# CHAPTER 7

# DETERMINANTS OF INFANT AND CHILD MORTALITY: AN ANALYSIS CONTROLLING FOR FAMILY AND COMMUNITY FRAILTY EFFECTS

## 7.1 Introduction

This chapter analyses data on maternal, socioeconomic and environmental contamination variables associated with infant and child mortality in Zimbabwe during 1996 - 2005, taking into account frailty effects at family and community levels. In particular the chapter examines the extent to which the survival outcomes of siblings are associated net of the observed factors.

These associations with infant and child survival outcomes even after accounting for different known determinants of mortality has been attributed to unobserved heterogeneity or frailty.[45,69] Frailty is the variance in mortality caused by unobserved factors.

Frailty models address the situation where the same individual may experience the hazard more than once, raising the possibility that due to some unmeasured and perhaps unknown cause (that is, a cause of "unobserved heterogeneity"), some subjects may be more likely than others to experience repeated hazards. This likelihood is the ‘frailty’ of these subjects and in standard Cox models are unmeasured effects.[70] Frailty models are supported by STATA but not by SPSS.

## 7.2 Measurement of the Family and Community Frailty Effect

We outline in this section the statistical estimation of the frailty models for infant and child mortality. The Cox regression frailty equation shown below builds on the standard hazard model equation presented in Chapter 3. We show in the equation below the addition of specific

random effects for family ($v_i$) and community ($w_i$) to the standard Cox regression equation to allow for frailty effects. The equation used to estimate the frailty effects is given by:

$$h_{ijk}(t_{ijk} / v_i w_{ij}) = v_i w_i \lambda_{ijk}(t_{ijk}) = v_i w_{ij} \lambda_0(t_{ijk}) \exp(\beta^i x_{ijk})$$

where $\lambda_0(t_{ijk})$ represents the baseline hazard and $\exp(\beta x_{ijk})$ is the relative risk associated with covariates $x_{ijk.}$ We assume that the random effects $v_i$ and $w_{ij}$ follow the gamma distribution with a mean of one. The assumption of the random effects being gamma-distributed follows previous research on unobserved heterogeneity, which makes use of this distribution[70].

We used the STCOX command in STATA to compute the coefficients for the family and community frailty effects for infant and child mortality. We fitted the following two models to the data:

- Model I: Single random effect to allow for clustering by family;
- Model II: Single random effect to allow for clustering by community

The estimated coefficients from the two models were interpreted just as in a standard hazard model, while the estimated parameters describing the distributions of the frailty effects were interpreted as variances of the frailty distribution. If the variance is zero, observations from the same family or community are independent. A larger variance implies greater heterogeneity in frailty across families or communities and greater correlation among individuals belonging to the same family or community. A frailty value of one indicates that the measured covariates in the model do not explain any variation in mortality. In this

case all the variation in mortality is attributable to unmeasured or immeasurable factors.

## 7.3 Family and Community Frailty Models

Section 7.4.1 reports on the analysis of data on the clustering of infant deaths at the family and community levels. In turn section 7.4.2 analyses family and community frailty for child deaths. The results on the impact of maternal, socioeconomic and environmental factors on infant and child mortality are presented in Tables 7.1 and 7.2, respectively, with two frailty models fitted to these data. Model I includes a single random effect to allow for clustering of deaths by family. Model II is based on clustering of deaths at the level of community.

### *7.3.1 Infant Mortality*

Section 7.4.1 presents frailty models for infant mortality. The results regarding the various relationships between infant mortality and the independent variables are shown in Table 7.1. In this section we draw comparisons with the standard hazard model for infant mortality that was presented in Table 6.5.

#### *7.3.1.1 Family Clustering Model*

The frailty effect for infant mortality at the family level is 0.056 (see Table 7.1). This family frailty effect for infant mortality is not statistically significant. This implies that the risks of infant deaths between households in the 2005-06 ZDHS survey sample do not significantly differ even after controlling for a number of unknown determinants of infant mortality. The family frailty effect estimate indicates that the death of one infant in a household due to unobserved family characteristics is associated with a 5.6 percent increase in the risk of the index child dying relative to what it would be if that child were alive. This family frailty

effect is not substantial and further implies that the measured covariates in model I of Table 7.1 account for 94.4 percent of the variation in infant mortality at the family level in the 2005-06 ZDHS survey.

Three patterns emerge when comparing the results from the Cox proportional hazard model of infant mortality presented in model III of Table 6.5 with the hazard model incorporating a single random effect for each family presented in model I of Table 7.1. First, there is a small increase in the magnitude of the coefficients for sex of child, type of birth (singleton or multiple), wealth status and piped drinking water after introducing the family random effect. Second, there is a small decrease in the coefficients for birth order and preceding birth interval, maternal age and flush toilet. Third, the coefficients for place of residence, maternal education and paternal education levels remain largely unchanged.

*7.3.1.2 Community Clustering Model*

This community frailty effect for infant mortality is 0.101 and this value is not statistically significant (see model II of Table 7.1). This community frailty effect implies that the unexplained variation in infant mortality by the covariates in model II at the community level is 10.1 percent. This further indicates that the covariates in model II explain 89.9 percent of the variation in infant mortality at the community level in the 2005-06 ZDHS survey. The community effect represents the association among children residing in the same community that is a consequence of their shared physical, disease, cultural, and socioeconomic environment.

Overall, the effects of individual - level covariates that are indicative of high risk families tend to be underestimated in the standard model that includes no correction for clustering. Communities characterised by high frailty have more deaths. Covariates that are exogenous and essentially

independent of community factors, most notably child sex and maternal age, change the least between the standard hazard model and the community frailty model.

*7.3.2 Child Mortality*

Section 7.4.2 presents frailty models for child mortality. The results regarding the various relationships between child mortality and all maternal, socioeconomic and environmental contamination variables are presented in Table 7.2. The standard hazard model for child mortality was presented in Table 6.10.

*7.3.2.1 Family Clustering Model*

The results of the parameter estimates presented in Table 7.2 are largely in line with theoretical expectations and patterns observed for child mortality. The family frailty effect for child mortality is 0.086 and is non-significant. This implies that the unexplained variation in child mortality by the covariates in the model at the family level is 8.6 percent. The observed maternal, socioeconomic and environmental contamination variables in model I of Table 7.2 explain 91.4 percent of the family variation for child mortality in Zimbabwe. We further observe from comparison of data in Table 7.1 and Table 7.2 that family frailty is larger for child mortality than infant mortality. Overall, a comparison of the strengths of the relationships between model III of Table 6.10 and model I of Table 7.2 shows marginal differences in the relative risks for the covariates in these two models. Two distinct patterns emerge in model I of Table 7.2, which allow us to study the family random effect on child mortality.

First, there is an increase in the coefficients depicting the impact of maternal and environmental contamination variables on child mortality.

Second, there is a decrease in the coefficients depicting the impact of socioeconomic variables on child mortality.

We observe that children of birth order 2-5 and short preceding birth interval are 31 percent more likely to die in child age than those of birth order 2-5 and long preceding birth interval. This relationship is however not significant. The lack of a U-shaped relationship between maternal age and child mortality is maintained in the presence of the random effect for the family in model I. However, giving birth at a young age that is, less than 20 years continues to be a risk factor in the presence of the family random effect. The sex of the child also continues to have a very marginal effect on child mortality even in the presence of the family random effect.

Similar to the results in Table 6.10, the impact of the type of birth variable on child mortality marginally increased though it is not significant. Living in the rural areas similarly increases the risk of child death by a third relative to living in the urban areas. Compared to Table 6.10, the impact of maternal education on child mortality is marginally decreased in the presence of the family random effect. Attaining secondary and higher education for mothers reduces the risk to child death by 41 percent relative to no education.

Unlike in model III of Table 6.10 where attaining secondary and higher education for fathers reduces the risk to child death by 33 percent, attaining secondary and higher education in Table 7.2 reduces the risk to child death by 32 percent in the presence of the family random effect. The coefficients depicting the impact of wealth status on child mortality shows in Table 7.2 that children born to mothers in households classified as “middle” experience 24 percent higher mortality risk of dying in child age than children born to mothers in households classified

as 'poor'. As previously noted in this study, this could be due to differential application of childcare practices.

The impact of the availability of piped water and a flush toilet on child mortality is marginally reduced in the presence of the family random effect as shown in model I of Table 7.2. The availability of piped water for the household reduces child mortality risk by 32 percent in the presence of the family random effect although the relationship is not significant. A similar observation is made for the availability of a flush toilet in the dwelling. Access to a flush toilet significantly reduces the risk to child death by 58 percent ($p<0.01$).

We now turn to section 7.4.2.2 where we present the results on the impact of all the independent variables on child mortality after allowing for the community random effect.

*7.3.2.2 Community Clustering Model*

Table 7.2 depicts the impact of all maternal, socioeconomic and environmental contamination variables on child mortality allowing for the community frailty effect (model II). We also draw comparisons with the standard model II of Table 6.10 in this section. The community frailty effect for child mortality is 0.155 and is statistically significant ($p< 0.05$). This means that the unexplained variation in child mortality by the covariates in the model at the community level is 15.5 percent. These results imply that the observed measured maternal, socioeconomic and environmental contamination variables in model II of Table 7.2 explain 84.5 percent of the variation in child mortality at community level. These results suggest that there is greater significant variation between communities in the risk of child mortality that is not accounted for by the measured maternal, socioeconomic and environmental contamination

factors in model II than in the standard model. In addition, the community frailty effect is larger in childhood than in infancy.

The differences in the strength of the relationships between the covariates in model III of Table 6.10 and model II of 7.2 are marginal. We further found that model II exhibits marginal increases in the effects of the covariates on child mortality from the preceding model in Table 7.2. Two diverse patterns are similarly observed after introducing the community random effect in model II: an increase in the coefficients for maternal and environmental contamination covariates and a decrease in the coefficients for socioeconomic covariates. The direction of the hazard ratios in model II is analogous to that observed in model I.

The results in model II show that the elevated child mortality risk is associated with children of birth order 2-5 and short intervals, maternal age of less than 20 years, multiple births, living in the rural areas and being born to mothers of 'middle' wealth status. A lower risk to child death is associated with secondary and higher maternal and paternal education, availability of piped water and a flush toilet for the household. The relationships of the covariates with child mortality are largely non-significant after introducing the community frailty effect with the exception of that for flush toilet which is significant at $p<0.01$.

## 7.4 Concluding Remarks

Chapter 7 presented an analysis of family and community frailty effects. We observed that family and community variance of random effects are higher for child mortality than infant mortality. Only the variance of random effect for child mortality is significant. This indicates that there is substantial variation in the risk of child death among communities in the 2005-06 ZDHS sample that is not taken into account by the covariates of the standard model. We found that there were marginal changes in the coefficients of the covariates for both infant and child

mortality after we introduced the family and community random effects. As observed in the Cox proportional standard hazard models, maternal factors were predominant in the infancy phase while socioeconomic and environmental contamination factors were predominant during the childhood phase. We further found that many of the relationships between the covariates and mortality were not significant, but they were often in the expected direction.

This study has demonstrated that child mortality risks vary due to unobserved factors at the community levels. Unobserved heterogeneity at the community level has been shown to be higher than that at the family level. Children in the same community live under similar climatic, environmental, socioeconomic and cultural conditions and are exposed to the same illness and diseases prevalent at community level.[68] The results of our frailty models suggest that the effects of community factors such the availability of hospitals and natural and economic resources are likely to be important. A major limitation of the DHS data is that it does not collect community information. Future studies should therefore endeavour to include community factors where they are available.

We now turn to Chapter 8, which presents the overall discussion of the findings, conclusions of the study and recommendations. Chapter 8 will also deal with the implications of the findings for child health policy formulation, programming and further research.

Table 7.1: Impact of Maternal, Socioeconomic and Environmental Contamination Variables on Infant Mortality, Controlling for Family and Community Frailty, 1996-2005 (2005-06 ZDHS), Zimbabwe

| Covariate | Family Frailty Model (Model I) | | Community Frailty Model (Model II) | |
|---|---|---|---|---|
| | **Relative Risk** | **Confidence interval** | **Relative Risk** | **Confidence interval** |
| *Birth order and preceding birth interval*[1] | | | | |
| First births | 1.090 | 0.845-1.418 | 1.090 | 0.851-1.416 |
| 2-5 and short | 1.391 | 0.949-2.027 | 1.322 | 0.945-2.021 |
| 2-5 and medium | 1.477 | 0.945-2.308 | 1.477 | 0.945-2.308 |
| 2-5 and long | 1.000 | ---------- | 1.000 | ---------- |
| 6+ and short | 2.851*** | 1.512-5.134 | 2.815*** | 1.626-5.212 |
| 6+ and medium | 1.132 | 0.547-2.185 | 1.190 | 0.595-2.274 |
| 6+ and long | 1.323 | 0.896-1.858 | 1.374 | 0.821-1.932 |
| *Maternal age* | | | | |
| <20 years | 1.145 | 0.685-1.594 | 1.149 | 0.771-1.674 |
| 20-29 years | 1.088 | 0.795-1.381 | 1.012 | 0.731-1.352 |
| 30-39 years | 1.000 | ---------- | 1.000 | ---------- |
| 40-49 years | 1.077 | 0.611-1.892 | 1.079 | 0.617-1.898 |
| *Sex of child* | | | | |
| Female | 0.988 | 0.796-1.162 | 0.962 | 0.816-1.176 |
| Male | 1.000 | ---------- | 1.000 | ---------- |
| *Type of birth* | | | | |
| Multiple | 2.041*** | 1.537-2.725 | 2.187*** | 1.532-2.742 |
| Singleton | 1.000 | ---------- | 1.000 | ---------- |
| *Residence* | | | | |
| Rural | 1.329 | 0.761-2.295 | 1.334 | 0.765-2.297 |
| Urban | 1.000 | ---------- | 1.000 | ---------- |
| *Maternal education* | | | | |
| No education | 1.000 | ---------- | 1.000 | ---------- |
| Primary | 1.082 | 0.725-1.752 | 1.085 | 0.721-1.748 |
| Secondary and higher | 1.073 | 0.632-1.721 | 1.092 | 0.615-1.753 |

[1] Preceding birth interval: short <= 18 months, medium 19-23 months, long 24+ months.

Table 7.1 (Continued)

| Covariate | Family Frailty Model (Model I) | | Community Frailty Model (Model II) | |
|---|---|---|---|---|
| | **Relative Risk** | **Confidence interval** | **Relative Risk** | **Confidence interval** |
| *Paternal education* | | | | |
| No education | 1.000 | ---------- | 1.000 | ---------- |
| Primary | 1.121 | 0.784-1.495 | 1.125 | 0.781-1.503 |
| Secondary and higher | 1.127 | 0.821-1.575 | 1.131 | 0.815-1.582 |
| *Wealth index* | | | | |
| Poor | 1.000 | ---------- | 1.000 | ---------- |
| Middle | 1.089 | 0.832-1.381 | 1.093 | 0.837-1.379 |
| Rich | 1.082 | 0.715-1.629 | 1.088 | 0.719-1.639 |
| *Piped drinking water* | | | | |
| Yes | 0.903 | 0.523-1.321 | 0.927 | 0.539-1.342 |
| No | 1.000 | ---------- | 1.000 | ---------- |
| *Flush toilet* | | | | |
| Yes | 0.610 | 0.335-1.129 | 0.636 | 0.339-1.133 |
| No | 1.000 | ---------- | 1.000 | ---------- |
| ***Variance of Random Effect*** | | | | |
| **Family Frailty Effect** | **0.056** | | | |
| Standard Error | 0.043 | | | |
| **Community Frailty Effect** | | | **0.101** | |
| Standard Error | | | 0.037 | |

*p<0.05, **p<0.01, ***p<0.001

Source: Author's calculations Zimbabwe Central Statistical Office/ Macro International Inc21

Table 7.2: Impact of Maternal, Socioeconomic and Environmental Contamination Variables on Child Mortality, Controlling for Family and Community Frailty, 1996-2005 (2005-06 ZDHS), Zimbabwe

| Covariate | Family Frailty Model (Model I) | | Community Frailty Model (Model II) | |
|---|---|---|---|---|
| | **Relative Risk** | **Confidence interval** | **Relative Risk** | **Confidence interval** |
| *Birth order and preceding birth interval*[1] | | | | |
| First births | 0.620* | 0.221-0.974 | 0.633* | 0.332-0.968 |
| 2-5 and short | 1.314 | 0.515-3.375 | 1.325 | 0.511-3.268 |
| 2-5 and medium | 0.821 | 0.330-2.037 | 0.832 | 0.321-2.029 |
| 2-5 and long | 1.000 | ---------- | 1.000 | ---------- |
| 6+ and short | 0.921 | 0.115-6.672 | 0.927 | 0.119-6.675 |
| 6+ and medium | 0.733 | 0.085-5.189 | 0.736 | 0.091-5.292 |
| 6+ and long | 0.981 | 0.496-1.878 | 0.984 | 0.498-1.874 |
| *Maternal age* | | | | |
| <20 years | 1.425 | 0.776-2.691 | 1.418 | 0.754-2.629 |
| 20-29 years | 0.912 | 0.571-1.462 | 0.896 | 0.552-1.392 |
| 30-39 years | 1.000 | ---------- | 1.000 | ---------- |
| 40-49 years | 0.267 | 0.083-1.132 | 0.261 | 0.059-1.117 |
| *Sex of child* | | | | |
| Female | 1.033 | 0.751-1.443 | 1.031 | 0.735-1.417 |
| Male | 1.000 | ---------- | 1.000 | ---------- |
| *Type of birth* | | | | |
| Multiple | 1.496 | 0.657-3.423 | 1.502 | 0.643-3.433 |
| Singleton | 1.000 | ---------- | 1.000 | ---------- |
| *Residence* | | | | |
| Rural | 1.291 | 0.541-2.928 | 1.315 | 0.561-2.952 |
| Urban | 1.000 | ---------- | 1.000 | ---------- |
| *Maternal education* | | | | |
| No education | 1.000 | ---------- | 1.000 | ---------- |
| Primary | 0.761 | 0.365-1.577 | 0.752 | 0.368-1.556 |
| Secondary and higher | 0.590 | 0.281-1.269 | 0.589 | 0.269-1.258 |

*p<0.05, **p<0.01, ***p<0.001

[1] Preceding birth interval: short <= 18 months, medium 19-23 months, long 24+ months.

Table 7.2 (Continued)

| Covariate | Family Frailty Model (Model I) | | Community Frailty Model (Model II) | |
|---|---|---|---|---|
| | **Relative Risk** | **Confidence interval** | **Relative Risk** | **Confidence interval** |
| *Paternal education* | | | | |
| No education | 1.000 | ---------- | 1.000 | ---------- |
| Primary | 0.625 | 0.364-1.125 | 0.619 | 0.339-1.088 |
| Secondary and higher | 0.684 | 0.375-1.182 | 0.679 | 0.363-1.167 |
| *Wealth index* | | | | |
| Poor | 1.000 | ---------- | 1.000 | ---------- |
| Middle | 1.239 | 0.804-1.901 | 1.232 | 0.801-1.909 |
| Rich | 1.058 | 0.542-2.018 | 1.054 | 0.558-2.029 |
| *Piped drinking water* | | | | |
| Yes | 0.683 | 0.318-1.119 | 0.689 | 0.321-1.123 |
| No | 1.000 | ---------- | 1.000 | ---------- |
| *Flush toilet* | | | | |
| Yes | 0.423** | 0.163 0.931 | 0.427** | 0.169 0.933 |
| No | 1.000 | ---------- | 1.000 | ---------- |
| ***Variance of Random Effect*** | | | | |
| **Family Frailty Effect** | **0.086** | | | |
| Standard Error | 0.042 | | | |
| **Community Frailty Effect** | | | **0.155*** | |
| Standard Error | | | 0.045 | |

*p<0.05, **p<0.01, ***p<0.001

Source: Author's calculations Zimbabwe Central Statistical Office/ Macro International Inc21

# CHAPTER 8

# DISCUSSION OF FINDINGS, CONCLUSIONS AND RECOMMENDATIONS

## 8.1 Introduction

This chapter describes and discusses the major findings of the research. The chapter concludes with recommendations towards health policy and child health programming in Zimbabwe.

## 8.2 Summary and Discussion of Major Findings

### *8.2.1 Levels and Trends of Infant and Child Mortality*

In this section the findings on levels and trends of infant and child mortality are discussed. Firstly, the direct estimates are discussed, followed by the indirect estimates. Finally, the results obtained from the application of the multiple-spline regression technique to data from multiple demographic sources in Zimbabwe are discussed.

We studied the trends in infant and child mortality with direct estimates from DHS-type surveys conducted in Zimbabwe between 1988 and 2005 and found the following results. Whereas neonatal, infant and child mortality declined during the period 2001-2005, postneonatal mortality stagnated at 36 deaths per 1,000 live births during this period. Under-5 mortality declined from 102 deaths per 1,000 live births during the 1995-1999 period to 82 deaths per 1,000 live births during the 2001-2005 period.[21] The majority of the decline in under-5 mortality would appear to be the result of the decline of 40 percent in child mortality during 2001-2005. Overall, the findings on the decline between 1995-1999 and 2001-2005 were unexpected. Infant and under-five mortality rates were expected to have increased during this period due to the direct and indirect impact of HIV and AIDS and the worsening economic and political conditions in Zimbabwe during the 1995-2006 period.

Several reasons can be mentioned making it likely that the decline in under-5 mortality in the late 1990s and early 2000s in not genuine. First, the vaccination coverage and nutritional status of under-five children in Zimbabwe worsened between 1994 and 2005-06. For instance, the percentage of children aged 12 to 24 months who had not received any vaccinations was more than five times higher in 2005-06 than in 1994 (4 percent and 21 percent, respectively). It is therefore difficult to accept that childhood mortality could have declined between 1999 and 2005 in Zimbabwe.

Second, we found that there is a discrepancy between infant and under-5 mortality estimates referring to the 5-9 year-period preceding the 2005-06 ZDHS survey with those for the 0-4 year-period preceding the 1999 ZDHS survey.[14,21] These rates should be more or less similar, but they are not.

Third, we hypothesise that the difference in mortality estimates between the 1999 and 2005-06 ZDHS surveys could be due to the excess mortality among women of childbearing age that occurred from 1993-1999 to 2000-2005. The 2005-06 ZDHS survey report states that adult mortality has continued to rise between 1996-1999 to 2000-2005 by around 40 percent among women and 20 percent among men. Adult female mortality is highest among women in the 30-39 year age group whose mortality doubled to 23 deaths per 1,000 during the 1993-1999 and 2000-2005 periods. It could be that these mothers are “missing” in the 2005-06 ZDHS survey and this means that their children – with higher than average mortality rates - were also not included.

One interesting observation from this study is that the mortality gap by rural-urban and sex of child differentials in Zimbabwe has narrowed since 1995. For instance, the child mortality rate decreased to 21

deaths per 1,000 live births for both sexes during the period 1995-1999 from levels of 35 and 31 deaths per 1,000 live births during the 1990-1999 period, for males and females, respectively. Under-five mortality decreased to 72 and 64 deaths per 1,000 births from 100 and 69 deaths per 1,000 live births in rural and urban areas, respectively. Similar declines were recorded with respect to all the subcomponents of under-five mortality during 1990-1999.

The closing of the mortality gap between rural and urban areas could possibly be due to the effectiveness of the programmes for equitable distribution of health facilities aimed at removing the discrepancies between rural and urban areas in Zimbabwe. The population in rural areas has had better and improved access to health facilities since independence in 1980 in Zimbabwe. The narrowing of the male-to-female mortality gap could also be partly due to the diminishing gender inequality.

We found that there are still substantial provincial variations in under-five mortality in Zimbabwe. What is intriguing is that the under-five mortality rate in Harare (65 per 1,000 live births) was higher than that for Bulawayo (45 per 1,000 live births).

In order to evaluate the plausibility of the direct estimates, we also computed indirect estimates of infant and under-five mortality. Whereas direct estimates are computed directly from the data, indirect techniques use models and/ or consistency checks to estimate demographic parameters. A comparison of direct and indirect estimates of infant and under-five mortality revealed that although the indirect estimates were somewhat higher than the direct estimates, the differences were small, giving credence to the correctness of the direct estimates.

The results from the multiple-spline regression analysis showed that the data points from the 2005-06 ZDHS survey deviate from the other three rounds of the ZDHS surveys. The regression analysis showed that infant and under-five mortality declined during the period from 1960 to 1990 and that the decline in mortality stalled from the 1990s to 2005.

Having discussed the major findings on the levels and trends of infant and child mortality, we now turn to the next section which discusses the major findings on the determinants of infant and child mortality.

*8.2.2 Determinants of Infant and Child Mortality*

We used the Cox proportional hazards method to estimate the bivariate and multivariate coefficients on the impact of a number of factors on infant and child mortality. In general, differences in the strength of the relationships using bivariate and multivariate relationships were small which means in the discussion we deal with both of them together. We conducted the bivariate and multivariate analysis in this study for both infant and child mortality. These results will be discussed simultaneously. We will discuss the determinants of infant mortality and add those for child mortality in so far as the results from the determinants of infant mortality are different from those of child mortality.

We found that in many cases the relationships between independent variables and infant and child mortality were in the expected direction but for only a few variables was the relationship statistically significant. This is illustrated in the tables in chapter 6 and 7. There is at the end of this section a hypothesis on why the relationships between the independent and dependent variables were smaller than expected. We now look at the impact of the independent variables on infant and child mortality in more detail.

*8.2.2.1 Maternal Variables and Mortality*

We found that there is a substantial elevated mortality risk to infants of birth order 6 and more and infants born after short birth intervals, that is, intervals of less than 18 months. What we have found is in accordance with other findings on the influence of birth order and birth spacing.

It has been firmly established in the demographic literature that infant and child mortality are higher for births spaced at short intervals and high parity births. Short birth intervals increase the number of children of more or less similar (comparable) ages in the household. Alam[71] argues that this consequently heightens the susceptibility of children to infectious diseases due to their physical proximity and leads to siblings' competition for household resources including individualised maternal care.

Results from this study indicated that children born to young (less than 20 years) and older (40-49 years) mothers experienced 20 percent higher mortality risk than those born to mothers aged 30-39 years. Maternal age therefore still remains an important factor impacting on infant mortality in Zimbabwe where the median age at childbearing is 26 years.[21] Children born to women in their mid-twenties, which is close to the median age at childbearing in Zimbabwe, exhibit the lowest mortality risks relative to women aged 30-39 years. The effects of maternal age at birth are both physiological and sociopsychological. The immature reproductive systems of young mothers and the depleted physiological systems of older mothers due to repeated pregnancies makes them susceptible to pregnancy complications and bearing low birth weight babies both of which are associated with a higher risk of child death. Family planning could mitigate the negative effects of young and old maternal age at birth on child survival.[72]

We found that female infants are associated with less than 2 percent lower mortality relative to male infants. This is consistent with the observations made earlier that the mortality gap by sex of child differentials in Zimbabwe has narrowed since 1995.

We also found that children from multiple births have an elevated mortality risk of 2.06 and 1.49 times higher during infancy and child age, respectively, compared to singleton births. The relative risk is highly significant for infant mortality and is not significant for child mortality. These findings are consistent with literature documenting the elevated mortality risk associated with twins in infancy.[19]

*8.2.2.2 Socioeconomic Variables and Mortality*

Living in the rural areas is associated with an elevated risk to child death of 1.26 times high relative to living in the urban areas. These findings are expected given the discrepancies between rural and urban areas in terms of health infrastructure. It was also noted in the study of trends in under-five mortality that the mortality gap by rural-urban differentials in Zimbabwe has narrowed since 1995.

We observed that the impact of education is higher in childhood than in infancy although none of these relationships are statistically significant. Attaining secondary and higher education reduces the risk of child death by 40 and 33 percent for mothers and fathers, respectively, relative to no education. The modest impact of maternal schooling on infant mortality in Zimbabwe during the period 1996-2005 is worth commenting. This finding is consistent with results from an analysis of Egyptian data by Casterline et al[73] where maternal education did not have a discernible impact on infant mortality. The lack of an impact of maternal education on infant mortality seems to be observed only in the 2005-06 ZDHS survey. The data for the other 3 rounds of the ZDHS

surveys conducted in 1988, 1994 and 1999 show that maternal education has a depressing impact on infant and child mortality.

Previous research has identified maternal education as the socioeconomic factor of central importance in determining children's survival chances. Maternal education is often used to indicate mother's level of skills and knowledge to effectively utilise the available health and childcare resources. According to Caldwell, education supplies women with the knowledge and skills to raise healthy children, provides women with higher income through higher earnings or selective mating, increases the value of time, alters preferences, and changes the structure of intrahousehold relationships.[24,43,74] Cleland and van Ginneken[24] further argue that "The inverse education-mortality relationship is found in all major regions of the developing world; the association is very pronounced, but appreciably closer in childhood than in infancy; and even a modest exposure of the mother to formal schooling is associated with reduced risks of death in most contexts. It is thus important both for a better understanding of health determinants and for practical policy reasons, to reassess whether the education-mortality relationship is a causal one, and if so, to identify the pathways of influence" (p.1359). Mothers with more education are also more able to overcome the adverse effects of some factors such as very young and old maternal age and short birth intervals.

We further found that the impact of wealth status on infant mortality is not as discernible as that on child mortality. The impact of wealth status on child mortality revealed that children born to mothers in households of middle wealth status had mortality risk of 1.24 times high relative to those born to mothers in poor households. This relationship was however not significant. This could be due to use of different child-care practices in these two groups.

*8.2.2.3 Environmental Contamination Variables and Mortality*

Our results revealed that access to sanitation facilities namely piped drinking water and flush toilet, are associated with low infant and child mortality. However, the relationships are only significant for the presence of a flush toilet and only for child mortality. Having access to piped drinking water and flush toilet facilities reduce child mortality by 39 and 60 percent, respectively, relative to having none of these facilities.

Previous research suggests that the type of toilet facility and source of drinking water reflect the level of environmental contamination, which determines the transmission of infectious agents to children.[48,75,76] These factors also serve as a proxy for information unavailable from the survey including household hygiene, standards of cleanliness, food preparation and storage practices.[77,78]

Our study confirms, therefore, that since the majority of the population in Zimbabwe (69 percent) relies on unsafe water sources, conditions of use, including manner of storage and whether families treat the water before using it, are important factors in determining infant and child survival in Zimbabwe.

*8.2.2.4 HIV/AIDS and Infant and Child Mortality*

The results on the inclusion of HIV/AIDS in the multivariate models indicate that HIV/AIDS does have an influence on infant and child mortality in Zimbabwe. A unit increase in HIV prevalence significantly increases the odds of dying in infancy by 10 percent and in childhood by 62 percent in the presence of maternal, socioeconomic and environmental contamination variables. However, these results should be treated with caution as they only suggest that HIV among mothers has an effect on under-5 mortality. These results are consistent with those by Hill, Bicego and Mahy[5] who found that the prevalence of

HIV/AIDS was strongly associated with elevated mortality child mortality risks in Kenya in the 1990s.

Marindo and Hill[20] however observe that it is difficult to measure the impact of HIV/AIDS on under-5 mortality from surveys that utilise individual reports from the mothers. They observe that for the child to be infected with HIV/AIDS the mothers have to be infected as well. Child mortality estimates are likely to be understated as the mothers of infected children are likely to have died before the time of the survey and are therefore not present to report the deaths of their infected children in the survey (ibid). However, our results are important in as far as they show the potential direct and/or indirect influence that HIV/AIDS could have on infant and child mortality. Programming for child health has to include efforts to reduce the spread of HIV/AIDS in Zimbabwe, especially if the Millennium Development Goal (MDG4) is to be achieved.

As already mentioned in Chapters 4 and 5, we hypothesise that the decline in the under-5 mortality estimates between 1999 and 2005-06 is not genuine and could be due to the increase in mortality among women of reproductive age between 1996-1999 and 2000-2005. We earlier observed in this study that adult mortality among women of childbearing age increased by 40 percent between 1996-1999 and 2000-2005. Close to 60 percent of these extra deaths were to women aged 30-44 years.[21] Therefore, a group of older women who would have had children with higher than average mortality rates were “missing” from the 2005-06 ZDHS survey. It is probably the finding that there were a number of “missing mothers” from the survey which not only led to the possible underestimation of the true levels of infant and child mortality in the 2005-06 ZDHS survey, but also to the lack of expected relationships between infant and child mortality and the independent variables in the

2005-06 ZDHS survey. For example, the 1994 and 1995 ZDHS surveys show a stronger impact of maternal education on under-5 mortality. This impact completely disappears in 2005-06. We elaborated already on this point in the final section of Chapter 5. Further research is required to determine the plausibility of the mortality estimates from the 2005-06 ZDHS survey.

In this section we discussed the major results on the determinants of infant and child mortality. The next section discusses results on the frailty hazard models.

*8.2.3 Frailty Hazard Models*

Results presented in Chapter 5 and 6 have shown that infant and child mortality vary due to the measured maternal, socioeconomic, environmental contamination and personal illness control factors even though the relationships between the independent variables and mortality were often not statistically significant. In this section we discuss the findings on frailty at the family and community level. Frailty, in the infant and child mortality models presented in Chapter 7, represents a child's susceptibility to the risk of death. It captures the total effect of all factors that influence the child's risk of death that are not included in the baseline hazards presented in chapter 6.

Since the models presented in chapter 6 can account for observed covariates, the frailty effects presented in Chapter 7 represent unmeasured or immeasurable effects on infant and child mortality. Zimbabwe provides one of the better settings in which to estimate the unobserved family and community effect. This is because the capacity to be able to measure the clustering of mortality risk is much greater in settings with relatively high fertility and high mortality.[71,79,80] It is the association among siblings' survival that allows the estimation of the

family and community frailty effect.[81,82] We found that there is marginal difference between the coefficients of the standard models and those representing the effect of frailty for both infant and child mortality. The frailty effects at the family level for infant and child mortality were 5.6 and 8.6 percent, respectively. The frailty effects were not statistically significant. This implies that the risks of infant and child deaths between households in the 2005-06 ZDHS survey sample do not significantly differ even after controlling for a number of known determinants of infant and child mortality.

The frailty effects at the community level for infant and child mortality were 10.1 and 15.6 percent, respectively. We further found that only the frailty effect at community level and for child mortality was statistically significant. This implies that the risks of child deaths between communities in the 2005-06 ZDHS survey sample significantly differ even after controlling for a number of known determinants of child mortality. It further implies that the variables in the child mortality model explained 84.4 percent of the community variation in child deaths in the 2005-06 ZDHS survey sample.

We now compare the results from this study with those of Guo[80] who estimated a family random effects model for infant mortality using Guatemalan data and Zenger[83] who estimated a similar model using data from Bangladesh and Curtis et al[84] who studied family frailty in Brazil.

We find that the family frailty effect is much smaller for Zimbabwe (0.056) than for Guatemala (0.610) and much larger for Bangladesh (close to zero). In contrast, Curtis et al[84] report the presence of large and highly significant family frailty effects in a study of postneonatal mortality in Brazil. All these results call for further research to determine

the effect of unobserved heterogeneity in sub-Saharan Africa. Child health policy and programming in Zimbabwe should focus on those aspects that make some communities more prone to morbidity and mortality relative to other communities.[85]

## 8.3 Conclusions

The findings from this study on the levels and trends of childhood mortality revealed that infant and under-five mortality declined in Zimbabwe in the period 1999-2005. This decline is difficult to accept and should be treated with caution due to a number of reasons that were mentioned in the previous section. What is also intriguing in our findings in this study is the disappearance of mortality gaps by sex and rural-urban differentials in Zimbabwe. However, we found that provincial differentials in infant and child mortality still exist in Zimbabwe.

The findings on the determinants of childhood mortality illustrate that the maternal variables are more important during the infant age (0-11 months) than during the childhood age (12-59 months). Infant mortality risk is increased for children of higher birth orders and short preceding birth interval. Higher parity and short preceding birth spacing is clearly risky for the child and is also harmful to the mother's health.

We also found that socioeconomic and environmental factors are more important during the childhood age than during infancy but many of these relationships were not statistically significant. This study supports health policy initiatives stimulating the use of family planning methods to increase birth intervals. This would lead to a reduction in infant and child mortality in Zimbabwe. Many women, particularly those living in rural areas or those without formal education are aware of the benefits of family planning, but because of some social, economic, and cultural factors do not practice it. These women may not even be aware of the

advantages of birth spacing using modern contraception. However, women living in urban areas or who have higher educational levels are more likely to use modern family planning methods.

We found that family frailty for both infant and child mortality (5.6 and 8.6 percent) is not substantial and is non-significant. There is however substantial community frailty with respect to infant and child mortality. The magnitude of the unexplained variation in infant mortality by the measured independent variables at community level is 10.1 percent while that for child mortality is 15.6 percent. Only the last finding is statistically significant. These results suggest that there is considerable variation among communities which indicates that child deaths are likely to be clustered in certain communities in Zimbabwe. Appropriate child health programmes should target the most vulnerable communities in Zimbabwe in order to advance child survival prospects and to achieve the Millennium Development Goals.

## 8.4 Limitations

This section gives an overview of the limitations that could have affected this study. The birth history data in the DHS surveys is collected retrospectively and mothers with greater levels of education are probably less likely to omit births that end in death than are mothers with less education. Hence the effect of education on child survival obtained from the DHS surveys may be underestimated.

The analysis of independent variables in this study is restricted to 10 years before the 2005-06 ZDHS survey, that is, 1996-2005 so that the hazard ratios are based on a sufficient number of cases in each category to ensure statistically reliable estimates. A potential problem with analysing births from a ten-year window period is that many of the independent variables refer to conditions or characteristics at the time of

the survey. These may not be the actual conditions under which the children were exposed to the risk of death. Our analysis however focused on independent variables which are likely to reflect accurately actual conditions under which children were exposed to the risk of death even though they were recorded at the time of the interview.

For example, the maternal variables (birth order, preceding birth interval, maternal age and type of birth) that we analysed are fixed for each child and refer to the situation under which the child was exposed to the risk of death even though the information was collected at the time of the survey. Furthermore, women's education is usually completed before or upon the birth of her first child.

8.5 Recommendations

In this section we provide the recommendations emanating from this research.

1. In the light of the conclusions drawn from this study, we recommend that the DHS programme should consider using not only direct, but also indirect techniques of estimating childhood mortality in order to facilitate comparison with estimates from censuses and other demographic surveys. Indirect mortality estimates refer to a specific time location, which is important in studying mortality levels and trends. The DHS programme uses life-table probability techniques to estimate childhood mortality. These estimates refer to 0-4, 5-9 and 10-14 years preceding the survey. They do not have a clearly defined reference point in time. We assume, therefore, that the DHS mortality estimates refer to the mid-point of the interval in years preceding the survey. This reference point is less precise than in the case of use of indirect techniques.

2. In countries such as Zimbabwe where the HIV prevalence had in 2005-06 declined to 18.1 percent[21] and still very high, we recommend that results of research projects mentioning a decline in under-5 mortality should be interpreted with caution. It may not necessarily be a true reflection of trends in child health.

3. The results suggest that HIV/AIDS either directly or indirectly influences the levels of under-5 mortality in Zimbabwe. Programmes to reduce under-5 morbidity and mortality should be intertwined with those targeting the control of the spread of HIV/AIDS in Zimbabwe.

4. A contraceptive prevalence rate of 58 percent is encouraging for Zimbabwe. Family planning programmes should therefore be aimed at educating women and men with low educational levels and those in rural areas about the potential benefits of long-term birth spacing and encouraging them to use birth spacing methods. These programmes need to be made more accessible to both urban and rural women. Furthermore, given the findings from this study, an improvement in family planning service provision will definitely enhance child survival in Zimbabwe.

5. Policies that weaken and remove the social, economic, and cultural barriers to the use of contraception and promote appropriate breastfeeding and are directed at women in rural areas with low educational levels, are especially needed in Zimbabwe. One such policy is to enhance the training of traditional midwives and also train traditional healers and absorb them into the public health system.

6. The multi-spline robust regression method can ably reconcile differences in values from multiple data sources by extrapolating a linear regression line. On this basis, we recommend use of this method to estimate childhood mortality levels and trends in Zimbabwe.

7. On-going data collection programmes, such as the Demographic and Health Surveys should continue to gather information on morbidity and mortality in under-5 children. These survey programmes continue to contribute to the knowledge-base of child health conditions and use of health services in developing countries.

8. Additional research is needed to determine the plausibility of the recent decline in childhood mortality in Zimbabwe before a conclusion is accepted that mortality declined over the period between the 1999 and 2005-06 ZDHS surveys.

9. Further research is required to test the hypothesis that the unexpected decline in under-5 mortality and the change in the relationships of the covariates with mortality are probably due to the “missing children” and “missing mothers” in the 2005-06 ZDHS survey.

10. The determinants of adult female mortality should also be more thoroughly studied, particularly with the high levels of AIDS - related adult female deaths in countries such as Zimbabwe. A more refined analysis could uncover interesting insights on the impact of independent variables on adult female mortality.

11. Multiple births are strongly negatively associated with infant survival in Zimbabwe independent of other risk factors. This evidence suggests that improving maternal and child health services, screening for high-risk pregnancies and making referral services for these conditions more accessible, particularly to the rural women and children, will be key to improving child survival in Zimbabwe.

12. The results from this study are expected to assist policy makers and programme managers in the child health sector to formulate appropriate strategies and interventions to improve the situation of under-five children in Zimbabwe. In particular, child health interventions should be expanded by means of health programmes such as the Integrated Management of Childhood Illness (IMCI).