Changing Neighbourhood and Infant Mortality in Rural India

by

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Abstract

Childhood mortality rates in India are concentrated along geographic and family lines. Five of twenty-six states account for more than half of the under-5 deaths in the country. Within states, a small fraction of villages account for most deaths. Within villages, some families experience multiple child deaths, while others experience none. This paper uses two waves of an all-India health survey to examine the factors (causal as well as correlated) that affect infant mortality and how this has changed over time.

The results show that the observed death clustering found in the data can be explained as one due to a causal mechanism (scarring) acting via the death of the previous sibling increasing the mortality risk of the index child only during the period 1986-1992. This effect is not present in the 1993-99 period. About 86-87% of families are estimated to have an elevated mortality risk due to a death of a previous sibling in infancy. This is in contrast to standard models that impose the restriction that such a scarring effect elevating the mortality risk is present in every household.

1. Introduction

India was the first country in the world to launch a family planning programme in the world.[1] She also had made health education and promotion an integral part of all national health and family welfare programmes since then, with successive 5-year plans (FYP) providing funding for planned development for nationwide improvement of health status of mothers and their children.[2] In spite of this, India still lags behind many developing countries in terms of a basic indicator of health, social and economic development of a country – the infant mortality rate. Of the 25.2 million children born in 2002 in India, 1.69 million died in infancy, before reaching their first birthday.[3] That is, around 7% of the children born in 2002 in India died before reaching their first birthday.

India had committed herself to reducing infant mortality rate (IMR) to 27 by 2015 (World Bank, 2004) by signing up to the Millennium Development Goals (MDG).[4] Due to her size and because she contributes nearly 25% of global child deaths, India occupies a crucial position in the global scenario of the MDG. The UN is actively monitoring progress towards the MDG. Given the current high rates of infant mortality in India, Policy makers and health planners are concerned about the issue of whether India is on track to meet the MDG target.[5] The importance placed by India in trying to reduce infant mortality deaths in order to meet the MDG target is also reflected in the current 10th 5-Year Plan (FYP) which has set a very ambitious goal of reducing IMR to 45/1000 by 2007 and 28/1000 live births by 2012.

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1 For example, see the Ministry of Health and Family Welfare of India website www.mohfw.nic.in.

2 Planning Commission of India website: www.planningcommission.nic.in; and also World Health Organisation, Country Health Profile for India, 2004.

3 UNICEF www.unicef.org/sowc04/files/Table1_english.xls.

4 Infant Mortality rate is measured as the number of children dying during the first 12 months per 1000 live births.

5 Claeson *et al.* (2000) argue that the rate of decline of IMR has slowed down in the period 1993-97 relative to 1981-92. According to the World Bank (2004) report, if the IMR decline experienced during the period 1971-2000 is maintained in the future, the IMR for India as a whole would only fall to a level of 46 instead of the target of 27 by 2015.

In order to achieve an overall reduction in IMR, it is important to recognise the high heterogeneity present in the incidence of infant mortality in India. Indian states differ significantly from each other with respect to demographic, social and economic indicators (Dreze and Sen, 1997) as well as the availability of health care infrastructure. It is therefore not surprising that infant mortality is concentrated in just a few states. The IMR of 68 for the country masks differences across states in India. For example, IMR was the lowest in the state of Kerala at 14 when it was highest at 98 in the State of Orissa in 2004 (World Bank, 2004).

There are also intra-state variations in IMR with rural areas exhibiting higher IMR relative to urban areas with a small fraction of villages accounting for most infant deaths. For example, around fifth of the villages experience around 50% of infant deaths (World Bank, 2004). The institutional structure in India flows downwards from the Department of Family Welfare of a state to district family welfare offices to primary health centres that are responsible for a group of villages and finally to the sub-centres at the village level. This contributes to inter-village differences within a state. The villages also differ in terms of provision of basic infrastructure – access to clean water and proper sanitation as well as electricity. The recognition of the role played by these factors in reducing infant mortality is not new (Leipziger, *et al*, 2003, van der Klaaw and Wang, 2006) and identifying the villages with high IMR is crucial (World Bank, 2004). Over 70% of the children are born to women living in rural areas. Thus, accounting for the availability of various health care facilities as well as other infrastructure is crucial in the identification of the correlates with the infant deaths for effective interventions required for reducing infant mortality.

Within villages, some families experience multiple infant deaths, while others experience none. Infant death clustering at the family level is well known now (see for example Arulampalam and Bhalotra (2006a, 2006b) and the references therein). The issue

here is whether the observed death clustering can be partly explained by a causal effect due to the death of a previous infant. A particular [causal] effect that has interested researchers operates through the death of a child modifying both birth spacing and birth spacing effects on the mortality risk of the subsequent child. The death of a child tends to shorten the time to the next birth because the mother stops breastfeeding and, thereby, is able to conceive sooner than otherwise before the mother is able to recuperate physiologically from the birth. This then elevates the mortality risk of the subsequent child. The mortality risk of the index child can also *fall* if there are learning effects or if there was an intervention (for example, increasing birth spacing voluntarily) to reduce the risk due to the death of the preceding child. If the family-level clustering in deaths that is observed in the data reflects such a causal process, then there are clear implications for policy such as that improving access to birth-spacing methods will reduce death clustering and overall mortality rates. Identification of this causal effect is important if there is such a process present.

The heterogeneities found at the state, village and family levels pose a major challenge to achieving the targets for health policy makers in meeting the overall MDG target for infant mortality. A better understanding of the determinants of infant mortality and the role played over time by these factors is crucial for the identification of strategies that can be effectively targeted at the most vulnerable group of people in order to achieve an overall reduction in infant mortality.

The main objective of this paper is to identify the factors at various levels (village, household and individual) that had contributed to the observed differences in infant mortality over time. The survey used is the National Family Health Survey (NFHS) of India. The survey collected information from mothers aged 13-49 at the time of the survey and therefore suffers from two problems. First, sample includes children born across four decades and thus does not constitute a representative sample of children born. For example,

among children born 20 years prior to the survey date, the data will only include mothers who were aged 29 or younger at the time of their birth. The second problem is that the sample information collected does not tell us where the mother was resident at the time of birth or death of the children born some years prior to the survey date. The analysis therefore uses two rounds of the survey and restricts to children born during a short time interval prior to the survey. The first survey (NFHS I) was carried out during the period 1992-93 and the second survey (NFHS II) during the period 1998-99.[6]

The paper also makes an additional methodological contribution to the issue of death clustering. To capture the causal effect due to the death of the previous sibling, an indicator for the survival of the previous child is included in the equation for mortality risk of the index child. This paper extends the standard specification which imposes a constant causal effect to allow for heterogeneous effect and the estimation technique used delivers an estimate of this 'scarring' effect for each household. The 'scarring' effects are empirically determined for each household and the methodology allows for identification of any positive learning effects associated with an infant death.

The data are described in Section 2, where descriptive statistics show a remarkable degree of death clustering amongst siblings. Section 3 sets out the econometric model and discusses estimation issues. The results are presented in section 4 and Section 5 concludes.

2. Data, Sample and Variables

In order to look at trends in infant mortality both rounds of the National Family Health Survey of India (NFHS-I and NFHS-II) are used. NFHS-I (NFHS-II) interviewed 89,777 (92,300) ever-married women aged 15-49 in 1992-93 (1998-99) and recorded complete

[6] The period between the two surveys saw the enactment of the 73rd constitutional amendment act 1992, which decentralised the provision of Family Welfare Programmes to the domain of the village administration in order to establish health infrastructure in rural areas (similar provisions were made for urban areas). However, unfortunately the data are not rich enough to identify how the village level health infrastructure had evolved during the latter 6 years due to the policy change.

fertility histories for the mothers amongst them, including the time and incidence of child deaths. NFHS-I (NFHS-II) was conducted in 25 (26) Indian states and accounts for 99 percent of India's population. For details on sampling strategy and context, see www.nfhsindia.org. The NFHS is one of a series of fairly comparable Demographic Health Surveys (DHS), available for about sixty-nine low and middle-income countries. The DHS surveys are freely available to researchers at www.measuredhs.com.

During the period 1988-1992, infant mortality (death before age 1) was 79 per 1000 live births (IIPS, 1995). Although India saw a decline of infant mortality rate to 68 during the five years preceding the second survey in 1998-99 (IIPS and ORC Macro, 2000), there has not been much research which had looked at the roles played by changing characteristics of the women and facilities available over time.

As discussed in the Introduction, one of the objectives of this paper is to look at the changing effects of neighbourhood and village health facilities on infant mortality. For this purpose, the village files provided with the NFHS data are used. The bias caused by the assumption that the mortality risk during the six years preceding the survey is related to factors observed at the time of the survey should not be high given the lack of mobility of households resident in villages and the slow evolvement of infrastructure in India.

Some useful summary statistics for the selected sample is provided in Table 1. In the data, approximately 3 in 4 families lived in rural areas and 3 in 4 children were born in rural areas. There is a significant rural/urban difference in the probability of infant death. For a child born during 1986-1992 in urban India, the probability of death was 0.077 and this reduced to 0.065 if the child was born during the next six years. However, in rural India, the probability of death was 0.113 during the period 1986-92, an increase of 3.6 percentage points for an urban born child. In rural India this was 9.53.

What about the death clustering found in the data? One way to measure death clustering is to define this as the difference in the conditional probabilities of infant death conditional on death and survival, respectively, of the preceding sibling. The two relevant probabilities are given in rows [5] and [6] for rural India. The difference of these two conditional probabilities which is defined as clustering is in row [7]. In spite of the slight slowing down of the unconditional probability of death over this sample period, the measure of death clustering has hardly changed over this period. Overall, the figures indicate a remarkable degree of death clustering. These, however, are simply the observed tendencies in the data. Estimation of the statistical model discussed below will allow one to disentangle clustering effects into correlated risks amongst siblings (inter-family heterogeneity) and, conditional upon this, a causal effect of the death of one sibling on the risk of death of the next sibling (scarring). [7] As discussed in the Introduction, a death of an infant can result in an increased mortality risk for the subsequent child. However, the mortality risk of the index child can also *fall* if there are learning effects or if there was an intervention (for example, increasing birth spacing voluntarily) to reduce the risk due to the death of the preceding child. Since the data are not rich enough for the analysis presented here to allow for endogenous interventions, the standard model is generalised to allow for the possibility of positive or negative effect from the death of the previous child on the mortality risk of the index child.

3. Econometric Model

The models used for the analysis belong to the general class of dynamic random effects probit. The first model, named **Model 1**, is the conventional model that allows for random family/woman specific factors in the intercept; the second model, named **Model 2,**

[7] See Arulampalam and Bhalotra (2006a) for a discussion of various possible reasons for scarring in the context of infant mortality.

additionally allows the death of a previous child in infancy on the index child to be specific to the family/woman.

Let there be n_i children in family *i.* For child *j* ($j=2,\ldots,n_i$) in family *i* ($i=1,2,\ldots, N$), the probability that the child will die in infancy is modelled as a random effects panel probit. That is

$$Prob(y_{ij}=1) = \Phi[\, \boldsymbol{x_{ij}}'\beta + \gamma\, y_{ij-1} + \alpha_i\,] \qquad (1)$$

where Φ is the cdf of a standard normal distribution and $y_{ij}=1$ if infant *j* dies in family *i.* A random intercept α_i, is included to account for family-specific unobserved characteristics. This picks up any correlation of death risks among siblings. The model also includes the *observed* survival status of the previous sibling, y_{ij-1}, the coefficient on which picks up scarring. $\boldsymbol{x}$ is a vector of strictly exogenous observable child specific characteristics that influence infant death and β is the vector of coefficients associated with $\boldsymbol{x}$. Family or neighbourhood (village) specific variables are not included in $\boldsymbol{x}$**.** The parameters of the child-specific covariates β are first estimated using maximum likelihood under the assumption that $\alpha_i \sim N(\alpha,\sigma^2_\alpha)$; then Bayes shrinkage estimate of α_i are estimated; and finally, estimated α_i are regressed on a set of characteristics that are common across all children. This model is named **Model 1.** The details of this method of estimating α_i is discussed below after discussing the issue of 'initial conditions problem' and the method of dealing with it in this context.

Initial Conditions Problem

In the survey, women aged 15-49 in 1992/93 and 1998/99 were interviewed and retrospective data on their birth histories were collected. A well-recognised problem with retrospective data, when an age cut-off is used to select the interviewees, is the selectivity issue. The interviewees may be a representative sample as of the survey date, but will not be

so for earlier years (Rindfuss, *et. al.*, 1982). For this reason and for reasons of recall bias, only information on children born within six years of the survey is used providing two non-overlapping samples in terms of calendar years. This left truncation of the data by calendar time occurs at different points in the birth history of different households, creating complications due to the fact that the start of the sample does not coincide with the start of the stochastic process under study. This is a well recognised initial condition problem (e.g. Heckman, 1981; Wooldridge, 2002). The problem arises essentially because $y_{ij\text{-}1}$ and α_i are necessarily correlated and the model is recursive and the left truncation results in an endogenously truncated sample. Heckman (1981) was the first one to propose a method for correcting for selection bias caused by left truncation in these models. Unfortunately, the application of this technique requires specially written software. There are two other techniques currently available (Orme, 1997, 2002; Wooldridge, 2002) which are easily implemented using commercially available standard software. However, these two techniques only use information from the second child onwards as the analysis is conditioned on the first observation. An observation window of six years is not wide enough to provide us with many women with more than three children in the sample: a woman needs to have at least three children during the six year period in order to contribute at least two observations to the estimation. The paper therefore uses Heckman's method to account for the 'initial condition' problem.

Given the above assumptions and the left-truncated sample, and dropping $\boldsymbol{x}$ and the index i for convenience, the joint probability of the observed sequence of binary outcomes, conditional on y_1 and α is

$$P(y_n,\ldots,y_2 \mid y_1, \alpha)=P(y_n|y_{n-1}, \alpha)\, P(y_{n-1}|y_{n-2}, \alpha)\ldots P(y_2|y_1, \alpha) \qquad (2)$$

where y_1 now refers to the first observation in the sample for family i rather than the first child for this family. Although y_1 is observable, α is not. An assumption regarding $P(y_1,\alpha)$ is

now required. Note, if there were no unobserved heterogeneity α_i , then the initial condition y_1 could be treated as exogenous, and the model given by equation (1) could be estimated using the sample of children j ($j=2,\ldots,n$). Alternatively, even in a dynamic model that incorporates unobserved heterogeneity, the initial conditions problem is avoided if the time dimension of the panel (n_i) is large (Hsiao (1986), pp170.). However, n_i in the model is given by the number of births of mother i observed during the sample period, and this cannot be assumed to tend to infinity. As a result, consistent estimation requires endogenisation of the initial condition. Note, in the absence of guidance from economic theory, there are no *a priori* reasons to expect one method to be superior to the other.

Heckman's approach

This is the oldest suggestion for dealing with the initial conditions problem. Since one requires $P(y_1|\alpha)$ in $P(y_n,\ldots,y_2,y_1|\alpha) = P(y_n,\ldots,y_2|y_1,\alpha)\ P(y_1|\alpha)$ prior to marginalising with respect to the unobserved α, Heckman suggested that one could use an approximation to model the process generating the first observation (y_1) in the sample using the same form of the equation used for the rest of the observations but with some restrictions. That is assume,

$$P(y_{i1}=1|\alpha_i) = \Phi[\mathbf{z_i}'\boldsymbol{\lambda} + \theta\ \alpha_i] \qquad i=1,\ldots,N \qquad (3)$$

where $\mathbf{z_i}$ is a vector of exogenous covariates. In principle, the vector of covariates in $\boldsymbol{x}$ and $\boldsymbol{z}$ need not be the same, and θ need not equal one. Equations (1) and (3) together specify a complete model for the infant survival process. Assuming a particular distribution for the unobservable α one can marginalise the likelihood with respect to this α. A conventional assumption for the distribution of α is the Normal distribution. Heckman provides some simulation results to show that this approximation works relatively well.

In this model, the contribution to the likelihood function for family i is given by

$$L_i = \int \left(\Phi\left[\left(\mathbf{z_{i1}}'\boldsymbol{\lambda} + \theta\alpha_i\right)\left(2y_{ij}-1\right)\right] \prod_{j=2}^{n_i} \Phi\left[\left(\mathbf{x_{ij}}'\boldsymbol{\beta} + \gamma y_{ij-1} + \alpha_i\right)\left(2y_{ij}-1\right)\right] \right) f(\alpha_i)\, d\alpha_i \qquad (4)$$

where $f(\alpha)$ is the probability density function of the unobservable family-specific heterogeneity.

Estimation of the random effect α_i

α_i is the unobserved individual specific random effects in the model. Then, by Bayes' theorem,

$$f(\alpha_i \mid y_{i1},...,y_{in_i}) = \frac{f(y_{i1},...,y_{in_i} \mid \alpha_i) f(\alpha_i)}{f(y_{i1},...,y_{in_i})} \quad (8)$$

Thus, $E(\alpha_i \mid y_{i1},..,y_{in_i}) = \int \alpha_i f(\alpha_i \mid y_{i1},..,y_{in_i}) d\alpha_i = \frac{\int \alpha_i f(y_{i1},...,y_{in_i} \mid \alpha_i) f(\alpha_i) d\alpha_i}{f(y_{i1},...,y_{in_i})}$ (9)

$f(y_{i1},...,y_{in_i} \mid \alpha_i)$ is the conditional likelihood and $f(y_{i1},...,y_{in_i})$ is the marginal likelihood which are obtained during the maximisation of the likelihood function. The estimated $E\left(\alpha_i \mid y_{i1},...,y_{in_i}\right)$ is known as the Bayesian shrinkage estimate (Goldstein, 2003). These estimates are then regressed on a set of child-invariant factors such as religion, caste, education of the mother and father and some village level variables. The main advantage of this procedure compared to the usual procedure of entering these factors directly in equation (1) is that one is able to identify patterns of clustering given the pattern of infant deaths in that particular family as the estimates condition on the observed sequence $y_{i1}, y_{i2},..,y_{in}$.

Model 2: The previous conventional model is generalised by allowing the effects of the death of the previous child to be heterogeneous across families. Whether this effect is positive (i.e. a genuine 'scarring' effect) or negative (i.e. there are some positive learning) will be empirically determined in this model. Equation (1) becomes

$$Prob(y_{ij}=1) = \Phi[\, \boldsymbol{x_{ij}}'\boldsymbol{\beta} + \gamma_i y_{ij-1} + \alpha_i \,] \quad (10)$$

Additionally, assume $\begin{pmatrix} \alpha_i \\ \gamma_i \end{pmatrix} \sim N\left(\begin{pmatrix} \alpha \\ \gamma \end{pmatrix}, \begin{pmatrix} \sigma_\alpha^2 & \sigma_{\alpha\beta} \\ \sigma_{\alpha\beta} & \sigma_\beta^2 \end{pmatrix}\right)$ (11)

This model allows for heterogeneous intercept as well as heterogeneous scarring effect. The same two-step procedure discussed earlier is used to obtain the correlates between the unobserved and observable family specific factors.

4. Results

The dependent variable and the survival status of the preceding child were both coded as binary variables that are unity if the child dies before the age of 12 months and zero otherwise. Children who were younger than 12 months at the time of the survey were dropped from the sample because they had not had 12 months of exposure to mortality risk. Mothers who had multiple births during the sample periods are also excluded.

Child-specific regressors in the model include a dummy for the child born during the first half of the sample period, the birth order of the index child, gender, and the age of the mother at birth of the index child. The latter is expected to capture effects of the physiological condition of the mother at the relevant time. All of the above regressors were also included in the equation for the first observation. The mother's age at birth of each of her children was averaged over all children and was additionally included in this equation along with its square. . In the second stage regression where the estimated random effects were regressed on family-specific covariates, indicators for the educational attainment of each of the mother and father, religion, caste, village facilities and various neighbourhood characteristics were included. These are discussed later.

The model estimates are provided in Table 2. The first panel in Table 2 presents results for 1992/3 and the second for 1998/9. Model 1 (Column [1])) only allows for heterogeneous intercept and Model 2 (Column [2]) allows for both heterogeneous intercept as well as heterogeneous 'scarring'.

First note that the results are very similar across the two specifications of the model (Models 1 and 2). The test for exogeneity of the survival status of the previous sibling is given by a test of $\theta=0$. This is rejected only in the model that imposes homogeneity of scarring effect (Model 1) stressing the importance of addressing this issue. The scarring effect is only significant in the 92/93 sample and not in the 1998/99 sample. However, the estimated ρ which is the variance of the unobserved family-specific error variance as a proportion of total variance, is very much reduced in the 1992/93 sample. In fact it is only significant at 10%. In the 1998/99 sample, about 18% of total variance is attributed to the unobserved family specific random error. The main cause of the observed clustering of deaths in the raw data seems to be due to the causal effect of scarring in the 1992/93 sample but unobserved factors common across siblings in the 1998/99 sample. The calculated marginal effects imply that among children born during the 1986-1992, the probability of a child dying in infancy was 5.5 percentage points higher for the index child if the preceding child had died in infancy compared to when the preceding child survived (Table 3 Column [1]). Although this effect is estimated to be 0.043 in the second sample, the scarring coefficient was not significantly different from zero.

Children born to younger mothers are exposed to higher probability of death, *ceteris paribus*. We also find that infant mortality rates are coming down over time. For example, in the 1992/93 sample, children born during 1986-89 period had a probability of death in infancy which was 1.8 percentage points higher compared to those children born during 1990-92 period, *ceteris paribus*. The figure for the second sample was 3.3 implying a larger drop in infant mortality in the first sample relative to the second sample. Relative to the first born, the other children face a smaller risk of death in infancy *ceteris paribus*.

A plot of the kernel density of various estimated family specific effects α (from Model 1 and 2) and the woman-specific scarring effects (from Model 2), are provided in

Figures 1 to 3. Note, these plots are not the plot of the unconditional distribution of alpha which one would find in the population, but a plot of the estimated expected value of alpha for each family that conditions on the observed outcomes for each woman in the sample. Infant death is a rare event. A large positive value of α in Model 1 implies a large probability of death. Hence, one would expect to find estimated α to be greater than zero only for a small number of families.

The correlation between the estimated family/woman specific heterogeneity in the two models is 0.754 in the first sample and 0.960 in the second sample. The high correlation of 0.96 is not surprising since the scarring effect was found to be insignificant in the second sample and thus allowing for scarring heterogeneity does not make a lot of difference.

The estimated correlation between the two heterogeneity components in Model 2 is -1.00 in the first sample and -0.855 in the second sample. Persistence found in infant mortality in families may be due to measured as well as unmeasured family heterogeneity or due to genuine scarring. Hence, one would expect to find a negative correlation between the two estimated heterogeneity components in Model 2.

Finally, about 86-87% of families are estimated to have an elevated mortality risk due to a death of a previous sibling in infancy. This is in contrast to standard models that impose the restriction that such a scarring effect elevating the mortality risk is present in every household.

Second Stage Estimates

The second stage regressions of the estimated heterogeneity effect on various child invariant covariates are provided in Table 3. Demographic characteristics, household facilities, village characteristics and village health facilities variables are included in this regression. In addition, regional dummies; North (Rajasthan, Haryana, Punjab), East (Orissa, Bihar, West Bengal), West (Gujarat, Maharashtra), Central (Madhya Pradesh, Uttar Pradesh), and South

(Andhra Pradesh, Karnataka, Tamil Nadu, Kerala) are also included. We discuss the effects from Model 1 first before discussing the results from Model 2.

Children born to mothers who live in the Northern, Eastern and Central regions face a higher mortality risk relative to children born in the South, *ceteris paribus*. This regional difference persists over time except in the Eastern region. Children born into non-hindu families face a lower mortality risk. This effect has been consistently observed in other research too (Arulampalam and Bhalotra, 2006a and 2006b).

Since independence, India has consciously pursued affirmative action policies to provide preferential treatment to historically economically and socially backward scheduled castes and tribes (Bajpai, 2003). The higher risk faced by children born into scheduled tribe groups found in the first sample is not present in the second sample.

Both maternal as well as paternal education is found to be strongly negatively associated with infant mortality risk. There has been a small increase in the female literacy rate during the observation period of about 8 percentage points from a very low level of 29% in the 1986-92 sample.

One of the keys to economic development is better infrastructure. There was only a very small increase in the proportion of households who had access to electricity, from 40% in the first sample to 47% in the second sample. Access to electricity is estimated to reduce the mortality risk in both samples. As expected, access to proper sanitation facility is found to reduce the mortality risk. 93% of women in the second sample reported as having access to water from a tap, well or a pump. However, there was no significant effect from having access to water.

Beside the standard demographic indicators the analysis also included some neighbourhood characteristics. The 'community' or the 'neighbourhood' is taken to be the village in which the woman lives. These were all calculated prior to the selection of children

born in the 6 years preceding the surveys. The neighbourhood characteristics included were: proportion of educated women in the village, proportion of women ever used contraceptives, proportion of women sterilised and the average number of children born to a woman.

Women who live in villages where average educational level is relatively high or where there is small family norm, may have lower fertility than they would otherwise have (Kravdal, 2002 and 2004). Short birth spacing has been shown to increase mortality (Hobcraft, 1992). Preceding birth interval is not included in the model due to the endogeneity of this variable.[8] Given the absence of information on the contraceptive availability, the proportion of women ever used contraceptives and the proportion of women sterilised were included. Children born to women who live in villages where the fertility norm is relatively high are found to face a higher mortality risk in both samples. Surprisingly, higher proportion of educated women in the village is positively associated with increased mortality risk.

Variables reflecting access to health care were included in the analysis. These were indicator variables for different types of health care facilities available in the village. The presence of a health clinic or a dispensary is associated with a lower mortality risk. These presumably provide contraceptive advice and could be the reason for the negative association.

Another surprising result is the effect found for villages that have access to an all weather road. This was found to be associated with a high mortality risk in the first sample.

Next turn to the estimates given in [2] to [3] for the two samples. These refer to Model 2 results where each woman is allowed to have a different scarring parameter. The interesting aspect of this model specification is that instead of estimating an average scarring

[8] See Bhalotra and van Soest for a structural model that accounts for the presence of preceding birth interval in the neonatal mortality risk equation.

parameter for every family in India, this model allows for families to be able to reduce the scarring through learning effects. If the previous child had died in infancy, then the two effects from [2] and [3] will have to be added to evaluate the total effect on the mortality risk of the index child. This is provided in column [4]. No surprisingly, covariates are found to have opposite effects on the two estimated heterogeneity variables. As before a positive effect of a covariate implies an elevated mortality risk for the index child, *ceteris paribus*.

There are some very interesting results. Eastern and Central regions which were found to do badly relative to the Southern region now are found to be doing very well. The estimated scarring effects for families living in the Eastern and Central regions is now very large and negative implying some sort of interventions taking place to reduce the mortality risk in families that have seen a death of an infant. However, this differential effect across regions is not present in the second sample.

Children born to either a Hindu or a Christian family are now found to have very high scarring parameter in the first sample. The overall effect due to a death of an infant in these families results in an increased mortality risk to the next child. However, this is reversed in the second sample.

Scheduled caste families do badly in both samples when there is an infant death in the family.

Scarring parameter is found to be positively associated with paternal education and dominating the negative effect of the family unobserved characteristic. However, this is reversed in the second sample.

The household and village infrastructure variables that were found to play a significant role in families who had experienced an infant death in the first sample are found to have almost no effect in the second sample. This is also the same with the village health facilities.

6. Conclusions

Given the slow progress shown towards meeting one of the Millennium Development Goal target of reducing child mortality by two thirds by 2015 from its level in 1990, the Government of India had been continuously implementing various health policies recently. This paper uses two rounds of the National Family Health Surveys for India, in order to identify the effects of various demographic, socio-economic, neighbourhood characteristics as well available health facilities on infant mortality over time to shed some light on why the rate of decline of infant mortality observed in the 70s and 80s has slowed down.

Estimation results show that the observed death clustering found in the data can be explained as one due to a causal mechanism acting via the death of the previous sibling increasing the mortality risk of the index child only during the period 1986-1992. This effect is not present in the 1993-99 period. About 86-87% of families are estimated to have an elevated mortality risk due to a death of a previous sibling in infancy. This is in contrast to standard models that impose the restriction that such a scarring effect elevating the mortality risk is present in every household.

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1992-93 Sample

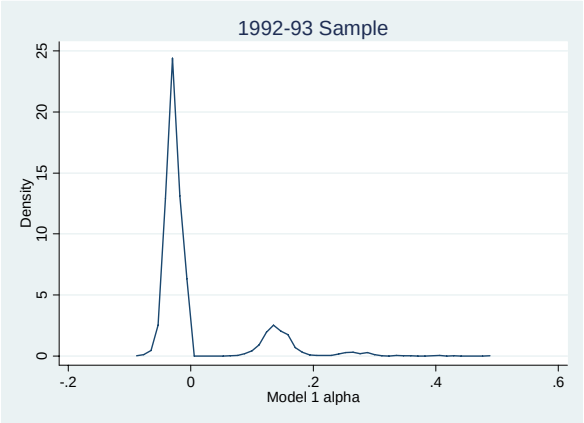

Figure 1a

1998-99 Sample

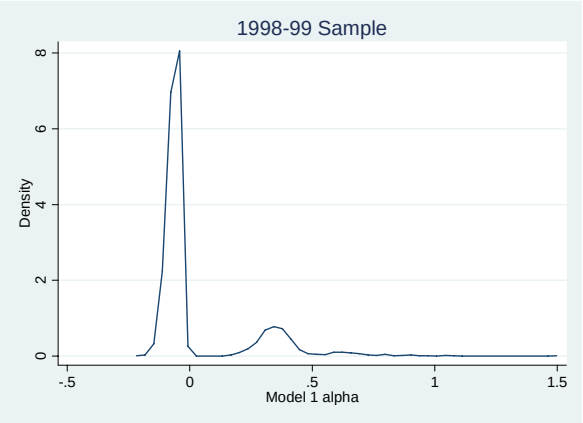

Figure 1b

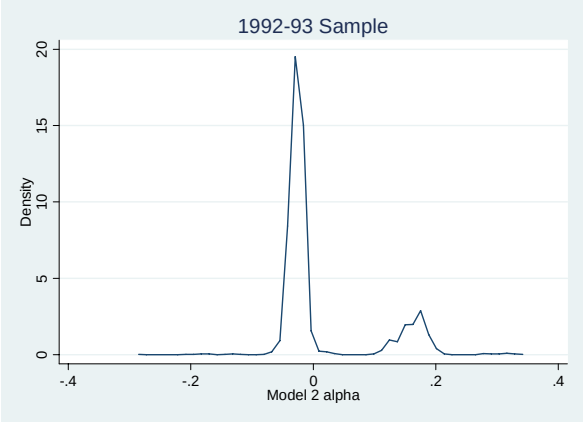

Figure 2a

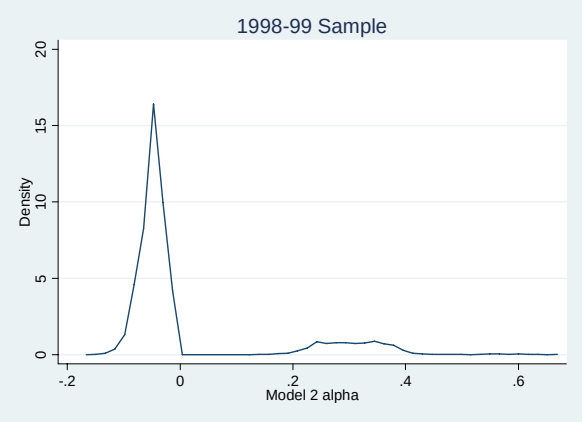

Figure 2b

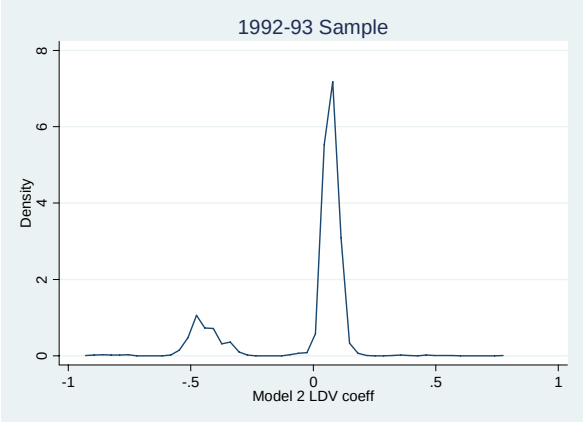

Figure 3a

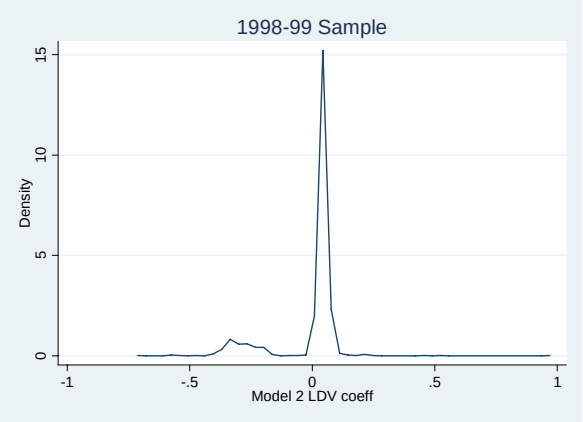

Figure 3b

Table 1

		1992-93	1998-99
[1]	% women living in rural India	72.29	70.30
[2]	% of children born in Rural India	74.19	72.76
[3]	Rural: Probability of infant death x 100	11.34	9.53
[4]	Urban: Probability of infant death x 100	7.73	6.45
[5]	Rural: Prob of death given previous sibling's death x100	24.47	23.17
[6]	Rural: Prob of death given previous sibling's survival x100	9.00	7.76
[7]	Rural: Death clustering { [5]-[6] }	15.47	15.41
[8]	Rural: Total number of children in the sample	44,580	46,179
[9]	Rural: Total number of mothers in the sample	26,519	26,886
[10]	Rural: % first born	24.54	24.72
[11]	Rural: % second born	22.35	23.64
[12]	Rural: % third born	18.10	18.24
[13]	Rural: % Mothers aged 18 or less at the beginning of the sample	22.91	17.30
[14]	Rural: % Mothers aged 19-20 at the beginning of the sample	16.35	15.18
[15]	Rural: % Mothers aged 21-25 at the beginning of the sample	30.76	35.69
[16]	Rural: % Mothers aged 26-30 at the beginning of the sample	16.83	19.30
[17]	Rural: % Mothers with first born during the sample period	41.26	42.45
[18]	Rural: % Mothers with one child in the sample period	47.88	46.08
[19]	Rural: % Mothers with two children in the sample period	37.84	38.19
[20]	Rural: % Mothers with three children in the sample period	12.68	13.78

Notes: (i) The above figures refer to children born during the preceding 6 years from the survey year, 1986-1992 and 1992-1998.

Table 2 – Maximum Likelihood Estimates for Model of Infant Mortality

	1992/93		1998/99	
	Model 1	Model 2	Model 1	Model 2
	[1]	[2]	[1]	[2]
Main equation variables				
female	0.043	0.044	0.063**	0.063**
Birth order – second or third	-0.335***	-0.333***	-0.424***	-0.416***
fourth or higher	-0.233**	-0.235**	-0.169	-0.167
Child's birth year is first half	0.246***	0.255***	0.353***	0.349***
Age at birth of mother of index child	-0.093***	-0.091***	-0.137***	-0.132***
Age at birth of mother of index child squared /1000	1.669***	1.648***	2.261***	2.182***
Previous child died – SCARRING (marginal effect	0.328*** (0.050)	0.653***	0.077 (0.008)	0.300
Intercept	-0.124	-0.155	0.435**	0.419*
Prop of women with positive est'd unobserved characteristic	0.154	0.142	0.144	0.144
Prop of women with positive est'd scarring effect		0.859		0.869
σ^2_α variance of unobserved family level heterogeneity (s.e)	0.093 (0.060)	0.084 (0.047)	0.265 (0.073)	0.179 (0.120)
ρ proportion of σ^2_α in total variance				
σ^2_γ variance of unobserved scarring heterogeneity (s.e)		0.617 (0.235)		0.431 (0.259)
Covariance between α and γ (see text for details)		-0.227 (0.097)		-0.147 (0.121)
Correlation between α and γ (see text for details)		-0.999		-0.531
Maximised Value of the Log Likelihood	-11,998.41	-11994.86	-11,485.00	-11483.67
Number of Mothers	26,519	26,519	26,886	26,886
Number of Observations	44,580	44,580	46,179	46,179

Notes: (i) Sample used is all children born during the preceding six years of the survey; (ii) Column [1] refers to the model with random intercept and [2] to the model with random intercept as well as random coefficient on the survival status of the previous sibling. Initial conditions problem is addressed using Heckman's method. (iv) ***,**, * coefficient significant at 1%, 5% and 10% respectively.

Table 3 – Regression of estimated unobserved heterogeneity (coefficient x 100)

	1992/93					1998/99				
	Dependent Variable					Dependent Variable				
	Mean	Intercept heterog Model 1	Intercept heterog Model 2	Scarring heterog Model 2		Mean	Intercept heterog Model 1	Intercept heterog Model 2	Intercept heterog Model 2	
	[1]	[2]	[3]	[4]	[3] + [4]	[1]	[2]	[3]	[4]	[3]+[4]
Region – base South	0.18					0.15				
North	0.15	0.165	0.185	-0.501	-0.317	0.16	0.629*	0.582**	-0.567**	0.014
East	0.21	0.407***	0.402***	-1.092***	-0.690***	0.23	-0.299	-0.162	0.019	-0.143
West	0.07	-0.037	-0.055	0.149	0.094	0.06	0.097	0.157	-0.204	-0.047
Central	0.26	0.855***	0.922***	-2.506***	-1.584***	0.23	0.914**	0.861***	-0.853***	0.008
North-East	0.13	-0.327*	-0.266	0.723	0.457	0.17	-0.439	-0.307	0.086	-0.221
Religion –Base Hindu	0.76					0.78				
Muslim	0.11	-0.431***	-0.396***	1.076***	0.680***	0.11	-1.062***	-0.809***	0.543**	-0.266***
Other	0.12	-0.630***	-0.494***	1.342***	0.848***	0.11	-0.584*	-0.369	0.058	-0.311***
Caste	0.70					0.41				
Scheduled Caste	0.17	-0.183	-0.213*	0.578*	0.365*	0.19	0.369	0.158	0.164	0.322***
Scheduled Tribe	0.13	0.306**	0.12	-0.325	-0.206	0.19	-0.043	-0.074	0.086	0.012
Other Backward Caste						0.31	0.281	0.193	-0.104	0.089
Parental Education										
Mother – some education	0.29	-0.171*	-0.057	0.155	0.098	0.37	-0.756***	-0.565***	0.380**	-0.184**
Father – some education	0.58	-0.402***	-0.308***	0.836***	0.529***	0.66	-0.698***	-0.582***	0.443***	-0.139*
Household facilities										
House is pucca or semi pucca	0.39	-0.247***	-0.254***	0.690***	0.436***	0.56	-0.13	-0.086	0.039	-0.046
Has electricity	0.40	-0.254***	-0.294***	0.799***	0.505***	0.47	-0.594***	-0.461**	0.308*	-0.152*
No electricity (village with electricity)	0.20	-0.077	-0.114	0.309	0.195	0.18	-1.121	-0.738	0.341	-0.397
Has access to water (not from river)	0.07	-0.076	-0.056	0.152	0.096	0.93	0.282	0.199	-0.162	0.036
Has access to a toilet	0.18	-0.222**	-0.282***	0.766***	0.484***	0.23	-0.577**	-0.515***	0.492***	-0.023
Neighbourhood (village) Characteristics										
Proportion of educated women	0.26	0.787***	1.036***	-2.816***	-1.780***	0.34	1.258**	0.924*	-0.553	0.371*
Prop of women ever used contraceptives	0.40	-0.722	-1.329***	3.612***	2.283***	0.47	-0.12	-0.27	0.281	0.011
Average number of children/family	3.76	0.262***	0.235***	-0.639***	-0.404***	3.63	0.384**	0.309**	-0.235*	0.074
Prop women sterilised	0.30	0.141	0.665	-1.808	-1.143	0.34	-1.41	-0.938	0.626	-0.313

Table 3 Continued

	1992/93					1998/99				
	Dependent Variable					Dependent Variable				
	Mean	Intercept heterog Model 1	Intercept heterog Model 2	Scarring heterog Model 2		Mean	Intercept heterog Model 1	Intercept heterog Model 2	Intercept heterog Model 2	
	[1]	[2]	[3]	[4]	[3] + [4]	[1]	[2]	[3]	[4]	[3]+[4]
Village Health Facilities										
Primary Health Centre	0.12	-0.205	-0.153	0.417	0.263	0.13	-0.186	-0.159	0.135	-0.024
Sub Centre	0.30	0.02	0.094	-0.254	-0.161	0.30	0.033	0.067	-0.144	-0.077
Community Health Centre						0.05	-0.226	-0.204	0.095	-0.109
Hospital	0.15	-0.185	-0.259**	0.705**	0.446**	0.07	0.002	-0.02	0.08	0.06
Dispensary/clinic	0.33	-0.174*	-0.162*	0.441*	0.278*	0.96	-0.07	-0.065	0.062	-0.003
Village Health Guide	0.39	-0.077	-0.111	0.302	0.191	0.33	-0.236	-0.199	0.223	0.024
Birth Attendant	0.46	-0.001	-0.025	0.067	0.042	0.57	0.033	0.006	0.024	0.03
NGO Family Planning Health Clinic	0.17	0.222*	0.114	-0.309	-0.196					
Mobile Health Unit	0.16	-0.113	-0.078	0.211	0.134	0.11	0.027	0.116	-0.153	-0.038
Village Infrastructure										
Has electricity	0.78	-0.082	-0.118	0.32	0.203	0.80	-1.08	-0.712	0.336	-0.376
Has an all weather road	0.50	0.192**	0.168*	-0.457*	-0.289*	0.77	0.263	0.276*	-0.253*	0.023
Intercept		-0.431	-0.344	0.934	0.591		0.558	0.262	0.084	0.346
Number of Observations	26,519					26,886				
R-squared		0.02	0.02	0.02	0.02		0.01	0.01	0.01	0.00

Notes: (i) * significant at 10%; ** significant at 5%; *** significant at 1%.