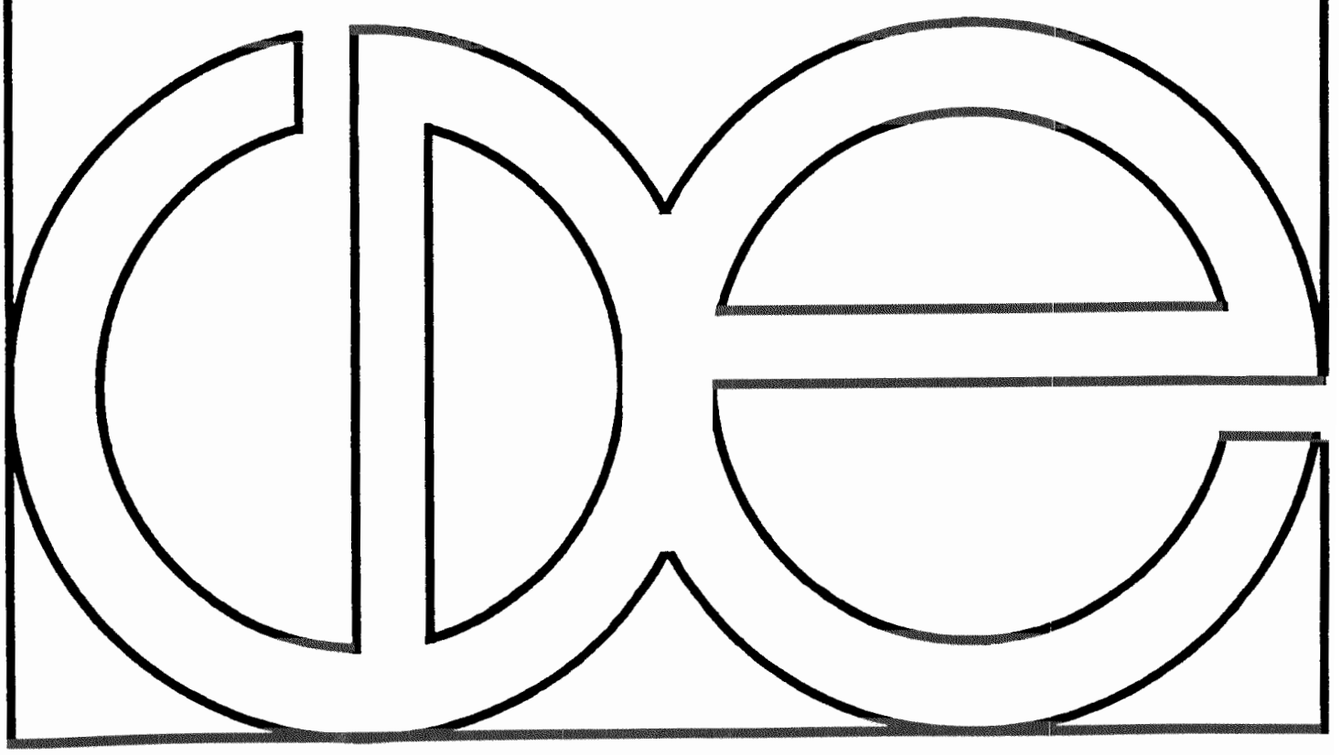
Center for Demography and Ecology

University of Wisconsin-Madison

EPIDEMIOLOGY AND CONTROL OF INFANT AND EARLY CHILDHOOD MALARIA IN AFRICA: A COMPETING RISK ANALYSIS

Barthélémy Kuate Defo

CDE Working Paper 93-13



EPIDEMIOLOGY AND CONTROL OF INFANT AND EARLY CHILDHOOD MALARIA IN AFRICA: A COMPETING RISKS ANALYSIS

Barthélémy Kuate Defo

Center for Demography and Ecology
University of Wisconsin-Madison
4412 Social Science Building
1180 Observatory Drive
Madison, WI 53706
(Correspondence address)
&
Department of Preventive Medicine
University of Wisconsin-Madison Medical School
504 N. Walnut Street
Madison, WI 53705

This research was made possible by a fellowship from the Rockefeller Foundation. Computations were performed using facilities of the Center for Demography and Ecology at the University of Wisconsin-Madison, which are supported by the Center for Population Research of the National Institute for Child Health and Human Development (HD-5876).

ABSTRACT

This paper investigates the determinants of early child malarial morbidity and mortality and the extent to which determinants of malaria-associated mortality are also risk factors of mortality ascribed to causes other than malaria.

Two sets of findings emerge from this study. First, the prevalence and severity of malaria in the first year of life is greater than in the second year of life. Second, malaria-associated mortality covaries with household conditions, parity of the mother, prenatal care visits, infant feeding practices, antecedent child morbidity, and immunization status. To be sure, these factors are also important determinants of mortality related to other causes.

EPIDEMIOLOGY AND CONTROL OF INFANT AND EARLY CHILDHOOD MALARIA IN AFRICA: A COMPETING RISKS ANALYSIS

1. Introduction

In most tropical countries, malaria is a major public health problem in childhood and thereafter (MacDonald, 1957; Edington, 1967; Balint and Anand, 1979; Brabin, 1983; Fleming et al., 1979; Molineaux, 1985; Najera, 1979, 1989). Malaria transmission is higher in sub-Saharan Africa than anywhere else in the world and remains one of the most difficult epidemiological challenges in sub-Saharan Africa, where the situation appears to be changing for the worse in many countries (Ofosu-Amaah, 1991). Malaria morbidity is mainly a feature of infancy and may account for 30-35 percent of the cases in persons seeking health care at rural dispensaries in Africa (Bradley, 1991).

There is a possibility that the slow progress in malaria control in Africa is partly due to the fact that emphasis has been placed on chemotherapy and chemoprophylaxis as the most effective means of control, and very little is known about the socio-environmental and epidemiological determinants of this life-threatening condition. In fact, studies in Congo at Kinkala and in Burkina Faso at Bobo Dioulassou showed little effect of intense chemoprophylaxis and chemotherapy on malaria mortality (Feachem and Jamison, 1991). It is against this background that this study is designed. The general strategy is based on the "intermediate framework" approach (Mosley and Chen, 1984; van Norren and van Vianen, 1986;

Johansson and Mosk, 1987). We start from the perspective that when analyzing diseases, it is essential to distinguish between exposure to contagion and resistance to infection.

2. Major relations

(a) The role of household factors

Household factors are expected to exert a relatively direct impact on malaria morbidity and mortality because this condition is closely related to the risk of exposure to infectious agents (germs, vectors, etc) and the extent to which residents are protected from environmental pollution and degradation. Such effects have been suggested by previous investigators (Hinkle and Wolff, 1957; Mims, 1991; Molineaux, 1985; Mosley, 1982; Pelassy, 1976; Bagster, 1949; MacDonald, 1957). The household factors involve: a) construction materials of the dwelling; b) source of power (electricity) and water supply; and c) extent of overcrowding.

El Samani et al. (1987) found that malaria was inversely associated with ownership of a refrigerator, and indicators of crowding were the best predictors of the risk of malaria. Population density was found to be an important factor in malaria transmission. Fewer cases of the disease were observed in households with three or more rooms and more malaria was observed in households with more than five people. Fewer cases of malaria were reported from children of monogamous households. This may well be a direct function of the human reservoir of the parasite where the vector is present (Mims, 1991; MacDonald, 1957). Similar results have been shown in previous investigations where the incidence of malaria was

significantly higher in crowded houses (e.g., Banguero, 1984).

(b) The role of maternal reproductive patterns

Maternal reproductive patterns (age at childbearing and parity) have been suggested as a potential determinant of malaria (MacLeod, 1988). In areas that are endemic for malaria, the prevalence of parasitaemia is higher among pregnant than nonpregnant women, especially among primiparae (Gilles et al., 1969; Bray and Anderson, 1979; Brabin, 1983). These studies suggest a greater susceptibility of primiparae to malaria. The disease is then transmitted to their children through the placenta (MacGregor and Avery, 1974; MacDonald, 1957).

(c) The role of breastfeeding patterns

Many studies have shown that the pattern of breastfeeding is a major determinant of growth faltering and the prevalence of infectious diseases in most developing countries (Rowland et al., 1978; Majumdar and Ghose, 1982; Mata, 1978; Mckigney, 1971; Gibson, 1990; Ogra and Ogra, 1978; Winikoff, 1982; Feachem and Koblinsky, 1983; Feachem and Jamison, 1991; Morley, 1973; Monjour et al., 1982; Welsh and May, 1979). The typical epidemy of these diseases has been shown to follow the period of weaning (Morley, 1973; Jelliffe, 1973; Jelliffe and Jelliffe, 1978; Kirkwood, 1991a, 1991b; Goldman and Smith, 1973; Hanson and Winberg, 1972; Greenwood et al., 1972; Greenwood et al., 1981a, 1981b; Mata et al., 1967; Mata, 1978; Rowland et al., 1978; Wenlock, 1979, 1981; Clemens et al., 1990; Boerma et al., 1991).

Nonetheless, the nature, direction and magnitude of the effects of full, partial and no breastfeeding remain conjectural. The protective influence of breastfeed-

ing against the hazards of disease and the environment should not be taken for granted. Whether the protective effect of breast milk is from its conferred anti-infective properties, improved hygiene, or from nutritional factors per se is not entirely clear from studies to date. Demonstration of potential mechanisms of breast milk protection from malarial transmission are of greatest interest if they actually operate in the community. In Kenya, it has been shown that in healthy areas where malaria is not prevalent, both full and partial breastfeeding reduces mortality (Eelens, 1983). In the areas where malaria is rife, only full breastfeeding has any effect on mortality.

(d) The role of antecedent morbidity

Later infection-associated childhood mortality may be related to infectious experiences during the first months of life. In developing countries, the majority of childhood deaths are a consequence of recurrent bouts of infectious diseases and nutritional deficiencies (Koster et al., 1981; Mosley, 1985; Aaby et al., 1993; Balint and Anand, 1979). The role of infectious disease in mediating the effect of the family dwelling on child mortality has been noted by others (Feachem, 1981). A disease might coexist with other diseases, the higher incidence of which diseases can be due to reinfections (i.e., cases in which the immunity that had been acquired did not last for very long). There might be also 'endogenous' infection (reinfection is the result of an old infection) and 'exogenous' infection (reinfection comes from an active case) for diseases which do not have the life-long immunity property of measles. In all these instances, later childhood mortality may be related to infectious experiences during the earlier months of life. Clearly, children are

rendered more delicate by previous infectious diseases. Indeed, many viral, bacterial or protozoal infections impair cell-mediated immunity, humoral immunity or the functions of neutrophil polymorphs, the former being the most frequently affected (Greenwood and Whittle, 1981a, 1981b; Kimati et al., 1981). *P. falciparum* malaria and measles are among the most notorious and are commonly complicated by pneumonia, gastrointestinal infections and respiratory infections (Fleming and Werblinska, 1982). The role of intercurrent infections and other health conditions of the child in the exacerbation of mortality is not well documented, particularly their potential causality. It has been suggested by previous investigators that there might be variations in immunological responses by individuals to repeated attacks of malaria in different localities as well as within localities (Jensen et al., 1984; Hinkle and Wolff, 1957; Kimati et al., 1981; MacDonald, 1957; Monjour et al., 1982; Russell et al., 1963; Smedman et al., 1986; Wilson et al., 1950; Carswell et al., 1981). Thus, we hypothesize a role of previous episodes of malaria and other infections on the prevalence and severity of malaria. Experimental work on the interaction of malaria with other infections in laboratory animals certainly supports the existence of a synergistic effect on mortality under some circumstances, but the scale of that effect has not been assessed from African data (for a review, see Feachem and Jamison, 1991).

(e) The role of immunizations: the 'replacement' mortality hypothesis debate

Many studies have shown that immunization against the six major diseases of the Expanded Programme on Immunizations (EPI) is of paramount importance to infant and child health and survival, and that measles vaccine saves the lives of

children both in developed and developing countries (Clemens et al., 1988; Center for Diseases Control, 1982; Feachem and Jamison, 1991; Aaby and Clements, 1989; Koenig et al., 1990, 1991; Feachem and Koblinsky, 1983; Boerma et al., 1991; Koster et al., 1981). However, the influence of measles vaccine in reducing mortality remains controversial. An argument has been made that measles vaccination did not result in improved overall survival in Zaire (Kasongo Project Team, 1981). The effectiveness of measles vaccine in preventing overall mortality has also been questioned with arguments that a variety of other factors contribute to infant and childhood mortality. Thus, previous investigations of the effect of measles vaccine on prevention of mortality have resulted in conflicting data, and some investigators have suggested that 'replacement' mortality occurs (i.e., if measles is prevented, high-risk children might die of other causes) (Hendrickse, 1975; Kasongo Project Team, 1981). Other data have suggested that measles is not only a major cause of immediate mortality, but also that the residual effects of measles contribute to malnutrition and increased mortality from other diseases for many subsequent months (Morley, 1973; Kimati et al., 1981; Hull et al., 1983; Koster et al., 1981; Aaby et al., 1993). In this sense, a reduction in measles morbidity will result in reduction in other morbidities (which occur as complications of measles or not), and their mortalities, leading to a reduction in overall mortality.

On the other hand, it has been suggested that measles vaccine failures appear most often due to primary, rather than secondary, vaccine failure resulting in waning immunity (Brunell, 1990). Since vaccine is immunogenic in most recipients it appears likely that host factors (possibly interferon induced by concomitant viral

illnesses) and not vaccine, may be responsible for some of these primary vaccine failures. In these conditions, a low immunity level conferred by measles vaccine may be due to other factors (that need to be controlled), not to measles inefficacy. Hence, a control for such factors should boost the magnitude of the protective effects of measles vaccine in those situations. Unfortunately, the Zaire study of the Kasongo Project Team (1981) and similar negative studies have evaluated the effects of measles vaccine on future mortality due to other diseases, without accounting for the potential inhibiting influences of preceding illnesses of children which might have affected the immunologic power of the vaccine over time. Other reasons for low seroconversion rates have been suggested and include interference by antibodies in breast milk (Domok et al., 1973) or by other enteroviruses in the gut (Jacob and Christopher, 1975). Many other conditions, such as high prevalence of malaria and other parasitic diseases might alter the immune response (Bottiger et al., 1981). Two studies of live attenuated measles vaccine found an unaltered humoral response with malaria infection (Kimati et al., 1981; Gilles et al., 1983), while Smedman et al. (1986) found augmented antibody response to live attenuated measles vaccine in children with *P. falciparum parasitaemia.* Thus, the role of malaria and other morbid conditions in the efficacy of measles vaccine needs further investigation. We will control for a number of these potential inhibiting factors in order to evaluate the effects of immunizations on malaria-associated mortality versus other mortalities.

3. Data and methodological considerations

(a) Data

Information on factors associated with childhood infectious diseases in sub-Saharan Africa is relatively scarce. Studies carried out so far in the region with some detailed information about these conditions are of variable lengths of follow-up, frequency of visits, the type of data (current illness or recall), the outcome measures (episodes per child, average daily prevalence, percentage of illness episodes, episodes per child per year), and the age range covered, and are generally of small sample size (for a review, see Feachem and Jamison, 1991). Since mortality is rarer than illness, estimating mortality requires larger sample sizes than estimating morbidity, a requirement which faces serious problems with most of the available data. Mortality due to malaria has been generally measured in three ways: a) from clinical records of the cause of death; b) by observing the rise in mortality during malaria epidemics; and c) by determining the fall in mortality when malaria is brought under control.

The Yaoundé Infant and Child Mortality Survey (YICMS) of 9774 children, with information on maternal and child episodes of illnesses collected at 1, 4, 8, 12, 16, 20 and 24 months after birth combines most of the advantages of previous methodologies and offers a unique opportunity to tackle the epidemiology and control of childhood malaria. The data have been assessed and found to be of good quality (Kuate Defo, 1992a). The YICMS appears to be the most comprehensive survey where a given health condition was just one aspect of a more general health concern with the causes of morbidity and mortality on a large-scale longitudinal

population-based study. Set up to obtain accurate data on the major causes of childhood morbidity and mortality as well as maternal morbidity, the survey's aim was to improve maternal and child health in Cameroon. Classification of causes of morbidity and mortality was based on lay reporting, a procedure generally followed in large-scale longitudinal population-based studies of the IFORD-type (Kirkwood, 1991a, 1991b; Cantrelle et al., 1986; Aaby et al., 1993; Halabi et al., 1992). Classification of causes of morbidity and mortality was based on symptoms and causes according to the child's mother or surrogate and followed the 8th revision of the International Classification of Diseases (ICD). The basic question asked at time t was: a) for survivors regarding their illnesses: 'has this child (name of the child) been sick since the last interview?' If yes, the question was asked about the nature of the first two most important (severe) conditions experienced by the child; and b) for deceased children regarding their causes of death: 'what was the cause of death of this child (name of the child).' The major advantage of this strategy (as opposed to the WHO/CDD or DHS strategy) is that it allows the person to be interviewed to be natural and not implicitly forced to search through his/her mind for something that may not be the natural declaration under normal interviewing conditions. In this fashion, the true morbidity is likely to be observed and reported as sensed by the parents, while in the WHO/CDD or DHS case, there is a high probability of misclassification and overestimation of prevalence towards specified diseases as confirmed by analyses of the DHS surveys (for a review, Boerma et al., 1991).

The discussion of clinical aspects of malaria is beyond the scope of this study.

There is sufficient evidence to argue that the quality of data on causes of death is quite accurate. First, a cross-classification of causes of death according to the source of information (hospital diagnosis vs. mother's report) has shown that in the YICMS, there was consistency in most cases regarding the reporting of causes of infectious diseases (measles, diarrhea, acute respiratory infections, and malaria) (Kuate Defo, 1988). Second, while in cause-specific morbidity and mortality analyses we expect some random misclassification of both cases and non-cases, this could only reduce the associations observed towards the null value and we would probably have observed larger measures of association had we used more accurate diagnostic procedures. Thus, the effects of any misclassification will be to produce conservative estimates, not an association between an exposure variable and the outcome if one does not exist (Chavance et al., 1992).

(b) Methodological considerations

Despite the heavy toll of malaria on lives of infants and children in developing countries, there is little agreement regarding the risk factors of malaria-associated morbidity and mortality. Also, the effects of putative risk factors of malaria morbidity and mortality are difficult to gauge because of methodological flaws in previous studies. Furthermore, the mechanism behind the suggested associations between exposure and outcome is not well established. The purpose of this study is to search for direct and indirect influences of strategic variables on the outcomes of interest. It is likely that failure to control for mutually confounding effects can lead to erroneous inferences about both the statistical significance and effect magnitude. Thus, poorly controlled studies will generally overestimate the magnitude

of effects, and the overall variance in effect magnitudes will be larger than that due to sampling variation alone under the null hypothesis that they are equivalent. An independent causal effect will be considered present if the effect magnitude on any of the outcome variables is greater than zero and sampling variation could be excluded.

A thorough test of the causal question requires studies that allow strong inference about the essential attributes of a cause, namely: association, time order, and direction. These attributes of causes form a hierarchy: once associations are established, the tests of the causal hypothesis need to be severe enough to eliminate doubt, first about the time sequence of the study variables and then about the direction between putative cause and effect. Direction implies consequential change: change in the outcome must be shown to be consequent to change in the supposed causal factor. The construction of variables used in this study is designed to reflect the time sequence of exposure variables vis-à-vis the outcomes of interest.

We use a continuous-time hazards modelling within a framework of competing risks analysis. This approach is designed to disentangle simultaneously the determinants of mortality ascribed to malaria versus other mortalities, while adjusting for other factors that may be correlated with infant and child survival. The continuous-time approach allows us to evaluate each exposure variable consistently with its exposure length. For instance, children who survived longer were more likely to have received immunizations than children who died early, independently of the effects of other variables.

The rationale for a competing risks analysis is that a certain number of children

will die anyway, possibly for other ultimate causes (e.g., malaria, diarrhea, nutritional diseases, acute respiratory infections, meningitis) and the immediate cause of death may be malaria, if present, or some other cause, if malaria is absent. Both malaria and, say, acute respiratory infections (ARI) may be sufficient causes, and only one (malaria) is necessary. In fact, analysis of cause-specific mortality data is complicated by the possibility of interaction between one cause and other causes of death (Bailey, 1975; Becker, 1989). Because of these interactions, it is virtually impossible to use conventional hazards models to estimate the true impact of, say, malaria on mortality rates (Gill, 1992). Eliminating malaria may prevent some deaths due to other causes (e.g., ARI); however, the malnourished children who are most apt to die from measles are also at increased risk of death from other causes. Cause-specific analysis of mortality differentials is critical for targeted health policies. Ignoring causes of death in the study of mortality is somewhat akin to ignoring the key variable in mortality analyses. Since underlying cause is defined in such a way that several causes are mutually exclusive and exhaustive, any variation in mortality from all causes combined is necessarily attributable to variation in mortality for one or more causes of death.

Since a number of conditions that may lead to death due to malaria cannot possibly be captured by measured variables selected for this study (see Table 1), we will examine the extent to which unmeasured heterogeneity can influence our conclusions. The work done to date on human populations leaves little doubt that population heterogeneity can have a significant impact on measures of mortality and mortality differentials (Rogers, 1992; Trussell and Rodriguez, 1990; Vaupel et

al., 1979). Because both the direction and the magnitude of the resulting biases remain ambiguous, more empirically grounded research is needed before effective tools to deal with unobserved heterogeneity can be developed. This work is designed to contribute to this effort.

4. Statistical methods: duration models and unobservables

(a) Multistate hazards models with duration dependencies

An individual's health history is assumed to evolve from birth to death. Once a child is born, he/she is at risk of dying of any cause of death. Let $\tau = 0$ be the calendar time initiating the birth-to-death process. We define a finite-state continuous time process $Y(\tau), \tau \succ 0$, $Y(\tau) \in E$, where the set of possible attained states is finite ($E = 0, 1, 2, ..., C, C \prec \infty$). An element of E defines the number of states. $Y(\tau)$ is state attained at τ. Transitions occur on or after $\tau = 0$. Sample-attrition through dropouts is assumed to have no influence on this process so we abstract from it in our presentation (Kuate Defo, 1992a; Kuate Defo and Palloni, 1992).

The key for multistate duration models is the conditional hazard. Define $H(\tau)$ as the relevant conditioning set at time τ. The relevant past of each individual including the history of the process up to time τ may be part of $H(\tau)$.

We assume that all durations $T_1, T_2, ..., T_C$ conditional on the appropriate history H have absolutely continuous distributions. Thus each random variable can be described by a conditional density which integrates to a conditional distribution function. If an individual becomes at risk for the jth state at time $\tau(j-1)$, the

conditional hazard at duration t_j is defined to be

$$h_j(t_j \mid H(\tau(j-1)+t_j) \tag{0.1}$$

As a consequence of our assumption that T_j given H is a random variable with an absolutely continuous distribution function, we may integrate (0.1) to form the survivor function

$$S(t_j \mid H(\tau(j-1)+t_j)) = exp[-\int_0^{t_j} h_j(u \mid H(\tau(j-1)+u))du] \tag{0.2}$$

Assuming the validity of (0.2), the birth-to-death process evolves in the following way. An individual at risk of one of the competing events at $\tau = 0$ remains so for a random length of time governed by the survivor function

$$Pr(T_1 \succ t_1 \mid H(\tau(0)+t_1)) = exp[-\int_0^{t_1} h_1(u \mid H(\tau(0)+u))du] \tag{0.3}$$

At calendar time $T(1) = \tau(1)$, the individual experiencing that event moves to the state $Y(\tau) = 1$. In the general case, $Y(\tau) = k-1$ for $\tau(k-1) \preceq \tau \prec \tau(k)$ and $T_k = T(k) - T(k-1)$ is governed by the conditional survivor function

$$Pr(T_k \succ t_k \mid H(\tau(k-1)+t_k)) = exp[-\int_0^{t_k} h_k(u \mid H(\tau(k-1)+u))du] \tag{0.4}$$

The conditional density function of duration $T_k = t_k$ is

$$g(t_k \mid H(\tau(k-1)+t_k)) = h_k[t_k \mid H(\tau(k-1)+t_k)][S(t_k \mid H(\tau(k-1)+t_k)] \quad (0.5)$$

If the H include all relevant conditioning information, the conditional density of $(T_1, T_2, ..., T_C)$ given $H(\tau(0) + \sum_{i=1}^{C} t_i)$ is

$$g(t_1, t_2, ..., t_C \mid H(\tau(0) + \sum_{i=1}^{C} t_i)) = \prod_{k=1}^{C} g(t_k \mid H(\tau(k-1)+t_k)) \qquad (0.6)$$

(b) The unobservables

In the continuous-time multistate hazards specifications described so far, we have assumed that all covariates are measured. This is unlikely to be the case if unobserved population heterogeneity is present. Athough it has long been recognized that different populations may face significant mortality differentials, only recently have researchers begun to examine the influences of unobserved population heterogeneity for the analysis and interpretation of demographic data, aimed at making explicit the possible biases occurring when unknown variables relevant to the process under study are omitted. There is a suggestion that heterogeneity in individual frailty can have substantial influence on observed age patterns of demographic processes through the process of selection (Vaupel et al., 1979; Rogers, 1992; Trussell and Rodriguez, 1990; Trussell and Richards, 1985; Alter and Riley, 1989; Berhman et al., 1990). In this study, we explore the possibility that unmeasured heterogeneity might bias our estimates. For example, heterogeneity in risks of death may imply a selection process in which those births with the lowest risks have a survival advantage (good health). In the YICMS data for example, one

could claim that when studying the birth-to-death process, the cohort of survivors to any given survival time do not represent a random sample of the cohort of survivors at an earlier survival time, and that instead an underlying selection results in a biased sample with overrepresentation of low risk individuals. Clearly, not all the children to be analyzed had the same level of frailty at birth and/or over time, either because of their natural resistance or their acquired immunity (e.g., through immunizations). Such bias may occur whenever the variables under study are correlated with the probability of surviving to a given survival time.

The few papers that fit models with unobservables generally assume that they can be summarized by a scalar random variable θ which is time-invariant with distribution $F(\vartheta)$; θ is assumed independent of H(0), the initial state of the process (for a review, see Heckman and Singer, 1985). The conventional model with unobservables augments (0.6) so that densities are defined conditional on $H(\tau)$ and ϑ:

$$g(t_k \mid H(\tau(k-1)+t_k),\vartheta) = h_k(t_k \mid H(\tau(k-1)+t_k),\vartheta)S(t_k \mid H(\tau(k-1)+t_k),\vartheta) \tag{0.7}$$

The conditional density of $T_1, T_2, ..., T_C$ given $H(\tau(0)+\sum_{i=1}^{C} t_i)$ is then

$$g(t_1,t_2,....,t_C \mid H(\tau(0)+\sum_{i=1}^{C} t_i)) = \int_{\underline{\theta}} \prod_{k=1}^{C} g(t_k \mid H(\tau(k-1)+t_k),\vartheta)dF(\vartheta) \tag{0.8}$$

where $\underline{\theta}$ is the support of θ.

Unobservables unknown to the individual being studied may be relevant compo-

nents of model specification. In a study of health, it is implausible that individuals know their own θ.

Throughout this study, we approximate the jth conditional hazard using the following functional form:

$$h_j(t_j \mid H(\tau(j-1)+t_j), \vartheta) = exp[\gamma_{0j} + \sum_{k=1}^{K_j} \gamma_{kj}(\frac{t_j^{\lambda_{kj}} - 1}{\lambda_{kj}}) + Z\beta_j + f_{j\vartheta}] \qquad (0.9)$$

where Z includes all observed regressors. The hazard specification (0.9) encompasses a variety of widely used models. Setting $\beta = 0$, $k_j = 1$, and $f_j = 0$, (0.9) specializes to a Weibull model if $\lambda_{1j} = 0$ and to a Gompertz hazard if $\lambda_{1j} = 1$. Specification (0.9) extends previous duration models by allowing for general time-varying covariates and by introducing unobserved heterogeneity component θ that is correlated across spells (Heckman and Walker, 1990). Empirically, we estimate distribution $F(\vartheta)$ by the nonparametric maximum likelihood (NPMLE) procedure described in Heckman and Singer (1984). This procedure approximates any distribution function of unobservables with a finite mixture distribution.

The model selection criterion is concerned with survival time. In most cases, a Gompertz or a Weibull is selected for most demographic processes. We will use the Weibull distribution which has been found to approximate very well the observed mortality in the YICMS (Kuate Defo, 1992b). Indeed, the Weibull function is widely used in reliability theory and, with appropriate choice of parameters, it can describe any demographic phenomenon that declines with age (negative slope) or increases with age (positive slope). The difference between the Gompertz and the

Weibull models is seen most clearly when one takes the logarithms of their corresponding hazard functions. When these logarithms are compared, the logarithm of the Weibull hazard function is linear in the logarithm of survival time, whereas the logarithm of the Gompertz hazard function is linear in survival time. Thus, for appropriately chosen parameters, Weibull and Gompertz hazard functions differ only in the pace of change, the Weibull function decreasing more rapidly during younger ages and more slowly during older ages than the Gompertz function.

5. Results

(a) Age-specific morbidity and mortality ascribed to malaria

Table 2 presents the age-specific prevalence, case fatality rate (CFR) and mortality of malaria. The condition is most prevalent after the first 4 months of life, but most fatal in the neonatal period (CFR = 19%). Its prevalence rises gradually throughout the first year of life, from 2.2% in the first month to 20.4% at 8-11 months. The data suggest that malaria is most severe in its acute phase across all age segments. Moreover, it is most severe in the first year of life: the CFR is 19%, 9%, 10%, and 8% at 0, 1-3, 4-7, and 8-11 months, respectively; in constrast, the CFR is only 2%, 4% and 1% at 12-15, 16-19 and 20-23 months, respectively. While the focus of vertical strategies to reduce infant mortality has been generally restricted to well-known childhood diseases like measles and diarrhea, the data here suggest that the contribution of malaria to infant mortality might be much more important than usually thought. This could be explained by the fact that the condition is so common in Africa that it is conventional wisdom to assume

that all groups are equally at risk. The level of malaria-associated mortality 4.9, 1.5 and 6.4 per 1,000 live births for the first year, second year and first two years of life, respectively.

(b) Influences of household factors

The Weibull estimates of transitions from birth to malaria mortality and other mortalities are shown in Table 3. Modern wall is inversely related to malarial mortality, while overcrowding is a very strong risk factor of malaria-associated deaths (model 1). The protective effects of modern wall are totally explained by the ethnic identity of the mother (model 3). It is possible that the type of materials used for the wall, even within an urban community, still reflect some underlying designs which are ethnically driven; this is likely to be the case particularly in popular neighborhoods in Yaoundé which are generally built along ethnic lines. The direct influences and statistical significance of overcrowding on malarial mortality are unaffected by controls for measured covariates (models 1 through 9) ($p \prec .01$), and a control for unmeasured heterogeneity marginally alters its statistical significance ($p \prec .05$). Regarding mortality due to other causes: modern roof and availability of electricity in the household are protective to child survival, while overcrowding has robust and direct effects ($p \prec .01$) on mortality. Modern roof has direct protective effects on mortality, while the effects of power source (electricity) operate to some extent through the mother's ethnic identity (model 3) and are totally captured by health conditions of the child (model 8).

(c) Influences of maternal reproductive patterns

It has been suggested that in areas where malaria is endemic-like, the prevalence

of parasitaemia is high among primiparae, and because malaria of the placenta is high among these groups, it is expected that their children are at significantly higher risks of malaria morbidity and mortality than children of multiparae. These conjectures are examined in Table 4. Our results support the view that first-born children are at a disadvantage vis-à-vis malarial mortality. The strongest evidence to reinforce this argument is found when control is introduced for child health (model 4) and child immunization status (model 5). In model 4, while parity shows no influences on mortality due to other causes, it appears clearly that first-born children experienced significantly higher malaria-associated mortality compared to their counterparts of higher birth order. This cannot be explained by the conventional wisdom that first-born children generally have higher mortality than other children, since such a claim is not substantiated for mortality due to causes other than malaria in this model. Furthermore, there is an increased advantage with increased parity as regards malarial mortality in all models controlling for measured covariates. Model 6 indicates that these differences are explained by unobservables, probably owing to the frailty of children who contact malaria of the placenta from their mothers and carry that underlying disadvantage over time.

(d) Influences of prenatal care visits

Table 5 presents the estimates of influences of lack of prenatal care on the propensity of children to move from birth to death due to malaria and other conditions. The results strongly indicate that lack of prenatal care visits puts children at greatest risks not only of mortality due to other causes, but also to malarial mortality. These findings are unaffected by controls for measured covariates (models 2

through 8), or for unmeasured heterogeneity (model 9). It appears that prenatal care visits should be stressed for all women, in view of its strong and direct impact on child survival. Such visits are likely to detect high risk pregnancies, to treat malaria during pregnancy with appropriate therapy, and to control the condition so as to minimize or eliminate the risk of malaria of the placenta.

(e) Influences of breastfeeding regimens

Nutritional status has been suggested to differentiate children in terms of their malarial mortality risks. We examine such a possibility using feeding methods. The results are shown in Table 6. Full and partial breastfeeding are protective to child survival, particularly as regards other mortalities. The inverse relations between full and partial breastfeeding and malaria are mediated to some extent by mother's ethnic identity (model 3), suggesting that differences in feeding practices among ethnic groups explain some of the effects of breastfeeding on malarial mortality. The statistical power of the inverse relationship between full breastfeeding and malaria-associated mortality is eliminated after a control for maternal reproductive patterns and survival status of preceding child (model 6), shifted upwards once child health status is controlled (model 8) ($p \prec .01$), reduced ($p \prec .05$) once immunization status of the child is controlled (model 9), and eliminated after a control for unmeasured heterogeneity (model 10). The inverse relation between partial breastfeeding and malarial mortality is eliminated only after a control for unmeasured heterogeneity. Overall, full and partial breastfeeding have basically indirect effects on mortality due to malaria, but direct ones on mortality attributable to other causes.

(f) Influences of antecedent morbidity

Table 7 presents the estimated effects of antecedent illnesses on transitions from birth to malaria-associated death and death ascribed to other causes. It appears that recurrent attacks of malaria are significant risk factors of malarial mortality, even after other preceding childhood diseases (model 1), socio-economic background of the mother (model 2), neighborhood of residence (model 3), and maternal reproductive patterns and survival status of the preceding child (model 4) are controlled ($p \prec .01$). But a control for prenatal care visits (model 5) eliminates the statistical significance of preceding malaria; this finding reinforces the significance of prenatal care visits as a health care strategy to reduce malarial mortality, as noted above. Antecedent respiratory infections (RI) are directly related to malarial mortality ($p \prec .01$), but show no effects on other mortalities (model 1). The statistical significance of the RI-malaria-associated mortality is somewhat reduced ($p \prec .05$), while preceding diarrhea gains statistical power ($p \prec .05$) once prenatal care visits are controlled (model 5). It appears clearly that prenatal care visits covary with intercurrent infections which have influences on malarial mortality. As regards malarial mortality, the disadvantage of children with previous infections (malaria, RI, diarrhea) is eliminated after a control for unobservables (model 7), but poor health status at birth and preceding diarrhea continue to put children at significant risk of mortality due to other causes, even after measured and unmeasured covariates are accounted for.

(g) Influences of full immunization

Table 8 presents the estimated effects on transitions from birth to malarial

mortality and from birth to other mortalities of full immunization. There is ample evidence that full immunization is directly protective against mortality due to causes other than malaria, even after unobserved heterogeneity is controlled ($p \prec .01$). Full immunization also shows protective effects against malarial mortality in the primary association model ($p \prec .05$). The statistical power of full immunization is reduced to some extent ($p \prec .10$) once the mother's ethnicity is controlled (model 3); this finding suggests that behavioural factors have some mediating impact of immunization effects on child survival. The residual protective effects of full immunization against malarial mortality completely vanish once dwelling characteristics are controlled. This result suggests that even though immunizations are protective to child survival, it is likely that improvements of housing conditions bear more significance in reducing malaria mortality than full immunization of the child.

(h) The influences of specific vaccines: the 'replacement' mortality hypothesis revisited

The following age schedule of immunization is used in Cameroon: DPT and poliomyelitis No. 1 plus BCG at 6-8 weeks, DPT and poliomyelitis No. 2 one month later, DPT and poliomyelitis No. 3 one month later, and measles at 9 months. Table 9 examines the possibility that specific vaccines can improve mortality due to causes other than the underlying cause for which a particular vaccine is meant. As regards malarial mortality, the data show that the second and third dose of DPT-polio and measles vaccine confer significant protection against malaria-associated mortality (model 1); moreover, the magnitude and statistical power of the protec-

tive effects of measles vaccine are much more important than that of DPT-polio. The protective effects of DPT-polio against malaria mortality are eliminated once a control for maternal background characteristics is introduced (model 2), but those of measles vaccine remain, although somewhat reduced. Thus, the protective effects of measles vaccine against malaria mortality cannot be explained by the socio-economic characteristics of the mother or her ethnic affiliation (model 2), by her neighborhood of residence in the city (model 3), or by household factors (model 4). Although once prenatal care is controlled measles vaccine loses its explanatory power, the magnitude of its protective effects remains the highest compared to that of other vaccines (model 5). These results are not an artifact of unmeasured covariates (model 6). Therefore, it appears that measles vaccine could prevent not only deaths due to measles, but also deaths due to other causes in general and to malaria in particular. These results are very important because they settle the 'replacement' mortality debate by showing that measles vaccine not only prevents measles deaths, but also reduces total mortality by reducing mortality associated with other childhood diseases, such as malaria in this case.

(i) Impact of unobservables and risk assessment of modifiable factors

Table 10 presents the Weibull estimates of selected modifiable factors from the full model (model with all measured variables) but without accounting for unobservables (model 1) and from the full model correcting for unmeasured heterogeneity (model 2), as well as the public health indicators derived from the estimated effects of these covariates. A comparison of these two models reveals that unmeasured heterogeneity has virtually no critical impact on the estimated effects of the

study key variables. Improvements in housing conditions (no overcrowding) could prevent as much as 83% of malarial deaths and 78% of deaths due to other causes which are attributable to overcrowding.

6. Discussion

The purpose of this study was to investigate the determinants of early child malarial morbidity and mortality and the extent to which determinants of malaria-associated mortality are also determinants of mortality due to causes other than malaria, in an effort to identify areas where a single health intervention might be beneficial vertically (i.e., control of the underlying health problem) and horizontally (i.e., control at the same time of other health conditions not directly targeted by the program).

The widespread availability of some antimalarials, chiefly chloroquine, has undoubtedly affected the childhood mortality from malaria. However, the spread of drug-resistant strains of *P. falciparum* is greatly altering this optimistic view, particularly in countries like Cameroon, Ghana, and other West African states (Bradley, 1991). Two sets of findings emerge from this study. First, among children under two years of age, the prevalence and severity of the disease among infants is greater than among toddlers. Second, malaria-associated mortality covaries with: a) household conditions; b) parity of the mother; c) prenatal care visits; d) infant feeding practices; e) antecedent infections; and f) immunization status of the child in general and measles vaccine in particular.

Other potential risk factors of malarial morbidity and mortality such as quarter

of residence and season of the year were considered; but these factors had no explanatory power on the prevalence and severity of malaria (results not shown). These findings may reflect the endemic character of malaria in Yaoundé and throughout the year.

Overcrowding and lack of prenatal care have direct positive relationships with malarial mortality, as well as with mortality due to other causes. Primiparae and antecedent episodes of respiratory infections, malaria and diarrhea, have indirect positive relationships with malaria-associated deaths. Modern wall, full and partial breastfeeding, full immunization status, DPT-polio 2 and 3, and measles vaccine have inverse indirect relationships with deaths attributable to malaria.

Of interest is the change in the attack rate of malaria with age of the children found in this study. It is believed that during early infancy, children are protected by maternal immunoglobulins in breast milk (Welsh and May, 1979; McKigney, 1971). They gradually become susceptible and more exposed to infection as early as the first couple of months after birth (Jelliffe and Jelliffe, 1978; Woodruff et al., 1983; Hanson and Winberg, 1972). Our findings that malaria is more prevalent at 4-7 months and 8-11 months than in the first 4 months or in the next 12 months are both consistent with the immunity conferred by breastmilk in the first months of life, increased susceptibility in the following months, and partial immunity thereafter.

It appears that the lack of prenatal care visits, besides its deleterious effects on child survival (and as regards malaria-associated mortality in particular) also covaries with a number of other risk factors of malarial mortality such as mater-

nal background (e.g., ethnicity), antecedent infections, and immunizations. Thus, prenatal care checkups should be stressed for all women, in view of the strong and direct impact on child survival. Such checkups are likely to detect early pregnancy at risk, to treat malaria during pregnancy with appropriate therapy, and to control the condition so as to minimize or eliminate the risk of placental malaria and its consequences on the fetus such as short gestational duration, weight loss, or both. Clearly, prenatal care attendance reduces malarial morbidity and mortality in infancy, perhaps through health education and malaria prophylaxis.

Blacker (1991) speculates that 'crowding within households seems unlikely to be a major factor in malaria transmission.' Our analysis, on the contrary, strongly indicates that crowding has an independent effect on malaria mortality. The number of persons sharing the room with the index child is not only an indicator of large space per person and less crowding, but also reflects well being and prestige, social status and deprivation of families. Our finding of strong and direct positive relationship of malarial mortality with a child from a home with more than three persons sharing the room with him indicates an effect of the socioeconomic well-being and better living conditions in the family and/or household. These results support the hypothesis that the risk of malaria is related to living conditions. The risk of malaria depends on the presence of an infected human reservoir, breeding of the vector and a susceptible recipient of the parasite. The first two of these factors are more likely where there are low-standard living conditions, crowded homes and poverty, which are conducive to high malarial morbidity and mortality because they facilitate the spread of infections and vector, lead to the pollution

of the environment and of water supplies, and result in a lower standard of child care.

This study has assessed the potential role of vaccines in preventing infant and child morbidity and mortality. Of all vaccines of target EPI diseases, measles vaccine has the greatest explanatory power on survival chances associated with morbidity and mortality under two years of age. The data strongly indicate that measles vaccine is the optimal prophylaxis for reducing simultaneously measles mortality and mortality due to other causes. Read from the perspective of the 'replacement' mortality hypothesis, these results contradict the view that those children who are prevented from dying by measles vaccine are the ones who remain at greatest risk of dying of other childhood diseases and that the benefits conferred by measles immunization are thus eventually decreased or lost entirely. Along with the findings for full immunization status, it appears clearly that children fully immunized or children who have received their measles vaccine have better survival chances than their unimmunized counterparts. These findings provide solid grounds to support immunization programs in infancy and childhood.

Table 1: Definition of Selected Variables, YICMS, 1978-1980

Variables♣	Definition
MEDU	1 if mother educated
EMPM	1 if mother employed before and since marriage
MSIT	1 if mother not married
SINCO	1 if household income between 20,000 and 50,000 CFA francs
TINCO	1 if household income greater than 50,000 CFA francs
CESETH	1 if ethnic groups of the regions of Centre and South
LITSWETH	1 if ethnic groups of the regions of Littoral and South-West
ESETH	1 if ethnic groups of the region of East
NORETH	1 if ethnic groups of the region of North
MOKOLO	1 if quarter of residence is Mokolo
BRIQUETERIE	1 if quarter of residence is Briqueterie
NKOLDONGO	1 if quarter of residence is Nkoldongo
DRY	1 if dry season of birth
WALL	1 if wall is modern
ROOF	1 if roof is modern
FLOOR	1 if floor is modern
ELEC	1 if elec is modern
PIPEDWAT	1 if piped water
WELLWAT	1 if is well water
CROWDING	1 if more than 3 persons share the bedroom with the index child
FAGE	1 if maternal age at child's birth < 20 years
LAGE	1 if maternal age at child's birth > 34 years
SECO	1 if birth order 2-3
THIR	1 if birth order 4+
PRES	1 if preceding child deceased
CARE	1 if prenatal care
FBF	1 if the index child was fully breastfed in the age interval immediately preceding the age interval of estimation
PBF	1 if the index child was partially breastfed in the age interval immediately preceding the age interval of estimation

Table 1 (con):

FC	1 if the following conception was reported to have occurred in the age interval immediately preceding the age interval of estimation
HEBI	1 if the index child had poor health status at birth
RESP	1 if the index child had suffered from acute respiratory infections in the age interval immediately preceding the age interval of estimation
MALA	1 if the index child had malaria in the age interval immediately preceding the age interval of estimation
DIA	1 if the index child had diarrhea in the age interval immediately preceding the age interval of estimation
ROUVAX	1 if the index child had measles vaccine prior to the age interval of estimation
BCG	1 if the index child had BCG vaccine prior to the age interval of estimation
DPT1	1 if the index child had DPT no. 1 prior to the age interval of estimation
DPT2	1 if the index child had DPT no. 2 prior to the age interval of estimation
DPT3	1 if the index child had DPT no. 3 prior to the age interval of estimation
IMAL	1 if the index child was fully immunized prior to the age interval of estimation

♣ All variables were coded as dummies.

Table 2: Age-specific Malarial Morbidity and Mortality, YICMS 1978-1980

Age at Infection (in completed months)		Age at Death (in completed months)						
		0	1-3	4-7	8-11	12-15	16-19	20-23
0								
	Cases	215	211	195	185	170	162	156
	CFR	19	--	5	--	--	--	--
1-3								
	Cases	--	885	877	816	773	736	725
	CFR	--	9	1	1	--	1	--
4-7								
	Cases	--	--	1445	1431	1348	1294	1250
	CFR	--	--	10	1	--	--	--
8-11								
	Cases	--	--	--	1458	1447	1380	1320
	CFR	--	--	--	8	1	1	--
12-15								
	Cases	--	--	--	--	1219	1216	1174
	CFR	--	--	--	--	2	2	--
16-19								
	Cases	--	--	--	--	--	1276	1271
	CFR	--	--	--	--	--	4	--
20-23								
	Cases	--	--	--	--	--	--	1362
	CFR	--	--	--	--	--	--	1
Q(x)		0.4	1.0	1.9	1.6	0.5	0.8	0.2

Note: The case fatality rate (CFR) is expressed in per 1,000 malaria cases.
The probablity of dying at age x (Q(x)) is expressed in per 1,000 live births.

Table 3: Weibull Estimates of Influences of Household Characteristics on Mortality due to Malaria and Other Causes, YICMS 1978-1980

Variables	Model 1 Transitions		Model 2 Transitions		Model 3 Transitions	
	1 ---> 2	1 ---> 3	1 ---> 2	1 ---> 3	1 ---> 2	1 ---> 3
WALL	-1.178¶	-.093	-1.250¶	-.024	-1.247	-.062
	(.720)	(.180)	(.798)	(.181)	(.910)	(.183)
ROOF	.143	-.652*	.178	-.607*	.166	-.589*
	(.803)	(.179)	(.869)	(.180)	(.945)	(.182)
FLOOR	-.186	-.008	-.197	.129	-.174	.126
	(.483)	(.126)	(.499)	(.127)	(.519)	(.127)
ELEC	.302	-.304§	.283	-.226§	.309	-.220¶
	(.443)	(.129)	(.458)	(.130)	(.507)	(.132)
PIPEDWAT	.595	.056	.561	.128	.565	.135
	(.781)	(.235)	(.888)	(.236)	(1.057)	(.237)
WELLWAT	.035	.176	.066	.151	.070	.160
	(.573)	(.165)	(.659)	(.166)	(.810)	(.168)
CROWDING	1.657*	1.415*	1.665*	1.462*	1.649*	1.465*
	(.361)	(.092)	(.374)	(.094)	(.383)	(.094)
L	3800		3721		3715	

Variables	Model 4 Transitions		Model 5 Transitions		Model 6 Transitions	
	1 ---> 2	1 ---> 3	1 ---> 2	1 ---> 3	1 ---> 2	1 ---> 3
WALL	-1.272	-.046	-1.279	-.041	-1.271	-.054
	(.955)	(.183)	(.993)	(.183)	(1.096)	(.185)
ROOF	.142	-.571§	.182	-.570*	.207	-.511§
	(1.022)	(.183)	(1.168)	(.185)	(1.249)	(.187)
FLOOR	-.172	.126	-.205	.123	-.207	.090
	(.557)	(.127)	(.609)	(.128)	(.662)	(.129)
ELEC	.309	-.220¶	.304	-.228¶	.336	-.214¶
	(.511)	(.132)	(.580)	(.133)	(.667)	(.134)
PIPEDWAT	.593	.111	.567	.107	.563	.092
	(1.084)	(.238)	(1.318)	(.240)	(1.356)	(.242)
WELLWAT	.094	.141	.092	.140	.085	.135
	(.818)	(.169)	(1.118)	(.171)	(1.191)	(.172)
CROWDING	1.654*	1.462*	1.757*	1.494*	1.760*	1.489*
	(.391)	(.094)	(.443)	(.095)	(.509)	(.096)
L	3713		3702		3656	

Table 4: Weibull Estimates of Influences of Reproductive Patterns on Mortality due to Malaria and Other Causes, YICMS 1978-1980

Variables	Model 1 Transitions		Model 2 Transitions		Model 3 Transitions	
	1 ---> 2	1 ---> 3	1 ---> 2	1 ---> 3	1 ---> 2	1 ---> 3
FAGE	-.050	.154	-.096	.140	-.058	.163
	(.848)	(.126)	(.858)	(.127)	(.853)	(.127)
LAGE	.661	.167	.629	.171	.674	.183
	(.668)	(.171)	(.748)	(.172)	(.758)	(.172)
SECO	-.635	-.207¶	-.675	-.208¶	-.620	-.191
	(.682)	(.130)	(.696)	(.131)	(.694)	(.131)
THIR	-1.211¶	-.233¶	-1.223¶	-.236¶	-1.150¶	-.236¶
	(.651)	(.145)	(.706)	(.147)	(.706)	(.147)
L	3702		3656		3642	

Variables	Model 4 Transitions		Model 5 Transitions		Model 6 Transitions	
	1 ---> 2	1 ---> 3	1 ---> 2	1 ---> 3	1 ---> 2	1 ---> 3
FAGE	.737	.098	.533	.054	-.117	.056
	(.739)	(.125)	(.716)	(.124)	(.940)	(.124)
LAGE	.954	.193	1.576$	.209	.689	.198
	(.762)	(.172)	(.706)	(.172)	(.885)	(.173)
SECO	-1.295$	-.149	-1.479$	-.199¶	-.678	-.194
	(.643)	(.128)	(.627)	(.128)	(.775)	(.129)
THIR	-1.924$	-.176	-2.293*	-.239¶	-1.265	-.232
	(.716)	(.146)	(.617)	(.146)	(.834)	(.148)
L	3565		3539		3495	

Note: Variables are abbreviated as shown in Table 1. Standard errors are in parentheses underneath the estimates.

♣ 1 ----> 2: Birth to Malaria Mortality;
1 ----> 3: Birth to Other Mortalities;

Model 1 =FAGE+LAGE+SECO+THIR+PRES+MEDU+EMPM+MSIT+SINCO+TINCO+CSETH+LITSWETH+ESETH+
+NORETH+MOKOLO+NKOLDONGO+WALL+ROOF+FLOOR+ELEC+PIPEDWAT+WELLWAT;
Model 2 = Model 1 +FBF+PBF+FC;
Model 3 = Model 2 +CARE;
Model 4 = Model 3 +HEBI+RESP+MALA+DIA;
Model 5 = Model 4 +IMAL (full model)
Model 6 = Full model correcting for unmeasured heterogeneity.
L = - Log-Likelihood.
* p <.01 ; $ p <.05 ; ¶ p <.10.

Table 5: Weibull Estimates of Influences of Prenatal Care on Mortality due to Malaria and Other Causes, YICMS 1978-1980

	Model 1		Model 2		Model 3	
Variables	Transitions		Transitions		Transitions	
	1 ---> 2	1 ---> 3	1 ---> 2	1 ---> 3	1 ---> 2	1 ---> 3
NOCARE	1.496*	1.032*	1.764*	.683*	1.772*	.682*
	(.478)	(.161)	(.558)	(.180)	(.560)	(.180)
L	3933		3849		3846	
	Model 4		Model 5		Model 6	
Variables	Transitions		Transitions		Transitions	
	1 ---> 2	1 ---> 3	1 ---> 2	1 ---> 3	1 ---> 2	1 ---> 3
NOCARE	1.988*	.809*	1.930*	.816*	1.923*	.806*
	(.596)	(.183)	(.618)	(.183)	(.637)	(.184)
L	3699		3688		3642	
	Model 7		Model 8		Model 9	
Variables	Transitions		Transitions		Transitions	
	1 ---> 2	1 ---> 3	1 ---> 2	1 ---> 3	1 ---> 2	1 ---> 3
NOCARE	2.075*	.816*	2.105*	.790*	1.950$	.787*
	(.640)	(.193)	(.612)	(.192)	(.860)	(.196)
L	3565		3539		3495	

Note: Variables are abbreviated as shown in Table 1. Standard errors are in parentheses underneath the estimates.
♣ 1 ----> 2: Birth to Malaria Mortality;
1 ----> 3: Birth to Other Mortalities;
Model 1 = NOCARE;
Model 2 = Model +MEDU+MSIT+EMPM+SINCO+TINCO+CSETH+LITSWETH+ESETH+NORETH;
Model 3 = Model +MOKOLO+NKOLDONGO;
Model 4 = Model +WALL+ROOF+FLOOR+ELEC+PIPEDWAT+WELLWAT+CROWDING;
Model 5 = Model +FAGE+LAGE+SECO+THIR+PRES;
Model 6 = Model +FBF+PBF+FC;
Model 7 = Model +HEBI+RESP+MALA+DIA;
Model 8 = Model +IMAL (full model);
Model 9 = Full model correcting for unmeasured heterogeneity.
L = - Log-Likelihood.
* p <.01 ; $ p <.05 ; ¶ p <.10.

Table 6: Weibull Estimates of Influences of Breastfeeding Regimens on Mortality due to Malaria and Other Causes, YICMS 1978-1980

	Model 1		Model 2		Model 3	
Variables	Transitions		Transitions		Transitions	
	1 ---> 2	1 ---> 3	1 ---> 2	1 ---> 3	1 ---> 2	1 ---> 3
FBF	-.904$	-1.303*	-.923$	-1.256*	-.930¶	-1.246*
	(.396)	(.119)	(.439)	(.120)	(.525)	(.121)
PBF	-1.199*	-1.102*	-1.237*	-.985*	-1.269$	-.994*
	(.438)	(.125)	(.462)	(.125)	(.523)	(.125)
L	3903		3821		3814	
	Model 4		Model 5		Model 6	
Variables	Transitions		Transitions		Transitions	
	1 ---> 2	1 ---> 3	1 ---> 2	1 ---> 3	1 ---> 2	1 ---> 3
FBF	-.931¶	-1.245*	-.989¶	-1.240*	-.981	-1.238*
	(.532)	(.121)	(.622)	(.125)	(.660)	(.125)
PBF	-1.271$	-.992*	-1.336$	-.988*	-1.332¶	-.989*
	(.545)	(.126)	(.690)	(.129)	(.761)	(.129)
L	3812		3667		3657	

Table 6 (con):

Variables	Model 7		Model 8		Model 9	
	Transitions		Transitions		Transitions	
	1 ---> 2	1 ---> 3	1 ---> 2	1 ---> 3	1 ---> 2	1 ---> 3
FBF	-1.020	-1.238*	-2.224*	-1.121*	-2.073§	-1.075*
	(.682)	(.125)	(.702)	(.128)	(.706)	(.128)
PBF	-1.352¶	-.996*	-2.735*	-.931*	-2.427*	-.882*
	(.774)	(.130)	(.776)	(.133)	(.788)	(.133)
L	3642		3565		3539	

Variables	Model 10	
	Transitions	
	1 ---> 2	1 ---> 3
FBF	-1.151	-1.075*
	(.806)	(.129)
PBF	-1.468	-.880*
	(1.200)	(.134)
L	3495	

Note: Variables are abbreviated as shown in Table 1. Standard errors are in parentheses underneath the estimates.

♣ 1 ----> 2: Birth to Malaria Mortality;
1 ----> 3: Birth to Other Mortalities;

Model 1 = FBF+PBF;
Model 2 = Model 1 +MEDU+MSIT+EMPM+SINCO+TINCO;
Model 3 = Model 2 +CSETH+LITSWETH+ESETH+NORETH;
Model 4 = Model 3 +MOKOLO+NKOLDONGO;
Model 5 = Model 4 +WALL+ROOF+FLOOR+ELEC+PIPEDWAT+WELLWAT+CROWDING;
Model 6 = Model 5 +FAGE+LAGE+SECO+THIR+PRES;
Model 7 = Model 6 +FC+CARE;
Model 8 = Model 7 +HEBI+RESP+MALA+DIA;
Model 9 = Model 8 +IMAL (full model)
Model 10 = Full model correcting for unmeasured heterogeneity.
L = - Log-Likelihood.
* p <.01 ; § p <.05 ; ¶ p <.10.

Table 7: Weibull Estimates of Influences of Antecedent Morbidity on Mortality due to Malaria and Other Causes, YICMS 1978-1980

Variables	Model 1 Transitions♣ 1 ---> 2	Model 1 Transitions 1 ---> 3	Model 2 Transitions 1 ---> 2	Model 2 Transitions 1 ---> 3	Model 3 Transitions 1 ---> 2	Model 3 Transitions 1 ---> 3
HEBI	1.286	2.618*	2.250$	2.616*	1.942¶	2.635*
	(1.151)	(.119)	(1.068)	(.121)	(1.098)	(.121)
RESP	1.769*	-.157	2.476*	-.161	2.459*	-.167
	(.224)	(.112)	(.193)	(.112)	(.195)	(.112)
MALA	2.435*	-.410$	3.647*	-.405$	3.589*	-.419$
	(.394)	(.174)	(.200)	(.176)	(.191)	(.176)
DIA	.478	.138	-.126	.149	-.218	.154
	(.564)	(.140)	(.483)	(.141)	(.488)	(.141)
L	3687		3853		3846	

Variables	Model 4 Transitions 1 ---> 2	Model 4 Transitions 1 ---> 3	Model 5 Transitions 1 ---> 2	Model 5 Transitions 1 ---> 3	Model 6 Transitions 1 ---> 2	Model 6 Transitions 1 ---> 3
HEBI	1.777	2.573*	.298	2.489*	.408	2.507*
	(1.170)	(.123)	(1.326)	(.127)	(1.358)	(.125)
RESP	1.800*	-.151	1.087$	-.057	1.175$	-.043
	(.293)	(.112)	(.493)	(.114)	(.520)	(.113)
MALA	2.775*	-.387$	.667	-.322¶	.607	-.252
	(.286)	(.176)	(.703)	(.179)	(.696)	(.179)
DIA	.028	.145	1.707$	.245¶	1.758$	.306$
	(.531)	(.142)	(.591)	(.145)	(.655)	(.145)
L	3700		3565		3539	

Variables	Model 7 Transitions 1 ---> 2	Model 7 Transitions 1 ---> 3
HEBI	.728	2.506*
	(1.654)	(.165)
RESP	.437	-.045
	(.680)	(.113)
MALA	.738	-.252
	(.854)	(.180)
DIA	.339	.302$
	(1.300)	(.145)
L	3495	

Note: Variables are abbreviated as shown in Table 1. Standard errors are in parentheses underneath the estimates.
♣ 1 ----> 2: Birth to Malaria Mortality;
1 ----> 3: Birth to Other Mortalities;
Model 1 = HEBI+RESP+MALA+DIA+MEDU+MSIT+EMPM+SINCO+TINCO+WALL+ROOF+FLOOR+ ELEC+PIPEDWAT+WELLWAT;
Model 2 = Model 1 +CSETH+LITSWETH+ESETH+NORETH;
Model 3 = Model 2 +MOKOLO+NKOLDONGO;
Model 4 = Model 3 +FAGE+LAGE+SECO+TINCO+PRES;
Model 5 = Model 4 +CARE+FBF+PBF+FC;
Model 6 = Model 5 +IMAL (full model);
Model 7 = Full model correcting for unmeasured heterogeneity.
L = - Log-Likelihood.
* p <.01 ; $ p <.05 ; ¶ p <.10.

Table 8: Weibull Estimates of Influences of Full Immunization on Mortality due to Malaria and Other Causes, YICMS 1978-1980

Variables	Model 1		Model 2		Model 3	
	Transitions		Transitions		Transitions	
	1 ---> 2	1 ---> 3	1 ---> 2	1 ---> 3	1 ---> 2	1 ---> 3
IMAL	-.943§	-1.268*	-.968§	-1.123*	-.893¶	-1.095*
	(.446)	(.166)	(.456)	(.168)	(.475)	(.168)
L	3907		3832		3828	
	Model 4		Model 5		Model 6	
Variables	Transitions		Transitions		Transitions	
	1 ---> 2	1 ---> 3	1 ---> 2	1 ---> 3	1 ---> 2	1 ---> 3
IMAL	-.897¶	-1.091*	-.923	-1.089*	-.948	-1.090*
	(.486)	(.168)	(.674)	(.169)	(.745)	(.170)
L	3826		3682		3657	
	Model 7		Model 8		Model 9	
Variables	Transitions		Transitions		Transitions	
	1 ---> 2	1 ---> 3	1 ---> 2	1 ---> 3	1 ---> 2	1 ---> 3
IMAL	-.888	-1.043*	-1.168¶	-1.075*	-.919	-1.072*
	(.929)	(.171)	(.755)	(.170)	(1.100)	(.171)
L	3614		3539		3495	

Note: Variables are abbreviated as shown in Table 1. Standard errors are in parentheses underneath the estimates.
♣ 1 ----> 2: Birth to Malaria Mortality;
1 ----> 3: Birth to Other Mortalities;
Model 1 = IMAL;
Model 2 = Model 1 +MEDU+MSIT+EMPM+SINCO+TINCO;
Model 3 = Model 2 +CSETH+LITSWETH+ESETH+NORETH;
Model 4 = Model 3 +MOKOLO+NKOLDONGO;
Model 5 = Model 4 +WALL+ROOF+FLOOR+ELEC+PIPEDWAT+WELLWAT+CROWDING;
Model 6 = Model 5 +FAGE+LAGE+SECO+THIR+PRES+CARE;
Model 7 = Model 6 +FBF+PBF+FC;
Model 8 = Model 7 +HEBI+RESP+MALA+DIA (full model);
Model 9 = Full model correcting for unmeasured heterogeneity.
L = - Log-Likelihood.
* p <.01 ; § p <.05 ; ¶ p <.10.

Table 9: Weibull Estimates of Influences of specific Immunizations on Mortality due to Malaria and Other Causes, YICMS 1978-1980

Variables	Model 1		Model 2		Model 3	
	Transitions		Transitions		Transitions	
	1 ---> 2	1 ---> 3	1 ---> 2	1 ---> 3	1 ---> 2	1 ---> 3
BCG	-.450	-.580	-.247	-.281	-.258	-.286
	(2.028)	(.302)	(2.350)	(.300)	(2.238)	(.300)
DPT1	.170	.087	.099	-.091	.087	-.087
	(2.027)	(.297)	(2.352)	(.296)	(2.241)	(.296)
DPT2	-2.278*	-.355	-.096	-.325	-.028	-.322
	(.658)	(.230)	(.741)	(.231)	(.779)	(.231)
DPT3	-2.118§	-.582¶	-.589	-.500¶	-.576	-.496¶
	(.913)	(.318)	(.966)	(.319)	(.966)	(.319)
ROUVAX	-3.144*	-.289	-2.076¶	-.184	-2.019¶	-.184
	(1.040)	(.207)	(1.131)	(.209)	(1.132)	(.208)
L	3990		3831		3828	

Variables	Model 4		Model 5		Model 6	
	Transitions		Transitions		Transitions	
	1 ---> 2	1 ---> 3	1 ---> 2	1 ---> 3	1 ---> 2	1 ---> 3
BCG	-.326	-.266	-.247	-.274	-.190	-.252
	(2.226)	(.299)	(1.794)	(.300)	(1.630)	(.299)
DPT1	.138	-.114	.032	-.099	-.002	-.081
	(2.228)	(.297)	(1.795)	(.297)	(1.636)	(.296)
DPT2	-.057	-.344	-.080	-.334	-.093	-.344
	(.849)	(.233)	(.964)	(.234)	(1.046)	(.234)
DPT3	-.563	-.487	-.585	-.498	-.534	-.477
	(1.016)	(.321)	(1.144)	(.321)	(1.587)	(.321)
ROUVAX	-1.916¶	-.176	-1.913	-.175	-1.855	-.166
	(1.248)	(.209)	(1.531)	(.209)	(1.553)	(.210)
L	3684		3659		3616	

Note: Variables are abbreviated as shown in Table 1. Standard errors are in parentheses underneath the estimates.

♣ 1 ----> 2: Birth to Malaria Mortality;
1 ----> 3: Birth to Other Mortalities;

Model 1 = BCG+DPT1+DPT2+DPT3+ROUVAX;
Model 2 = Model 1 +MEDU+MSIT+EMPM+SINCO+TINCO+CSETH+LITSWETH+ESETH+NORETH;
Model 3 = Model 2 +MOKOLO+NKOLDONGO;
Model 4 = Model 3 +WALL+ROOF+FLOOR+ELEC+PIPEDWAT+WELLWAT+CROWDING;
Model 5 = Model 4 +FAGE+LAGE+SECO+THIR+PRES+CARE;
Model 6 = Full model.
L = - Log-Likelihood.
* p <.01 ; § p <.05 ; ¶ p <.10.

Table 10: Sensitivity of Estimates to Unobserved Heterogeneity and Risk Assessment of Modifiable Risk Factors of Malaria-associated Mortality and other Mortalities, YICMS 1978-1980

	Model 1		Model 2		Risk Assessment	
Variables	Transitions		Transitions		Transitions	
	1 ---> 2	1 ---> 3	1 ---> 2	1 ---> 3	1 ---> 2	1 ---> 3
WALL	-2.232§	-.087	-1.291	-.079		
	(1.008)	(.177)	(1.279)	(.178)		
ROOF	-.197	-.446	.209	-.446		
	(1.089)	(.183)	(1.447)	(.185)		
FLOOR	-.273	.036	-.251	.035		
	(.639)	(.125)	(.721)	(.129)		
ELEC	.802	-.075	.444	-.075		
	(.728)	(.130)	(1.039)	(.131)		
PIPEDWAT	1.490	.213	.627	.203		
	(1.322)	(.233)	(1.827)	(.234)		
WELLWAT	.116	.211	.148	.210		
	(1.198)	(.160)	(1.551)	(.163)		
CROWDING	2.282*	1.508*	1.797§	1.507*	83♬	78♬
	(.562)	(.097)	(.726)	(.099)		
FAGE	.533	.054	-.117	.056		
	(.716)	(.124)	(.940)	(.124)		
LAGE	1.576§	.209	.689	.198		
	(.706)	(.172)	(.885)	(.173)		
SECO	-1.479§	-.199	-.678	-.194		
	(.627)	(.128)	(.775)	(.129)		
THIR	-2.293*	-.239¶	-1.265¶	-.232		
	(.617)	(.146)	(.834)	(.148)		
NOCARE	2.105*	.790*	1.950§	.787*	86♬	54♬
	(.612)	(.192)	(.860)	(.196)		
FBF	-2.073§	-1.075*	-1.151	-1.075*	--	66♪
	(.706)	(.128)	(.806)	(.129)		
PBF	-2.427*	-.882*	-1.468	-.880*	--	59♪
	(.788)	(.133)	(1.200)	(.134)		
FC	.081	.325§	.169	.323*	--	28♬
	(.615)	(.164)	(.793)	(.164)		
HEBI	.408	2.507*	.728	2.506*	--	92♬
	(1.358)	(.125)	(1.654)	(.165)		
RESP	1.175§	-.043	.437	-.045		
	(.520)	(.113)	(.680)	(.113)		
MALA	.607	-.252	.738	-.252		
	(.696)	(.179)	(.854)	(.180)		
DIA	1.758§	.306§	.339	.302§	--	26♬
	(.655)	(.145)	(1.300)	(.145)		
IMAL	-1.168¶	-1.075*	-.919	-1.072*	--	66♪
	(.755)	(.170)	(1.101)	(.171)		
L	3539		3495			

Note: Variables are abbreviated as shown in Table 1. Standard errors are in parentheses underneath the estimates.
♣ 1 ----> 2: Birth to Malaria Mortality;
1 ----> 3: Birth to Other Mortalities;
Model 1 = Full Model;
Model 2 = Full model correcting for unmeasured heterogeneity.
♪ Preventable Fraction (PF) for negative risk factors from Model 2.
♬ Attributable Fraction (AF) for positive risk factors from Model 2.
L = - Log-Likelihood.
* p <.01 ; § p <.05 ; ¶ p <.10.

References

Aaby, P., Andersen, M. and Knudsen, K. 1993. "Excess mortality after early exposure to measles." *International Journal of Epidemiology* 22(1): 156-62.

Aaby, P. and Clements, J. 1989. "Measles immunization research: a review." *Bull. World Health Organ.* 67: 443-48.

Alter, G. and Riley, J.C. 1989. "Frailty, sickness, and death: models of morbidity and mortality in historical populations", *Population Studies* 43: 25-45.

Anderson, R.M., and May, R.M. 1982. " Directly transmitted infectious diseases: control by vaccination." *Science* 215: 1053-60.

Bagster, W.D. 1949. "Malaria incidence in Central and South Africa.", In: *Malariology*, Boyd, M.E. (ed.), vol. 2. Philadelphia and London: W.B. Saunders Company, pp. 800-809.

Bailey, Norman T.J. 1975 (Second edition). *The mathematical theory of infectious diseases and its applications.* New York: Hafner Press.

Balint, O. and Anand, K. 1979. "Infectious and parasitic diseases in Zambian children." *Tropical Doctor* 9: 99-103.

Banguero, H. 1984. "Socio-economic factors associated with malaria in Colombia." *Social Sciences and Medicine* 19: 1099.

Becker, N.G. 1989. *Analysis of infectious disease data.* New York: Chapman and Hall.

Behrman, J., Sickles, R. and Taubman, P. 1990. "Age specific death rates with covariates: Sensitivity to sample length and unobserved frailty." *Demography* 27(2): 267-84.

Blacker, J. 1991. "Infant and child mortality: Development, environment, and custom". In: Feachem, R.G. and Jamison, D.T. (eds.), *Disease and Mortality in Sub-Saharan Africa.* Oxford: Oxford University Press, pp. 75-86.

Boerma, J.T., Sommerfelt, A.E. and Rutstein, S.O. 1991. "Childhood morbidity and treatment patterns." *DHS Comparative Studies* 4. Institure for Resource Development, Columbia, Maryland.

Brabin, B.J. 1983. "An analysis of malaria in pregnancy in Africa." *Bulletin of the World Health Organization* 61: 1005-1016.

Bradley, D.J. 1991. "Malaria", In: Feachem, R.G. and Jamison, D.T. (eds.), *Disease and Mortality in Sub-Saharan Africa.* Oxford: Oxford University Press. pp. 190-202.

Bray, R.S. and Anderson, M.J. 1979. "Falciparum malaria and pregnancy." *Transactions of the Royal Society of Tropical Medicine and Hygiene* 73: 427-431.

Cantrelle, P., I.L. Diop, M. Garenne, M. Gueye and S. Sadio. 1986. "The profile of mortality and its determinants in Senegal, 1960-1980", In: *Determinants of mortality change and differentials in developing countries.* New York: United Nations, Population Studies, no. 94, pp. 1-4.

Carswell, F. Hughes, A., Palmer, R. et al. 1981. "Nutritional status, globulin titers and parasitic infections of two populations of Tanzanian school children." *American Journal of Clinical Nutrition* 34: 1291.

Centers for Disease Control. 1982. "Recommendations of the Immunization Practices Advisory Committee: measles prevention." *Morbidity and Mortality Weekly Report* 31: 217-231.

Chavance, M. Dellatolas, G. and Lellouch, J. 1992. "Correlated nondifferential misclassifications of disease and exposure: application to a cross-sectional study of the relation between handedness and immune disorders." *Int. J. Epidemiol.* 21: 537-546.

Clemens, J.D., Stanton, B.F., Chakraborty, J., et al. 1988. "Measles vaccination and childhood mortality in rural Bangladesh." *Am J Epidemiol* 128: 1330-1339.

Edington, G.M. 1967. "Pathology of malaria in West Africa." *British Medical Journal* i: 715.

Eelens, F. 1983. *Impact of breast-feeding on infant and child mortality with varying incidence of malaria - Evidence from the Kenya Fertility Survey 1977-78.* Interuniversity Programme in Demography Working paper 1983-3.

El Samani, F.Z., Willett, W.C. and Ware, J.H. 1987. "Nutritional and sociodemographic risk indicators of malaria in children under five: a cross-sectional study in a Sudanese rural community." *Journal of Tropical Medicine and Hygiene* 90: 69-78.

Feachem, R. 1981. "The water and sanitation decade." *Journal of Tropical Medicine and Hygiene* 84(2).

Feachem, R. and Jamison, D. (eds.) 1991. *Disease and Mortality in Sub-Saharan Africa.* Oxford: Oxford University Press.

Feachem, R. and Koblinsky, M. 1983. "Interventions for the control of diarrhoeal diseases among young children: measles immunization." *Bulletin of the WHO,* 61:641-652.

Fleming, A. and Werblińska, B. 1982. "Anaemia in childhood in the guinea savanna of Nigeria." *Annals of Tropical Paediatrics* 2: 161-173.

Fleming, A., Storey, J., Molineaux, L., Iroko, E. and Attai, E. 1979. "Abnormal haemoglobins in the sudan savanna of Nigeria. I. Prevalence of haemoglobins and relationships between sickle cell trait malaria and survival." *Ann. Trop. Med. Parasitol.* 73: 161-72.

Gibson, R.S. 1990. *Principles of nutritional assessment.* New York: Oxford University Press.

Gill, R.D. 1992. "Multistate life-tables and regression models." *Mathematical Population Studies* 3(4): 259-276.

Gilles, H.M., Greenwood, B.M., Greenwood, A.M., et al. 1983. "The Malumfashi Project." *Trans R Soc Trop Med Hyg* 77: 24-31.

Gilles, H.M., Lowson, J., Sibelas, M. et al. 1969. "Malaria, anaemia and pregnancy." *Annals of Tropical Medicine and Parasitology* 63: 245.

Goldman, A.S. and Smith, C.W. 1973. "Host-resistance factors in human milk." *Journal of Pediatrics* 82: 1082-1090.

Greenwood, B.M. and Whittle, H.C. 1981a. "Nutrition, infection and immunity." In: *Immunology of Medicine in the Tropics,* London: Edward Arnold.

Greenwood, B.M. and Whittle, H.C. 1981b. "The immune response to infection." In: *Immunology of Medicine in the Tropics,* London: Edward Arnold.

Greenwood, B.M., Bradley-Moore, A.C., Palit, A. and Bryceson, A. 1972. "Immunosuppression in children with malaria." *Lancet* 1: 169-72.

Halabi, S., Zurayk, H., Awaida, R., Darwish and Saab, B. 1992. "Reliability and validity of self and proxy reporting of morbidity data: a case study from Beirut, Lebanon." *International Journal of Epidemiology* 21: 607-612.

Hanson, L.A. and Winberg, J. 1972. "Breast milk and defence against infection in the newborn." *Archives of Diseases in Childhood* 47: 845-848.

Heckman, J. and Singer, B. 1984. "A method for minimizing the impact of distributional assumptions in econometric models for duration data." *Econometrica* 52: 271-320.

Hendrickse, R.G. 1975. "Problems of future measles vaccination in developing countries." *Trans R Soc Trop Med Hyg.* 69: 31-34.

Hinkle, L.R., Jr and Wolff, H. G. 1957. "The nature of man's adaptation to his total environment and the relation of this to illness." *Arch. Intern. Med.* 99: 442-460.

Hull, H.F., Williams, R.J. and Oldfield, F. 1983. "Measles mortality and vaccine efficacy in West Africa." *Lancet* 1: 972-975.

Jelliffe, D.B. and E.F.P. Jelliffe. 1978. *Human Milk in the Modern World.* Oxford: Oxford University Press.

Jelliffe, D.B. 1968. *Infant Nutrition in the Subtropics and Tropics.* Geneva: WHO.

Jensen, J.B., Hoffman, S., Boland, M. et al. 1984. "Comparison of immunity to malaria in Sudan and Indonesia: Crisis form versus merozoite-invasion inhibition." *Proceedings of the National Academy of Science* 81: 922.

Johansson, S.R. and Mosk, C. 1987. "Exposure, resistance and life expectancy: Disease and death during the economic development of Japan, 1900-1960." *Population Studies* 41: 207-235.

Kasongo Project Team. 1981. "Influence of measles vaccination on survival pattern of 7-35 month old children in Kasongo, Zaire." *Lancet* 1: 764-767.

Kimati, V., Loretu, K., Munube, G. and Kimboi, F. 1981. "The problem of measles virus response with reference to vaccine viability, age, protein energy malnutrition and malaria in the tropics." *J Trop Pediatr* 27: 205-9.

Kirkwood, B.R. 1991a. "Diarrhea." In: Feachem, R.G. and Jamison, D.T. (eds.), *Disease and Mortality in Sub-Saharan Africa.* Oxford: Oxford University Press, pp. 134-157.

Kirkwood, B.R. 1991b. "Acute Respiratory Infections." In: Feachem, R.G. and Jamison, D.T. (eds.), *Disease and Mortality in Sub-Saharan Africa.* Oxford: Oxford University Press, pp. 158-172.

Koenig, M., Fauveau, V. and Wojtyniak, B. 1991. "Mortality reductions from health interventions: the case of immunization in Bangladesh", *Population and Development Review* 17, 1:87-104.

Koenig, M., Khan, M., Wojtyniak, B., Clemens, J., et al. 1990. "The Impact of measles vaccination on childhood mortality in Matlab, Bangladesh." Population Council, Programs Division Working Paper no.3.

Koster, F.T., Curlin, G.C., Aziz, K.M.A., Hague, A. 1981. "Synergistic impact of measles and diarrhoea on nutritional and mortality in Bangladesh." *Bull WHO* 59: 901-908.

Kuate Defo, B. 1992a. "Mortality and attrition processes in longitudinal studies in Africa: An appraisal of the IFORD Surveys." *Population Studies* 46: 327-348.

Kuate Defo, B. 1992b. "Interplay of the determinants of low birthweight and child morbidity and mortality."Center for Demography and Ecology, University of Wisconsin, Working Series Paper No. 92-13.

Kuate Defo, B. and Palloni, A. 1992. "Determinants of infant and early childhood mortality in Cameroon." Center for Demography and Ecology, University of Wisconsin, Working Series Paper No. 92-23.

Kuate Defo, B. 1988.. *Mortalité Infanto-Junénile à Yaoundé: Essai d'Approche Causale.* Les Enquêtes sur la Mortalité Infantile et Juvénile, Vol. 3, Tome 2. Yaoundé: IFORD.

MacDonald, G. 1957. *The Epidemiology and Control of Malaria.* Oxford: Oxford University Press.

MacGregor, J. and Avery, J. 1974. "Malaria transmission and fetal growth." *British Medical Journal* 3: 433-436.

MacLeod, C.L. (ed.). (1988). *Parasitic infections in pregnancy and the newborn.* Oxford: Oxford University Press.

Majumdar, A.S. and Ghose, A.C. 1982. "Protective properties of anticholera antibodies in human colostrum." *Infection and Immunity* 36: 962-965.

Mata, L.J. 1978. *The children of Santa Maria Cauque: a prospective field study of health and growth.* Cambridge: MIT Press.

McKigney, J. 1971. "Uniqueness of human milk: economic aspects." *American Journal of Clinical Nutrition* 24: 1005-1012.

Mims, 1991. "The origin of major human infections and the crucial role of person-to-person spread." *Epidemiol Infect* 106: 423-433.

Molineaux, L. 1985. "La lutte contre les maladies parasitaires: le probleme du paludisme, notamment en Afrique." In: *La Lutte Contre la Mort.* Vallin, J., and Lopez, A. (eds.). Travaux et Documents no. 108. Paris: Presses Universiataires de France, pp. 111-140.

Monjour, L., Bourdouillon, F., Schlumberger, M., et al. 1982. "Étude de l'immunité humorale et cellulaire après vaccination antitétanique chez l'enfant africain malnutri et paludéen. 1. Étude de la réponse en anticorps antitétanique." *Bull WHO* 60: 589-96.

Mosley, W.H. 1985. "Biological and socio-economic determinants of child survival. A proximate determinants framework integrating fertility and mortality variables." *International Population Conference.* IUSSP, Florence. pp. 189-208.

Mosley, W.H. 1982. "Biological contamination of the environment by man. In: *Biological and Social aspects of mortality and the length of life,* Preston, S.H. (ed.), Liege: Ordina Editions, pp. 39-67.

Mosley, W. Henry. & L. C. Chen. 1984. "An Analytical Frame work for the Study of Child Survival in Developing Countries." *Population and Development Review,* Supplement to vol. 10, 1984, pp. 25-45.

Morley, D. 1973. *Pediatrics Priorities in the Developing World.* Massachusetts: Butterworths.

Najera, J.A. 1989. "Le paludisme et l'action de l'OMS." *Bulletin de l'Organisation Mondiale de la Santé* 67(4): 347-363.

Najera, J.A. 1979. "A suggested approach to malaria control and to the methodology applicable in different epidemiological situations based on experience in the Americas." *Bulletin of the Panamerican Health Organization* 13: 223-234.

Ndkuyeze, A., Munzo, A., Stewart, J., et al. 1988. "Immunogenicity and safety of measles vaccine in ill African children." *Int. J. Epidemiol.* 17: 448-455.

Ofosu-Amaah, S. 1991. "Disease in Sub-Saharan Africa: An overview", in Feachem and Jamison (eds.), pp. 119-121.

Ogra, S.S. and Ogra, P.L. 1978. "Immunologic aspects of human colostrum and milk." *J Pediatr* 92: 550-555.

Pelassy, P. 1976. "Caractéristiques de la pollution atmosphérique particulaire à Yaoundé (Cameroun) pendant la grande saison sèche 1974/1975: ses conséquences sanitaires." *Bull Organ Mond Santé* 54: 507-512.

Rogers, A. 1992. "Heterogeneity and selection in multistate population analysis." *Demography* 29(1): 31-38.

Rowland, M., Barrell, R., and Whitehead, R. 1978. "The weanling's dilemma: Bacterial contamination in traditional Gambian weaning foods." *Lancet* 1: 136-8.

Russell, P.F., West, L.S., Maxwell, R.D., and MacDonald, G. 1963. *Practical Malariology.* 2d ed. London: Oxford University Press.

Smedman, L., Silva, M., Gunnlaugsson, G. et al. 1986. "Augmented antibody response to live attenuated measles vaccine in children with Plasmodium falciparum parasitaemia." *Annals of Tropical Paediatrics* 6: 14-153.

Trussell, J. and Rodriguez, G. 1990. "Heterogeneity in demographic research." In: *Convergent Issues in Genetics and Demography,* edited by Adams, J., Lam, D.A., Hermalin, A.I. and Smouse, P.E. New York: Oxford University Press, pp. 111-132.

Trussell, J. and Richards, T. 1985. "Correcting for unobserved heterogeneity in hazard models: An application of the Heckman-Singer procedure to demographic data." In: *Sociological Methodology,* edited by Tuma, N. Jossey-Bass: San Francisco, pp. 242-276.

van Norren, B. and van Vianen, H.A.W. 1986. *The malnutrition-infections syndrome and its demographic outcome in developing countries. A new model and its application.* PCDO/Programming Committee for Demographic Research, Publication no. 4. The Hague: Department of Demography.

Vaupel, J., Manton, K.G. and Stallard, E. 1979. "The impact of heterogeneity in individual frailty on the dynamics of mortality." *Demography* 16(3): 439-454.

Welsh, J.K. and May, J.T. 1979. "Anti-infective properties of breast milk." *Journal of Pediatrics* 94: 1-9.

Wenlock, R.W. 1981. "Endemic malaria, malnutrition and child health." *Food policy* 6: 105.

Wenlock, R.W. 1979. "The epidemiology of tropical parasites in rural Zambia and the consequences for public health." *Journal of Tropical Medicine and Hygiene* 82: 90.

Wilson, D.B., Garnham, P.C.C. and Swellengrebel, N.H. 1950. "A review of hyperendemic malaria." *Tropical Diseases Bulletin* 47: 677-698.

Winikoff, B. 1982. "Weaning: nutrition, morbidity and mortality consequences." In: *Biological and Social aspects of mortality and the length of life.* Preston, S.H. (ed.), Liege: Ordina Editions, pp. 113-150.

Woodruff, A.W., Adamson, E., El Suni A., et al. 1983. "Infants in Juba, Southern Sudan: The first six months of life." *Lancet* ii: 262.

Mailing address:
Center for Demography and Ecology
University of Wisconsin
1180 Observatory Drive #4412
Madison, WI 53706-1393
USA