household income and mother's education, to 0.16 in Table 4 which include these two important household-level covariates. This finding is consistent with our results in the gamma models. Furthermore, the z-score for π_1 (the probability that a family belongs to the first group) is reduced from 1.76 to 1.34 when family income and mother's education are added, an indication of the uncertainty about the two-group division. This finding also is consistent with our results in the gamma models.

DISCUSSION AND CONCLUSIONS

Estimates of Mortality Determinants and Their Inferences

A closer look at the case of child mortality using the general results described earlier explains the lack of change in parameter estimates. Equation (8), derived from equation (7) when a gamma distribution is assumed for the familial effects, gives the ratio of the observed hazards for a two-sample problem, where the relative risk for Sample 1 in the numerator is $e^{x\beta}$ and for Sample 2 in the denominator is 1:

$$R(t) = \frac{\lambda_1(t,e^{x\beta})}{\lambda_2(t,1)} = \frac{e^{x\beta} + \phi^{-1}\Lambda_0^*(t)e^{x\beta}}{1 + \phi^{-1}\Lambda_0^*(t)e^{x\beta}}, \quad (8)$$

where $\Lambda^*_0(t)$ is the cumulative hazard function corresponding to $\lambda_0^*(t)$ (the unmixed hazard, as defined in equation (5)), in equation (7). This simple expression (Equation (8)) provides a convenient means of examining the influences of unobserved mortality determinants on parameter estimates.

If ϕ^{-1}, the gamma variance, is 0, implying that all families have the same risk, $R(t)$ will be the constant $e^{x\beta}$ and the model will be a standard proportional hazards model. A larger ϕ^{-1} will imply a greater departure from proportionality. This departure depends heavily on the size of the baseline hazard as well as on unobserved determinants of mortality. When $\lambda_0^*(t)$ approaches 0, the hazards of the groups approach proportionality. On the other hand, a higher level of baseline hazards signifies greater deviation from proportionality. To explain the lack of change in parameter estimates, we must show that the size of the unobserved mortality determinants and baseline hazards is not large enough to generate a substantial change.

In Table 5 we report the results from a simulation. It contains the ratios of the observed hazard in a group with $e^{x\beta}=2$ to that in a reference group with $e^{x\beta}=1$, for two levels of baseline hazards at Months 1 through 20. The results in Columns 2 and 3 are calculated using equation (8). The levels of baseline hazards are set at 0.004 and 0.377, the respective estimated baseline hazards for Months 24 through 59 and for the first month from the multivariate model in Table 3. The variance of gamma frailty (0.22), used in both Columns 2 and 3, also comes from the model. Even though we use the estimated parameters, the exercise is hypothetical. In both cases (Columns 2 and 3), we assume, contrary to fact, that the baseline hazard remains constant from birth to Month 20. We emphasize the large difference in the level of baseline hazard between Columns 2 and 3. When baseline hazards are high (Column 3), the hazards for the two groups clearly are not proportional and the ratio of hazards converges quickly towards unity, so the parameter β is reduced rapidly towards 0. With a low baseline hazard and the same frailty, however, the hazards for the two groups are largely proportional over the 20 months we have considered. The simulation shows that moderate frailty has a dramatic impact on the proportionality of hazards only when the baseline hazard is high. This finding suggests that the biases in estimates of mortality determinants from unobserved heterogeneity are likely to be small so long as

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Table 5. Illustrative Calculations Showing the Ratios R(t) of Observed Hazards in a Group with $e^{x\beta} = 2$ to a Reference Group with $e^{x\beta} = 1$[a]

Duration in months	Ratio of Hazards with Low Baseline Hazard ($\lambda = 0.004$)	Ratio of Hazards with High Baseline Hazard ($\lambda = 0.377$)
1	1.99	1.83
2	1.99	1.71
3	1.99	1.63
4	1.99	1.56
5	1.99	1.50
6	1.98	1.46
7	1.98	1.42
8	1.98	1.38
9	1.98	1.36
10	1.98	1.33
11	1.97	1.31
12	1.97	1.29
13	1.97	1.28
14	1.97	1.26
15	1.97	1.25
16	1.96	1.24
17	1.96	1.23
18	1.96	1.22
19	1.96	1.21
20	1.96	1.20

[a] For different levels of baseline hazards held constant from Month 1 through Month 20, with $\phi^{-1} = 0.22$.

neither the unobserved familial effect nor the baseline hazard is much larger than what we found in the Guatemalan data.

Standard errors are a different story. Although most of the deflations in the values of standard errors that we found are moderate, still they could result in dramatically different interpretations of parameter estimates when the z-ratios estimated by the standard model are just below the usual level of significance. The z-ratio for birth order is reduced from 1.85 to 1.61 in Table 3 and from 1.64 to 1.44 in Table 4 by correction for dependence. The z-ratios for previous birth intervals of 24–35 months and 36 or more months in Table 4 are reduced respectively from 1.72 to 1.55 and from 1.99 to 1.89. Reductions on a comparable scale may well cause a researcher to draw a different inference on a parameter estimate. Besides, there is no guarantee that the inflation in z-ratios will not be considerably larger in another analysis.

Parental Competence and Shared Genetic Effects

When the variance of the unobserved familial effects, ϕ^{-1}, and its associated z-ratio, are estimated respectively to be 0.14 and 1.16, we have suggested that the estimated familial effects *net* of household socioeconomic status are relatively unimportant, at least in this Guatemalan data set. We interpret 0.14 as the sum of the additive genetic effects shared among siblings (50% of the total additive genetic factors), a small portion of interaction

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effects among genes, effects of parental competence, and other household effects that are not measured by household income and mother's education.

This interpretation implies that the effect of parental competence cannot be larger than 0.14 in this Guatemalan population and probably is much smaller. This conclusion does not strongly support Das Gupta's (1990) argument that parental competence is a major source of variation independent of household income and mother's education. Even so, we caution against generalizing our results to other countries and other circumstances. Child-rearing practices in Guatemala may be very different from those in India. The effect of parental competence also may be attenuated by the fact that the births in a family in the Guatemalan data set can extend over a 15-year period.

Similarly, if the unobserved nonsocioeconomic effects at the household level (including parental competence) are nonnegligible, the genetic effect within this total should be much smaller than 0.14. Thus we expect the noninteractive genetic effect shared among siblings to be fairly small. We also expect the total noninteractive genetic effect, which is twice the size of the shared effect, to be quite moderate. More generally, we may ask what conclusions we can draw about the total effect (both additive and interactive) of genetic factors. This total effect should be fairly moderate unless the interactive effects dominate the additive effects. Although almost nothing is known about the interactive genetic effects on child mortality, behavioral geneticists have found very few such effects on behavioral measures for which data are available (Plomin and Daniels 1987; Plomin et al. 1980, p. 224).

Why do shared genetic factors, or even a combination of shared and nonshared genetic factors, seem to play a relatively minor role in child survival in a developing country? One explanation lies in the causes of early human mortality. Most such mortality in a developing country seems to be related to environmental factors. Genetic factors unfavorable to early survival have been kept low by natural selection because those who die young do not have a chance to pass on their unfavorable genes. During the period 1985–1990, the infant mortality rate was still as high as 122 per 1,000 in Ethiopia, 138 in Malawi, and 162 in Afghanistan, while in many developed countries, such as Japan, Denmark, and Sweden, infant mortality was as low as about 6 per 1,000 (United Nations 1991). Most likely the two groups of countries differ in the amount of care they can offer to their children rather than in the genetic makeup their children inherit.

The statistical model used in this analysis assumes independence between genetic and environmental factors, but do familial genetic factors and parental competence coexist and compensate for each other? More specifically, parents may recognize an unfavorable genetic inheritance in their children, and accordingly may make extra efforts to keep them healthy, thus counterbalancing the genetic disadvantages. If this is the case, the total familial effects will not be the sum of genetic and environmental factors, and the interpretation of the total effects as the upper bound of the shared additive genetic factors will not be guaranteed.

Evidence from aggregate statistics, however, seems to be inconsistent with this scenario. Although infant mortality in the United States has been reduced dramatically since 1940, infant deaths related to genetic disorders have not declined (Crandall 1976). Recall that the percentage of infant deaths due to genetic disorders or malformations in the United States (40%) is much higher than that in Bangladesh (12%). It seems that infant deaths due to genetic disorders cannot be averted easily even by the huge improvements in social and economic conditions that have occurred in Western countries in recent decades, or at least not as easily as infant deaths due to environmental causes. Himsworth (1984) made similar observations on the decline in child mortality in Britain in this century. Although some counterbalance between genetic effects and parental competence is always possible, the interpretation of the total effect as the upper bound of the familial genetic factors should hold true unless the counterbalance takes place on a massive scale. From a practical point of

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view, it is perhaps less important to know whether and how much genetic factors are moderated by parental competence than to know that family genetic factors are of very limited significance to early child survival with or without assistance from parental efforts.

Again we caution against generalizing the results of this analysis to other developing countries. Quite different familial genetic mechanisms may be at work elsewhere. The prevalence of consanguineous marriage, for example, differs substantially from country to country. We mentioned earlier that our child mortality data were collected in six Guatemalan Latino communities. Although we do not know the prevalence of consanguinity, we know that extended families are less important for Latinos than for Indians in Guatemala. Thus the proportion of consanguineous marriages among Guatemalan Latinos may well be considerably lower than that among peoples in other parts of the world. The proportion of consanguineous marriage is as high as 60% in Saudi Arabia (Swailem et al. 1988) and 47% in South India (Rao and Inbaraj 1979). Familial genetic factors in parts of the world with high prevalences of consanguineous marriage may play a more important role than in locales with low consanguinity.

This work has implications for a wide variety of social and demographic phenomena. Familial environment can be important for outcomes such as fertility behavior, contraceptive practice, educational attainment, and criminality. The difficulty with family influences is that familial effects other than general socioeconomic status are very difficult to observe. The approach adopted here addresses this difficulty by taking advantage of the fact that siblings must share at least a significant portion of familial effects. As a result, the importance of those effects can be assessed in addition to basic household socioeconomic characteristics.

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