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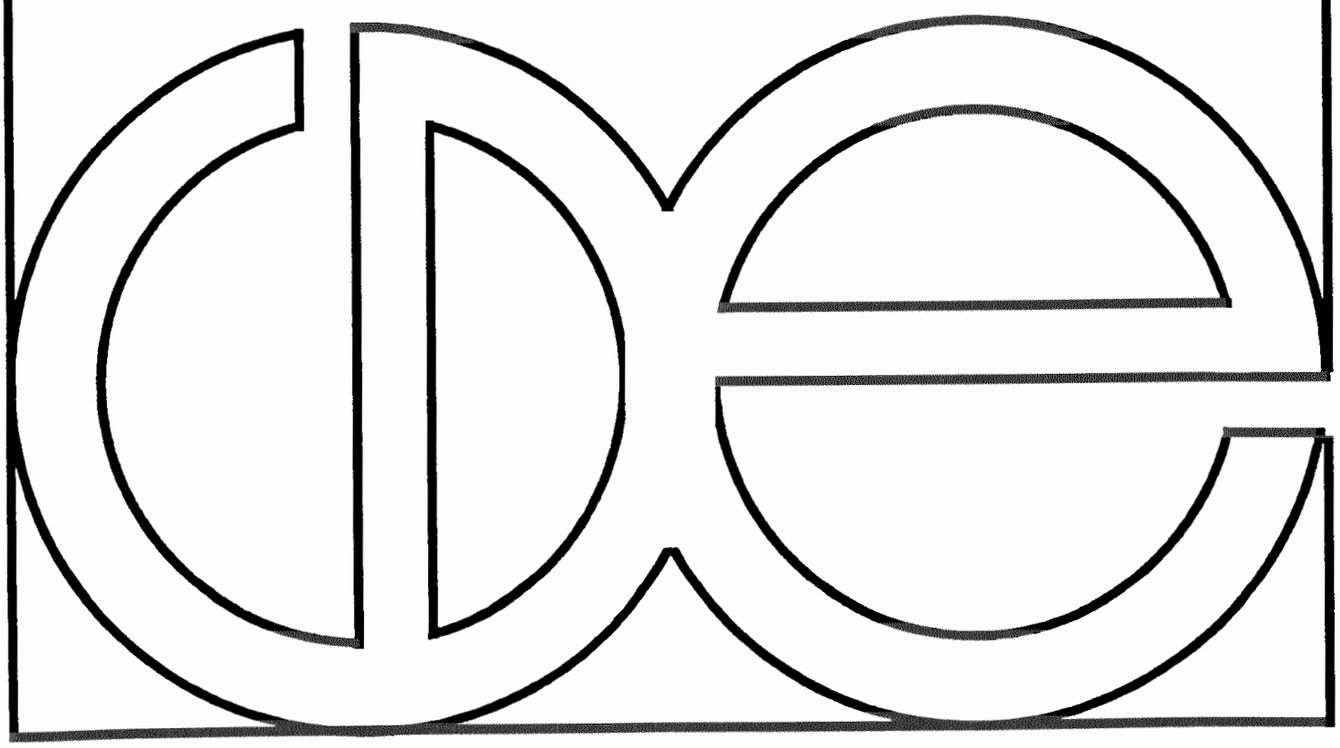

DETERMINANTS OF INFANT AND EARLY CHILDHOOD MORTALITY IN CAMEROON

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ABSTRACT

We use information on child health and survival for a sample of children in Cameroon to assess the influence of selected determinants. The data were collected through a panel design with repeated interviews every three or four months after the birth of a child during a two-year period beginning on January of 1978.

We propose two-state hazard models with and without unmeasured heterogeneity to verify hypotheses about the effects of breastfeeding and length of following conception on infant and early childhood mortality while controlling for potentially confounding factors. Our results confirm conjectures formulated by other researchers who have used information from both retrospective and prospective surveys. Our results suggest that the effects of breastfeeding and premature following conception on mortality risks during early childhood are strong and unlikely to be due to spurious relations, simultaneity biases, unmeasured characteristics, or selective losses to follow up.

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1. THE IFORD SURVEYS AS A SOURCE FOR THE ANALYSIS OF EARLY CHILD MORTALITY.

Despite their obvious importance as rich data sources on child health and mortality before age two, the *Enquêtes sur la Mortalité Infantile et Juvénile* carried out in some African countries during the late 1970s and early 1980s under the auspices of IFORD (*Institut de Formation et de Recherche Démographiques*) remain grossly underanalyzed. Admittedly, the focus on large cities (Ouagadougou, Cotonou, Bamako, Bobo Dioulassso, Brazzaville, Lomé and Yaoundé) and on births that took place within hospitals makes them less attractive than large scale, representative surveys such as the World Fertility Surveys (WFS). Furthermore, as is common in panel studies carried out in developing countries, sizeable levels of sample losses pose severe limitations to the robustness of inferences drawn from the studies. In compensation, however, the IFORD surveys contain a wealth of information on a number of characteristics such as episodes of illnesses, feeding practices, birth weight, pre- and peri-natal care that are rarely, if ever, available in larger scale surveys. If these characteristics were marginal to theories of determinants of infant and child health

and survival, one could probably disregard the surveys at least as potentially significant sources for the analysis of mortality. But this is hardly the case. Some of the most important findings from WFS about the effects of pace of childbearing and breastfeeding, for example, (Hobcraft et al. 1983, 1984; Cleland and Sathar 1984; Palloni and Millman 1986; Rutstein 1984; Lantz et al. 1992; Retherford et al. 1989; Hobcraft 1992) can be called into question with arguments that at least partially invoke the hypothetically strong influence of unmeasured factors such as birth weight and recurrent illnesses (Potter 1989; Retherford et al. 1989; Lantz et al. 1992; Habicht et al. 1986; Miller 1989; Gribble 1993) neither of which were appropriately measured in WFS.

In this paper we attempt to fully analyze the information from the IFORD survey carried out in Yaoundé between 1978 and 1980 to test hypotheses about selected determinants of early child mortality. We show that, despite convincing arguments to the contrary, the effects of breastfeeding and length of following conception remain important even after a series of controls available from the Yaoundé survey are introduced. Our analysis pays special attention to two issues. The *first* is the selection of appropiate models for the estimation of effects. We argue that single and multistate hazard approaches are, in theory at least, the best choice. We also propose the use of alter-

native models with unmeasured heterogeneity to partially address difficulties engendered by omitted covariates. The *second* issue is to assess the potentially damaging influence of selective losses to follow up. Although we devote considerable attention to this problem and suggest alternative treatments, we find no evidence whatsoever indicating that our inferences could be badly in error as a result of sample losses. This conclusion must, however, remain tentative since the proper model for testing our conjectures is beyond the realm of the conventional hazard models that we use here.

The plan of the paper is as follows: In section 2 we describe the relations between pace of childbearing, breastfeeding, illnesses and mortality, and the difficulties involved in the estimation of effects. In section 3 we discuss characteristics of the data source and evaluate its main shortcomings. In section 4 we present modelling strategies and discuss the estimates and their implications.

2. THE RELATIONS BETWEEN BREASTFEEDING, PACE OF CHILDBEARIING AND HEALTH AND SURVIVAL.

2.1. Breastfeeding and early child mortality.

The health advantages of uninterrupted lactation during the first year of the life of a child seem to have been amply recognized (Habicht et al. 1986;

Jelliffe and Jeliffe 1978; Palloni and Millman 1986). Breastmilk confers immunities to the child, contains a high percentage of the nutritional substances required for the infant's growth and is hygienic (Plank and Milanesi 1973; Millman 1985). Early introduction of supplementation and bottle feeding which leads to reduced intensity of breastfeeding may, under most conditions prevailing in Africa, Latin America and parts of Asia, increase exposure to illnesses, diminish the reservoir of immunities and curtail the capacity to promptly recover from bouts of infections and parasitic diseases (Ajello 1982; Mosley 1985).

Since the initiation, duration, intensity, and type of breastfeeding are partially influenced by the infant's health status at birth (Millman and Cooksey 1987), the onset of illness and the consequent reduction in suckling (Butz et al. 1983), and maternal behavior with an independent impact on the health of the child (Potter 1988; The Cebu Team 1989), isolating the 'pure' effects of breastfeeding is a difficult task and requires detailed information on changing characteristics of the child and the mother.

a) Health conditions at birth, breastfeeding, and survival.

If health problems develop as a result of or around the time of birth, an infant may be prevented from starting lactation at all or the pattern of breastfeeding may not be intense enough to secure an adequate supply of milk.

If subsequent health complications develop and the child dies, both health deterioration and death will appear to be associated with lack of or poor lactation when in fact both are the result of pre-existing health conditions. An optimal solution to this problem is to control for health conditions at birth. Birth weight is a good proxy for them and should be used whenever available. A still imperfect but yet safe alternative is to focus on conditional survival beyond some arbitrarily selected minimum threshold age. If health conditions at birth do not at all or only weakly affect health and survival after the selected age, this solution should be quite efficient. But the evidence that we have available is that conditions at birth are likely to have non-trivial detrimental effects throughout and even past the end of the first year (Haaga 1989). While displacing the threshold age beyond which the analysis is done gradually reduces the biases, it also removes progressively larger segments of infancy to be analyzed. In the analyses that we describe in section 4 we use both strategies: we first estimate models for the entire age span from 0 to 24 months while controlling for birth weight and then we restrict estimation to the age segment 1-24 and do not control for birth weight.

b) Illnesses and the impairment of lactation.

The onset of an illness may impair the child's capacity or stimulus to suckle and that, rather than any changes in the pattern of lactation, could be responsible for progressive weakness and eventually death. In some societies the onset of an illness frequently involving high fevers and diarrhea will trigger immediate discontinuation, regardless of the effects of the disease on the child capacity to breastfeed. Naturally, it is possible that inadequate feeding practices facilitate the encroachment of repeated illnesses and that these, in turn, lead to further deterioration of breastfeeding and eventually death. The point that we are stressing, however, is that under certain conditions and irrespective of the event(s) that set(s) in motion the causal chain, the pattern of lactation will be affected by the same factors that facilitate illness and possibly death. Regardless of whether breastfeeding changes as a result of physiological impairment or due to maternal behavior, there is an artifactual relation involving health conditions, lactation and mortality. If not properly modelled, the estimated effects of breastfeeding will contain large upward biases. An ideal study design addressing this problem should retrieve information on the timing of onset, duration and resolution of important illness while at the same time including observation on the changeable patterns of breastfeeding. This

makes possible a control for presence (absence) of illness while assessing the magnitude of the effects of lactation on their onset and on child survival.

c) Breastfeeding as a behavioral choice.

Breastfeeding, like any other maternal behavior influencing health care, is chosen (or avoided) deliberately by women. If maternal behaviors with effects on child health are connected to each other and to unmeasured characteristics that determine the infant's mortality risks, breastfeeding will appear to cause mortality when in fact there is no relation or, alternatively, the measured relation could turn out to be considerably weaker than what in fact is. If women who choose not to breastfeed are systematically selected among those with poor birth outcomes, a relation between breastfeeding and mortality will be observable even though it only reflects the effect of birth conditions on maternal behavioral choices. Similarly, if women with a modern outlook, more aware and knowleadgeable about health care practices choose to reduce or altogether abandon breastfeeding, the measured relation will be weaker than what it should be. There are two ways of solving the problem: the first is to model simultaneously the choice of breastfeeding practices as well as child health and child mortality. The second is to control for the characteristics that influence both outcomes.

Although these cautionary notes are important, we should note that it is unlikely that the very strong effects of breastfeeding observed across so many societies with sharply different practices of contraception and availability of health care could be due entirely to artifacts.

2.2. Pace of childbearing and early child mortality.

Like the evidence relating breastfeeding to infant mortality, the evidence suggesting a close connection between pace of childbearing and early child mortality is very strong and generalized (Hobcraft et al. 1983, 1985; Palloni and Millman 1986; Rutstein 1984; De Sweemer 1984; Cleland and Sathar 1986; Pebley and Stupp 1986; Winikoff 1983; Hobcraft 1992; Lantz et al. 1992; Boerma et al. 1992; Muhuri and Menken 1992). Unlike the relation involving breastfeeding, however, the unsettled issues have less to do with the magnitude of the effects than with the mediating or intervening mechanisms interlocking one variable to the other.

A preceding interval is generally defined as the time elapsed between the occurrence of a birth of order n and the occurrence of the (n+1)th birth. If the birth interval is too short, the health conditions of the (n+1)th birth may be impaired. This could occur as a result of several mechanisms:

i) A short birth interval may reflect a short birth-to-conception interval

which in itself could lead to short gestational age. Since sub-standard length of gestation is strongly associated with high levels of neonatal mortality, the relation between birth interval and mortality could be attributable to the effects on the gestational process. Although this remains a plausible mechanism (National Research Council 1989; Haaga 1989) there is no good evidence unequivocally pointing to its operation. Whether or not the mechanism is real, short gestational lengths will disproportionaly contribute to the pool of the shortest of birth intervals. Thus, even if there is no linkage between birth-to-conception interval and length of gestation, a short birth interval will appear to be correlated with higher mortality as it is a proxy for short length of gestation (Miller 1989) However, the power of this artifact to explain the observed association with mortality in the context of developing countries is of reduced import (Hobcraft 1992; Hobcraft et al. 1985; Wolfers and Scrimshaw 1975; Pebley and Stupp 1987; Miller et al. 1992).

ii) The deleterious effects of short birth intervals may be a result of maternal depletion and the consequent impaired capacity of the mother to secure adequate intrauterine growth. Maternal depletion is believed to take place when there is insufficient recovery time from the last pregnancy. A weaker pattern of breastfeeding is the physiological counterpart of maternal

depletion but its effects are felt post-natally rather than influencing the nature of the birth outcome as maternal depletion does (National Research Council 1989).

iii) A short birth interval may increase sibling competition for resources or, alternatively, augment the opportunities for transmission of infectious disease (Mosley 1985; Aaby 1988; Mims 1991).

iv) Finally, the relation may be spurious and attributable to the correlation between mortality conditions to which children born to the same mother are exposed. Indeed, if the death of the nth child accelerates the conception of the (n+1)th, either through a volitional response or as an indirect, non-intended result of premature return to menses, a significant fraction of short birth intervals will be drawn from households experiencing relatively high mortality conditions. Since the survival conditions of two siblings are in part at least jointly determined, the relation between length of interval and survival could reflect the association between exposure to common conditions and fertility replacement behavior.

A following birth interval is defined as the time elapsed from the time of the index birth (the birth whose health we investigate) and the occurrence of the following conception. In some cases only conceptions leading to a live

birth are considered, whereas in others all conceptions are taken into account. The effects of following intervals on infant and early childhood mortality have been largely attributed to sharply disrupted or outright cessation of lactation. However, other mechanisms may be operating. First, physical depletion of the mother, whether it is accompanied by changed breastfeeding or not, could significantly reduce the quality of child care received by all but especially the youngest of a pair of successive siblings. Second, sibling competition for resources as well as for maternal care can, as it did in the case of preceding interval, impair the health conditions of a recently born child.

Like the case involving previous interval, the relation between the timing of the following conception and mortality could be the result of artifacts. It is well known that the death of the child will terminate breastfeeding and increase sharply the likelihood of return to menses. Indeed, a most powerful predictor of the onset of menses is the death of the child (Jones and Palloni 1990). In societies with little or no use of contraception this chain of events may lead to a more rapid conception. If so, it is the death of a child that generates the conditions for a short birth-to-conception interval rather than the other way around. To avoid the resulting simultaneity biases, it is necessary to observe and define precisely the timing of the events implicated in the

relation. As shown below, it is difficult to totally circumvent the problem, but the task is made easier with the data collected by the Yaoundé-Iford Survey (YIS).

Studies that document strong effects of preceding birth interval on infant and early childhood mortality are numerous. But the best known are those that have used WFS and DHS data (Hobcraft et al. 1983, 1984; Rutstein 1985; Hobcraft 1992; Boerma et al. 1992; Boerma and Bicego 1992). While the magnitude of the effects of following interval is universally large, the effects of preceding birth interval are more variable and attain importance in countries of Africa and Asia but much less so in Latin America (Palloni and Millman 1986). The geographic variability may reveal the operation of alternative mechanisms or simply reflect the presence of different errors and biases.

3. THE DATA SOURCE: ADVANTAGES AND SHORTCOMINGS.

3.1. Design of the panel study.

The Yaoundé-Iford Survey (YIS) was carried out in Yaoundé, Cameroon, from January 1978 to January 1981. The survey was designed to measure

levels and patterns of mortality during the first two years of life, and its demographic, biomedical, cultural and socioeconomic determinants. The survey also retrieved information on infectious diseases, nutritional deficiencies in infancy, and changes in maternal and child health. The information was collected through a two-stage process of follow-up interviews. The first stage was initiated with the registration at maternity wards (hospitals) of newly born children whose mothers were residents of Yaoundé. The information gathered at this stage included demographic, cultural and socio-economic information about the parents, characteristics of the pregnancy and of the newly born child. The second stage consisted of seven waves of interviews to the mothers or their surrogates. The interviews took place 1, 4, 8, 12, 16, 20 and 24 months after birth. These interviews were designed to collect information on characteristics of the household, and health conditions of the mother and the child.

As shown in Table 1, a total of 9774 births were identified. At the end of the survey, 6 percent (576) of the children had died and 34 percent (3346) had been lost to observation. Fully one-third (1201) of the drop-out cases were lost to observation during the first month, that is, before the first interview following release from the hospital. Close to four-fifths of the children who

died (434) did so during the first twelve months of life.

[Table 1 about here]

We estimate the conditional probabilities of dying for successive inter-wave periods using a life table with two independent competing risks, mortality and sample losses. The level of infant mortality implied by the data is about .054 and the probability of dying before age two is .077.

[Table 2 about here]

3.2. How representative is the information from YIS?

As in most African countries, in Cameroon the levels of mortality within the boundaries of large cities are lower than for the sorrounding areas and the countryside. Infant mortality for Cameroon as a whole during the year 1978 was estimated from WFS and stood at about .106. This is roughly 1.9 times higher than the level estimated for Yaoundé from YIS. Clearly, the data from YIS cannot be representative of the entire country. But, is it a good reflection of conditions prevailing in Yaoundé? The evidence available to us suggests a positive answer. First, using WFS data, Ferry (1983) showed that approximately 90 percent of births in Yaoundé occurred in the 11 maternity wards

surveyed by YIS. Second, in separate studies Disaine and Garsen (1983) and Garsen (1985) estimate that about 88 percent of the population of Yaoundé was covered by YIS. Finally, Houehougbe (1985) and Garsen (1985) argue that the fraction of births that occurred outside the maternities surveyed by YIS is likely to have a neglegible impact on mortality estimates. In fact, if we conservately assume that between 10 and 12 percent of the births are missed and that these experience the highest levels of infant mortality observed for any group in the sample, we are led to errors of about 3.72 per 1,000 births, which is below half the length of a 95 percent confidence interval constructed with the estimated sampling error. Thus, although the mortality conditions assessd by YIS do not reflect national conditions, they should provide a very good yardstick indeed to evaluate conditions in Yaoundé and in similar cities in Cameroon.

3.3. The problem of selective losses to follow-up.

Admittedly, the most serious problems with the YIS data are the level and pattern of losses to follow up. The magnitude of the problem can be fully appreciated with a simple illustration: the estimated level of neonatal mortality is about .016 when losses to follow up are considered as censored cases. However, the estimate increases to .141 if all those who were lost to

observation before the first wave of household interviews had all died during the first month of life.[1] A similarly disconcerting range of estimates can be constructed for mortality in infancy and before age two. Although the assumption that all losses correspond to deaths is probably an unrealistic extreme, it is a benchmark to calibrate the dimensions of the problem.

The soundness of the study design not withstanding (van de Walle 1988), YIS collected information that has not been received favorably arguably because the magnitude of sample losses is considered to be close to devastating for an accurate appraisal of mortality conditions. In several studies dealing with the IFORD surveys in general, the problem of sample losses was singled out as a major conundrum. Rather than being random, so goes the argument, losses to follow up are related to mortality risks throughout the span of two years but particularly during the first month. Hence, careless use of the data could lead to serious underestimation of the levels of mortality and to under (over) estimates of the effects of important determinants (Mbacke and van de Walle 1989; van der Pol 1986; Ouaidou and van de Walle 1987). In a recent

[1] In an interesting exercise, Brouard has explored the effects of alternative hypotheses about the nature of the sample losses on estimates of infant mortality; however, he does not deal with the issue of biases on the estimated effects of mortality determinants (Brouard 1985).

publication exploring similar data from Burkina-Faso, Trussell and colleagues suggest that "the losses are extremely damaging to the study of mortality, as it is not possible to distinguish between a loss and a death, but the nature of the biases involved in a study of fertility is less obvious" (Trussell et al. 1989). In this paper, we argue that a more sanguine evaluation is possible and that a great deal of accurate information can be retrieved from the Survey inspite of the losses.

The mechanisms generating a loss to follow-up can be classified in three types. The first type is the least destructive for statistical inferences and refers to a loss that occurs due to factors unrelated to the phenomenon of interest (illness and mortality). This type of loss can be easily handled with models for independent risks to yield adjusted estimated levels and effects. The second type of mechanism corresponds to losses that occur due to factors that are directly or indirectly related to ill-health and mortality. This can occur, for example, if an eligible household cannot be located due to lack of sufficient address information and if (unmeasured) socioeconomic conditions affect the nature of the information about the elicited residence as well as illness and mortality. The third type of mechanism includes cases where the death of a child itself triggers a permanent or transient residence change that leads to a

final sample loss. It has been hypothesized that this phenomenon is likely to be in operation in Yaoundé since members of some ethnic groups are still bound to ritualistic practices that enforce changes of residence in the aftermath of the death of an infant (Brouard 1985). While the effects of the second type of loss can in theory at least be adequately purged by controlling for factors that create the association between sample losses and the event of interest, the third type is far less tractable and, as we illustrate later on, requires special estimation procedures.

Information about factors associated with sample losses is useful to at least elaborate a profile of those who drop out and to formulate hypotheses about the event in question and the events of interest to us. Table 3a displays the distribution of losses to follow-up by wave and by reasons for loss. Note that the heaviest losses during the first wave fall in the 'no information' category but that its importance is considerably reduced in subsequent waves. In fact, for waves two and higher, changes of residence but specially migration account for the bulk of losses. It is highly likely that most of the cases in the 'no information' category in the first wave at least are associated with lack of proper initial address. In fact, as displayed in Table 3b, about 71 percent of the losses that occur prior to the first wave of household interviews are directly

attributable to lack of address. Under these conditions one could argue that losses in the first month of life are more likely to belong to the first or second type of error than to the third one. Conversely, losses experienced in subsequent waves are more likely to be generated by a mechanism that leads from death of a child to sample loss. Regrettably the association between timing of the loss and mechanisms producing it cannot be studied since the individual information on which Table 3a is based was not included in the individual records released in the public use tape. In Section 4 we propose estimated ranges for the errors associated with losses to follow-up.

[Tables 3a and 3b about here]

3.4. The characteristics of the sample.

Table 4 displays information on selected background variables that will be used in subsequent analyses. In all cases we define categorical variables and show frequencies corresponding to the initial (maternity) sample, the sample for the first wave (month 1) and the sample at the onset of the second year of life (month 12). Although attrition due to death and losses to follow-up could conceivably result in important shifts of the observed distributions for

the variables, Table 4 reveals a strong sample stability and no changes worthy of notice.

[Table 4 about here]

4. MODELS AND ESTIMATION OF THE RELATIONS.

While we are chiefly interested in the effects of breastfeeding and pace of childbearing, we locate these factors within a more complex causal framework that includes proximate as well as background variables. Some of these variables were either not measured in the YIS or presented problems and were not included in the final analyses. The YIS lacks information on length of preceding birth interval so it is not included in subsequent analysis. Unlike commonly available retrospective data, YIS includes information on health conditions at birth (e.g., birth weight), episodes of illness experienced during interwaves period (diarrhea and measles being the two most important), and information on supplementation and feeding. The models we formulate are designed to minimize some of the problems of simultaneity, spuriousness and selection that were discussed before.

4.1. Two-state hazard models in a context of independent competing risks.

The simplest model we pose is a two-state model for the risk of dying at any time within the course of the study:

$$\mu(t; X(t)) = \mu_0(t) exp(\beta X(t)) \tag{1}$$

Where $\mu(t; X(t))$ is the risk of dying in (t, t+dt) for an individual with a vector of characteristics $X(t)$, $\mu_0(t)$ is a baseline hazard and β is a vector of effects.

There are two assumptions on which this model rests. The first is that the relevant covariates are those explicitly recognized and that there are no omitted covariates affecting mortality risks (conditional homogeneity). The second assumption is that, conditional on measured covariates, sample losses can be treated as a competing and independent risk (conditional independence). Neither of these two assumptions is likely to be correct. In particular, the independence assumption is tantamount to asserting that losses to follow-up are totally explained by the first mechanism described in Section 3, a somewhat naive assumption. However, as we will show later, violations to these two assumptions are unlikely to change much our inferences regarding

the effects of breastfeeding and following conception.

Although for most variables that change over time we have available information to the month, we have adopted a conservative strategy to minimize errors of response. In particular, we will estimate effects assuming that we observe events in discrete segments: 0, 1-3, 4-7, 8-11, 12-15 and 16-23. With one exception to be noted later, when a death occurs within a segment it is assigned to the middle of the segment. Experiments with alternative solutions, including the use of observed age at death, do not change our results in any significant way. Censoring times are assigned to the middle of the segment also because, unlike the timing of death, their exact values are not known. We include four time dependent covariates: breastfeeding, timing of the following conception, episodes of diarrhea and episodes of measles. Again, with one exception that will be described later, we use a general rule to assign values to the time dependent covariates. This rule is expressed as follows: let $\mathbf{X(t)}$ be the value of the time dependent covariate at time $\mathbf{t}$ and $\mathbf{t} < \mathbf{T}$, where $\mathbf{T}$ is the lower value of an age segment selected for analysis (T= 0, 1, 4, 8, 12, 16). Its value can be either 0 or 1 depending on the occurrence (non-occurrence) of an event *before* the child enters the segment $\mathbf{(T,T+n)}$. For example, the value of breastfeeding for a child who survives to age 4 months will be 1 if he/she

stopped breastfeeding any time *before* month 4 and 0 otherwise. Similarly, the value of following conception will be 1 if the mother experienced a conception sometime before the child became 4 months old.[2]

Estimation of model (1) proceeds under two different assumptions about the baseline hazard, $\mu_0(t)$. One was that it could be represented as a piece-wise constant with nodes at 1-3, 4-7, 8-11, 12-15, and 16-23. The second was that it could be efficiently represented by a Gompertz hazard. In what follows we discuss results obtained under the latter representation since it requires a smaller number of parameters. It is important to emphasize that none of the conclusions we reach would change if instead we select the piece-wise model or even a Gompertz with ages at death expressed in months. The baseline model reproduces infant mortality quite well predicting a level of about .057 instead of the observed level of .054.

The first five columns of Table 5 display the results of estimating suc-

[2]A further fine-tuning of the definition of timing of following conception is, however, required. Since in each of the segments there are following conceptions associated with infant deaths that occur within the same age segment, we identify and flag a following conception as occurring before the death whenever the death was preceded by return to menses. The idea behind this coding scheme is that if menses precedes the death of a child, even if by a short time, it is more likely that conception would have preceded the death rather than the other way around.

cessively more complicated models for the age segment 0 to 24 months. The sixth column of the table shows the estimates obtained when we constrain the sample only to those who are under observation at the beginning of the first month of life.

The first (column 1) contains only the effects of background covariates. Among them are remote determinants such as ethnicity and more proximate determinants such as birth order and age of mother at birth. It also includes a measure of relatively poor health care at the time of birth (main maternity) and a control for status of address that should attenuate the biases associated with losses to follow-up due to the second source of losses discussed above. The second model (column 2) includes the effects of breastfeeding and following conception but omits the possibly confounding effects of antecedent illnesses. The effects of breastfeeding are fairly strong and statistically significant even though we are controlling for birth weight. The effects of a following conception are strong and in the expected direction. The relative risk associated with breastfeeding discontinuation is about 1.73 while the relative risk associated with a following conception is 1.54. Although these are hefty impacts, they are within the range estimated with very different data sets (Lantz et al. 1992; Palloni 1986; Palloni and Millman 1986; Hobcraft et al. 1983). It is important

to note that these effects represent average effects over a 24-month period even though, as hypothesized above, the effects of both breastfeeding and following conception may change over time.[3]

Does a preceding illness inflate the effects of breastfeeding? To answer this question we introduce two control variables measuring the presence of illness in a preceding period. We singled out two illnesses that are the most important during this stage in the life of African children, measles and diarrhea. The results in model 3 (column 3) show that the direct impact of measles is very strong whereas that of diarrhea is quite weak. While this is interesting in itself, the crucial point is another one: although the magnitude of the coefficient for breastfeeding is reduced, as should be expected, the changes are hardly noticeable. This is an important result since it strongly supports the contention that estimates for breastfeeding obtained without controlling for preceding illness are likely to be genuine rather pure artifacts.

How large would the bias of the estimates of breastfeeding effects be if we did not control for birth weight? The fourth model (column 4) provides an answer: they would be very small. In fact, the differences in the estimates are trivial (-.55 versus -.61). This finding is in agreement with others of similar

[3]We show later that the effects of breastfeeding and of following conception decrease steadily with age.

nature in the literature. Working with different countries but with comparable models and definitions, Montgomery (Montgomery et al. 1986), Millman (Millman and Cooksey 1987) and Goldman (Goldman et al. 1989) confirmed that the effects of breastfeeding remain strong even after controlling for birth weight.

Although there are indications that birth weight is a good proxy for health status at birth (note the behavior of the effects for the variables multiplicity, main maternity and early age of mother at birth), one could still insist that there are health conditions at birth that may impede normal breastfeeding that are not captured by birth weight. To circumvent some of the biases one could estimate a model for the mortality risks conditioning on survival to the first month. The estimates of breastfeeding should be less contaminated by the influence of health status at birth. The results of such a model are displayed in the last column of the table. Note that the magnitude of the coefficient for breastfeeding moves sharply downward though remaining statistically significant. The relative risks of death after breastfeeding discontinuation drop from 1.73 to 1.42 but they are still of considerable magnitude.[1]

[1]It is important to note that whether there is a bias or not, one should expect that the effects of breastfeeding conditional on survival to the first month would be lower than the unconditional effects since, by hypothesis, the beneficial effects of breastfeeding decline

The final problem raised with regard to the effects of breastfeeding is that as an outcome of a behavioral choice, the practice of lactation may be a proxy for other (unmeasured) behaviors that are associated with health care. The net result could be either an attenuation of effects (most likely) or an upward bias (least likely). Testing this proposition requires modelling simultaneously the choice of breastfeeding pattern and mortality risks. An alternative is to control for variables that are responsible for the association between those behaviors. In our models we have included several background characteristics such as ethnic group and education that are good proxies for the likelihood of traditional (modern) behaviors. In addition, we estimated models where we controlled for accessibility and use of medical care. Since the effect of breastfeeding remains unaltered we conclude that it is unlikely that its magnitude or direction could be biased as conjectured.[5]

Column 5 of the table displays the estimates after the inclusion of the indicator for the timing of following conception and column 6 shows the results of omitting breastfeeding among the covariates. As expected, the effect of timing of following conception is strong and positive. Although a control for

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sharply over time.

[5]Similar results have been obtained by other researchers using DHS and WFS data. See Boerma et al. 1992; Lantz et al. 1992.

breastfeeding attenuates the effects of following conception (compare estimates for FC in columns 3 and 5), its effects remain strong and significant. This means that breastfeeding is not the only (nor the most important) mediating mechanism between conception and infant death. This result confirms findings from the WFS and other data sets all of which uniformely indicate that timing of following conception has a strong influence on survival outcomes but that its operation has little to do with shifts in the patterns of lactation induced by the onset of a new pregnancy (Palloni and Millman 1986; Lantz et al. 1992). Recent work on DHS data reveals a similar pattern in relation to the effects of previous birth intervals (Boerma et al. 1992).

An important issue that can be addressed with YIS data is whether the definition of the timing of following conception that has been used in previous studies (Palloni and Millman 1986; Retherford et al. 1989; Hobcraft et al. 1984) does in fact lead to underestimates of effects. This is likely to occur when the definition of timing of following conception does not take into account events occurring within the age segment of reference but only those prior to its initiation (Lantz et al. 1992). Thus, for example, for the segment (**T**, **T+n**) the conventional definition of following conception relies on information on conceptions before **T** and does not reflect those occurring within the age

segment even though these may indeed have deleterious consequences for the health of a child. Of course the problem only exists when, as in YIS, the timing of some events is not in continuous form or, as in cases involving analysis of WFS and DHS, when the analytic model forces a discrete definition of segments. Our results, however, indicate that the conventional definition of the timing of following conception leads to estimated effects that are larger than those estimated with the fine-tuned definition of following conception (results not shown). The implication of this finding is either that the conjecture stated above is incorrect or that the procedure we used to infer a following conception from the detailed data on ages at death and of timing of ressumption of menses is too coarse to enable us to fine-tune the indicator of timing of following conception.

Are the effects of breastfeeding and following conception fixed over time?

The pattern of effects over time could be revealing of the mechanisms that may be in operation. Thus, for example, a decreasing impact of the effects of the timing of following conception is consistent with the interpretation that breastfeeding is the key mediating mechanism but it does not follow at all from an interpretation that assigns more importance to sibling competition

or deterioration of maternal care. Similarly, a decreasing effect of breastfeeding would be revealing of an increased gap between nutritional demands and nutritional content of breastmilk. Indeed, it has long been suspected that the benefits of breastfeeding should begin to wear down before the end of the first year of life whether their source is nutritional intake or enhanced immunocompetence. In previous studies the estimated effects of both variables are sharply reduced after the sixth month of life (Palloni and Millman 1986; Palloni and Tienda 1986).

To test for the variability of the effects of breastfeeding and following conception over time we estimate two models where the corresponding indicators were allowed to have different effects throughout the first and second year of life. This was achieved by defining a set of interaction effects between the variable of interest and time. The first model (model I) with interaction effects does not allow for unmeasured heterogeneity whereas the second model (model II) does so. The results, displayed in Table 6, are consistent with expectations, namely, that the effects of both breastfeeding and following conception diminish with age. Note that the relative risks for those not breastfeeding decrease from 10.1 (exp(2.31)) at ages below 4 months to .44 (exp(2.31-3.14)) at ages above 16 months. Similarly, the relative risks accom-

panying an early conception drop from about 44.3 to about 2.25 over the same age segments. Although at least some of the sharp decline in the effects of breastfeeding is due to an attenuation of the biases affecting the estimates of breastfeeding effects at early ages, the second column of Table 6 indicates that the estimates of interaction effects are robust to the presence of unmeasured characteristics.

4.2. Models with unmeasured heterogeneity.

The possibility that neither episodes of illness or birth weight capture well health conditions that simultaneously prevent breastfeeding and increase mortality risks is certainly not farfetched. Within a hazard model framework, however, it is possible to test whether or not our results are sensitive to omitted *fixed* covariates. This can be done estimating a two-state model with non-parametric heterogeneity (Heckman and Singer 1984). We choose non-parameteric heterogeneity since when parametric models are used the results are very sensitive to the distribution imposed on the unknown covariate(s) (Trussell and Richards 1985). In addition, since we are fairly certain that the duration structure of early child mortality is well represented by a Gompertz, we are in a position to minimize the identification problems that plague hazard

models with unmeasured heterogeneity (parametric or non-parametric).[6]

We estimate two models. The first model is one where all variables are included but we add unmeasured heterogeneity. The estimates of breastfeeding and timing of following conception are then compared to those of an equivalent model with no unmeasured heterogeneity (Model 3 in Table 5). If the estimated effects show no change, our inferences regarding breastfeeding and following conception receive further confirmation. Instead, if the hypotheses described in Section 2 were correct and indeed birth weight was a poor proxy for health conditions, we would expect important changes in the effects of one or both variables.

In the second model we exclude birth weight but include unmeasured heterogeneity. The results are then compared with those of a model where birth weight is included but unmeasured heterogeneity is omitted. If birth weight were tapping properly health conditions that are otherwise unmeasured, the results of one should be quite close to those from the other.

A comparison of columns 1 and 2 in Table 7 shows that the estimates

[6]To the extent that models with unmeasured heterogeneity are only estimable when the unmeasured covariates are fixed, the introduction of heterogeneity will do little to resolve the problem posed by unmeasured illnesses since their incidence and seriousness varies over time.

of breastfeeding and following conception when we introduce non-paramteric heterogeneity with two points of support (additional points of support could not be estimated) are virtually unchanged. A comparison of columns 3 and 4 in the same table reveals a similar type of stability: the model with no birth weight and no unmeasured heterogeneity leads to virtually the same estimates of breastfeeding and following conception than a model with unmeasured heterogeneity. There are two alternative interpretations for these results. The first is that all the unmeasured heterogeneity that can be captured with fixed covariates is indeed captured by birth weight. This would explain why the estimates in columns 3 and 4 are so much alike. The second interpretation is that birth weight is a mediocre proxy for unmeasured health conditions and that these conditions are related to some of the covariates in the model. Such relation violates a fundamental assumption of the models with unmeasured heterogeneity and there is no reason to expect that results should be different when heterogeneity is taken into account than when it is not.

These tests lead to an ambiguous conclusion: either there are no sources of heterogeneity affecting our results (even in the absence of controls for birth weight) or, if they exist at all, they are neither measured by birth weight nor do they belong to the class of covariates that are *fixed* and independent of

measured covariates.

4.3. Modelling losses to follow-up: dependent and informative censoring.

Are the results that we obtain robust to different processes leading to sample losses? The answer to this question is positive if the assumption of independent censoring is accurate or, alternatively, if losses to follow-up are generated by what we called mechanism *No 1* in Section 2.

If mechanism *No 2* is the most important, the procedures that we have followed may be in error since then the censoring mechanism becomes at least informative. However, if we control for all factors that cause the association between censoring and mortality, we could invoke again an argument of independent censoring: conditional on the factors that cause the association, censoring and the event of interest are, in fact, independent. Among the variables that we controlled for are income, education, residence and the status of the elicited address, all of which are good indicators of conditions that may increase (decrease) the likelihood of loss to follow-up and increase (decrease) the opportunities for ill-health. These controls, however, alter slightly the value of estimates and standard errors. In particular, the inclusion (exclusion) of a dummy variable for missing address affects only trivially the estimates of the time-dependent covariates and is itself statistically insignificant.

An alternative procedure to assess the magnitude of the problem is to utilize a selection model to investigate to what extent the probability of sample loss is correlated with the risk of dying. Since selection models have been formulated for several combinations of polytomous and continuous variables but not within a hazard context, we reformulate our model slightly without any loss of generality. The reformulation defines three age segments, 1-6, 6-12 and 12-24. We then predict the probability that a child will drop out before reaching the beginning of each segment and then estimate the conditional probability of dying within each segment after correcting for selection. The equation for the conditional probability of dying in each segment includes a correction factor which is equivalent to the predicted hazard of being lost to follow-up within the age segment. A comparison of the estimates with and without correction for selection (not shown, available on request) reveal changes of only modest magnitude. We conclude from this exercise that selective attrition is likely to be unimportant. But what if the assumptions on which the selection model rests are not appropriate? The consequences of this failure are explored below.

Lurking in the background flickers the most ominous possibility of all, namely, that censoring is a direct result of the death of the child. This was identified before as a mechanism of type *No 3*. The operation of the mechanism

can be formalized as follows: an individual is exposed to two types of censoring. The first is conventional, non-informative and independent. The second is censoring that follows with some probability as a direct result of the death of the child. In this case one could imagine the operation of a random mechanism that assigns children deaths into two groups: those that are identified as deaths and those that are confused with censored cases. The likelihood of the sample will then be composed of the product of three quantities: the likelihood for true censored cases, the likelihood for identified deaths and the likelihood for deaths that are confused with censored cases. More formally, the likelihood for a case i is given by:

$$L_i = exp[-\int_0^{t_i} \mu(v)dv](\mu(t_i)\rho_i)^{c}_{1i}(\mu(t_i)(1-\rho_i)^{c}_{2i} \qquad (2)$$

where c_{1i} is 1 if the individual belongs to the class of well identified deaths, c_{2i} is 1 if the individual belongs to the class of deaths that are mixed with censored cases, and ρ_i is the probability that the random mechanism assigns the death to the class of identifiable deaths.[7] The estimation problem is obvious: we have no observational basis to determine which among the *observed* censored cases

[7]The mechanism could conceivably be dependent on characteristics of the individual, such as ethnic group. To allow for this possibility the probability ρ is denoted with a subscript.

belongs to one class and which to the other. Thus the likelihood is undefined. Assuming that ρ is unity restores the tractability of the problem even though one must continue to assume independent and non-informative censoring.

Although exact estimates are not possible, we can estimate the upper and lower bound of the range within which the estimates must be contained. Indeed, one extreme of the range is retrieved from a model that assumes that all censored cases correspond to survivors, that is, that none among them died before the second year of life (ρ equals unity or, equivalently, all deaths are well identified). Another extreme of the range is to assume that ρ is identically zero, that is, that all censored cases correspond to unidentified deaths. To be sure, the range thus established is not a true range but a 'pseudo' range (see below). Columns 1 and 2 in Table 8 display the estimated coefficients of selected variables under these two extreme assumptions in the entire sample. Columns 3 and 4 display the the estimates corresponding to the sample that survived to age 1 month. With the exception of the effects for following conception, the effects of all variables are quite robust and involve fairly narrow ranges. But the effects of following conception may swing widely: if all censored cases are survivors, the relative risks are equivalent to 2.32 (exp(.84)); if all censored cases correspond to unindentified deaths, the relative

risks reverse direction and become .58. This can only happen if, as it is indeed the case in our sample, most among children whose mothers do not experience an early conception are NOT drop-outs.

The pseudo-ranges established above are, in all likelihood, unnecessarily wide and conservative. Although they are useful to confirm the important role of breastfeeding, birth weight and illnesses, they cast some doubt on the validitiy of the effects of following conception.

The estimation of an exact and less conservative range requires a formidable amount of computation since one would need to calculate the likelihood function for the sample under all possible assignments of the censored cases. This may be an impossibly taxing task in many computing environments. In fact, if there are C censored cases, the likelihood function would have to be computed 2^C times to generate the full range of possibilities.

5. CONCLUSIONS.

The most important result of this paper is that YIS fully confirms the findings from WFS, DHS and other data sources regarding the influence of breastfeeding and birth intervals on infant and early child mortality. In particular, the effects of lactation during infancy are strong and in the expected direction. The timing of following conception exerts a powerful effect on the

mortality risks experienced by the index child. Neither of these effects is altered when we account for measured or unmeasured sources of heterogeneity. This confirms the conjecture made by several researchers that the importance of breastfeeding and timing of following (and preceding) conception (birth) are more than artifacts of the data, models used or measurement errors.

While most tests suggest that the substantial losses to follow-up in YIS do not affect the results to any great extent, we are unable to provide an efficient range for the estimates of interest. However, a conservative 'pseudo-range,' obtained under extreme assumptions about drop-outs, indicates that our estimates for breastfeeding are not too far off the mark but that more uncertainity ought to be placed on the estimates of effects of following conception.

Similarly, the estimation of models with unmeasured heterogenity helps to dismiss the potential influence of some artifacts. There is a possibility, however, that the effects of breastfeeding and following conception are contaminated by omitted time-varying covariates or fixed covariates with strong associations with measured variables. Since the statistical tools for dealing with these two problems are not available at this time, our conclusions cannot pass the screening of tests stronger than those already applied.

Table 1. Evolution of Yaounde Sample by Wave and Child's Status

	Wave 0	**Wave 1**	**Wave 2**	**Wave 3**	**Wave 4**	**Wave 5**	**Wave 6**	**Wave 7**
Alive	9729	8398	7727	7146	6658	6343	6049	5852
Dead	45	130	69	79	111	53	50	39
Dropout	--	1201	602	502	377	262	244	158
Total	9774	9729	8398	7727	7146	6658	6343	6049

Table 2. Life Table of Children in Yaounde

Age (X,X+n) in months	(0,1)	(1,4)	(4,8)	(8,12)	(12,16)	(16,20)	(20,24)	Total
Q(X,X+n) (in p. 1000)	19.1	8.5	10.6	16.0	8.1	8.0	6.5	74.4

Table 3a. Proportional Distributions of Losses to Followo-Up by Wave and by Reasons Explaining the Loss

Reason for Loss	Wave 1	Wave 2	Wave 3	Wave 4	Wave 5	Wave 6	Wave 7	Total
No information	979	78	31	46	12	62	9	1217
	(.82)	(.13)	(.07)	(.12)	(.05)	(.25)	(.06)	
Moved residence	93	224	212	123	71	61	40	824
	(.08)	(.37)	(.42)	(.33)	(.27)	(.25)	(.25)	
Outmigration	129	300	259	208	179	121	109	1305
	(.11)	(.50)	(.52)	(.55)	(.68)	(.50)	(.69)	
Total	1201	602	502	377	262	244	158	3346

Source: Adapted from F. Gubry et al. (1987).

Table 3b. Proportional Distribution of Losses to Follow-up by Status of Residence Address

Status of Residence	Wave 1	Wave 2	Wave 3	Wave 4	Wave 5	Wave 6	Wave 7	Total
Known	3535	178	196	156	107	95	81	1166
Unknown	848 (.71)	424 (.71)	306 (.61)	221 (.59)	155 (.60)	149 (.61)	77 (.49)	2180

Table 4. Mean Values of Selected Background Variables by Age Intervals

Variables	0 - 1 month	1 - 12 months	12 - 24 months
Main Maternity	0.53	0.51	0-.50
Ethnic1	0.40	0.41	0.40
Ethnic2	0.20	0.21	0.20
Ethnic3	0.06	0.05	0.05
Unknown Address	0.52	0.49	0.46
Married Mother	0.72	0.74	0.76
Mother's Education	0.85	0.86	0.85
Number of Observations	9774	8398	6658

Table 5. Estimated Effects of Background and Proximate Determinants on Early Child Mortality [a]

	I	II	III	IV	V	VI
Intercept	-5.04(.22)	-4.88(.22)	-4.86(.22)	-4.68(.22)	-5.15(.22)	-6.16(.32)
Slope	-.07(.007)	-.10(.008)	-.12(.09)	-.12(.008)	-.12(.008)	-.07(.010)
Main Maternity	.38(.09)	.37(.09)	.36(.09)	.44(.09)	.30(.09)	.34(.11)
Ethnic1	.12(.16)	.001(.16)	-.02(.16)	.04(.15)	.05(.16)	.00(.20)
Ethnic2	.42(.11)	.18(.11)	.05(.12)	.18(.12)	.14(.12)	.27(.14)
Ethnic3	.35(.14)	.20(.14)	.18(.14)	.24(.14)	.25(.14)	.25(.17)
Unknown Address	.07(.10)	.16(.10)	.17(.10)	.17(.10)	.09(.10)	.09(.12)
Married Mother	-.34(.11)	-.22(.11)	-.23(.11)	-.25(.11)	-.35(.11)	-.34(.14)
Mother's Education	-.27(.12)	-.36(.12)	-.40(.12)	-.36(.12)	-.35(.12)	-.14(.16)
Teenage	.16(.12)	.17(.12)	.17(.12)	.22(.12)	.12(.12)	.20(.16)
Parity 2,3	-.10(.12)	.05(.12)	.06(.12)	-.06(.12)	.02(.12)	.14(.15)
Parity 4+	-.24(.14)	-.02(.13)	-.02(.13)	-.18(.13)	-.08(.13)	.10(.17)
Multiple Birth	1.09(.14)	.17(.14)	.08(.14)	.77(.14)	.31(.14)	.02(.24)
Season	-.23(.09)	-.20(.09)	-.26(.09)	-.26(.09)	-.29(.09)	.20(.10)
Breastfeeding	--	-.85(.10)	-.90(.10)	-.94(.10)	--	-.33(.11)
Birth Weight	--	1.44(.10)	1.47(.10)	--	1.52(.10)	.88(.14)
FC	--	.72(.15)	.81(.15)	.80(.15)	1.02(.15)	.68(.15)
Diarrhea	--	--	.17(.14)	.17(.15)	.16(.15)	.09(.15)
Measles	--	--	2.21(.10)	2.67(.10)	2.17(.10)	2.35(.11)
Negative Log Likelihood	3721	3661	3404	3493	3450	2560
Number of Parameters	14	17	19	18	18	19
N	9774	9774	9774	9774	9774	8398

[a] Numbers in parentheses are estimated standard errors.

Table 6. Models with True Dependent Effects of Breastfeeding and Following Conception

	I[a]	II[b]
Following Conception		
Main Effects	3.79(.39)	3.65(.41)
Effects at 4-7	-1.70(.39)	-1.63(.40)
8-11	-1.36(.29)	-1.29(.31)
12-15	-.91(.35)	- .98(.35)
16+	-2.98(.39)	-2.98(.41)
Breastfeeding		
Main Effects	-2.31(.25)	-2.20(.25)
Effects at 4-7	1.29(.30)	1.16(.30)
8-11	2.20(.29)	2.02(.29)
12-15	2.21(.32)	2.02(.33)
16+	3.14(.33)	2.95(.33)
Negative Log Likelihood	3165	3166
Number of Parameters	27	30
N	9774	9774

[a] Model I is like Model III in Table 5, except for the introduction of the interaction effects for age segments 4-7, 8-11, 12-15, and 16+.

[b] Model II is like Model I, but includes control for unmeasured heterogeneity (2 support points)

Table 7. Models With Unmeasured Heterogeneity

	I[a]	II	III[b]	IV
Intercept	-4.86(.22)	7.74(.40)	-4.68(.22)	-6.39(32.38)
Slope	-.12(.09)	-.08(.01)	-.12(.01)	-.12(.01)
Main Maternity	0.36(.09)	0.41(.12)	0.44(.09)	0.45(.09)
Ethnic1	-.02(.16)	0.01(.21)	0.04(.15)	0.03(.16)
Ethnic2	0.05(.12)	0.09(.15)	0.18(.12)	0.19(.12)
Ethnic3	0.18(.14)	0.17(.18)	0.24(.14)	0.27(.14)
Unknown Address	0.16(.19)	0.14(.13)	0.17(.10)	0.16(.10)
Married Mother	-.23(.11)	-.26(.15)	-.25(.11)	-.26(.11)
Mother's Education	-.40(.12)	-.35(.16)	-.36(.12)	-.34(.12)
Teenage	0.17(.12)	0.09(.17)	0.22(.12)	0.20(.12)
Parity 2,3	0.06(.12)	-.06(.17)	-.06(.12)	-.06(.12)
Parity 4+	-.07(.13)	-.15(.19)	-.13(.14)	-.17(.14)
Multiple Births	0.08(.14)	0.56(.24)	0.77(.14)	0.77(.15)
Season	-.26(.09)	-.23(.10)	-.26(.09)	--
Breastfeeding	-.90(.10)	-.93(.13)	-.94(.10)	-.94(.11)
Birth Weight	1.47(.10)	2.46(.19)	--	--
FC	0.81(.15)	0.81(.17)	0.80(.15)	0.78(.15)
Diarrhea	0.17(.14)	0.24(.10)	0.17(.17)	0.14(.17)
Measles	2.21(.10)	2.64(.12)	2.67(.11)	2.16(.12)
Factor	-	4.49(.25)	--	2.00(28.8)
Probability	-	0.84(.02)	--	0.37(4.52)
Negative Log Likelihood	3904	3353	3493	3492
Number of Parameters	19	21	18	20
N	9774	9774	9774	9774

[a] From Table 5, Column III

[b] From Table 5, Column IV

Table 8. Selected Estimates From Models that Assume Different Survival Outcomes for Dropouts[a]

Variable	Model I	Model II	Model III	Model IV
Breastfeeding	-.91(.10)	-.77(.04)	-.33(.11)	0.09(.04)
Birth Weight	1.48(.10)	0.32(.05)	0.88(.14)	0.21(.07)
FC	0.84(.15)	-.55(.10)	0.70(.15)	-.63(.10)
Diarrhea	0.18(.14)	0.04(.06)	0.09(.15)	0.00(.07)
Measles	2.23(.103)	0.07(.08)	2.37(.11)	0.13(.09)
Negative Log Likelihood	3413	15795	2565	11893
Number of Observations	19	19	19	19
N	9774	9774	8398	8398

[a] Models I and III assume that all dropouts survive to the end of the follow-up. Models II and IV assume that all dropouts die during the interwave period in which they drop out.

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