SOCIO-ECONOMIC STATUS AND CLUSTERING OF CHILD DEATHS IN RURAL PUNJAB

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ABSTRACT

This paper explores alternate models for testing for child death clustering, and tests for it in different socio-economic and educational strata, using data from Punjab. Significant evidence of clustering is found only amongst the lowest strata, which show considerable positive and negative deviance in child survival. As maternal education and socio-economic status rise, the negative deviance in factors related to child mortality appear to be removed. The extent of clustering is quite high in this population, indicating substantial potential for reducing child mortality by focussing services on high-risk households. Family building factors such as short birth intervals and high parity births do not raise child mortality, when families are disaggregated by level of risk. They seem to be an *effect* rather than a *cause* of clustering of child deaths.

RÉSUMÉ

Ce texte explore des modèles alternatifs pour tester la proximité de la mortalité infantile (clustering), tout en tenant compte des niveaux socio-économiques et éducationnels de la population. Nous utilisons des données du Penjab (Inde). Des indications significatives de proximité (clustering) ne se présentent que parmi les couches inférieures de la population qui montrent d'importantes déviations, positives et négatives, de mortalité infantile. A mesure que l'éducation maternelle et le statut socio-économique s'améliorent, les déviations négatives par rapport aux facteurs reliés à la mortalité infantile semblent disparaitre. Le niveau de proximité (clustering) est assez élevé dans la population, ce qui indique qu'il existe un potentiel important de réduction de la mortalité infantile en orientant les services vers les ménages à risques élevés. Les facteurs qui contribuent à la formation de la famille tels les courts intervalles entre naissances ou le nombre d'enfants déjà nés n'accroissent pas la mortalité des enfants, une fois que les familles sont décomposées selon le niveau de risque. Il semble qu'il s'agisse d'un *effet* plutot que d'une *cause* de proximité (clustering).

RESUMEN

Este documento estudia los modelos alternativos de análisis de la proximidad (cluster) en la mortalidad infantil realizando pruebas en distintos estratos socio-económicos y educacionales, usando datos del Punjab. La evidencia más significativa de proximidad (cluster) se encuentra solo entre los estratos más bajos, los cuales muestran considerables desviaciones, tanto positivas como negativas, en la supervivencia infantil. A medida que aumentan, tanto la educación maternal como el estatus socio-económico, la desviación negativa de los factores relativos a la mortalidad infantil desaparece. La extensión de la proximidad (cluster) es bastante alta en dicha población, indicando un considerable potencial para reducir la mortalidad infantil concentrando los servicios en las familias de alto riesgo. Los factores que intervienen en la construcción de la familia, tales como los cortos intérvalos de tiempo entre cada nacimiento o la cantidad (paridad, igualdad) de ellos, no aumentan la mortalidad infantil, cuando las familias se disgregan según el grado de riesgo. Parece ser más un *efecto* que una *causa* de proximidad (cluster) de mortalidad infantil.

RESUM

Aquest document analitza els models alternatius d'anàlisi de la proximitat (cluster) a la mortalitat infantil realitzant proves a diferents nivells socioeconòmics i educacionals, utilitzant dades del Punjab. L'evidència més significativa de proximitat (cluster) es troba només entre els nivells més baixos, els quals mostren considerables desviacions, ja sigui positives com negatives, en la supervivència infantil. A mesura que l'educació i el nivell socioeconòmic aumenten la desviació negativa dels factors relatius a la mortalitat infantil desapareix. L'abast de la proximitat (cluster) és bastant alt en l'esmentada població, i indica un considerable potencial per a reduir la mortalitat infantil concentrant els serveis en les famílies d'alt risc. Els factors que intervenen en la construcció de la família, com són els intervals curts de temps entre cada naixement o la quantitat dels mateixos, no fan aumentar la mortalitat infantil, quan les famílies es disgreguen segons el nivell de risc. Sembla més un *efecte* que una *causa* de proximitat (cluster) de mortalitat infantil.

SOCIO-ECONOMIC STATUS AND CLUSTERING OF CHILD DEATHS IN RURAL PUNJAB[1]

I INTRODUCTION

Several studies of child mortality in recent years have focussed on the question of death clustering, or inter-family heterogeneity in child mortality (Das Gupta 1990; Curtis et al.1993; Guo 1993; Ronsmans 1993; Zaba and David 1994). This hypothesis is consistent with indications from analysis of World Fertility Survey data (Meegama 1980; Hobcraft et al.1985), as well as from other studies in various settings[2].

This question can have considerable implications for reproductive health and child survival programs. In India, as in many other countries, health services are made available to people largely in response to their demand. If child deaths tend to be heavily concentrated amongst some families, this would suggest that substantial improvements in child mortality could be made by adopting the more cost-effective technique of focussing health care resources more specifically on the subset of families who are at high risk of losing children.

Given the academic and policy interest of the question of familial risk, it is important to examine the matter carefully. There are several possible reasons why such a finding can be an artifact of the method of data analysis. For example, we should expect to find clustering when analyzing data derived from an agglomeration of study sites, as is often the case with country-level data. The people in these study sites may be subject to very different conditions of life which can affect their mortality levels, such as access to health services, nutrition, and exposure to disease. The same consideration applies to a localized study conducted in a setting where mortality levels have changed over time: here, aggregating different age-groups of women will have an effect similar to that of aggregating samples from different regions.

Another obvious reason for clustering within a population is that families of different socio-economic standing are subject to different mortality regimes. For example, Guo (1993) found that most of the clustering in his data from Guatemala was accounted for by the household's economic status and mother's education. As he indicates, finding clustering due to such well-known factors (Caldwell 1979; Ware 1984; Cleland and van Ginneken 1988) does not add to our existing understanding of child mortality. Any group of families will always show clustering, which will disappear if we control for all the relevant factors making for child mortality. Finding clustering is of analytical interest only if this points towards hitherto unanalysed factors in child mortality.

This paper explores alternate models for testing for clustering, and tests for it in different strata of socio-economic status, mother's education and child's age at death, using data from Punjab. The child's gender is also added because it is known that in Punjab, girls in families with more than one daughter suffer selective discrimination and excess mortality (Das Gupta 1987), and this could well be repeated for more than one girl in a family. Significant evidence of clustering is found only amongst the lowest socio-economic and educational strata. The relationship between familial risk and family building factors such as short birth intervals and high parity births is also explored, as these are well known to be associated with elevated risk of child mortality. This association appears to be an artifact of selection of high-risk mothers into short birth intervals and higher parities, following child loss. Thus short birth intervals and high parity births seem to be an *effect* rather than a *cause* of clustering of child deaths.

II DATA SOURCES AND THE STUDY AREA

The data used in this paper were collected in a study of eleven villages in Ludhiana District, Punjab[3]. The survey data used in the present analysis were collected in 1984, and draw on (1) pregnancy histories of 3630 ever-married women aged 15-59 in the study population, and (2) a household census, with information on household socio-economic status and household structure. For the present analysis, interviews were carried out with the mothers and other members of households with multiple child deaths, as well as "matched" families of similar socio-economic status with no child deaths, focussing on the circumstances of family life and childbearing.

The study area and its region have been undergoing rapid improvement in child survival in recent decades. In the State of Punjab as a whole, the infant mortality rate fell from around 129 per thousand livebirths in 1972 to 66 in 1984 (Sample Registration Scheme 1985:15). In the study villages, it fell from about 156 per thousand livebirths in 1957-59 (Wyon and Gordon 1971:181) to 62 in 1979-84. Fertility has also been declining. In Ludhiana District, to which these study villages belong, the Total Fertility Rate has fallen from 5.2 in 1955 to 3.7 in 1980 (Das Gupta 1994). In the study villages in 1984, the Total Fertility Rate was 3.26.

The study area is quite homogeneous with regard to the external environment of health. The villages are in three groups, located between the towns of Khanna and Ludhiana. Both these towns as well as other nearby towns offer a range of public and private health services. The villagers can reach these easily because all the villages have all-weather roads and regular bus services to the urban centers. Access to Primary Health Centres and sub health centres is also quite homogeneous: each village has one either inside the village or in a neighbouring village no further than two miles away. The sanitary conditions are similar in all the villages, and the drinking water is almost entirely taken from handpumps, drawing on the same underground water sources. This homogeneity of the external environment of health is important from the point of view of studying death clustering, as most of the differences observed can reasonably be assumed to be caused by factors pertaining to the household, rather than to the external environment.

While the villages are quite homogeneous in terms of their environment of risk, there have been substantial changes in this environment over time, as evidenced by the decline in child mortality. Under these circumstances, aggregating age-groups of mothers would tend to artificially exaggerate the heterogeneity found between mothers. For this reason, the analysis has been restricted to a small age-range of women, which helps to ensure that the children were subject to a fairly homogeneous external environment of risk.

The age-group of women selected for analysis here is those aged 40-49. There are 674 women of this age-group in the sample, and their children experienced a child mortality rate of 126.7 per thousand livebirths. The advantage of this age-group is threefold. Firstly, given that fertility beyond age 35 is very low, the children of these women have almost all been subject to the full five years of exposure to child mortality. The children of the younger women are more mixed in duration of exposure. Secondly, these women have completed their childbearing, which means that they are homogeneous in terms of stage of family building. This is especially important for the negative binomial test for clustering used here. Thirdly, the children of these older women were subject to higher levels of child mortality than those of younger women. As child survival rates improve, multiple child loss becomes a rare phenomenon, and the question of death clustering becomes redundant, although there can be variation in health[4]. Where the analysis is less affected by mortality decline, larger samples are obtained by using wider age-groups of women. For example, women aged 35-59 are used for the cross-tabulations of mortality by birth interval and by birth order, and women aged 40-59 are used for the regression analysis (with a control for age).

An earlier analysis of the determinants of child mortality in this population found that children who had dead siblings (born before or after the index child) had a significantly higher probability of dying, even after controlling for a wide range of socio-economic, biological and behavioural factors (Das Gupta 1990). This looked at the risk of individual children. The present analysis focusses more explicitly on *familial risk*, where the unit of analysis is the whole sibling-set.

Table 1 gives some basic information on child mortality by socio-economic status and mother's education, for women aged 40-49. As expected, child mortality is inversely related to socio-economic status.

III TESTS FOR CLUSTERING

Death clustering means that there is greater heterogeneity in the distribution of child deaths than would be expected, if deaths were randomly distributed. The question then becomes one of which model to use to obtain a benchmark model of the random distribution of deaths. The binomial and the negative binomial models are used here, and it is argued that their validity must be assessed in the context of the particular population studied.

1. The binomial model

An obvious candidate for generating the expected distribution of deaths is the binomial model, which estimates the distribution of failures to be expected for a given number of trials and a given probability of failure. This model has been used by others to examine clustering in child deaths (Ronsmans 1993; Zaba and David 1994). Applied to child mortality, it estimates the expected distribution of deaths, for a given number of livebirths who are subject to a given probability of child mortality. For a large sample, this distribution should (almost) exactly coincide with the observed one, provided the model is correct.

To use the model as a benchmark, it is appropriate to posit the same probability of failure for all families within the group being studied. That is, the underlying mortality level used is the same for all the families within the group, since the null hypothesis is that all their births are subject to the same probability of dying. The expected distribution of deaths (= the number of failures) is calculated separately for each parity (= the number of trials). This expected distribution is weighted by the number of families of that parity. Then the failures are aggregated across parities to obtain an expected distribution of deaths, to be compared with the observed distribution of deaths for the group of families being studied.

The binomial model presumes that fertility decisions are made, by and large, without a clear notion of the outcomes of such decisions. This ex-ante "hoarding" approach is most likely to be found where mortality rates are generally high (not only during infancy), and/or the desired number of surviving children is high (see Preston 1978).

The hypothesized difference between the observed and the expected distributions, if death clustering exists, is that there will be more women in the observed than in the expected distributions who have no child deaths, and who have several child deaths: that is, a comparison of the two distributions will show "fat tails" at both ends.

2. The negative binomial model

In contrast to the "hoarding" approach, it may be the case that childbearing is targeted towards a certain number of surviving children. In the extreme variant of the targetting model, people try to replace dead children until they reach their targeted number of surviving children, and then stop childbearing. In this case, it is more appropriate to use the negative binomial

model to generate a random distribution of deaths.

The negative binomial model estimates the number of trials necessary in order to obtain a given number of successes, under conditions of a given probability of failure. In this application, it estimates the number of births that would be required in order to reach a particular targeted number of surviving children, given the existing probability of child death. Thus this model tests the difference between the observed distribution of deaths and that which should obtain under conditions of pure targeting towards a given number of surviving children.

Again, to obtain a benchmark, the probability of dying is kept constant for all the families in a given group, that is, it is assumed that the underlying mortality level is the same for all the families within the group. The expected distribution of deaths is calculated separately for each "surviving parity" group. Then the deaths are aggregated across the "surviving parities", to obtain an expected distribution of deaths for all families within the group.

The negative binomial model is a stringent test for clustering. It assumes that the number of surviving children a family has *is* the number it was targeting towards. This model can only be tried on families which have completed their childbearing. Amongst younger age-groups of women, many may not yet have completed their childbearing, and therefore their number of surviving children cannot be assumed to represent their target.

Just as in the case of the binomial distribution, the negative binomial relies on an extreme model. In reality, a combination of the binomal and the negative binomials might serve as a more accurate benchmark, the specific weights accorded to each varying according to the population concerned.

3. Choosing a model

To find out which model more accurately fits the population studied, we have to determine the extent to which childbearing is targeted towards reaching a certain number of surviving children in the study area. The fertility decline in this area increases the likelihood that people are successfully targeting towards a certain number of surviving children. Evidence of trying to shape the size of completed families emerges from previous analyses in the study area (Das Gupta 1987; 1994), as well as from data from a cluster of nearby villages (Faruqee and Sarma 1983:164). Although older women had higher fertility because they were childbearing at an earlier stage of the fertility transition, it is possible that they too may have been successful targeters in childbearing.

The question of targeting needs to be examined separately for different subgroups of the population, as they may differ in their fertility goals. Indeed, we know that there are substantial fertility differentials amongst subgroups of this population (Das Gupta 1994; Wyon and Gordon 1971), which gives us some basis on which to explore differences in targeting of childbearing. Multivariate analysis of fertility in this study area (Das Gupta 1994) indicates that women's education and household socio-economic status are the most important criteria for differentiating groups in terms of fertility behaviour.

Two tests are used to look for evidence of successful targeting amongst these women:

Test 1

A good test for targeting is to compare the proportion of children dead amongst the last births in families with the proportion dead out of all the births. Under the assumption of targeting, where people replace dead children and stop childbearing after reaching a given number of surviving children, we should expect a very high proportion of survivors amongst the lastborn children, relative to the mortality of all births. (There would, of course, be some cases in which the last child died after the mother was too old or for other reasons unable to

replace the child.)

Looking at the overall sample of women aged 40-49, the child mortality rate of the lastborn children is somewhat lower than the average (Table 2), though the difference is not sharp enough to constitute strong evidence of targeting[5]. Sharper evidence of targeting is revealed when we look at landownership and education groups separately. Clearly the landowners and the educated women were either more target-oriented in childbearing, or were more successful in achieving their targets, than the landless and the uneducated women. This is consistent with the fact that the landowners and the educated women are more advanced in their fertility decline than the others and their mortality rates are also lower (Table 1), making their targets lower and easier to meet.

Test 2

Another way of looking for evidence of targeting is to compare the observed distributions of the number of surviving children with the expected (random) distribution of surviving children, which is based on an equal probability of losing children. Here the binomial model is used to determine the number of surviving children, under the assumption that fertility is <u>not</u> targeted towards a certain number of surviving children. Thus if women did indeed target their fertility, we would expect to find a greater heaping in some surviving parity groups in the observed than in the expected distributions of the numbers of surviving children.

This tests for targeting, quite independently of whether there is clustering in deaths or not. Put another way, it should not be assumed that the binomial and observed distributions obtained here for the number of surviving children are just the converse of those obtained for the distributions of dead children. This would be the case only if the total numbers of children born were very similar across all families. Since this is not so, this test is, in principle, completely independent of the counterpart test used for clustering, which depends on the distribution of dead children.

Given the strong son preference in this society, and the evidence for aiming for a given number of sons rather than children (Das Gupta 1994), it is necessary also to compare the observed and expected distributions of surviving *sons*. Once again, we look separately at landless and landowning families.

The observed and expected distributions of the numbers of surviving children and sons are shown in Graphs 1 and 2. Graph 1 shows a little extra concentration at parity 4 in the observed distribution of surviving children, but overall the observed and expected distributions correspond fairly closely. Breaking this down by landowning status, we can see that some concentration is evident only amongst the landowners, not the landless. This pattern is repeated when we look at surviving sons (Graph 2): there is somewhat greater evidence of concentration (at parity 2) in the observed as compared to the expected distributions, and this is evidenced only amongst the landowners, not the landless. Thus it seems that the landowners show some evidence of successful targeting (more around the number of surviving sons than the number of surviving children), while the landless do not.

This is not to say that the landless and uneducated did not have targets regarding their family size. The speed with which their fertility has declined in recent years suggests that they had targets and have been revising them downwards. The most plausible interpretation is that these older women <u>had</u> targets, but they were too high to make it possible for the large majority to be successful in meeting their goals. Many of these older women wanted two or even three surviving sons. Under the high child mortality regimes prevailing for lower socio-economic groups at the time (Table 1), such high targets would result in a substantial proportion of women being unable to reach their targets during their fecundible years. This would result in a situation in which, though women had some targets in mind, the inability of many to attain these targets created patterns of childbearing which do not satisfy the criteria of targeting, and

are effectively indistinguishable from a non-targeting population.

The results of the two tests suggest that the binomial model is clearly the appropriate one to use for the lower socio-economic groups. For older women taken as a whole, the binomial model is again the more appropriate one to use. However it is less clear which the more appropriate model would be in the case of the higher socio-economic groups, namely the landowners and the educated mothers. These groups show more evidence of successful targeting than the others, as they are further advanced in the fertility transition as well as the mortality transition, thus being better placed to achieve their desired family size. The ideal model would be one which allows for imperfect targeting, i.e. which lies somewhere between the binomial and the negative binomial, to generate the expected distribution of deaths. For the present, we will use the binomial model for all groups, and the negative binomial only for the higher socio-economic groups.

IV RESULTS BASED ON THE BINOMIAL MODEL

Taking all the women aged 40-49 together, the pattern of the observed as compared with the expected (binomial) distributions has "fatter tails" at both ends (Table 3). This is what we would expect to find if there was indeed a clustering of deaths. There are families which experience fewer child deaths than would be expected on the basis of their numbers of births (positive deviants), and families which experience more child deaths than expected (negative deviants).

1. The extent of clustering

Table 3 shows that many more families have no deaths, or only one death, than the model predicts. In terms of proportions of families, these positive deviants are the largest category of deviants from the expected distribution. There is some convergence between the observed and expected figures amongst families with two child deaths, although there are a few positive deviants here as well. The negative deviants are a very small proportion of families, and are concentrated amongst those with three or more deaths.

Although they are a small proportion of families, these negative deviants account for a large proportion of total child deaths. While only 3.71% more of all women fall in the category of three or more deaths than expected, 23% more of all child deaths fall in this category than expected. Thus we could say that 23% of all child deaths are attributable to belonging to a high-risk family, and that less than 4% of families fall in this high-risk category.

2. The effect of socio-economic status

To remove some of the effect of socio-economic differentials when testing for clustering, the sample of women was stratified by socio-economic level and each group tested separately for clustering. Of course, it would be useful to have a larger sample, which would permit stratification into narrower socio-economic groups. Each socio-economic stratum shows significant clustering (Table 4).

The lower socio-economic groups (poorer, landless) show greater divergence between the observed and expected figures than do the higher socio-economic groups, indicating that they experience greater clustering than the higher socio-economic groups. This suggests that while clustering is due to some hidden factor which is <u>not</u> simply socio-economic status, this hidden factor nevertheless appears to be positively correlated with socio-economic conditions. For example, improvements in socio-economic status may homogenize the domestic hygiene and health care practices of a household such that negative deviancy in risk-prone behaviours is reduced.

Landowning status seems to be a sharper indicator of socio-economic differentials than per capita income. There is less divergence between the observed and the expected figures for the landowners than for the rich. That is, stratifying by landownership removes more of the clustering between families. Landownership is therefore the main indicator of socio-economic status used in the subsequent analysis.

The greater cutting power of landownership could be simply because data on income is far less accurate than that on landholding. It could also be due to the fact that in this rural setting, the fact of owning land distinguishes people according to their permanent income (i.e. their expected income over time, as opposed to their current per capita income level). Even more importantly, whether or not people own land distinguishes them sharply in terms of social status, with concomitant differences in access to information, expectations, and ability to mobilize resources from the outside world.

3. The effect of the mother's education

The mother's education is a powerful factor influencing the clustering of child deaths (Table 4). When education groups are analyzed separately, it emerges that educated women do not differ significantly amongst themselves in their experience of child mortality. However, the children of uneducated women show significant clustering in mortality. As in the case of socio-economic status, this suggests that whatever hidden factors may underlie clustering in child mortality, they are removed by receiving education. Besides, educated women have both low fertility and low child mortality, which as discussed earlier minimizes the chances of having multiple child deaths. Even if they discriminate more heavily against female children because they are more advanced in fertility decline (Das Gupta 1987), their overall likelihood of having multiple deaths remains low.

Stratifying by two criteria of low status (landlessness and uneducated), the data continue to show significant clustering in child mortality, which confirms that it is amongst low socio-economic groups that we need to explore further, with larger and more detailed datasets, to understand the causes of this clustering.

4. The effect of the child's gender

Stratifying by the child's gender (Table 5), we find that boys as well as girls show significant clustering in child mortality. Examining the effect of gender in each socio-economic group, it appears that there continues to be significant clustering in the mortality of both boys and girls amongst the rich and the poor, as well as amongst the landless. Amongst the landowners, though, there is no significant clustering in boys' deaths. This suggests that the landowners may not experience clustering in child mortality beyond that caused by discrimination against girls, that is, their apparent clustering is an artifact of their own actions to ensure their desired sex compostion of children. Amongst the low socio-economic stratum, however, the clustering is not attributable simply to the fact that there is selective discrimination against daughters in some families in this society.

5. Clustering by age at death

For a closer look at the nature of child death clustering in this population, it is useful to see whether there is clustering during both the neonatal and postneonatal periods. This can provide clues to the reasons for clustering, because the biological and behavioural factors underlying the mortality in these two age groups are somewhat different.

There is significant clustering in mortality during both the neonatal and postneonatal (1-

59 months) period (Table 6)[6]. This is true not only amongst the total sample of women, but also amongst the lowest socio-economic stratum of landless uneducated women. However, the postneonatal period shows greater clustering (greater divergence between the observed and expected distributions) than the neonatal period, and postneonatal deaths account for a large part of total child deaths. This suggests that factors related to postneonatal mortality, which are typically related to childcare practices, may be more responsible for death clustering than biological factors, which predominate during the neonatal period.

V NEGATIVE BINOMIAL MODEL RESULTS

The tests of targeting suggested that the negative binomial model may be applicable to the landowners and the educated women in this sample. The results obtained by fitting this model to the data for women aged 40-49 are shown in Table 7. This shows no significant clustering in child mortality amongst the children of educated mothers. The landowners show significant clustering, but the variance between the observed and expected distributions is small. These results are similar to those from the binomial model, showing robustness for this sample of families under either set of assumptions regarding targeting.

Given the nature of the negative binomial model, the righthand tail is fatter than in the binomial model, so that the divergence between the observed and expected distributions is smaller than in the binomial model. Also, the negative binomial model does not accept the possibility of the target being nil, that is, women with no surviving children. Thus the negative binomial analysis excludes the few women who lost all their children[7], which in turn leads to some underestimation of clustering in child loss.

VI FAMILIAL RISK AND CHILD MORTALITY ASSOCIATED WITH SHORT BIRTH INTERVALS AND HIGH PARITY

1. Familial risk and birth intervals

One possible reason for finding death clustering in families may be that some mothers may have shorter birth intervals than others. Short birth intervals are well-known to be associated with higher mortality (Hobcraft et al. 1985). Differences between women in the pace of childbearing may, then, be the cause of much of their observed clustering in child mortality, and this is explored here.

Proneness to having short birth intervals may have behavioural or biological causes. Behavioural reasons could be that some mothers' pattern of family formation is less carefully structured to avoid short birth intervals. It can also be a purely biological effect, in that some of the women with early child losses will have short birth intervals because earlier cessation of breastfeeding results in earlier resumption of ovulation.

The results of this analysis are very interesting. As expected, the aggregate data show the familiar pattern of higher mortality with shorter preceding birth intervals (Table 8, last column). However, this relationship disappears when the data are disaggregated by the number of child deaths experienced by the family. Child mortality rises sharply with the family's overall experience of child loss, and is little affected by the length of the birth interval for any given risk group of families.

It seems that, at least in this population, the positive relationship between child mortality and length of birth interval is an artifact of the preponderance of short intervals amongst families which are subject to a high risk of child loss (Table 9). *Their proneness to child loss is unrelated to the length of the preceding birth interval.* Families with no dead

children contribute disproportionately to the births with long birth intervals, while those with multiple child deaths contribute disproportionately to the births with short birth intervals. Families with three or more child deaths are more than twice as likely to have birth intervals shorter than 18 months, than those with no child deaths. This is probably partly due to biological reasons following a child death (earlier cessation of breastfeeding resulting in earlier resumption of ovulation), and partly due to efforts to "replace" dead children in order to reach a certain number of surviving children.

Looking at the relationship between birth intervals and child survival from the perspective of death clustering throws an entirely new light on the causality involved in this relationship. It throws further light on the findings by Hobcraft et al.(1985: 375) from a cross-country analysis of pairs of births, that a child's risk of dying was greater if the previous child had died, even if the birth interval between these children was not a short one. Data from different settings (Ronsmans 1993; Curtis 1993) also indicate that the relationship between birth intervals and child mortality is strongly influenced by differences in familial risk.

2. *Concentration of excess mortality at higher birth orders*

Birth order is another family-building factor commonly found associated with mortality differentials. The typical pattern is that child mortality falls and then rises with increasing parity of the child. Some of the mechanisms whereby this could take place include competition for resources in crowded families, and the more biological effect of maternal depletion. This pattern is found for the aggregate sample of women in Khanna (Table 10).

However, analyzing the effect of parity for families of different levels of risk (Table 10), we find that *from the first parity onwards*, "high-risk" families experience far higher levels of child mortality than others. The probability of a child dying does not rise with birth order, but varies greatly according to the level of familial risk. In fact, in this sample, child mortality actually declines steadily with parity at any given level of familial risk. One reason for this could be that women are able to cease childbearing when their children survive, which means that their last births will reflect higher survival rates than their earlier births. Another possible reason could be that greater precautions are taken to avoid child loss as they reach higher parities, reflecting increasing anxiety to reach their desired number of surviving children. The possibility that there may also be biological factors at play needs to be investigated.

The divergence between the levels of child mortality between families of different "levels of risk" increases with parity. This may be partly because women in high-risk families are forced into ever higher parities in order to reach some acceptable number of surviving children, and their children continue to be exposed to the families' elevated levels of risk. By contrast, women in lower-risk families experience enough survivals to be able to stop childbearing.

The pattern of rising mortality with parity is created by the fact that high-risk families contribute disproportionately to the high-parity births (Table 11). There is a concentration of high-risk families amongst those reaching higher parities, since they are trying to reach a given number of surviving children. Those who reach their desired surviving family size stop childbearing, while their less fortunate counterparts go on to higher parities in an effort to reach their desired family size. The result is an apparent rise in mortality amongst high parity children (Table 10, last column), and a concentration of excess child mortality amongst higher-parity families (Table 12). Again, just as with birth intervals, the familiar direction of causality is reversed.

The selection into higher parities of women who experienced deaths amongst their early livebirths has been commented on by researchers working in varied settings, for example in the French village of Meulan (Lachiver 1969, quoted in Knodel 1978), rural Punjab in the 1950s

(Wyon and Gordon 1971) and presentday rural Senegal (Ronsmans 1993).

VII THE CORRELATES OF FAMILIAL RISK

In order to study the correlates of familial risk, an index of familial susceptibility to child death was constructed. This index is based on both the number of births as well as the number of deaths a family has, and not just the *ratio* of deaths to the number everborn. Thus, for example, a family losing two out of five livebirths is ranked as more susceptible to child loss than one who lost two out of eight livebirths[8]. More significantly, the measure distinguishes between families with the same ratio of loss but different parities, so for example a family experiencing a 100% loss at parity 3 is ranked as more susceptible than one with the same ratio, but a parity of 1.

One way to account for this is to compare the actual experience of a family with the probability of that occurrence, where the probability of death is calculated from group averages (Garenne 1992). Thus if a family has x deaths from y births, any inverse function of Probability (deaths $\geqslant$ x | y births) will suffice. To calibrate the degree of susceptibility between zero and infinity, the following particular form is used:
$S(x,y) \equiv -\ln$ [Probability (deaths $\geqslant$ x | y births)]
where S (x,y) means "susceptibility of a family with x deaths from y births", and where the probability is calculated using population averages.

The independent variables used are as follows:

1. The woman's age, used not as an explanatory variable, but as a control to avoid overestimation of the influence of these variables on familial risk. Without this control, some spurious correlation would be engendered by the fact that the circumstances of mortality and health care under which these children were born changed over time.

2. The household's per capita income, logged.

3. The household's socio-economic status and the parents' educational level (landownership, husband's occupation, woman's education, and her husband's education). These can influence the household members' access to information, effectiveness in using health services and other factors influencing their ability to care for their children.
   (i) Landownership is introduced as the dummy variable "Landowner", positive for households who own land.
   (ii) Husband's occupation is defined as a dummy, positive if he is a wage labourer (agricultural or non-agricultural). This occupation is associated with low income and low social status.
   (iii) Education is measured in single years of completed schooling.

4. Specific behaviours relating to health care utilization (Antenatal tetanus and BCG), domestic health care (Rehydrate diarrhoea) and domestic hygiene (Uses soap).
   (i) The variable on antenatal tetanus immunization is defined as a dummy variable which is positive if one or more pregnancies leading to a livebirth was protected by prenatal tetanus immunization.
   (ii) "BCG" is a dummy, defined as positive if one or more children were immunized with BCG vaccine. Defining this variable in terms of the whole set of livebirths minimizes the inherent problem that the vaccine is usually given within a few weeks of the child's birth, so that if a particular child dies during the neonatal period it is unlikely to have had a chance to receive the immunization. Even if some of a family's children die very early, the other children are available to have been immunized.
   (iii) "Rehydrate diarrhoea" is a dummy variable, defined as positive if the mother gave

her children plenty of water to drink if they had diarrhoea.

(iv) "Uses soap" is also a dummy variable, defined as positive if the mother always washes her hands with soap after defecating.

These are at best very crude proxies for any "hidden factors" relating to health care which might account for death clustering. Unfortunately, more detailed data on the specifics of health practices in the past were not collected.

Ordinary Least Square regressions were run on these variables, using the data for women aged 40-59. These women share with 40-49 year old women the fact that their childbearing is complete. The distortion which could be introduced by expanding the sample to include these women, namely that the age-group is so wide as to include childbearing under different levels of prevailing child mortality and different conditions of availability of health care, is minimized by introducing a control for age.

Two sets of regressions were run (Table 13). The first includes only the variables relating to age, socio-economic status and education. The second set adds to these variables those relating to health care behaviours, to see how the effect of the other variables change when these are introduced.

The results show that the socio-economic indicators which combine both social as well as economic status, such as landownership and occupational status, are significantly correlated with familial risk. Being a landowner is significantly negatively associated with familial risk. If the father is of low occupational status, a wage labourer, the family has a significantly higher level of risk. The purer measure of economic status (per capita income) has no significant independent relationship with susceptibility to losing children, after controlling for the other indicators of socio-economic and educational status included in this analysis.

Improved health care practices seem to be one of the mechanisms whereby people of higher education or socio-economic status achieve lower risk. This is suggested by the fact that the significance of landownership falls when the health care variables are introduced. This is especially true of the mother's education, which is significantly negatively associated with risk in the first regression, but ceases to be significant when the health care variables are introduced. The husband's educational level is not significantly associated with susceptibility to child mortality.

Turning to the health care variables, the use of immunization is strongly and significantly negatively related to risk. The two variables need to be interpreted separately, because they are different in nature. The use of prenatal tetanus immunization has a direct effect on child mortality by preventing deaths from neonatal tetanus, which was a major cause of mortality in this population (Wyon and Gordon 1971). This variable is also likely to be related indirectly to child mortality, in that women who obtained prenatal immunization are also likely to have received other forms of antenatal care. Such women may also have made better preparations for delivery and postnatal care than women who did not avail of antenatal immunization.

BCG immunization, on the other hand, provides limited protection against tuberculosis, which was in any case not a major cause of child mortality in this population (Wyon and Gordon 1971). Despite this lack of direct effect on child survival, there is a strong negative relationship between the use of BCG immunization and the likelihood of losing children. One possible explanation is that some other aspects of health care are positively correlated with using BCG. For example, such people may be better informed about child health care, and more active in using health services to ensure that their children remain healthy. Immunization is not thrust upon people: they have to make some effort to have it done. The level of commitment required to get children immunized with BCG is all the greater because this vaccine causes a large boil lasting for several weeks: few people would inflict this on their

babies if they were not strongly committed to using whatever methods are at hand to maintain their children's health. The domestic health care practices (the use of rehydration, the use of soap) have the expected direction of relationship with familial risk, but fall short of being significant at the 5% level.

In brief, these regressions indicate that (i) household health care practices are related to susceptibility to losing children, and (ii) susceptibility is affected by household social and economic status, rather than income level alone. It should be noted, though, that the R2 of the regressions in Table 13 are low. This may be partly due to the complexity of the dependent variable, that is the susceptibility to child loss. Another potential problem is the particular form of the susceptibility function. If the true relationship is not linear (as assumed in a linear regression), a low R2 may result.

Part of the explanation for the low R2 obtained here must also lie in the fact that what we are trying to study is by nature quite subtle and elusive. Many different factors or combinations of factors can lead to greater susceptibility to child loss, and families may vary widely as to which particular factors account for their experience. Moreover, the important factor may be one which underlies some of the variables but is inadequately captured by them, as would be the case if the important factor were the effectiveness in responding to a child's illness. It is intrinsically difficult to adequately capture the factors making for heterogeneity in risk of child loss. For example, in the case of domestic health care, there are many aspects of care which can lead to a child's death or survival. This could relate to any of a wide range of aspects of illness prevention and treatment, including hygiene, crowding, illness management and effectiveness in utilization of health care services. Even within a family, different children may have been lost for different reasons. This makes it a formidable task to identify and adequately measure the factors making for greater susceptibility to child mortality.

On the other hand, the direct tests of clustering (Tables 4-5) suggest that there is a correlation between the "hidden factors" underlying death clustering and well-known determinants of child mortality such as maternal education and socio-economic status. Positive and negative deviance from the model are greater among the lower socio-economic strata than among the higher ones, and are greatest amongst the lowest stratum of families which are both landless as well as having uneducated mothers (Table 4). As maternal education or socio-economic status rise, the negative deviance in the "hidden factors" appears to be removed. This could result, for example, from greater variability in health practices amongst lower socio-economic groups than amongst higher ones. For the higher socio-economic groups, the more risky practices may be removed by factors such as improved living conditions and access to information. Their practices are more homogeneously grouped around the lower-risk end of the spectrum, where a substantial section of the lower socio-economic groups also belong. Graph 3 is a hypothetical scatterplot, illustrating how the variance in the "hidden factors" may come down as mother's education or socio-economic status rises.

VIII QUALITATIVE INSIGHTS INTO HIGH-RISK HOUSEHOLDS

As discussed above, it is extremely difficult to collect data on the precise reasons for multiple child loss. This problem extends also to efforts to collect qualitative data on the circumstances of multiple child loss, as it is hard to frame an exhaustive set of questions on what factors led to the losses. In this study, qualitative data were collected by visiting some households which had experienced multiple losses, and having an open-ended conversation with the mother and subsequently others in the household. Similar interviews were carried out in households of similar socio-economic status which had not experienced child deaths. These interviews give some insight into the complexity of circumstances making for susceptibility to child loss, and some of them are summarized here. Some individuals who had proved to be well-informed and perceptive about conditions of village life were also asked if they had any

insights into the problem. In particular, a long discussion with the headman of one of the study villages proved especially helpful.

The headman thought about the households he knew who had suffered multiple child loss, and came up with the following observations relating to his understanding of the profile of high-risk households:

1. Families which are unconvinced of the benefits of modern health care. They do not familiarize themselves with the more effective forms of health care available, but depend more casually on "folk" medicine.

2. Families with cumulative stresses. To illustrate this, he described a family impoverished through the poor financial management of the father. He said that this not only impoverished them, but also demoralized them, and that they also managed other aspects of their lives badly. For example, they left some pesticide lying around carelessly, and one child swallowed some. The other villagers rallied around to provide transportation to the hospital and to pay for the medical costs, but it was too late and the child died. He attributed this family's other child deaths to their inability to obtain the right care at the right time.

3. Multiple deaths related to not wanting too many female children. This was especially interesting because families never admit this directly about themselves, even though others in the village often know about their actions.

Our interviews with the households broadly corroborated the headman's view of the association between the risk of multiple child loss and domestic management, while adding other perspectives on the household dynamics associated with this phenomenon. The following are some examples of the interviews:

1. A case in which the couple feel overwhelmed by their responsibilities. The husband has been unable to enter the new economic niches opened up in this region, which have transformed the financial situation of most people in these villages. He continues in the artisan work traditional to his caste, work which is now poorly paid relative to other occupations. He works long hours, and the wife feels both worried about him as well as unable to cope with the household on her own. Both husband and wife feel not only overwhelmed by, but also lacking in control over the circumstances of their lives. They do the best they can, on all fronts, but do not expect to always succeed in what they would like to achieve.

2. A case of a low-caste family, in which the parents seemed to be well-informed about the outside world, with good communication and cooperation between husband and wife. The mother was able to describe the circumstances of her children's illnesses and subsequent deaths with clarity, as well the efforts she and her husband made to seek treatment for the children. It was unclear from the interview what could have gone wrong, and why the couple had had multiple deaths. One possibility is that theirs was a case of "bad luck", and that they had experienced multiple loss through the workings of random chance.

3. A case of a highcaste family, in which the bereaved mother was trying to change her behaviour to avoid further child loss. In this joint family household, one woman had had multiple child deaths, while her two sisters-in-law's children were all alive. She and her sisters-in-law had tried to think of what might have contributed to her child loss, and how she could alter her behaviour to avoid this. One obvious precaution they had decided on was more careful use of antenatal care, followed by delivery in a clinic. It was noteworthy that they attributed the loss to alterable behaviours, rather than to

fate, sin or genetic disposition.

4. On one visit, we made a very illuminating mistake. In looking for a household with multiple deaths, we mistakenly entered one with the same name of head of household and caste but a different identification number. The courtyard was neatly swept, with the beds put away, and everything about the house looked clean and well-organized. Just as I was thinking how unlikely it seemed that this household had suffered multiple child deaths, my assistant came up looking profoundly embarrassed and said we had made a mistake, the woman had had no child deaths.

The woman cheerfully directed us to the correct household. Here the first sight was that of a filthy courtyard. The cattle were kept next to the cooking area, and flies were settling around the droppings and on the children with running noses who were sitting and playing on this ground. The mother mentioned that apart from the children who had died, two of those who were alive had had polio: she had not had them immunized, as most villagers had.

The headman's analysis was especially important because he was familiar with the households and the circumstances of their lives from before their tragedies befell them. Thus he could be surer of the fact that many of these households suffered cumulative stresses and/or had poor domestic management *before* losing children. In contemporary interviews, it is difficult to know whether the perceptible stress and demoralization is a result or a cause of the deaths. Households with multiple deaths commonly show considerable stress. This is reminiscent of the findings in Newcastle (Spence et al.1954) that children in families suffering from stress related to employment or illness seemed especially vulnerable to poor health.

It is important to clarify what is meant here by "poor management". It would be wrong to suggest that the parents are to be blamed for their children's deaths. "Poor management" can result from poor judgement or bad luck in husbandry. As the interviews show, this can result in various forms of stress and demoralization, culminating in tragedy. An important reason for "poor management" is the fact that the health services themselves are very difficult to negotiate. A bewildering plethora of forms of treatment face the potential patient, with different implications in terms of finances and the number of visits required. It is far from clear what is the most effective treatment available. Those who are lucky enough to place greater faith in the public health care system despite the delays and frustrations involved, or find a good private practitioner, are the more fortunate. Even then, it takes much confidence and skill to be effective in obtaining care from these health personnel. There are many reasons why people who are less fortunate or well-informed may select a poor source of health care or find it difficult to follow up on hastily-given instructions.

To improve these conditions, it is essential that the health services take a more active role in delivering health care to the population they serve, rather than leaving the onus on the client. People will always vary in their innate abilities, and it is up to the providers of care to tailor their provision of services to ensure that this does not result in differential mortality rates.

IX CONCLUSIONS

Significant clustering in child mortality was found only amongst the lower socio-economic and educational strata in this population. Most families in these strata succeeded in avoiding child loss despite their social and economic disadvantages, while a few were less fortunate. This heterogeneity may be due to greater variation in behaviours related to child survival than amongst the higher groups. Improvements in socio-economic status and in education appear not only to reduce child mortality substantially, but also to reduce whatever hidden factors cause the differences in the risk of losing children. Besides, education reduces fertility as well as mortality dramatically, minimizing the chances of experiencing multiple child deaths.

The extent of clustering is quite high. In the population as a whole, as well as amongst the lowest socio-economic stratum, 23% of child mortality can be attributed to being born into a high risk family (with 3 or more child deaths), while less than 4% of families fall into this category. Susceptibility to child mortality is correlated with health care practices, socio-economic status and mother's education. The multivariate analysis suggests that improved health care practices are a major route whereby child loss is averted by educated mothers in particular, and also by higher socio-economic groups, over and above the protection of better standards of living. This is consistent with the fact that these groups have greater exposure to information, and are better placed to obtain effective care from the health services.

This discussion does not in any way suggest that parents are somehow to be blamed for their losses. In any population, we should expect to find variation in peoples' abilities. Where health services are more aggressive in providing care, they succeed in reducing mortality despite this. By contrast, where health services require aggressive efforts by clients to receive services, there is far more scope for falling through the cracks. Thus, for example, death rates were very low in Newcastle in the late 1940s, although Spence et al.(1954) found considerable variation between parents in childrearing practices and in children's health status.

One implication of heterogeneity in child loss could be that the overall level of fertility in the population is raised. This would follow if people's perception of the need for insuring against child mortality were based on the experience of the higher-risk families, rather than on the average risk of the group or their own particular experience. The data indicate that, at least in this area, people respond more to their own experience of risk than that of others, a situation which is conducive to more rapid fertility decline.

In testing for clustering in child mortality, the binomial model has commonly been used. Here we point out that in populations in which families are targeting fairly successfully towards a certain number of surviving children, it is more appropriate to use the negative binomial model to derive the expected distribution of child deaths.

Some interesting results emerge when the relationship between birth interval length, parity, and child survival is analyzed from the perspective of families' proneness to child loss. The disaggregated data show no increase in child mortality with shorter birth intervals or higher parity. These relationships are found in the aggregate data because higher-risk families are more exposed to short birth intervals and more likely to reach higher parities in an effort to reach their desired family size. Thus short birth intervals and higher parity births do not appear to have an independent effect on raising the risk of child mortality: at least in this population, they seem to be an *effect* rather than a *cause* of greater familial susceptibility to child loss.

This finding is pertinent to population policies, as the rationale for extending family planning programmes is based partly on the fact that child survival is understood to be negatively affected by short birth intervals and higher parities. While wider birth spacing and smaller family size have many benefits for both mother and child, the belief that these will in

themselves improve child survival appears to be ill-founded.

Also from a policy perspective, the results indicate a considerable potential for reducing child mortality by focussing on high-risk households. Substantial reductions in child mortality can be achieved by using the simplest possible information for targeting families for extra attention. In this sample for example, if health workers had used the first death as a marker for targeting intervention, and were successful at preventing all subsequent deaths, they would have reduced child mortality by 66%. In fact, even if the health workers had focussed on families after they had had two deaths, they would have needed to focus on only 16% of the families, and potentially averted 35% of child deaths. Amongst the lowest socio-economic strata (landless and uneducated women), focussing on families after they had had two deaths could potentially have averted 46% of their child deaths.

To improve child survival, it is important to intervene in ways which will help high-risk families to avoid child loss. This will also go a long way towards reducing fertility. Women whose children survive stop childbearing at lower parities, while those who experience child losses are pushed into a much more stressful reproductive cycle of shorter birth intervals and high fertility, along with further child losses. This causes considerable stress for a woman, her children and the household as a whole. Intervening to help high-risk families will have a major impact on lowering both child mortality as well as fertility, and improving reproductive health.

TABLE 1 **RELATIVE RISK OF CHILD MORTALITY BY SOCIO-ECONOMIC STATUS AND MOTHER'S EDUCATION, WOMEN AGED 40-49**

	N	Mean parity	Proportion dead	Relative risk
By income level:				
1 (lowest)	65	5.66	0.1685	1.33 *
2	230	5.63	0.1291	1.02
3	289	4.78	0.1230	0.97
4 (highest)	90	3.54	0.0846	0.67 *
By landownership:				
Landless	363	5.65	0.1483	1.17 *
<4 hectares	188	4.24	0.1028	0.81
4+ hectares	123	4.19	0.0777	0.61 *
By woman's education:				
0 years	566	5.21	0.1315	1.04
1+ years	108	3.81	0.0922	0.73 *

* Significant at 5% level

TABLE 2 **CHILD MORTALITY RATE OF LAST BIRTHS AMONGST WOMEN AGED 40-49**

	Last births	All births	Last births/All births
All	69.3	126.7	54.70
Landless	112.5	148.3	75.86
Landowners	32.7	93.0	35.16
No education	77.3	131.5	58.78
1+ years of education	27.5	92.2	29.83

TABLE 3 **DISTRIBUTION OF CHILD DEATHS AMONGST WOMEN AGED 40-49, USING THE BINOMIAL MODEL**

		Number of deaths			
		0	1	2	3+
% of mothers:	Observed	62.54	21.53	9.74	6.19
	Expected	52.63	34.24	10.65	2.48
	O-E	9.91	-12.71	- 0.91	3.71
% of deaths:	Observed	-	33.95	30.70	35.35
	Expected	-	54.05	33.63	12.32
	O-E	-	-20.10	- 2.93	23.03

(chi-square = 170.2655 ; $p < .0000$)

TABLE 4 **DISTRIBUTION OF CHILD DEATHS BY SOCIO-ECONOMIC STATUS, WOMEN AGED 40-49, USING THE BINOMIAL MODEL**

		Number of child deaths			
		0	1	2	3+
By income:					
	poorer				
% of mothers:	Observed	57.81	22.47	12.88	6.85
	Expected	47.10	36.14	13.15	3.62
	O-E	10.71	-13.67	- 0.27	3.23
(chi-square = 119.6496 ; p < .0000)					
	richer				
% of mothers:	Observed	67.73	21.09	6.71	4.47
	Expected	59.90	31.18	7.60	1.31
	O-E	7.83	- 10.09	- 0.89	3.16
(chi-square = 71.6592 ; p < .0000)					
By landowning status:					
	landless				
% of mothers:	Observed	52.09	23.47	14.15	10.29
	Expected	40.00	37.43	16.72	5.85
	O-E	12.09	-13.96	- 2.57	4.44
(chi-square = 106.3513 ; p < .0000)					
	landowning				
% of mothers:	Observed	71.11	20.44	6.54	1.91
	Expected	66.16	27.82	5.34	0.68
	O-E	4.95	- 7.38	1.20	1.23
(chi-square = 124.7842 ; p < .0000)					
By mother's education:					
	Uneducated				
% of mothers:	Observed	59.75	23.02	10.19	7.03
	Expected	49.69	35.31	11.95	3.04
	O-E	10.06	-12.29	- 1.76	3.99
(chi-square = 120.6337 ; p < .0000)					
	Educated				
% of mothers:	Observed	77.06	13.76	7.34	1.83
	Expected	70.18	25.32	4.13	0.37
	O-E	6.88	-11.56	3.21	1.46
(chi-square = 10.10969 ; p < .2574)					
Uneducated as well as landless women:					
% of mothers:	Observed	51.59	23.32	13.43	11.66
	Expected	39.00	37.44	17.27	6.29
	O-E	12.59	-14.12	- 3.84	5.37
(chi-square = 96.05666 ; p < .0000, N= 283)					
% of mothers:	Observed	-	25.29	29.12	45.59
	Expected	-	40.55	37.41	22.04
	O-E	-	- 15.26	- 8.29	23.55

TABLE 5 DISTRIBUTION OF CHILD DEATHS BY CHILDREN'S GENDER AND SOCIO-ECONOMIC STATUS, WOMEN AGED 40-49, USING THE BINOMIAL MODEL

		Number of child deaths			
		0	1	2	3+
By gender:					
	boys				
% of mothers:	Observed	76.44	17.17	4.41	1.98
	Expected	71.61	24.48	3.56	0.35
	O-E	4.83	- 7.31	0.85	1.63
(chi-square = 122.9658 ; p < .0000)					
	girls				
% of mothers:	Observed	75.00	16.72	7.14	1.14
	Expected	70.29	25.34	3.93	0.44
	O-E	4.71	- 8.62	3.21	0.70
(chi-square = 44.30511 ; p < .0000)					
By income and gender:					
	Poorer boys				
% of mothers:	Observed	74.29	17.51	5.93	2.26
	Expected	68.59	26.41	4.49	0.51
	O-E	5.70	- 8.90	1.44	1.75
(chi-square = 32.90518 ; p < .0000)					
	Poorer girls				
% of mothers:	Observed	71.68	17.34	10.12	0.87
	Expected	66.28	27.97	5.09	0.66
	O-E	5.40	-10.63	5.03	0.21
(chi-square = 33.28618 ; p < .0000)					
	Richer boys				
% of mothers:	Observed	78.95	16.78	2.63	1.64
	Expected	75.30	22.01	2.53	0.16
	O-E	3.65	-5.23	0.10	1.48
(chi-square = 16.82888 ; p < .0319)					
	Richer girls				
% of mothers:	Observed	79.26	15.93	3.33	1.48
	Expected	75.63	21.59	2.56	0.22
	O-E	3.63	- 5.66	0.77	1.26
(chi-square = 14.72472 ; p < .0647)					
By landownership and gender:					
	Landless boys				
% of mothers:	Observed	69.31	20.13	6.60	3.96
	Expected	61.98	30.33	6.67	1.02
	O-E	7.33	-10.20	-0.07	2.94
(chi-square = 46.97307 ; p < .0000)					

	Landless girls				
% of mothers:	Observed	67.91	18.92	12.16	1. 01
	Expected	62.23	30.34	6.45	0.98
	O-E	5.68	-11.42	5.71	0.03

(chi-square = 29.56924 ; p < .0002)

	Landowning boys				
% of mothers:	Observed	82.54	14.65	2.54	0.28
	Expected	80.99	17.52	1.44	0.06
	O-E	1.55	- 2.87	1.10	0.22

(chi-square = 7.960239 ; p < .4373)

	Landowning girls				
% of mothers:	Observed	81.56	14.69	2.50	1.25
	Expected	78.53	19.38	1.97	0.13
	O-E	3.03	- 4.69	0.53	1.12

(chi-square = 21.36217 ; p < .0062)

TABLE 6 DISTRIBUTION OF CHILD DEATHS BY AGE AT DEATH AMONGST WOMEN AGED 40-49, USING THE BINOMIAL MODEL

		Number of deaths			
		0	1	2	3+
All women:					
	Neonatal deaths				
% of mothers:	Observed	82.74	13.13	2.80	1.33
	Expected	78.89	18.78	2.15	0.17
	O-E	3.85	-5.65	0.65	1.16
(chi-square = 44.04992 ; p < .0000)					
	Deaths at age 1-59 months				
% of mothers:	Observed	73.60	16.52	6.78	3.10
	Expected	66.55	27.32	5.38	0.75
	O-E	7.05	-10.80	1.40	2.35
(chi-square = 111.9410 ; p < .0000)					
Uneducated as well as landless women:					
	Neonatal deaths				
% of mothers:	Observed	80.57	14.13	3.18	2.12
	Expected	74.73	21.92	3.08	0.28
	O-E	5.84	- 7.79	0.10	1.84
(chi-square = 15.17030 ; p < .0339; N= 283)					
	Deaths at age 1-59 months				
% of mothers:	Observed	61.48	20.85	11.31	6.36
	Expected	51.93	34.50	10.99	2.58
	O-E	9.55	-13.65	0.32	3.78
(chi-square = 46.19459 ; p < .0000; N= 283)					

TABLE 7 DISTRIBUTION OF CHILD DEATHS AMONGST WOMEN AGED 40-49, USING THE NEGATIVE BINOMIAL MODEL

		Number of child deaths			
		0	1	2	3+
All women					
% of mothers:	Observed	59.41	22.67	10.37	7.56
	Expected	53.46	29.58	11.69	5.28
	O-E	5.95	- 6.91	- 1.32	2.28
(chi-square = 38.69504 ; p < .0000)					
Educated women:					
% of mothers:	Observed	77.06	13.76	7.34	1.83
	Expected	72.59	21.71	4.78	0.92
	O-E	4.47	- 7.95	2.56	0.91
(chi-square = 13.08236 ; p < .109)					
Landowning women:					
% of mothers:	Observed	71.31	20.49	6.28	1.91
	Expected	68.76	23.89	5.91	1.45
	O-E	2.55	- 3.40	0.37	0.46
(chi-square = 25.65723 ; p < .0012)					

TABLE 8 **CHILD MORTALITY RATE BY BIRTH INTERVAL AND FAMILIAL RISK women aged 35-59**

Length of preceding interval	Number of child deaths of the mother 0	1	2	3+	All
< 18 months	-	152	363	469	221
18-23 months	-	174	303	454	151
24+ months	-	151	292	467	119

TABLE 9 **DISTRIBUTION OF BIRTHS BY BIRTH INTERVAL AND FAMILIAL RISK women aged 35-59**

Length of preceding interval	Number of child deaths of the mother 0	1	2	3+	All
< 18 months	5.06	10.90	12.96	19.84	9.04
18-23 months	13.06	15.70	16.79	20.98	15.01
24+ months	81.89	73.40	70.25	59.18	75.94
All	100	100	100	100	100

TABLE 10 **CHILD MORTALITY RATE BY PARITY AND FAMILIAL RISK women aged 35-59**

Birth Order	Number of child deaths of the mother 0	1	2	3+	All
1	-	309	412	602	150
2	-	247	351	580	131
3	-	154	370	532	116
4	-	148	310	500	120
5-6	-	145	252	446	136
7+	-	88	180	322	151

TABLE 11 **DISTRIBUTION OF BIRTHS BY PARITY AND FAMILIAL RISK women aged 35-59**

Birth Order	Number of child deaths of the mother 0	1	2	3+	All
1	21.66	17.20	14.68	11.68	18.64
2	21.80	17.36	14.78	12.70	18.87
3	19.57	16.79	14.78	12.59	17.55
4	15.31	16.01	13.92	12.70	15.01
5-6	16.54	22.06	24.76	22.90	19.58
7+	5.13	10.59	17.08	27.44	10.35

TABLE 12 **DISTRIBUTION OF BIRTHS AND CHILD DEATHS, BY PARITY OF MOTHER, WOMEN AGED 40-49**

Parity	% of births (=% of expected deaths)*		% of deaths		% births/%deaths	
1	0.35		0.23		1.52	
2	3.56		1.88		1.89	
3	7.83		3.05		2.57	
4	17.21		10.56		1.63	
5	17.21		15.02		1.15	
6	21.00		20.42		1.03	
7	12.25		14.55		0.84	
8	10.68		18.54		0.58	
9	6.14	20.58	8.69	34.27	0.71	0.60
10	2.37		4.69		0.51	
11+	1.39		2.35		0.59	

* Note that the % of births is equal to the % of expected deaths in the binomial model, which assumes that each birth is subject to the same probability of dying.

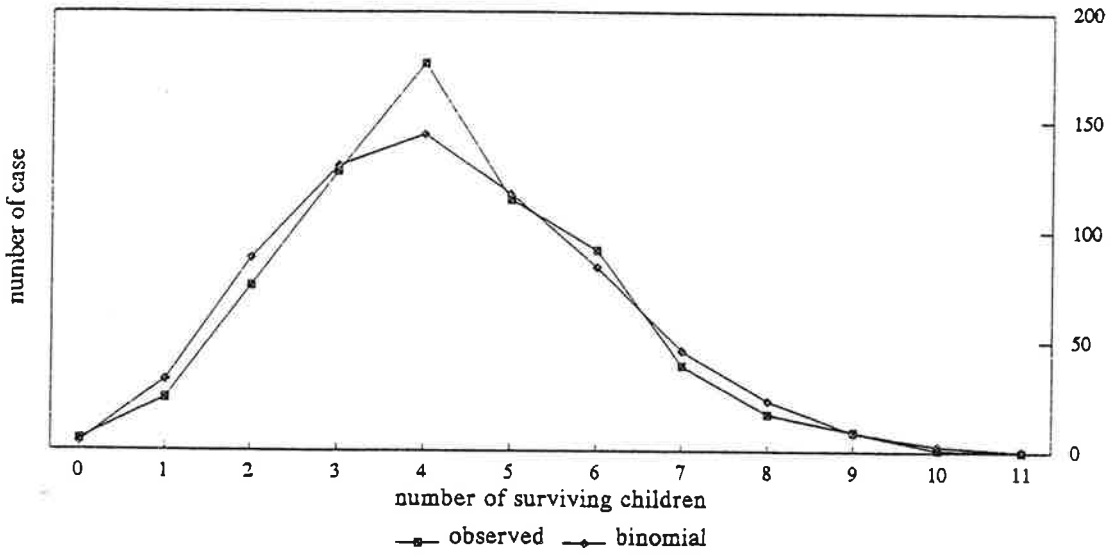
TABLE 13 REGRESSION ANALYSIS OF THE CORRELATES OF SUSCEPTIBILITY TO CHILD LOSS, WOMEN AGED 40-59

	Model 1		*Model 2*	
Explanatory Variable	Coefficient	t-value	Coefficient	t-value
Intercept	0.486774	0.517	1.555525	1.672
Woman's age	0.037115	3.527**	0.021508	2.106*
Socio-economic status				
Per capita income	-0.130426	-1.246	-0.119261	- 1.180
Landowner	-0.422300	-2.642**	-0.311130	- 2.023*
Husband wage labourer	0.623463	3.584**	0.530219	3.172**
Education				
Woman's education	-0.058862	-2.134*	-0.026160	-0.974
Husband's education	0.002948	1.811	0.002291	1.466
Health care				
Antenatal tetanus immunization			-0.088322	- 1.903*
BCG			-1.176397	- 9.696**
Rehydrate if diarrhoea			-0.199019	- 1.643
Uses soap			-0.161488	- 1.412
R2	0.0806		0.1586	
F value	15.612		20.069	

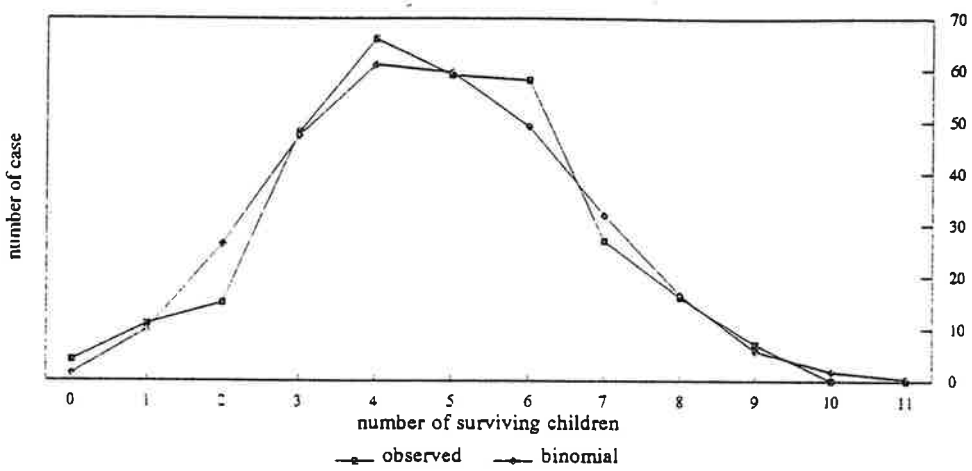
* Significant at 5% level, ** significant at 1% level

GRAPH 1 DISTRIBUTION OF SURVIVING CHILDREN OF WOMEN AGED 40-49

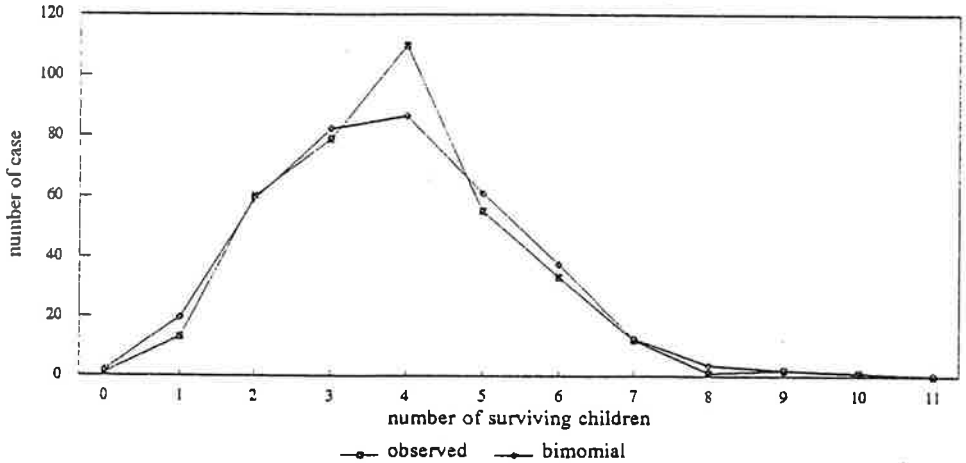
OBSERVED vs. BINOMIAL DISTRIBUTION



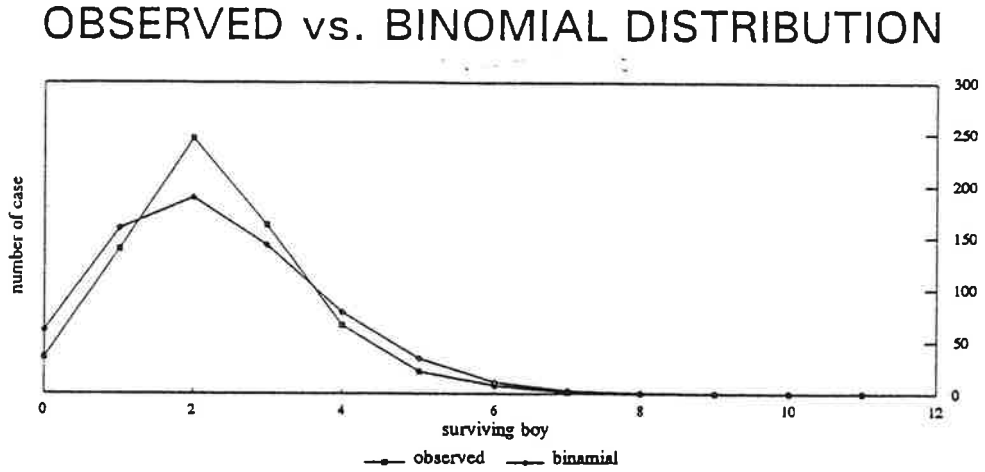
OBSERVED LANDLESS vs. BINOMIAL DISTR.

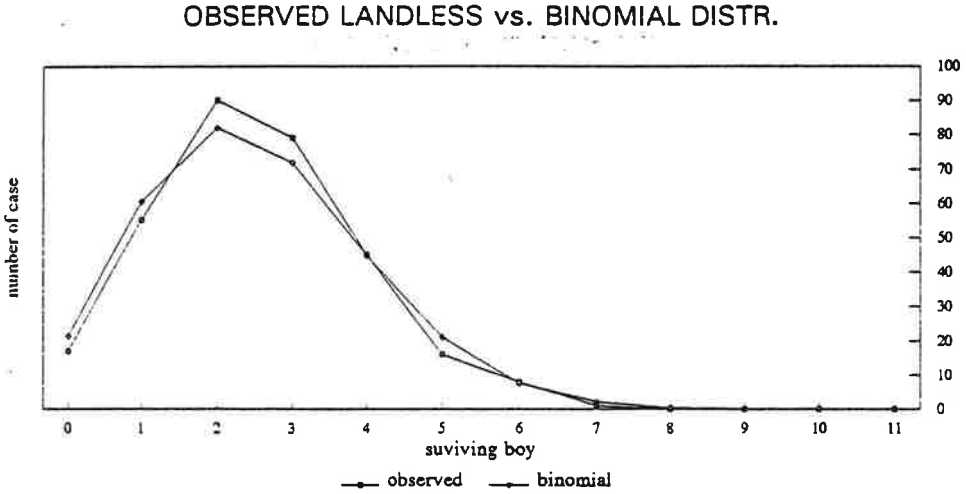


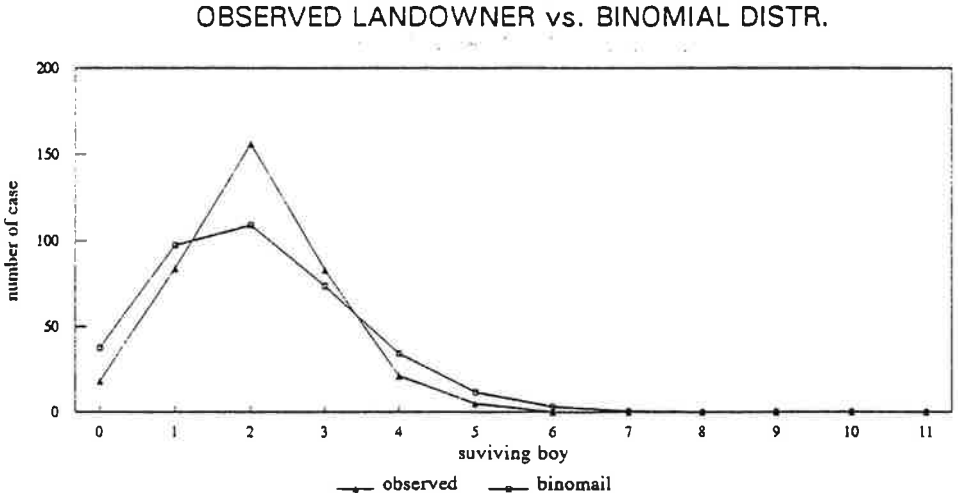
OBSERVED LANDOWNER vs. BINOMIAL DISTR.

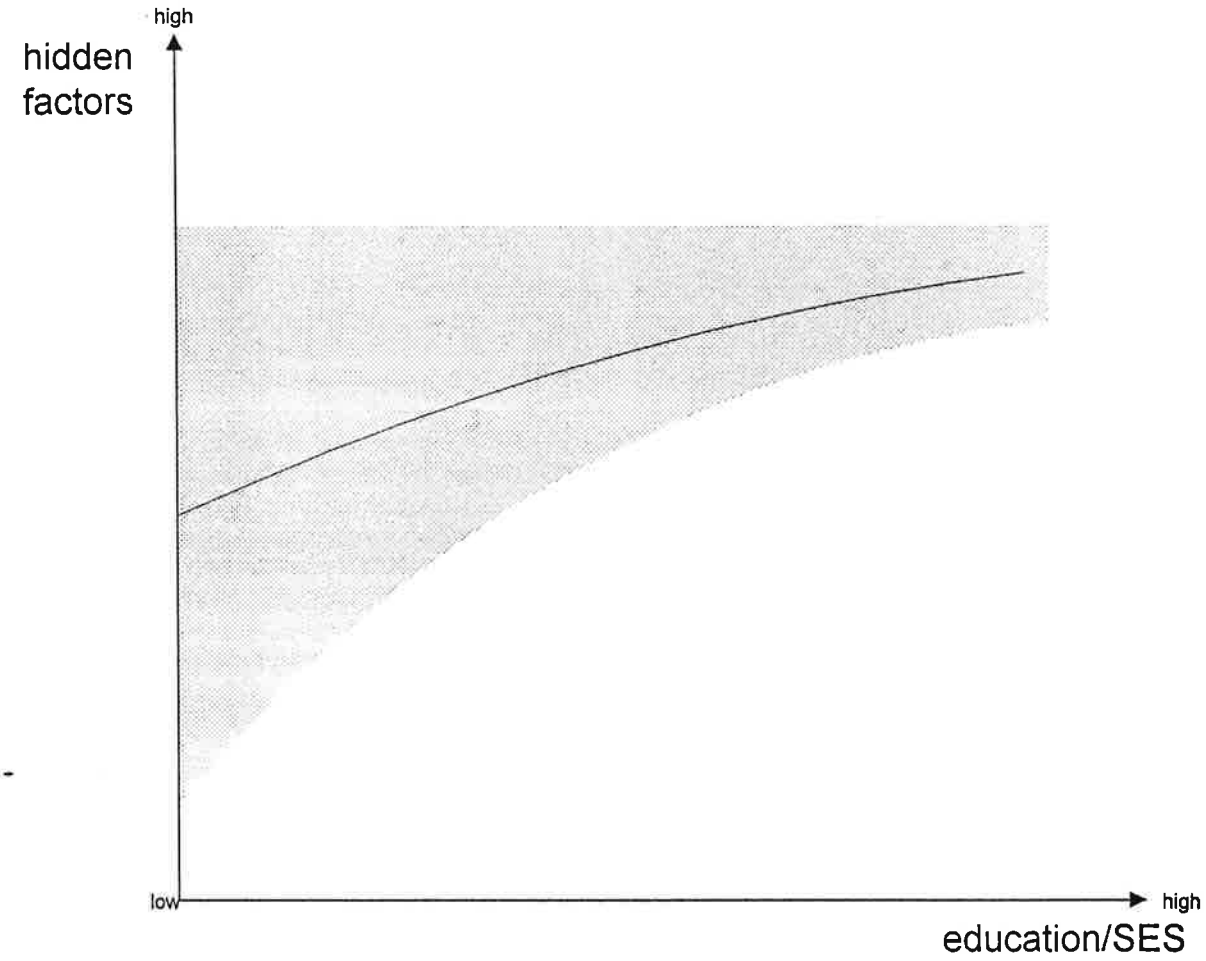


GRAPH 2 DISTRIBUTION OF SURVIVING SONS OF WOMEN AGED 40-49









Graph 3: Hypothetical scattergram of the reduction in variance of "hidden factors" underlying child mortality with improvement in education/socio-economic status.

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Notes

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2. See, for example, the data from Guinea-Bissau (Aaby 1991); Bangladesh (Welch 1974), India (Sapru et al. 1987); Kenya (Voorhoeve et al.1984); and nineteenth century Sweden (Imhof 1986).

3. See footnote 1.

4. For example, Spence et al.(1954) found differentials in familial risk of poor health status amongst children, although levels of child mortality were so low as not to show multiple deaths.

5. The last-born children are of higher parities and therefore *ceteris paribus* (if women were not targeting) may be susceptible to somewhat higher mortality than the average for all livebirths, if it is true as generally believed that higher parity births are at greater risk of dying. However, as is discussed below, the literature has perhaps exaggerated the intrinsic effect of parity on survival, because much of this relationship may be an artifact of a selection bias: namely, that higher-parity births are concentrated amongst mothers who carried a higher risk of child loss from the inception of their childbearing lives.

6. Disaggregating for age at death, the crossover from positive to negative deviance takes place at 2+ deaths, as opposed to 3+ deaths for the overall 0-59 month mortality.

7. This is why the observed distributions in the negative binomial analysis differ a little from those in the binomial analysis.

8. Susceptibility is not an easy thing to measure. At first sight, a simple proxy appears to be the death rate experienced by a particular family. But this measure has the following problem: a family with a single child, which died, would be branded with the same degree of susceptibility as one which lost all five children born to them. Clearly, the susceptibility of the latter is greater, given the larger number of data points.