that resistance to the detrimental factor increases with age. It is unfortunately impossible to differentiate statistically between these interpretations

Since the observations are live births, there is frequently more than one observation per mother. This creates the possibility that influences on different infants born to the same mother may be correlated. We partially control for this by including variables specific to the infant and variables specific to the mother or household. However, unmeasured mother-specific influences may remain. We ignore this problem because computer programs for treating it when the dependent variable is dichotomous are as yet unavailable. The

[7] Olsen and Wolpin (1981), in a separate analysis of mortality in the MFLS, attempt to account empirically for the effects of sample heterogeneity. Their restriction to a subsample of Malay children and many other differences make comparisons with our findings difficult.

resulting coefficient estimates reported below are unbiased on this account, but the reported standard errors may be smaller than is actually the case. Hence, the true precision of estimates may be less than is reported here. This is probably more true for family variables (for example, stable maternal characteristics like schooling level) than for child characteristics, since the former are likely to be more highly correlated across infants in the same family.[8]

ESTIMATING THE EFFECT OF BREASTFEEDING ON MORTALITY IN THE FIRST YEAR OF LIFE

One of the key mortality determinants of interest here is breastfeeding. Estimating this relationship is complicated by three special considerations.

- Variations in breastfeeding that occur after an at-risk period cannot affect mortality during that period, and should not be used in regression analyses of that mortality.
- If a child dies before supplementation or weaning, breastfeeding is short because the child died rather than vice versa.

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[8] Variance-components estimation makes a larger difference the greater the proportion of variation in the dependent variable that arises from within-family error components relative to across-family errors, and the larger the groups (families) into which observations (live births and infant deaths) are clustered. In a variance-components analysis of length of breastfeeding in these data (Butz and DaVanzo, 1981), the estimated value of ρ, the proportion of the total residual variance due to the family-specific component, is between .48 and .58. A variance-components analysis of birthweight in these data (DaVanzo, Habicht, and Butz, 1981) estimates ρ at around .33. Several considerations would indicate a value near the lower figure in the present case of infant mortality.

- A third factor, say illness, may cause the child both to stop breastfeeding and, frequently with a lag, to die. The feeding change does not cause the death.

We handle these complications in the following manner for our analyses of the subperiods of infancy. We truncate the length of total breastfeeding (full plus supplemented) to be no longer than the start of the period at risk. In the regressions for 8-28 days, for example, children who breastfed longer than seven days are given the value of seven days. Otherwise children who died before the end of the 28-day period would necessarily have breastfeeding shorter than the maximum possible for children who survived the entire period. Similarly the weaning/supplemental-milk variables apply only if the child stopped full (unsupplemented) breastfeeding before the beginning of the at-risk period. This procedure of using previous breastfeeding to explain subsequent mortality is comparable to the method used by Scrimshaw et al. (1968) and by Plank and Milanesi (1973). However, it amounts to assuming that how the infant was fed on days 8 and after does not affect its mortality through day 28. The assumption may be considerably more extreme for the 2-6 and 7-12 month samples, where we are unable to estimate the effects of breastfeeding and choice of first supplemental food after four weeks or after six months of age, respectively, on the infant's subsequent survival probability.

In addition to this truncation we also exclude from each particular subsample a few infants (14 in all) who died in the at-risk interval under consideration and whose length of truncated breastfeeding was more than 87.5 percent of their length of life. These infants, who had to

have died near the start of a particular at-risk period, are excluded from our analysis because it is very likely that the same condition both caused them to stop breastfeeding and then killed them. Their cessation of breastfeeding was probably not responsible for their deaths [9]

Because we cannot truncate to the beginning of the period for infant mortality as a whole, we use a different method to examine the effects of breastfeeding over the whole of infancy. Length of total breastfeeding (full plus partial) is truncated at 12 months for each infant who in fact breastfed longer. For dead infants who breastfed 87.5 percent or more of their lives (i.e., who were still breastfeeding shortly before they died) the amount of time they would have breastfed had they survived was assigned from breastfeeding expectancies computed from infants who breastfed less than 87.5 percent of their lives.[10] These breastfeeding expectancies, analogous to life-expectancies in a life table, are unbiased estimates of how much longer an infant would breastfeed (up to one year) conditional on the length of breastfeeding when it was interrupted.[11] For children who lived less than one week

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[9] These data do not include the morbidity information required to directly identify babies who both ceased breastfeeding and subsequently died because of a third cause. However, examination of the data indicates that, in addition to the infants whose duration of breastfeeding was the same as their length of life, another substantial proportion stopped breastfeeding only shortly before they died. The derivation of the 87.5 percent level is discussed in Section IV.

[10] We also assigned expected values to a small number of infants whose mothers reported that they did not breastfeed or stopped breastfeeding because the child was dying. This information is only available for children who breastfed for three months or less.

[11] For dead infants who fully breastfed 87.5 percent or more of their lives and stopped at time t (at or just before dying), we impute length of breastfeeding from the sample of infants who full breastfed at least t months but breastfed less than 87.5 percent of their lives. The imputation is the average length of full and partial breastfeeding for these surviving infants. For infants who breastfed 87.5 percent or more of their lives and were partial breastfeeding when they stopped at time

and did not initiate breastfeeding, we assign the unconditional means of full and partial breastfeeding of all surviving infants in the sample. This procedure assumes that these infants did not breastfeed because they had no chance. These imputed breastfeeding lengths are thus identical to those of the surviving children whose breastfeeding was neither prohibited at initiation nor interrupted; therefore this procedure will not bias the estimates of weaning's effect on survival.[12]

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t, we use the sample of infants who breastfed less than 87.5 percent of their lives, and breastfed at least t months, but full breastfed less than t months. For these babies we compute their average length of partial breastfeeding as the difference between the imputed total breastfeeding and their actual value of full breastfeeding.

[12] An alternative way to avoid including the effect of death on duration of breastfeeding in estimating the effect of breastfeeding on death would be to exclude from the analysis those infants whose breastfeeding was interrupted by death and its antecedents. However, this method would inflate the estimated association between breastfeeding and death by excluding those infants who were breastfeeding when they died, hence underestimating the mortality rates of longer breastfeeders.

III. EMPIRICAL RESULTS

Tables 1 through 9 present the linear probability model estimates that are the focus of this paper. In Tables 1 through 4, the first column contains coefficient estimates for deaths of live-born infants during the first week postpartum. Column 2 shows estimates of determinants of deaths during the remainder of the first 28 days for those who survived the first week, while columns 3 and 4 concern mortality during months 2 through 6 and months 7 through 12. Column 5 presents estimates for infant mortality--the death of a live-born infant at any time during the first 12 months.

Each table is designed to highlight the effects of a different set, or combination of sets, of explanatory factors. Table 1 contains factors usually considered in the literature to be the most proximate correlates of mortality: biological attributes at birth, such as the baby's birthweight, sex, and birth order. Table 2 adds to the variables in Table 1 various biological influences that can directly affect infant mortality through known mechanisms, but in contrast to those in Table 1 are postnatal and can be controlled by parental behavior; duration of breastfeeding is an example. Tables 3 and 4 examine the influences of indirect, less proximate, family characteristics such as mother's schooling, household income, and ethnicity. We first report their total estimated influences in Table 3, where we omit from the regressions the more direct and proximate determinants in Tables 1 and 2 through which they might work. Then, in Table 4, we control the variables in Tables 1

Table 1

CHANGES IN THE INFLUENCE OF MORTALITY DETERMINANTS DURING THE FIRST YEAR OF LIFE: BIOLOGICAL ATTRIBUTES AT BIRTH

(Dependent variable = Dummy that equals one if infant died during indicated period; method of estimation = OLS; t-statistics are in parentheses beneath coefficients)

Explanatory Variable	Mortality in				
	First Week (n = 5573 live births)	Days 8-28 (n = 5471 survivors of 1st week)	Mos. 2-6 (n = 5345 survivors of 1st 4 weeks)	Mos. 7-12 (n = 5152 survivors of 6th month)	First 12 Mos. of Life (n = 5357 live births)
Maternal Characteristics					
Age spline (yrs.)					
≤ 18	-.0162 (-4.54)	-.00420 (-1.84)	-.00301 (-0.79)	-.00125 (-0.41)	-.0220 (-3.60)
18-40	.00000 (0.00)	-.00050 (-1.70)	-.00082 (-2.70)	-.00058 (-1.48)	-.00215 (-2.66)
>40	.00512 (0.89)	.00047 (0.13)	-.00316 (-0.53)	.00744 (1.43)	.0113 (1.08)
Proportion other interpregnancy intervals < 15 mos.	.0235 (2.91)	.00324 (0.63)	.00549 (0.65)	-.0141 (-2.06)	.0103 (0.73)
Proportion stillbirths of all births	.0262 (1.26)	.0258 (1.94)	.0405 (1.84)	-.0222 (-1.24)	.0644 (1.80)
Preceding interpregnancy interval < 15 mos. (D)	.00790 (1.60)	.00677 (2.17)	.0204 (3.94)	.00548 (1.31)	.0382 (4.46)
Child Characteristics					
Sex = male (D)	.00821 (2.48)	.00045 (0.22)	.0175 (5.08)	.00600 (2.17)	.0324 (5.63)
Birthweight spline (kg.)					
≤ 2 kg.	-.316 (-8.10)	-.197 (-6.81)	.0455 (0.86)	.0335 (0.77)	-.259 (-3.41)
2 - 2.5 kg.	-.0259 (-1.17)	-.00080 (-0.06)	-.0913 (-3.77)	-.00138 (-0.07)	-.150 (-3.84)
2.5 - 3.5 kg.	-.00219 (-0.35)	-.00829 (-2.09)	-.0124 (-1.89)	-.00988 (-1.88)	-.0311 (-2.85)
> 3.5 kg.	-.00154 (-0.18)	.00177 (0.33)	-.00592 (-0.67)	.0116 (1.65)	.00416 (0.28)
Birth order	-.00085 (-0.85)	.00020 (0.33)	.00150 (1.44)	.00169 (2.02)	.00263 (1.50)
Intercept	.944 (9.68)	.481 (7.02)	.0179 (0.15)	-.0329 (-0.33)	1.03 (5.68)
R^2	.0289	.0193	.0174	.0044	.0347
Mean Dependent Variable	.0158	.0060	.0163	.0099	.0478

Notes: D = Dummy

The samples exclude live births occurring closer to the date of interview than the end of the period at risk of mortality; e.g., babies born four months before the interview are not included in the 2-6 month (or later) samples, but are included in earlier samples.

Table 2

CHANGES IN THE INFLUENCE OF MORTALITY DETERMINANTS
DURING THE FIRST YEAR OF LIFE: POSTNATAL BIOLOGICAL INFLUENCES

(Equations also control the biological attributes at birth in Table 1, but these coefficients are not presented here)

Explanatory Variable	Mortality in				
	First Week	Days 8-28	Mos. 2-6	Mos. 7-12	First 12 Mos.
Breastfeeding[a]					
Mos. unsupplemented breastfeeding	na	-.0676 (-5.04)	-.0203 (-3.53)	-.00302 (-3.35)	-.00535 (-4.73)
Mos. supplemented breastfeeding	na	-.0243 (-1.64)	-.00701 (-1.27)	-.00103 (-1.63)	-.00215 (-3.31)
House Characteristics					
Piped water (D)	-.00054 (-0.15)	-.00500 (-2.18)	-.00128 (-0.34)	-.00743 (-2.43)	-.0129 (-2.02)
Toilet (D)	-.0102 (-2.34)	-.00146 (-0.53)	-.0165 (-3.59)	-.0105 (-2.83)	-.0421 (-5.51)
Density (persons/room)	-.00097 (-0.99)	.00171 (2.79)	.00023 (0.23)	.00039 (0.47)	.00167 [illegible]
Year of Child's Birth (e.g., 65 for 1965)	-.00047 (-1.91)	-.00003 (-0.20)	-.00075 (-2.83)	-.00091 (-4.16)	-.00228 (-5.05)
R^2	.0306	.0266	.0238	.0132	.0506

NOTE: D = dummy.

a = For the regressions in columns (2)-(4), the breastfeeding variables measure the durations of unsupplemented and supplemented breastfeeding before the beginning of the interval. For more detail on this and the measurement of breastfeeding for the analysis of infant mortality, see pp. 11-14.

Table 3

CHANGES IN THE INFLUENCE OF MORTALITY DETERMINANTS DURING THE FIRST YEAR OF LIFE: EFFECTS OF INDIRECT FAMILY INFLUENCES EXCLUDING THE BIOLOGICAL ATTRIBUTES AT BIRTH AND POSTNATAL BIOLOGICAL INFLUENCES THROUGH WHICH THEY MAY OPERATE

Explanatory Variable	Mortality in				
	First Week	Days 8-28	Mos. 2-6	Mos. 7-12	First 12 Mos. of life
Indirect Family Influences					
Mother's education (yrs.)	-.00067 (-1.09)	.00009 (0.24)	-.00009 (-0.14)	-.00139 (-2.71)	-.00137 (-1.83)
Household's income (log average household income per adult)	-.00338 (-1.34)	-.00033 (-0.21)	.00032 (0.12)	-.00171 (-0.82)	-.00494 (-1.13)
Household composition					
Number children less than 2 years old	-.00106 (-0.30)	.00364 (1.65)	.00785 (2.15)	.00496 (1.70)	.0147 (2.40)
Number grandparents	.00069 (0.28)	-.00072 (-0.46)	-.00214 (-0.83)	-.00102 (-0.50)	-.00288 (-0.67)
Number other non-nuclear-family relatives	-.00049 (-0.39)	-.00099 (-1.25)	.00025 (0.19)	-.00069 (-0.67)	-.00167 (-0.77)
Child born in hospital (D)	-.00698 (-1.23)	.00328 (0.92)	-.0104 (-1.75)	-.00383 (-0.80)	-.0158 (-1.57)
Indirect or Direct Influences Outside Family's Control					
Ethnicity					
Chinese (D)	-.00992 (-2.49)	-.00177 (-0.71)	-.0181 (-4.41)	-.00831 (-2.54)	-.0392 (-5.69)
Indian (D)	.00225 (0.42)	.00190 (0.56)	-.00109 (-0.19)	-.0107 (-2.39)	-.0101 (-1.09)
Sex of child = male (D)	.00863 (2.59)	.00003 (0.02)	.0169 (4.90)	.00567 (2.06)	.0310 (5.36)
Year of child's birth	-.00056 (-2.40)	-.00023 (-1.59)	-.00068 (-2.76)	-.00048 (-2.42)	-.00203 (-4.92)
Intercept	.0767 (3.22)	.0234 (1.56)	.0537 (2.16)	.0584 (2.92)	.216 (5.19)
R^2	.0071	.0021	.0134	.0094	.0247

NOTE: D = dummy.

Table 4

CHANGES IN THE INFLUENCE OF MORTALITY DETERMINANTS DURING THE FIRST YEAR OF LIFE: ALL DIRECT AND INDIRECT INFLUENCES

Explanatory Variable	Mortality in First Week	Days 8-28	Mos. 2-6	Mos. 7-12	First 12 Mos. of Life OLS	Logit
BIOLOGICAL ATTRIBUTES AT BIRTH						
Maternal Characteristics						
Age spline (yrs.)						
≤ 18	-.0123 (-3.27)	-.00485 (-2.00)	.00200 (0.50)	.00268 (0.83)	-.0101 (-1.57)	-.0481 (-0.45)
18-40	.00065 (1.23)	-.00058 (-1.75)	.00011 (0.19)	.00027 (0.62)	.00034 (0.37)	.00553 (0.24)
>40	.00041 (0.71)	.00147 (0.40)	-.0029 (-0.49)	.00722 (1.43)	.0122 (1.16)	.257 (1.18)
Proportion other interpregnancy intervals < 15 mos.	.0286 (3.47)	.00274 (0.53)	.0113 (1.31)	-.0117 (-1.67)	.0166 (1.15)	.413 (1.25)
Proportion stillbirths of all births	.0197 (0.93)	.0300 (2.24)	.0364 (1.63)	-.0204 (-1.13)	.0671 1.88	.856 (1.52)
Preceding interpregnancy interval < 15 mos. (D)	.0113 (2.12)	.00495 (1.47)	.0203 (3.65)	.00447 (0.99)	.0359 (3.90)	.648 (3.47)
Child Characteristics						
Sex = male (D)	.00856 (2.59)	.00021 (0.10)	.0174 (5.07)	.00604 (2.19)	.0324 (5.68)	.770 (5.44)
Birthweight spline (kg.)						
≤2 kg.	-.322 (-8.23)	-.190 (-6.56)	.0467 (0.88)	.0308 (0.71)	-.275 (-3.66)	-2.20 (-2.44)
2-2.5 kg.	-.0225 (-1.01)	-.00278 (-0.19)	-.0884 (-3.66)	-.00169 (-0.09)	-.143 (-3.70)	-1.57 (-2.55)
2.5-3.5 kg.	-.00219 (-0.34)	-.00843 (-2.11)	-.0123 (-1.87)	-.00989 (-1.86)	-.0297 (-2.71)	-.873 (-3.29)
>3.5 kg.	-.00357 (-0.42)	.00116 (0.22)	-.00866 (-0.98)	.00988 (1.40)	-.0011 (-0.07)	.0706 (0.18)
Birth order	-.00157 (-1.33)	-.00080 (-1.07)	.00010 (0.08)	.00027 (0.27)	-.00211 (-1.02)	-.0572 (-1.15)

Table 4 -- continued

CHANGES IN THE INFLUENCES OF MORTALITY DETERMINANTS DURING THE FIRST YEAR OF LIFE: ALL DIRECT AND INDIRECT INFLUENCES

Explanatory Variable	Mortality in First Week	Days 8-28	Mos. 2-6	Mos. 7-12	First 12 Mos. of Life OLS	Logit
POSTNATAL BIOLOGICAL INFLUENCES						
Breastfeeding						
Mos. unsupplemented breastfeeding	N.A.	-.0686 (-4.89)	-.0249 (-4.23)	-.00343 (-3.74)	-.00618 (-5.42)	-.239 (-4.46)
Mos. supplemented breastfeeding	N.A.	-.0219 (-1.44)	-.0112 (-1.99)	-.00166 (-2.47)	-.00383 (-5.49)	-.0806 (-5.14)
House characteristics						
Piped water (D)	.00522 (1.30)	-.00623 (-2.46)	.00028 (0.07)	-.00324 (-0.96)	-.00313 (-0.45)	-.0887 (-0.51)
Toilet (D)	-.00467 (-1.02)	-.00125 (-0.43)	-.0113 (-2.35)	-.00715 (-1.84)	-.0288 (-3.64)	-.425 (-2.65)
Density (persons/rm.)	-.00151 (-1.49)	.00216 (3.39)	-.00041 (-0.38)	.00013 (0.16)	.00081 (0.46)	.0176 (0.54)
Year of Child's Birth	-.00039 (-1.42)	-.00001 (-0.08)	-.00097 (-3.32)	-.00082 (-3.46)	-.00229 (-4.68)	-.0444 (-3.94)
INDIRECT FAMILY INFLUENCES						
Mother's education (yrs.)	-.00075 (-1.10)	.00005 (0.11)	.00008 (0.11)	-.00089 (-1.55)	-.002 (-1.68)	-.0707 (-2.18)
Household income	-.00310 (-1.18)	.00183 (1.10)	.00063 (0.23)	-.00221 (-1.00)	-.00204 (-0.45)	-.0487 (-0.43)
Household composition No. children less than 2 yrs. old	-.00293 (-0.76)	.00213 (0.88)	.00239 (0.60)	.00330 (1.02)	.00233 (0.35)	.0657 (0.43)
No. grandparents	-.00053 (-0.21)	-.00219 (-1.40)	-.00325 (-1.25)	-.00175 (-0.84)	-.00720 (-1.68)	-.164 (-1.63)
No. other non-nuclear family relatives	-.00050 (-1.36)	-.00161 (-2.03)	.00035 (0.27)	-.00049 (-0.47)	-.00198 (-0.91)	-.0588 (-0.93)
Child born in hospital (D)	-.00764 (-1.36)	.00312 (0.88)	-.0118 (-1.99)	-.00402 (-0.83)	-.0193 (-1.93)	-.648 (-2.28)
Ethnicity Chinese (D)	-.0108 (-2.43)	.00064 (0.22)	-.0193 (-4.10)	-.00807 (-2.10)	-.0418 (-5.22)	-1.18 (-5.46)
Indian (D)	-.00826 (-1.41)	.00005 (0.01)	-.00891 (-1.46)	-.00987 (-2.01)	-.0319 (-3.15)	-.652 (-2.86)
COMMUNITY INFLUENCES						
Rurality (scale: 10 = most urban, 40 = most rural)	.00020 (1.12)	-.00001 (-0.11)	-.00026 (-1.41)	-.00011 (-0.75)	.000019 (0.06)	-.00173 (-0.23)
INTERCEPT	.939 (9.21)	.481 (6.80)	.0193 (0.15)	-.0100 (-0.10)	1.09 (5.84)	7.65 (2.83)
R^2	.0342	.0300	.0298	.0163	.0606	-
χ^2	-	-	-	-	-	297.

and 2 to inspect how these indirect influences change when the more direct ones are controlled.

Although conceptually useful, the division of variables into biological attributes at birth, postnatal biological influences, and indirect family influences is necessarily arbitrary. For example, a preceding short interval, which appears in Table 1 as a biological attribute at birth, may affect the risk of mortality through both biological and behavioral mechanisms. Below we discuss these ambiguities where they arise and attempt, whenever possible, to separate the biological and behavioral influences empirically.

Section IV examines in more detail the influences of breastfeeding and use of other infant foods on mortality, as well as the interacting effects of breastfeeding and type of household water and sanitation.

The large amount of empirical information in Tables 1 through 4 lends itself most naturally to two modes of organizing a discussion. One is to describe the changing structures of mortality determinants during the first year of life. The other is to describe the relationships between more proximate and less proximate determinants of mortality, thereby elucidating the direct and indirect roles of particular characteristics of infants and families. The first mode calls for comparisons within tables, the second for comparisons across tables. We attempt a compromise, describing the key features within successive tables but not hesitating to compare estimates across tables. The discussion assumes that hypotheses are subject to one-tail tests and applies a five-percent level of significance.

BIOLOGICAL ATTRIBUTES AT BIRTH

Maternal Age

The three splined variables for mother's age allow very young mothers (<18) and very old ones (>40) to exhibit different infant mortality risks from those in the more prime childbearing ages. The relationships estimated by the spline functions in Table 1 are graphed in Fig. 2 as solid lines.[13]

The last column in Table 1 and panel (e) of Fig. 2 suggest that infants born to very young mothers face significantly elevated risk of infant mortality. This is widely recognized in the literature (United Nations, 1973; Puffer and Serrano, 1975; Chase, 1972; Nortman, 1974; Deschamps and Valantin, 1978). The linear probability of death falls by 22 deaths per thousand for each additional year of mother's age in this range (before age 18), above and beyond any effect of maternal age on birthweight (DaVanzo, Habicht, and Butz, 1981), birth order, and the other biological influences taken into account in Table 1. Risk of infant mortality also falls significantly between ages 18 and 40, but much less steeply than before age 18. Mortality risk may then rise with increasing maternal age for very old mothers, though the Age >40 slope is not significantly different from zero.

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[13] A spline allows one to estimate connected linear segments (in Table 1, three in the case of age) as an approximation to a nonlinear function. Each coefficient is the slope of that particular linear segment. Each splined variable in this analysis was chosen from more general specifications in order to reflect the particular nonlinearities that characterize that variable's association with mortality. The graphs in Fig. 2 show mortality differentials associated with young and old maternal age relative to mean maternal age; i.e., the mortality differential is arbitrarily set to zero at mean maternal age (25).

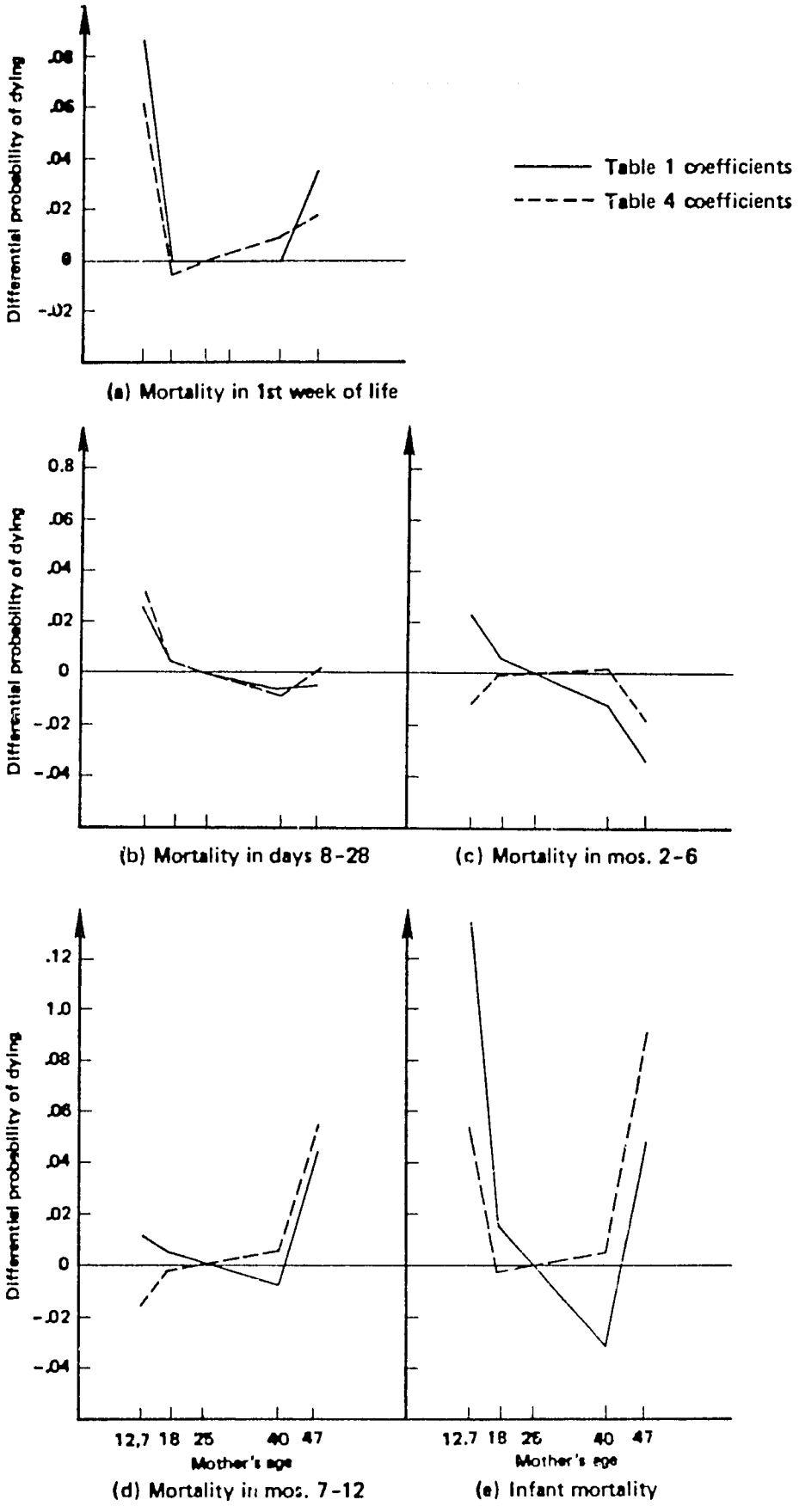

Fig. 2 – Maternal-age splines for each subperiod of first year of life

The value of examining the changing structure of mortality determinants during subperiods of infancy is first revealed in Table 1 by the clear indication that the sharp decrease in mortality risk with age among very young mothers is entirely concentrated in the first month of life. Thereafter, both young and prime-age mothers show an equal improvement in infant survival with increasing maternal age, which is most marked and statistically significant betweeen the second and sixth months of infancy. Older mothers show an increased risk of infant death during the first week of life (albeit not statistically significant), as is well recognized in the literature (Nortman, 1974; Puffer and Serrano, 1975). Babies born to older mothers also have higher mortality in the second half of infancy. This result, which is significant at the 10-percent level, has not been previously reported, perhaps because deaths during the second half of infancy are usually not separately identified.

Examining the age patterns in Table 4, shown in dashed lines in Fig. 2, reveals the degree to which the maternal age effects in Table 1 are due in fact to associated socioeconomic and behavioral variables. The first month' mortality of babies born to young mothers is not much affected. Beyond the first month, improvements in infant survival with increasing maternal age among prime-aged mothers (18 to 40 years) are much less in Table 4 than in Table 1 (in fact, the coefficients are positive, though insignificant). These changes suggest that these age effects are probably mediated by behavioral factors, perhaps associated with mother's increasing experience with infant care.

The poor survival of babies born to older mothers during the last half of infancy is little changed in Table 4. There is, however, little theoretical basis for believing that the poor survival in months 7-12 of babies born to older mothers is due to purely biological reasons. It might arise from a higher incidence of unwanted births at these ages.[14] This behavioral interpretation is reinforced by the fact that, where sociocultural influences are favorable, high maternal age, even coupled with high parity, favors total infant survival (Voorhoeve, Muller, and W'Oigo, 1979). The behavioral factors in question, however, are apparently not captured by the additional variables in Table 4.

Factors Associated with Short Interpregnancy Intervals

The common finding that a short interpregnancy interval preceding a child's birth is associated with higher mortality risk is difficult to interpret. It could be due to (a) gestational prematurity (reviewed in DaVanzo, Habicht, and Butz, 1981), (b) nutritional depletion of the mother (reviewed in DaVanzo, Habicht, and Butz, 1981), (c) competition of a previous young infant for the mother's attention,[15] (d) other factors associated with less adequate child care, including shortened

[14] We attempted to investigate this directly by using survey information on the number of additional children desired and the number that would be desired if the woman could begin her married life again. This indicator of a child's "being unwanted" is highly correlated with its mortality experience. We feel that most of the statistically highly significant ($p < .001$) correlation between the two is spurious rather than behavioral, due the to the fact that many mothers reported that they wanted the number of living children they had at the time of the survey.

[15] This behavioral hypothesis would apply only if the child born at the beginning of the interval survived. We separately examine these hypotheses in Tables 3 and 4 in which we control for the number of living children less than two years old.

breastfeeding and a consequent rapid cessasation of postpartum sterility (Habicht et al., 1981), (e) an overall tendency of the mother to have short birth intervals (Bakketig, Hoffman, and Harley, 1979), which may result in her becoming pregnant soon after the birth in question and hence may have detrimental effects on breastfeeding the child in question.

In Table 1 we attempt to proxy recurrent gestational prematurity by two variables: the proportion of the woman's other interpregnancy intervals that are less than 15 months[16] and the proportion of her births that are stillbirths. These proxies will identify women who have recurrent gestational premature babies (Terris and Gold, 1969) and perinatal deaths (Schlesinger et al., 1972). We would expect these proxies to pick up a significant proportion of first week and other neonatal (first month) deaths because gestational prematurity should be most pernicious during the first month of life. Indeed, this appears to be the case for "proportion other interpregnancy intervals < 15 months," whose influence is only significant in the first week of life, indicating that many deaths associated with short intervals are probably due to prematurity, as was observed by Terris and Gold (1969) in New York City. This fact is now appreciated in the medical literature (Wray, 1971; Wolfers and Scrimshaw, 1975; Fedrick and Adelstein, 1973), but is sometimes overlooked in the family planning literature (e.g. Bhalla et al., 1974).

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[16] The proportion variable excludes the intervals preceding and following the birth under consideration. The former is included as a separate variable. The latter is excluded from the proportion variable because this interval may be short following a child's death (due to replacement or shorter breastfeeding). Hence its inclusion could cause simultaneity bias.

"Proportion of stillbirths" has less of an immediate effect than expected from the literature (Schlesinger et al., 1972) and more of a protracted effect up to six months than would be expected if it is indeed a proxy for gestational prematurity. The lower immediate effect might be due to the fact that repeated gestational prematurities are controlled for by the variable, "proportion other interpregnancy intervals < 15 months," in this analysis. However, Bross and Shapiro (1982) find in U.S. data a significant effect of prior fetal deaths on postneonatal deaths but not on neonatal deaths, after accounting for birthweight but not for gestational age. Thus, proportion of stillbirths may not be a good proxy for prematurity, but may reflect some other longer lasting pernicious influence on the infant.

Finally, we include the dummy indicating whether the interpregnancy interval preceding the birth under consideration was short (< 15 months). Even when the just-discussed proxies of recurrent premature births are taken into account, there remains a detrimental effect of the interval preceding the birth in question being short. This effect extends far longer than would be expected if it were due to gestational prematurity and suggests that some of the other factors mentioned above (b through e on p. 25) may be the cause.

When other behavioral influences are controlled (Table 4), both short-interval variables are more significant in the first week of life than they were in Table 1. This pattern suggests that the behavioral and environmental factors included in Table 4 mitigate the effects of prematurity when these factors are not controlled in Table 1.

The relationship between birthspacing and infant mortality presented in Table 1 holds birthweight constant. However, length of previous interval is associated with infant mortality through intermediating variations in birthweight. This occurs because a preceding short interval is an important correlate of low birthweight in this sample (DaVanzo, Habicht, and Butz, 1981). Thus, when birthweight is not controlled (not shown), the detrimental influence of a preceding short interval is even larger than in Table 1, especially for mortality in the first week. This is compatible with both the prematurity hypothesis already discussed and with the maternal depletion hypothesis. DaVanzo, Habicht, and Butz (1981) do find evidence of a maternal depletion effect on birthweight among poor women in this sample.

Sex of Child

Male infants show higher mortality risk throughout their first year, especially in the second to sixth months. Higher male infant mortality is a nearly universal finding, except where girls are discriminated against (United Nations, 1973). The higher mortality of boys is generally ascribed to biological mechanisms.

Birthweight

By far the most important correlate of mortality in Table 1 is birthweight. This is a universal finding (reviewed by United Nations, 1973; and DaVanzo, Habicht, and Butz, 1981) even when the effect of gestational age is accounted for (as is attempted here by controlling for short pregnancy intervals and recurrent stillbirths).

Our regressions include birthweight as a spline function that allows for different slopes over the range of birthweight. The relationships estimated by these splines are graphed in Fig. 3 for infant mortality. Among very low birthweight babies, those weighing less than two kilograms, the linear probability of mortality in the first week falls 31.6 deaths per thousand births with each additional 100 grams of birthweight. In the second through fourth weeks postpartum the decline in mortality risk is also substantial--19.7 deaths per thousand for every 100 grams of additional birthweight. Thereafter, extreme low birthweight appears to have no effect, probably because of small sample size.

Babies weighing between 2000 and 2500 grams at birth are also at higher mortality risk, but mostly in the second through sixth months. In that period, the heaviest babies in this range (2500 grams) face a mortality probability that is 46 per thousand lower than that faced by the smallest babies in this range (2000 grams). This is compatible with two possibilities. The first is that some of these low-birthweight babies were stunted because of intrauterine pathology from which they subsequently died. This seems unlikely because these infections should then be reflected in higher death rates at younger ages also. The other possibility is that these small neonates remained small infants and were less able to survive infectious illnesses in post-neonatal infancy (Mata, 1978).

This latter possibility may also explain why even babies within the "normal" birthweight range of 2500 to 3500 grams who survive their first week are more likely to survive the rest of their first year the heavier

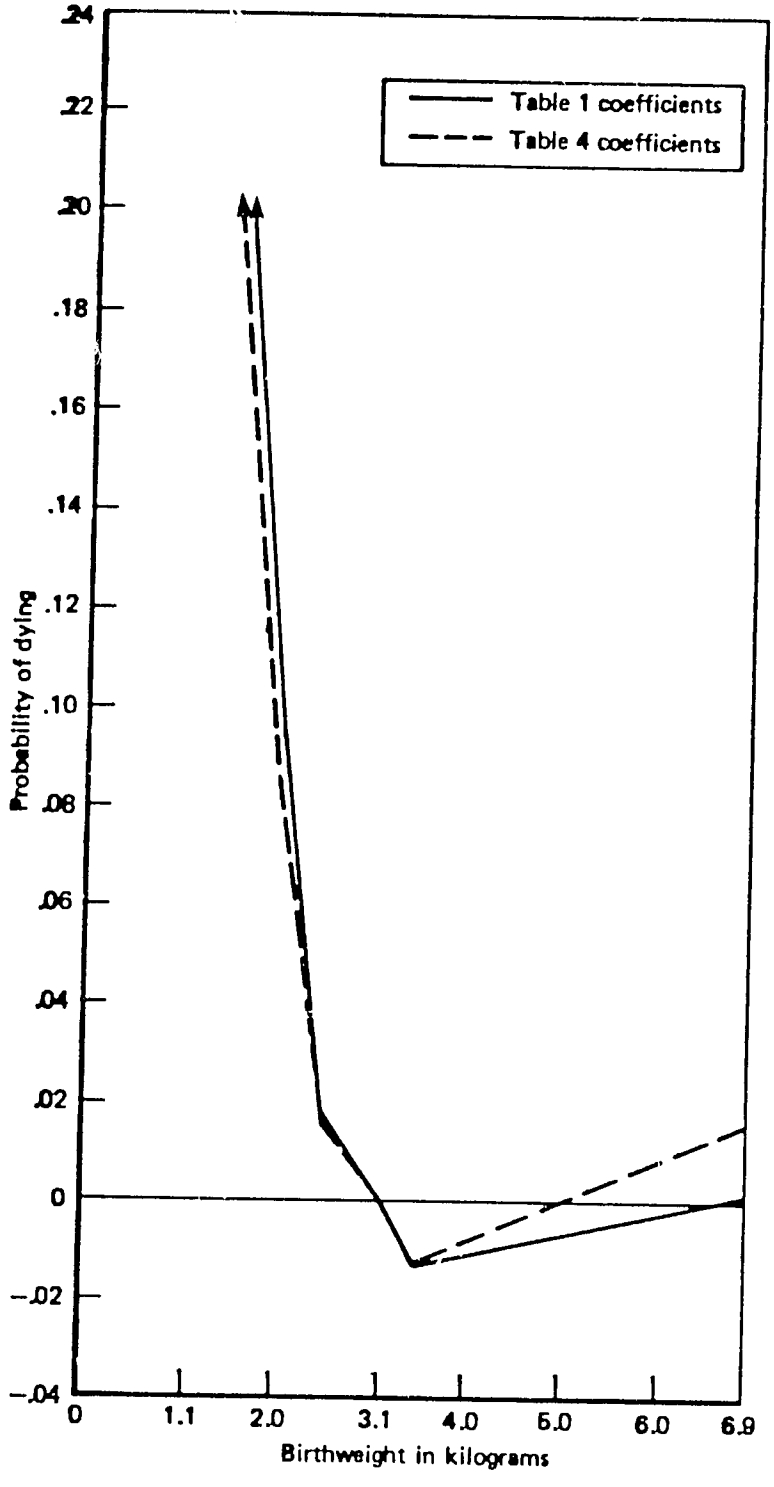

Fig. 3 – Birthweight splines for infant mortality

they were at birth. Their risk gradient is fairly constant over the year but much flatter than among babies weighing less than 2500 grams at birth.

Comparing Table 1 with Table 4 indicates that taking behavioral and environmental factors into account does not affect the relationship between birthweight and mortality.

Birth Order

In these data the child's birth order is unrelated to its mortality in the first six months of life when the mother's age and the child's weight at birth are controlled (Table 1).[17] Without these controls, first-born babies appear to face elevated risk in the first month of life (not shown), as is generally found (United Nations, 1973), even in developed countries (Feldstein and Butler, 1965). In these data, this association is due both to the younger age of mother and lower birthweight (DaVanzo, Habicht, and Butz, 1981) of first-borns, and hence vanishes when these variables are controlled.

When only biological attributes at birth are controlled (Table 1), higher-order births that survive the first six months are more likely to die in the next six months than are babies of lower birth order. Others also have found higher infant mortality with higher birth order (United Nations, 1973). In our data the birth order effect attains statistical significance only for the 7-12 month subperiod. However, this detrimental effect of high birth order disappears entirely when the

[17] We experimented with various nonlinear specifications that allowed estimation of mortality differentials for first order and high order (>7) births, in addition to a linear relationship. When mother's age and child's birthweight are controlled, only the linear term was ever statistically significant.

behavioral and other biological variables in Table 4 are controlled.[18] That the birth-order effects in Table 1 become stronger in later periods of life and disappear when behavioral variables are controlled suggest that the birth-order influences in Table 1 are primarily behavioral.

Hence, higher death rates of higher-order births appear due to pernicious behavioral factors, while higher death rates of first-order births are due to detrimental biological influences. We placed birth order in Table 1 as a biological attribute at birth because it is usually interpreted in the literature as influencing mortality through biological mechanisms. Our results suggest that this interpretation should be reexamined.

POSTNATAL BIOLOGICAL INFLUENCES

The regressions presented in Table 2 add to the biological attributes at birth in Table 1 several postnatal biological factors, some of which are more subject to behavioral influences. These are the lengths of full (unsupplemented) and supplemented breastfeeding, whether the infant's house had piped water or a toilet sanitation system, and the number of persons per room in the house. Year of child's birth is also entered here as a proxy for public health changes not otherwise controlled. The coefficients on the biological attributes at birth are not shown in Table 2. They change very little from Table 1, as one would expect if they are indeed proximately related to mortality.

[18] In fact the association between birth order and mortality in the first month is negative, though insignificant, in Table 4. Analysis not shown indicates that this is due to a detrimental effect of first birth order.

Breastfeeding

Columns (2) through (4) of Table 2 present estimates of the association between prior lengths of breastfeeding (measured in months) and mortality in subperiods of infancy.[19] Because the breastfeeding variables refer only to practices before the beginning of the at-risk period, they are not relevant in explaining mortality in the first week of life and hence are excluded from column 1 of Table 2.

Infants who fully breastfeed (i.e., without supplementation) longer have substantially reduced mortality risk in this sample. The effect is greatest in the first month, with infants who fully breastfeed throughout their first week of life having 16 less deaths per thousand in days 8-28 of life than those who do not initiate full breastfeeding.

Infants who survive their first month likewise have reduced mortality risk in the next five months, the longer they full-breastfed in the first month. The estimated risk difference between breastfeeding the entire first four weeks and not breastfeeding at all is 20 deaths per thousand in this second-through-sixth month period. Finally, infants who survive their first six months continue to have better subsequent prospects the longer they full-breastfed in those first six months. In this period, the mortality difference between six months of full breastfeeding and none at all is 18 less deaths per thousand children alive at six months.

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[19] To obtain the mortality in days 8-28, for example, one multiplies the monthly mortality rate (regression coefficient) times the duration of breastfeeding in the seven days previous to the 8-28 day period. Thus children who full breastfed for the whole of the first week had 16 per thousand (= 0676 x (7/30 months)) less deaths during the rest of the month than did those who never breastfed during the first week of life.

Multiplying these coefficient estimates by .23 (weeks per month) indicates that the average beneficial effect of an additional week of full breastfeeding falls from 16 deaths per thousand for mortality in 8-28 days to 1.8 deaths per thousand for mortality in months six through twelve. This decline might reflect the result of heterogeneity in the changing samples of surviving infants: Those most likely to die for whatever reasons are missing from the later samples; those who remain may be less vulnerable to environmental insults, including those associated with feeding. More likely this pattern reflects an actual lessening in the importance of breastfeeding during the first year of life (Rowland et al., 1981).

Very long full breastfeeding (more than 6 months) can be detrimental to a child's health (Plank and Milanesi, 1973) for it does not provide enough energy to the older infant (Rowland et al., 1981). One would therefore expect to see the effect of full breastfeeding on mortality become positive in the last months of the first year, but our sample has an insufficient number of deaths in this period for such an investigation.

In contrast to these results for full breastfeeding, the estimated effects of supplemented (partial) breastfeeding on mortality do not reach statistical significance in the analyses of subperiods of infancy in Table 2. The differences between the full- and partial-breastfeeding coefficients are statistically significant in all subperiods. As with full breastfeeding, the effects of partial breastfeeding, though always insignificant, attenuate in size over the course of the first year of life.

In column 5 we present estimates of the effects of unsupplemented and supplemented breastfeeding on infant mortality as a whole. As discussed in Sec. II, durations of breastfeeding are measured differently for the analysis of overall infant mortality than they are for the analyses of subperiods of infancy. For infant mortality, the effects of both unsupplemented and supplemented breastfeeding are statistically significant. The latter is about 40% the magnitude of the former, and the difference between the two is statistically significant.

When additional influences on mortality are controlled in Table 4, the breastfeeding effects become stronger for mortality in months 2-6 and 7-12 and for infant mortality as a whole. This is especially true for partial breastfeeding. These results are consistent with the fact that Malaysian women who breastfeed more tend to have characteristics, e.g., less education, low household income, Malay ethnicity (Butz and DaVanzo, 1981), that are associated with higher infants' mortality, other things the same (see Table 4).

The above findings regarding the poor post-neonatal prognosis of those who never begin breastfeeding but survive the first week are similar to those reported from the Punjab (Scrimshaw et al., 1968). That report, however, did not take into account the fact that some of the association between mortality and initiation of breastfeeding may be because illness and death cut breastfeeding short. Consequently, that report overstated the effect of breastfeeding on neonatal mortality. A similar mistake, although partially recognized, was made in a report using data from Egypt (Janowitz et al., 1981).

The findings reported here are most comparable to those reported in a retrospective study by Plank and Milanesi (1973) for rural Chile. They also analyzed the effect of previous breastfeeding on subsequent mortality. They found a protective effect of 31.3 per thousand less deaths during the rest of infancy among those infants fully breastfeeding at one month of age; our comparable estimate is 28.2 per thousand less deaths (Table 4).[20] They found no protective effect (4.5 per thousand less deaths) from one month of partial breastfeeding, in contrast to our statistically significant estimate of 12.9 per thousand fewer deaths (Table 4).[21] Their estimate for those fully breastfeeding at six months was 9.9 per thousand less deaths in the rest of infancy compared with our 20.6 per thousand (Table 4: 6 months x .00343); for those partially breastfeeding at six months they found 5.9 per thousand less deaths compared with our statistically significant 10 per thousand.

In Section IV we investigate the relationships between infants' feeding and mortality and discuss their significance in considerably more detail.

Type of Water and Sanitation

Better sanitary conditions are often considered to have been a major cause of 19th Century health improvements in today's developed countries (McKeown, 1965). Yet, a recent review of intervention studies

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[20] This estimate derives from the following: -.0282 = -.0249 + (1-.0163) (-.0034), where -.0249 = coefficient of full breastfeeding in 2-6 month regression, .0163 = death rate in months 2-6, and -.0034 = coefficient of full breastfeeding in 7-12 month regression.

[21] -.0129 = -.0112 + (1-.0163)(-.0017).

in developing countries showed little benefit from improving water quality and quantity (Hughes, 1981). Unfortunately, most of these studies did not consider the educational, socioeconomic, nutritional, or medical case backgrounds of the recipients. When these factors are taken into account in the statistical analysis of cross-sectional data on populations, the presence of sanitation does seem to have a beneficial effect, especially during post-neonatal infancy (Meegma, 1980).

In our data 43 percent of the sample births occurred to households with piped water, some exclusive to the household and some shared with other households. Some of these systems consisted of interior piping and some of exterior standpipes. The other 57 percent of births occurred to households whose water came from wells, rivers, or canals. Preliminary analysis showed that the most important distinction for mortality in these data is between piped and non-piped water. This distinction is reported in Table 2. Infants born to households with piped water experienced significantly lower mortality in the first month and the last six months of the first year than did infants in houses using other water sources. Overall, in the first year, the difference in mortality is about 13 deaths per thousand live births, which is statistically significant, in contrast to a non-significant 5 deaths per thousand live births found in Kenya (Anker and Knowles, 1977).

Household toilet sanitation systems are reported in the MFLS to be either flush or nonflush and either exclusive to the household or not.[22] Households without toilets dispose either in pits, bushes, or

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[22] Because the relationship between sanitation and infant mortality is probably mediated through water quality, the sanitation variables are probably more appropriately measured at the community level, rather than the household level. This data set does contain

rivers. The gross distinction between a toilet system of any type and disposal of any other type is the most important distinction for mortality in these data. Seventy-nine percent of births in this sample occurred to households with toilet sanitation. These babies were significantly less likely to die throughout their first year, except in the last three weeks of the first month, when no significant difference emerges. The infant mortality rate of these babies is 42 deaths less per thousand live births than that of babies in houses without toilet sanitation. This is about double the effect described in Sri Lanka (Meegma, 1980) (statistically significant) and Kenya (Anker and Knowles, 1977) (statistically insignificant). Hence the type of sanitation emerges as a much more important influence on infants' mortality than source of water in these Malaysian data.[23]

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information on community prevalence of the different sanitation systems, but this information describes conditions only at the time of the survey, in 1976-1977. We attempted to use these variables for the 1046 births after 1970 that occurred in the same community in which the mother was interviewed. The community characteristics should be most accurate for these observations. However, the house-level sanitation variables give consistently stronger estimates. This may be because these community characteristics have changed rapidly and did not apply to births even a few years before the interview date. It may also be due to the small number of infant deaths in recent years (only 43 since 1970) compared with 256 deaths in the whole sample. We therefore use the house-specific data that are relevant for the time of each birth for the whole sample. In the year of the interview these are highly correlated with community prevalence of toilet systems.

[23] In preliminary analyses we also included an interaction term indicating that the house had both piped water and toilet sanitation, to allow for the possibility that the two effects may not be simply additive as in Table 2. This interaction was insignificant for all subsamples except 2-6 months, where the coefficient was positive and significant and nearly equal in absolute magnitude to the water coefficient. In this subsample, both toilet and piped water alone significantly reduce mortality, but the combination has no greater effect than just toilet alone. Hence, the addition of piped water once the household already has a toilet has no effect on mortality in the 2-6-month subsample. Adding a toilet once the household already has piped water causes a small, but statistically insignificant, reduction in mortality.

Because income and other socioeconomic characteristics of families are not controlled in Table 2, it may be that the association between these water and sanitation variables and infant mortality is due more to other factors associated with socioeconomic status than to fecal-borne disease. In Table 4, which includes socioeconomic variables, some of the estimated effects of water and sanitation do indeed weaken. For water, only the effect in days 8-28 remains significant. The effect seen in Table 2 for months 7 through 12 is apparently due instead to other socioeconomic influences. Although attenuated, the effects of toilet sanitation remain significant after the first month and for infant mortality as a whole in Table 4. Hence, babies born to households with toilet systems experience infant mortality rates 29 per thousand lower, even when a number of potentially confounding variables are controlled in Table 4.

We will see in the next section that the effects of water and sanitation on mortality depend markedly on the infant's breastfeeding experience.

House Density

Babies born into relatively crowded houses appear to experience higher risk of mortality in the last three weeks of the first month postpartum. This effect is small but statistically significant, with houses containing six persons per room having an 8-28 day mortality probability elevated by 8.5 deaths per thousand (.0017 x 5) compared with houses with one person per room. This effect is even somewhat stronger when potentially confounding socioeconomic variables are controlled in Table 4. The physiological rationale for this effect is,

however, not clear. Crowding is thought to adversely affect health and survival through increased contagion of respiratory diseases such as tuberculosis and pneumonia (Gorosomov, 1968) and through increased contact and fomite transfer of orally ingested pathogens (Wray, 1971). However, one would not expect these contagions to begin and cease only during the last three weeks of neonatal life.

Year of Child's Birth

The year of the child's birth is introduced in Table 2 as a proxy for other influences accompanying socioeconomic development, above and beyond those associated with factors presented in Table 1 and changes in breastfeeding, housing, and sanitation. The birth-year coefficients in Table 2 show significant declines in mortality over time for all at-risk intervals except the second through fourth week of life. Overall, the linear probability of infant mortality, as measured in these data, has fallen about 23 deaths per thousand per decade since 1945, when the effects of changes in the biological correlates of mortality are controlled. (The simple uncontrolled trends are shown in Fig. 1.) This decline is similar to that reported in the Malaysian Vital Statistics (Government of Malaysia, various years). The rate of decline remains the same when indirect family influences are also controlled (Table 4).

Across subperiods of the first year of life, the mortality decline is largest in the last eleven months of the first year of life. The rates of decline shown in Table 2 are generally unaffected when the other postnatal biological influences in Table 2 are not controlled (not shown) and are little changed when indirect family influences are also controlled (Table 4).

The declines over time in mortality in the first week of life may be due to improvements in delivery and neonatal care. The declines after the first month of life may be the result of improvements in family welfare and environmental conditions not identified elsewhere in Table 4. Apparently these latter improvements have not affected mortality in the first month of life.

INDIRECT FAMILY INFLUENCES

Table 3 documents the effects of several family-level factors that may be indirectly associated with infant mortality. Many socioeconomic or demographic analyses of infant mortality consider only these types of factors, since they lack data on some of the more proximate influences examined here. To assess the total effects of these indirect factors, the regressions in Table 3 do not control for the more proximate variables through which these indirect factors might operate. Table 4 then admits these controls--the same variables in the regressions of Tables 1 and 2--to distinguish those indirect influences that do work through more proximate causes from those that have independent influences.

Mother's Education

Education of parents, especially of the mother, is a well-established correlate of infant mortality everywhere (Bairagi, 1980; United Nations, 1973; Cochrane et al., 1980; Chase, 1973) but not necessarily of neonatal mortality (Niswander, 1972). This pattern is evident in Table 3 where infants of more highly educated mothers enjoy lower mortality risk during the last six months of the first year of life.[24] Thus maternal education has a more marked effect on mortality

[24] Father's education is not significantly related to mortality when the other variables in Table 3 or 4 are controlled.

during the latter half of infancy than before--similar to Bross and Shapiro's (1982) conclusion for the United States after they had controlled for birthweight.

When we control for more proximate determinants including birthweight (Table 4), the statistical significance of maternal education generally falls, although the effect on total infant mortality remains significant. This pattern indicates that the influence of mother's schooling on infant mortality is somewhat mediated through the more direct determinants we are able to control, but that a small and significant direct effect remains. It may be that more highly educated mothers spend a higher quality of time with their infants. DaVanzo and Lee (1978) find that mothers' education is not related to the quantity of time they spend caring for their chilren in this sample.

Household Income

Similarly, the association of household income with infant mortality is less when the other variables included in Table 4 are controlled. In these data, even the simple correlation between income and infant mortality is statistically insignificant[25]. Income is generally negatively associated with infant mortality in macro-level analyses (reviewed in United Nations, 1973) and even in micro-level

[25] This income measure is constructed from retrospective reports of hours worked and wages of the mother and her husband, and from his retrospective reports of extraordinary income. All other income sources, such as income from family farming and cottage industry, were not documented in the retrospective instruments. Furthermore, many of the women surveyed were no longer married to the same husbands who were present at the birth of some of their children. These former husbands could not be surveyed. Although we attempted to correct for these deficiencies through complex imputation procedures that made use of the more detailed data on families' income and wealth at the time of the survey, we feel that the resulting income variable is probably measured with more error than any other variable in this study.

analyses when the income range is broad (Gortmaker, 1979; Anker and Knowles, 1977). However, household income is generally a less important determinant of infant mortality than is education and is often unimportant when maternal education is taken into account (Cochrane et al., 1980; Bairagi, 1980).

All that can be concluded is that family income is more strongly related to infant mortality without the other controls in Table 4 than with them, though the relationship never attains statistical significance. This is in contrast to the significant effect in these data of low income on birthweight (DaVanzo, Habicht, and Butz, 1981), which is probably a consequence of stunted intrauterine growth due to malnutrition among the poor. This nutritional effect is apparently not particularly pernicious to infant survival (Taylor, Kielman, Parker, et al, 1978; Lechtig and Klein, 1980) once birthweight is controlled; it is not reflected here in increased mortality.

Household Composition

The household composition variables in Table 3 are entered to examine whether the presence of other young children or of individuals who can substitute for the parents in child care is associated with infants' mortality. The first variable, represented here by the number of children less than two years old in the household, is frequently entered in infant mortality regressions to represent competing demands on mother's attention and other family resources (see Wray, 1971). The estimated coefficients in Table 3 are positive after the first week of life and statistically significant in months two through six and for infant mortality as a whole. This is consistent with the usual interpretation. However, when more proximate mortality influences are

controlled in Table 4, this variable becomes completely insignificant. In particular, its association with mortality in Table 3 is entirely accounted for by the dummy variable for previous short birth interval, whose effect, we argue above, is biological in origin.

The two variables reflecting presence of grandparents and other non-nuclear family relatives do not affect mortality when the more proximate determinants are not controlled (Table 3). But when the proximate determinants are controlled (Table 4), infant mortality is significantly[26] reduced by the presence of grandparents, and mortality in the second through fourth weeks of life is reduced by the presence of other relatives. This pattern, of a less proximate variable gaining significance when more proximate variables are controlled, may have emerged here because Malaysian families without the advantage of relatives to help with the baby compensate in other ways (that are controlled in Table 4).[27] By the same token, families faced with otherwise detrimental circumstances may elicit the help of grandparents and other relatives. Hence, no association of mortality with grandparents' presence or other relatives is visible by inspecting only main effects in Table 3.[28]

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[26] One could hypothesize that grandparents' presence could either increase or reduce infants' mortality risk. In this case a two-tail test would be appropriate and this coefficient would not be significant at the 5% level.

[27] A similar change occurred above for the breastfeeding variables.

[28] The fact that grandparents are still alive (and present in the household) could simply reflect a low-mortality environment. However, in this case, an association between grandparents' presence and infants' mortality should emerge most strongly in the Table 3 regression that controls for fewer other covariates. This does not occur.

Infant's Institution of Birth

We examined associations with four places of birth--hospital, estate clinic, private maternity clinic, and home. In the regression that includes only these and other indirect family influences, only hospital birth is statistically significant and only in months 2 through 6.[29] However, when biological attributes at birth and postnatal biological influences are also controlled, hospital-born babies have significantly lower infant mortality as a whole than babies not born in hospitals. Tables 3 and 4 show this comparison, excluding the other birth places, whose coefficients are never significant.

This association between hospital birth and subsequent mortality is concentrated in the second-through-sixth months of infancy. The association may occur because of continuing care through hospitals, or because hospital personnel can identify, at birth, conditions that tend to become lethal in months two through six. Neither explanation seems convincing. The result may simply be due to self-selection of mothers who tend to give their infants better care. This explanation is also probably the source of the association between better medical care and infant mortality reported elsewhere (Chase, 1973).

That this hospital birth variable is significant in Table 4 but not in Table 3 suggests that couples tend to select a hospital as the birthplace when they expect one or more of the more direct mortality influences to be detrimental. In Table 3, where these other influences are not controlled, hospital birth is not significantly associated with

[29] One could hypothesize that hospital birth could either increase or decrease the probability of mortality relative to home birth. In this case a two-tail test would be appropriate and this coefficient would not be significant at the 5% level.

infant mortality. Only when the other controls are added does the association emerge.

Ethnicity

Finally, Table 3 includes dummy variables for Chinese and Indian ethnicity to estimate the residual influence of ethnicity (relative to the reference group Malays). During the period relevant to these data, Malaysian vital statistics show that Chinese infants had higher survival rates than Indian infants, who in turn had higher survival rates than Malay babies (Government of Malaysia, various years). Our data show the same results: Chinese babies enjoy significantly lower mortality risk through most of the first year of life when the other variables entered in Table 3 are controlled. Indian babies also experience lower mortality than Malays in the last six months of the first year of life. These ethnic differences that remain after we control for the other indirect family influences in Table 3 are similar to those that exist when these controls are absent (not shown).

In Table 4, when we also control the biological correlates from Tables 1 and 2, the Indian mortality risk generally falls relative to Malays'. The Table 3 estimates imply that the Indian infant mortality rate is 10 fewer deaths per thousand live births than Malays', while Table 4 estimates imply 32 fewer deaths per thousand live births. The main control variables that account for this change are birthweight and previous short interpregnancy intervals. Indian babies in the MFLS sample weigh, on average, 240 grams less at birth than Malay babies (DaVanzo, Habicht, and Butz, 1981), and Indian mothers have a higher incidence of very short interpregnancy intervals than the other ethnic groups (DaVanzo and Haaga, 1981). When these differences are

controlled, infant mortality is significantly less among Indians than among Malays.

In contrast to the Indians, the Chinese advantage over Malays shown in Table 3 is affected little by the controls introduced in Table 4. This advantage appears to be quite independent of the measured behavioral and environmental influences that might be thought to lie behind it. Something else, unmeasured or poorly measured in these data (e.g., whether the household boils its drinking water), must account for the difference.[30]

COMMUNITY INFLUENCES

The MFLS data include detailed information on characteristics of communities that might affect the mortality of their residents--e.g., types of sanitation, prices and availabilities of infant foods, and access to various types of medical care. We have examined the relationships of these community variables with infants' mortality, but few significant or systematic relationships have emerged. This is probably because the community data refer to the time of the survey (1976) and hence are most applicable to births that occurred within a few years of the survey; however, there are not many infant deaths in this period. In future work, we hope to collect additional data that offer a better historical perspective on levels and changes in community characteristics that might affect mortality. In the meantime, we include in Table 4 a variable measuring the rurality of the place where the family lived when the child was born. This variable derives from

[30] One contributing factor could be differential reporting of deaths due to cultural differences in willingness to discuss the dead. However, the fact that Malay and Chinese infant motality rates in these data are similar to those in Vital Statistics suggests that this factor is unimportant in these data.

the retrospective residence history in the Female Life History questionnaire and, hence, is applicable to all births.

Although many studies of infant mortality in developing countries find important rural/urban differentials (United Nations, 1973), none emerge in Table 4 when other biological and family influences are controlled. However, when no other variables or only the biological attributes at birth in Table 1 are controlled (not shown), rural babies are significantly more likely to die in the first week and last six months of infancy. Hence, it is not rurality *per se*, but postnatal biological influences (e.g., type of water) and indirect family influences (e.g., mother's education) correlated with rurality, that increase the mortality risk of babies born in rural areas.

These findings are consistent with a prominent hypothesis to explain the sudden reversal between urban and rural death rates that occurred in many countries near the beginning of this century (reviewed in United Nations, 1973). It is hypothesized that rural conditions are inherently healthier, but that investments in schools and in public health and medical facilities were biased toward urban areas, thereby lowering their mortality. Our findings support this interpretation for the modern case of Malaysia. When we control for education, birthplace, and type of water and sanitation, there is no significant mortality difference between rural and urban areas. Given the actual configuration of these characteristics, however, urban mortality is significantly less.[31]

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[31] In addition to the variables reported or discussed here, we investigated the roles of other factors which were never statistically significant and hence not reported here. These were the mother's hours of work (other than housework) in the child's first year of life, the occupation of that job, and its distance from her home; other dimensions of household composition (presence of other children by sex and age, presence of servants); incidence of epidemics during the child's first

COMPARISONS OF LOGIT AND THE LINEAR PROBABILITY MODEL

The last column of Table 4 shows coefficient estimates from a logit regression explaining infant mortality. This is the correct procedure for investigating factors whose effects on mortality occur through relative risk, as opposed to attributable risk. Corresponding logits on subsamples cannot be run because of the small numbers of deaths and their tendency to concentrate at particular values of explanatory variables.

A comparison of the last two columns of Table 4 shows that none of the main conclusions drawn from the OLS regression change when logit is used. Hence our estimates and conclusions for infant mortality are unaffected by estimation technique. It is likely that the same would also be true for the subperiods of the first year of life.

PROPORTION OF VARIANCE EXPLAINED BY THE REGRESSIONS

The proportion of variation in mortality explained by these linear probability models is low, from less than one percent to 3.5 percent in Table 1. Even in Table 4, which includes at one time all the variables examined in this analysis, the highest R^2 is .0606. There is of course considerable variation in infant mortality that can never be accounted for statistically. Apart from this, prospective studies should be expected to explain more of the variation in mortality than can this retrospective study, simply because the former are able to measure variables more precisely. They are closer to the relevant time period and use objectively measured, rather than subject-reported, variables.

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year of life; and the type of health care the people in the community generally seek when sick.

Bearing in mind these limitations of a long-term retrospective study, it is interesting to note tha' the regressions reported here account for less variation in mortality during the last six months than during the early part of the first year of life. This may be because disease and the non-breastfeeding components of the diet are important influences on mortality in the last six months of the first year; neither of these factors is well documented beyond the initial weaning period in these data.

IV. INFANT FEEDING AND INFANTS' MORTALITY

Lengths of full and supplemented breastfeeding are two of the three variables most significantly related to the linear probability of infant mortality in Table 4. Those estimates imply that use of foods other than breastmilk is associated, at least under some conditions, with elevated mortality. In this section we seek to identify some of these conditions. We begin in Table 5 by estimating the associations between mortality and use of alternative supplemental and weaning foods common in Malaysia. We then investigate in Tables 6 through 8 the interactions between breastfeeding and types of house water and sanitation, to test the hypothesis that short or no breastfeeding is most pernicious to infants' health when the water supply is likely to be contaminated. Finally, Table 9 reports the sensitivity of the estimated breastfeeding-mortality relationship to different methods of constructing samples and variables.

INFANT FOODS AND MORTALITY

The MFLS reports the first food other than breastmilk fed to mothers' first and most recently born children. From data on length of full breastfeeding, one can compute the length of time over a given period in the baby's first year when supplementary food was given. The data do not report which foods, other than the first, were used or for how long. However, by assuming that the reported first food was used throughout the periods under consideration, we can estimate, though imperfectly, the statistical associations between use of different infant foods and mortality.[32] As with breastfeeding, we only consider use of these foods <u>before</u> the beginning of each at-risk period.

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[32] The distinction in the data between full and supplemented

Table 5 presents these estimates for mortality in days 8-28, months 2-6, and months 7-12, controlling for all the variables in Tables 1 and 2.[33] Use of each of the five types of foods considered in Table 5 is associated with significantly higher mortality in at least one of the subperiods of the first year of life compared with full breastfeeding. Use of infant formula is significantly associated with higher mortality in all three periods of infancy. In the first month and months 7-12, however, use of non-milk appears even more pernicious. Other milk (primarily cow and goat milk) is also worse than infant formula in the first period. Finally, condensed or evaporated milk is significantly worse than full breastfeeding in months 2 through 6. The last category in the table (type of food unknown) is the duration of supplemented or substitute feeding for children other than the mother's first- and last-born, and for a few cases in which the mother could not report the type of food. These coefficients lie within the range of the others because most unknown types were actually the same as those recorded.

Most substitute or supplementary foods show declining associations with mortality over the first half year. For condensed or evaporated milk, however, this pattern reverses in the second period. This larger and statistically significant coefficient for months 2 through 6, therefore, implies an important mortality association. All substitute

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breastfeeding allows construction, for each food type, of both the length of supplemental feeding and the length of substitute feeding. Because the associations of the two variables with mortality were similar for each type of food, we combined the measures into one--total length of use--for each food.

[33] An infant mortality regression analogous to those in the last columns of the previous tables is not presented, both because length of the alternative feeding period is correlated with length of life and because our assumption of continuing use of the first alternative food is least correct in the last six months of infancy.

Table 5

INFLUENCES OF ALTERNATIVE TYPES OF SUPPLEMENTARY OR SUBSTITUTE INFANT FOODS

Explanatory Variable: *Type of supplementary or substitute food (months)*	Mortality in Days 8-28	Mortality in Months 2-6	Mortality in Months 7-12
Infant formula	.0441 (2.50)	.0233 (3.32)	.00268 (2.56)
Condensed or evaporated milk	.0193 (0.85)	.0228 (2.67)	.00098 (0.85)
Other milk	.0994 (1.86)	-.00234 (-0.13)	.00135 (0.66)
Non-milk	.0766 (2.44)	.0103 (1.00)	.00376 (2.96)
Don't know type of food	.0604 (5.01)	.0136 (2.77)	.00251 (2.96)
R^2	.0269	.0243	.0137

NOTE: Equations also control the biological attributes at birth in Table 1 and the postnatal biological influences in Table 2 except for breastfeeding, but these coefficients are not presented here.

or supplementary foods show a marked decline in their association with mortality from the first to second half of the first year, as would be expected.

Although a number of these estimates are significantly different from zero, they fail in every case but one to be pairwise significantly different from each other. The exception is that use of non-milk is significantly worse than use of condensed or evaporated milk for survival in months 7 through 12.

Hence the general absence of full breastfeeding is associated with higher mortality, but the differential harm in feeding one food rather than another cannot be significantly estimated in these data. Rather than carry these distinctions farther, we therefore revert in the remainder of this section to studying relationships with lengths of full and supplemented breastfeeding.

INTERACTIONS BETWEEN BREASTFEEDING AND WATER AND SANITATION VARIABLES

Table 6 enters the breastfeeding, water, and toilet variables from Table 2 along with their interactions.[34] If shorter breastfeeding is particularly dangerous in houses without piped water or toilet sanitation, it should emerge in these estimates. The statistically significant, positive interaction coefficients indicate that this is the case with toilet sanitation.

In households with neither a toilet nor piped water, the effects of reducing breastfeeding by one month are estimated by the main-effects coefficients at the top of the table, all statistically significant. In

[34] These estimates derive from regressions that also controlled for the other variables in Tables 1 and 2.

Table 6

INTERACTIONS BETWEEN BREASTFEEDING AND WATER AND TOILET VARIABLES

Explanatory Variable	Days 8-28		Mos. 2-6		Mos. 7-12		Mos. 0-12	
Breastfeeding								
Mos. unsupplemented breastfeeding	-.186	(-4.80)	-.0947	(-5.93)	-.00672	(-2.68)	-.0179	(-5.45)
Mos. supplemented breastfeeding	-.130	(-3.16)	-.0569	(-3.82)	-.00306	(-1.94)	-.0112	(-7.64)
House characteristics								
Piped water (D)	-.00675	(-1.22)	-.00763	(-0.87)	-.0158	(-2.66)	-.0337	(-3.30)
Toilet (D)	-.0271	(-3.04)	-.0674	(-4.89)	-.0184	(-2.03)	-.125	(-7.87)
Interactions								
Mos. unsupplemented breastfeeding x piped water	.0246	(0.89)	.0131	(1.09)	.00257	(1.39)	.00501	(2.16)
Mos. unsupplemented breastfeeding x toilet	.120	(2.86)	.0774	(4.44)	.00285	(1.05)	.0118	(3.34)
Mos. supplemented breastfeeding x piped water	-.0171	(-0.55)	.00622	(0.54)	.00175	(1.37)	.00322	(2.45)
Mos. supplemented breastfeeding x toilet	.124	(2.75)	.0505	(3.07)	.00128	(0.73)	.00920	(5.53)
R^2	.0289		.0284		.0142		.0604	

NOTE: Equations also control for biological attributes at birth in Table 1, and house-density and year-of-birth variables in Table 2.

houses that do have a toilet and/or piped water, however, the effects of reducing breastfeeding by one month are estimated as the sum of the main-effects coefficient and the appropriate interaction coefficient. For example, mortality among infants with no toilet or piped water is increased by 94.7 deaths per thousand in months 2 through 6 if a child does not breastfeed during the first month. This harmful effect of not breastfeeding during the first month on mortality in the ensuing five months is reduced to 81.6 deaths per thousand (0.0947 - 0.0131) if piped water is present, to 17.3 deaths per thousand (0.0947 - 0.0774) if there is a toilet, and to a very small 4.2 deaths per thousand (0.0947 - 0.0131 - 0.0774) when there are both.

The interaction effects in Table 6 can be more systematically inspected in Tables 7 and 8, where we present the mortality differentials due to the presence or absence of certain combinations of breastfeeding, water, and sanitation, calculated from the estimates in Table 6. The third column of Table 7, for example, shows that in months 7 through 12 the estimated mortality differentials associated with having toilet sanitation or piped water depend on the child's length of breastfeeding. With no breastfeeding at all, presence of a toilet is significantly associated with 18.4 fewer deaths per thousand. This effect is smaller (10.8 deaths per thousand), but still significant, for infants who partially breastfed for six months. But, for infants who fully breastfed for six months, there is no significant mortality differential at all associated with presence of a toilet. A similar pattern emerges for piped water in the 7-12 period; again the beneficial effect disappears with 6 months of either full or supplemented breastfeeding.

Table 7

MORTALITY EFFECTS OF PIPED WATER AND TOILET SANITATION
DEPENDING ON BREASTFEEDING

(Table entries are changes in mortality associated with presence of toilet sanitation or piped water compared with the absence of that feature; t-statistics are in parentheses)

Mortality Differences Associated with:		Changes in Mortality in Days 8-28	Months 2-6	Months 7-12	First Year BF = 28 days	BF = 6 Mos.
Toilet Sanitation (compared with no toilet) with full or supplemented breastfeeding*						
No	No	-.0271 (-3.04)	-.0674 (-4.89)	-.0184 (-2.02)	-.125 (-7.87)	-.125 (-7.87)
No	Yes	.0018 (0.34)	-.0203 (-2.99)	-.0108 (-2.18)	-.116 (-7.98)	-.070 (-7.74)
Yes	No	.0009 (0.23)	.0048 (0.57)	-.0013 (-0.10)	-.113 (-7.73)	-.054 (-2.70)
Piped Water (compared with no piped water) with full or supplemented breastfeeding*						
No	No	-.0068 (-1.22)	-.0076 (-0.87)	-.0148 (-2.66)	-.0337 (-3.30)	-.0337 (-3.30)
No	Yes	-.0107 (-2.26)	-.0018 (-0.28)	-.0043 (-0.90)	-.0305 (-3.28)	-.0144 (-2.07)
Yes	No	-.0010 (-0.30)	.0046 (0.45)	.0006 (0.07)	-.0287 (-3.06)	-.0036 (-0.27)

*"Yes" for full or supplemented breastfeeding means that it lasts 1 week in the 8-28 day sample, 28 days in the 2-6 month sample, 6 months in the 7-12 month sample, and 28 days or 6 months (as indicated) in the first year sample. "No" means that no breastfeeding of that type occurs.

Table 8

MORTALITY EFFECTS OF REDUCED BREASTFEEDING DEPENDING ON TYPE OF WATER AND SANITATION
(Table entries are changes in mortality associated with reductions in breastfeeding; t-statistics are in parentheses)

		Changes in Mortality in:				
		Days 8-28	Months 2-6	Months 7-12	First Year	
Full Breastfeeding Reduced to Zero from:		(1 week)	(28 days)	(6 Months)	(28 days)	(6 Months)
with toilet and piped water						
No	No	.0433 (4.81)	.0884 (5.93)	.0403 (2.68)	.0167 (5.45)	.107 (5.45)
No	Yes	.0376 (3.70)	.0761 (4.44)	.0249 (1.41)	.0120 (3.33)	.0773 (3.33)
Yes	No	.0154 (2.91)	.0161 (1.83)	.0232 (3.03)	.0057 (3.97)	.0366 (3.97)
Yes	Yes	.0097 (2.39)	.0039 (0.54)	.0078 (0.93)	.0010 (0.59)	.0065 (0.59)
Partial Breastfeeding Reduced to Zero from:		(1 week)	(28 days)	(6 Months)	(28 days)	(6 Months)
with toilet and piped water						
No	No	.0303 (3.16)	.0531 (3.82)	.0184 (1.95)	.0104 (7.64)	.0672 (7.64)
No	Yes	.0343 (3.09)	.0473 (2.92)	.0079 (0.70)	.00744 (4.29)	.0479 (4.29)
Yes	No	.0014 (0.25)	.0060 (0.70)	.0107 (1.82)	.0019 (2.04)	.0120 (2.04)
Yes	Yes	.0054 (1.14)	.0002 (0.03)	.0002 (0.05)	.0011 (1.29)	.0073 (1.29)

Most of the combinations in Table 7 follow this general pattern, which is summarized for infant mortality as a whole in the last two columns. Presence of piped water or toilet sanitation is most important in reducing infant mortality for women who breastfeed little or not at all. This importance is less where breastfeeding is initiated and maintained for a month (column 4), and much less where breastfeeding lasts for six months (column 5). Presence of toilets is more important than piped water in nearly every instance.

Table 8 uses the estimates in Table 6 to make a related point: that breastfeeding declines are less harmful in households with either toilets or piped water and much less harmful in households with both. In actuality, women in this Malaysian sample who breastfeed very little or not at all tend disproportionately to live in houses with modern water and sanitation facilities. Of babies who did not breastfeed, 91.4 percent were born into homes with toilet sanitation, compared with 76.9 percent of babies who did breastfeed. The comparable figures for piped water are 68.4 percent for non-breastfed babies and 37.7 percent for breastfed babies. These results suggest that many Malaysian infants suffer little mortality risk on average from the lack of breastfeeding. However, lack of breastfeeding is dangerous in communities without modern water and sanitation facilities, where water used in mixing alternative foods is more likely to be contaminated.

SENSITIVITY OF BREASTFEEDING EFFECTS ON MORTALITY TO ALTERNATIVE METHODS OF ESTIMATION

Section II discussed the statistical difficulties in estimating the effects of breastfeeding on mortality. Table 9 examines the sensitivity of estimated effects of breastfeeding on infant mortality to several alternative estimation methods. The first three rows of panel A use reported lengths of breastfeeding and compare estimates from three

TABLE 9

INFLUENCES OF BREASTFEEDING ON INFANT MORTALITY: COMPARISONS ACROSS DIFFERENT SAMPLES AND MEASUREMENT OF BREASTFEEDING

(Table entries are coefficients (and t-statistics) of breastfeeding variables from equations that also controlled for the biological attributes at birth and postnatal biological influences in Tables 1 and 2. Dependent variable = dummy that equals 1 if infant died in first 12 months of life)

A. BREASTFEEDING VARIABLES = DURATION OF BREASTFEEDING

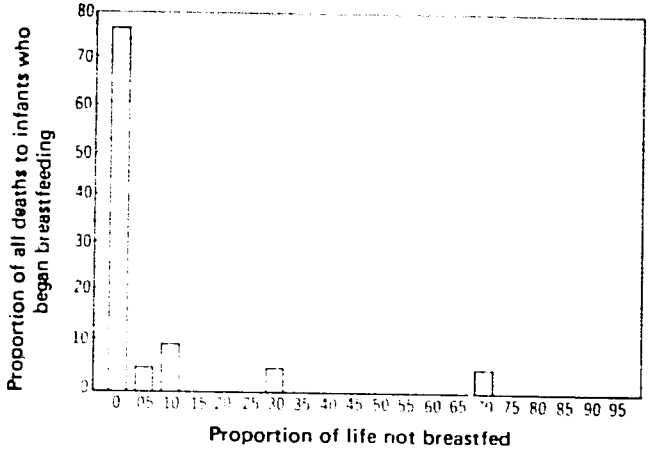
	Months of Unsupplemented Breastfeeding	Months of Supplemented Breastfeeding
Breastfeeding = actual value		
Sample		
All live births (n=5357)	-.0117 (-10.76)	-.0112 (-18.09)
Excluding cases where length of breastfeeding = length of life (n=5217)	-.0048 (-6.01)	-.0042 (-9.21)
Also excluding cases where breastfeeding stopped shortly before death (n=5178)	-.0036 (-5.45)	-.0037 (-9.72)
Breastfeeding = imputed value		
Sample		
All live births (n=5357)	-.0054 (-4.73)	-.0022 (-3.31)

B. BREASTFEEDING VARIABLE = DUMMY THAT EQUALS 1 IF CHILD WAS BREASTFED

Sample	
All live births (n=5375)	-.0798 (-9.96)
Excluding cases where breastfeeding was not begun because baby died (n=5298)	-.0177 (-2.38)

samples. The first sample includes all live births. The second excludes mortality cases where the length of breastfeeding equals the length of life or where the mother reported that she stopped breastfeeding (or did not begin) because the child died.

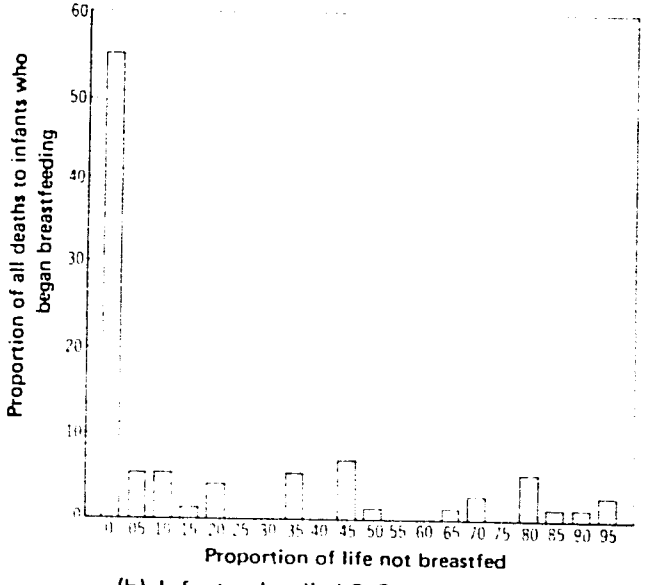
The third sample excludes, in addition, cases where breastfeeding stopped shortly before death. We do this because examination of Figure 4 shows that more deaths occur shortly after the cessation of breastfeeding than would be expected, given the more uniform distribution of deaths following longer periods of nonbreastfeeding. Under the conditions of water quality, sanitation, and infant feeding practices common in Malaysia, there is no reason to expect that cessation of full or supplemented breastfeeding could kill an infant in so short a period. Alternatively, there are many lethal diseases and conditions that in the final stages weaken an infant so much that it cannot nurse, even quite apart from the associated hospitalization that, when it occurs, nearly always halts nursing. It is reasonable to assume from the pediatric and public health evidence that the average time required for many diseases to induce death increases proportionately with an infant's age. Hours are often critical in the first day and week of life, while later in the first year, days and weeks can pass with most conditions. One would therefore expect to observe a large accumulation of deaths shortly after the cessation of breastfeeding, with the accumulation spreading over a somewhat longer period the older the infant was at death. Figure 4 in fact shows this pattern. Most babies who began breastfeeding and subsequently died had either not been weaned when they died or died shortly after weaning. Most of this



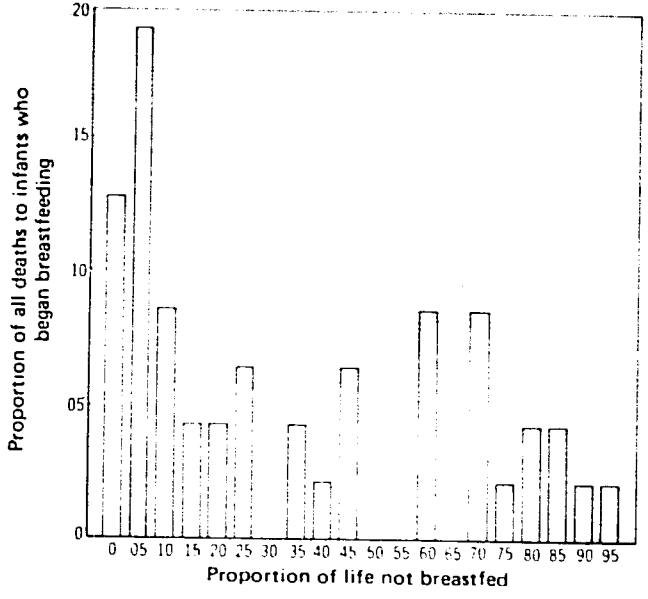
(a) Infants who died 8-28 days after birth

Notes : If length of breastfeeding is greater than the beginning of the at-risk interval (e.g. 7 days for 8-28 day mortality), it is truncated to equal the length of time until the beginning of the at-risk interval.

The entries on the horizontal axis are midpoints; e.g. the bar centered on .10 shows the proportion between .075 and .125.



(b) Infants who died 2-6 months after birth



(c) Infants who died 7-12 months after birth

Fig. 4 — Frequency distribution of proportion of life not breastfed for infants who began breastfeeding

excess accumulation of deaths appears to occur within a period after weaning that constitutes no more than 12.5 percent of the infants' length of life. We conclude that these infants who nursed until shortly before death are more likely both to have ceased nursing and to have died because of third causes than to have died because they ceased nursing. Accordingly, if infants whose duration of breastfeeding was the same as their length of life are dropped from the sample for the purpose of estimating breastfeeding coefficients, so should these other infants be dropped. In the data presented in row 3 of Table 9, we exclude infants who died whose breastfeeding ceased within 12.5 percent of their age at death.

The sample of all live births (row 1) yields the largest estimated effects of full and partial breastfeeding, with coefficients about three times those for the smallest sample (row 3). The row 2 estimates are in between. These differences between rows 1 and 2 are due to the fact that some babies had short breastfeeding because they died. Row 3 estimates are even smaller because they exclude cases where a third factor may be the reason for the infant's death.

The last estimates in panel A are based on the total sample again, but use an imputed value of breastfeeding lengths for children whose breastfeeding length was equal to or only slightly less than their length of life. (This imputation procedure is described in Sec. II.) The estimates of the influence of full breastfeeding for this method are smaller in absolute magnitude than those in the first row of Table 9, where actual breastfeeding values are used for this same sample, but are larger in absolute magnitude than those in the second and third rows of

Table 9, which are based on samples that exclude cases subject to spurious correlation. The estimates of the absolute magnitude and significance of the effects of supplemented breastfeeding are lowest in the fourth row.[35] Only in row 4 is the effect of supplemented breastfeeding significantly smaller than that of unsupplemented breastfeeding, as we found for subperiods of the first year of life in Table 2 using another method.

We prefer the method used in row 4, because, unlike those for rows 2 and 3, it does not exclude from the sample observations on children who died. Two-thirds of the infant deaths are excluded from the sample that remains in row 3. Excluding these cases not only causes an enormous loss of information. More importantly, it biases upward the statistical significance of the breastfeeding coefficients because it artifactually reduces the number of deaths among children with long breastfeeding. However, including these cases without any adjustment (in row 1) severely biases the estimates of breastfeeding's effects. For these reasons, all the estimates of breastfeeding's influence on infant mortality presented earlier in this paper are based on the method used in row 4 of Table 9.

Panel B of Table 9 reports a similar experiment where breastfeeding is characterized by a simple dummy variable indicating whether or not the child was breastfed. Some studies (e.g., Scrimshaw, Taylor, and Gordon, 1968; Janowitz et al., 1981),[36] particularly those from

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[35] This may be because our restrictions for the row-3 estimates failed to exclude all cases subject to spurious correlation between length of partial breastfeeding and length of life.

[36] Janowitz et al. (1981) recognize the possibility of reverse causation, but their test for its effect (excluding from the sample <u>all</u> babies who breastfed less than 3 months, regardless of whether they died or survived) does not address the issue directly.

hospital- or clinic-based samples, use such a variable. We compare the coefficient of this dummy variable for the total sample with that for a sample which excludes the babies whose mothers reported that the child was not breastfed because it died. The results suggest that 78 percent of the statistical association between infant mortality and the initiation of breastfeeding arises in fact from infants who did not begin breastfeeding because they died before having a chance.

Studies that do not correct for spurious correlation between infant mortality and initiation and duration of breastfeeding will produce overestimates of the mortality-inhibiting effects of breastfeeding. These Malaysian results suggest that the biases can be extremely large.

V. IMPLICATIONS

The findings reported in this paper have implications for future research, for risk screening and targeting of interventions, and for program and policy initiatives. We conclude by discussing each in turn.

IMPLICATIONS FOR RESEARCH

The analyses reported herein have used retrospective life-history data from the Malaysian Family Life Survey to investigate biomedical, behavioral, and environmental influences on mortality during the first year of life. The data, despite being mothers' reports of events many years earlier, produce many statistical associations with infant mortality that are consistent with the clinical and epidemiological literature. Examples are the elevated mortality risk associated with very young maternal age, low birthweight, short previous birth interval, and male sex of the child. These corroborations with carefully collected clinical data suggest that retrospective data can yield valid conclusions about influences on infant mortality.

This paper moves beyond these findings to investigate other less well-known interrelationships with infant mortality in Peninsular Malaysia. These extended investigations were facilitated by several features of this research:

- The joint investigation of biological and behavioral factors that together influence mortality risk. This investigation reveals, for example, significant associations of mortality with child's birth order and whether another child aged less

than two was in the home. However, these associations disappear when the infant's birthweight and the presence of a preceding short birth interval are controlled. The latter two variables have their influences through biological mechanisms. Without controlling for their influences, a researcher might erroneously conclude from the associations of the former two variables with mortality that behavioral factors such as mother's time spent with the infant are important.

- The investigation of changing structures of mortality determinants over the course of the first year of life. This reveals that such biological factors as low birthweight are more important early in the first year of life, while such behavioral and environmental factors as mother's education or types of water and sanitation system are more important later.
- The investigation of interactions between more and less proximate influences on mortality. Three types of interactions emerge that are of special interest. One occurs when the effect of a less proximate determinant is mediated by a more proximate determinant. For example, when proximate determinants are not controlled, Indians' infant mortality is not significantly different from Malays'. When these more proximate correlates are controlled, Indian infant mortality is significantly lower than Malays'. This is because Indians have lower birthweights and shorter intervals preceding their births. Another interaction of interest arises when no association between mortality and a determinant is detected in one kind of family, but interaction variables reveal an

association in other kinds of families. For instance, in the second through sixth months of infancy lack of toilet facilities does not emerge as a strong correlate of mortality among fully-breastfed infants, but is a serious threat to non-breastfed infants. Finally, differentiating between proximate and less proximate determinants of mortality identifies determinants which would otherwise be hidden. For example, the beneficial effect of the presence of grandparents and other relatives is only evident in regressions that control for more proximate mortality influences. This apparently occurs because Malaysian households without the advantage of grandparents or other relatives to help with the baby compensate in other ways. Similarly, households with high mortality risk may compensate through the presence of grandparents and relatives for help.

- The investigation of breastfeeding's effects on infant mortality. When sources of spurious correlation are removed, these estimated effects decline significantly. Studies that do not remove these sources of bias can produce serious overestimates of the mortality-inhibiting effects of breastfeeding.

IMPLICATIONS FOR SCREENING AND TARGETING INTERVENTIONS

In addition to suggesting potentially effective interventions, the results reported above also point to specific characteristics of mothers and infants that may be associated with elevated risk of infant mortality. As part of a risk screen, these characteristics might be monitored to anticipate increasing risks or to identify particular communities, families, or infants to which an intervention could be targeted. Table 10 summarizes the risk-screening characteristics that

Table 10

RISK-SCREENING CHARACTERISTICS

Screening Characteristics	Increases Mortality in: First Week	Days 8-28	Months 2-6	Months 7-12	First 12 Months of Life
Household characteristics					
No piped water				x	x
No toilet sanitation	x		x	x	x
High density (large number of persons per room)		x		x	x
Low income	x				x
No nonnuclear family relatives					x
Maternal characteristics					
Mother less than 18 years old	x	x[a]			x
Preceding short birth interval (<15 months)		x	x		x
History of short birth intervals	x				
History of stillbirths		x	x		x
Little and no education				x	x
Mother is Malay:					
Compared with Chinese mothers	x		x	x	x
Compared with Indian mothers				x	
Child characteristics					
Sex is male	x		x	x	x
Low birthweight	x	x	x		x
High birth order				x	
Little or no unsupplemented breastfeeding[b]	NA	x	x	x	x
Little or no supplemented breastfeeding[b]	NA				x
Child not born in hospital			x		x

[a]Furthermore, in this subperiod, mothers in their late teens or twenties have higher mortality risk than mothers in their thirties.

[b]Especially in households without piped water or toilet sanitation.

are important in these data. The purpose of a risk screen is merely to identify those at elevated risk (so that one can intervene to reduce mortality), rather than to infer why the screening characteristic is associated with higher mortality. Hence, one may argue that risk-screening characteristics should be based on total effects (simple correlations), rather than partial ones.[37] Therefore, the list in Table 10 includes some variables that are not important in regressions that control other variables, but that are significantly related to mortality without controls.[38] Before these candidates for risk indicators can be used, their actual sensitivity-specificity characteristics (Habicht et al., 1982) must be determined, so that the costs and benefits of the screening and the consequent interventions can be compared. Even indicators with rather low correlation coefficients in a population study such as reported here can be turned into good screening indicators when they are collected and judged appropriately.

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[37] Actually, the best risk-screening characteristics take into account knowledge available at the time of screening and should be tailored to the specific screening situation. For instance, if one were screening after birth, one should consider the mortality risk conditional on birthweight. In that case, Malay infants would be identified as being at higher risk than Indian infants. By contrast, if one were screening before birth, Indian/Malay ethnicity would not be a factor. The data in Tables 1-4 and 10 show the range of screening possibilities with these data.

[38] Variables are included in Table 10 if their coefficients are significantly different from zero at the 5-percent level (one-tail test) in a regression that includes only that variable (or the set of which it is a member, e.g., for mother's age), the child's year of birth, and a constant term.

PRINCIPAL IMPLICATIONS FOR PROGRAM AND POLICY INITIATIVES

This study has yielded a number of findings with plausible implications for policies and programs. Some of these findings are sufficiently consistent with the findings of other studies, or are sufficiently supported by biomedical or behavioral theory, that, in our opinion, program and policy initiatives can now reliably take them into account.[39] Here are the principal implications from these findings:

- Water and sanitation improvements will have their greatest impact on public health if focused on areas where mothers breastfeed little or not at all, or where the mothers appear, on the basis of research there or elsewhere, to be at risk of reducing their breastfeeding in the near future. These findings are consistent with theory and are so strongly robust in this study that we feel they have immediate program implications, even though they are new.
- Policy and program initiatives of whatever type to increase breastfeeding will have their greatest effect in reducing infant mortality if applied selectively to populations whose water and sanitation systems are poor. Present theory expects some benefit from breastfeeding even in infants with access to pure water, but our estimates indicate that these benefits are relatively small.

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[39] As with findings from any nonexperimental study, it would be wise to verify causality through scientific field trials (Habicht, 1979; Habicht and Butz, 1980).

- Previous studies of the effectiveness of breastfeeding in reducing infant mortality have produced overestimates. Due to methodological flaws, these studies assigned to short breastfeeding the responsibility for many deaths that must have been due instead to other causes. Our finding is that length of breastfeeding is one of the strongest correlates of infant mortality, but no stronger than several other factors that public programs can influence. Therefore, policies to encourage breastfeeding should not be pursued at the expense of other program and policy initiatives to reduce infant mortality.
- Family planning services should focus on very young and older women to reduce infant mortality, as well as to reduce fertility. Even though our interpretation of the causes of increased mortality to very young and very old mothers may be new, the literature contains ample evidence to substantiate our findings that these mothers tend to lose their infants more than other mothers.

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