Effects of Family Frailty on Child Mortality: Ivory Coast Experience

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Abstract
This article examines the impact of family-level clustering on under-five mortality risk by a Bayesian approach. A proportional hazard model with multiplicative random effect is applied to a sample of 6,804 children's survival times. This data set results from the 1998/99 Demographic and Health Survey conducted in Ivory Coast. When the frailty is Gamma distributed, a variance of 0.32 is obtained, which indicates that family membership significantly affects child mortality risk.

Introduction
Infant mortality rate has globally decreased in West Africa: from 156 per 1,000 in 1960 to 107 in 1998, a figure of nearly 31% (UNICEF 2001). Determinants of child mortality have been intensively studied (Pebley and Stupp 1987; Martin et al. 1983). Among others, the most discussed factors are the sex of the child, its birth order, the survival status of the previous child, the previous/subsequent birth interval, the mother’s schooling and marital status, the maternal age and the household characteristics. However, major studies have not considered the dependence between mortality risks for children belonging to the same family (Manda 1999; Kuate Debo 1992; Akoto and Tabutin 1990). Observations from members of the same family share certain characteristics which are not captured by the covariances included in the standard model (Guo and Rodriguez 1992). Ignoring such family-level correlation may lead to biased parameters estimates.

Various sources of unobserved family frailty (Vaupel et al. 1979) have been identified, which can be of genetic, socioeconomic or behavioural form. In effect, children from the same parents may inherit common genetic factors (Guo 1993) which may affect their natural defence system and increase their susceptibility to infection. Moreover, some women repeatedly experience complica-

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tions in pregnancy and delivery which affect the baby's health (Curtis et al. 1993). In addition, mothers usually adopt similar childcare behaviours for all their children and multiple deaths in a family may be due to insufficient parental education in childcare (Das Gupta 1990). For example, the mother does not have enough knowledge and resource in the use of health services and the detection of childhood diseases. In some cases, the mother even lacks competence in appropriate breastfeeding practice. Socioeconomic factors are related to the family environmental and cultural behaviour. In addition, unobserved heterogeneity may also operate at the community level (Sastry 1997). However, the use of "cluster" variable stratification in this data has not proven to really define any community classification (Kuate-Defo 2001). Hence, in the present study shared frailty is studied at the family level only.

Previous studies related to correlated sibling mortality mostly used the Expectation Maximization (EM) algorithm (Sastry 1997; Curtis et al. 1993; Guo and Rodriguez 1992). Although the EM is fast, its results heavily rely on the choice of starting values. Hence, the algorithm may sometimes converge toward a local maximum instead of the global one (Robert and Casella 2002). To circumvent this problem, a full Bayesian approach can be used. The goal of this work is to investigate the effect of family heterogeneity on child mortality risk from a Bayesian perspective using data from Ivory Coast.

The structure of the paper is as follows. Section 2 describes the data and the covariates of interest. The Bayesian model and the computation approach are presented in section 3. The results are discussed in section 4. Section 5 contains some concluding remarks. Further results of the Markov Chain Monte Carlo (MCMC) inference are shown in the appendix.

Data and Covariates

The data used in the present study result from the Demographic and Health Survey (DHS 2001) program conducted in Ivory Coast (between September 1998 and March 1999). A representative sample of 3,040 women, aged 15–49 years, was interviewed. The survey questionnaire included a complete birth history, as well as information on maternal education, household, and related subjects. Of this sample, a total of 6,804 single births from 1,935 women occurred. The study data is constituted by this reduced sample in which 1,022 children (15%) died by the time of the interview. Infant mortality in Ivory Coast was about 10.7% in 1998 (UNICEF 2001). Figure 1 shows child mortality trends in Ivory Coast from 1979–1998. A reversal in the downward mortality trend starts by end 1989. Mortality among children under five years increased by more than 7% in the 1990–1998 period.

The effects of the following covariates are studied: the mother’s age at the child’s birth, the sex of the child, the child’s birth order, the survival status of the previous child, the duration of breastfeeding and the length of the preceding and succeeding birth intervals. Maternal age (at birth of the child) is a commonly used covariate. Previous studies usually showed that children born from women at

Figure 1: Child Mortality Trends, Ivory Coast, 1979 – 1998.

Source: DHS Statscompiler www.measuredhs.com
(Note: Infant mortality refers to mortality for children aged less than 1 year.)

2 The cluster number is “the number identifying the sample point as used during the fieldwork. This variable may be a composite of several variables in the questionnaire.” (DHS Individual Recode Data File, www.measuredhs.com.)
youngest and oldest age are subject to highest risk of death (Sastry 1997; Pebley and Stupp 1987; Trussell and Hammerslough 1983). However, Martin et al. (1983) found that children born from the oldest women have a lower mortality risk in data from Indonesia and Pakistan. Lalou and Legrand (1997) also concluded to the same puzzling effect of mother’s age on child mortality in Bamako, Mali. The sex of the child is also a significant covariate: mortality used to be higher among boys than girls, at least during the first months of life (Trussell and Hammerslough 1983). Large birth order is generally risky for child survival, but first-born children also experience a high mortality rate (Hobcraft et al. 1985). Findings from several studies demonstrate that short preceding and succeeding birth intervals largely increase child mortality risk (Guo 1993; Miller et al. 1992; Pebley and Stupp 1987). However, Koenig et al. (1990) found a lower effect of short birth spacing. A variable related to whether the previous child died or did not prior to conception of the reference child was also used in the model. The duration of breastfeeding was not included in the study, since that variable contains a nonnegligible proportion of missing values (maybe due to lack of memory for earlier births).

Information (taken at the time of interview) on household income, father/mother’s occupation and education level has also been omitted because such information might have changed during the time preceding the interview. Nor was the source of drinking water included, since more than 10% of the women did not answer the question.

Table 1 gives the summary statistics of the variables used in the study. The sample of children contains almost as many girls as boys. On average, mothers gave birth at age 26 years and approximately 12% of the children experienced the death of a previous sibling. The table also shows that most interbirth periods are long (24 months and 12 months for preceding and succeeding interval).

Table 2 shows the distribution of children by family. The 1,935 mothers having births represent the family subdivision. The number of births per mother varies between 1 and 14, with a mean of 3.52. A nonnegligible level of clustering is expected, since more than 90% of the children belong to families that contribute two or more births (Sastry 1997). Nonparametric analysis of child survival times, obtained using the Kaplan-Meier survival curves (Kaplan and Meier 1958), are shown in Figure 2. Female children have lower risk of death, as well as children whose previous sibling was alive by index’s child birth. There is also greater survival chance for children with longer preceding or succeeding birth interval.
A parametric model is used to quantify the effect of each covariate on child mortality risk. The distribution of the model parameters are estimated through a Bayesian approach.

**The Bayesian Model**

Denote by \( t_{ij} \) the random survival time of the \( j^{th} \) child from family \( i \) and \( \Theta=(\mathbf{\beta}, w_i) \) the unknown parameters of the model corresponding to the data. The parameter \( w_i \) represents the family random effect and \( \mathbf{\beta}=(\beta_1, \beta_2, \ldots) \) is the vector of fixed effect coefficients. The family random effect is assumed to act on the conditional hazard \( h(t_{ij} | \mathbf{\beta}, w_i) \) in the following multiplicative way

\[
h(t_{ij} | \mathbf{\beta}, w_i) = w_i \lambda_0(t_{ij}) \exp(\beta_1 X_{1ij} + \beta_2 X_{2ij} + \ldots). \tag{1}
\]

Our aim is to find the distribution of the family effect \( w_i \). The Bayesian approach updates the prior belief (\( \pi(\mathbf{\beta}, w_i) \)), using the data in order to obtain the posterior distribution, which represents new beliefs after having observed the data (Gelman et al. 1995). The posterior distribution of \( \Theta \) conditioned on the data is proportional to the product of the likelihood function, and the prior distribution (\( \pi \) stands for the distribution of interest and \( l \) for the likelihood)

\[
\pi(\mathbf{\beta}, w_i | t_{ij}) \propto \pi(\mathbf{\beta}, w_i) \times l(t_{ij} | \mathbf{\beta}, w_i). \tag{2}
\]

**The Likelihood Function**

Previous studies have shown that the effect of the chosen covariates on child mortality does not have equal importance over the whole period of childhood (Sastry 1997; Guo and Rodriguez 1992). Hence the study time period is split into five intervals with cut points at 3, 6, 12 and 24 months (based on preliminary analysis not shown here). Within each interval \( I_n \), the baseline hazard is assumed constant: \( \lambda(t_{ij}) = \lambda_n \) for \( t_{ij} \in I_n \). Under that assumption, the likelihood function coincides with that of a Poisson distribution with mean \( \lambda_n E_{ij} \), where \( E_{ij} \) and \( \lambda_n \) denote, respectively, the time lived in the interval \( I_n \) and the hazard function for the \( j^{th} \) child from the \( i^{th} \) family (Laird and Olivier 1981).

### Table 2: Distribution of Deaths by Family Size, Ivory Coast (1998/99 DHS).

<table>
<thead>
<tr>
<th>Number of Births per Mother in 10 Year Period</th>
<th>Number of Children Who Died</th>
<th>Total Mothers</th>
<th>Children</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>1</td>
<td>464</td>
<td>61</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>259</td>
<td>75</td>
<td>10</td>
</tr>
<tr>
<td>3</td>
<td>172</td>
<td>78</td>
<td>20</td>
</tr>
<tr>
<td>4</td>
<td>116</td>
<td>64</td>
<td>25</td>
</tr>
<tr>
<td>5</td>
<td>96</td>
<td>53</td>
<td>19</td>
</tr>
<tr>
<td>6</td>
<td>60</td>
<td>49</td>
<td>19</td>
</tr>
<tr>
<td>7</td>
<td>38</td>
<td>41</td>
<td>18</td>
</tr>
<tr>
<td>8</td>
<td>22</td>
<td>25</td>
<td>9</td>
</tr>
<tr>
<td>9</td>
<td>17</td>
<td>10</td>
<td>16</td>
</tr>
<tr>
<td>10+</td>
<td>3</td>
<td>11</td>
<td>10</td>
</tr>
<tr>
<td>Total</td>
<td>1247</td>
<td>467</td>
<td>146</td>
</tr>
<tr>
<td>% of children</td>
<td>64.5</td>
<td>24.1</td>
<td>7.6</td>
</tr>
<tr>
<td>% of deaths</td>
<td>45.7</td>
<td>28.5</td>
<td>14.4</td>
</tr>
</tbody>
</table>

(Note: Analysis times are in years.)
The Prior Specification
To compute the prior distribution \( \pi(\alpha, w_i) \), a widely used conjugate prior is adopted for the family frailty (Guo 1993): \( w_i \sim \text{Gamma}(\tau, \tau) \), with \( \tau \sim \text{Gamma}(1,1) \) for simplicity. The matrix of fixed effects \( \alpha \) follows a multivariate normal distribution with zero mean and low precision \( \Sigma: \ \alpha \sim \text{Normal}(0, \Sigma) \).

The Posterior Distribution
The posterior distribution, resulting from \( \theta \), has a form which makes it complicated to sample directly from it. Hence, a Markov Chain Monte Carlo (MCMC) simulation is performed using the defined priors and likelihood functions. The Markov chain algorithm, after a suitable initial burn-in period, is expected to reach a stationary distribution which is the same as the desired posterior distribution (Gelman et al. 1995). Following previous studies on a hierarchical model (Robert and Casella 2002; Bolstad and Manda 2001), the distributions of the nodes, conditional on all the parameters, are assumed independent of each other.

The Computation
The algorithm suggested by Bolstad and Manda (2001) can be used to sample from the posterior density of the family frailty. In the present study, we used our own algorithm (available on request), written by using the Bayesian software WinBugs (Spiegelhalter et al. 2003).

After 2,000 simulations for burn-in, 5,000 iterations were performed on four chains with different starting values. The Gelman-Rubin factors (Gelman and Rubin 1992) were used as convergence criterion. This factor describes how much the increase in the number of iterations may improve the
estimates. Values under 1.2 correspond to approximate convergence of the Markov chain (Congdon 2003). Factors very close to 1 were obtained for the fixed effects. Figure 3 shows the Gelman-Rubin (GR) factor for the family frailty and its variance. The GR factors ranged between 0.7 and 1.3 during the first thousand iterations, and reached 1 after 2,500 iterations. These results do not contradict the convergence observed with the chains’ historical time series.

Results

Figure 4 shows the values of the baseline hazard, by time interval. Similarly to previous studies on child mortality (Bolstad and Manda 2001; Sastry 1997; Guo and Rodriguez 1992), the child mortality risk is higher (0.2) in the first two months of life, and then decreases over age. The relative risk of death for children three months is 1.22 times higher than the risk after 24 months. This may be due to inadequate delivery conditions and lack of prenatal vaccination in order to guarantee child immunity against childhood diseases, among other causes. Households (nutrition and hygiene) and maternal health status may also contribute to high mortality risk at lower age.

The first column of Table 3 shows the results of the piecewise exponential model, which ignores the family clustering. The table indicates that mortality risk is slightly higher among boys than girls, as it was found in some previous studies (Bolstad and Manda 2001; Trussell and Hammerslough 1983). The relative risk of death for girls is 0.76 times the mortality risk among boys. The results also show that children whose previous sibling died experienced a risk of death 2.02 times higher than the risk for those with the immediate sibling alive. For the length of the preceding birth interval, the reference group has a preceding birth interval greater than 24 months. A preceding birth interval less than 18 months increases by nearly two times the child mortality risk. First births are also 1.5 times riskier.

A child whose younger sibling is born in a less than 12-month period is five times more likely to die than a child with a succeeding birth interval greater than 12 months. In effect, that child may receive less attention and care due to the mother’s new pregnancy. Maternal age does not seem to be a determinant in the sample: relative risks of 0.97 and 1.00 are obtained for linear and squared effect respectively. A positive correlation ($\rho = 0.7$) is found between maternal age squared and death of previous child. Birth order is strongly negative correlated to maternal age squared ($\rho = -0.84$) as well as to linear maternal age ($\rho = -0.72$).

The last column of Table 3 depicts the results of the proportional hazard with multiplicative family random frailty. The posterior distribution of the family heterogeneity has a mean variance of 0.32 after controlling for the model covariates. This result suggests that child risk of death increases by 32% in a family where one child has died, compared to the mortality risk for a child belonging to a family where no sibling died (Guo 1993). This value lies in the range of family heterogeneity obtained in previous studies: Guo and Rodriguez (1992) found a variance of 0.22 for family random effect in Guatemala; Sastry (1997) obtained 0.516 for northeast Brazil; Bolstad and Manda (2001)...
reported a variance of 0.843 for Malawi. The mortality risk associated with child sex remains unchanged, as well as the risk related to maternal age, succeeding and preceding birth intervals. However, the relative effect of the birth order on mortality risk slightly increased (from 0.8 to 1) by including the family frailty term. Moreover, the posterior mean for the survival status of the previous child is lower in the model with frailty: a relative effect of 2.02 against 1.4 (when the model incorporates family frailty). A similar result was found by Sastry (1997) in Brazil and Guo (1993) in Guatemala.

**Concluding Remarks**
In this study, a Bayesian approach is used to explore the effects of family membership on child mortality risk. A proportional hazard model with multiplicative family frailty has been applied to data from the Ivory Coast 1998/99 Demographic and Health Survey. This study indicates that the death of the previous sibling increases two times the relative mortality risk of the following child. A possibility for this is that the premature death of a child exposes the nonbreastfeeding mother to the risk of new pregnancy, while her body is still physically (and mentally) weak. The interconception periods may then be shortened. As a consequence, children may have small weight at delivery due to improper fetal development (Scrimshaw 1996), and they may also experience high risk of transmission of infectious diseases (Ronsmans 1995).

This study also indicates that short interbirth periods, independent of previous sibling status, increase child risk of death. Indeed, reduced birth spacing not only affects the mother’s health condition during each pregnancy, but also leads to large family size, where competition between siblings for nutritional and affective resources may occur. Hence, adequate policies should focus on teaching women family planning, which is not widely applied even if its benefits are known. The results indicate that the child’s birth order affects his or her chance of survival. Sex difference in mortality risk was also found, with girls’ risk of death being 0.76 times lower than the mortality risk of boys. Similar results were found in, for example, Sastry (1997) and Guo (1993).

In the pathways of previous researches (Sastry 1997; Trussell and Hammerslough 1983), children born from teenaged or old mothers were expected to have a higher mortality risk. However, in this sample, maternal age did not produce a significant impact on child survival (the relative risk associated with maternal age is 0.97). One explanation may lie in the customs still prevailing in the

### Table 3: Comparison between Standard Proportional Hazard Model and Multiplicative Model with Family Gamma Frailty, Ivory Coast (1998/99 DHS).

<table>
<thead>
<tr>
<th>Covariates</th>
<th>Model Without Family Effects</th>
<th>Model With Family Random Effects</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Posterior Beta Mean</td>
<td>Posterior Beta Mean</td>
</tr>
<tr>
<td><strong>Sex of child</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Boy*</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Girl</td>
<td>0.76 (1.17)</td>
<td>0.75 (1.07)</td>
</tr>
<tr>
<td><strong>Previous child survival status</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Alive*</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Death</td>
<td>2.02 (1.42)</td>
<td>1.39 (1.12)</td>
</tr>
<tr>
<td><strong>Maternal age</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Linear effect</td>
<td>0.97 (1.04)</td>
<td>0.97 (1.01)</td>
</tr>
<tr>
<td>Squared effect</td>
<td>1.00 (1.01)</td>
<td>1.00 (1.01)</td>
</tr>
<tr>
<td>Birth order</td>
<td>0.82 (1.19)</td>
<td>1.03 (1.03)</td>
</tr>
<tr>
<td><strong>Succeeding birth interval</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Long*</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Short</td>
<td>5.18 (1.36)</td>
<td>4.58 (1.21)</td>
</tr>
<tr>
<td><strong>Preceding birth interval</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>First child</td>
<td>1.51 (1.27)</td>
<td>1.42 (1.11)</td>
</tr>
<tr>
<td>Short</td>
<td>1.99 (1.22)</td>
<td>1.78 (1.13)</td>
</tr>
<tr>
<td>Medium</td>
<td>1.39 (1.22)</td>
<td>1.30 (1.12)</td>
</tr>
<tr>
<td>Long*</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Variance of Family Frailty</td>
<td>0.324 (0.095)</td>
<td></td>
</tr>
</tbody>
</table>

* indicates the reference group. **Standard deviation in parenthesis
region, where childcare is not solely restrained to the nuclear family, but is an extended family issue. Hence, the lack of experience (or financial resources) due to a mother’s young age is overcome by other family members’ contributions. Important family random effect is found with a variance of 0.32, compatible with other findings (Sastry 1997; Guo and Rodriguez 1992).

The Bayesian approach used in the study produces results consistent with previous works. Furthermore, this simple method offers various extensions worth studying: spatial representation of the national region with high child mortality risk can be produced. Such information is needed for targeted policy programs. However, the data set must contain clear spatial localization of each respondent.

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References


Appendix A

Kernel density for (a) Family Frailty and its (b) Variance, Ivory Coast (1998/99 DHS).